DEATH DUE TO ANAESTHESIA -

ITS INCIDENCE AND SOME ASSOCIATED FACTORS.

by

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TO

MARY

AND TO

THE MEMORY OF

MY LATE PARENTS

ACKNOWLEDGMENTS.

I wish to express my gratitude to the present Head of the Department of Anaesthetics of the University of Cape Town, Professor A.B. Bull, and to his predecessor, Dr. C.S. Jones - now of the University of New South Wales, for their guidance, help and support in this project.

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1) The Council for Scientific and Industrial Research

and

2) The Anglo-American Corporation and De Beers Consolidated Mines Anaesthetic Research Fund.

The former provided the major grant which enabled the initiation of this survey and the completion of the first or pilot period. Grants from the latter provided assistance essential to the documentation of the continuing period of this survey to date. I wish to acknowledge my gratitude to both these donors.

I wish to acknowledge the help and cooperation I have received from successive Medical Superintendents of Groote Schuur Hospital, Dr. B. de Wet, Dr. N. Cloete and Dr. J.G. Burgher, which made possible the establishment of the means by which I am notified of all post-operative deaths occurring in the hospital.

Dr. L.S. Smith, the State Pathologist, and his predecessor, Professor R. Turner, willingly allowed me access to the records of autopsy examinations conducted by their Department, a priviledge accorded me also by the Department of Pathology of the University of Cape Town. I wish to acknowledge the very essential help I received from both these sources.

The persons most involved in the conduct of this survey are undoubtedly members of the staff of the Department of

Anaesthetics of Groote Schuur Hospital between the years 1956 to 1965. These have provided the grist for the mill, the forum for discussion and have always willingly provided me with the clinical information I sought. To them I acknowledge my gratitude. Without their help and willing cooperation this survey would have been completely impossible.

Lastly I wish to thank my wife for the immense help she has given me in the typing and compilation of this script.

CONTENTS.

CHAPTER 1.

RAISON D'ETRE.

On the 16th October, 1846, at the Massachusettes General Hospital the successful use of general anaesthesia for a surgical operation was publicly demonstrated for the first time.[109, 162] The state of anaesthesia was produced by the inhalation of the vapour of diethyl ether. Here indeed was the anodyne, the lack of which had barred any really significant advance in surgery. John Collins Warren, the surgeon concerned in this successful demonstration of general anaesthesia by William Morton, could write subsequently with justifiable enthusiasm "If Ambroise Paré and Louis and Dessault and Cheseldin and Hunter and Cooper could see what our eyes daily witness how they would long to come among us and perform their exploits once more".[147] After witnessing the first major operation to be performed under general anaesthesia - the amputation of a leg - Henry Bigelow, one of the spectators present also at Morton's first demonstration, exclaimed with prophetic insight "I have seen something today that will go round the world".[64] Indeed, once demonstrated, the knowledge and use of this discovery, fulfilling as it did so great a need, spread with amazing rapidity around the civilized world. Just over two months later operations under general anaesthesia were performed in England in places as far apart as Dumfrieshire and London.[175] We have records of the use of ether anaesthesia here in Cape Town, within six months of Morton's public demonstration, by a dentist, a Mr. Raymond, for dental extraction.[155] The first major surgical operation in South

Africa under general anaesthesia by the inhalation of ether vapour - the amputation of a leg - was performed by Dr. W.G. Atherstone in Grahamstown on the 16th June, 1847, just eight months after the Boston demonstration.[75]

It soon became apparent, however, that this great boon to mankind was a mixed blessing. As early as 1847 reports of deaths occurring during or shortly after the administration of general anaesthesia with ether vapour had appeared in America, Europe and England.[28] [100] From accounts some of these deaths seem to have been asphyxial while with others the inhalation of ether vapour appears to have had little causative association. To these were soon added reports of the apparently unpredictable, almost whimsical, sudden deaths which occurred during the induction of anaesthesia with chloroform. In his monograph 'On Chloroform and other Anaesthetics' published in 1858, John Snow includes reports of no fewer than fifty deaths associated with chloroform anaesthesia.[164]

The first reported, and probably best known of these deaths associated with chloroform anaesthesia, that of Hannah Greener on the 28th of January, 1848, in Winlaton, County Durham, England,[17] serves to stress the essential tragedy of a death due to anaesthesia itself. Hannah was a fit girl of fifteen years of age. She was anaesthetised for nothing more serious than the avulsion of an ingrowing toenail. From accounts her sudden death seems to have been caused by ventricular fibrilation resulting solely from causes related to the induction of general anaesthesia with chloroform. Here indeed is the problem that still confronts us today. Anaesthesia, in itself non-therapeutic, which serves but to render much of the surgeon's healing ministrations possible

and bearable to the patient, can of itself completely frustrate the ultimate success of such surgery. Sykes recently estimated - and is of the opinion that it is an under-estimate - that in the first hundred years of anaesthesia, 1846 - 1946, there were in England and Wales 24,378 deaths associated with anaesthesia.[176] Many of these must in fact have been caused by the anaesthetic. Today the objectives of anaesthesia have extended far beyond the mere obliteration of pain. Whilst seeking to ensure the maximum safety and comfort for the patient, the anaesthetist aims to provide for the surgeon conditions adequate for operation on any part of the body, controllably preserving at the same time the patient's general physiological homeostasis.

In the latter objectives advances over the years have been spectacular. Pari passu surgical initiative and advances in anaesthetic techniques have proceded to the present stage where there is literally no organ of the body that cannot be approached by the surgeon with facility. In general, however, it is the first objective - the safety of anaesthesia for the patient - that is basically the most important. In spite of the advances that have taken place in anaesthetic practice the Lancet has expressed the opinion, in a leading article, as recently as 1962 that "the most obvious risks of an operation are those associated with anaesthesia".[9]

A crude measure of this aspect of the safety of anaesthesia for the patient can be provided by a knowledge of the incidence of death directly due to anaesthesia. Computation of the incidence of death due to anaesthesia with any degree of accuracy, as against that more simply associated with anaesthesia and operation, is regrettably difficult. Yet when the vast number of patients annually subjected to surgery and anaesthesia is borne in mind the importance of our having some estimate of

this incidence becomes obvious.

Jones[106] has estimated, using the year 1957 as a sample, the annual number of anaesthetics administered in South Africa and South West Africa to be in excess of 385,667. This means that of the total population of South and South West Africa, at least one in every thirty persons is subjected to anaesthesia and operation in any one year. The American Hospital Association estimated the number of operations performed (and, presumably therefore, the number of anaesthetics given) in the United States of America during the year 1.10.48 - 30.9.49 to be 7,076,953.[4] If allowance is made for non-reporting hospitals the annual figure for operations performed and anaesthetics given in the United States at this time was probably of the order of 8,000,000.[18] In England the annual number of operations performed and anaesthetics administered is approximately 3,000,000, estimated from a survey conducted in 1954[83] (see footnote ϕ). Accepting the approximations inherent in these figures, they do indicate that the number of people submitted to operation and anaesthesia

ϕ The statement in this report of the Registrar General that "this means a total of about 8,000,000 administrations of anaesthetics per year in all hospitals in England" is completely misleading. This estimate is based on a tabulation of anaesthetic agents administered alone or in combination. The correct number of anaesthetics administered, in the generally accepted sense, can be computed from the figures quoted of the number of operations performed during the sample periods. Calculated on this basis the number of anaesthetics administered in England in 1954 was 2,915,224.

every year is so great that an associated mortality as low as 0.5 deaths per 1,000 anaesthetics would achieve the dimensions of a public health problem. In spite of this there was, and still is, a grave paucity of information on the incidence of death due to anaesthesia.

This state of affairs was highlighted in 1954 by the publication of Beecher and Todd's monumental survey of 'Deaths associated with Anaesthesia and Surgery'.[18] This investigation analysed on a contemporary basis the mortality associated with approximately 600,000 anaesthetics given at ten university hospitals in the United States of America during the five year period 1948 - 1952 (incl.). The survey, and in particular the inferences Beecher and Todd made from it, excited much criticism. Yet at the time there was no survey of deaths associated with anaesthesia that could be matched with that of Beecher and Todd either with regard to size or scope. As this survey dealt with deaths associated with anaesthesia in the United States of America it would have been of interest to know of the experience of other countries. As it was the mortality consequent on the use of the muscle relaxant drugs about which they expressed particular concern, surveys conducted since the introduction of these drugs into clinical anaesthetic practice in 1942[86] would have been especially apposite. At this time most of the few available surveys of deaths associated with anaesthesia had also been conducted in the United States. In England the only survey of similar scope being undertaken at this time was that commenced by the Association of Anaesthetists of Great Britain and Ireland in 1949.[76] This survey was concerned with elucidating the common causes of anaesthetic death but was not designed to estimate the actual incidence of death due to anaesthesia. Though the Registrar General of England and Wales had recorded annually since 1868 the number of deaths associated with

anaesthesia[176] no attempt had been made to classify these into those more precisely due to anaesthesia itself and those due to other causes. In fact, except for one survey from Manchester published by Hill and Hunter in 1948,[97] the last major survey in which an attempt was made to establish the incidence of death due to, and not simply associated with, anaesthesia in England was that published by the British Medical Association Committee on Anaesthetics in 1900.[32]

In South Africa official disquiet had been caused from time to time by the impression that the incidence of death due to anaesthesia was high. A committee appointed by the Minister of the Interior "to investigate the causes of the high death rate in the Union from anaesthetics and generally to make recommendations more especially with a view to the possibility of measures being introduced which may have some practical effect in lessening the mortality" published their report in 1936.[169] This committee, surveying hospital records placed at its disposal covering the years 1931 - 1935, did attempt to establish what the incidence of deaths due to anaesthetics was at this time. The report of the National Health Service Commission published in 1944 contains in that section on recommendations regarding the administration of anaesthetics, the statement that "we are impressed by the evidence showing that there is an unnecessary mortality from this cause (anaesthetics)", though what this evidence was or what the incidence of death due to anaesthesia was, is not quoted.[170] A thesis on 'Death associated with Anaesthesia' was presented to the University of the Witwatersrand in 1947 by Melzer.[126] Part of the investigation on which this thesis was based established the incidence of death associated with anaesthesia only. Except for a short paper published in 1953 by Barlow et al. on the anaesthetic mortality at Coronation Hospital, Johannesburg,[16] there was little concrete information

available on the incidence of death due to anaesthesia in South Africa at the time of the publication of the Beecher and Todd survey - this, in spite of the periodic disquiet at the alleged high incidence of death due to anaesthesia in South Africa.

As a long term project, therefore, I thought it would be instructive and informative to survey and evaluate the incidence of death due to anaesthesia or to which anaesthesia was significantly contributory, in Groote Schuur Hospital, the principle teaching hospital of the University of Cape Town. To this end I was stimulated, supported and encouraged by the then Head of the Department of Anaesthetics of this hospital, Dr. C.S. Jones, support and encouragement that has been continued by his successor, Professor A.B. Bull. Some financial support was provided for this investigation by the Council for Scientific and Industrial Research ø which body itself, at this time, had commenced examining the problem on a wider national scale. [168, 165]

The pages that follow contain an acccount of this investigation. It covers to date a period of eight years which is divided into two parts:-

1. The five year period 1956 - 1960 (incl.)
   This period is regarded as the pilot survey.
2. The three year period 1963 - 1965 (incl.)- the continuing survey.

   (The gap of two years between the two periods surveyed resulted from the absence of the author from Groote Schuur Hospital.)

This survey continues. It serves, as do surveys like it, to reflect and monitor our standards of clinical anaesthesia and so provides a vital means of measuring and evaluating the safety of anaesthetics for the patient and consequently progress in anaesthesia.

ø In subsequent text - C.S.I.R.

CHAPTER 2.

THE COLLECTION OF DATA.

This study has been conducted at the Groote Schuur Hospital, Cape Town. This general hospital is the principle teaching hospital of the Medical Faculty of the University of Cape Town. The accommodation for in-patients of 850 beds during the first period of the survey had been increased to approximately 1200 beds during the second period. Groote Schuur Hospital is a general hospital in the widest sense, and the work of the Anaesthetic department provides a very adequate cross section of general anaesthetic practice. Though the department of Anaesthetics does provide anaesthetic services for other hospitals in the teaching hospitals group, in the interest of more efficient management, this study was confined to the Groote Schuur Hospital. During the major part of this survey anaesthetics administered in other hospitals in the teaching hospitals group accounted for approximately 10% only of the total number of anaesthetics given by the department.

The surgery for which anaesthetics were administered covered the whole spectrum of modern surgical practice. It included operations of all types from the most simple to the most complex, from Casualty out-patient surgery to Cardiac surgery with cardiopulmonary by-pass. It included general surgery and the surgery of all the present day surgical specialities. Specialised Paediatric surgery is one branch of surgery not fully represented in this survey. A specialised

Paediatric hospital, the Red Cross War Memorial Hospital, opened its doors in the year this survey commenced. However most of the surgery in children in the surgical specialities, Neurosurgery, E.N.T., Ophthalmological and Orthopaedic surgery continued to be performed at Groote Schuur Hospital and so provided a fairly adequate paediatric population for the purpose of this survey. No anaesthetic services for operative Obstetrics were included in the first period of this survey as no Obstetrics was conducted at Groote Schuur Hospital during this time. A 120 bed maternity wing was opened at this hospital in the interim between the two survey periods. Anaesthetic services provided for operative Obstetrics in this block is included in the second period of this survey. Though most of the anaesthetics given were for standard surgical procedures, the advancing fringes of surgery were always in evidence. The years covered by this survey saw the birth and development of Open Heart surgery, major vascular surgery and the increase of almost epidemic proportions in serious multiple injuries, predominantly from motor vehicle accidents, requiring surgical treatment. The annual distribution of the numbers and types of operations for which anaesthetics were given during this survey is tabulated in Table 1.

The patients included in this survey were multiracial, reflecting the population of the country and being predominantly of three racial types, (i) European, (ii) Cape Coloured, (iii) African.

To evaluate the problem of deaths associated with anaesthesia ideally, all deaths occurring during anaesthesia and operation and during the total hospital stay of the patient after operation should be studied. But the adoption of so wide a field of study with the vast investment of time and facilities

TABLE 1.

SURGERY FOR WHICH ANAESTHETICS WERE ADMINISTERED

FIRST PERIOD

Type of Operation	1956	1957	1958	1959	1960
Gastric Resection	335	251	232	203	227
Biliary System	176	228	197	204	206
Colon and Rectum	89	78	67	59	129
Adrenalectomy	26	14	12	15	9
Oesophagectomy	13	36	27	28	34
Sympathectomy	71	63	71	45	65
Aortic Aneurysm and major Vascular Surgery	-	10	35	156	144
Other General Surgery	3019	2942	2830	2553	2459
Major Jaw and Tongue	15	43	28	35	25
Cardiac	61	62	66	84	187
Other Intrathoracic	53	84	72	87	60
Intracranial and Spinal	332	285	162	212	372
Other Neurosurgical	272	251	369	359	840
Mastoidectomy	188	195	158	166	199
Microsurgery of Ear	7	1	5	98	122
Tonsillectomy	2131	1780	2024	2250	1747
Nasal Sinuses	90	282	99	304	290
Laryngectomy	4	5	11	5	4
Open Bone Operations	843	793	880	936	953
Manipulations	798	710	690	712	865
Nephrectomy	41	39	25	48	30
Transabdominal Urol.	186	134	164	180	182
Other Urological	370	472	354	420	638
Hysterectomy	397	507	545	549	585
Wertheim and Pelvic Exenteration	32	52	42	34	9
Vesicovaginal Fistula	40	66	40	53	40
Colporrhaphy	113	108	129	92	119
Other Gynaecological	3364	3062	3232	3662	3665
Plastic Surgery	477	581	393	401	455
Ophthalmological	520	575	556	543	573
Casualty Surgery	1941	2371	2482	2584	2569
	16004	16080	15997	17077	17802
					82960

SECOND PERIOD

Type of Surgery	1963	1964	1965
General	4199	4396	4766
Cardio-Thoracic a.Open card.	64	82	92
b. closed card. & thoracic	83	90	81
Neurosurgery	1301	1531	1537
Orthopaedic a.Open ops.	726	1129	1137
b.Manipulation	895	932	971
Gynaecological	4588	5310	5395
Obstetric	326	718	822
Urological	1970	2088	2042
E.N.T.	3062	2903	2760
Ophthalmological	1385	1338	1282
Plastic Reconstructive	993	1040	1147
Casualty & Minor G.A.	1310	1580	1636
L.A.	1183	1094	798
	22085	24231	24466
			70782

TOTAL ANAESTHETICS - 8 YEARS - 153742.

involved was not considered practicable. The identification of anaesthesia as a contributory cause of death is often difficult. This difficulty increases the further the death of the patient is removed in time from the actual administration of the anaesthetic. This is especially so beyond the more immediate post-operative period and after the patient has regained consciousness. In general it is true to say that nearly all deaths that result specifically from anaesthesia, or to which anaesthesia has been significantly contributory, are acute deaths which occur during the administration of an anaesthetic or in the immediate post-anaesthetic period. Accepting this premise the study of deaths associated with anaesthesia may be limited to a study of those deaths that occur during anaesthetic and operation and the immediate post-operative period, including those patients who fail to regain consciousness after anaesthesia, provided it is realised that by so doing some late post-operative deaths to which anaesthesia may have been contributory will be missed. Examples of such late post-operative deaths would be the death of a patient from bronchopneumonia resulting from causes related to the anaesthetic administration, the death of a patient from meningitis following a spinal anaesthetic or the death of a patient from acute necrosis of the liver following the administration of an anaesthetic drug with hepatotoxic properties. An example, with particular South African relevance, would be the death of a patient following an attack of acute porphyria precipitated in a susceptible subject by the administration of a barbiturate anaesthetic.[57] I have evidence that one such death due to porphyria did occur in a patient anaesthetised in this hospital during this survey.[58] (The actual attack of acute porphyria commenced after the patient's discharge from hospital following an anaesthetic for a minor gynaecological

involved was not considered practicable. The identification of anaesthesia as a contributory cause of death is often difficult. This difficulty increases the further the death of the patient is removed in time from the actual administration of the anaesthetic. This is especially so beyond the more immediate post-operative period and after the patient has regained consciousness. In general it is true to say that nearly all deaths that result specifically from anaesthesia, or to which anaesthesia has been significantly contributory, are acute deaths which occur during the administration of an anaesthetic or in the immediate post-anaesthetic period. Accepting this premise the study of deaths associated with anaesthesia may be limited to a study of those deaths that occur during anaesthetic and operation and the immediate post-operative period, including those patients who fail to regain consciousness after anaesthesia, provided it is realised that by so doing some late post-operative deaths to which anaesthesia may have been contributory will be missed. Examples of such late post-operative deaths would be the death of a patient from bronchopneumonia resulting from causes related to the anaesthetic administration, the death of a patient from meningitis following a spinal anaesthetic or the death of a patient from acute necrosis of the liver following the administration of an anaesthetic drug with hepatotoxic properties. An example, with particular South African relevance, would be the death of a patient following an attack of acute porphyria precipitated in a susceptible subject by the administration of a barbiturate anaesthetic.[57] I have evidence that one such death due to porphyria did occur in a patient anaesthetised in this hospital during this survey.[58] (The actual attack of acute porphyria commenced after the patient's discharge from hospital following an anaesthetic for a minor gynaecological

operation).

Though there is little or no indication in the literature as to the precise incidence of late post-operative deaths to which anaesthesia is considered contributory, I did not consider the number involved would be commensurate with the practical difficulties entailed in a survey of the total post-operative period. I decided, therefore, to limit this survey to the period of anaesthetic and operation and immediate post-anaesthetic phase in the belief that the incidence of death due to anaesthesia during this period would not differ very greatly from the incidence of death due to anaesthesia in the overall surgical course of the patient.

Accordingly, I sought to obtain relevant information on all patients :-

1. who died during the administration of an anaesthetic.
2. who died within twenty-four hours of the administration of an anaesthetic.
3. who, having been conscious before an anaesthetic, died without regaining consciousness thereafter.

The period of twenty-four hours was chosen arbitrarily as being the period most likely to reveal the cases relevant to this study without the investigation becoming unmanageably diffuse. In his investigation of deaths associated with anaesthesia Brown[35] reported that 89% of deaths occurred during or within two hours of the operation. Of the deaths associated with anaesthesia reported to the Anaesthetic Deaths Committee of the Association of Anaesthetists of Great Britain and Ireland 77% had occurred during the operation or within thirty minutes thereafter.[81] An existing hospital routine provided the means by which initial information on all deaths associated with anaesthesia in the hospital could be obtained. Whenever a patient dies at Groote Schuur Hospital a small certificate of

identity is completed by the admission officer for attachment to the body. This certificate gives details of the patient's name, age, ward, disease, date of admission and date and time of death. The format of this certificate was modified to include the question 'If operated upon' (see Fig. 1). Instructions were then issued by the Hospital Superintendent that should the answer to this question be 'Yes', a duplicate certificate was to be completed and sent to me. By this means I was kept informed of all the operative and post-operative deaths that occurred in the hospital. With this information, consultation of the registers of operations kept in all operating theatres revealed both the name of the anaesthetist concerned and the time interval between the operation on the patient and his death. If the death had occurred during the operation and anaesthesia or within twenty-four hours, I directly approached the anaesthetist concerned and requested him to submit to me full clinical details of the pre-anaesthetic condition of the patient, details of the conduct of the anaesthetic and clinical course of the patient until death. For this purpose use was made of a roneoed form designed by Professor O.V.S. Kok of the C.S.I.R. Anaesthetic Deaths Research Unit in Pretoria for the collection of information (see Fig. 2). This form was itself an adaption from that used by the Committee on Anaesthetic Deaths of the Association of Anaesthetists of Great Britain and Ireland for their investigation.[76] Use of the C.S.I.R. forms facilitated duplication of case records for submission to the C.S.I.R. Anaesthetic Deaths Research Unit with which body I was co-operating while conducting this independent survey.

Of those patients who died more than twenty-four hours after having been anaesthetised, I kept details only of the operation performed. From these I computed the total number of post-operative deaths. To obtain information on the third category

G.S.H. 290

GROOTE SCHUUR HOSPITAL/HOSPITAAL

NOTIFICATION OF DEATH
IN KENNISSTELLING VAN STERFGEVAL

COMPLETE BY HAND WHEN LABEL NOT AVAILABLE
VOLTOOI SKRIFTELIK INDIEN ETIKET NIE BESKIKBAAR IS NIE

NAME
NAAM..... AGE
OUD.....

FOLDER No.
OMSLAG Nr..... BIRTH
GEB..... RACE
RAS..... SEX
GESLAG.....

ADM.
TOEL..... DEPT..... WARD
SAAL.....

Address
Adres.....

.....

Notified
In kennis gestel .....

Doctor-in-charge
Dokter-in-bevel .....

If operated upon
Indien geopereer ..... Nature
Aard .....

Is P.M. wanted?
Word Lykskouing verlang? .....

Time of Death
Tyd van Dood ..... Date
Datum .....

THIS SLIP TO BE HANDED IN TO ROOM 102, FIRST FLOOR, MAIN BUILDING.

McMaster Wbg./11736/4/64/100 Bks.

FIGURE 1.

Form for Notification of a Death.

FIGURE 2.

(see reverse and facing pages)

Form used for the collection of data on details of patient's condition, operation and anaesthetic.
This is a slight modification of the C.S.I.R. Anaesthetic Deaths Research Unit form. The modifications are :-

1. the addition of a space for 'operation'

and

2. the addition of the following questions :
   a) Did patient regain consciousness ?
   b) Was there any respiratory inadequacy ?

Strictly Confidential. Number .....

RECORD OF DEATH ASSOCIATED WITH ANAESTHESIA.

1. HOSPITAL OR INSTITUTION .....

PATIENT'S HOSPITAL NO. ..... WEIGHT .....AGE.....

SEX ..... RACE ..... OCCUPATION.....

DATE OF ADMISSION ..... ALCOHOL OR DRUG ADDICTION. .....

* STATUS OF SURGERY e.g. Specialist; General Practitioner; Registrar or Intern.

* STATUS OF ANAESTHETIST e.g. Specialist; General Practitioner; Registrar or intern. (N.B. No names need be mentioned.)

* Underline status of doctor.

DATE OF OPERATION .....DURATION OF OPERATION.....

PRE-OPERATIVE DIAGNOSIS .....

OPERATION .....

POST-OPERATIVE FINDINGS .....

---

2. CONDITION OF PATIENT BEFORE OPERATION .....

TEMPERATURE ..... PULSE.....

RESPIRATION ..... B.P. .....

(a) CIRCULATORY .....

(b) RESPIRATORY .....

(c) URINARY .....

(d) OTHER SYSTEMS (e.g. Liver or spleen enlarged?/Was Jaundice present ?) .....

---

3. SPECIAL PRE-OPERATIVE TREATMENT (e.g. blood, gastric lavage, insulin) .....

---

4. (a) PREMEDICATION: DRUGS USED .....

Time of giving ..... Satisfactory/Unsatisfactory.

(b) METHOD OF INDUCTION .....

.....

(c) METHOD OF MAINTENANCE .....

.....

.....

(d) RELAXANT DRUGS (Details of dosage and antidotes used).....

.....

.....

5. TIMES OF -

(a) Starting induction .....
(b) Starting operation .....
(c) Ending operation .....
(d) First untoward signs .....
(e) Respiratory failure .....
(f) Circulatory failure .....
(g) Cessation of restorative efforts .....

---

6. DETAILED ACCOUNT OF (a) ADMINISTRATION and (b) RESTORATIVE METHODS. (Please attach copy of blood pressure, pulse etc. if available).

---

7. DID PATIENT REGAIN CONSCIOUSNESS? .....

---

8. WAS THERE ANY RESPIRATORY INADEQUACY? .....

---

9. SUMMARY OF POST-MORTEM FINDINGS (Please attach copy of pathologist's report, microscopical and macroscopical).

---

10. IF INQUEST HELD, MAGISTRATE'S VERDICT. (To be forwarded if not available).

---

11. REMARKS.

..... 19...

.....
Signature.

---oOo---

of patients studied, those who failed to regain consciousness after anaesthesia but may have survived more than twenty-four hours post-operatively, the co-operation of all members of the Department of Anaesthetics was obtained by a request from the Head of the Department that all members submit to me the same information on any such patients that they had anaesthetised on the roneoed form described.

Clinical documentation was completed when possible by the findings at autopsy. Where deaths fell within the provision of the Inquest Act of 1919 or Clause 86 of the Medical, Dental and Pharmacy Act No. 13 of 1928,[74] autopsy was performed by the Government Pathologist from whom I obtained details. Where this was not the case, the necessary legal permission for post-mortem examination was sought from the patient's relatives. In cases where this was obtained, autopsies were performed by the Department of Pathology of the University of Cape Town. Records of these post-mortems were subsequently available to me.

The adoption of the above criteria which are similar to those adopted by various anaesthesia study commissions in the United States of America,[149, 137, 139] means that cases are entered into the study entirely on the basis of factual events. No matter of opinion is involved in the entry of a case into the study. Discussion and opinion do not antecede but follow on the entry of a case into the study. In common with many countries, South Africa has adopted legislature requiring compulsory inquests in all cases of death occurring during the administration of an anaesthetic or to which an anaesthetic may have contributed. The relevant section of the South African Medical and Dental and Pharmacy Act, Clause 86 of Act 13 of 1928 reads as follows :-

"The death of a person whilst under the influence of a general anaesthetic or local anaesthetic or to

> which the administration of the anaesthetic has been a contributory cause, shall not be deemed to be a death from natural causes within the meaning of the Inquest Act of 1919, or the Births, Marriages and Deaths Registration Act of 1923 or any amendment of these Acts."

In their studies of deaths associated with anaesthesia in South Africa, the Orenstein Committee[169], Kok[165] and Melzer[126] have all made use of these criteria to define the association of a death with anaesthesia. However, other than for those deaths that occur during the actual administration of an anaesthetic, e.g. those occurring in a post-operative period, an initial opinion and decision as to the contribution of anaesthesia to the death is necessary before the submission of the case to autopsy and inquest and so entry of such a case into the study. This decision is not made by the person conducting the study. This same criticism applies to criteria which do not adequately define deaths 'associated with anaesthesia' adopted by the Anaesthetic Deaths Committee of the Association of Anaesthetists of Great Britain and Ireland[76]. Other than in cases of death occurring during the actual administration of an anaesthetic, the adoption of such vague criteria must involve, to some extent, an opinion as to the cause of death and the contribution of anaesthesia thereto before the entry of such a case into the study. This may result in some deaths with a less obvious association with anaesthesia escaping the net of the investigator. With reference to the legal provision for Inquests on deaths occurring in association with anaesthesia, it must be borne in mind that these provisions are framed not so much for the investigation of causation, incidence and prevention of deaths due to anaesthesia as for the seeking out and prevention of

medical negligence. In these circumstances the deaths with the less obvious association with anaesthesia are perhaps not as relevant as they would be to the present study.

BACKGROUND DATA

In order to relate the numbers of deaths due to anaesthesia to the parent population of patients anaesthetised, I sought information on the following :-

1. The total number of anaesthetics administered at Groote Schuur Hospital. This information was derived annually from the registers of operations kept in all operating theatres.
2. The age and sex of all patients anaesthetised.
3. The numbers of uses of various anaesthetic agents and techniques.
4. The physical status of patients anaesthetised.

To obtain this information a small pocket data card was designed on which every anaesthetist could easily and simply record the essentials of every case that he personally anaesthetised. The design of this card is illustrated in Fig. 3. As each card was completed by the individual anaesthetist, so it was returned to me and a new one obtained. The details supplied by these cards were then recorded. At the completion of the pilot survey period the value and relevance of this information to the purpose of the survey was re-examined. In the light of this I decided that the value of classifying deaths to which anaesthesia was contributory, in relation to the drugs used, was not commensurate with the vast amount of time demanded for the collection and tabulation of this information. The use of the pocket anaesthetic data cards was dropped therefore in the subsequent continuation of the survey. Details of the number of operations and the sex of the patients was then obtained directly from the registers of operations. Regrettably the omission of the use of the data card did mean that for the continuing survey the age distribution of the patients anaesthetised was not obtainable directly.

Age Sex	OPERATION	INHALATION							INTRAVENOUS					TECHNIQUE							RISK				Resuscitation		NOTES
		N_2O	O_2	Ether	Trilene	Et. Chlor.	Chlorof.	Cyclo.	Pent.	Relaxant	Prostigmine	Pethidine	Largactil	N. Resp.	I.P.P.R.	Endotrach.	Spinal	Epidural	Nerve Block	Field Block	1	2	3-4	Emergency	Vasopressor	Blood	

NOTE.—Use 1 line per case. Mark relevant square with X. N.Resp.—Normal Spontaneous Resp. All Assisted or Controlled Resp.—I.P.P.R. Epidural—Lumbar, Caudal. If case emergency mark Risk & Emergency Square. List under notes all drugs or techniques not indexed.

FIGURE 3.

Pocket Anaesthetic Data Card.

CHAPTER 3.

THE CLASSIFICATION AND ASSESSMENT OF DATA.

THE GENERAL CLASSIFICATION OF DATA.

In any biological study where the observations are compounded from the interaction of many variables, the classifying of data into meaningful groups from which valid inferences can be made is frequently a most difficult problem. In examining the data collected on deaths associated with anaesthesia the principle problem with which I am concerned is to establish the number of such deaths in which the administration of an anaesthetic and all that this implies was a major causative factor and so to establish, with a knowledge of the background anaesthetic population, the actual incidence of death to which anaesthesia is significantly contributory.

In many, if not most instances, deaths associated with anaesthesia result ultimately from a combination of circumstances of which anaesthesia is but one. It may be extremely difficult to assess accurately the individual gross responsibility of these various factors. The commonest and most important of these associated circumstances is the physical status of the patient. Grave systemic disturbance, often not directly connected with the surgical disease, may be present. Such disease, even if not itself responsible for the death of the patient, will by its deleterious effects on the patient's physiological homeostatic mechanisms, make the giving of an anaesthetic safely a task of increasing difficulty. The other important circumstance in what we may regard as the environment of the anaesthetic is the magnitude of and possible difficulties, errors or mishaps

in the surgical procedure for which the patient is anaesthetised. Many workers take cognisance of this environment of the anaesthetic by the provision of separate categories for these factors in the classifications they adopt. An example of a classification of deaths associated with anaesthesia that takes into account these three broad factors - the anaesthetic, the patient's disease, the surgical operation and permutations of these - is that of Waters and Gillespie[183], used also by Dornette and Orth.[54]

1. Deaths due to the operation.
2. Deaths due to the patient's disease.
3. Deaths due to the operation and the patient's disease.
4. Deaths due to anaesthesia.
5. Deaths due to operation and anaesthesia.
6. Deaths due to anaesthesia and the patient's disease.
7. Deaths due to anaesthesia, the patient's disease and operation.

The classifications adopted by many workers are variations of this type.[169, 150, 18, 103, 98, 45, 154, 42.] An example of a slightly different classification is that adopted by the Association of Anaesthetists of Great Britain and Ireland for their study of deaths associated with anaesthesia.[81] This classification is of especial interest in that it has been adopted subsequently by two other bodies working in this field viz. the South African Council of Scientific and Industrial Research Anaesthetic Deaths Research Unit[165] and the special committee appointed to investigate deaths due to anaesthesia in New South Wales.[134] Categories in this classification are as follows :-

1. Those deaths where it is reasonably certain that the death was caused by the anaesthetic agent or technique or in other ways within the province of the anaesthetist.
2. Similar cases in which there is some element of doubt whether agent or technique are entirely responsible.

3. Those deaths due to surgical and anaesthetic technique.

4. Surgical deaths.

5. Inevitable deaths e.g. cases of severe peritonitis but in which the surgical and anaesthetic technique is satisfactory.

6. Fortuitous deaths e.g. pulmonary embolism.

7. Cases that could not be assessed.

8. Cases in which the data presented was inadequate. No opinion.

Consideration of such classifications led me to the belief that a simpler classification would be more relevant to the purpose of this study. Anaesthesia purports to be a controllably reversible process. In seeking to establish the incidence of deaths due to anaesthesia I feel that the culpability of the anaesthetic should be considered in the context not only of the fit patient but also of the more seriously ill and aged patient with which ever advancing surgical initiative presents it. I decided therefore to classify all deaths associated with anaesthesia in which the administration of the anaesthetic itself or factors within the compass of the anaesthetist's responsibility could be shown to have played a significantly causative role in one overall group. It is from this group that the more precise incidence of death to which anaesthesia is contributory is calculated. It is examination of the clinical details of these cases that should point the lessons from which improvements in clinical anaesthesia must follow.

From its very nature there are few, if any, deaths that occur in association with anaesthesia to which it can be said that anaesthesia has made no contribution. However, where the conduct of the anaesthetic appears faultless, where other circumstances appear to have provided adequate reason for death and a contributory role of the anaesthetic, if present at all, is minor, no useful purpose is served by classifying these deaths

with those of anaesthetic etiology. All these deaths are identified in a second large group which for the major purpose of this study is excluded from further discussion.

Lastly there is a third group of not uncommon cases that require separate identification. These are deaths from which the anaesthetic and its management, though apparently faultless, cannot be excluded as a significantly contributory factor. This may be because death has occurred during anaesthesia. Or it may be that the final collapse of circulatory homeostasis in a patient, moribund before anaesthesia was induced, appears to have been finally precipitated unavoidably by the known effects of anaesthetic drugs and anaesthesia e.g. vasodilatation. This group of deaths may be regarded as 'inevitable deaths'[81] or deaths to which anaesthesia may be regarded as 'necessarily and unavoidably contributory'. In these circumstances precision of assessment of the exact contributory role of the anaesthetic is not only impossible to make but is also of little clinical importance. All cases in which death occurred following a cardiac arrest during anaesthesia but where the contributory role of anaesthesia was minor are also classified in this group, even though death may have occurred in the post-operative period after the termination of the anaesthetic.

Attempts were always made to obtain data sufficient for the death to be classified. I provide no category in the classification for 'uncertain cases'. If there was great uncertainty as to the precise category in which a death should be placed I concluded that any contributory role played by the anaesthetic was in all probability not major or significant and classified such cases in the second group.

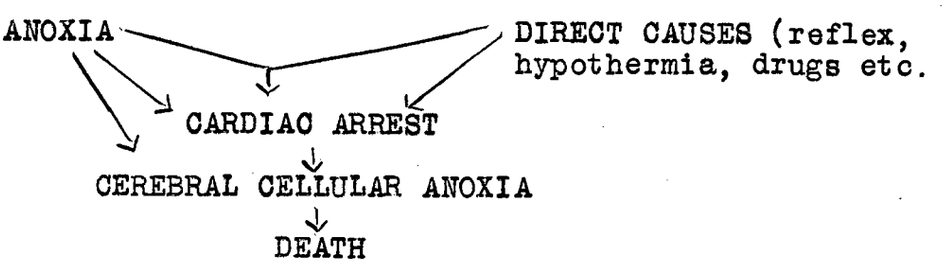
<u>The Classification of Causes of Death</u>.

The anaesthetist, it has been said, conducts the patient

he anaesthetises through the 'valley of the shadow of death' (Psalm XXIII).[87] This idea arises from the profound and reversible direct depression produced by anaesthetic agents of the very seat of life, the brain. Inseparable from this are the effects the administration of anaesthetic drugs have on those two interrelated vital systems, the circulatory and respiratory, the continuous and efficient functioning of which is essential to the viability of every cell in the body. Death follows when disturbance of the function of either or both of these is of sufficient magnitude to deprive the brain of oxygen for as little as from three to eight minutes. Even a state of partial anoxaemia, we are told by Haldane, "means not a mere slowing down of life, but progressive and perhaps irreparable damage to living structure."[92] This, in its stark simplicity, is the mechanism by which the administration, more usually the maladministration, of an anaesthetic may directly cause death.[5]

Though severe anoxia may directly cause irreversible cerebral damage,[44] the final common pathway to the ultimate cerebral anoxia and death is most often through the mechanism of arrest of the circulation from cardiac arrest, either asystole or ventricular fibrillation. Besides anoxia which is a most common factor in its causation, cardiac arrest has a wider etiology including such factors as autonomic reflexes, acidosis, hypothermia, the effects of certain drugs, changes in the intra/extracellular potassium concentration ratios and direct handling of the heart. Most of these factors are of more consequence in the causation of cardiac arrest in an environment of myocardial anoxia.[130, 172]

This simple concept of the causes of death due to anaesthesia may be illustrated by the following diagram :-



This fundamental pathological state of anoxia arises when the cellular oxygen utilisation and demand exceeds the amount of oxygen available. Nunn and Freeman[135, 136] express 'available oxygen' as the product of :-

i. Arterial oxygen saturation.

ii. Cardiac output.

iii. Haemoglobin concentration.

iv. Oxygen carrying capacity of Haemoglobin.

In normal values this would be :-

$$\text{Available Oxygen ml./minute at B.T.P.S.} = \frac{95}{100} \times 5250 \times \frac{15}{100} \times 1.34 = 1000 \text{ ml./min. (approx.)}$$

If any one of these factors is reduced, the available oxygen is reduced proportionately. If two or three factors are reduced simultaneously the available oxygen is reduced as the product of these factors. This may be regarded, in essence, as a modification of Barcroft's classification of anoxaemia,[15] but stated in terms that stress the interaction of the factors concerned with the provision of oxygen to the cells, and the synergism of derangements in these. To relate Nunn and Freeman's concept of 'available oxygen' to Barcroft's classification of anoxaemia, deficiency in arterial oxygen saturation may be regarded as equivalent to Anoxic Anoxia, that resulting from deficiency of cardiac output as Stagnant Anoxia and that due to deficiency in haemoglobin concentration as Anaemic Anoxia. I prefer to regard deficiency in cardiac output - Barcroft's stagnant anoxia - resulting as it does in poor tissue perfusion, as causing tissue ischaemia and use for the state of anoxia so

arising the term ischaemic anoxia. Difficulties arise in the presentation and discussion of clinical cases where anaesthesia has been significantly contributory to death because the factors that produce the final anoxia are so often inextricably mixed. Using this concept of 'available oxygen' the cases in this survey are presented and classified in terms of the factor deficiency of which was considered most prominent.

Within the framework of this general classification cases are further classified with regard to the more precise clinical fault or 'departure from accepted practice'[81] that led to the particular state of anoxia. Such faults are legion and are not detailed here.

In this classification the presentation of cases of cardiac arrest presents some difficulty. In some cases the etiological factors that result in a cardiac arrest are apparent before the event occurs. More often these escape notice until the dramatic occurrence of cardiac arrest. In these circumstances the causal factors, though they may be elucidated in retrospect on occasion, are often obscure. In these cases beyond the fact that the conduct of the anaesthetic may appear in general to be contributory in the absence of more obvious causes, there may be no certainty as to a precise cause. In this survey cases of cardiac arrest are classified in relation to the type of anoxia that was considered causal. Those cases for which some direct cause other than anoxia was responsible or where the etiology was uncertain, are classified separately under the heading 'Cardiac Arrest'.

Those patients who suffered cardiac arrest from causes related to the anaesthetic but, because of correct resuscitative procedures, survived into the immediate post-operative period only to die of continuing circulatory failure, are classified in relation to the cause of the original cardiac arrest. For

those anaesthetic contributory deaths that do not fall into any of these categories a sub-division 'Miscellaneous' is provided.

Preventibility.

Looked at broadly, deaths due to anaesthesia are a function of three factors :- [131]

1. the skill of the anaesthetist.
2. the condition of the patient.
3. the agent used.

Of these the skill of the anaesthetist is the most important - skill which is compounded of many parts, training, clinical experience and ability. The condition of the patient, which will respond to some extent to skilful pre-operative treatment, may often be something over which the anaesthetist will have no final control. As regards the agent used, the anaesthetist should have sufficient knowledge of the drugs he uses clinically, of the actions, side-actions and methods of counteracting these, that any deaths that may be said to result from so-called inherent toxicity of the drug used become basically deaths due to lack of skill. If we accept that lack of skill on the part of the anaesthetist is the most important factor in the causation of death due to anaesthesia, it follows that many deaths due to anaesthesia are preventable. This tenet is strongly ennunciated by Macintosh [119] "I hold there should be no deaths due to anaesthetics". This aspect of 'preventibility' must be stressed in any survey of deaths due to anaesthesia. It is in this sector that improvements must be made. Clinical anaesthesia is not an exact science. In the assessment of any particular death that is considered due to anaesthesia, it is often difficult to say accurately that such a death is definitely preventable. We seek always to identify 'departures from accepted practice'. [81] This in itself must change with the passing of time and the accummulation

of further knowledge and experience. On examination of the circumstances of any death in the light of present knowledge we may be able to say that such a death was 'probably preventable'. More frequently the circumstances may allow only the conjecture that such a death was 'possibly preventable'. In some circumstances, though we may conclude virtually by exclusion that a particular death was in large measure due to the administration of an anaesthetic, examination of the conduct of the anaesthetic may fail to reveal any obviously correctable fault. We are then able to give no verdict as to its preventability. This should not, however, detract from the premise that ideally deaths due to anaesthesia are preventable. The deaths allocated to group 1. are, therefore, sub-divided into the following three accessory groups :-

i. probably preventable

ii. possibly preventable

iii. no verdict

The first two sub-divisions include those cases best described, as suggested by Morton,[131] as "those deaths for which a reasonably satisfactory explanation can be provided and for which counter measures are practicable". The last sub-division includes those cases "which cannot at present be fully explained and for which counter measures are either lacking or largely empirical".
It is only in relation to the management of the anaesthetic that the aspects of 'preventibility' are classified and commented on. Those pertaining to the surgical management of the patient are not discussed.

In summary the classification adopted in this survey is as follows :-

1. <u>Anaesthetic Contributory Deaths</u>

   Those deaths to which anaesthesia was considered to have been contributory to a significant degree. These deaths are then further classified as to -

The cause of death

I. ANOXIA — A. Anoxic Anoxia.
B. Ischaemic Anoxia

II. CARDIAC ARREST — A. Direct causes other than anoxia.
B. Uncertain etiology.

III. MISCELLANEOUS

The more precise clinical fault is then identified.

Preventibility

i. Probably preventable.

ii. Possibly preventable.

iii. No verdict.

2. Deaths due to other causes.

Those deaths occurring in association with anaesthesia which are due to the patient's disease or factors related to the surgical operation and to which the anaesthetic was considered either non-contributory or, at most, a minor contributory factor.

3. Inevitable Deaths.

Deaths to which anaesthesia was considered to be 'necessarily and unavoidably contributory'.

THE ASSESSMENT OF DATA

"Felix qui potuit rerum cognoscere causas - but the only person who really knows the cause of an anaesthetic death is the anaesthetist" (Report of the Ministry of Health of England and Wales)[83].

In any death associated with anaesthesia, assessment of the contributory role the anaesthetic and its management may have played is fraught with difficulty. While consideration must be given to the ways in which the known actions of anaesthetic drugs and errors or misjudgments in their administration are known to lead to death, judgments must be made from clinical accounts of circumstances which have been subject to swift change. Because

of this, the monitoring of various vital parameters is often incomplete. Being acute, the factors that may lead to anaesthetic contributory death leave no pathognomonic changes discernible at autopsy.[5, 138] That most basic of mechanisms of death due to anaesthesia - gross anoxic anoxia - may result in no histological change if death is rapid.[44A, 115] The events that may precede a cardiac arrest, e.g. hypotension, cardiac arrythmia autonomic reflexes, leave no trace.[160] Even as gross a finding as vomitus in the respiratory tract may require careful interpretation

In this assessment of the contributory role of anaesthesia in the associated deaths I have adopted with Dripps and co-workers the attitude that

> "there is nothing to be gained in a mortality study by omitting a particular death merely to lower a statistical death rate. Avoiding responsibility or taking refuge in the fact that a patient was desparately ill prior to anaesthesia and operation may improve one's mortality figures, but it will not advance general knowledge or change one's own practice. On the other hand one should not resort to self-flagellation, assuming responsibility for a fatality merely because an anaesthetic was administered and death occurred."[55]

In addition to the actual administration of the anaesthetic, consideration must be also given to the ways that errors of commission or omission in the wider field of responsibility that has, by practice, become that of the anaesthetist may have caused death, e.g. the failure to replace adequately by transfusion blood lost during the operation, or the administration in error by the anaesthetist of incompatible blood. Examination of this aspect involves clarification of what may be considered the extent of the anaesthetist's responsibility. This is something that is difficult to define precisely as the compass of the

responsibility of the anaesthetist must vary with time and place, depending on contemporary practice. Probably one of the clearest statements of what may be considered the responsibilities of the anaesthetist is that written by Bellamy Gardiner[21] which, though published as long ago as 1916, is well worth repeating. He writes

> ".....the services of a practised administrator.... should relieve the operator of all responsibility with regard to the patient's general condition during operation. The anaesthetist therefore has in many instances to undertake duties of considerable gravity and should be thoroughly equipped not only by individual qualification but physically by possessing perfect senses of sight, hearing, keen scent and gentleness of touch. To his share fall the provision and accurate manipulation of the best drugs and apparatus for the administration of the different vapours; the detection of symptoms and physical signs of disease which will affect the subsequent anaesthesia; the choice of the particular anaesthetic or sequence of anaesthetics most suitable to the patient and operation in hand; the protection of the body from external harm; the regulation of atmospheric temperature; the resort to stimulants and methods of resuscitation in cases of failing vitality; the safe transference to bed and the supervision during recovery from insensibility;..... following complications such as haemorrhage or the need of operative procedures other than those first contemplated which would require a more prolonged administration, it may rest with the anaesthetist to decide whether the patient's condition is such that they may be safely undertaken."

For the purposes of this study I have defined the responsibilities of the anaesthetist as involving attention to all matters that have a bearing on the maintenance of the patient's general physiological homeostasis during and immediately after the performance of a surgical operation, under any form of anaesthesia. This is similar to the definition used by Ruth and co-workers.[150] Though the overall responsibility for the patient's treatment and well being is the surgeon's, it is delegated in this respect to the anaesthetist.

In the assessment of the data collected on deaths associated with anaesthesia, though the final opinion was my own, the opinions of members of the Department of Anaesthetics of this hospital were sought. A regular weekly departmental meeting provided the forum for clinical discussions of these cases. In some instances the opinion of the surgeon concerned was also sought.

That these assessments were made by anaesthetists, usually without the help of the opinion of surgeon, physician or pathologist may be considered to be a weakness in this study. Many think the ideal method of assessment is that by an 'anaesthetic death study committee' which would include, together with anaesthetists, representatives of the various other specialities with which anaesthesia is concerned. The Orenstein Committee[169] of 1938, to which reference has been already made, is the only example of the setting up of such a composite committee here in South Africa. The composition and functioning of such committees, which have become popular in America, has been well described by Ruth,[149] Pallin[137] and Phillips and Frazier.[139] There are anaesthetic death study committees active in eighteen states of the U.S.A.[140] Two Australian examples worth quoting are that set up at the Royal Adelaide Hospital in 1935[35] and the more recently constituted special committee on anaesthetic deaths in New South

Wales.[134] Such a scheme was not feasible in this instance. With the precedent that in many published surveys of deaths associated with anaesthesia[60,183,126,97,16,54.] including those of the C.S.I.R. Anaesthetic Deaths Research Unit[165] and of the Association of Anaesthetists of Great Britain and Ireland[81] the assessments were made by anaesthetists, the method of assessment here described was adopted.

In this work it is necessary to refer often to 'Death associated with anaesthesia' and 'Death to which anaesthesia was considered a significant contributory factor'. These phrases are cumbersome and are abbreviated to read 'anaesthetic associated death' and 'anaesthetic contributory death' respectively. The former term has no implication as to the precise cause of the death, and includes all deaths associated with an anaesthetic be they due to the patient's disease, the operation or the anaesthetic per se. The second term refers only to those deaths occurring in association with an anaesthetic which have been caused in large measure by factors for which the anaesthetic and its management was responsible.

CHAPTER 4.

CAUSES OF ANAESTHETIC CONTRIBUTORY DEATH.

"The causation of a given death is always multiple sothat the necessity of selecting a single cause for purposes of tabulation has become a distressing problem." (Treloar)[179]

During the eight year period covered by this survey 1,749 patients died following surgical operation at Groote Schuur Hospital. Of these patients 354 died during or within 24 hours of operation or, having been conscious before, failed to regain consciousness after having received an anaesthetic. These patients, who form the basis of this study, were distributed over the two periods into which the survey falls and were classified into the groups described as follows :-

TABLE 2.

PERIOD	Total Operative & Post-Op. Mortality	Operative & 24 hr. Post-Op. Mortality	Group 1	Group 2	Group 3
1. 1956 - 1960 incl.	1096	207	36	131	40
2. 1963 - 1965 incl.	653	147	15	91	41
	1749	354	51	222	81

The clinical details, classification and commentaries validating such classification of all cases together with notes on aspects of Preventibility in Group 1 cases are included in Volume II. The causes of death in cases classified in Group 1

and those in Group 3 will be discussed briefly here.

I. ANOXIA.

A. ANOXIC ANOXIA.

Anoxic Anoxia is probably the commonest mechanism responsible for death due entirely to the administration, or more commonly maladministration, of an anaesthetic. 45% of all anaesthetic contributory deaths in this survey were due to this cause. The real figure may well be higher as anoxic anoxia was a probable causative factor in some of the deaths classified under the heading of cardiac arrest.

Cases are presented here in relation to the manner in which anoxic anoxia arose during the course of the anaesthetic administration - see Table 3.

Anoxic anoxia during the course of an anaesthetic may arise from :- [153]

1. deficient concentration of oxygen in the inhaled gases or atmosphere - atmospheric anoxia.
2. deficient effective pulmonary ventilation - tidal anoxia. This may be due to - a. respiratory tract obstruction.
   b. induced neuromuscular block without the provision by the anaesthetist of adequate mechanical pulmonary ventilation.
   c. respiratory centre depression.
3. decrease in the efficiency or number of functioning alveoli - alveolar anoxia. In his classification, Saklad [153] used this term to cover certain pathological conditions such as consolidation of the lung. It is pertinent here to remember the hypoxia that may result from the altered ventilation/perfusion ratios in the lung associated with I.P.P.R.ø [38, 43, 173] Though this in itself was not

ø I.P.P.R. - Intermittent Positive Pressure Respiration.

TABLE 3.

DEATHS DUE TO ANOXIC ANOXIA - PARTICULAR CAUSES

CAUSES OF ANOXIC ANOXIA	No. of Cases.	% of Anaes. Contributory Deaths	Identity of Cases
1. ATMOSPHERIC ANOXIA	1	2.0	110/2
2. TIDAL ANOXIA			
a.) RESPIRATORY OBSTRUCTION	12	23.5	
i. Vomiting and Regurgitation (3 cases)			71/1, 97/1, 135/1.
ii. Secretional Bronchial Obstruction (2 Cases)			30/1, 104/1.
iii. Complications of Endotrachael Intubation (4 Cases)			68/1, 12/2, 47/1, 112/1.
iv. Inadequate Post-op. Supervision (3 Cases)			54/1, 74/2, 80/2.
b. RELAXANT ASSOCIATED DEATHS	9	17.6	
i. Post Relaxant Respiratory Abnormality (8 Cases)			20/1, 91/1, 140/1, 157/1, 169/1, 39/2, 70/2, 116/2.
ii. Neostigmine Cardiac Arrest (1 Case)			84/2
3. ALVEOLAR ANOXIA - PULMONARY OEDEMA	1	2.0	136/2
TOTAL	23	45	

identified as a cause of death in any cases here presented it may have played a part in some. Besides causing anoxia of themselves such conditions will have compounded the effect of any existing tidal anoxia.

Though it is anoxia that is stressed here, most of the conditions - certainly those that result in Tidal Anoxia - will have been associated with hypercarbia and respiratory acidosis. Many of the clinical effects of underventilation are due to respiratory acidosis and it is synergistic with anoxia in the production of catastrophe.[59, 118, 151, 39, 129.]

1. ATMOSPHERIC ANOXIA
Case 110/2

The only case in this survey wherein anoxia from this cause was identified as a major contributory cause of death was due to faulty connection of the anaesthetic circuit. This resulted in no oxygen reaching the patient. He totally rebreathed his own exhalations for a period of several minutes. Before the commencement of the anaesthetic this patient was critically ill.

2. TIDAL ANOXIA

a.) RESPIRATORY TRACT OBSTRUCTION

i. Vomiting, Regurgitation and Aspiration - Cases 71/1, 97/1, 135/1

Deaths due to inhalation of vomitus or regurgitated stomach content or blood feature in most surveys of death due to anaesthesia[150, 77, 98] especially anaesthesia for obstetric surgery.[84, 85, 127, 128] In the continuing survey of anaesthetic deaths conducted by the Association of Anaesthetists of Great Britain and Ireland, 11% of the first 1,000 deaths reported to them[81] and 8% of the subsequent 600[82] were ascribed to this cause.

Kok and Mullan[168] attributed 52 of the 1,000 deaths they reviewed to aspiration of vomitus.

It is interesting that in this survey the 3 deaths ascribed to this cause all occurred during the first period of the survey, during which time anaesthesia for obstetric services was not included. During the second period in which anaesthesia for obstetrical services was included no deaths that could be ascribed to vomiting and aspiration occurred.

In both periods of this survey the occurrence of vomiting during anaesthesia was reported rarely by the anaesthetist in relation to patients included in this study. In most instances where it occurred treatment was prompt and effective. In only 3 cases was the aspiration of vomitus identified as a major contributory factor to the patient's death.

The assessment of the inhalation of vomitus as a contributory factor to a patient's death must be based primarily on the clinical history rather than on the ultimate autopsy findings, though these are helpful. It is known that not only can the regurgitation and inhalation of stomach content occur as a terminal event in patients dying of other causes, but, conversely, in many deaths undoubtedly due to this cause, autopsy has failed to show stomach content in the air passages.[81] This latter observation applies to two of the cases in this survey (97/1 and 135/1) in which vomiting and aspiration was identified as contributory to the patients' deaths. In neither of these cases was aspirated material identified in the respiratory tract at autopsy. The remaining case no. 71/1 is an example of silent gastric regurgitation and aspiration.[23,46] In contrast to the former cases this diagnosis was based on the autopsy findings. The clinical events supported the diagnosis. Only the latter patient suffered a cardiac arrest in the operating room. All three deaths were regarded as preventable.

ii. Secretional Bronchial Obstruction - Cases 30/1, 104/1

Both of these patients were children, a circumstance where the smallness of the bronchial anatomy is a factor which markedly worsens the degree of obstruction which may result from bronchial secretions. In case no. 30/1, following the inhalation of a foreign body, the administration of open drop ether, without the prior administration of atropine, provoked copious bronchial secretions causing gross respiratory embarrassment. In case no. 104/1, though gross bronchiectasis was the primary cause of the difficulties encountered, laryngospasm occurring during the induction of anaesthesia was a factor contributory to the final fatal anoxia.

Both the patients suffered cardiac arrest in the operating room.

Though both deaths were regarded as preventable, in the case of no. 104/1 this is a harsh judgment.

iii. Complications of Endotracheal Intubation - Cases 68/1, 12/2, 47/1, 112/1

In the above four cases death was directly due to preventable complications of endotracheal intubation.

Case 68/1 and 12/2 are examples of the dire consequences that may follow endotracheal intubation performed when none of the desiderata essential for safe endotracheal intubation are present. These desiderata are the presence of either deep general anaesthesia or complete muscular relaxation or adequate topical analgesia of the larynx and trachea. In each of these cases endotracheal intubation, performed during light general anaesthesia, was followed by such severe spasm of the muscles of respiration in expiration, together with bronchospasm, that pulmonary ventilation was rendered impossible for a considerable time. Case 12/2 suffered a cardiac arrest at the time. Case 68/1

survived operation but died later of irreversible cerebral damage. Edwards et al[81] identified nine similar cases in their survey. Aspects of preventability are discussed in the commentary on each case.

Cases 47/1 and 112/1 were tragic examples of complete respiratory obstruction following kinking of the endotracheal tube in curarised patients.[69] Failure to recognise the complications timeously was, in each case, due to the inexperience of the anaesthetist. In the second case recognition of the respiratory obstruction was delayed until cardiac arrest had occurred. In the first, though recognition of the respiratory obstruction and its correction were in time to prevent cardiac arrest, irreparable cerebral damage had already occurred. Both patients survived for some days post-operatively in the coma of irreversible anoxic cerebral damage.

iv. Inadequate post-operative Supervision - Cases 54/1, 74/2, 80/2

It is fundamental to safe anaesthetic practice that a patient's emergence from the anaesthetic state be well supervised.[188] Yet the number of deaths due to inadequate post-operative supervision still reported is indeed surprising.[111, 79, 82.] All three cases attributed to inadequate post-operative supervision in this survey were associated with the use of muscle relaxants. After the use of such agents, though respiration may appear to be of adequate depth following pharmacalogical reversal of neuromuscular block, residual muscle weakness may remain.[189, 36.] This may result in the inability of the patient to cough strongly and so expel bronchial secretions or in the inability of the patient to overcome minor degrees of respiratory obstruction by an extra respiratory effort. This factor of mild residual curarisation was probably a contributory factor in each of these cases. Case 54/1 had

bronchiectasis and had been anaesthetised for bronchography. Case 74/2 who was grossly obese and short necked, was placed supine on a theatre trolley immediately post-operatively and was not observed. Pharyngeal respiratory obstruction is known to have occurred in case 80/2 whilst in transit from operating theatre to ward. Case 74/2 suffered cardiac arrest while still in the operating room and the other two cases mentioned suffered cardiac arrest shortly after their return to the ward. All three deaths are regarded as preventable.

b.) RELAXANT CONTRIBUTORY DEATHS

i. Post Relaxant Respiratory Abnormality -
Cases 20/1, 91/1, 140/1, 157/1, 169/1, 39/2, 70/2, 116/2

ii. Neostigmine Cardiac Arrest
Case 84/2

The introduction of muscle relaxant drugs into clinical anaesthetic practice by Griffith and Johnson in 1942[86] initiated the most sweeping advances in anaesthesia. It is important that we have some idea of the incidence of mortality following the use of relaxant drugs as well as knowledge of clinical factors with which such mortality is associated. The identification of the use of relaxant drugs and all that this implies as a factor contributory to the death of a patient poses many difficulties. These are discussed in Chapter 8. I have chosen here to identify as relaxant contributory deaths those which follow the use of a relaxant drug and an I.P.P.R. technique during anaesthesia

1. after which anaesthesia the patient has failed to respire spontaneously with a normal respiratory pattern and the tidal volume has not achieved its pre-operative level or adequacy.
2. for which respiratory abnormality there is no other obvious

TABLE 4.

FACTORS ASSOCIATED WITH 9 RELAXANT ASSOCIATED DEATHS.

FACTOR			Number of Cases	Identity of Cases
LESION	Pancreatitis	7	3	91/1, 140/1 70/2
	Peritonitis		2	157/1, 84/2
	Strangulated Inguinal Hernia		2	39/2, 116/2
	Umbilical Hernia		1	20/1
	Diffuse Carcinomatosis		1	169/1
PHYSICAL STATUS	Fair		3	20/1, 39/2, 70/2
	Poor		6	91/1, 140/1, 157/1,169/1, 84/2, 116/2
ABDOMINAL DISTENSION			6	91/1, 140/1, 157/1, 39/2, 84/2, 116/2
METABOLIC ACIDOSIS (Probable)			7	91/1, 140/1, 157/1, 39/2, 70/2, 84/2 116/2
EFFECT OF NEOSTIGMINE	Nil		6	91/1, 140/1, 157/1, 169/1, 70/2,116/2
	Transient		2	20/1,39/2
	Cardiac Arrest		1	84/2
CONSCIOUSNESS	Recovered Consciousness		4	91/1, 140/1, 169/1, 39/2
	Failed to recover	"	5	20/1, 157/1, 70/2,84/2,116/2.
TIME OF DEATH.	Operating room death		1	84/2
	Less than 12 hours post-operatively		5	91/1,140/1, 157/1,39/2,70/2
	12-24 hours Post-operatively		1	169/1
	More than 24 hours post-operatively		2	20/1, 116/2

explanation such as, for example, anoxic cerebral damage following cardiac arrest.

3. for which respiratory abnormality no efficient pulmonary ventilation was provided by the anaesthetist.
4. for which death no other obvious cause was apparent even if efficient pulmonary ventilation was provided.
5. which may be associated with the pharmacological antidotes to relaxant drugs.

The nine cases listed at the head of this section fulfilled these criteria. Some factors common to these cases are tabulated in Table 4. No one of these nine patients could be classed as a 'Good' anaesthetic risk - i.e. Physical Status Grade 1.[152] Six of these nine patients were considered poor anaesthetic risks pre-operatively whilst the remaining three were considered 'fair'. While all involved abdominal operations, seven of the nine were acute abdominal emergencies and six of these suffered marked abdominal distension. Three suffered acute pancreatitis, two peritonitis and two others acute intestinal obstruction, i.e. seven had lesions which would produce rather similar patterns of fluid and electrolyte deficiencies. This aspect was treated pre-operatively. In all cases spontaneous respiration did recommence but manifested a 'tracheal tug' of varying severity. In all eight who manifested persistent respiratory abnormality, little or no improvement followed administration of neostigmine - the respiratory abnormality appeared neostigmine resistant.[101] Though not investigated it is probable that seven cases had concomitant acidosis.[34] In none of these cases was the volume of respiration adequately measured or monitored post-operatively, the anaesthetist contenting himself with clinical assessment and the absence of cyanosis before return of the patient to the ward. Four of the eight patients who had persistent respiratory

abnormality did regain consciousness after anaesthesia.

Only one of this group, 70/2, was treated with mechanical pulmonary ventilation, However, inadequacies in the application of this otherwise correct form of treatment were probably contributory to the patient's death.

Case 116/2 was associated with gross bronchiectasis. This was doubtless a major contributory cause of the hypoventilation and hypoxia that resulted in cardiac arrest immediately post-operatively, but there was also no doubt of the 'curarised' pattern of respiration that followed anaesthesia.

In one case, 84/2, fatal cardiac arrest followed and appeared to be precipitated by the injection of neostigmine administered for the reversal of residual curarisation.[120] Though an adequate dose of atropine was administered the patient had been permitted to hypoventilate for some time before the administration of neostigmine, circumstances shown by Riding and Robinson[146] to render the administration of neostigmine dangerous.

Five other cases not classified in Group 1, 159/1, 51/2, 56/2, 63/2, 82/2, displayed respiratory inadequacy after anaesthesia involving the use of muscle relaxants. Each of these cases was treated with efficient mechanical pulmonary ventilation post-operatively. In each case there was an adequate reason for the death of the patient. Three of these had peritonitis and one acute pancreatitis.

3. ALVEOLAR ANOXIA
Case 136/2

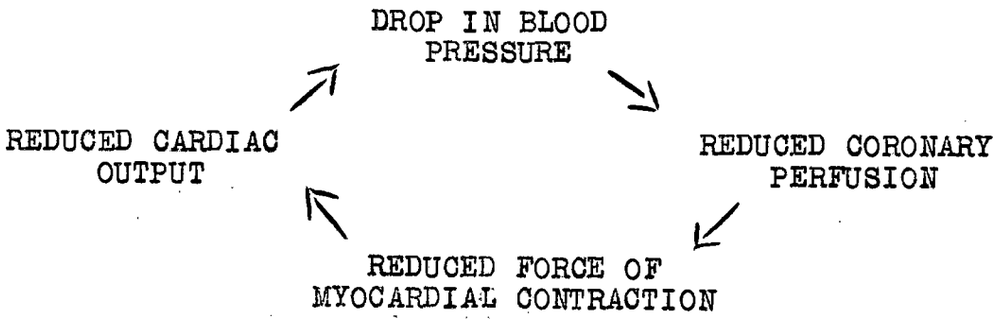
In case 136/2 anoxic anoxia followed the onset of pulmonary oedema due to over-transfusion. This over-transfusion was the result of an error of judgment on the part of the anaesthetist. This error was the acute replacement during operation of blood that

had been lost during the forty-eight hours preceding operation and for which blood loss the patient had already compensated.

B. ISCHAEMIC ANOXIA. - A failure of circulatory homeostasis.

In the presence of a normal uptake of oxygen by the blood, normal oxygenation of the body depends on the maintenance of an adequate perfusion of the tissues. There is presently no practical method applicable in clinical conditions of monitoring tissue blood flow. During clinical anaesthesia the existence of deficient tissue perfusion is deduced from the observation of a persistent lowering of the systolic blood pressure. This is a parameter that can be easily monitored. This deduction, though not always valid especially when the auto-regulatory mechanisms of tissue blood flow are considered, can be defended on the grounds of clinical practicability. The systolic blood pressure is a function of the cardiac output and the peripheral resistance.[108] A fall in blood pressure may reflect a fall in either or both of these parameters. The fact that the blood pressure falls implies that these derangements are no longer compensated. This may be either because they are of too great a magnitude or because the homeostatic mechanisms are depressed or obtunded by the drugs or techniques of anaesthesia. Where, on clinical grounds, this latter is thought to be an important factor in the failure of a patient's circulatory homeostasis, the anaesthetic and its management is regarded as a factor significantly contributory to the patient's death. In acute deaths, such as embraced by this survey, the two organs of which the perfusion has failed are the heart and/or the brain. In the case of the latter, though local auto-regulatory mechanisms are the primary factors controlling blood flow, cerebral blood flow will fall once the systemic systolic blood pressure has fallen below 60 - 70 mm. Hg.[89, 33, 99.] Once ischaemia of

sufficient severity occurs, changes in cerebral cells rapidly become irreversible. In the case of the myocardium the volume of coronary blood flow is very dependant on the mean level of aortic blood pressure and the cardiac rate.[108] In the conditions of clinical anaesthesia a vicious cycle may follow a marked fall in mean systemic blood pressure.



Unless measures to raise the mean blood pressure and so coronary perfusion are adopted, the myocardium may soon suffer ischaemia sufficient to derange its function and result in ventricular fibrillation/cardiac arrest especially in the presence of any pathological narrowing of the coronary arteries.[62] Such derangement of function is often initially reversible. The rapid and correct application of methods of cardiac resuscitation - now standardised[130,110] - will re-establish normal heart beat. Failure to apply such measures timeously is regarded as a fault in anaesthetic and surgical management.

The venous return is an important factor affecting cardiac output. The maintenance of the patient's blood volume and so venous return by the adequate replacement by transfusion of blood lost at operation is considered a duty of the anaesthetist. There are occasions when the rate at which blood is lost from the operative site exceeds the rate at which it is possible for the anaesthetist to replace it. In these circumstances, the death of the patient is regarded as due primarily to a surgical cause- though when death has occurred during operation the case is classified in Group 3.

TABLE 5.

FACTORS ASSOCIATED WITH 12 CASES OF INTRACTABLE HYPOTENSION.

ASSOCIATED FACTORS	Number of Cases	Identity of Cases
Derangement of Cardiac Function	8	3/1, 29/1, 39/1, 52/1, 70/1, 164/1, 7/2, 23/2.
Derangement of Cerebral Function	4	7/1, 8/1, 10/1, 191/1.
Age and Degenerative Vascular Disease. Patients over 65 years of age	6	7/1, 29/1, 39/1, 52/1, 7/2, 23/2.
Pre-operative Hypovolaemia	4	3/1, 39/1, 70/1, 7/2.
Inadequate Replacement of Blood	4	10/1, 39/1, 164/1, 7/2.
Thiopentone Sodium	7	3/1, 7/1, 8/1, 29/1, 191/1, 7/2, 23/2.
Associated use Promethazine and erect posture	1	191/1
Use I.P.P.R.	8	3/1, 7/1, 8/1, 29/1, 39/1, 70/1, 164/1, 7/2
Apparently causally related	1	70/1
Induced Hypotension	1	10/1
Massive Subarachnoid Block	1	52/1
Consciousness.		
Operating room death	5	3/1, 29/1, 52/1, 164/1, 23/2.
Recovered Consciousness	3	39/1, 70/1, 7/2
Failed to regain Consciousness	4	7/1, 8/1, 10/1, 191/1

HYPOTENSION DURING ANAESTHESIA

Of the 51 deaths to which the anaesthetic and its management were considered a significant contributory factor, 12 cases (23.5%) followed episodes of severe and refractory hypotension during anaesthesia. These fall into two main clinical groups depending on whether the resultant ischaemia led primarily to:-

a. Derangement of cardiac function
3/1, 29/1, 39/1, 52/1, 70/1, 164/1, 7/2, 23/2 (8 cases).

b. Derangement of cerebral function
7/1, 8/1, 10/1, 191/1 (4 cases).

Of those in the first group episodes of hypotension were followed by cardiac arrest during operation in all but case 7/2. Five of these seven cases (3/1, 29/1, 52/1, 164/1, 23/2) died in the operating room while two (39/1, 70/1) were successfully resuscitated, survived the operation, regained consciousness after anaesthesia only to die of continuing circulatory failure in the immediate post-operative period. In case 7/2 induction of anaesthesia precipitated inexorable circulatory failure which caused the death of the patient in the immediate post-anaesthetic period. All four cases in the second group, after episodes of hypotension during the anaesthetic, manifested irreversible cerebral damage by a failure to regain consciousness after anaesthesia.

When hypotension is observed to follow the induction of anaesthesia it is often impossible to identify the cause precisely. This is because of the simultaneous or sequential use during the induction of anaesthesia of a number of drugs, any of which may of itself cause hypotension, not to mention the imposition of other factors such as I.P.P.R. and induced respiratory alkalosis. In spite of this it is pertinent to comment on some of the associated clinical factors listed in table 5.

(1) Age and Degenerative Vascular Disease.

Half of these 12 patients (7/1, 29/1, 39/1, 52/1, 7/2, 23/2) were over the age of 65 years. Degenerative vascular disease was a probable associated factor.

(2) Pre-operative Hypovolaemia.

This - a factor stressed recently by Dinnick [82] - was probably present in four cases (3/1, 39/1, 70/1, 7/2). ϕ see Footnote. Though attention to the patient's state of hydration is primarily part of the surgical pre-operative management it has important implications for the safety of clinical anaesthesia. The anaesthetist should himself assess the state of hydration of the patient and his blood volume, in consultation with the surgeon.

(3) Inadequate Replacement of Blood lost during Operation.

This deficiency in clinical anaesthetic management was considered an important factor in four cases (10/1, 39/1, 164/1, 7/2). Case 10/1 underwent a neurosurgical operation for which a technique of induced hypotension was used. The anaesthetist made inadequate provision for the rapid replacement of blood that became necessary during the operation. The resultant profound hypotension caused irreversible ischaemic brain damage. In the remaining three cases it appears from the events described that the anaesthetists concerned were in error in their clinical estimates of blood loss during operation.

ϕ Footnote This was present also in a fifth case, 135/1. Because of vomiting and aspiration during induction of anaesthesia the patient was classed in the anoxic anoxia group, even though hypovolaemia was an important factor in the hypotensive state which ensued.

Consequently, the amounts of blood transfused were in the terms of Hingson et al[98] "too little, too late".

(4) Thiopentone Sodium.

This agent used almost routinely for the induction of anaesthesia is known to cause hypotension.[61, 144, 56, 6.] Seven cases (3/1, 7/1, 8/1, 29/1, 191/1, 7/2, 23/2) displayed progressive hypotension immediately following injection of thiopentone. In most of these, active treatment of the hypotensive condition was tardy and when finally undertaken - usually the administration of a vasopressor drug- proved ineffective. In only one case (3/1) was the actual dose of thiopentone considered excessive. Important associated factors were:-

(a) Age and degenerative vascular disease. Four of these patients were over the age of 65 years. In these, degenerative vascular disease was a probable associated factor.

(b) Pre-operative hypovolaemia was present in two cases (3/1, 7/2).

(c) Obstructive jaundice, with consequent derangement of liver function was a factor in two cases (7/1, 8/1).

Intractable hypotension leading to death following the injection of thiopentone sodium is especially noted by Edwards et al[81] who note no fewer than 107 cases in the first 1,000 deaths reported to them. While Kok and Mullan[168] regard 96 of the 1,000 deaths they reviewed as due to this mechanism. In one other case (122/1) cardiac arrest followed immediately on the injection of thiopentone down a cardiac catheter. This case is classified in the following section under the heading 'Drug induced Cardiac Arrest'. Thiopentone may well have played a part in many other deaths in this study but the immediate association appeared less obvious than in the cases singled out here.

(5) I.P.P.R.

The technique of I.P.P.R. and factors associated with it can lead to hypotension. [121, 122, 143.] An I.P.P.R. technique was associated with 8 of these 12 cases. Because of the multiple factors involved, it was impossible to identify any causal relationship between the commencement of I.P.P.R. and the onset of hypotension except in one case, 70/1. In this case induction of anaesthesia was followed by a period of 35 minutes during which the patient was permitted to breathe spontaneously. During this time there was comparative circulatory homeostasis. Following the institution of I.P.P.R., an immediate deterioration in the patient's circulatory state occurred, ending in cardiac arrest.

This patient was in poor condition and probably hypovolaemic before the induction of anaesthesia.

(6) Massive Spinal Block.

Acute hypotension and fatal cardiac arrest followed induction of caudal extradural block in case 52/1. This appeared to be the result of inadvertent thecal puncture resulting in the occurrence of a massive subarachnoid block.

HYPERTENSION DURING ANAESTHESIA - due to intravenous urea.

In this overall group of deaths due to ischaemic anoxia it is convenient to classify two cases (72/2, 111/2) in which death followed from the effects of massive subarachnoid haemorrhage including probable cerebral ischaemia. This massive subarachnoid haemorrhage was in each case due to rupture of an intercranial berry aneurysm following an episode of hypertension during anaesthesia involving the use of intravenous urea (urovert). The use of intravenous urea as an agent to reduce brain bulk and tension has become a standard ancillary to anaesthetic

techniques for neurosurgery. It is known that before the diuresis commences following administration of the urea, the blood pressure may rise.[142] It is recognised also that intracranial berry aneurysms that have leaked previously may rupture following a rise in blood pressure. While anaesthetising a patient for the operation of ligation of such a berry aneurysm care must be taken to avoid circumstances that might provoke a sudden rise in blood pressure. For these reasons it is thought that following the use of intravenous urea in the anaesthetic technique for such an operation a rise in blood pressure should be anticipated and prevented if necessary. This may be done by the use of drugs such as halothane or ganglioplegic agents. In each of the cases reported here a large rise in systolic blood pressure followed the infusion of intravenous urea. This rise in blood pressure, for which no preventative steps were taken caused rupture of the berry aneurysm, subarachnoid haemorrhage, abandonment of the operation and the subsequent death of the patient. As this reaction to the infusion of intravenous urea may be anticipated and prevented, both these deaths are regarded as preventable.

One other death, 204/1, was the result of an intracerebral haemorrhage which followed episodes of hypertension during aortic surgery. However as anaesthetic responsibility, aspects of preventibility and the part played by the surgical procedure were less clear cut this case was classed in Group 3.

II. DEATHS FOLLOWING CARDIAC ARREST.

- due to DIRECT CAUSES OTHER THAN ANOXIA and those of UNCERTAIN ETIOLOGY.

Though many direct causes of cardiac arrest other than obvious anoxia have been described[130] they do not feature prominently in those deaths to which the anaesthetic and its management was regarded as contributory. This is for the simple

TABLE 6.

ANAESTHETIC CONTRIBUTORY CARDIAC ARREST.

ETIOLOGICAL FACTORS	Number of Cases	Identity of Cases
DIRECT CAUSES OTHER THAN ANOXIA		
Reflex (vagal)	1	11/1
Drug Induced -	2	
Thiopentone (1 case).		122/1
Ether (overdose) (1 case).		130/1
Hypothermia (inadvertent)	1	13/2
Ventricular Tachycardia -	2	
Digitalis induced (1 case).		63/1
Hyperventilation Myocardial Ischaemia (1 case).		100/1
UNCERTAIN ETIOLOGY	7	
Possible Etiological Factors -		
Anoxic Anoxia (2 cases).		46/1, 61/2
Hypotension & Anoxic Anoxia (3 cases).		2/1, 81/1, 90/1
Deep Ether Anaesthesia (1 Case).		84/1
Water Intoxication (1 Case).		182/1
SUMMARY. Cardiac Arrest GROUP 1.		
A. ANOXIC ANOXIA	9	110/2, 71/1, 30/1, 104/1, 12/2, 112/1, 74/2, 84/2, 116/2
B. ISCHAEMIC ANOXIA	7	3/1, 29/1, 39/1, 52/1, 70/1, 164/1, 23/2

reason that such direct causes are usually factors over which the anaesthetist has no direct control. The majority of such deaths are classified in Group 3. Where factors associated directly with the management of the anaesthetic are causally related to cardiac arrest it is usually through the mechanisms of anoxia and/or ischaemic anoxia. In all, of the 51 anaesthetic contributory deaths, 27 were associated with cardiac arrest during operation and anaesthesia. Of these 16 are included in the 37 anaesthetic contributory deaths due to anoxic and ischaemic anoxia already discussed. In a further 4, direct causes other than anoxia could be identified. In another 7 the more precise etiology was uncertain though the anaesthetic and its management were regarded as significantly contributory factors (Table 6).

Reflex Cardiac Arrest.

Only one case, 11/1, appears to be an example of this entity frequently identified in many surveys of cardiac arrest.[158, 145, 172, 159.]

In this case cardiac arrest occurred during a pneumonectomy immediately the bronchus was clamped. There is evidence that there was underventilation with probable hypercarbia and hypoxia.[39.]

Drug Induced Cardiac Arrest.

Though doubtless the action of drugs administered by the anaesthetist may have played a contributory role in many other cases in this survey, in only two did the occurrence of cardiac arrest appear directly related to the administration of a particular drug. In case 122/1 the direct injection of a dose of thiopentone down a cardiac catheter to supplement inadequate basal narcosis caused immediate cardiac arrest.[63.] Case 130/1 was the tragic result of inattention. While using a relaxant-I.P.P.R. technique of anaesthesia for an abdominal hysterectomy, the anaesthetist,

instead of turning the control knob of the 'soda-lime' cannister of a Coxeter-Mushin circle absorption unit, turned on the in-circuit ether vaporiser. Having done so, he failed to notice any signs of the progressively deepening anaesthesia until cardiac arrest from ether overdosage occurred. Case 84/2 may also be regarded as qualifying for inclusion here. In this case cardiac arrest followed immediately on injection of neostigmine for reversal of curarisation. This case has, however, been included with relaxant contributory deaths already discussed.

Hypothermic Cardiac Arrest.

Inadvertent cardiac hypothermia from the rapid transfusion of cold blood [30, 29] was the probable cause, together with circulatory hypovolaemia, of cardiac arrest in case 13/2. It is because this was the result of an error of judgment on the part of the anaesthetist that the anaesthetic and its management is regarded as contributory to this fatality. Inadvertent hypothermia from the massive transfusion of cold blood in cases of massive haemorrhage features prominently as a cause of cardiac arrest in patients classed in Group 3.

Ventricular Tachycardia - Cases 63/1, 100/1.

Though these two patients did not develop cardiac arrest during anaesthesia it is convenient to classify them here as the ventricular tachycardia that caused their death after operation commenced during anaesthesia. In case 63/1 ventricular tachycardia was precipitated by the intravenous administration of digoxin during anaesthesia for anterior resection of the rectum. In case no. 100/1 hyperventilation during induction of anaesthesia for cystoscopy appeared to precipitate its onset. In each case, though the patient regained consciousness after anaesthesia, the ventricular tachycardia proved refractory to treatment and proceeded to ventricular fibrillation in the immediate post-

operative period.

UNCERTAIN ETIOLOGY.

Though the conduct of the anaesthetic was regarded as a contributory factor in the causation of cardiac arrest - and death - in 7 other cases, the more precise etiology of the cardiac arrest was uncertain. Probable causative factors are listed in Table 6.

The inclusion here of case 182/1 illustrates the difficulties that arise in classifying deaths associated with anaesthesia in terms of etiology. This patient was anaesthetised for a trans-urethral resection of prostate. Water intoxication with circulatory overload,[125] together with septicaemia or pyaemia from absorption of bladder irrigating fluid through sectioned infected prostatic venous sinuses, is a probable causative factor in this patient's death. As such it should be classified as being due to surgical causes. But as some aspects of the anaesthetic management, particularly resuscitation, are open to criticism the death is classed together with the anaesthetic contributory deaths. A similar case 129/2, also thought to be due to water intoxication from this same cause, is classed as a surgical death because, in this case, the signs of water intoxication were more obvious while the conduct of the anaesthetic passed scrutiny.

III. MISCELLANEOUS.

Incompatible Blood Transfusion.

During this survey two deaths associated with anaesthesia occurred as a direct result of the transfusion of incompatible blood. In one case 147/1 the error responsible for the patient's receiving incompatible blood was a failure in identification of the patient at the time blood for grouping and cross-matching was taken from the patient. As the anaesthetist was in no way

TABLE 7.

TYPE OF SURGERY, GROUP 3.

OPERATION			Number of Cases	% of Group 3.
CARDIAC SURGERY			31	38
	Closed	20		
	With By-Pass	11		
MAJOR VASCULAR SURGERY			15	19
	Aortic Aneurysm			
	Abdominal	9		
	Thoracic	1		
	Post-op. dehiscence	1		
	Other	4		
MULTIPLE INJURIES			6	7
ABDOMINAL CONDITIONS			9	11
	Acute	6		
	Not Acute	3		
OESOPHAGEAL			3	4
	Varices	2		
	Carcinoma	1		
GYNAECOLOGICAL			5	6
	Genital Carcinoma	4		
	Tubal Insufflation	1		
NEUROSURGICAL			7	9
THORACIC			3	4
	Pulmonary Surgery	1		
	Intrathoracic Haemorrage	2		
OTHER			2	2
	T.U.R.	1		
	Manipulation of hip	1		
	TOTAL		81	100

concerned with, nor responsible for, this procedure, the anaesthetic management was not regarded as contributory to this death.

In the second case 176/1 the error responsible for the transfusion of incompatible blood was a failure on the part of the anaesthetist to identify the patient and to check with care the identity of the blood to be transfused before administration. As this aspect of blood transfusion is definitely within the ambit of the anaesthetist's clinical responsibility, the anaesthetic management is regarded as contributory to this latter patient's death.

CAUSES OF DEATH IN PATIENTS CLASSED IN GROUP 3

Though anaesthesia is not considered contributory, other than unavoidably, to deaths classed in Group 3, as the majority (74%) of these deaths occurred during anaesthesia brief comment on the causes of these deaths is relevant.

Type of Surgery (see Table 7).

Eighty-one deaths were classed in Group 3. No fewer than 46 (57% of the group) involved cardiac or major vascular surgery. Abdominal procedures (11%) were the next most common followed by neurosurgical which were responsible for 9% of cases. Six cases (7%) were associated with operations necessitated by severe multiple injuries.

Probable Causes of Death.

Of 81 cases included in this group, 67 suffered cardiac arrest during operation and anaesthesia. While 60 died in the operating room, 7 were initially successfully resuscitated only to die in the

TABLE 8.

CAUSE OF DEATH - GROUP 3.

CAUSE.	Number of Cases.	Identity of Cases	
		Cardiac Arrest during Surgery.	Post-op. Death. No operative Cardiac Arrest.
Massive Haemorrhage. Massive Transfusion	39	4/1, 165/1, 78/1, 173/1, 127/2, 134/2, 140/2, 13/1, 15/2, 64/1, 98/1, 150/1, 151/1, 83/2, 118/2, 6/2, 93/2, 87/2, 97/2, 119/2, 134/1, 102/1, 9/2, 16/2, 19/2, 77/2, 117/2, 50/2, 98/2, 42/2, 79/2, 81/2, 123/2, 125/2, 64/2, 78/2. (36 cases).	88/1, 51/2, 201/1. (3 cases).
Factors concerned with cardiac surgery. Direct Trauma to heart. Elective cardiac arrest.	17	26/1, 146/1, 179/1, 193/1, 68/2, 35/1, 76/1, 198/1, 113/1, 133/1, 161/1, 194/1, 30/2, 167/1, 92/2. (15 cases).	24/1, 29/2. (2 cases).
Prolonged Hypotension. Irreversible Shock. (metabolic acidosis)	13	168/1, 89/1, 37/1, 143/2, 48/2, 63/2, 88/2, 121/2. (8 cases).	27/1, 48/1, 42/1, 159/1, 56/2. (5 cases).
Coronary Thrombosis	2	25/2, 104/2. (2 cases).	
Pulmonary Embolism	2	126/2, 18/2 (2 cases).	
Air Embolism	1	206/1	
Cerebral Ischaemia. Carotid thrombosis/ stenosis	2	2/2	205/1
Post mitral valvotomy. Cerebral embolus	1		99/1
Pontine Haemorrhage Hypertension during anaesthesia.	1		204/1
Brain Stem Oedema	2	14/1	109/1
Incompatible transfusion	1	149/1	
	81	67 cases.	14 cases.

immediate post-operative period.

The remaining 14 patients died in the post-operative period, most of them failing to regain consciousness. The apparent causes of death are summarised in Table 8.

Massive Haemorrhage
Massive Transfusion.

Occurring in nearly half the cases in this group, 39, massive haemorrhage was the factor most commonly associated with cardiac arrest. The extent to which cardiac arrest and ultimately death were due to hypovolaemia or to the deleterious effects of the concomitant massive transfusion of stored blood [66] was impossible to assess.

One of the more important and more easily monitored effects of massive transfusion - inadvertent hypothermia - [30, 29] was known to be present in just over one third, 14, of the cases of cardiac arrest associated with massive haemorrhage and transfusion (see Table 9). It may have been present in more.

TABLE 9.

INADVERTENT HYPOTHERMIA AND MASSIVE TRANSFUSION

HYPOTHERMIA	Number of Cases	Identity of Cases
PROBABLE (patient felt very cold	5	6/2, 9/2, 16/2, 19/2, 79/2
DEFINITE (Oesophageal Temperature monitored)	9	201/1, 51/2, 77/2, 81/2, 93/2, 97/2, 98/2, 125/2, 123/2
TOTAL	14	

In the group in which oesophageal temperatures were monitored these ranged from 28° C. - 32° C. at the time of cardiac arrest.

Until fairly recently the inadvertent hypothermia that

followed massive rapid transfusions of stored blood was in large measure unavoidable. The means of warming such blood at the speed required and with the necessary safety were not available. However, many devices for this purpose have now been described.[24] In the future when such devices are widely accepted and available the anaesthetic management may be regarded as faulty when a death associated with massive haemorrhage during operation appears to have resulted from the hypothermia of massive transfusion rather than from the hypovolaemia of massive haemorrhage if no means have been adopted to avoid such cooling. Case 79/2 is an example of such a death.

Factors associated with Cardiac Surgery.
Direct Trauma to Heart.

Seventeen deaths were due to factors other than massive haemorrhage associated with cardiac surgery. These will not be commented on further.

Prolonged Hypotension
Irreversible Shock.

Thirteen patients suffered cardiac arrest during operation or died post-operatively after prolonged hypotension not the immediate result of massive haemorrhage. In most instances this could be described as the syndrome of 'irreversible shock'.

In all but four this could be said to be present before anaesthesia was commenced. The surgical lesion most commonly present in this group was peritonitis which was present in four cases whilst a fifth had acute intestinal obstruction. Five cases suffered lesions which were the result of degenerative vascular disease - three had major vascular occlusions and two involved aortic aneurysms. Recent work[177, 34] indicates that metabolic acidosis was the probable common denominator in these thirteen cases. Active treatment of this condition was only undertaken (unsuccessfully) in one of these cases, no. 63/2. Now that the means for the rapid biochemical diagnosis and treatment of this

condition have become more readily available[12] and its clinical importance is realised, the failure to treat it actively may in future be regarded as a fault in the anaesthetic and surgical management, though in these particular cases it cannot be said that such treatment would necessarily have prevented their deaths.

Coronary Thrombosis.

Cardiac arrest was associated with autopsy evidence of recent coronary occlusion in two cases in this Group, 25/2, 104/2. The operation in each case was not of great magnitude, the course of anaesthesia appeared untoward. Blood loss at operation was slight and there were no episodes of hypotension. No satisfactory explanation for the occurrence of these coronary occlusions is apparent in the clinical account of these anaesthetics. Ischaemic heart disease has a profound effect on post-operative mortality.[11] Several of the post-operative deaths, classed in Group 2 in this survey, were the result of myocardial infarction. These are so classed as anaesthetic agents per se appear to have no effect on the incidence of post-operative myocardial infarction and the contributory role that other aspects of the anaesthetic may have is not known.[178]

Pulmonary Embolism.

In two cases cardiac arrest followed pulmonary embolism. It is perhaps worthy of comment that in both cases pulmonary embolism followed movement and manipulation of the patient some days after fractures of the leg.

There is little that requires comment in the remaining cases in this Group except to note that two, nos. 149/1 and 204/1, have been referred to already under the respective headings of 'incompatible transfusion' and 'hypertension during anaesthesia'.

In conclusion it must be realised that in many cases in this Group the assessment of the extent to which the anaesthetic and

its management was contributory to the patient's death and the extent to which this was avoidable was extremely difficult.

SUMMARY.

The causes of the 51 anaesthetic contributory deaths are summarised in Table 10.

Not surprisingly deaths due to anoxic anoxia constitute the greatest proportion, 45%. It must be appreciated that, though these deaths are classified as due to anoxic anoxia, respiratory acidosis must have been coexistent and is synergistic with the effects of anoxia in the production of catastrophe. More than half of the deaths due to anoxic anoxia were the result of respiratory obstruction. A little under a half of these deaths were due to causes related to the use of muscle relaxant drugs - these constituted 17.6% of all anaesthetic contributory deaths. The evaluation of the fact that a substantial proportion of all anaesthetic deaths is related to the use of relaxant drugs is difficult. As will be discussed in Chapter 8, it probably means little more than that the muscle relaxant drugs are now used very often.

Another mode of anaesthetic contributory death that emerges from this survey with some prominence is that which follows progressive, prolonged and intractable hypotension following the induction of anaesthesia. In some cases death followed the precipitation of cardiac arrest by this phenomenom while in others, though cardiac arrest did not supervene during operation, death followed post-operatively from what appeared to be irreversible cerebral damage.

TABLE 10.

CAUSES OF ANAESTHETIC CONTRIBUTORY DEATH.

MECHANISM		CAUSE OF DEATH	Number of Deaths	Percentage of Anaes. Contrib. Deaths.
I	ANOXIC ANOXIA	Atmospheric Anoxia	1	2.0
ANOXIA		Tidal Anoxia		
	45%	Resp. Obstruction.	12	23.5
		Vomiting & Regurg. 3		
		Secretional bronch. obstruc..... 2		
		Endotracheal intubation..... 4		
		Inadequate post-op supervision... 3		
		Relaxant Contributory Deaths	9	17.6
		Post-relax. resp. abnormality..... 8		
		Neostigmine card. arrest..... 1		
		Alveolar Anoxia		
		Pulmonary Oedema	1	2.0
	ISCHAEMIC ANOXIA			
	27%	Prolonged Hypotension Derangement of cardiac function Circulatory failure	8	15.7
		Prolonged Hypotension Cerebral damage (ischaemic)	4	7.8
		Hypertension Ruptured Berry aneurysm Cerebral damage	2	3.9
II				
CARDIAC ARREST (causes other than anoxia)	25%	Direct Causes	4	7.8
		Uncertain Etiology	7	13.7
		Ventricular Tachy-cardia	2	3.9
III				
MISCELLANEOUS	2%	Incompatible blood transfusion	1	2.0
			51	99.9

Hypertension resulting from factors in the anaesthetic technique is also noted as being responsible for anaesthetic contributory death. In two instances, noted here, episodes of hypertension, regarded as preventable, were related to the use of intravenous urea.

Of all the anaesthetic contributory deaths 53% (27 cases) were associated with cardiac arrest during operation and anaesthesia.

It is relevant here to summarise the causes of fatal cardiac arrest during operation and so evaluate the importance of anaesthetic factors in its causation. This is done in Table 11. (Note 7 of these patients survived operation to die in the immediate post-operative period). This shows that factors directly related to the anaesthetic and its management are responsible for 29%, less than one third, of all fatal operating room cardiac arrests. The commonest factors responsible for fatal cardiac arrest during operation and anaesthesia were the associated effects of gross haemorrhage and massive transfusion.

TABLE 11.

CAUSES OF FATAL CARDIAC ARREST DURING OPERATION.

	CAUSES	Number of Cases	% of Cases of Cardiac Arrest
ANAESTHETIC CONTRIBUTORY 29%	Anoxic Anoxia	9	9.5
	Ischaemic Anoxia	7	7.4
	Reflex	1	1.1
	Drug Induced	2	2.1
	Hypothermia	1	1.1
	Uncertain Etiology	7	7.4
OTHER CAUSES 71%	Gross Haemorrhage Massive Transfusion	36	35.3
	Cardiac Surgery (cause other than haemorrhage)	15	15.9
	Prolonged Hypotension	8	8.5
	Pulmonary Embolism	2	2.1
	Air Embolism	1	1.1
	Coronary Thrombosis	2	2.1
	Carotid Thrombosis Cerebral Ischaemia	1	1.1
	Brain Stem Oedema	1	1.1
	Incompatible Transfusion	1	1.1
	TOTAL	94	99.9

CHAPTER 5.

CLASSIFICATION OF SURVEYS OF DEATH ASSOCIATED WITH ANAESTHESIA.

"It is never easy to assess comparative mortality and morbidity figures following anaesthesia ....." (Wylie)[188]

In their survey of 'Deaths associated with anaesthesia and surgery' published in 1954, Beecher and Todd[18] brand such deaths as being of public health concern, computing that twice as many deaths are associated with anaesthesia annually in the United States of America as result from poliomyelitis. This allegation was reiterated on an even wider basis by Campbell[40] in 1960, when he stated that deaths associated with anaesthesia were more numerous "than suicides, homicides and vehicular accidents". The Registrar General of England and Wales,[83] in his report on the state of the nation's health in 1954, computed that deaths associated with anaesthesia were responsible for 1 in every 1,000 deaths occurring in the population. It is against this challenging background that we must examine the incidence of death associated with and that due to anaesthesia as revealed both by this survey at Groote Schuur Hospital and those published by other centres over the years.

Deaths associated with anaesthesia, for comparative purposes, can be considered only in the context of the surgery of the time. The anaesthetic mortality statistics published by those pioneers of anaesthesia, Snow,[164] Buxton,[37] Gardiner,[20, 19] Hewitt[96] and Gwathney,[90] during the latter half of the last century and the early part of this, though of great interest, are

of little value to us for purposes of comparison for surgical practice, of which anaesthesia is the handmaiden, has changed vastly during this time. Probably the most important advances in the last century that have led surgery to progress to its present state have been :-

1. The discovery of anaesthesia.
2. The introduction of aseptic surgical techniques.
3. The use of blood transfusion.
4. An understanding of fluid and electrolyte homeostasis.
5. The discovery of antibiotics.

Though improvements continue in each of these spheres, the basis to all of them can be considered to have been laid by 1930.[162,102] It is from this date, 25 years before the start of this Groote Schuur Hospital Survey, that the incidence of deaths associated with anaesthesia will be examined for comparative purposes.

When examined together, it is immediately apparent that there is a grave lack of comparability between the various surveys of deaths associated with anaesthesia published over the years. This is the result of a lack of uniformity in the classification and assessment of data by various workers and in the varying perioperative time periods surveyed.

1. <u>Assessment</u>.

a) Many authors report only on deaths associated with anaesthesia and make no attempt to identify those more precisely due to anaesthesia or to which anaesthesia may be considered contributory.

b) Because of the very dynamic nature of clinical anaesthesia, etiological diagnoses made in cases of death associated with anaesthesia usually are made retrospectively by the reviewer. Absence of adequate monitoring of vital functions or records thereof in circumstances in which the evidence of subsequent

autopsy helps little, leads to diagnoses being perhaps more subjective than objective. This criticism may be considered as conferring the virtue of objectivity on the former method of reporting. However, though objective, with this former method the net is cast too wide and the result is too unselective for worthwhile inferences to be made.

c) Assessment of the culpability of the anaesthetic in any particular catastrophe depends so much on a concept of the anaesthetist's responsibility that, once again, complete objectivity is impossible to attain. The survey by Hill and Hunter of deaths associated with anaesthesia at the Manchester Royal Infirmary, published in 1948[97] is an example of this. Here the authors adopt a broad and clear classification of -

(1) Deaths due to anaesthesia

(2) Deaths due to operation and disease for which it was performed

(3) Deaths due to other causes

Twenty of 77 deaths are ascribed to anaesthesia. However two deaths due to aspiration of vomitus, to which, on the criteria used in this survey, the anaesthetic would be regarded as contributory, are included in Group 3. The degree of responsibility for the prevention of 'bronchial spill' that is the anaesthetist's, is called in question by the classification of two deaths from this cause in the same section. One is led to question the extent of the contributory role of the anaesthetic in deaths listed in Group 2 under the headings of 'Shock' and 'Thyroidectomy'.

2. Classifications.

In many surveys where etiological diagnoses of the deaths enumerated are made, these are subsequently classified in such a way as to render it difficult, if not impossible, for the reader to distinguish those that may be considered as falling

within the ambit of anaesthetic responsibility and those due to the other major broad factors involved, viz. the condition of the patient, his existing disease, and surgical factors.

For example, Ament[3] classifies cases in his survey under the diagnoses of :

Primary reflex cardiac arrest
Anoxia
Haemorrhage
Drug Toxicity
Pathological deaths
Multiple causes.

Though, on the whole, anaesthetic responsibility may be accepted for deaths falling under the category of anoxia, the degree of anaesthetic responsibility implied by the other headings is impossible to assess. Where causes of cardiac arrest are enumerated, fatal and recovered cardiac arrests are lumped together rendering the mortality rate incomputable. In another example Briggs, Sheldon and Beecher[31] use a similar broad unselective classification of :

Cardiac arrest
Hypoxia
Haemorrhage.

Though from such classification of causes of cardiac arrest, anaesthetic culpability can perhaps be assumed in such categories as 'increasing depth of anaesthesia' and 'hypoxia', no accurate inference can be made from such categories as 'cardiovascular collapse', 'cardiac disease' and 'moribundity'.

3. Peri-operative Time Period included in Survey.

Though all include deaths occurring during the period of operation and anaesthetic itself, published surveys show marked variability in the length of post-operative period of which survey is made for mortality. It will be shown that no comparison between surveys which cover differing peri-operative time periods is valid. The peri-operative time periods covered by the various published surveys vary as follows :-

a) The immediate period of anaesthesia and operation only. Some few of such surveys include deaths occurring in the recovery room as this is usually part of the theatre suite - 'operating room deaths'.

b) To deaths occurring in this immediate period of anaesthesia and operation are added deaths that follow failure of the patient to regain consciousness after anaesthesia. A minor variation of this is the inclusion of those cases only that fail to regain consciousness and die after a cardiac arrest has occurred during anaesthesia.

c) In addition to the mortality occurring during the immediate period of operation and anaesthesia, deaths occurring during an arbitrary immediate post-operative period, most usually 24 hours or the day of, plus the day following operation, are examined. Some surveys also include those deaths that occur after this period in patients who fail to regain consciousness after anaesthesia. It is this time period that is covered by this survey at Groote Schuur Hospital.

d) Some few surveys cover not only the operative but also the entire post-operative period, i.e. they survey the contribution of anaesthesia to operative mortality in toto. This type of survey is perhaps the ideal.

4. Type and Scope of Surgery covered.

There are certain basic skills necessary to the administration of any anaesthetic - even for the most minor surgical procedure. But skill, training and experience in anaesthesia are more severely tested, and the margin of clinical error becomes less, the wider the scope of surgery and the more prolonged the operation involved. Further, the skill and ability of the anaesthetist is, by and large, more severely tested by the poor, inexperienced surgeon than by the skilful. This consideration, the type of surgery conducted in a hospital and the status of the surgical as well as the anaesthetic staff are factors difficult to evaluate, but which must also mitigate against valid comparisons of anaesthetic mortality between the various published surveys.

Though it is the contribution of anaesthesia to all operative mortality in which we are interested, there is some virtue in confining an anaesthetic mortality survey to certain specific standard operations. For example, the survey of Landelius[111] is confined to gastric, biliary and appendix surgery, that of Wylie and Key[190] is confined to thoracic surgery, while many are

confined purely to obstetrical anaesthesia.[84, 85, 73, 128, 50.]

Many surveys are selective in other ways that render comparisons invalid. For example, whilst covering cases in a general hospital, Briggs, Sheldon and Beecher[31] exclude consideration of neurosurgical cases, Lysford[117] of John Hopkins Hospital, specifically excludes Emergency Outpatient Surgery.

5. Type of Anaesthesia.

It is obvious too that inferences as regards the general incidence of anaesthetic death cannot be made from surveys that confine themselves to specific anaesthetic techniques. For example, in their excellent survey on the role of anaesthesia in surgical mortality, Dripps, Lamont and Eckenhoff[55] confine themselves to patients who were anaesthetised with spinal anaesthesia or general anaesthesia involving the use of relaxants.

6. Context.

Because they fail to take cognizance of the background surgical population from which they derive, many surveys of anaesthetic mortality lack context. Though these are of no value at all in defining the size of the problem of deaths due to anaesthesia many are of immense clinical value in elucidating and enumerating the many ways in which anaesthesia or its mis-management may lead to death and how, perhaps, these may be avoided. An example of this type of survey is that sponsored by the Association of Anaesthetists of Great Britain and Ireland on which reports have been published by Edwards et al[81] and Dinnick.[82]

For presentation and comparison, the surveys of death associated with anaesthesia are divided up into broad groups that are considered roughly comparable. Comparison between groups is not considered valid. The surveys presented are classified on the basis of two broad criteria - the 'completeness' of the

survey and the perioperative time period covered.

1. The scope or completeness of the survey -

A. Those surveys are considered 'complete' surveys in which

i. the total number of deaths associated with anaesthesia is recorded.

ii. these are examined and classified as to cause and the contribution to them of anaesthesia:

a) broadly grouped etiologically as -

deaths due to anaesthesia or to which anaesthesia is considered contributory.

deaths due to the surgery undertaken.

deaths due to existing disease.

b) those to which anaesthesia is considered contributory are analysed as to more precise cause, e.g. respiratory obstruction, inadequate blood replacement, etc., and an opinion is formed as to whether they are considered preventable or not.

iii. the number of operations and anaesthetics with which these deaths are associated is recorded.

B. Those surveys are considered 'incomplete' in which one or more of the above desiderata is omitted.

2. The operative and post-operative time period covered by the survey.

A. Those in which the time period covered includes

i. the operative period.

ii. the immediate post-operative period of at least 24 hours or maybe longer.

iii. those patients who, having been conscious before, failed to regain consciousness after anaesthesia even when death occurred later than the arbitrary time period chosen.

B. Those which cover only the operative and anaesthetic period

with the possible inclusion of those patients who failed to regain consciousness after anaesthesia.

Though most of the surveys presented cover all types of surgery, some that are quoted have a limitation of surgery. These will be identified in the Tables presented. Those surveys covering specific anaesthetic techniques only are omitted from these general surveys. In terms of the above criteria, the surveys presented will be grouped as follows :-

1. Complete surveys - all deaths analysed as to cause together with enumeration of background surgical population.
   A.) Those that cover the operation and an immediate post-operative period together with those patients who failed to regain consciousness after anaesthesia.
   B.) Those surveys that cover the operative period only, the so-called 'operating room deaths'.
2. Incomplete surveys.
   A.) Those in which deaths associated with anaesthesia are not analysed as to cause but statistics of the background surgical population are presented.
   B.) Those in which deaths associated with anaesthesia are analysed as to cause but which lack statistics of the background surgical population.

   It has not been thought worthwhile to sub-divide the incomplete surveys on the basis of the perioperative time period covered by them as has been done with the complete surveys.

CHAPTER 6.

THE INCIDENCE OF DEATH ASSOCIATED WITH AND THAT DUE TO ANAESTHESIA.

"As a killer, anaesthesia must be ranked with poliomyelitis, scarlet fever, tetanus, acute rheumatic fever, brain tumours, appendicitis and accidental death from poisoning or shooting". (Davis - 1957).[47]

This present survey of death associated with anaesthesia at Groote Schuur Hospital is one of the group that I have classified as 'complete surveys covering the operative and an arbitrary post-operative period'. There are few similar surveys with which direct comparison can be made. In the course of this chapter the statistics that emerge from this survey will be presented and selected in such a way as to allow of some comparison with the other groups of surveys classified according to the criteria described in Chapter 5. This will demonstrate also how such selection can change the apparent incidence of anaesthetic contributory death from its true value. The incidence of anaesthetic associated and anaesthetic contributory deaths will be expressed as deaths per 1,000 anaesthetics.

It must be noted that in general the incidence of anaesthetic contributory deaths are of the order of one death per 1,000 anaesthetics or less. If any comparisons are to be made, it must be realised that, with all the variables involved in their computation, differences of incidence of less than 0.1 deaths per 1,000 anaesthetics are almost certainly meaningless. Differences of as much as 0.2 deaths per 1,000 anaesthetics would only just pass the test of statistical significance at a 5% level.

If, for example, we compared two surveys of 100,000 cases each, with a mortality rate of the order of 0.5 deaths per 1,000 anaesthetics, the standard error of the difference would be 0.099 per 1,000 demanding a difference between the mortality rates of 0.19 per 1,000 to establish a difference significant at a probability level of 5%. If the surveys we compared comprised 50,000 cases each, the standard error of the difference would be 0.14 per 1,000 requiring a difference between the mortality rates of 0.27 cases per 1,000 to establish a difference significant at a 5% level.

1.A. COMPLETE SURVEYS COVERING THE OPERATIVE AND ARBITRARY POST-OPERATIVE PERIOD.

The incidence of anaesthetic contributory death at Groote Schuur Hospital during the eight years of this survey, together with the relevant statistics, is presented in Table 12.

TABLE 12.

INCIDENCE OF ANAESTHETIC CONTRIBUTORY DEATH AT GROOTE SCHUUR HOSPITAL.

	Number of Anaesthetics.	Anaesthetic Contrib. Deaths	Incidence of Anaesthetic Contrib. deaths per 1,000 anaesthetics.
1st Period (5 years) 1956-1960 incl.	82,960	36	0.43
2nd Period (3 years) 1963-1965 incl.	70,782	15	0.21
1st and 2nd Period (8 years)	153,742	51	0.33

For purposes of comparison, information from eleven similar

surveys together with that from this survey is summarised in Table 13. I regard the incidence of anaesthetic contributory death computed from these surveys as the best estimate of the true incidence of anaesthetic contributory death that we have with the present method of evaluation.

Some entries in this Table require clarification.

In the survey conducted by the Baltimore Anaesthetic Study Committee (Phillips et al)[140], it was found that of 1,024 operative and post-operative deaths reviewed, 196 were considered anaesthetic contributory deaths. These figures are quoted in brackets in Table 13. They considered that cases referred from outside the Baltimore area would distort their estimate of the incidence of anaesthetic contributory deaths. To overcome this difficulty they used for their estimate the number of these cases in which the patient was a resident of Baltimore together with an estimate of the number of operations performed annually in Baltimore. It is these figures which are reflected in the Table.

The effect on the incidence of anaesthetic contributory deaths that differences of classification and assessment of data between authors may have has already been referred to. The survey of Hingson et al[98] is an example of differences that may arise from aspects of classification. In this survey deaths are assessed in relation to anaesthetic culpability as being :-

Group 1. Anaesthesia - the sole cause in mortality.

Group 2. Death due to surgical causes.

Group 3. Anaesthesia - a major factor in mortality.

Group 4. Anaesthesia - a minor factor in mortality.

If Groups 1 and 3 are regarded as reflecting anaesthetic contributory deaths, the incidence would be estimated as 0.3 deaths per 1,000 anaesthetics. If to these the deaths classified in Group 4 are added - and this depends entirely on the author's assessment of 'minor factor' - the incidence of anaesthetic

TABLE 13.

INCIDENCE OF ANAESTHETIC ASSOCIATED AND ANAESTHETIC CONTRIBUTORY DEATH

IA. Complete Surveys which include a Post-Operative Period.

Author. Ref.no. Place. Yr.of Public-ation.	Years inclu-ded in Survey	Number of Anaesths. admini-stered	No.of Deaths		Incidence of Death per 1000 anaesths.		% Anaes. contr./ Anaes. assoc.
			Anaes. assoc.	Anaes. contr.	Anaes. assoc.	Anaes. contr.	
Groote Schuur Hosp.	1st period 1956-60 5 yr.	82960	207	36	2.49	0.43	17.4
	2nd period 1963-65 3 yr.	70782	147	15	2.08	0.21	10.2
South Africa 1966	Period 1 plus 2 8 yrs.	153742	354	51	2.30	0.33	14.4
Kok & Mullan. 168. S.A.1963	1956-62 7 yrs.	1002712	1000	492	0.94	0.49	49.2
Clifton and Hotten. 42. Australia 1963	1952-62 10 yrs.	205640	162	52	0.78	0.25	31.4
Martines-cu 124. Rumania 1962	1951-60 10 yrs.	21469	127	29	5.9	1.34	22.8
Schapira et al. 154. U.S.A. 1960	1952-56 5 yrs.	22177	200	27	9.02	1.21	13.5
Phillips et al. 140. U.S.A. 1960	5½ yrs.	Estima-ted for Balti-more	(1024)	135 (196)		0.4	
Hingson et al. 98. U.S.A. 1956	1945-54 10 yrs.	136043	127	41 (59)	0.93	0.30 (0.43)	32.3
Beecher & Todd. 18. U.S.A. 1954.	1948-52 5 yrs.	599548	7977	384	13.30	0.64	4.8

CONTINUED OVERLEAF.

TABLE 13 (CONTD.)

Author. Ref.no. Place. Yr.of Publication.	Years included in Survey	Number of Anaesths. administered	No.of Deaths		Incidence of Death per 1000 Anaesths.		% Anaes. contr./ Anaes. assoc.
			Anaes. assoc.	Anaes. contr.	Anaes. assoc.	Anaes. contr.	
Dept. Anaesths. Jo'burg. 105. South Africa. 1953	1951-52 2 yrs.	92326	79	8 (40)	1.87	0.19 (0.94)	10 (50)
Brown. 35. Australia 1950	1936-50 15 yrs.	166397	142	133	0.85	0.79	93
Trent & Gaster. 180. U.S.A. 1944	1930-43 12½ yrs.	54128	38	27	0.70	0.50	78.9
Orenstein Committee 169. South Africa 1936	1931-35 5 yrs.	203159	318	177	1.57	0.82	55.6

contributory death would be 0.43 deaths per 1,000 anaesthetics, one third higher than the former. It is the former figures that are listed in the Table whilst the latter are mentioned in brackets.

Of great interest from a South African point of view is the survey of anaesthetic associated deaths at the Johannesburg Hospital.[105] This exemplifies the vast difference in the incidence of anaesthetic contributory deaths computed that may arise from the author's assessment of anaesthetic culpability. In this survey the incidence of anaesthetic contributory death is reported as 0.19 deaths per 1,000 anaesthetics. The clinical details of cases included are appended to the report. If these are assessed by the criteria used in this Groote Schuur Hospital survey 32 deaths, additional to the 8 so assessed by the author, would be regarded as anaesthetic contributory deaths. The additional deaths assessed as anaesthetic contributory arise as follows :-

From Group A - (Pre-operative conditions main factor)
21 of 42 deaths so classified.

From Group B - (Operation main factor)
3 of 19 deaths so classified.

From Group D - (Deaths due to mechanical factors)
all 8 cases so classified.

The addition of these cases would result in the incidence of anaesthetic contributory deaths in this survey being estimated as 0.94 deaths per 1,000 anaesthetics, more than four times greater than that originally reflected. The figures that arise from my assessment of the clinical records appended to the report are included in brackets in the Table.

A South African survey of anaesthetic mortality that is of some importance for purposes of comparison is that conducted by the Orenstein Committee whose report was published in 1936.[169] This was a special Committee under the chairmanship of A.J. Orenstein appointed by the Minister of the Interior to investigate

the causes of the alleged high death rate in the Union of South Africa from anaesthetics at this time. The figures quoted in Table 13 are those given in the annexure to this report. I have regarded as anaesthetic contributory deaths both those listed as 'deaths on the table ascribed solely to the anaesthetic' and 'deaths ascribed to the anaesthetic and shock'. Using this total and the number of operations listed I have estimated the incidence of anaesthetic contributory deaths as shown in the Table (0.82 per 1,000). In their conclusions, however, the Committee used a different estimate of the incidence of anaesthetic contributory deaths (0.93 deaths per 1,000 anaesthetics). This estimate was based on an extrapolation from the proportion of medico-legal autopsies in which death was regarded as due to anaesthesia.

The three South African surveys quoted, those of the Orenstein Committee, the Johannesburg Hospital and Kok and Mullan, included deaths associated with anaesthesia in terms of Section 86 of the Medical, Dental and Pharmacy Act of 1928, while Brown[35] in Australia used the similar Coroners Act as his criterion for inclusion of cases. Of the other surveys, with the exception of Beecher and Todd, the post-operative time period included was, in general, 24 hours or the day of and the day following operation. Beecher and Todd included the whole hospital stay post-operative period in their survey.

The estimates of the incidence of anaesthetic contributory deaths tabulated in Table 13 are illustrated graphically in Figure 4 (the unshaded additions to the Hingson survey and the Johannesburg Hospital survey represent the figures shown in brackets in Table 13).

The incidence of anaesthetic contributory death estimated from these complete surveys and this present one ranges from the 1.34 deaths per 1,000 anaesthetics of Martinescu[124] down to the

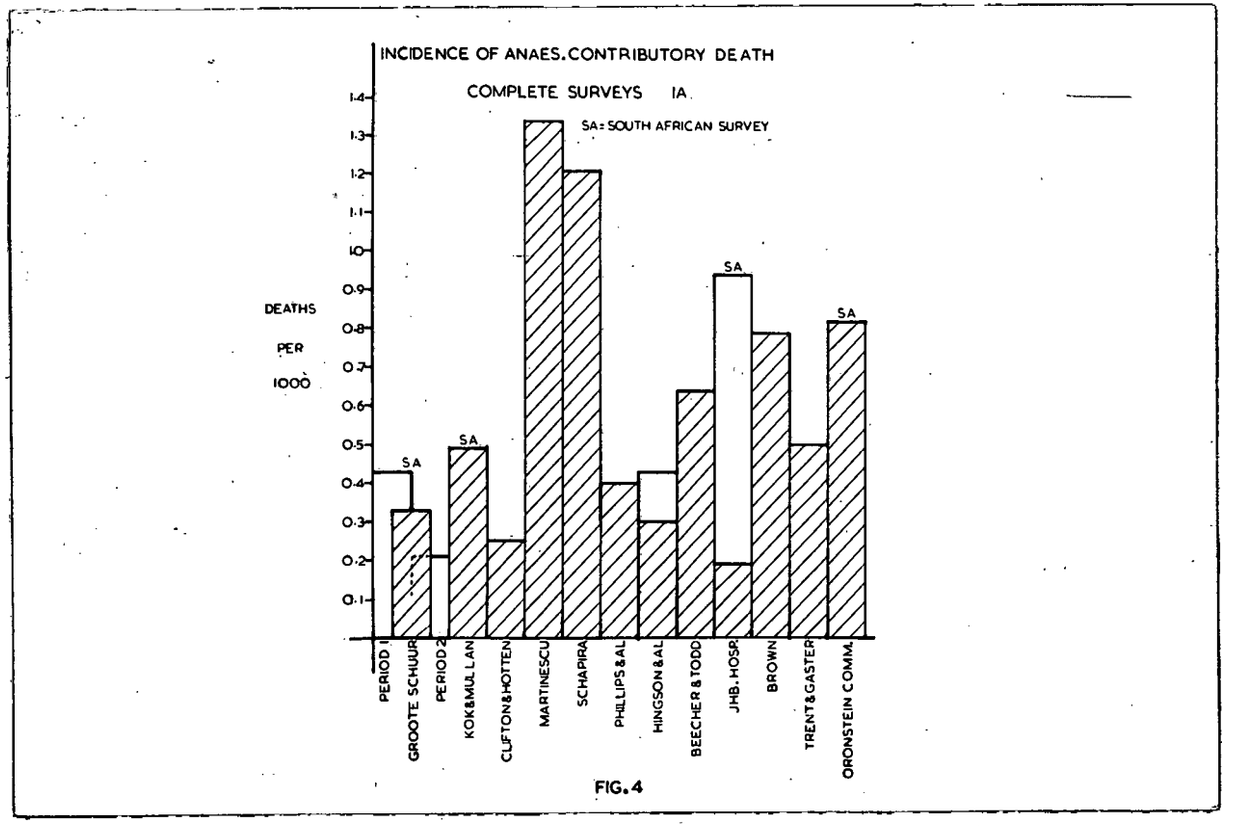

INCIDENCE OF ANAES. CONTRIBUTORY DEATH
COMPLETE SURVEYS IA
SA = SOUTH AFRICAN SURVEY
DEATHS PER 1000
1.4
1.3
1.2
1.1
1.0
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
SA
SA
SA
SA
PERIOD 1
GROOTE SCHUUR
PERIOD 2
KOK & MULLAN
CLIFTON & HOTTEN
MARTINESCU
SCHAPIRA
PHILLIPS & AL
HINGSON & AL
BEECHER & TODD
JHB. HOSP.
BROWN
TRENT & GASTER
ORONSTEIN COMM.

FIG. 4

0.25 deaths per 1,000 anaesthetics of Clifton and Hotten.[42] (For reasons stated the figure of 0.19 per 1,000 given by the Johannesburg Hospital report is not accepted). Though there is little uniformity in the values calculated, it may be noted that of the 12 surveys summarised all but two are less than 1 death per 1,000 anaesthetics while a half, including this present Groote Schuur Hospital survey, give estimates of 0.5 deaths per 1,000 anaesthetics or less.

Of particular South African interest is the fairly close similarity in the incidence of anaesthetic contributory death revealed by this survey at Groote Schuur Hospital (0.33 per 1,000) and that by the contemporary survey conducted by the anaesthetic deaths research unit of the C.S.I.R. (0.49 per 1,000) (Kok and Mullan).[168] In that this latter survey covered all Transvaal Provincial Hospitals, together with some hospitals, including Groote Schuur Hospital, in the other three Provinces of South Africa, and included both teaching and non-teaching hospitals, the slightly lower incidence reflected in this survey could be expected.

The Orenstein Committee, in its report published in 1936,[169] concluded that "in the Union of South Africa deaths due solely to the administration of anaesthetics or in which the administration of anaesthetics was a contributory cause are approximately 0.93 per 1,000 operations. This ratio is higher than in other parts of the world and is one which it should be possible to reduce considerably". Considering the vast changes that have occurred in surgical initiative in the thirty years since this report was published, it is extremely encouraging to note that the incidence of anaesthetic contributory death computed by Kok and Mullan and that from this present survey are but 52% and 30% respectively of this figure.

The publication in 1954 by Beecher and Todd[18] of their study

of death associated with anaesthesia and surgery at ten University hospitals in the United States of America can be regarded, to some extent, as the starting point of this survey at Groote Schuur Hospital. It is apposite, therefore, to note that the incidence of anaesthetic contributory death in this survey is overall approximately one half of that computed by Beecher and Todd though it must be borne in mind that the post-operative period included by Beecher and Todd was longer than that included in this survey. But, as will be shown later, the vast majority of anaesthetic contributory deaths occur during or very shortly after anaesthesia.

1.B. COMPLETE SURVEYS THAT COVER OPERATING ROOM DEATHS ONLY.

The time period to which most investigators confine themselves in surveying deaths associated with anaesthesia is the period of anaesthesia and operation only - the so-called 'operating room deaths'. It is interesting to see how this method of selection reduces the incidence of anaesthetic contributory death reflected from that which results when the immediate post-anaesthetic period is included in the survey. If the cases in the Groote Schuur Hospital survey are selected on the basis of operating-room deaths only, the incidence of anaesthetic contributory death is as follows - (see Table 14).

TABLE 14.

	No. of Anaesthetics	Total op.-room deaths	Anaes. Contrib. op.-room deaths	Anaes. Contrib. op.-room deaths. Incidence per 1,000 anaesthetics
Period 1	82,960	38	13	0.15
Period 2	70,782	41	6	0.08
TOTAL	153,742	79	19	0.12

The fallaciousness of this method of estimating the incidence of anaesthetic contributory death is demonstrated by two observations -

1. With this method of selection some of the more glaring examples of anaesthetic contributory deaths are omitted. Examples of such deaths are cases nos. 8/1, 47/1, 81/1, 84/1, 130/1, 176/1, 80/2, 136/2.

2. The incidence of anaesthetic contributory death estimated with this method of selection (0.12 per 1,000) is only one third, approximately, of the value estimated when a post-operative period is included.

Inclusion of those patients who failed to regain consciousness following administration of an anaesthetic, appears to produce less distortion of the incidence revealed and, certainly in the case of the Groote Schuur Hospital survey, produces a figure that is nearer the true incidence. But selection on this basis does result in the apparent incidence of anaesthetic contributory death being lower than the true incidence. Selected on this basis, the Groote Schuur Hospital survey produces the following figures (see Table 15).

TABLE 15.

	No. of Anaesthetics	Total o.r.d. and f.r.c.	Anaesthetic contrib. o.r.d. and f.r.c.	Anaesthetic contrib. death o.r.d. and f.r.c. Incidence per 1,000 anaesthetics.
Period 1	82,960	65	26	0.32
Period 2	70,782	52	11	0.15
TOTAL	153,742	117	37	0.24

o.r.d. - operating room death.
f.r.c. - failed to regain consciousness.

From this one may infer that both these methods of selection, of which the former especially is widely used, lead to an incorrect estimate of the incidence of death due to anaesthesia.

The results of surveys of operating room deaths are summarised in Table 16. This gives a wider field of reference and comparison for the Groote Schuur Hospital survey. Differences between authors in assessment and classification of data already discussed apply here with equal force. One such example from this group, the survey of Hill and Hunter[97], has already been discussed in the previous chapter. The surveys are arranged chronologically in order of publication.

As I wish to quote but two surveys of operating room deaths which include patients who failed to regain consciousness, these are also included here. The relevant figures from the Groote Schuur survey (Table 15) are included in Table 16 in brackets.

The first of these, the survey of Barlow and associates[16], is of direct interest to us here in South Africa in that it was conducted at the Coronation Native Hospital in Johannesburg. It covers the seven years, 1945 - 1951. The overall incidence of death to which anaesthesia was significantly contributory of 2.29 deaths per 1,000 must be regarded as high. Examination of the annual anaesthetic death rate and closer consideration of the conditions pertaining at the hospital over the period covered, reveals quite strikingly the effect of the factor of availability of adequately trained staff on the anaesthetic mortality rate. This will be discussed later.

The other survey of this type, that of Dornette and Orth[54] conducted at the University of Wisconsin group of hospitals, is also worthy of comment. This survey, which reveals an incidence of death to which anaesthesia was contributory of 0.74 per 1,000 anaesthetics - three times as high as the comparable figure from the Groote Schuur Hospital survey - is from a centre that has consistently shown interest in the problem of deaths associated with anaesthesia. This survey covers the 12 years 1943 - 1954. The ten preceding years - 1933 - 1942 - were

TABLE 16.

INCIDENCE OF ANAESTHETIC ASSOCIATED AND ANAESTHETIC CONTRIBUTORY DEATH

IB. Complete Surveys. Operating Room Deaths

Author. Ref.no. Place. Yr.of Publication	Years included in Survey	Number of Anaesths. administered	No.of Deaths		Incidence of Death per 1000 Anaesths.		% Anaes. contr./ Anaes. assoc.
			Anaes. assoc.	Anaes. contr.	Anaes. assoc.	Anaes. contr.	
Groote Schuur Hosp.	Period 1 1956-60 5 yrs.	82960	38	13	0.46	0.15	
			(65)	(26)	(0.78)	(0.32)	
South Africa. 1966	Period 2 1963-65 3 yrs.	70782	41	6	0.58	0.08	
			(52)	(11)	(0.73)	(0.15)	
	Period 1 plus 2 8 yrs.	153742	79	19	0.51	0.12	24
			(117)	(37)	(0.76)	(0.24)	(31)
Dornette & Orth. 54. U.S.A. 1956	1943-54 12 yrs.	63105	108	47	1.71	0.74	43
Barlow et al. 16. South Africa. 1953.	1945-51 7 yrs.	10909	68	25	6.23	2.29	38
Boba. 26. U.S.A. 1965	1957-62 6 yrs.	61319	146	68	2.37	1.19	46
Natof & Sadove. 133. U.S.A. 1958	1953-56 4 yrs.	10724	23	9	2.14	0.84	38
Jacoby. 103. U.S.A. 1955	1947-54 8 yrs.	54000	34	5	0.63	0.09	14
Carman. 41. E.Africa 1955	1942-54 13 yrs.	9380	8	4	0.84	0.42	50
Dill. 51. U.S.A. 1948	1943-48 6 yrs.	10580	-	5	-	0.47	-

CONTINUED OVERLEAF.

TABLE 16 (CONTD.)

Author. Ref.no. Place. Yr.of Publication	Years included in Survey	Number of Anaesths. administered	No.of Deaths		Incidence of Death per 1000 Anaesths.		% Anaes. contr./ Anaes. assoc.
			Anaes. assoc.	Anaes. contr.	Anaes. assoc.	Anaes. contr.	
Hill & Hunter. 97. England 1948	1934-48 15 yrs.	124500	77	20	0.63	0.16	26
Waters & Gillespie 183. U.S.A. 1944	1933-42 10 yrs.	44891	47	29	1.04	0.65	62
Dealey. 49. Jamaica. 1943	1936-41 6 yrs.	19529	29	18	1.48	0.92	62
Lysford. 117. U.S.A. 1942	1931-41 11 yrs.	51392	75	20	1.5	0.39	27
Yannick. 191. U.S.A. 1939		3000	6	4	2.0	1.33	67
Henson. 95. USA. 1937	1933-36 4 yrs.	193046	-	193	-	1.00	-
Babcock. 14. U.S.A. 1932	1921-31 11 yrs.	90000	26	16	0.29	0.17	62

similarly surveyed by Waters and Gillespie.[183] This earlier survey was confined to operating-room deaths only. The incidence of anaesthetic contributory death was very similar being computed as 0.65 per 1,000 anaesthetics. These two surveys covering roughly similar time periods and conducted in the same group of hospitals are of particular interest in that they have as their dividing line the period of the introduction of curare into clinical anaesthetic practice. In view of the controversy over the influence of curare on anaesthetic mortality which will be discussed later, it is interesting to note that the difference in incidence of death in these two surveys is small, statistically not significant, and may be accounted for entirely by the differences in the method of selection used.

The remaining 10 surveys mentioned here require little comment insofar as they reflect an incidence of anaesthetic contributory death. In the surveys of Boba[26] and Natof and Sadove[133] the accent is on cardiac arrest rather than death in the operating room. I have derived the numbers, and consequently the incidence of operating room deaths and anaesthetic contributory deaths, from their work by subtracting the number of 'recoveries' listed. The surveys of Dill,[51] Dealey[49] and Yannick[191] include only small numbers of cases. In that of Jacoby et al,[103] 15% of the cases included involved local anaesthetic procedures whilst, in contrast, that of Lysford et al[117] excluded all cases conducted with local anaesthetic procedures as also all emergency outpatient surgery.

In terms of the incidence of anaesthetic contributory operating room death in general one may note that of the 15 surveys listed here (including this survey) all but 3 record an incidence of one death per thousand anaesthetics or less while approximately half record an incidence of 0.5 per thousand or less.

These estimates of the incidence of anaesthetic contributory operating room deaths are illustrated graphically in figure 5.

In this brief summary of statistics from complete surveys of deaths associated with anaesthesia, 26 surveys (including this from Groote Schuur Hospital) have been presented. Looking at the incidence of anaesthetic contributory death revealed by these surveys it is obvious that we cannot validly deduce an estimate of an overall general incidence of anaesthetic contributory death. Too many variable factors are involved in their computation. These have been discussed. The most that can be said is that of these twenty-six surveys all except five produce an estimate of the incidence of anaesthetic contributory death that is one per thousand or less whilst half of these surveys (including this survey and that of Kok and Mullan) produce an estimate of 0.5 deaths per 1,000 anaesthetics or less.

2. INCOMPLETE SURVEYS.

Regrettably the majority of surveys of deaths associated with anaesthesia published since 1930, as do most surveys published before this date, fall into the group I call 'incomplete surveys. They are incomplete in that the deaths are -

a. either not classified as to cause or are classified as to cause in such a manner that it is impossible to evaluate the role of the management of the anaesthetic per se. Examples of these have already been given.

b. classified as to cause but no cognisance is taken of the background surgical population from which they arise.

The purpose of many of these studies was an elucidation of

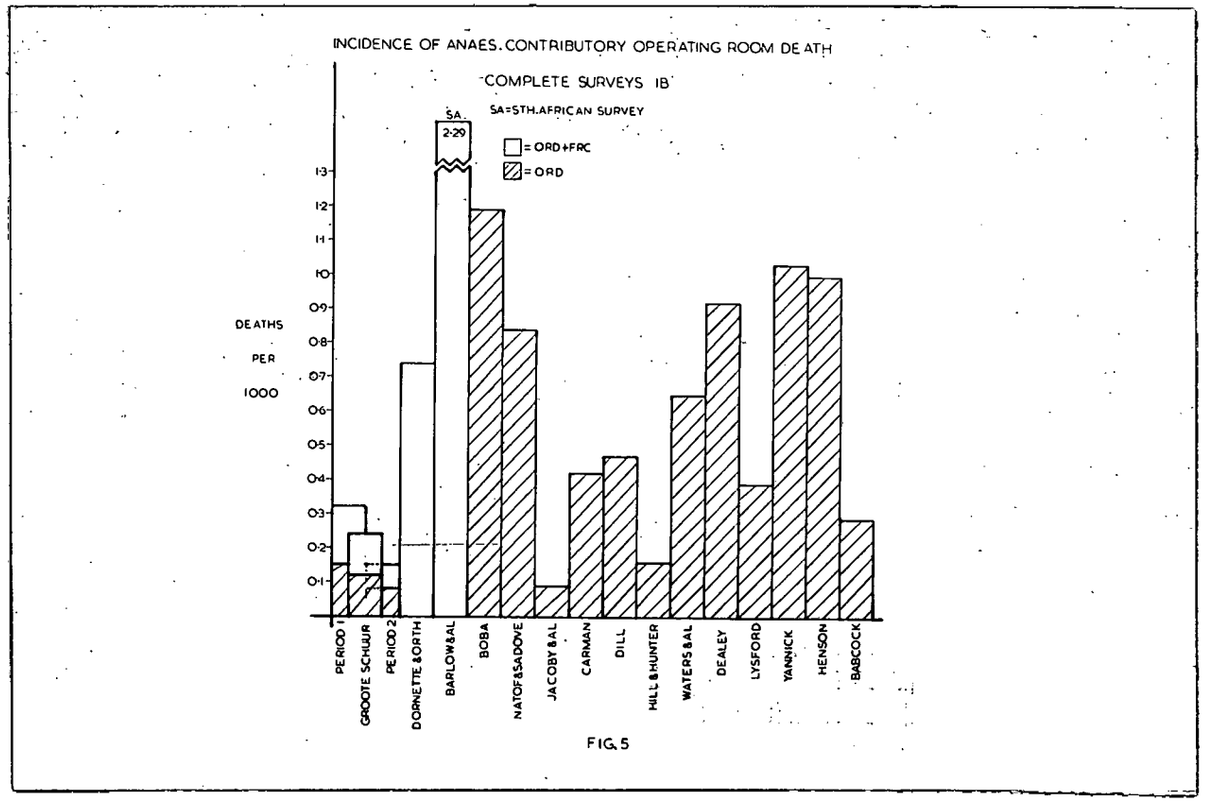

INCIDENCE OF ANAES. CONTRIBUTORY OPERATING ROOM DEATH
COMPLETE SURVEYS IB
SA=STH. AFRICAN SURVEY
= ORD+FRC
= ORD
SA 2·29
DEATHS PER 1000
1·3
1·2
1·1
1·0
0·9
0·8
0·7
0·6
0·5
0·4
0·3
0·2
0·1
PERIOD 1
GROOTE SCHUUR
PERIOD 2
DORNETTE & ORTH
BARLOW & AL
BOBA
NATOF & SADOVE
JACOBY & AL
CARMAN
DILL
HILL & HUNTER
WATERS & AL
DEALEY
LYSFORD
YANNICK
HENSON
BABCOCK

FIG. 5

the causes of death during anaesthesia and operation. As such some are of immense clinical value. But these studies are of no value in estimating the precise incidence of anaesthetic contributory death. From those in the former group one can estimate the incidence of death simply associated with anaesthesia and operation. This can be regarded as an index of crude operative mortality. Though this embraces deaths that result from the patient's disease, the operation and the anaesthetic, it is to some extent an index of overall surgical and anaesthetic standards. From studies in the latter group it is possible to estimate the proportion of deaths associated with anaesthesia and surgery which are due to anaesthesia. This can to some extent be regarded as an index of the importance of anaesthesia in surgical mortality.

The peri-operative time period included in these surveys varies from that of the period of operation and anaesthesia with the possible inclusion of those patients who failed to regain consciousness, to the inclusion of an arbitrary post-operative period. In many surveys the precise peri-operative time period included is not clear. All are presented together here.

2.A. INCOMPLETE SURVEYS NOT CLASSIFIED AS TO CAUSE.

I must stress again the marked effect that the varying peri-operative time period included in these studies has on the incidence of anaesthetic associated death computed as it has on that of the anaesthetic contributory deaths of the previous section. To illustrate this point the statistics from this Groote Schuur Hospital survey are selected and presented in Table 17 in relation to the inclusion of varying peri-operative time periods as -

1. period of operation, 24 hour post-operative period and patients who failed to regain consciousness.

2. period of operation only and patients who failed to regain consciousness.

3. period of operation only.

TABLE 17.

INCIDENCE OF ANAESTHETIC ASSOCIATED DEATH AT GROOTE SCHUUR HOSPITAL

EFFECT OF PERI-OPERATIVE TIME PERIOD INCLUDED IN SURVEY

	Number of Anaesthetics	Number of Anaesthetic Associated Deaths	Incidence per thousand of Anaesthetic Associated Deaths.
Period 1	82,960	1. 207 2. 65 3. 38	1. 2.49 2. 0.78 3. 0.45
Period 2	70,782	1. 147 2. 52 3. 41	1. 2.08 2. 0.73 3. 0.57
TOTAL (Periods 1 and 2)	153,742	1. 354 2. 117 3. 79	1. 2.30 2. 0.76 3. 0.51

Peri-operative Time Period.

1. Period of operation, 24 hours post-operatively and patients who fail to regain consciousness.

2. Period of operation only and patients who fail to regain consciousness.

3. Period of operation only - operating room deaths.

Omission of the deaths in the immediate post-operative period results in the estimate of the incidence of anaesthetic associated death being one third or one quarter of that which results with such inclusion depending on whether patients who fail to regain consciousness are included or not.

Statistics from 19 surveys of this type and estimates of the incidence of anaesthetic associated mortality are tabulated

chronologically in Table 18. For comparison similar figures from Group 1 surveys are included in Tables 13 and 16.

The effect that the inclusion of certain special risk categories of surgery may have on the crude anaesthetic associated death rate must also be borne in mind, e.g. cardiac surgery with cardio-pulmonary by-pass or major vascular surgery. Such surgery is included in this survey but not in the majority of others quoted.

The incidence of anaesthetic associated death computed from these surveys together with the similar estimates from the Group 1 surveys (see Tables 13 and 16) is illustrated for comparison in figure 6. Surveys including more than the twenty-four post-operative period are omitted from this histogram as also is the survey by Muir at Groote Schuur Hospital, as this is restricted. Whilst the vast majority of these surveys compute an incidence of anaesthetic associated death of 2 per 1,000 or less, approximately half of them (including this survey selected on an operating-room death basis) show an incidence of the order of 1 per 1,000 or less.

However, with the number and magnitude of the variables inherent in these estimates there is little value in any absolute comparisons between surveys from different centres and at different times.

Two surveys quoted in Table 18 are of South African interest. Firstly that of Melzer[126] published as a Thesis presented to Witwatersrand University in 1947. He examined, classified as to cause and commented on a consecutive series of 371 deaths occurring in association with anaesthesia in Johannesburg and presenting for medico-legal autopsy between the years 1941 - 1945. To obtain some indication of actual incidence of death associated with anaesthesia, to give his autopsy study some context, he computed the incidence of death during surgery at

TABLE 18.

INCIDENCE OF ANAESTHETIC ASSOCIATED DEATH.

2A. Incomplete Surveys not classified as to Cause.

Author. Ref.no. Place. Year of Publi-cation	Years included in Survey	Number of Anaesths. admini-stered.	Anaes. associated Deaths. Number.	Incidence per 1000 anaesths.	Remarks.
Groote Schuur Hosp. S.Afr. 1966	1956-60 1963-65 8 yrs.	153742	1. 355	2.30	1. o.r.d. 24 hrs.post-op. & f.r.c.
			2. 117	0.76	2. o.r.d. & f.r.c.
			3. 79	0.51	3. o.r.d.
Ament. 3. U.S.A. 1960	1949-58 10 yrs.	49618	79	1.59	o.r.d.
Mannix. 123 U.S.A. 1960	1947-58 12 yrs.	56000	21	0.38	o.r.d.
Shroff. 157. U.S.A. 1959	1948-57 10 yrs.	57132	59	1.03	o.r.d. & recovery room.
Briggs Sheldon and Beecher. 31.U.S.A. 1956.	1924-54 30 yrs.	189815	174	0.91	o.r.d.
Lewis & Brown. 113. U.S.A. 1955.	1943-54 12 yrs.	11705	17	1.5	o.r.d.
Carman. 41. E.Afr. 1955.	1934-37 4 yrs.	26357	29	1.1	post-op. period not stated.
Dillon. 52. U.S.A. 1949	1946-48 3 yrs.	28000	46	1.64	o.r.d.
Melzer. 126. S.Afr. 1947	1941-45 5 yrs.	63515	181	2.85	o.r.d.
Bishop 25. U.S.A. 1946.	4½ years.	20021	6	0.29	o.r.d.

CONTINUED OVERLEAF.

TABLE 18 (CONTD.)

Author. Ref.no. Place. Year of Publi-cation	Years included in Survey	Number of Anaesths. admini-stered	Anaes. Deaths. Number.	Assoc. Incidence per 1000 anaesths.	Remarks.
Gillespie 68. U.S.A. 1944	1932-42 10 yrs.	1.13011	1. 7	1. 0.53	o.r.d. 1. One hospital
		2.227546	2.283	2. 1.2	2. Many hospitals
Muir. 132 Groote Schuur Hosp. S.Afr. 1939	1938-39 1½ years.	2216	14	6.22	o.r.d.
Edwards. 60. England. 1938	1923-27	11871	30	2.52	o.r.d.
	1933-37	13821	21	1.51	
Kaye. 13. Australia. 1938	1932-36 5 yrs.	193977	89	0.46	post-op. period not stated. 8 hospitals.
Kaye. 13. Australia. 1938.	1932-36 5 yrs.	28313	22	0.77	post-op. period not stated. 1 hospital.
Kaye. 107. Australia. 1935	1919-29	13400	24	1.8	o.r.d. in-patients only.
	1929-34	17757	23	1.3	
Lotz. 114. U.S.A. 1937	1917-36 20 yrs.	32883	12	0.36	o.r.d.
Sykes. 174. England. 1933.	1-10 yrs.	266195	145	0.54	o.r.d.
Bortone 27 U.S.A. 1932	1930 1 yr.	39081	24	0.61	post-op. period not stated.
Rollison. 148. Australia. 1930.	1926-28 1½ yrs.	8043	6	0.74	o.r.d. & 24 hrs.

Note: f.r.c. - failed to regain consciousness.
o.r.d. - operating room death.

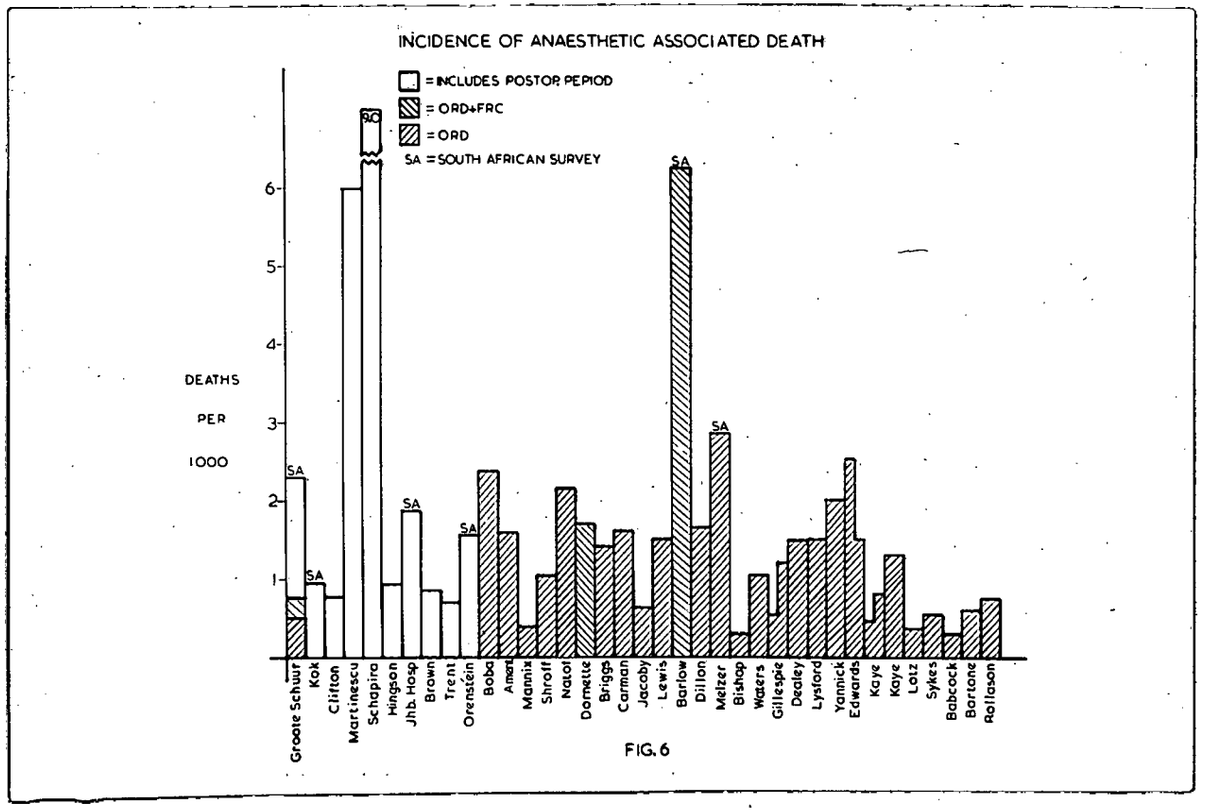

INCIDENCE OF ANAESTHETIC ASSOCIATED DEATH
= INCLUDES POSTOP PERIOD
= ORD+FRC
= ORD
SA = SOUTH AFRICAN SURVEY
DEATHS PER 1000
1
2
3
4
5
6
SA
SA
SA
SA
SA
SA
Groote Schuur
Kok
Clifton
Martinescu
Schapiro
Hingson
Jhb. Hosp
Brown
Trent
Orenstein
Boba
Amat
Mannix
Shroff
Natal
Domette
Briggs
Carman
Jacoby
Lewis
Barlow
Dillon
Melzer
Bishop
Waters
Gillespie
Dealey
Lysford
Yannick
Edwards
Kaye
Kaye
Latz
Sykes
Babcock
Bartone
Rollason

FIG. 6

the Witwatersrand University Medical School Teaching Hospitals Group during the same five years. The incidence computed by him of 2.8 deaths per 1,000 anaesthetics must be regarded as high. An analysis of these figures on a basis of the race of the patient, European as against Non-European, reveals a marked difference in incidence. Whereas the death rate ascribed to anaesthesia and surgery in the European patients was 1.48 deaths per 1,000 anaesthetics, an incidence which, though high, is of an order with that computed at other medical centres, the death rate in Non-European patients was 6.7 deaths per 1,000 anaesthetics - an incidence that would have given an Orenstein Committee food for thought. This figure, taken in conjunction with those published subsequently by Barlow et al,[16] already referred to, must be taken to indicate that, amongst many factors responsible, the provision of anaesthetic services in the Non-European hospitals must have been woefully inadequate at this time. This is a situation, one must hasten to add, that has been greatly improved since.

The rather rough and ready survey of Muir[132] is of great interest in that it was carried out 17 years before the start of this present survey in the self-same hospital. The survey was restricted to surgery falling into the broad general surgical field, the surgical specialities of orthopaedic surgery, urological surgery, gynaecological surgery, ophthalmological surgery and otorhinolaryngological surgery and casualty outpatient surgery being excluded. The high rate computed of 6.22 deaths per 1,000 cases is therefore not a true reflection of the overall rate of deaths associated with anaesthesia at that time. Selected on a similar basis, the incidence of death associated with anaesthesia that emerges from this present survey is 1.10 per 1,000 even with the inclusion of major vascular surgery which was not undertaken

in 1938.

2.B. INCOMPLETE SURVEYS, CLASSIFIED AS TO CAUSE - NO BACKGROUND DATA.

Lastly, in the surveys presented, is that large group that are incomplete in that the background figures of the number of anaesthetics, or operations from which they arise, are not known. Included in this group are a number of surveys based on the findings of autopsies conducted on patients who died during the course of an anaesthetic. Many of these autopsies were conducted in compliance with local legislation such as Section 86 of the South African Medical, Dental and Pharmacy Act of 1928 or the English or Australian Coroners' Laws which require that patients dying during the course of an anaesthetic be submitted to autopsy.

These surveys (summaries of which are presented in Table 19), of no use in establishing the incidence of anaesthetic contributory death, are often of immense clinical value in that they identify the factors and mistakes in clinical anaesthesia that do result in death. What can be computed from these surveys, as from the complete surveys, is the proportion of deaths associated with anaesthesia and operation for which anaesthesia itself is responsible, or, in other words, the relative role of anaesthesia in immediate surgical mortality. This proportion as a percentage is estimated as :-

$$\frac{\text{Number of deaths to which anaesthesia is contributory}}{\text{Total Number of deaths associated with anaesthesia}} \times 100$$

This index might almost be regarded as an index of the quality of the anaesthetic service. It should be noted that statistics from the surveys of Carman[41], Melzer[126], Bishop[25] and Kaye[107] each appear in more than one of the four tables of

TABLE 19.

ROLE OF ANAESTHETIC CONTRIBUTORY DEATH IN IMMEDIATE OPERATIVE MORTALITY.

2B. Incomplete Surveys - No Background Data.

Author. Ref.no. Place. Year of Publi-cation	Years included in Survey	DEATHS. Anaes. Assoc.	DEATHS. Anaes. contr.	% Anaes. contr./ Anaes. assoc.	REMARKS.
Groote Schuur Hosp. S.Afr. 1966	1956-60 1963-65 8 yrs.	355	51	14	Includes post-op. period 24 hrs.
Dilworth. 53 Australia 1965	1953-62 10 yrs.	80	31	39	Restricted to children. Total post-op. period.
Gain. 65. Canada. 1955.	1953	168	26	15	Includes 30 days post-op.
N.S.W. Committee on Deaths. 134. Australia. 1962	1960-62 2 yrs.	94	54	58	Australian Coroners Act.
A.A.G.B.I. Committee, England.					
Edwards. 81. 1956	1949-55	1000	589	59	Voluntary submission.
Dinnick 82. 1964	1956-63	600	400	66	
Pallin 137 U.S.A. 1951	1949-50 2 yrs.	103	23	23	Includes 1st post-op. day.
Ruth 150 U.S.A. 1947	11 yrs.	306	43	14	Includes day of and day following.
Bishop. 25. U.S.A. 1946	1933-36 4 yrs.	398	193	48	Operating Room deaths.

CONTINUED OVERLEAF.

TABLE 19. (CONTD.)

Author. Ref.no. Place. Year of Publi-cation	Years included in Survey	DEATHS. Anaes. assoc.	Anaes. contr.	% Anaes. contr./ Anaes. assoc.	REMARKS.
Campbell. 40. U.S.A. 1960	1957-59 3 yrs.	195	20	10	BASED ON PATIENTS PRESENTING FOR AUTOPSY.
Weiss. 185. U.S.A. 1960	1957-59 3 yrs.	200	57	28	
Simpson. 161. England. 1960	10 yrs.	100	26	26	
Simpson. 160. England. 1953		500	72	14	
Melzer. 126. S.Afr. 1947	1941-45	371	331	89	
Turner and Wilkinson. 181. Canada. 1942.	1931-40 10 yrs.	23	2	8	
Kaye. 107 Australia. 1935.	1929-34	22	14	64	

anaesthetic associated and contributory mortality. This follows the inclusion in each of these surveys of statistics appropriate to two of the different groupings used for these tables.

The value of the above calculation is, however, lessened by the selection that enters into many of the surveys. Deaths to which anaesthesia is contributory are acute. In general, therefore, one would expect the longer the post-operative period surveyed, the lower would be the proportion of surgical mortality for which anaesthesia could be held responsible. Conversely, the shorter the period surveyed the greater this proportion would be. In surveys based on the examination of deaths "occurring during anaesthesia or to which anaesthesia may have been contributory"[74] such as three of the South African surveys quoted, that of Orenstein,[169] Melzer[126] and Kok and Mullan[168] or that of the New South Wales Study Committee[134] and many of those based on autopsies, the percentage reflected of deaths for which anaesthesia is responsible, will be higher. The voluntary submission of information on deaths associated with anaesthesia, such as that used in the enquiry conducted by the Association of Anaesthetists of Great Britain and Ireland,[81,82] will result in similar selection. In this survey at Groote Schuur Hospital for the peri-operative period examined, i.e. the period of operation and 24 hours post-operatively, anaesthesia was considered a contributory factor in 14.4% of the anaesthetic associated deaths. The effect of varying the peri-operative time period surveyed is again apparent. If operating room deaths only are examined, anaesthesia was considered contributory to 24% while if this period is extended to include those patients who failed to regain consciousness, it was contributory to 31%.

When this quantitation of the role of anaesthesia in operative surgical mortality calculated from these surveys and

those in Group 1 (Tables 13 and 16) is examined generally the vast disparity that is evident between these surveys in all the aspects of this problem that have been discussed, is again the most striking feature.

Few surveys of anaesthetic mortality conform to the ideal of examining and relating anaesthetic mortality to the general overall operative mortality - mortality occurring within 30 days of operation. In addition to the classic survey of Beecher and Todd[18] already quoted in Group 1.A. in which anaesthetic contributory deaths were found responsible for 4.8% of the total surgical operative mortality, two other surveys are quoted here, those of Dilworth[53] in Western Australia and Gain[65] in Alberta, Canada. That of Dilworth, though not strictly comparable to the other surveys quoted here, in that it is restricted, is important to mention. This survey, conducted in Western Australia, was restricted to children under the age of 15 years who had undergone operations for potentially curable surgical lesions. Computing that anaesthesia and its mismanagement was responsible for 39% of the total surgical mortality in the surgery of children, she came to the challenging conclusion that anaesthesia is the biggest single cause of death in operations for potentially curable surgical lesions.

A particular look at this aspect of the surveys conducted in South Africa is of interest. The Orenstein Committee of 1936 found anaesthesia responsible for or contributory to 55.6% of operative mortality. Subsequent surveys, except that of Melzer, have found this proportion to be lower. Both the survey conducted at the Johannesburg Teaching Hospitals Group in 1951/52 and that larger and longer term survey of Kok and Mullan, show a very similar proportion, namely, of 50% (on my assessment) and 49% respectively. Though the actual anaesthetic death rate was high in the survey of Barlow et al at Coronation

Hospital, Johannesburg, the proportion of immediate surgical mortality for which anaesthesia was responsible was 38%. The very high proportion of operative deaths for which anaesthesia was responsible in the survey of Melzer - 89% - though perhaps indicative of a poor standard of anaesthetic practice at the time, was no doubt due in some measure, for reasons already discussed, to selection inherent in the way cases were included in the study.

PREVENTABILITY OF ANAESTHETIC CONTRIBUTORY DEATHS.

"There are possibly no accidents in the theatre that are not avoidable by any precaution that a reasonable man might take". (Simpson).[161]

In judging the preventability of anaesthetic contributory deaths we are bedevilled, as in all other aspects of this problem, by the difficulties inherent in the subjective assessment of clinical situations. This difficulty is exemplified by the classification used by two Australian workers in this field, Kaye[107] and Brown[35] -

Frankly preventable
Probably preventable
Possibly preventable
Possibly unavoidable
Unavoidable
Undetermined.

In the assessment of the preventability of deaths I have used three categories only -

Probably preventable

Possibly preventable

No verdict

On this basis the 51 anaesthetic contributory deaths in this survey were classified as follows :-

TABLE 20.

PREVENTABILITY OF ANAESTHETIC CONTRIBUTORY DEATHS.

Grade of Preventability	Number of Deaths	Percentage of Deaths	
Probably	18	35.3	92.1
Possibly	29	56.8	
No Verdict	4	7.9	
TOTAL	51	100	

To give these figures a more practical context, we may say that during the period of this survey at Groote Schuur Hospital, anaesthesia was contributory to 2.7% of the total operative and post-operative deaths (1,749) in a manner that was probably or possibly preventable. Consideration of anaesthetic mortality on this basis clearly indicates the importance of anaesthesia in the total surgical mortality.

Though most writers on the subject of anaesthetic deaths comment either directly or by implication on this aspect of preventability of anaesthetic mortality, very few actually calculate the proportion of deaths that are. Of the 55 surveys that have been quoted in this section, in only 8 is the proportion of anaesthetic deaths that are preventable quantitated. These estimates are summarised in the Histogram Figure 7. In all but two surveys the proportion of anaesthetic contributory deaths regarded as probably or possibly preventable was 50% or over.

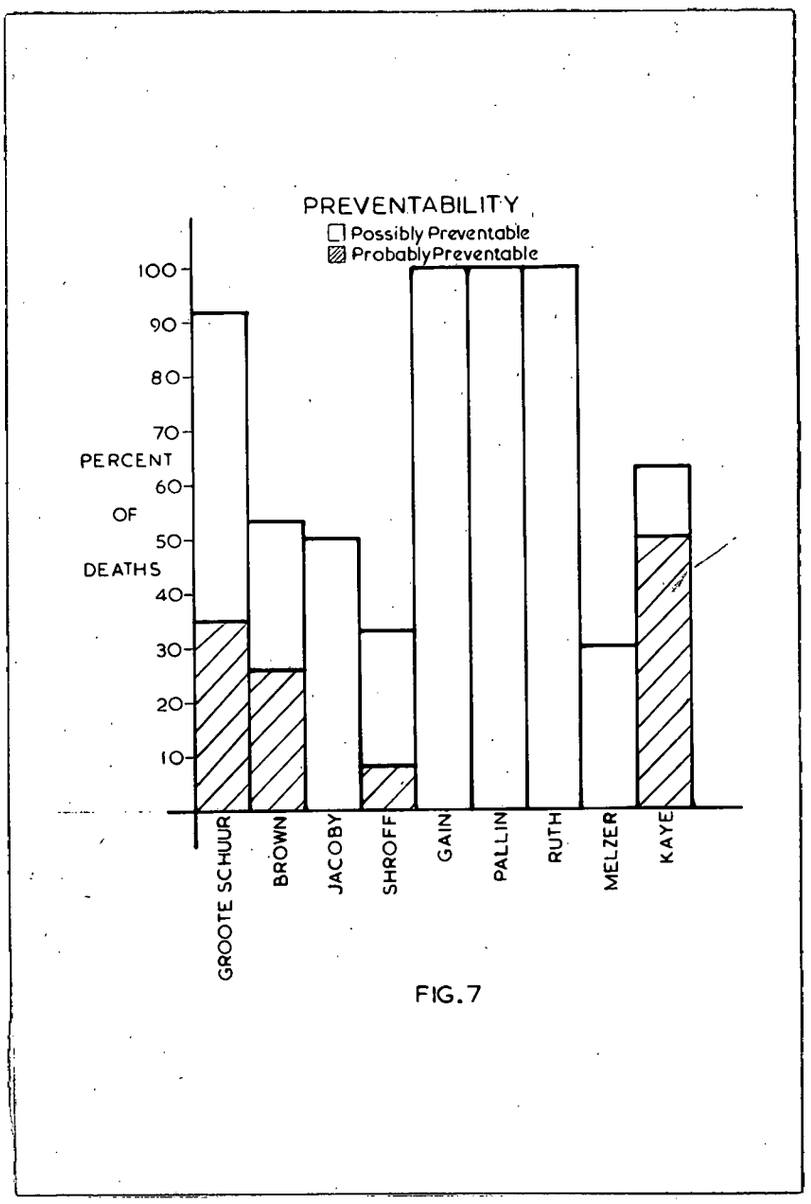

PREVENTABILITY
Possibly Preventable
Probably Preventable
PERCENT OF DEATHS
100
90
80
70
60
50
40
30
20
10
GROOTE SCHUUR
BROWN
JACOBY
SHROFF
GAIN
PALLIN
RUTH
MELZER
KAYE

FIG. 7

The two that reflect a slightly lower figure, those of Shroff[157] and Melzer[136] were not concerned with anaesthetic deaths alone but with operating room mortality. Ruth,[149] Pallin,[137] and Gain[65] regarded all the cases that they classed as anaesthetic contributory deaths as preventable. Dependent, as it is finally on the essentially subjective approach by which these cases are judged, the precise proportion of death considered preventable is not important. What is important is that all workers in this field, even those who attempt no concrete estimation of it, regard the major proportion of deaths to which anaesthesia is considered contributory to be of a type for which "a reasonably satisfactory explanation can be provided and for which effective countermeasures are practicable". (Morton).[131]

SUMMARY.

The incidence of anaesthetic contributory death is the yardstick by which we may measure the standard and quality of clinical anaesthetic practice. As such it is of value to compare that which we are evaluating in this survey with that of other centres. Regrettably a great number of variable factors influence this estimate of anaesthetic contributory mortality. It is impossible to assess how far differences in the anaesthetic contributory mortality rate between centres and at different times reflect differences in the standard of clinical anaesthetic practice or in what degree they reflect the influence of the variable factors discussed.

An attempt has been made to match the anaesthetic contributory mortality rate estimated from an 8 year survey at Groote Schuur Hospital with that from other centres. This has

been done in terms of

(1) Anaesthetic contributory mortality -

(a) estimated from deaths occurring in the operative and a 24 hour post-operative period.

(b) estimated from operating room deaths only.

(2) Crude anaesthetic associated mortality.

(3) The proportion of immediate surgical operative mortality due to anaesthetics.

In this comparison special reference has been made to other surveys conducted in South Africa.

Though only the broadest comparisons are possible, the standard of clinical anaesthetic practice reflected by this survey at Groote Schuur Hospital compares more than favourably with that of other countries.

When examined in relation to their 'preventability' the vast majority of anaesthetic contributory deaths are either probably or possibly preventable.

CHAPTER 7.

A MEASURE OF SAFETY.

"Given a properly trained and equipped anaesthetist, mortality and morbidity will fall regardless of the agent, technique or patient's condition". (Paul Wood as quoted by Gwathney).[91]

The aim of anaesthesia has always been to make surgery not only painless but safer for the patient. That advances in anaesthesia have allowed of improved operative access to any part of the body and thus encouraged ever greater surgical initiative is obvious. But the most important question we may ask is "has such advance in anaesthesia resulted in greater safety to the patient?". This is a most difficult question to answer in categorical terms. For as anaesthesia permits of surgical advance, indications for operation are widened and the surgeon presents an ever greater challenge to the anaesthetist in the type of patient he presents for anaesthesia for operations of increasing scope and complexity.

Examination of the surveys of anaesthetic mortality presented here shows so wide a disparity in the incidence reflected between various centres that no general trend is apparent with time. Considering the variables involved in their computation, this is not surprising. The optimists might regard the fact that there is no gross increase apparent in the incidence of anaesthetic mortality in the face of advancing surgical initiative as an indication of a reduction in anaesthetic mortality. It is salutory to remember that the

Anaesthetics Committee of the British Medical Association,[32] reporting in 1900 on their survey of all cases of administration of anaesthetics in England in the year 1892 - of which they collected details of 25,920 cases - could record an overall rate of death to which anaesthesia was considered contributory, of 1.11 per 1,000 cases out of a mortality of 3.12 deaths per 1,000 cases generally associated with anaesthesia.

The Registrar General of England and Wales has recorded 'Deaths associated with Anaesthesia' since 1868.[176] If we accept that deaths actually due to anaesthesia form a proportion, though not necessarily a constant proportion, of these, it is of some interest to see how the number of these deaths associated with anaesthesia has changed with the years in relation to the volume and type of surgery performed. This may be considered a guide, albeit a very rough one, to progress and safety in anaesthesia. Deaths associated with anaesthesia in England show a steady rise from the earliest days of anaesthesia up until 1938 (Figure 8) in which year a peak figure of 916 cases was recorded. During the same time there was a continued numerical increase in the number and magnitude of operations performed. It has not proved possible to enumerate the total surgical population with which these deaths were associated.[112] Goodman Levy, using a sample of London Teaching Hospitals between the years 1911 and 1923, noted that the volume of surgery had increased by 13% whereas, in the same period, deaths associated with anaesthesia recorded in England and Wales had increased by 62% and concluded that, at this time, "little or nothing had been effected in the way of reducing the general liability to death under anaesthesia".[176] Sykes, following the number of deaths associated with anaesthesia recorded by the Registrar General from the first records in 1868 up until 1946, used the numbers of operations performed at the General Infirmary,

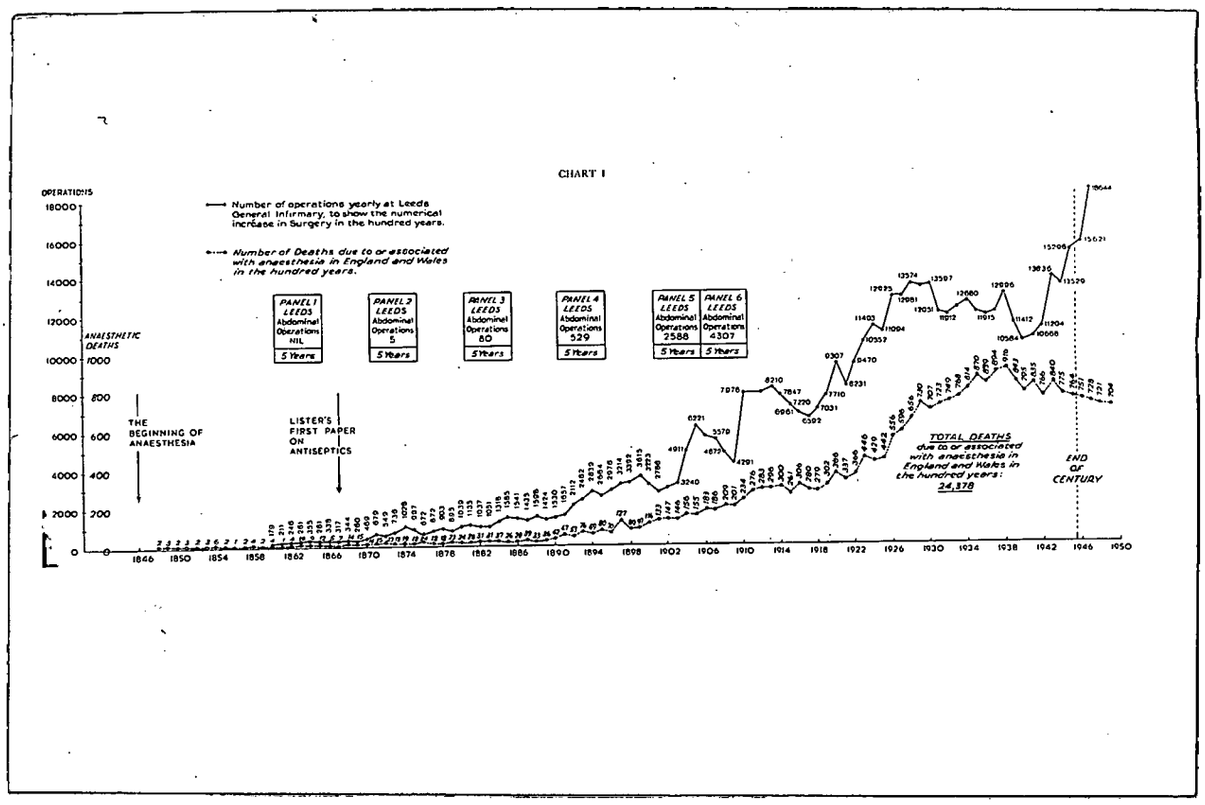


FIGURE 8. Deaths under or in association with Anaesthesia in England and Wales, 1846 - 1946. (Reproduced from 'Essays on the First Hundred Years of Anaesthesia' - Vol. II - by W. Stanley Sykes, by kind permission of E. & S. Livingstone Ltd., Edinburgh.)

Leeds, as an index of the increase in the number of surgical operations in general (Figure 8). This shows that up until 1938 the increase in numbers of deaths associated with anaesthesia was an accompaniment of an increase in the background surgical population. For the period examined by Goodman Levy, his assertion that the increase in deaths associated with surgery was disproportionate to the increase in surgery is supported by Sykes' figures from Leeds for the same period. If the deaths recorded by the Registrar General for the years 1910, 1911 and 1912 and 1922, 1923 and 1924 - the extremes of the period examined by Levy - are respectively averaged and the numbers of operations for the same period, in Leeds, likewise, the deaths associated with anaesthesia can be shown to have increased by 56% while the numbers of operations performed increased by 30% only in the same period. From 1938, the gross number of deaths associated with anaesthesia in England and Wales has fallen steadily (Figure 9) reaching a figure of 562 in 1953.[83] This decrease has occurred, in spite of the fact that during the same period the numbers of operations performed has increased at an even greater rate than before. Dawkins[48] estimates that the number of anaesthetics given in London teaching hospitals doubled in the 10 years preceding 1956. (This indicates a probable similar increase throughout England). That this decrease in anaesthetic associated mortality was concurrent with the period of the introduction of curare and relaxant techniques into clinical anaesthesia is of interest when juxtaposed with the findings of Beecher and Todd.[18] Together with this decrease in the crude death rate associated with anaesthesia, the Registrar General noted too, from confidential enquiries into maternal deaths in England and Wales during the years 1952 to 1957,[84, 85] a steady decrease in mortality from the complications of anaesthesia.

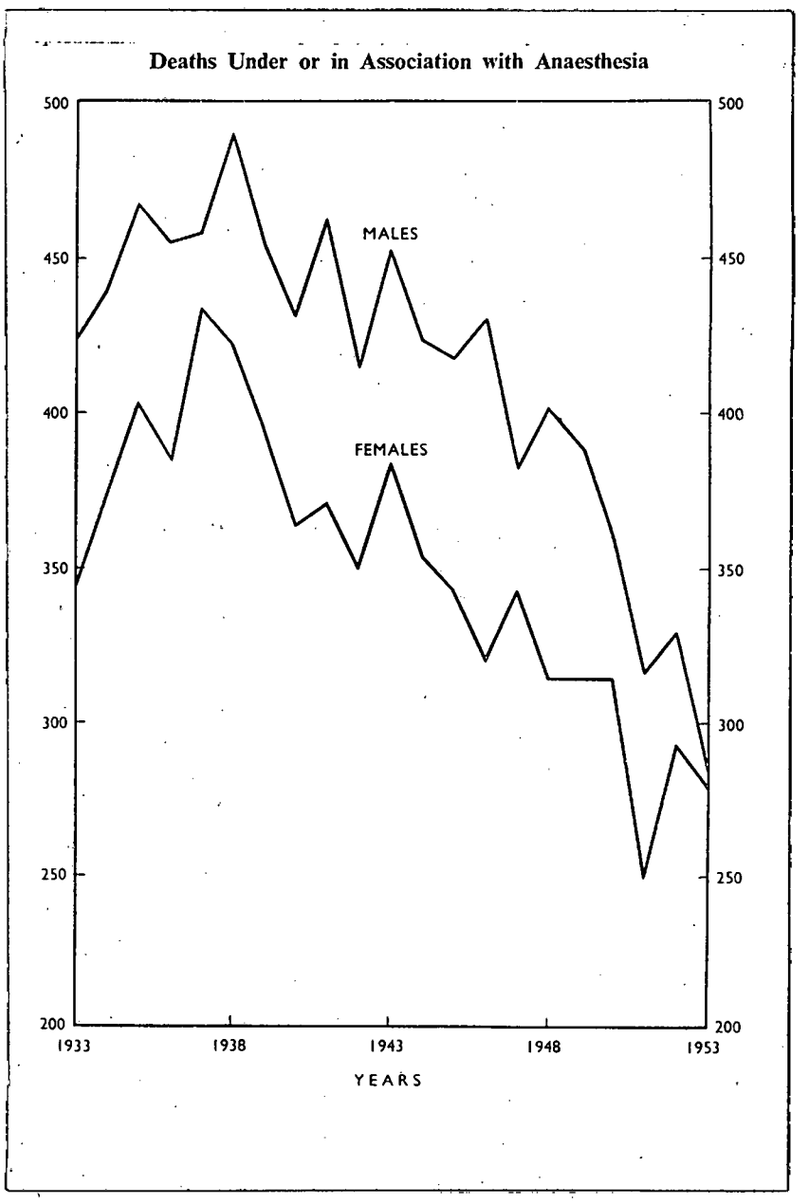


FIGURE 9. Deaths under or in association with Anaesthesia in England and Wales, 1933 - 1953. (Reproduced from the Report of the Ministry of Health for 1954 by kind permission of the Controller of Her Majesty's Stationery Office.)

Of particular interest in terms of this present Thesis is the situation as it exists here in South Africa. Examination of the surveys already quoted from this country does, in fact, show progress. Details abstracted from these surveys summarized in Table 21 is illustrated in the accompanying Histogram, Figure 10.

TABLE 21.

INCIDENCE OF ANAESTHETIC ASSOCIATED AND ANAESTHETIC CONTRIBUTORY DEATHS IN SOUTH AFRICA.

AUTHOR	Years included in Survey	Incidence per 1,000 Anaesthetics.	
		Anaesthetic Associated Deaths	Anaesthetic Contributory Deaths
Orenstein Committee	1931 - 1935	1.57	0.82
Melzer	1941 - 1945	2.85 Eur. 1.48 Non.Eur. 6.7	
Barlow et al	1945 - 1951	6.23	2.29
Johannesburg Hospital	1951 - 1952	1.87 Eur. 1.11 Non.Eur. 2.70	0.94
Kok and Mullan	1956 - 1962	0.94	0.49
Groote Schuur Hospital	1956 - 1960	2.49	0.43
	and	2.30	0.33
	1963 - 1965	2.08	0.21

It will be seen that there is no real difference between the incidence of anaesthetic contributory deaths reflected by the findings of the Orenstein Committee of 1936[169] (0.87 per 1,000) and that computed from the survey of the Johannesburg Teacing Hospitals Group[105] conducted 15 years later (0.94 per 1,000 -

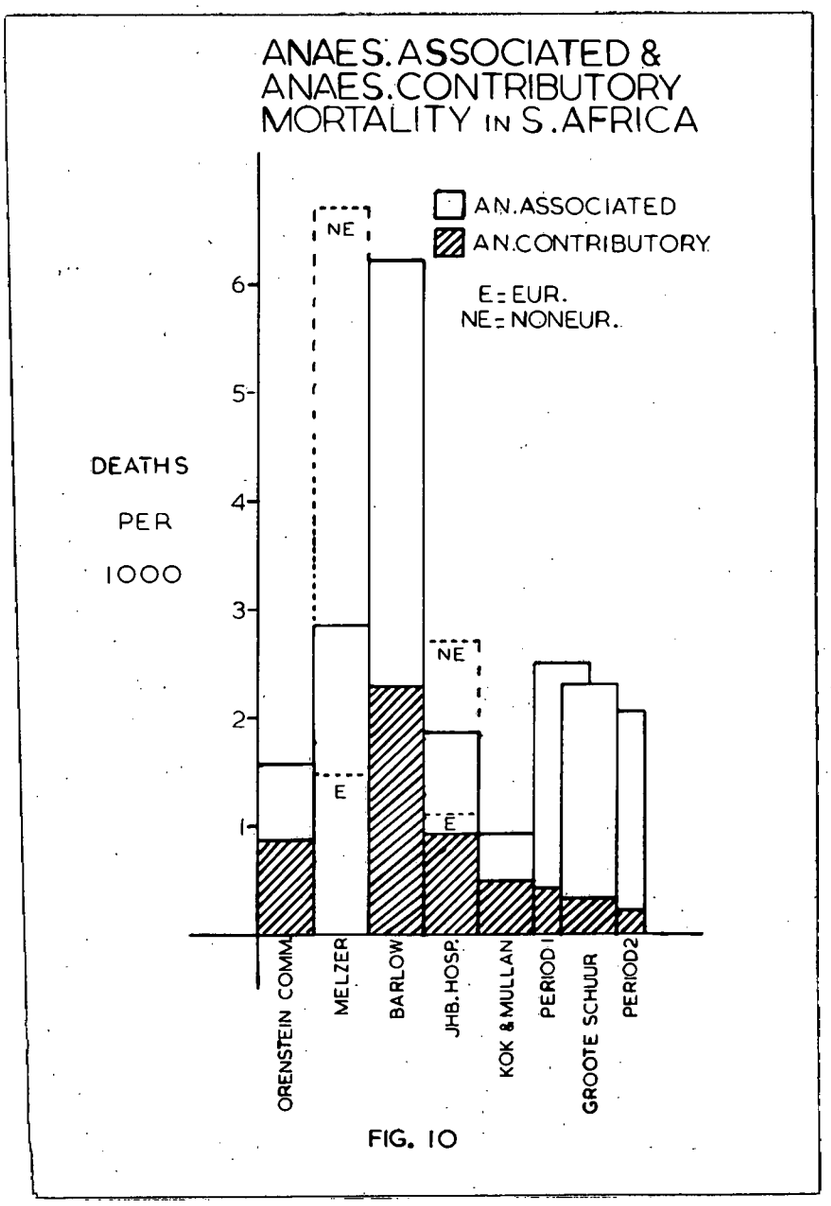

ANAES. ASSOCIATED &
ANAES. CONTRIBUTORY
MORTALITY IN S. AFRICA
AN. ASSOCIATED
AN. CONTRIBUTORY
E = EUR.
NE = NONEUR.
DEATHS
PER
1000
1
2
3
4
5
6
NE
E
NE
E
ORENSTEIN COMM.
MELZER
BARLOW
JHB. HOSP.
KOK & MULLAN
PERIOD 1
GROOTE SCHUUR
PERIOD 2

FIG. 10

my assessment). At the same time, the incidence of deaths associated with anaesthesia is shown to have increased slightly. A survey, conducted during the war years in the time between these two previous surveys, by Melzer[126] shows a marked increase in the number of deaths associated with anaesthesia, that in the Non-European being scandalously high (6.7 per 1,000). This finding was confirmed by the survey of the years 1945 - 51, conducted by Barlow et al at Coronation Non-European Hospital[16] where anaesthetic contributory deaths taken over the whole period of the survey had an incidence well over twice as high as that computed for South Africa by the Orenstein Committee - a fact that must be taken to indicate a distinct inadequacy in the anaesthetic services available to this section of the population at that time. However, in fairness, it must be said that the Orenstein Report does not separate figures for the incidence of death in the different race groups. During the period that was surveyed by Melzer, 1941 - 45; deaths associated with anaesthesia in the European (1.48 per 1,000) were much the same as the overall incidence of deaths associated with anaesthesia found by the Orenstein Committee. In that the incidence of death associated with anaesthesia computed by the Orenstein Committee (1.57 per 1,000) included all races, Melzer's figures probably indicate an increase, for the patient, in the hazards of surgery and anaesthesia at this time.

However, Melzer's survey covered the war years when there was a great drain on the country's man power, medical man power included. The subsequent survey in the Johannesburg Teaching Hospitals Group, 1951 - 52, showed a decrease in the deaths associated with anaesthesia from that found by Melzer - both overall, 1.87 per 1,000 as against 2.85 per 1,000, and in the incidence for individual broad racial groups, European 1.11 per

1,000 and Non-European 2.7 per 1,000 as against European 1.48 per 1,000 and Non-European 6.7 per 1,000 (this decrease in the anaesthesia associated mortality rate in the Non-European is particularly striking). However, the incidence of both death associated with anaesthesia and death due to anaesthesia at this time shows no improvement over that computed for South Africa in 1936. But when examined on an annual basis (Figure 11), the incidence of operating room deaths in the Johannesburg Central Hospitals Group from 1942 - 1952 does show a definite downward trend from 2.38 per 1,000 in 1942 to 0.80 per 1,000 in 1952. Likewise, when the incidence of death, both associated with and that due to anaesthesia, revealed by Barlow's survey at the Coronation Non-European Hospital[16] is examined on a yearly basis (Figure 12) a very marked decrease in incidence is seen. During the early part of the period covered by the survey, grave difficulties were experienced in providing adequately trained anaesthetic staff in sufficient numbers. This position was ameliorated to some extent over the period of the survey. This reduction of anaesthetic mortality pari passu with the increased availability of adequately trained staff, so well shown here, is commented on by many investigators including Bortone,[27] Edwards,[60] Jarman,[104] Waters and Gillespie,[183] Mannix,[123] Kok,[165] to quote but a few.

This indication of an improvement in the safety of anaesthesia and operation for the patient here in South Africa is further borne out by the results of both the survey of Kok and Mullan[168] - 1956 - 62 - and this survey at Groote Schuur Hospital which covers much the same period. An analysis by Kok[167] of the incidence of death associated with anaesthesia at 150 hospitals in South Africa during this time shows a progressive decrease (Fig. 13). The incidence of anaesthetic contributory death computed from Kok and Mullan's study[168] and that from the

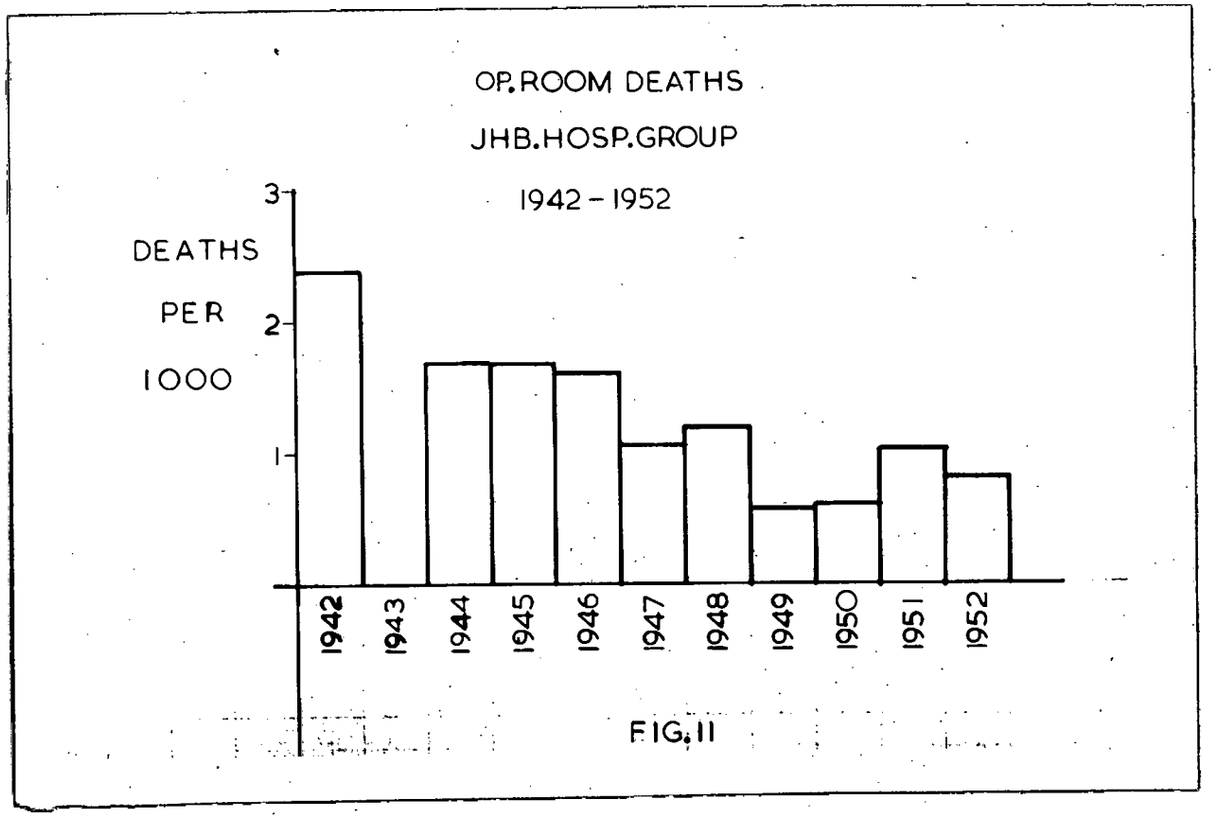

OP. ROOM DEATHS
JHB. HOSP. GROUP
1942 – 1952
DEATHS
PER
1000
3
2
1
1942
1943
1944
1945
1946
1947
1948
1949
1950
1951
1952

FIG. 11

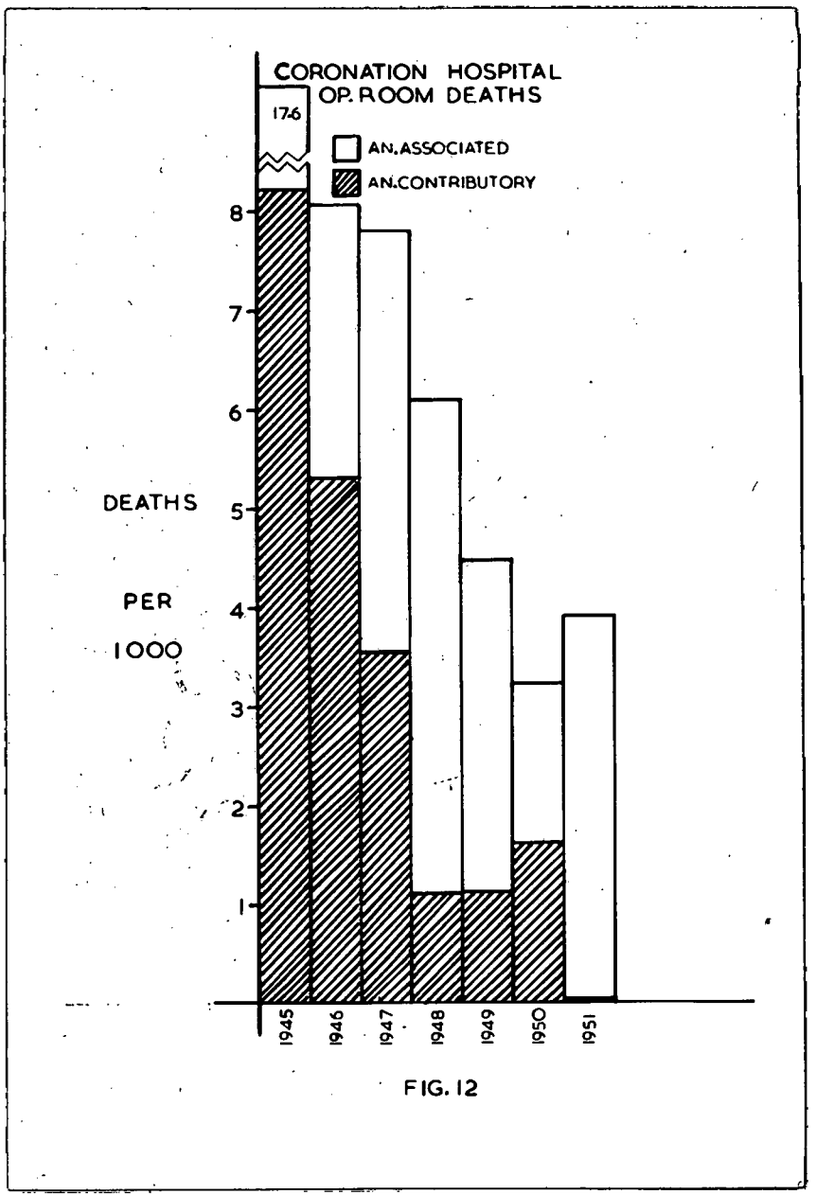

CORONATION HOSPITAL
OP. ROOM DEATHS
176
AN. ASSOCIATED
AN. CONTRIBUTORY
DEATHS
PER
1000
8
7
6
5
4
3
2
1
1945
1946
1947
1948
1949
1950
1951

FIG. 12

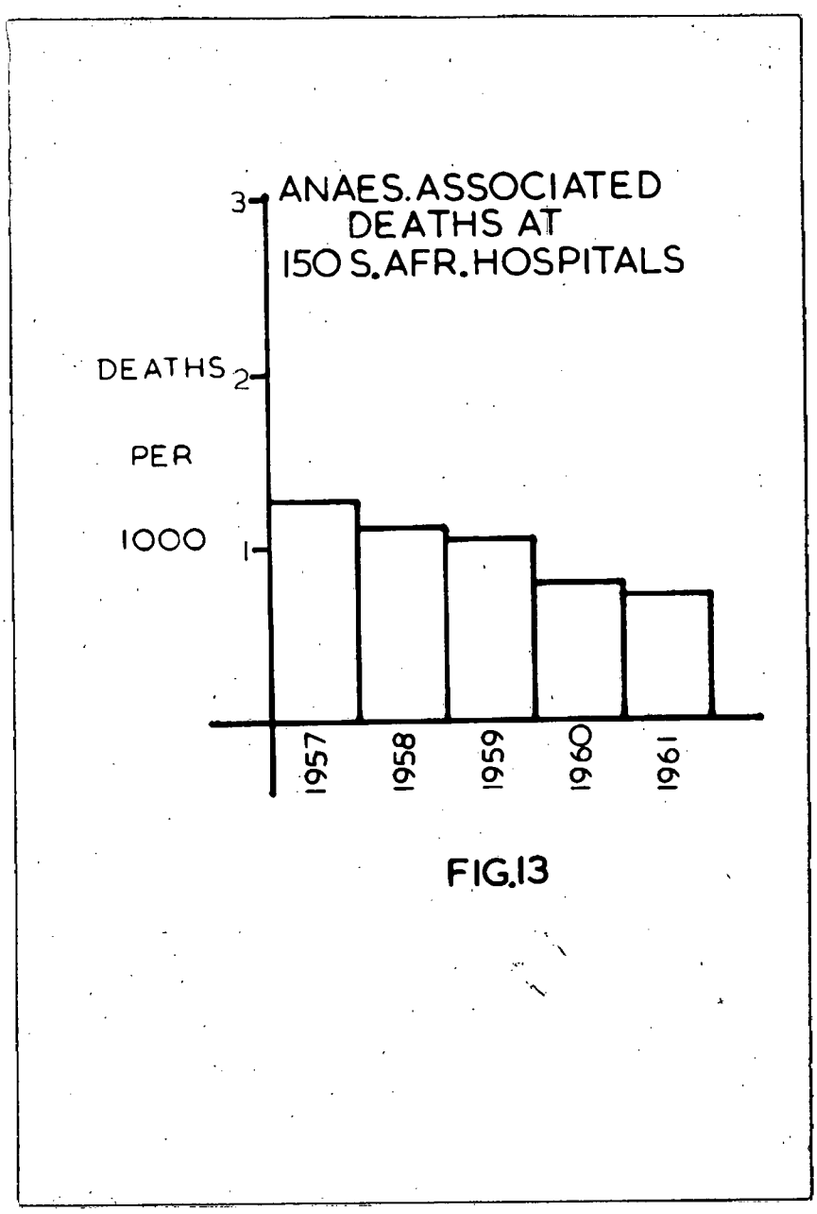

ANAES. ASSOCIATED
DEATHS AT
150 S. AFR. HOSPITALS
DEATHS
PER
1000
3
2
1
1957
1958
1959
1960
1961

FIG. 13

first period of this Groote Schuur Hospital survey are approximately a half only of that found by the Orenstein Committee[169] in 1936, whilst the incidence of anaesthetic contributory death in the second period of this survey is less than a quarter of the 1936 figure.

It has been noted that the basis of selection of cases used in this survey differed from other South African studies. These have all used the Section 86 of the Births, Marriages and Deaths Act of 1928 as their basis of selection. This implies some selection, on the basis of opinion of the anaesthetist, surgeon and perhaps Government pathologist, of those patients who die having survived the operation itself and regained consciousness, as to whether anaesthesia may or may not have been contributory before the case is submitted for examination and consequently inclusion in the survey. The use of an arbitrary post-operative time period as a basis of inclusion of patients in the Groote Schuur Hospital survey results in a greater number of cases being included in the survey and consequently a higher incidence of death associated with anaesthesia being reflected, viz. 2.30 per 1,000 as against 0.94 per 1,000 in Kok's[168] survey. Examined on an annual basis (Table 22, Figure 14) the incidence of death associated with anaesthesia at Groote Schuur Hospital shows little overall trend. The incidence of anaesthetic contributory death, however, shows a progressive diminution, the figure for the last year reviewed being one third approximately of that of the first year of this study.

The period since the Second World War, and more particularly since 1950, has seen a very definite advance in anaesthesia as a clinical speciality, in the length of period of training required of those adopting this discipline, in the academic and clinical standards demanded of such candidates before they

TABLE 22.

INCIDENCE OF ANAESTHETIC ASSOCIATED AND ANAESTHETIC CONTRIBUTORY DEATH AT GROOTE SCHUUR HOSPITAL - COMPUTED ON AN ANNUAL BASIS.

Year	Number of Anaesthetics	Number of Anaes. assoc. deaths.	Number of Anaes. contr. deaths.	Incidence per 1000 Anaes. assoc. deaths.	Incidence per 1000 Anaes. contr. deaths.
1956	16004	36	9	2.18	0.56
1957	16080	32	7	1.99	0.43
1958	15997	42	9	2.62	0.56
1959	17077	48	6	2.81	0.35
1960	17802	49	5	2.75	0.28
1963	22085	37	4	1.67	0.18
1964	24231	48	7	1.98	0.28
1965	24466	62	4	2.53	0.16
TOTAL	153742	354	51	2.30	0.33

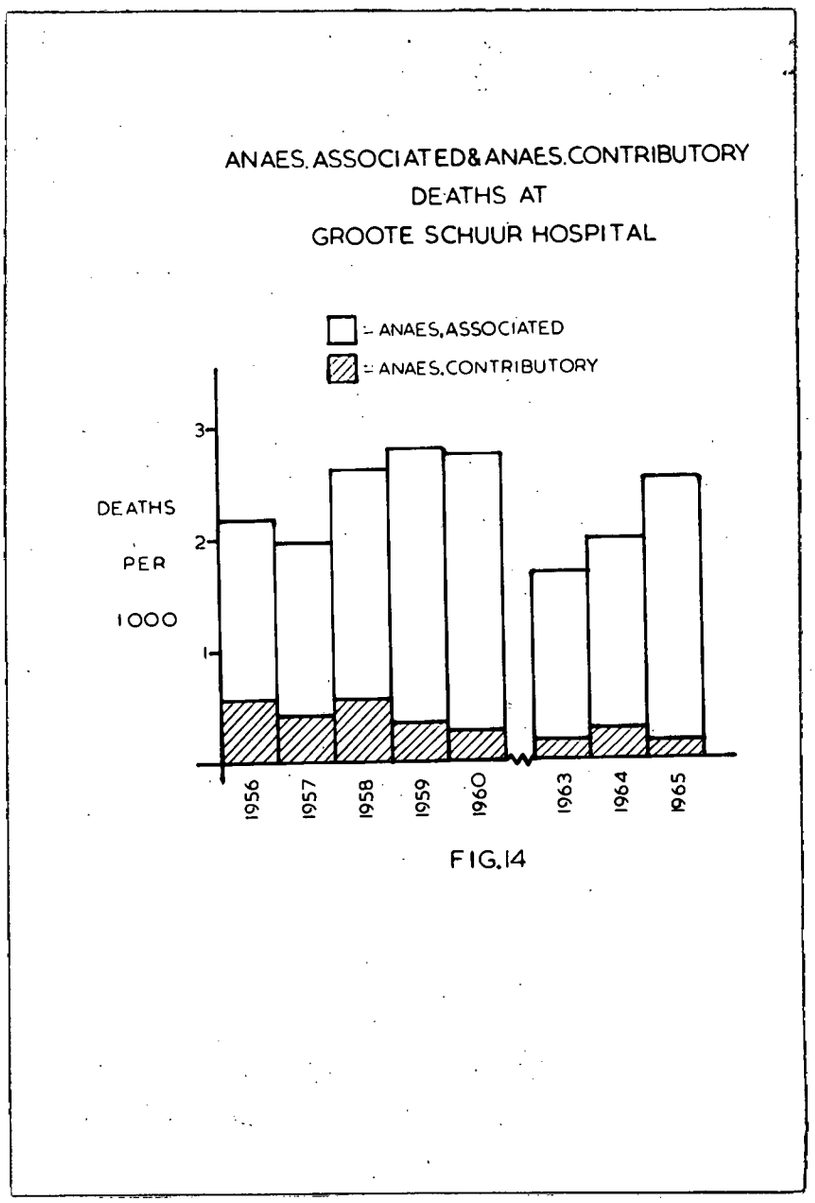

ANAES. ASSOCIATED & ANAES. CONTRIBUTORY
DEATHS AT
GROOTE SCHUUR HOSPITAL
= ANAES, ASSOCIATED
= ANAES. CONTRIBUTORY
DEATHS
PER
1000
3
2
1
1956
1957
1958
1959
1960
1963
1964
1965

FIG. 14

obtain diplomas of competency in anaesthesia and, in the numbers and quality of candidates presenting themselves for training. This world-wide trend is clearly mirrored in South Africa. Figure 15 shows the growth in number of specialist anaesthetists registered by the S.A. Medical and Dental Council since 1945 until the present.[171] A similar growth has occurred in the staff of the Anaesthetic Department at Groote Schuur Hospital during the period of this survey (Figure 16), a growth which culminated in the creation of a Chair of Anaesthetics at the University of Cape Town in 1965. It is important to note the increased proportion of trained specialist staff in relation to trainee anaesthetists. Evidenced by the decrease in incidence of death to which anaesthesia is considered contributory, these changes appear to have resulted in the increased safety of anaesthesia and surgical operation in general in spite of the growing surgical challenge.

Such advance towards the goal "that there should be no anaesthetic deaths" (Macintosh)[119] is gratifying. But the fact that anaesthetic contributory deaths do still occur and of these the majority are preventable, must goad us from any self-satisfied complacency. The figures quoted here reflect, on the whole, but a segment of the total population at risk throughout the country. It must be our goal that the patent benefits of advances in clinical anaesthesia in safety for the patient are further spread by the continued supply of more and better trained clinical anaesthetists.

SPECIALIST ANAESTHETISTS REGISTERED BY THE
S.A.MEDICAL & DENTAL COUNCIL

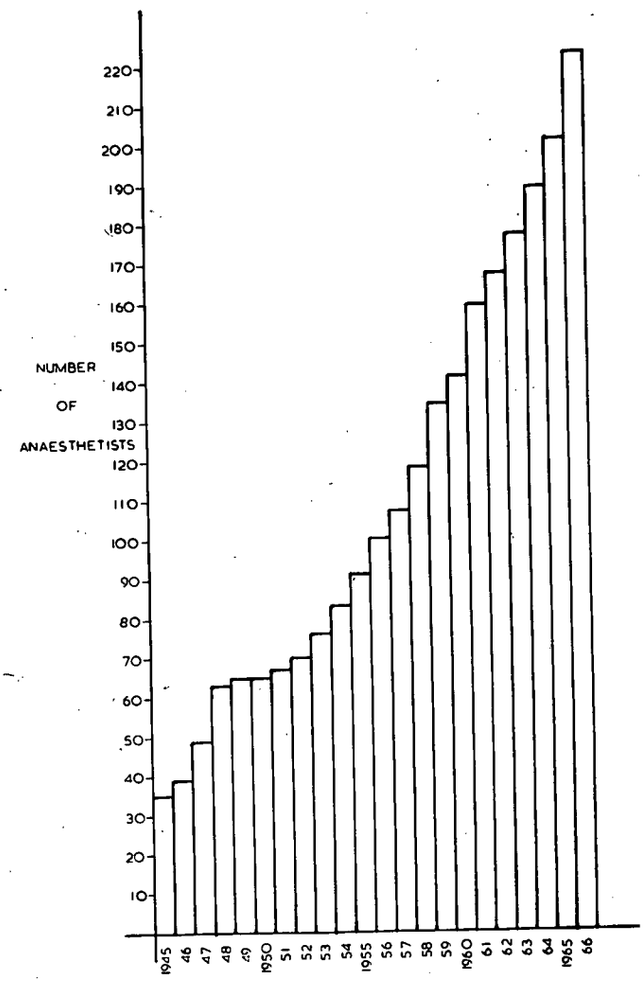


FIG. 15

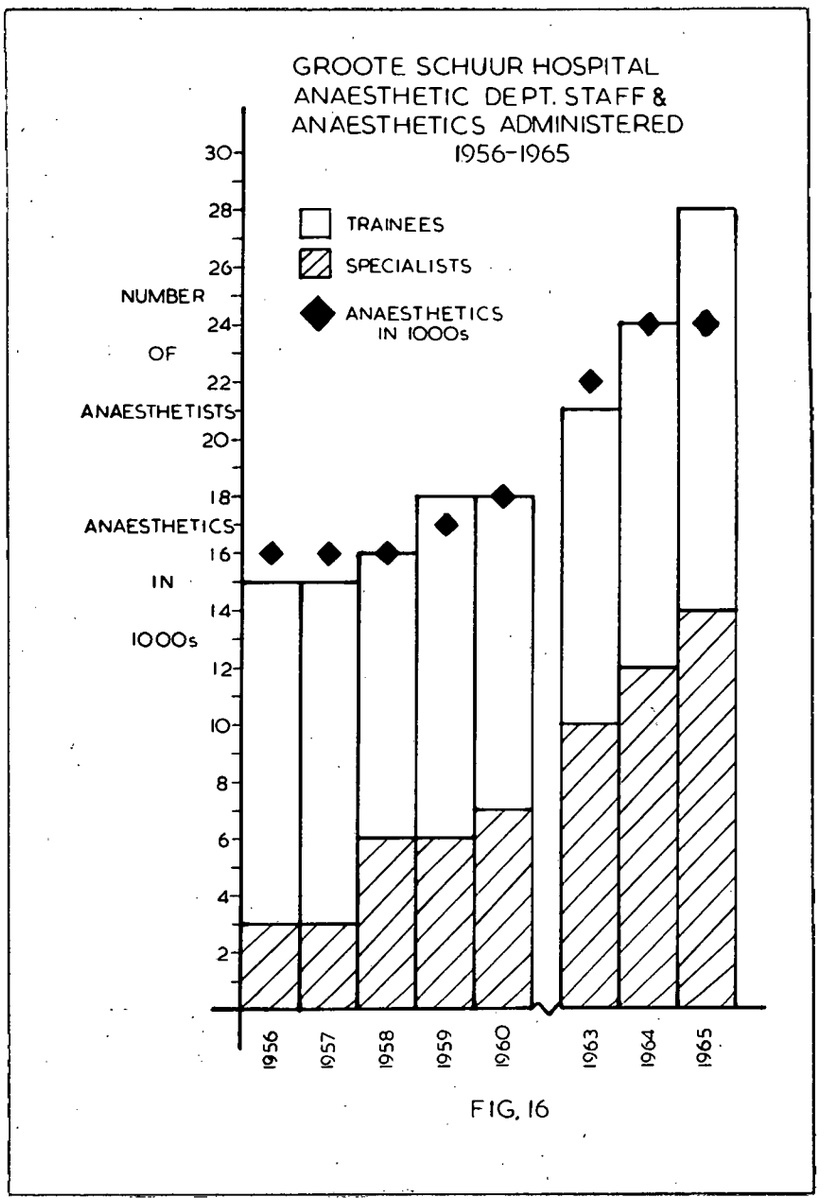

GROOTE SCHUUR HOSPITAL
ANAESTHETIC DEPT. STAFF &
ANAESTHETICS ADMINISTERED
1956-1965
TRAINEES
SPECIALISTS
ANAESTHETICS IN 1000s
NUMBER OF ANAESTHETISTS
ANAESTHETICS IN 1000s
30
28
26
24
22
20
18
16
14
12
10
8
6
4
2
1956
1957
1958
1959
1960
1963
1964
1965

FIG. 16

CHAPTER 8.

ANAESTHETIC CONTRIBUTORY DEATHS.
- SOME ASSOCIATED FACTORS -

An examination of anaesthetic contributory deaths in general, reveals, apart from the precise causes of such deaths, certain associated factors which appear to influence the incidence of such deaths. Associated factors that will be examined here include :-

1. The time in relation to the administration of the anaesthetic at which anaesthetic contributory deaths occur.
2. The physical status of the patient.
3. Status of the anaesthetist.
4. The sex of the patient.
5. The race of the patient.
6. The site of the operation.
7. The use of certain anaesthetic agents.

It will be seen that, in general, in contra-distinction to the disparity of the findings with regard to the more precise incidence of anaesthetic contributory deaths, most surveys of death associated with anaesthesia reveal a degree of agreement in their assessment of these associated factors.

1. Time of Death.

The inclusion of the immediate 24 hour post-operative period in the peri-operative period to be surveyed for anaesthetic associated mortality, together with the inclusion of those patients who failed to regain consciousness after anaesthesia has been discussed in an earlier chapter. An examination of the

time at which anaesthetic contributory deaths occurred in relation to the administration of the anaesthetic in the patients in this survey strongly supports the inclusion of this post-operative period. (see Table 23).

TABLE 23.

TIME OF DEATH IN RELATION TO ANAESTHETIC

Groote Schuur Hospital

	Time	Number of Deaths	Percentage of Anaesthetic Contributory Deaths.	
OPERATING ROOM DEATHS	During Induction	6	11.8	35.3
	During Operation	9	17.6	
	End of Operation	3	5.9	
POST OPERATIVE DEATHS	0 - 4 hrs.	10	19.6	64.6
	4 - 8 hrs.	8	15.7	
	8 - 24 hrs.	6	11.7	
	more than 24 hrs.	9	17.6	
	TOTAL	51	99.9	

While approximately one third of the anaesthetic contributory deaths occurred during anaesthesia and operation, two thirds occurred post-operatively. The proportion of anaesthetic contributory deaths that occurred during anaesthesia is somewhat lower than that quoted by those few authors who pay attention to this aspect of the problem. The time relationship of anaesthetic contributory deaths quoted by these authors is summarised in Table 24.

TABLE 24.

TIME OF ANAESTHETIC CONTRIBUTORY DEATH IN RELATION TO OPERATION.

AUTHOR Ref. No.	DURING OPERATION	POST OPERATIVE
Phillips et al 140.	36%	50% within operation plus 7 hours.
Edwards et al 81	57.4% Induction 25%	77% within operation plus 30 minutes.
Kok 168	56.5% Induction 12%	76.5% within operation plus 30 minutes.
Brown 35	59.4% Induction 16%	88.7% within operation plus 2 hours. 94% within operation plus 24 hours.
Beecher and Todd 18	40.5% Induction 10.5%	
Clifton and Hotten 42	Majority - Induction plus 30 mins.	94% within operation plus 4 hours. 98.7% fail to regain consciousness.

Of the patients who died post-operatively, a little over one half (18 out of 33 cases) failed to regain consciousness - 9 of these surviving beyond the 24 hour period. Of those 15 anaesthetic contributory deaths that occurred post-operatively after the patient had regained consciousness, all but one had occurred within 12 hours of the conclusion of the anaesthetic.

In view of the finding from this study that two thirds of the anaesthetic contributory deaths occurred post-operatively it is pertinent to recapitulate briefly here that anaesthesia was considered contributory in a significant fashion to only 24% of the actual operating room deaths and was responsible for only 29% of fatal operating room cardiac arrests.

The fact that the greater number of anaesthetic contributory deaths occur in the post-operative period probably is accounted for by improvements in methods of resuscitation applied once a catastrophe has occurred.

The induction has long been regarded as the most hazardous phase of any anaesthetic administration. Clifton and Hotten[42] state that where the anaesthetic is the principal etiological factor in a death, the death usually occurs during induction of anaesthesia or within 30 minutes. Though in this survey only 6 patients died during the actual induction of the anaesthetic, it is instructive to realise that the difficulties that subsequently led to death in a further 14 patients commenced during this phase of the anaesthetic, i.e. 39.2% of the anaesthetic contributory deaths resulted from difficulties commencing during the induction of anaesthesia.

Of the 9 patients who failed to regain consciousness after anaesthesia, yet survived beyond 24 hours post-operatively, all but one had suffered irreparable cerebral damage from grave anoxia during anaesthesia associated, in 5, with cardiac arrest. The well-known period of survival after such cerebral anoxia is well illustrated by the fact that 6 of these 9 cases died between the 4th and 6th day post-operatively.

2. Physical Status of the Patient.

Clinical anaesthesia has been described as a condition of 'physiological trespass'.[70] Where disease has already trespassed, the less trespass on the body's homeostatic mechanisms, by the anaesthetist, will be tolerated before breakdown occurs. The more disease has trespassed on the body's reserves, the greater the skill required by the anaesthetist to conduct the patient safely through the operation. In assessing the contributory role of anaesthesia in surgical mortality, it is relevant therefore to

test this in relation to the initial physical status of the patient - the better the physical condition of the patient the less acceptable is any mortality due to anaesthesia alone. In interpreting analyses of anaesthetic associated mortality in relation to the patient's physical status, one must be aware of the difficulties of assessment and the ease with which bias may occur. The more disease has deranged the body's homeostatic mechanisms, the more difficult it becomes to assess, except for gross errors, the contributory role of anaesthesia in any particular death. Where the physical condition of the patient is poor before anaesthesia is administered, this in itself often provides sufficient reason for death without too close an examination of the anaesthetic technique itself.

Probably the most widely used system of assessing the risk of anaesthesia per se in terms of the patient's physical status before anaesthesia is that devised by the American Society of Anesthesiologists.[152] The patient is classified in terms of his pre-operative physical condition as follows :-

1. An individual with no organic disease or localised disease which causes no systemic disturbance.
2. An individual with moderate systemic disturbance, e.g. mild diabetes mellitis.
3. An individual with severe systemic disturbance, e.g. poorly controlled diabetes, intestinal obstruction with electrolyte disturbance.
4. An individual suffering from systemic disease which is an imminent threat to life.

The above categories apply to patients presenting for elective surgery. For patients presenting for emergency surgery, those of the type that would be included in physical status 1 and 2 are classed as physical status 5 while those of physical status 3 and 4 are classed as physical status 6. Moribund patients are

identified as physical status 7. This classification of physical status takes no account of the magnitude of the operation or of the age of the patient as such. It appears also to allow of a degree of lattitude in interpretation.

It is doubtful though that any elaboration of this classification such as that devised by Ament[3] produces any greater clarity or value in assessment of the problem of anaesthetic deaths.

In assessing the physical status of the patient, I have aimed at greater simplicity of classification. On the basis of the physical condition, the patients are divided into two groups :-

1. Good - fair physical status. This is equivalent to groups 1 and 2 in the classification devised by the American Society of Anesthesiologists.
2. Poor - bad physical status. This is equivalent to groups 3, 4 and 7 in the American Society of Anesthesiologists classification.

Patients presenting for emergency surgery were classified into the same two categories, depending on their background physical state and the adequacy of pre-operative resuscitation, correction of fluid and electrolyte balance and restoration of blood volume. An analysis of the anaesthetic associated mortality in this survey in relation to the physical status of the patient is presented in Table 25.

TABLE 25

(see over)

TABLE 25

PHYSICAL STATUS OF PATIENTS.

PHYSICAL STATUS	Proportion of Patients Period 1 & Period 2			Incidence of Anaesthetic contributory Death in relation to Physical Status of Patient. 1st Period.		
	in Group 1. Anaes. Contri. Deaths	in Group 2. Deaths due to other Causes	in Group 3. Inevitable Deaths	Number of Patients.	Number of Anaes. Contri. Deaths	Incidence of Anaes. Contri. Death per 1,000
Good - Fair	49% (25)	18% (40)	25% (20)	79724 (96%)	18	0.22
Poor - Bad	51% (26)	82% (182)	75% (61)	3236 (4%)	18	5.55
	Numbers in brackets represent numbers of patients.					

This shows that one half of the patients who suffered anaesthetic contributory deaths were classed before anaesthetic as good-fair anaesthetic risks. The finding of the rather high proportion of patients in good physical status in anaesthetic contributory deaths is softened a little by calculation of the actual incidence of anaesthetic contributory deaths in relation to physical status. Estimated from data obtained in the first period of this survey, this shows the mortality rate of anaesthetic contributory deaths to be reasonably low, 0.22 per 1,000 anaesthetics in patients of good-fair physical status; while at the same time it is understandably high, 5.55 per 1,000 anaesthetics, in patients of poor-bad physical status.

The vast majority of patients presenting for anaesthesia

and surgery (96%) are of good-fair physical status. These patients are the most salvageable in the surgical terms and have, in general, the longest expectation of life. The figures presented here highlight the necessity for a reduction in anaesthetic contributory mortality in this group of patients especially. Though the incidence of anaesthetic contributory death cannot be directly calculated in relation to physical status for the 1963 - 65 period of this survey we may estimate it by extrapolation of the physical status distribution from the first period. Estimated on this basis the incidence of anaesthetic contributory death in patients of good-fair physical status for this period is 0.10 deaths per 1,000 anaesthetics. Experience of other authors is summarised in Table 26.

3. Status of the Anaesthetist.

The influence of the status or the seniority of the anaesthetist might have on the incidence of anaesthetic contributory death cannot be directly evaluated from this survey. Assignment of anaesthetists was varied in terms of the magnitude of the surgery involved and in terms of any anticipated difficulty - the more demanding cases being assigned to the more experienced anaesthetists. Further, the policy of the Anaesthetic Department was, and is, to provide supervision of trainee anaesthetists, especially the more junior, by experienced anaesthetists where possible and where necessary - though the administration of the anaesthetic will, in most instances, have been accredited in the operation registers and data cards to the trainee. This renders any enumeration of the distribution of anaesthetics administered on the basis of status of the anaesthetist inaccurate. Examination of the records of the anaesthetic contributory deaths in this survey (Table 27) shows that the administration of the anaesthetics was attributed to

TABLE 26.

DISTRIBUTION OF PHYSICAL STATUS OF PATIENTS IN ANAESTHETIC CONTRIBUTORY DEATHS.

AUTHOR	Physical Status				
	Good 1	Fair 2	Poor 3	Bad 4	
Proportion of Anaesthetic Contributory Deaths and Incidence per 1000					
Dripps et al. 55	0	15%	33.7%	41.2%	Moribund 10
Incidence per 1000	0	0.99	6.63	45.8	91.8
Briggs, Sheldon & Beecher. 31.					
Incidence per 1000	0.3		9.8		
Kaye 13.					
Incidence per 1000	0.3	0	2.0	34.6	
Proportions only of Anaesthetic Contributory Deaths.					
Clifton & Hotten. 42.	4.3%	8.7%	23.4%	63.6%	
Kok & Mullan. 168.	10.2%	28.8%	55.7%	5.3%	
New South Wales 134.	27.1%	14.5%	54.8%	3.6%	
Phillips et al. 140	36.7%		63.3%		
Brown. 35.	1.5%	18.8%	35.3%	40.2%	
Ruth et al. 150	31 %	21 %	41 %		

registrar or trainee anaesthetists on 38 occasions while in the remaining 13, consultant anaesthetists were responsible.

TABLE 27.

STATUS OF ANAESTHETIST IN RELATION TO ANAESTHETIC CONTRIBUTORY DEATHS.

PREVENTABILITY	Trainee Anaesthetist.	Specialist Anaesthetist.
Probably Preventable	13	5
Possibly Preventable	22	7
No Verdict	3	1
TOTAL	38	13

In most instances involving trainees, experienced help had been summoned once catastrophe had occurred. This finding may support the contention made in the previous chapter that the death rate from anaesthesia is reduced in relation to the experience and training of the anaesthetist. However, the lack of accurate knowledge of the frequency distribution of anaesthetics administered in relation to the experience of anaesthetists in this survey does not permit of any statistical proof of this.

4. The Sex of the Patient.

"Mere maleness influences unfavourably the resistance of the organism to disease". (Allen)[2].

The lesser viability of the male of the species in general[93] is very consistently demonstrated in all studies of deaths associated with anaesthesia. In one of the earliest authoritative monographs on anaesthesia 'On chloroform and other anaesthetics' published in 1858, John Snow[164] published the records of 50 deaths associated with anaesthesia. Of these, 29 were male.

In another early anaesthetic text, Hewitt[96] published collected figures of 210 deaths associated with anaesthesia. Of these 71% were in males. In neither of these instances was the sex distribution of the background surgical population enumerated. However, the sex distribution of the background surgical population at this time was published in the report of the Anaesthetics Committee of the British Medical Association published in 1900.[32] In the data collected by this Committee, the sex distribution of the background surgical population was 44% male and 56% female. While of the 29 deaths to which anaesthesia was considered contributory in this British Medical Association report, 60% were male. (This difference in sex distribution in anaesthetic contributory deaths can be shown to be statistically significant by a Chi square technique).

This preponderance of males in those dying in association with anaesthesia, as against the preponderance of females in the background surgical population continues unchanged. The sex distribution of anaesthetic associated mortality found in some recent surveys is presented in Table 28. In the case of two of these surveys in which the sex distribution of the background surgical population is included, those of Beecher and Todd[18] and Clifton and Hotten,[42] the statistical significance of this difference in sex distribution is demonstrable by the Chi square technique. This same preponderance of males in anaesthetic associated mortality is apparent in this present survey. In order to establish the sex distribution of the background surgical population, a representative sample was used. This was made up of the number of patients anaesthetised in 1964 classified as to sex, together with data on the sex of 15,561 randomly selected patients (roughly equal to the annual number of anaesthetics administered during the first period) anaesthetised during the years 1957, '58, '59 and '60. (The sex

TABLE 28.

SEX DISTRIBUTION OF ANAESTHETIC ASSOCIATED AND CONTRIBUTORY DEATHS.

Author	Anaesthetic assoc./contrib. Mortality.		Background Surgical Population.		
	Male.	Female.	Male.	Female.	
Boba 26.	52%	48%	-	-	of cardiac arrests.
Clifton & Hotten 42.	57.4%	42.6%	30.5%	69.5%	of anaesthetic assoc. deaths. $P < 0.001$
C.S.I.R. Kok 165.	60%	40%	-	-	of anaesthetic associated deaths.
Kok and Mullan 168.	54%	46%	-	-	
Dripps et al 55.	more deaths in males			more operations in females	
Phillips et al 140	54%	40%	-	-	
Campbell 40.	52%	48%	-	-	
Natof and Sadove 133.	70%	30%	-	-	of operating room deaths.
	73%	27%	-	-	of anaesthetic contrib.deaths.
Edwards et al. 81.	56.8%	43.2%	-	-	of anaesthetic assoc. deaths.
Bergner. 22	64%	36%	-	-	of cardiac arrests.
Beecher and Todd (18)	58%	42%	43%	57%	$P < 0.001$
Ministry of Health. Registrar General. 180.	more than female	less than male			of anaesthetic associated deaths.
Stephenson 172	61%	39%	-	-	of cardiac arrests.
Gordh 71.	more than female	less than male	-	-	of anaesthetic associated deaths.

CONTINUED OVERLEAF.

TABLE 28.(CONTD.)

Author	Anaesthetic assoc./contrib. Mortality.		Background Surgical Population.		
	Male.	Female.	Male.	Female.	
Brown 35.	62.4%	37.6%	less than in females	more than in males	of anaesthetic associated deaths.
Veal 182	60%	40%			of anaesthetic associated deaths.

of patient was not recorded on the data cards in 1956). The sex distribution of this sample together with that of the anaesthetic associated deaths and anaesthetic contributory deaths is presented in Table 29.

TABLE 29.

SEX DISTRIBUTION OF ANAESTHETIC ASSOCIATED AND ANAESTHETIC CONTRIBUTORY MORTALITY.

	MALE	FEMALE	
Surgical Population (sample)	41% (16719)	59% (23377)	
Anaesthetic Associated Deaths	60% (211)	40% (143)	ϕ
Anaesthetic Contributory Deaths	63% (32)	37% (19)	ϕϕ

ϕ P - less than 0.001

ϕϕ P - less than 0.005

Numbers in brackets are actual numbers of patients.

The preponderance of males both in the anaesthetic associated mortality and in anaesthetic contributory mortality is shown to be statistically highly significant by the Chi square technique.

Why there is an increased vulnerability of the male is difficult to say. Perhaps it is the high incidence of serious heart and circulatory disease in the male that renders him more vulnerable to the stress of anaesthesia. Perhaps this apparent increased vulnerability of the male is to some extent spurious in that, though the greater proportion of the background surgical population is female, many of the operations performed on females are minor gynaecological operations and, from the anaesthetic point of view, of a trivial nature, for example, cervical dilatation

and uterine curettage and uterine evacuation of retained products of conception. Examination of the operations performed during the period 1963 - 65 reveals that whereas 36% of operations performed on males were classed as minor, no less than 52% of operations performed on females were so classed and 28% of all operations on females were minor gynaecological operations. During this period the sex distribution of patients presenting for major operations was approximately equal. Yet of the anaesthetic deaths occurring in association with major surgery in the survey as a whole 64% were male. This difference in the sex distribution of anaesthetic contributory deaths in relation to major surgery just fails to achieve statistical significance at a 5% level ($X^2 = 3.24$ on 1 d.f. $0.05 < P < 0.10$.) In general, the reasons for this apparent increased liability to death in association with anaesthesia on the part of the male are too complex to be elucidated from a simple study such as this, covering in all so small a number of deaths. Though not strong, there is some evidence from the sex distribution of anaesthetic contributory deaths in this survey that "a price is paid for a beard and the presence of a functional testis". (Hamilton).[93]

5. <u>The Age of the Patient</u>.

Examination of the age distribution of the deaths associated with anaesthesia in this survey shows a definite vulnerability of certain age groups. This applies to those deaths to which anaesthesia was considered contributory and, to a lesser extent, to those deaths which were considered due to factors other than anaesthesia.

To obtain an estimate of the age distribution of the background surgical population with which to match the age distribution of those patients who died in association with anaesthesia, a

sample of just over one quarter of the total number of patients anaesthetised during the first period of this survey was used. Data on the age of 22,307 randomly selected patients was abstracted from the small anaesthetic data cards. The age distribution of these patients together with that of those patients who died in association with anaesthesia during the first period is presented in Table 30. This comparison is possible for the first period of this survey (1956 - 60) only. As a result of the abandonment of the use of the anaesthetic data cards during the second period (1963 - 65), though the age distribution of the anaesthetic associated mortality is available, that of the surgical population is not readily so. The differences in the age distribution of anaesthetic associated and contributory mortality and the surgical population is best seen in the accompanying histogram (Figure 17). In this the number of patients in each decade of age is expressed as a percentage of the total sample from which it comes. The differences in the age distribution of those patients who died in association with anaesthesia and the background surgical population are statistically highly significant. ($P < 0.001$ on a X^2 technique).

These differences appear to show a greater liability to death from causes related to anaesthesia in patients at the extremes of life. Patients in the first decade and again from the sixth decade and older, represent twice as great a proportion of the patients dying in association with anaesthesia as they do in the background surgical population. This apparent vulnerability of these age groups is shown in many surveys of anaesthetic associated mortality. Regrettably, most fail to match the numbers of deaths in age groups against the age distribution of the background surgical population. Comparisons are further invalidated by the differences in the age blocks in which various

TABLE 30.

AGE DISTRIBUTION - PERCENTAGES OF RESPECTIVE SAMPLES.

AGE. YEARS.	SURGICAL POPULATION.	ANAESTHETIC CONTRIBUTORY DEATHS.	ANAESTHETIC ASSOCIATED DEATHS.
0 - 9	10.1	19.4	12.9
10 - 19	12.3	0	8.8
20 - 29	22.4	0	10.5
30 - 39	18.3	11.1	12.9
40 - 49	13.6	19.4	13.4
50 - 59	10.9	16.6	14.0
60 - 69	7.1	19.4	16.4
70 - 79	3.8	8.3	6.4
80 plus	1.2	5.6	3.5
unknown			1.2

$P < 0.001$

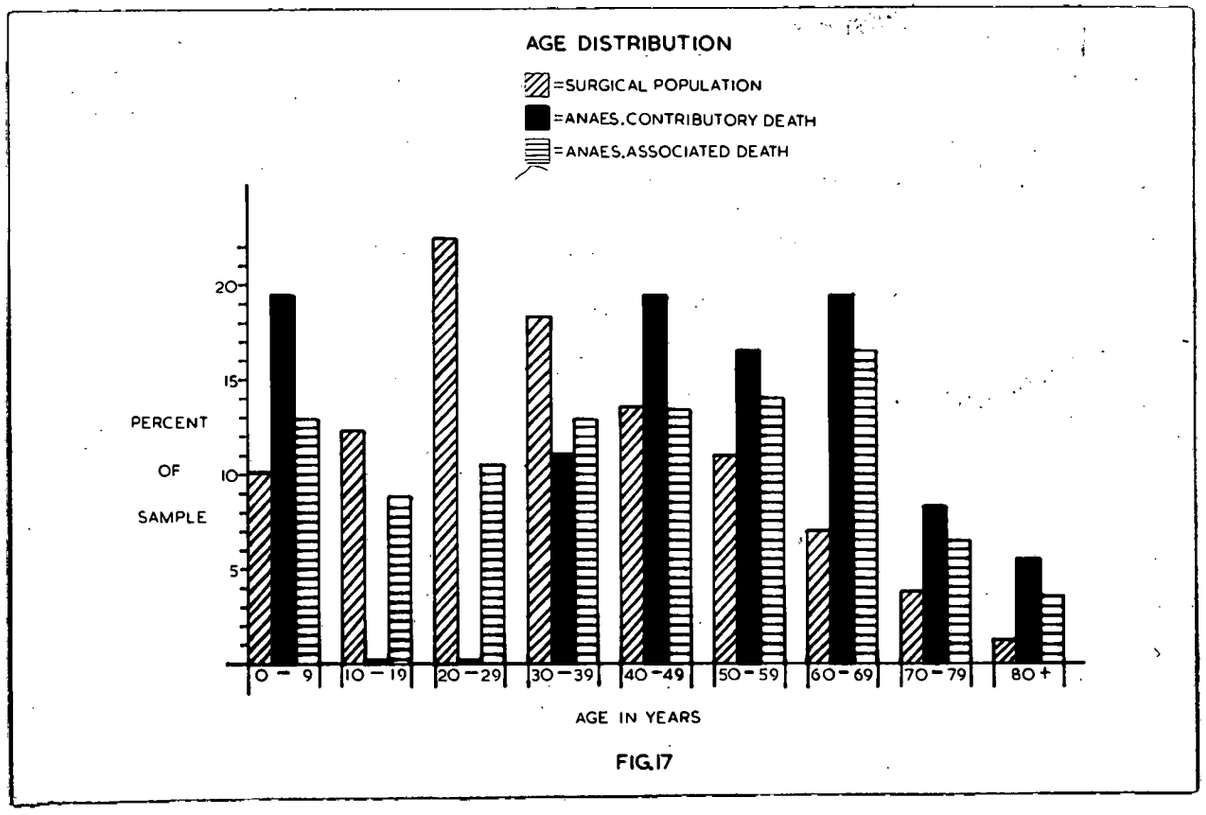

AGE DISTRIBUTION
=SURGICAL POPULATION
=ANAES.CONTRIBUTORY DEATH
=ANAES.ASSOCIATED DEATH
PERCENT
OF
SAMPLE
20
15
10
5
0 - 9
10 - 19
20 - 29
30 - 39
40 - 49
50 - 59
60 - 69
70 - 79
80 +
AGE IN YEARS

FIG.17

authors group the age distributions.

The increased death rate in the first decade of life is commented on by Stephenson,[172] West,[186] Gartner,[67] Hingson,[98] Schull[156] and the Committee on Anaesthetic Deaths of New South Wales.[134] In the survey of Beecher and Todd,[18] as in this survey, patients in the first decade comprise twice the proportion of the anaesthetic contributory deaths that they do in the background surgical population.

The more understandable increased liability to death in association with anaesthesia in the older age group of patients is commented on by the Committee on Deaths of the Association of Anaesthetists of Great Britain and Ireland,[78] Briggs, Sheldon and Beecher,[31] Schapira,[154] Schroff[157] and Brown.[35] The Baltimore Anaesthesia Study Committee,[140] in their estimate of the incidence of anaesthetic contributory death, showed that the incidence of deaths in the age group 65 - 74 was twice that in the age group 45 - 64, whilst that in patients over 75 was three times that in the 45 - 64 year age group.

The reasons for this increased liability to death in association with anaesthesia in patients in the older age groups are, to some extent, understandable. In all probability, it is a reflection of the increased incidence of concomitant degenerative vascular disease and pulmonary disease in this group of patients. The previously discussed factors of sex and physical status of the patient are interrelated with the effects of the age factor.

The reasons for the high incidence of anaesthetic contributory and associated mortality in patients in the first decade of life are less easy to see. In this survey the majority of deaths in this age group appeared to be associated with the element of anoxic anoxia. The higher metabolic rate and oxygen demand of children and the difficulties posed by the 'smallness' of their

anatomy may be factors. This, however, is surmise. No firm conclusions can be drawn from a study of this size and nature.

Robert Smith[163] of Boston, examining this question, poses three questions :

1. Is the higher mortality due to more heroic surgery in children ?
2. Are normal children poorer risks than adults ?
3. Do deaths represent inadequacy or errors on the part of the anaesthetist and surgeon ?

The answer he gives to the first two questions is 'No'. To the last he answers 'Yes'.

6. <u>The Race of the Patient</u>.

In a thesis presented to the Witwatersrand University in 1947, Melzer[126] produced evidence of an incidence of death associated with anaesthesia in the Non-European (Non-White) patient that was more than four times greater than that in the European (White). A report covering the teaching hospital of the same University for the years 1951 - 52[105] again showed a high incidence of death associated with anaesthesia in the Non-European patient, though the difference was much less marked. Though the greater proportion of the deaths reported in a subsequent wide survey of deaths associated with anaesthesia in South Africa - that of Kok and Mullan[168] - was Non-European, no estimate of the relative racial incidence was possible, as the racial distribution of the background surgical population was not stated.

From America, another country with a mixed population predominantly Caucasoid and Negroid, some authors[182, 95, 180, 51, 141,] have reported an increased incidence in the Negro as compared with the White of deaths associated with anaesthesia, though Beecher and Todd,[18] in their wide ranging survey of deaths

associated with anaesthesia at ten American University Teaching Hospitals, could find no difference in the racial distribution of deaths to which anaesthesia was considered contributory.

The results that emerge from an analysis of the racial distribution of deaths in association with anaesthesia in this survey are similar only to those of Beecher and Todd.[18] The racial distribution of the surgical population together with that of the anaesthetic associated and anaesthetic contributory deaths is presented in Table 31.

TABLE 31.

RACIAL DISTRIBUTION.

RACE	Surgical Population.	Anaesthetic Associated Deaths.	Anaesthetic Contributory Deaths.
White	41%	45%	53%
Non-White	59%	55%	47%

There is no significant difference in the racial distribution of the surgical population and that of the anaesthetic associated and contributory deaths in this study.

7. The Site of Operation.

Though there is truth in the adage that "there are no minor anaesthetics - it is only the surgical procedure that can on occasion be considered minor", it is apparent that the demands made on the skill of the anaesthetist by certain surgical procedures and by the type of patient involved are greater than by others.

An analysis of the site of operation in anaesthetic contributory deaths in this survey, showsthat no fewer than 39 of the 51 deaths involved had abdominal operations. (One of the 4 patients who died before operation commenced was also scheduled

for an abdominal operation). A list of operations with which the anaesthetic contributory deaths were associated is appended (Table 32). Of the background surgical population, approximately 18% presented for abdominal surgery. It would appear that in general, abdominal operations and the associated condition of the patient presenting for abdominal surgery make the greatest demand on the skill of the anaesthetist. The converse, that it is in the conditions associated with abdominal surgery that deficiencies on the part of the anaesthetist most frequently contribute to the death of the patient, is probably also true. In that group of patients whose death in association with surgery and anaesthesia was considered 'inevitable' or to whose death anaesthesia was considered 'necessarily contributory', the majority of operations were cardiac or major vascular surgery. In this latter group of patients, though it is the patient's disease and the surgical procedure involved that appeared to prove the greatest risk to life, it is obvious that the degree of skill and care demanded of the anaesthetist must be high. However, the former risk is of such an order and the circumstances involved are such that assessment of deficiencies in the latter are rendered more difficult. This finding that the conditions associated with abdominal surgery pose a severe test of the anaesthetist's skill has been reported by investigators such as Ruth,[150] Brown,[35] Kok and Kitay,[166] Kok and Mullan,[168] Dripps and co-workers[55] and the Anaesthesia Study Committees of Baltimore and New South Wales.[134] (This latter Study Committee singles out especially operations for intestinal obstruction).[140]

In surgery of the ear, nose and throat and of the eye, the surgical procedure should not of itself constitute any danger to the patient, except in the case of certain operations involving the respiratory pathway. In these circumstances, any deficiency on the part of the anaesthetist in terms of care and

TABLE 32.

SITE OF OPERATION.

Site of Operation	Operation	
ABDOMINAL	Gastrectomy	4
29	Exploration of Common Bile Duct	1
	Cholecystenterostomy	2
	Lacerated Liver	1
	Acute Abdomen (Pancreatitis 3 (Intestinal Obst. 3 (Appendix 1 (abscess	7
	Paraumbilical Herniorrhaphy	1
	Left Hemicolectomy	1
	Ant. Resection of Rectum	1
	Bilateral Adrenalectomy	1
	Aortico-Renal Endarterectomy	1
	Abdominal Hysterectomy	2
	Salpingectomy	1
	Caesarean Section	1
	Transvesical Prostatectomy	2
	Nephrectomy	1
	Dehisced Abdominal Wound	2
CARDIOTHORACIC	Cardiotomy-Pulmonary Stenosis	1
6	Pneumonectomy	1
	Bronchoscopy, Bronchogram	3
	Cardiac Catheterisation	1
PERINEAL	Circumcision	1
4	Cystoscopy	2
	Transurethral Prostatic Resection	1
ORTHOPAEDIC	Smith-Peterson Pin	1
2	Ulnar Nerve Suture	1

CONTINUED OVERLEAF.

TABLE 32. (CONTD.)

Site of Operation	Operation	
NEUROSURGICAL 5	Craniotomy	3
	Berry Aneurysm Ligation	2
EAR, NOSE AND THROAT SURGERY	Mastoidectomy	1
NO OPERATION PERFORMED 4	Operation Planned :-	
	Orchidectomy	1
	Amoebic Abscess of Liver	1
	Amputation of Leg	2

skill are all the more regrettable. Deaths from such deficiencies during these operations can more often than not be classed as preventable. Both Trent and Gaster[180] and Gartner[67] found a disappointingly large number of deaths during eye operations.

Mortality that has accompanied that common minor surgical procedure, tonsillectomy and adenoidectomy, is well documented by Ruth,[150] the Committee on Deaths of the Association of Anaesthetists of Great Britain and Ireland,[80] Wrigley,[187] Gain[65] and Dilworth.[53] It is encouraging to note that during the eight year period surveyed here, with one exception - a mastoidectomy -, there was neither anaesthetic contributory nor even anaesthetic associated mortality in surgery either of the ear, nose and throat or of the eye.

8. <u>The Use of Certain Agents</u>.

" It is the hand that gives the anaesthetic and the eye that watches its effects that matter more than the choice of agent, dosage and method." (Simpson)[160]

Though I doubt the value of classifying deaths resulting from anaesthesia in relation to the drugs used, the incidence of anaesthetic contributory death associated with two widely used groups of drugs does warrant some comment. These are :

1. The intravenous barbiturates.

2. The muscle relaxant drugs.

1. <u>Intravenous barbiturates</u>.

Introduced into clinical practice by Lundy in 1934[116] Thiopentone sodium is today used all but routinely as the agent for the induction of general anaesthesia. Many other ultra short-acting intravenous barbiturates have subsequently been introduced into anaesthetic practice but, by and large, none of

these have had sufficient clinical or pharmacological advantage over thiopentone to challenge it seriously, let alone displace it from clinical anaesthetic practice. However, other intravenous barbiturates are used and the heading of this section has been broadened to include them though thiopentone sodium is primarily the drug that is discussed.

In catastrophes following the administration of thiopentone or other intravenous barbiturates, though respiratory depression is a cause of death, many of the fatalities appear to result from the cardiac and vasomotor depression which follow injection of this drug. These reactions have been discussed earlier (Chapter 4.)

In an attempt to establish the incidence of death following the use of thiopentone sodium, Dundee[56] collected information on 192,881 administrations of thiopentone. From fatalities attributed to the use of thiopentone in these patients he estimated the incidence of death attributable to thiopentone as being 0.35 per 1,000 anaesthetics. But he has reservations about the accuracy with which the incidence of death attributable to any one anaesthetic agent can be established.

Both the major reports emanating from the Committee on Deaths Associated with Anaesthesia of the Association of Anaesthetists of Great Britain and Ireland stress the entity of circulatory failure following the use of an intravenous barbiturate for the induction of anaesthesia. Of the first 1,000 cases reviewed by Edwards et al,[81] in 107 of the 589 deaths to which anaesthesia was contributory, this mechanism was cited as the cause of death. The following 600 cases reported to the Committee were reviewed by Dinnick.[82] Of the 400 cases attributable to anaesthesia, 38 were considered to be due to the use of an intravenous barbiturate, usually associated with a muscle relaxant drug, and of these 26 displayed circulatory collapse.

The concomitant role of a hypovolaemic state in these patients is also stressed. Clifton and Hotten,[42] in their 10 year survey in deaths associated with anaesthesia at Prince Alfred Hospital, Sydney, Australia, attributed 17% of the anaesthetic contributory deaths to circulatory collapse after the administration of thiopentone.

In South Africa, reports from the C.S.I.R. Research Unit on anaesthetic deaths, stress the danger of the unskilful use of thiopentone.[165, 166] In this Unit's last report on deaths associated with anaesthesia, Kok and Mullan,[168] reviewing 1,000 deaths, attribute 19.5% of the anaesthetic contributory deaths to circulatory collapse following the use of thiopentone.

In this present study at Groote Schuur Hospital, 39 of the 51 anaesthetic contributory deaths had had anaesthesia induced with thiopentone sodium. Though its use may conceivably have been a contributory factor in many of these fatalities, in 7 cases the association of thiopentone with the subsequent death appeared closer. These constitute 14% of the anaesthetic contributory deaths. During the first period of the survey thiopentone was used for the induction of anaesthesia on 44,902 occasions while 5 of the above deaths occurred during this time. The incidence of fatality clearly due to the use of thiopentone is 0.11 per 1,000 anaesthetics involving its use. Considering the difficulties inherent in assessing the precise cause of an anaesthetic death where many drugs have been used, this figure should be regarded as the minimum incidence. The real incidence of anaesthetic death to which thiopentone is a major contributory factor is doubtless much higher.

Though we may not be able to express accurately the precise incidence of death to which the administration of thiopentone is contributory, it is important to realise that unskilful, injudicious and uncritical use of this universally used induction

agent is an important causative factor in many of the deaths to which anaesthesia is contributory.

2. The muscle relaxants.

Introduced into clinical anaesthetic practice in 1942 by Griffith and Johnson,[86] curare and other subsequently developed muscle relaxant drugs rapidly became a major weapon in the pharmacological armamentarium of the anaesthetist. The publication in 1954 by Beecher and Todd[18] of their startling finding that the incidence of death to which anaesthesia was contributory was six times greater when curare had been used as an adjuvant in the anaesthetic technique (2.7 per 1,000) than when it had been omitted (0.47 per 1,000) excited much discussion. The major conclusions that Beecher and Todd made from their study of deaths associated with anaesthesia were that (1) the use of curare as an adjuvant appeared to cause a gross elevation of the anaesthetic contributory death rate, (2) neither the condition of the patient, the experience of the anaesthetist nor the magnitude of the surgery had any effect on this incidence of death associated with anaesthesia. (3) Denying any increase in the incidence of clinical errors in the conduct of anaesthesia in those patients that received curare, they attributed the increased anaesthetic mortality rate to an 'inherent toxicity of curare'. This work was subjected to much criticism, mainly on the grounds that (a) the circulatory collapse that Beecher and Todd attributed to the use of curare, which they found occurred in spite of adequate artificial respiration, was contrary to accepted clinical and pharmacological opinion and (b) in their study, deaths assessed as due to 'respiratory failure' after the use of curare were not, but should have been, regarded as due to lack of skill.[8, 7, 1, 88, 10, 131.] The description by Hunter[101] in 1956, of an entity he called 'neostigmine resistant curarization' suggested a possible foundation for Beecher and

Todd's 'inherent toxicity'. However, no major survey of deaths associated with anaesthesia published since 1954 has supported these findings of Beecher and Todd.

Current opinion is possibly best expressed by Kok and Mullan[168] who, in their survey of deaths associated with anaesthesia state "We have gone carefully into our own deaths but can find no evidence to support the opinion that curare drugs have inherent toxicity which may result in cardiovascular failure . Most of our deaths can be explained and were due to overdosage or faulty technique". This survey, it should be noted, covered nearly twice the number of anaesthetics than was included in the controversial survey by Beecher and Todd.

One of the major difficulties that present in the evaluation of this problem is highlighted by Dripps and co-workers[55] when they state "We even questioned Beecher and Todd's contention that the use of muscle relaxants caused a mortality rate greater than if these adjuvants had been omitted". This is the difficulty of the lack of comparability in the test situation in which anaesthetic drugs are used.

Gordh[72] attempted to overcome this difficulty by examining at the Karolinska Sjukhuset, Sweden, (1) the gross surgical mortality, (2) the mortality after biliary surgery alone, both on an annual basis over a period commencing before the introduction of relaxant drugs in Sweden in 1944, to a time when their clinical use was well established - 1957. Finding that there was no change in the gross surgical mortality over the period 1946 - 57, while the use of the relaxant drugs had increased from no use to use in 3% of all anaesthetics, and that in the period 1944 - 57 the mortality after biliary surgery had been more than halved while, at the same time, the clinical use of relaxant drugs in anaesthesia for biliary surgery had increased from no use to use in 95% of anaesthetics, he concluded that

relaxant drugs do not increase the risk of anaesthesia. Dealing as he does with gross surgical mortality and not anaesthetic contributory mortality per se, Gordh relies, for the validity of his inference, on the assumption that a change in anaesthetic mortality would be reflected in the overall surgical mortality, an assumption which makes no allowance for improvements in surgical mortality that might have resulted from advances in surgical care.

Another difficulty that confronts us in the evaluation of this problem arises from the fact that rarely in modern clinical anaesthesia is any drug used alone. Relaxant drugs require in clinical use the addition of a drug, or drugs, to maintain anaesthesia itself, together with a technique that provides adequate pulmonary ventilation for the patient. Both of these factors impose changes of their own on the body's circulatory homeostasis - a circumstance which makes assessment of the responsibility for such changes difficult.

Further, though not obligatory, it is certainly safer when a relaxant drug is employed to utilise an endotracheal tube to ensure adequate control of the patient's airway. The use of an endotracheal tube has its own difficulties, pitfalls and dangers, some of which, if not recognised, can and do cause death. For example, during the first period of this present survey at Groote Schuur Hospital, two patients suffered fatal cerebral anoxia because the endotracheal tube, inserted to ensure patency of the patient's airway, became kinked, causing complete respiratory obstruction. In each case this catastrophe was not recognised timeously by the anaesthetist because of inexperience. It is probable that had the patient been breathing spontaneously, respiratory obstruction may have been recognised earlier and appropriate treatment applied. Here then a death has resulted from a technique which may be regarded as a necessary

corollary to the use of a relaxant drug and, in the face of the anaesthetist's inexperience, signs of the mishap could be said to have been masked by the action of the relaxant. Should such deaths be attributed to the use of a relaxant drug ? I think not. These deaths and deaths of a similar nature should be regarded as resulting from technical failure on the part of the anaesthetist, a failure that can be eliminated by training and experience.

The direct action of the relaxant drugs is muscle paralysis. Their use imposes on the anaesthetist the duty of providing the patient with adequate pulmonary ventilation for the duration of their action. Should death that results from inadequate pulmonary ventilation during anaesthesia be attributed to the use of the relaxant drug ? Again, I think not. Again, it is a failure of technique on the part of the anaesthetist that must bear the blame. However, deaths that follow the type of respiratory inadequacy or abnormality that may persist post-operatively following the use of a relaxant drug, especially in the circumstances of fluid and electrolyte depletion, and which respond poorly to existing forms of treatment, together with those due to drugs used as antidotes to the relaxants, are possibly the type of death that could be attributed to the use of relaxants. But even this argument is difficult to maintain. Many of these cases are the result of overdosage of relaxants in the clinical circumstances pertaining. Many will respond to the correction of abnormal acid base states.[34] Even those patients suffering from post-relaxant apnoea, which is not adequately reversed pharmacologically by means at the disposal of the anaesthetist, should not come to harm if adequate pulmonary ventilation is continuously provided until the action of the drug is terminated by its excretion. In these circumstances, one may again ask, should not this type of death

be also regarded as due to technical failure on the part of the anaesthetist, a failure correctable by training and experience ?

In their study, the figures from which Beecher and Todd made their inferences were derived by estimating the total number of deaths in which anaesthesia was contributory in patients who had received relaxant drugs as against those to which anaesthesia was contributory in the group of patients who had not.[98] Hingson and co-workers, making a similar calculation from their study of deaths associated with anaesthesia, found an incidence of death attributable to anaesthesia in patients in whom relaxant drugs had been used of 5.23 per 1,000 anaesthetics involving their use. This incidence is even higher than that estimated by Beecher and Todd. But, when they sought those deaths more directly attributable to the use of relaxant drugs, the incidence of such was 0.35 per 1,000 anaesthetics where those deaths to which it was a major contributory factor were considered and 0.52 per 1,000 when instances in which it was considered a minor contributory factor were included. Dripps and co-workers,[55] while finding no mortality at all in 6,000 healthy (Physical Status 1) patients who received muscle relaxants, found an overall relaxant contributory mortality of 0.82 deaths per 1,000 anaesthetics involving their use. When deaths were related to use of relaxant drugs, errors of omission or commission were always apparent.

It is pertinent to mention here again the results of two consecutive surveys of anaesthetic associated and anaesthetic contributory mortality, discussed in Chapter 6, which were undertaken at the same group of hospitals over a long period divided by the time of the introduction of curare into clinical anaesthetic practice. These surveys, undertaken at the University of Wisconsin group of hospitals by Waters and Gillespie[183] and Dornette and Orth,[54] show no real change in the incidence of

anaesthetic contributory death over this period.

From this present study, calculation of the incidence of relaxant contributory death may be made from the relevant data obtained during the 1956 - 60 period (see Table 33).

TABLE 33.

RELAXANTS AND ANAESTHETIC CONTRIBUTORY MORTALITY
1956 - 1960

	Anaesthetic Population	Anaesthetic Contrib. Deaths	Incidence Anaes. Contrib. Death per 1000	Relaxant Deaths	Incidence of Relaxant Death per 1000
Anaesthetics without Relaxant	68,358	16	0.23		
Anaesthetics with Relaxant	14,602	20	1.37	5	0.34
Anaesthetics with Relaxant and I.P.P.R.	10,935			5	0.45

If a calculation similar to that of Beecher and Todd is made, the incidence of anaesthetic contributory death in patients in whom relaxant drugs were used is 1.37 per 1,000 anaesthetics involving their use.

If we accept the 5 deaths that followed post-relaxant respiratory abnormality during the first period (see Chapter 4) as the only deaths more directly attributable to the use of relaxant drugs, the incidence of death to which the use of relaxant drugs was contributory is 0.34 per 1,000 anaesthetics involving their use. In patients who received no relaxant drug, the incidence of anaesthetic contributory death is 0.23 per 1,000 anaesthetics. In general, the scope and magnitude of operation and condition

of the patient anaesthetised were not comparable in these two groups of patients. If anything, the anaesthetic techniques used for the more prolonged and difficult operations involving, in general, patients of worse physical status, included the use of relaxant drugs. It appeared that the relaxant drugs were used in situations more testing of the skill of the anaesthetist. Bearing this in mind, it is interesting to note that there is overall a six-fold difference in the incidence of anaesthetic contributory deaths between that group of patients who received relaxants and those that did not - a difference qualitatively similar to that found by Beecher and Todd, but quantitatively at an actual level half of theirs. This difference (which is statistically highly significant) probably means no more than that the relaxant drugs are used in situations that make more demands on the anaesthetist's skill. When the deaths more directly attributable to the use of relaxant drugs alone are considered, though the incidence of death is higher (0.34 per 1,000) than that in cases that did not receive relaxant drugs, this difference does not achieve statistical significance.

In many instances a relaxant drug is used to facilitate intubation only and not as part of the anaesthetic technique for the operation as a whole. When a relaxant drug is used as a major part of an anaesthetic technique, an I.P.P.R. technique is invariably employed. In order to eliminate those cases in which the use of a relaxant drug was not a major part of the anaesthetic technique, only those cases where the use of a relaxant drug was associated with an I.P.P.R. technique were examined. In this instance, incidence of death more directly attributable to the use of relaxant drugs is 0.45 per 1,000 anaesthetics. Again, for the numbers in this survey, the difference between this and the incidence of anaesthetic contributory deaths in patients not receiving relaxant drugs cannot be shown to be statistically

significant.

During the second period of this survey 4 deaths only were more directly attributable to the use of relaxants (one of these being caused by the use of the necessary antidote - neostigmine). Though the incidence cannot be directly calculated for the number of uses of various drugs was not recorded during this period of the survey, as the number of anaesthetics given increased and indications for the use of relaxants have ever widened, the incidence of death more directly attributable to the relaxant drugs is probably lower than that estimated for the first period.

The use of relaxant drugs has become an essential part of the anaesthetist's pharmacological armamentarium. Their use, though deceptively easy, to be safe demands of the anaesthetist a thorough knowledge of their pharmacology, skilled judgment in their dosage and the mastery of certain technical procedures essentially associated with their use. Deaths to which use of a relaxant drug is attributable occur only when one of the above essential desiderata is missing.

Safety of newer Agents.

It is appropriate to consider here some of the criteria of comparison which should be adopted when the safety of newly introduced anaesthetic or adjuvant drugs is considered. It must be remembered that in general the overall incidence of death to which anaesthesia is contributory is of the order of 0.5 per 1,000 anaesthetics. Firm claims as to the safety or advantages a newly introduced agent may have over existing methods can be based only on the use of the agent in large numbers of cases. Disadvantages in the way of increased mortality may become obvious with smaller numbers only if at all gross.

If a new drug or technique can be shown to have a marked

advantage over existing methods of anaesthesia in the facilitation of certain surgical procedures, then advantages must be carefully weighed against any mortality attributable to the drug itself in relation to (a) the general mortality of the surgical procedure itself without their use and (b) the overall incidence of anaesthetic contributory mortality.

Lastly, in considering death associated with the use of any particular agent, one must be careful to distinguish those deaths which appear to result directly from some action of the drug from those that appear to result in some technical failure on the part of the anaesthetist. But here is the rub. Often, as experience with a new agent or technique increases so deaths that would initially have been attributed to the agent come to be recognised as due to technical failure or lack of skill on the part of the anaesthetist.

CHAPTER 9.

SUMMARY AND CONCLUSIONS.

A learned judge once said "It is a fact that to anaesthetise a human being, to deprive him of consciousness outright, is to take a considerable step along the road to killing him".[94] Yet in this country alone, in any one year, at least one in every thirty persons of the population requires to be anaesthetised for some surgical operation.[106] Whatever the technical advances of clinical anaesthesia the most important aspect from the patient's point of view is that it must be safe. It must not kill. Nowhere is the precept to do no harm more important than in anaesthesia.

The most fundamental index we have of the safety of anaesthesia is the incidence with which factors related to the anaesthetic cause or are contributory to a patient's death. To ascertain this incidence about which there is little relevant modern information a contemporary survey of the mortality associated with 153,742 anaesthetics administered at Groote Schuur Hospital over a period of eight years was undertaken and is here reported.

Information was collected on all patients -

1. who died during the administration of an anaesthetic.
2. who died within twenty four hours of receiving an anaesthetic.
3. who, having been conscious before an anaesthetic, died without regaining consciousness thereafter.

These were classed as anaesthetic associated deaths. Clinical information on these was sought concurently from the anaesthetist concerned together with the results of autopsy when obtainable.

This information was then assessed and classified in the broad terms of a simple classification -

1. Anaesthetic contributory deaths - deaths to which the anaesthetic and its management were considered to have contributed to a significant degree.
2. Deaths due to other causes.
3. Inevitable deaths or deaths to which the anaesthetic was considered to be necessarily and unavoidably contributory.

The anaesthetic contributory deaths were further classified as to cause, firstly in terms of the basic mechanism of death -

1. Anoxia - i. anoxic anoxia.
   ii. ischaemic anoxia.
2. Cardiac arrest - i. direct causes other than anoxia.
   ii. uncertain etiology.
3. Miscellaneous

Secondly, these anaesthetic contributory deaths were classified in terms of the more precise clinical fault or mishap which caused death, e.g. respiratory obstruction, due to kinking of endotracheal tube. Thirdly, aspects of the preventability of these anaesthetic contributory deaths were assessed as -

Probably preventable

Possibly preventable

No verdict

Clinical details of these anaesthetic associated deaths together with commentaries validating their classification and discussions of their possible preventability are included in volume 2.

In order to relate the anaesthetic contributory mortality to its parent surgical population, data was obtained on -

1. the total number of anaesthetics administered at Groote Schuur Hospital.
2. the age and sex of all patients anaesthetised.
3. the numbers of uses of various agents and techniques.
4. the physical status of all patients anaesthetised.

During the eight years there were 354 anaesthetic associated deaths, to 51 of which the anaesthetic and its management were considered to have been contributory. These were classified as to cause as follows :-

TABLE 34

CAUSE OF DEATH

Mechanism		Cause of Death	No. of Deaths	Percentage of Anaes. Contrib. Deaths
1. ANOXIA	ANOXIC ANOXIA 45%	Atmospheric Anox.	1	2
		Tidal Anoxia (Resp. Obstruction	12	23
		Tidal Anoxia (Relaxant Assoc. death	9	18
		Alveolar Anoxia — Pulmonary oedema	1	2
	ISCHAEMIC ANOXIA 28%	Derangement cardiac function	8	16
		Derangement cerebral function a) due to hypotension	4	8
		Derangement cerebral function b) due to hypertension	2	4
2. CARDIAC ARREST	28%	1. Direct Causes	4	8
		2. Uncertain etiology	7	14
		3. Ventricular tachycardia	2	6
3. MISCELLANEOUS		Incompatible blood transfusion	1	2

Not surprisingly deaths due to anoxic anoxia constitute the greatest proportion. It must be appreciated that though these deaths are classified as due to anoxic anoxia, hypercarbia must have been co-existent and is synergistic in the production of catastrophe. More than half of these deaths were due to respiratory

obstruction whilst a little under half were related to the use of relaxant drugs - these latter representing 17.6% of all anaesthetic contributory deaths. The fact that a substantial proportion of all deaths is related to the use of relaxant drugs is difficult to evaluate. This probably means little more than that relaxant drugs are used very often. Another mode of anaesthetic contributory death that emerges with some prominence is that which follows progressive, prolonged, intractable hypotension following the induction of anaesthesia. In some cases death followed the precipitation of cardiac arrest by this phenomenom, while in others, though cardiac arrest did not result at the time, death followed subsequently from irreversible cerebral damage. Hypertension resulting from factors in the anaesthetic technique is also noted as being responsible for anaesthetic contributory death. In two instances noted here the episodesof hypertension were related to the use of intravenous urea.

Of all these anaesthetic contributory deaths, 53% were associated with cardiac arrest during operation. On the other hand less than one third of all ultimately fatal operating room cardiac arrests were due to causes related to the anaesthetic. The factor responsible for the majority of these was the effects of gross haemorrhage and massive transfusion.

When anaesthetic contributory deaths are considered in relation to the background surgical population, the incidence that is reflected by this survey is 0.33 deaths per 1,000 anaesthetics. The survey falls into two periods, 1956 - 1960 and 1963 - 1965 inclusive. The incidence of anaesthetic contributory death for the former was 0.43 per 1,000 whilst in the latter it had dropped to 0.21 per 1,000.

Of these anaesthetic contributory deaths the vast majority, 92%, were considered either probably or possibly preventable.

Accepting the incidence of anaesthetic contributory death as a yardstick by which the standard of clinical anaesthetic practice can be judged, an attempt was made to compare the incidence of anaesthetic contributory death at Groote Schuur Hospital with the results of similar surveys of anaesthetic mortality conducted in other centres over the years. When these were examined closely, it became regrettably obvious that a great number of variable factors influenced the estimate of mortality rate that emerges from these surveys thus rendering any but the broadest comparison of incidence between surveys invalid. These factors are discussed and include -

1. Standards of assessment of data used.
2. Differences in classification of data.
3. Differences in the varying peri-operative time period included in these surveys.
4. Differences in the type and scope of surgery covered.
5. Limitation of the techniques of anaesthesia included.
6. Failure to measure the parent surgical population.

Examples are given of how these variables could influence the incidence of death computed. In particular, the differences that arise from varying the length of peri-operative time period which is surveyed for mortality are illustrated by appropriate selection of the cases from this survey. Three common variations of the period surveyed for anaesthetic associated mortality are illustrated. These are the inclusion of -

1. The period of operation, the immediate 24 hours thereafter, and the inclusion of those patients who die after failing to regain consciousness following anaesthesia, having been conscious before.
2. The period of operation only together with the inclusion

of those patients who fail to regain consciousness.

3. The period of operation only.

The first is the period included in this survey and yields an incidence of anaesthetic contributory mortality of 0.33 deaths per 1,000 anaesthetics. This is regarded as the best estimate of the incidence of anaesthetic contributory mortality. When cases are selected in terms of the second set of criteria the incidence reflected falls to 0.24 per 1,000 whilst that yielded by the third is approximately one third only (0.12 per 1,000) of the true incidence.

Though only the broadest comparisons are possible, the standard of anaesthetic practice reflected by this survey at Groote Schuur Hospital compares more than favourably with that reflected by 55 other surveys quoted. In these comparisons special reference is made to other surveys of anaesthetic mortality that have been conducted in South Africa.

Perhaps the most important question to which we must find an answer is "Have the advances which have been claimed for clinical anaesthesia resulted in greater safety for the patient ?". I have attempted to answer this question by examining the successive annual incidence of anaesthetic associated and anaesthetic contributory mortality from certain sample surveys. I have examined, in particular, the South African aspects of this problem. There appears to be definite evidence that anaesthetic contributory mortality is decreasing overall. It is pertinent to quote as an example figures from this survey in which the incidence of anaesthetic contributory death in the last year (1965), 0.16 per 1,000, was less than one quarter that of the first year (1956), 0.56 per 1,000. This increased safety of anaesthesia for the patient appears to be related to the increasing availability of adequately trained clinical anaesthetists and the better standards of training,

both clinical and academic, now available.

This survey portrays the situation as it exists in a single University teaching hospital - one well staffed with trained personnel. One cannot extrapolate these figures to the country as a whole. This is probably better represented in the work of the C.S.I.R. Anaesthetic Deaths Research Unit which covered in all 150 hospitals including teaching and non-teaching, large city and small town hospitals. This factor no doubt accounts for the higher incidence of anaesthetic contributory mortality shown by that survey. The major portion of the anaesthetics given for routine surgery in this country as a whole is administered by general practitioners,[106] persons who often have no special training in the discipline of anaesthesia. The patent benefits of advances in clinical anaesthesia in safety for the patient will only be spread by a continued supply of more and better trained anaesthetists. In outlying areas away from the larger centres which are now becoming 'specialist orientated' it becomes important to improve the training and skill of those practitioners who do administer anaesthetics. Possible practicable measures of accomplishing this are -

1. The provision for the short term attachment on a 'supernumerary staff' basis of interested practitioners to recognised teaching departments of anaesthetics. Such a scheme does exist at this Medical School but is not widely known.
2. The provision by teaching departments of what may be called 'visiting demonstrators' who could visit selected country hospitals for periods of up to a week giving practical demonstrations of accepted techniques of safe anaesthesia for the more routine surgery.

In this regard a sentiment expressed by Macintosh nearly twenty years ago still applies "I believe patients would be better off if

research on new drugs was halted for five years and attention directed to training young anaesthetists in the care of the unconscious patient and in the correct administration of time proved anaesthetics readily to hand in any hospital".[119]

Lastly I have examined the influence that certain factors associated with anaesthetics and operations have on the incidence of anaesthetic contributory death. Briefly I have concluded as follows -

1. Two thirds of those deaths to which anaesthesia is considered contributory occur after the conclusion of the anaesthetic and operation. Therefore surveys of anaesthetic mortality must include the immediate post-operative period as well as the period of operation.
2. The risk of death to which anaesthesia is contributory increases as the pre-anaesthetic physical status of the patient worsens.
3. Death to which anaesthesia is contributory occurs more frequently in the hands of the less experienced practitioner.
4. The liability to death both associated with and due to anaesthesia is greater in the male.
5. Anaesthetic contributory mortality is disproportionately high at the extremes of life - in the first decade and from the sixth decade onwards.
6. Anaesthetic contributory mortality is not influenced by the race of the patient.
7. The incidence of anaesthetic contributory death is higher when the anaesthetic is administered for an abdominal operation.
8. The use of two groups of drugs, the intravenous barbiturates and the relaxant drugs is examined in relation to anaesthetic contributory mortality. Both agents require skill, judgment and experience in their administration and

epitomise the adage 'there are no safe anaesthetics - only safe anaesthetists'. It cannot be demonstrated from my data that there is any significant difference in anaesthetic contributory mortality associated with the use of relaxant drugs but the need of skill in their use must be emphasised.

I believe that anaesthesia is ever providing a better and a safer service to the patient and to surgery. A pre-requisite to really establishing and monitoring such a claim is the continued systematic collection and assessment of factual data relating to clinical anaesthesia, its mortality and morbidity. A most important corollary to such a survey is the contribution made to the training and skill of anaesthetists by the dissemination of the clinical lessons that must emanate from such a study - for the value of history lies in the fact that by it we learn from the mistakes of others, learning from our own is a slow and, in terms of human life, an expensive process.

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VOLUME 2.

....BEING CLINICAL ACCOUNTS,
WITH COMMENTARY, DISCUSSION
AND CLASSIFICATION, OF ALL
CASES INCLUDED IN THIS STUDY.

Cases are identified by the block of 3 numbers in the top left hand corner of the script. These numbers represent in order :-

a) number of case in chronological order of entry into study.

b) The period 1 - 1956 - 1960
2 - 1963 - 1965

c) The year

The number in the adjacent space represents the group into which the case was classified. Classification is according to the criteria described in Chapter 3.

The 'Time of Death' in relation to the administration of the anaesthetic is recorded as :-

o.r.d. - operating room death - death occurring whilst the patient was anaesthetised.

< 24 - death within 24 hours of the administration of the anaesthetic.

> 24 - Death beyond the first post-operative 24 hours.

The sex of the patient is recorded as :-

m. - male.

f. - female.

The race of the patient is recorded as :-

E. - European.

C. - Coloured.

B. - Bantu.

Pre-operative assessment of anaesthetic risk is coded according to criteria described in Chapter 8, Section 2, Page 96.

Other abreviations used in the text are as follows :-

B.P. - Blood Pressure.

C.S.F.-Cerebro-spinal Fluid.

I.P.P.R. - Intermittent Positive Pressure Respiration.

d T.C. - d-Tubocurarine Chloride.

E.C.G. - Electrocardiogram.

E.E.G. - Electroencephalogram.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
1.1.1956	2	No comment.	< 24	Undetermined.	Yes

Name: Fred Benson. Age: 69. Sex: M. Race: C.

Disease: Carcinoma of lip, secondary gland in neck. Operation; Block dissection of neck.

Anaesthetic Risk: 2

PRE-OPERATIVE STATE:

Carcinoma of lip with secondary gland in neck. General physical state fair. Elective operation. B.P. 140/70 mm.Hg. Hb 13 mg.%. Respiratory and cardiovascular systems normal.

PREMEDICATION: Not recorded.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 200 mg. followed by nitrous oxide and oxygen in flow rates of 6 l. and 2 l./min. respectively, delivered via a Magill circuit with the patient breathing spontaneously. Trichlorethylene vapour was gradually added. Orotracheal intubation followed topical analgesia of larynx with 1% anethocaine. Anaesthesia was maintained with the same inhalational agents. During operation, blood was replaced as lost. The course of operation and anaesthesia was uneventful. At the conclusion of the operation and anaesthetic the patient regained consciousness within 5 minutes. Following a normal initial post-operative course, the patient was found dead during the night 12 hours post-operatively. Pethidine had been administered some 2 hours earlier.

AUTOPSY:

Other than for evidence of operation, there were no abnormal findings. No cause for death demonstrated.

COMMENT:

In the final analysis, cause of this death is undetermined. In that he had recovered adequately from the effects of the anaesthetic some hours before death, anaesthesia per se does not appear to have been contributory. Respiratory depression from the post-operatively administered Pethidine, together with the development of some upper respiratory tract obstruction from swelling at the operation site, are possible causes of this death. This latter was not obvious at autopsy. Some 30 minutes before death the patient's condition appeared normal to nursing staff.

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY.
2.1.1956	1	Possible	ORD	Cardiac arrest; uncertain aetiology	Yes

Name: Japhta Louw Age: 85. Sex: M. Race: C.

Disease: Gangrene of right leg. Operation: Planned above knee amputation (not done).

Anaesthetic Risk: 4, emergency.

PRE-OPERATIVE STATE:

This old patient was gravely ill. He had gross generalised arteriosclerosis with gangrene of the right leg. He appeared toxic and extremely emaciated. Pulmonary emphysema was present with poor respiratory excursion and poor air entry. He was not cyanosed. Blood pressure was recorded with difficulty, approximately 100 mm. Hg. systolic, with a pulse rate of 100/min.

PREMEDICATION:

Atropine 0.6 mg. by intramuscular injection 45 minutes pre-operatively. In addition, Omnopon 20 mg. was given in error.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen, with gradually added ether vapour, delivered via a Magill circuit with the patient breathing spontaneously. Within 5 minutes of commencement, during a quiet induction, there was a sudden cessation of respiration. IPPR was commenced immediately; a few respiratory gasps followed. Cardiac arrest was diagnosed. In view of the patient's age and gravely ill state, no cardiac massage was undertaken.

AUTOPSY:

1. Moist gangrene of the right lower leg.
2. Gross arteriosclerosis and calcification of aorta and coronary arteries. Coronary ostia almost occluded. Pericardial effusion. Calcified plaques in abdominal and thoracic aorta - gross sclerosis of other vessels.
3. Myocardium pale and flabby.
4. Massive bilateral pleural effusion and collapsed upper lobe.
5. Marked cirrhosis and fatty infiltration of liver.

COMMENT:

The immediate cause of death in this patient was cardiac arrest. The autopsy finding of pulmonary oedema indicates a period of left ventricular failure preceding this. There are two possible mechanisms to explain this. Vasodilation following the induction of anaesthesia may have led to a fall in systemic blood pressure, and therefore in aortic pressure. This, in the presence of the near complete obstruction of the coronary artery ostia demonstrated at autopsy, will have caused acute ischaemia of the myocardium and acute ventricular failure going on to cardiac standstill. The myocardium was pale and flabby at autopsy.

The fact that the blood pressure before anaesthesia was recorded with difficulty as approximately 100 mm.Hg systolic in this patient with gross arteriosclerosis, should have alerted the anaesthetist to the likelihood of a failing circulation and/or the presence of hypovolaemia. This condition should have been corrected before anaesthesia was commenced. Further, the monitoring of the patient's blood pressure during the induction of anaesthesia was not adequate.

The other ...

The other mechanism that may also have been contributory to this patient's death was anoxic anoxia, from inadequate pulmonary ventilation. Diagnosed as suffering from emphysema, this patient was found at autopsy to have bilateral pleural effusions and atelectasis of the right upper lobe of the lung. The anaesthetist appears to have been unaware of the presence of this pleural effusion. This, especially in the presence of the erroneous administration of 20 mg. Omnopon, may well have meant that the quiet induction described by the anaesthetist was in fact inadequate tidal ventilation. Because of the obvious faults in the anaesthetic management, anaesthesia is regarded as a significant causal factor in this patient's death, even though his disease is obviously also a major factor.

PREVENTABILITY:

Though this patient's death may be regarded as all but inevitable, in view of his grossly diseased state, the faults in the anaesthetic management are correctable. Their correction may have resulted in the patient's survival, at least of the operation. The death is therefore regarded as "possibly preventable".

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
3.1.1956	1	Possibly	ORD	Ischaemic anoxia; hypovolaemia; cardiac arrest.	Yes

Name: Isaac Goggle Age: 50 Sex: M Race: C

Disease: Carcinoma of the stomach. Operation: Total gastrectomy.

Anaesthetic Risk: 2

PRE-OPERATIVE STATE:

Carcinoma of the stomach with pyloric obstruction and profuse vomiting. Emaciated - weight 96 lb. - and malnourished. Respiratory and cardiovascular systems normal. B.P. 115/70 mm.Hg. No pre-operative electrolyte figures. Patient was deteriorating rapidly in spite of attempts to improve his condition pre-operatively.

PREMEDICATION:

Omnopon gr. ¼. Scopolamine gr. 1/200.

ANAESTHETIC:

Anaesthesia was induced with 350 mg. thiopentone sodium, followed by the inhalation of nitrous oxide and oxygen with gradually added ether vapour, administered via a Magill circuit, the patient breathing spontaneously. Following the induction ofanaesthesia, orotracheal intubation was performed, preceded by topical analgesia of the larynx and trachea with 1% amethocaine. Using gallamine, initial dose 80 mg., as the relaxant, an IPPR technique with carbon dioxide absorption was adopted for maintenance of anaesthesia. Minimal ether vapour was continued together with nitrous oxide and oxygen. After the induction of anaesthesia, the blood pressure commenced a steady progressive fall. Having been 100 mm.Hg systolic immediately before anaesthesia, it had fallen to a level of 70 mm. Hg systolic after 30 minutes. To this time the blood loss had been negligible. A vasopressor drug, methyl amphetamine 10 mg., was now administered with no effect. The patient was cold and clammy and blood pressure continued to fall, reaching a level of 60 mm.Hg after a further 10 minutes. The surgeon by now had transected the duodenum. Blood transfusion at a moderate rate was commenced. The initial transfusion of approximately 150 ml. blood caused the patient to rally and the blood pressure rose to 80 mm.Hg systolic. The pulse rate continued all this time at 120/minute. Improvement was short-lived and once again the blood pressure fell. At this stage the administration of ether was discontinued, a total of 3½ oz. had been used. A further 10 mg. methyl amphetamine was given with no effect. The operating table was tipped head down but, in spite of this, the blood pressure became irrecordable though the carotid pulse was palpable. The rapid transfusion of 1 pint blood at this stage had no effect. With the operation all but completed, 100 minutes after the induction of anaesthesia, the pulse became impalpable. Cardiac arrest in asystole was confirmed by palpation of the heart through the diaphragm. All anaesthetic agents were discontinued in favour of IPPR with oxygen, and cardiac massage was commenced. Spontaneous beating recommenced only to be followed shortly by ventricular fibrillation. Cardiac massage was recommenced and was followed shortly by spontaneous beating of the heart again. Shortly after this, cardiac arrest again supervened and all further resuscitative measures were of no avail.

Autopsy ....

AUTOPSY:

Nil significant found. Evidence of subtotal gastrectomy. Carcinomatous gland in porta hepatis. Bruising of pericardium, obviously from cardiac massage. Smell of ether in brain.

COMMENT:

The administration of an anaesthetic to this patient appears to have precipitated a state of inexorable peripheral circulatory failure. The prolonged hypotension so resulting, and consequent ischaemic anoxia, must be considered the principle cause of the cardiac arrest and the patient's ultimate demise.

The probable basis for this response to anaesthesia was hypovolaemia. The nature of the patient's lesion - carcinomatous pyloric obstruction with chronic vomiting - would have set the stage for this. The low blood pressure recorded immediately before anaesthesia (100 mm.Hg) should have alerted the anaesthetist. The response to anaesthesia is typical of the patient with hypovolaemia for which circulatory compensation, e.g. vasoconstriction, has been made. Once the compensating mechanisms have been disturbed by anaesthesia, the full picture of hypovolaemic circulatory failure becomes apparent.

The fault in this case was the failure of the anaesthetist to recognise this. This state of peripheral vascular failure should have been treated promptly, even though blood loss itself was minimal. Instead, treatment was tardy and, when some action was taken, a vasopressor drug was used. Only later was blood transfusion resorted to - and then it was a case of 'too little too late'.

Another fault apparent in the conduct of this anaesthetic was the dose of thiopentone with which anaesthesia was induced. For this emaciated (96 lb.) depleted patient, I feel a dose of 350 mg. is excessive. The steady circulatory deterioration commenced with its administration. This is a well recognised sequel of thiopentone administration in this type of case.

The administration of ether vapour should perhaps have been discontinued earlier in the anaesthetic, even though given in minimal concentration. This would have allowed of some vasoconstriction which would have assisted circulatory homeostasis.

The anaesthetic management is regarded as a significant factor in this patient's death, in the probable presence of inadequate preoperative preparation, especially with regard to hydration and electrolyte balance.

PREVENTABILITY:

The faults apparent in the conduct of the anaesthetic are correctable. A greater awareness of the derangements caused by the disease should have resulted in more correct and prompt treatment of the circulatory failure. Because of this, even though much of the difficulty in all probability resulted from inadequate preoperative preparation of the patient, this death must be regarded from the viewpoint of anaesthetic management as "possibly preventable".

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY.
4.1.1956	3	No comment	ORD	Uncontrollable haemorrhage from pulmonary artery. Secondary cardiac arrest.	Yes

Name; Magdalena M. van Rensburg. Age: 29. Sex: F Race: C.

Disease: Patent ductus arteriosus. Operation: Ligation of patent ductus arteriosus.

Anaesthetic Risk: 2

PRE-OPERATIVE STATE:

The patient's physical state was considered fair. Previous cardiac failure had been controlled by digitalisation.

PREMEDICATION: Not recorded.

ANAESTHETIC:

Anaesthesia was induced with 200 mg. thiopentone sodium followed by inhalation of nitrous oxide and oxygen with gradually added ether vapour. Following induction of light anaesthesia and topical analgesia of larynx, orotracheal intubation was performed. For maintenance of anaesthesia, an IPPR technique using nitrous oxide and oxygen only with carbon dioxide absorption (circle) was adopted, using dTC as relaxant.

Three hours after start, the pulmonary artery was torn during surgical closure of the ductus. Torrential, uncontrollable haemorrhage occurred. Blood was replaced as rapidly as possible to no avail. Secondary cardiac arrest, which would not respond to cardiac massage, ensued after some 30 minutes. Throughout operation, 14 pints of blood were transfused. Time from induction to death: 7 hours.

AUTOPSY:

Lungs: Right - areas of patchy collapse in apical lobe. Weight 610 gm. Left - partially collapsed. Weight 305 gm. Haematoma at root of lung. 250 ml. blood in left pleural cavity.
Heart: Enlarged. Weight 600 gm. with marked enlargement of the left ventricle.
Large blood vessels: Patent ductus has been firmly ligated, sewn and divided.
Brain meninges and cerebral vasculature - pale; smell of ether.

COMMENT:

This death resulted from surgical mishap. Anaesthesia is not regarded as contributory.

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY.
5.1.1956	2	No comment.	< 24	Cerebral contusion and laceration	Yes.

Name: Jennifer Lennox. Age: 8 Sex: F Race: E

Disease: Fractured skull with cerebral contusion. Operation: Carotid angiography and burrhole craniotomy.

Anaesthetic Risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was comatose and in very poor condition. Carotid angiography performed without anaesthetic.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

The patient was intubated orally after topical analgesia of the larynx. Nitrous oxide and oxygen in flow rates of 3 l. and 2 l./ minute respectively were administered, via a Magill circuit, the patient breathing spontaneously.

During operation, her condition deteriorated. The B.P. 130/60 mm. Hg at start, had fallen to 90 mm.Hg at the conclusion, while the pulse rate rose from 150/minute to 180/minute. At operation, gross cerebral contusion and laceration were found. The duration of operation was 1 hour. Following anaesthetic and operation the patient failed to regain consciousness. She died ½ hour post-operatively, in the ward.

AUTOPSY:

Comminuted fractures involving left parieto-occipital and right parietal regions of skull. Fracture involving anterior cranial fossa and left middle cranial fossa. Dura tense and brain pale. Bruising over whole left hemisphere extending to a depth of 1 inch in places. Haemorrhage in mid-brain and anterior corpus callosum. Haemopericardium with bruising over left ventricle 2 x 3 cms. Heart pale and contracted. Haemoperitoneum with lacerated spleen.

COMMENT:

This patient died of multiple injuries and the anaesthetic per se does not appear to have contributed. Judging from the drop in blood pressure with sharply rising pulse rate during the operation, the haemopericardium, ?tamponade and haemorrhage from the spleen would appear to be the more immediate causes of death as opposed to the cerebral laceration - though this is supposition.

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY.
6.1.1956	2	No comment	< 24	Cerebral cysticer-osis.	Yes.

Name: Johannes Adams. Age: 31 Sex: M Race: C

Disease: Cerebral cysticercosis. Operation: Carotid angiography. Craniotomy, excision of hydatid cysts.

Anaesthetic Risk: 2.

PRE-OPERATIVE STATE:

The patient had symptoms and signs of cerebral compression. His general physical state was good.

PREMEDICATION: Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia induced with thiopentone sodium 200 mg. and inhalation of nitrous oxide, oxygen, trichlorethylene, going on to ether via a Magill circuit, the patient breathing spontaneously. Oral intubation followed topical analgesia of larynx with amethocaine 1%.

During carotid angiography, apnoea followed injection of dye. IPPR was commenced. Following craniotomy and relief of cerebral compression, spontaneous respiration recommenced. Further course of anaesthesia uneventful. One hydatid cyst removed. The patient recovered consciousness post-operatively and breathed adequately. Death occurred 6 hours post-operatively, from causes unrelated to the anaesthetic.

AUTOPSY:

Surgical wound in scalp. Hole in left cerebral hemisphere. Hydatid cysts at anterior pole of left cerebral hemisphere and vertex of right cerebral hemisphere.

Detail: Extradural haematoma ½ cm. thick, 9 x 6 cm. under area of burrholes. Flap of dura under burrholes sutured. Hole in vertex 2 cm. from midline on left cerebral hemisphere 3 x 2 cm. equadistant from occiput and frontal lobe. Hydatid cyst 2 x 2 cm. directly opposite in same position on right cerebral hemisphere. Subdural haemorrhage 6 x 6 cm. underlying sutured area of dura. Hydatid cyst 1 x 2 cm. on anterior pole of left frontal lobe with calciferous wall.

COMMENT:

This patient recovered satisfactorily from the anaesthetic. Death appears to have resulted from the effects of surgery and the existing cerebral disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
7.1.1956	1	Possibly.	< 24	Profound hypotension. Ischaemic anoxia. Cerebral ischaemia.	Yes.

Name: Christoffel de Wet. Age: 80 Sex: M Race: E.

Disease: Obstructive jaundice, carcinoma of head of pancreas. Operation: Cholecystenterostomy.

Anaesthetic Risk: 3.

PRE-OPERATIVE STATE:

Poor: arteriosclerotic and grossly jaundiced. The patient had gross cirrhosis of liver. His weight was 130 lb.

PREMEDICATION:

ANAESTHETIC:

Following induction of anaesthesia with thiopentone sodium 150 mg., anaesthesia was deepened with inhalation of nitrous oxide, oxygen (6 l. and 2 l/minute respectively) with gradually added ether vapour, the patient breathing spontaneously. A carbon dioxide circle absorption system was used. Oral intubation was performed when moderate surgical anaesthesia had been induced. An IPPR technique was used following administration of 80 mg. gallamine, and administration of a trace of ether vapour was continued.

The patient's blood pressure - 180 mm.Hg systolic before anaesthesia - dropped during induction to 80 mm.Hg systolic. The pulse rate remained 100/minute until injection of gallamine caused it to rise to 120/minute. Methylamphetamine 6 mg. caused the B.P. to rise to 160-180 mm.Hg systolic. Further course of anaesthesia appears to have been uneventful during the remainder of the operation, which lasted 60 minutes. Blood loss during operation was minimal and no blood was transfused. At the conclusion of the operation, normal respiration resumed. Residual curarisation was adequately reversed with 0.5 mg. neostigmine, preceded by 0.6 mg. atropine.

While the patient was being removed from the operating table, a marked drop in B.P. to 80 mm.Hg systolic occurred. Cyanosis of the extremities with poor capillary refill became evident. 12 mg. methylamphetamine was administered with no effect. Noradrenaline 4 microgram./ml. was infused. B.P. recovered to 100 mm.Hg systolic but pulse became irregular, with short periods of asystole - probably the result of multiple extrasystoles. Following his return to the ward, the patient failed to regain consciousness completely, though pharyngeal reflexes returned. Circulatory failure persisted post-operatively. The administration of noradrenaline was continued, together with 500 ml. 5% dextrose in water. The patient died 7 hours post-operatively.

AUTOPSY:

Surgical wound right side of abdomen with stitches in situ. Rubber drain lateral to wound. Cholecystenterostomy. Deep jaundice. Both lungs congested. Peritoneal cavity contained 150 ml. clotted blood under right lobe of liver. Haematoma, about ½ cm. thick, in hepatorenal and gastrohepatic omentum.

Liver ....

Liver: Weight 1966 gm. Cirrhotic and greenish-yellow.
Gall bladder thickened and sutured to first loop of jejunum.
Pancreas: carcinoma of head.
Spleen: congested.

COMMENT:

The surgical procedure here was of little magnitude. Autonomic reflexes and inferior vena caval compression often associated with surgery in this region do not appear to have occurred.

There is no doubt that the circulatory failure was precipitated by the administration of the anaesthetic, probably on the basis of vasodilation inherent in the use of ether. The initial treatment of this state with a vasopressor may have been correct as an emergency measure, but the subsequent failure to transfuse blood and/or fluids in a patient who was probably hypovolaemic before surgery must be faulted - even though blood loss at operation was minimal.

The ultimate circulatory collapse that followed movement of the patient at the end of operation is in keeping with a state of hypovolaemia. Again, recourse was made to vasopressor drugs and not to blood volume expanders. This treatment was persisted with until death.

Judging from the autopsy findings, left ventricular failure was ultimately responsible for the patient's demise. This is not uncommon in circumstances where treatment with vasopressors is continued in the face of hypovolaemia. That the patient did not recover consciousness leads one to suspect that, in the presence of cerebral arteriosclerosis, the episodes of gross hypotension resulted in cerebral ischaemia. Though his poor physical status is doubtless a contributory factor to his death, considered in this context the management of the anaesthetic must be considered a significant contributory factor.

PREVENTABILITY:

Because of the midjudgment and mismanagement of this circulatory collapse, precipitated by the anaesthetic, this case must be considered "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY.	TIME OF DEATH.	CAUSE OF DEATH.	AUTOPSY
8.1.1956	1	Possibly.	< 24	Profound hypotension. Ischaemic anoxia. Cerebral ischaemia.	Yes.

Name: Morris Wertheim. Age: 49. Sex: M Race: E.

Disease: Stone in the common bile duct. Obstructive jaundice.

Operation: Laparotomy and exploration of the common bile duct.

Anaesthetic Risk: 2.

PRE-OPERATIVE STATE:

This obese patient had had a cholecystectomy 1 year previously. He now presented with obstructive jaundice of 5 days' duration. Weight - 230 lb. Serum bilirubin 10.5 mg.%. B.P. 110 mm.Hg sytolic. Other systems normal.

PREMEDICATION:

Pethidine 100 mg., Scopolamine gr. 1/150, by intramuscular injection 45 minutes before the anaesthetic.

ANAESTHETIC:

Immediately before induction of anaesthesia the patient's B.P. was 100 mm.Hg systolic. Anaesthesia was induced with the sequence thiopentone 400 mg., gallamine 120 mg., ventilation with oxygen, topical analgesia of the larynx, oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system, by an IPPR technique.

During the course of the operation one further dose of gallamine 40 mg. was administered. Immediately following induction of anaesthesia, peripheral cyanosis with slow capillary refill was noticed. The position and patency of the endotracheal tube, the volume of pulmonary ventilation and auscultated air entry were immediately checked. They appeared adequate. The patient was now positioned half rotated to the left and tilted 10° foot-down. The B.P. was now recorded as 80 mm.Hg. As the initial B.P. was but 100 mm.Hg systolic, no active treatment was adopted at this stage. After remaining at this level for 30 minutes the B.P. fell further. Two successive doses of methylamphetamine 6 mg. were administered intravenously without effect. Peripheral cyanosis was now more marked. Capillary refill time was very prolonged. The operating table was now levelled, then tilted head-down, without effect on the state of the circulation.

One pint of blood was transfused rapidly, though blood loss from the operative site was negligible. A drip infusion of nor-adrenaline 8 micrograms/ml. was commenced. The B.P. rose to 90 mm.Hg systolic.

Exploration of the common bile duct revealed a large impacted stone. This was removed. The operation was complete in 1¾ hours. At the conclusion of operation the patient's pupils were ¾dilated and reacted sluggishly to light. Gasping spontaneous respiration commenced. The tidal volume did not appear adequate. Injection of 2 mg. neostigmine preceded by atropine 0.6 mg. was followed by an increase in the tidal volume of ventilation to an adequate level,

but ...

but the gasping pattern of respiration persisted. The B.P. remained at a level of 90 mm.Hg in spite of the continued infusion of noradrenaline. The patient was kept in the operating theatre, breathing oxygen only, for 2 hours after operation. He failed to regain consciousness but, at the end of this time, pharyngeal reflexes were returning, as was the eyelid reflex. He was returned to the ward. There, his condition remained in status quo for 1½ hours, whereupon a rapid deterioration occurred and he died without regaining consciousness.

AUTOPSY:

Enlarged fatty liver. Enlarged heart. Coronary and aortic atheroma. Evidence of operation on the common bile duct.

COMMENT:

This death was undoubtedly due to the anaesthetic and its management. The onset of profound hypotension, with peripheral circulatory stasis, followed immediately on the administration of thiopentone and the induction of anaesthesia. For a man of this weight (230 lb.) the actual dose of thiopentone given - 400 mg. - does not appear excessive. The profoundness and persistence of the effect may have been related to impaired liver function, the result of biliary obstruction.

The 100 mg. pethidine administered as premedication may well have been synergistic with the thiopentone in the production of hypotension. Once hypotension had occurred, expectant treatment was continued for too long, and worse - the anaesthetist permitted the positioning of the patient in a head-up tilt. This error in judgement may have resulted from a failure of the anaesthetist to realise that the level of systolic B.P. recorded in this patient pre-operatively may not have been its normal level, but a subnormal level - the result of bilirubinaemia.

The autopsy revealed an enlarged heart, probably a sign of previous hypertension. It is postulated that the prolonged hypotension caused cerebral ischaemia and cellular anoxic damage. The subsequent further fall in systolic B.P. was due, no doubt, to myocardial failure following poor coronary perfusion.

The question arises whether the post-anaesthetic respiratory failure pattern displayed by this patient was not due to prolonged action of the relaxant drug used - 'neostigmine resistant curarisation'. This is a possibility, especially as the circulatory failure will probably have led to metabolic acidosis. However, hypotension and circulatory failure were the most prominent features throughout and this persisted despite adequate pulmonary ventilation during anaesthesia. Post-anaesthetic, the tidal volume of ventilation appeared adequate. The gasping pattern of respiration was in keeping with cerebral damage, as was the failure of the patient to regain consciousness after anaesthesia.

PREVENTABILITY:

Treatment of this gross hypotension was delayed for too long. A foot-down tilt of the patient should not have been permitted. In retrospect, the doses of both thiopentone and pethidine were large for this patient. These faults are correctable and this death is therefore regarded as "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY.	TIME OF DEATH.	CAUSE OF DEATH.	AUTOPSY
9.1.1956	2	No comment	> 24	Damage to anterior communicating artery.	Yes.

Name: Clara Markham Age: 49 Sex: F Race: E

Disease: Cerebral tumour. Operation: Bifrontal craniotomy, removal of supra-cellarmeningeoma.

Anaesthetic Risk: 3.

PRE-OPERATIVE STATE:

Poor: the patient displayed signs of cerebral compression. Level of consciousness was depressed but not comatose.

PREMEDICATION:

Atropine gr. 1/100. Largactil 50 mg. Pethidine 50 mg. Phenergan 50 mg. by intramuscular injection 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone followed by nitrous oxide, oxygen and ether. Larynx was sprayed with Anethaine and oral intubation performed. Hypothermia was induced by immersion in a bath of iced water. Temperature subsequently maintained at 88°F. Further anaesthetic course throughout the operation was relatively trouble-free.

Bifrontal craniotomy was performed, the tips of the frontal lobes excised and the supracellar tumour demonstrated. The optic nerve ran through the tumour. The left optic nerve was deliberately divided and the right was freed by dissection. Dissection of the posterior part of the tumour was difficult because of bleeding. The anterior communicating artery was torn at the junction with the right anterior cerebral. Temporary clips were applied at both anterior cerebral arteries and the bleeding point packed with muscle. On removal of the temporary clips, the anterior cerebral artery circulation appeared satisfactory.

The patient remained unconscious until death, 30 hours post-operatively. The temperature remained low - 90°F.

AUTOPSY:

Right temporal lobes softening. Pituitary enlarged. Streaky pontine haemorrhage. Intraventricular haemorrhage.

COMMENT:

There appears to be sufficient surgical cause to account for this death, borne out by the post-mortem evidence.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY.	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
10.1.1956	1	Possibly	> 24	Prolonged cerebral ischaemia due gross haemorrhage during hypotensive anaesthesia	Yes

Name: Dorothy Fraser Age: 41 Sex: F Race: E

Disease: Left olfactory groove in meningeoma. Operation: Craniotomy. Excision of frontal meningeoma.

Anaesthetic Risk: 2

PRE-OPERATIVE STATE:

The patient displayed signs of intracranial compression.

PREMEDICATION:

Largactil 50 mg. Phenergan 50 mg. Pethidine 50 mg. Atropine gr. 1/100, by intramuscular injection 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether. The larynx was sprayed with Anethaine and oral intubation was performed. Spontaneous respiration was maintained throughout using a Magill circuit. Hypotension was induced by means of an Arfonade drip infusion.

During a period of great operative blood loss, intravenous transfusion failed and great difficulty was experienced in re-establishing the intravenous infusion. Because of this, a period of 30 minutes (approximately) profound hypotension ensued during which the B.P. could not be recorded. Following the operation, the patient failed to recover consciousness. Three hours post-operatively she responded to painful stimuli. On the day following operation she could obey simple commands but could not speak. Slight paresis of the right side ensued. This later became more profound and the patient responded readily to stimulation. On the second post-operative day, breathing failed. Endotracheal intubation was resorted to and artificial respiration instituted. On the same day the wound was re-opened and the brain was found to be under severe tension. Therapy with hypnotic sucrose solution did not improve this. On the third post-operative day the patient's B.P. had dropped to between 60-80 mm.Hg systolic, and the pulse was hardly perceptible. A stellate block on the right side had no effect on the hemiparesis. The patient died on the fourth post-operative day.

AUTOPSY:

There was softening of the left frontal lobe of the brain, extending back to involve the caudate nucleus and internal capsule on the left side.

COMMENT ...

COMMENT:

It is difficult to say whether the period of hypotension during operation was a contributory factor to the fatal outcome in this case, as the tumour with its attachment to the hypothalamus itself was a considerable risk to life. But, on the grounds that one of the risks of a planned hypotensive anaesthetic technique is the lack of compensatory mechanism to severe haemorrhage, and that the failure of blood transfusion in this patient resulted in a period of 30 minutes' extremely profound hypotension, severe enough to have caused cerebral damage (evident on autopsy examination), the anaesthetic technique must share some of the responsibility for the patient's death. That the intravenous transfusion failed at a crucial moment may perhaps be classed as a mishap, but this might have been prevented had an alternative line been instituted at the commencement.

This case is classed in Group 1.

PREVENTABILITY:

Because the precaution of establishing an alternate infusion site - a 'fail-safe' precaution - may have avoided this death, it is regarded as "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
11.1.1956	1	Possibly	ORD	Cardiac arrest. Reflex vagal arrest.	Yes

Name: Howard Fukile Age: 2 Sex: M Race: B

Disease: Gross right pulmonary bronchiectasis. Operation: Right pneumonectomy.

Anaesthetic Risk: 3

PRE-OPERATIVE STATE:

This patient was assessed a very poor operative risk as he had pulmonary disease of sufficient magnitude to cause orthopnoea. Further, there was a profuse amount of purulent bronchial secretion. Grave difficulties had been experienced with an anaesthetic administered two weeks previously, while the child underwent a bronchogram.

PREMEDICATION: Scopolamine 0.2 mg.

ANAESTHETIC:

Anaesthesia was induced with open ether. When anaesthesia was of sufficient depth, oral intubation was performed. Bronchial toilette was performed. Anaesthesia was maintained with ether and oxygen delivered through a modified T-piece with infant reservoir bag. Respiration was assisted throughout but was not controlled. A degree of spontaneous respiration persisted throughout the operation. No relaxant was used.

The course of the operation and anaesthesia was relatively smooth and uneventful, the B.P. being maintained at between 100 and 110 mm.Hg systolic, the pulse rate 108/minute. When the right bronchus was clamped, a cardiac arrest occurred. Cardiac massage and intracardiac adrenaline restored the heart action after 41 minutes. However, the child's respiration ceased and cardiac arrest occurred once more. Death was presumed about 1 hour 20 minutes after commencing anaesthesia.

AUTOPSY:

Recent right pneumonectomy. Bronchial stump secure. Nothing else of note. Slight cerebral oedema. Right haemothorax approximately 30 cc. blood. Liver grossly enlarged with fatty infiltration. The specimen of right lung removed at operation revealed gross bronchiectasis.

COMMENT:

This death would appear to be primarily due to a reflex cardiac arrest resulting from hilar manipulation. As the respiration was never controlled, and in view of the gross pulmonary disease, it is possible that some degree of anoxia, hypercarbia and mediastinal flap were factors in providing the background on which vagal arrest might occur. The anaesthetic therefore is considered a contributory factor, together with the patient's disease, in the causation of this death.

PREVENTABILITY ....

PREVENTABILITY:

In view of the fact that properly controlled respiration, IPPR, may possibly have provided better ventilation and oxygenation, and entirely prevented a mediastinal flap, this death may be considered as "possibly preventable". Although this may be overstating the case, it stresses a possible clinical lesson to be learned.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
12.1.1956	2	No comment	< 24	Post-op. haemorrhage	Yes

Name: Maria Hilberg Age: 66 Sex: F Race: C

Disease: Obstructive jaundice. Operation: Exploration of common bile duct.

Anaesthetic Risk: 3

PRE-OPERATIVE STATE:

The patient had been cholaemic and was grossly jaundiced. She had ascites and had been treated with Digitalis. She was now clinically jaundiced, had severe ascites and a palpable cirrhotic liver. Prothrombin index 80. The patient had had two previous operative explorations of the common bile duct.

PREMEDICATION:

Pethidine 75 mg. Atropine gr. 1/100, by intramuscular injection 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with 250 mg. thiopentone followed by inhalation of nitrous oxide, oxygen and ether. The larynx was sprayed with Anethaine 1% and oral intubation was performed. Gallamine 60 mg. was then administered and an IPPR technique instituted with nitrous oxide, oxygen and ether through a carbon dioxide circle absorption circuit.

The operation was technically extremely difficult because of the gross and dense adhesions. Profuse bleeding occurred from these throughout the operation. Blood was replaced as lost, but the B.P. dropped steadily throughout the procedure. Methyl amphetamine 15 mg. administered intravenously had little effect on the drop in B.P. and a noradrenaline drip infusion was instituted. This produced a rise in B.P. from 60 to 100 mm.Hg systolic, at which level it was maintained to the end of the operation. Normal respiration was re-established at the conclusion of the operation. Neostigmine was not considered necessary.

Following discontinuation of the anaesthetic, the patient regained consciousness rapidly and was returned to the ward. Bleeding continued post-operatively, being voided from the abdomen through the drainage tubes. Twelve pints of blood was replaced but, despite this, the patient died 8 hours post-operatively.

AUTOPSY:

Gross cirrhosis of the liver. Enteroperitoneal haemorrhage. Enlarged heart. Evidence of recent surgery to abdominal cavity. Skin and subcutaneous tissues pale yellow. Right and left lungs: partial collapse, not congested. Heart weighed 400 gm. Left ventricular enlargement. Moderate atherosclerosis of coronary arteries. Moderate atherosclerosis of aorta. Peritoneal cavity contained 150 ml. blood. The liver was grossly cirrhotic and yellowish green. Bile ducts not seen, buried in a mass of fibrous tissue. Spleen - enlarged and congested, weighing 560 gm.

COMMENT:

Although this patient had a stormy operative course, she recovered from the anaesthetic, to all intents and purposes, immediately post-operatively. Death would appear to have resulted from severe operative and post-operative haemorrhage in the presence of gross liver disease, in a patient with a diseased heart. The anaesthetic is not considered contributory to the fatal outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
13.1.1956	3	No comment.	< 24	Cardiac arrest. Surgical manipulation. Massive post-op. haemorrhage.	Yes

Name: Decia Arries Age: 5 Sex: F Race: C

Disease: Tetralogy of Fallot. Operation: Pulmonary valvotomy.

Anaesthetic Risk: 4.

PRE-OPERATIVE STATE:

This patient, suffering from a classical tetralogy of Fallot, was considered a poor operative risk.

PREMEDICATION:

Pethedine 25 mg. Scopolomine gr. 1/200, by intramuscular injection 1 hour before anaesthetic.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen with gradually added ether and oral intubation was performed. Anaesthesia was maintained with ether and oxygen administered via Water's cannister. dTC was used as the relaxant. An IPPR technique was used throughout.

Following pulmonary valvotomy, performed through a right ventriculotomy, cardiac arrest ensued. Cardiac massage, intracardiac adrenaline, Atropine gr. 1/100 and procaine amide 350 mg. intravenously led to the re-starting of the cardiac action. Noradrenaline 1 cc. in a 100 cc. dextrose in water was administered. Respiration following closure of the thorax was irregular. This gradually improved. At the conclusion of the operation, Atropine gr. 1/100 followed by 0.5 mg. neostigmine were administered. Subsequently there was a slow improvement in respiration, which became more regular. However, the child failed to regain consciousness. Shortly after the return of the patient to the ward she suffered a sudden gross intrathoracic haemorrhage which led rapidly to her death.

AUTOPSY:

Surgical left thoracotomy wound plus a small haemothorax. Brain meninges and cerebral vasculature showed venous congestion. Right and left lungs congested. Heart: incision in right ventricle; varicose vegetations on pulmonary valve cusps with marked stenosis. Hypoplastic pulmonary artery and right ventricular hypertrophy. Tetralogy of Fallot. Small straw coloured peritoneal effusion.

COMMENT:

This death was due to haemorrhage. If this had not occurred, she might well have died from a cerebral anoxia which occurred at the time of cardiac arrest. Anaesthesia is not considered to have played any part in this patient's death. However, as cardiac arrest occurred while the patient was anaesthetised and she failed to regain consciousness, the case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
14.1.1956	3	No comment	ORD	Cerebral laceration. Subdural haematoma. Oedema of brain stem. Cardiac arrest.	Yes.

Name: Africa Syster Age: 30 Sex: M Race: B

Disease: Head injury. Subdural haematoma. Operation: Angiography followed by craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient was in extremis and comatose. Carotid angiography was performed without anaesthesia.

ANAESTHETIC:

The patient was intubated following topical analgesia of the larynx, using Amethocaine 1%. Patient commenced a continued violent coughing. Anaesthesia then induced with nitrous oxide, oxygen, trace ether. After one hour, respiration commenced failing. Respiration was then assisted, however, respiratory failure was progressive and ultimately failed completely. Cardiac arrest ensued. A torn sagittal suture was found at operation. Cardiac massage was of no avail.

AUTOPSY:

Subdural haematoma. Oedema of the brain stem. Laceration of the sagittal sinus.

COMMENT:

This death is considered to have been due to severe cerebral injury and brain stem oedema, the anaesthetic playing no more than a minor role, In that death occurred while the patient was anaesthetised, anaesthesia is regarded as "necessarily contributory".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
15.1.1956	2	No comment	< 24	Myocardial infarction with haemorrhage into atheromatous plaque.	Yes

Name: Lucy Wheeler Age: 58 Sex: F Race: E

Disease: Haematemesis. Operation: Partial gastrectomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was in an extremely poor state pre-operatively. She had had a severe maenatemesis and melaena from a duodenal ulcer. She was also in severe cardiac failure. She was resuscitated with blood transfusion. Digitalisation was commenced with Digoxin 0.75 mg.

PREMEDICATION:

Atropine gr. 1/100 by intramuscular injection 45 minutes before anaesthetic.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by nitrous oxide, oxygen and ether. She was intubated orally. Following administration of gallamine 100 mg., an IPPR technique with carbon dioxide absorption was used.

A partial gastrectomy was performed. The B.P. remained steady throughout the procedure. At the conclusion of operation, the patient recovered consciousness rapidly. Respiration was adequate. Neostigmine 1 mg. preceded by Atropine 0.6 mg. was given to ensure reversal of residual curarisation. Later there was some peripheral cyanosis. Oxygen was administered and a further dose of Digoxin 0.25 mg. was administered. The patient was returned to the ward in a deteriorating condition and died 1½ hours post-operatively.

AUTOPSY:

Myocardial infarction with a haemorrhage into an atheromatous plaque.

COMMENT:

The cause of this patient's death is obvious from autopsy. Anaesthesia is not considered to have played a contributory part. It is interesting to speculate at which stage the myocardial infarction occurred.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY.
16.1.1956	2	No comment.	< 24	?Pulmonary oedema.	No

Name: Johanna Ahrendse. Age: 41 Sex: F Race: C

Disease: Ruptured ectopic pregnancy. Operation: Laparotomy. Salpingotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient was shocked pre-operatively, following rupture of an ectopic pregnancy. Resuscitation with blood was commenced pre-operatively. Immediately before anaesthesia, B.P. was 80 mm.Hg systolic.

PREMEDICATION:

Pethedine 75 mg. Atropine gr.1/100.

ANAESTHETIC:

Anaesthesia was induced with the inhalation of nitrous oxide, oxygen and ether. Oral intubation was performed and gallamine 20 mg., followed by a further 20 mg. during operation, was administered. An IPPR technique was used throughout, via a carbon dioxide circle absorber.

Following clamping of the ruptured fallopian tube at operation and transfusion of blood, a rapid improvement in the patient's blood pressure occurred - to a level of 95 mm.Hg systolic with a pulse rate of 132/minute. At the conclusion of operation, Atropine gr. 1/100 followed by neostigmine 0.5 mg. was administered. Respiration was normal and the patient rapidly regained consciousness. She died 5½ hours post-operatively from clinical pulmonary oedema.

AUTOPSY:

There was no autopsy.

COMMENT:

That this patient died post-operatively from what appeared clinically to be pulmonary oedema leads one to suspect an element of over-transfusion in the ward post-operatively. The administration of an anaesthetic itself had nothing to do with this episode, and as such the anaesthesia is considered non-contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
17.1.1956	2	No comment	< 24	?Pulmonary oedema.	No.

Name: J.Davids. Age: 50 Sex: F Race: C.

Disease: Carcinoma of the uterus. Operation: Wertheim's hysterectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient was of a fair physical status pre-operatively. A previous chronic haemorrhagic anaemia had been corrected by a blood transfusion.

PREMEDICATION:

Atropine 0.6 mg.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether. Oral intubation was performed. Gallamine 60 mg. was administered and an IPPR technique was instituted via a circle carbon dioxide absorber. Anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique. A trace of ether was administered throughout.

The operative course was relatively uneventful. At the conclusion of the operation the patient regained consciousness rapidly and respiration was normal. Administration of neostigmine was considered unnecessary. The patient died 13 hours post-operatively.

As far as can be ascertained, the patient received 1 litre of dextrose in water, administered rapidly inadvertently just before death. Death was thought to have occurred from pulmonary oedema.

AUTOPSY:

No autopsy was performed.

COMMENT:

From the nature of this patient's death, it would appear that the anaesthesia played no part. Death seems to have occurred as a result of the inadvertent administration of excess fluid.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
18.1.1956	2	No comment	< 24	Gunshot wounds. ?Cerebral laceration.	Yes.

Name: Abraham Kotze Age: 55 Sex: M Race: E.

Disease: Gunshot wounds of the face.

Operation: Debridement of wounds. Immobilization of fractures. Reconstruction of face.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

General condition satisfactory. Local condition - there were severe lacerations of the face, fractures of the lower jaw, fragmented tongue and floor of mouth, and comminuted fracture of the hard palate exposing maxillary and nasal sinuses.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg. The larynx was sprayed with Anethaine 1% and oral intubation with a 10 cuffed tube was performed. Anaesthesia was maintained with nitrous oxide and oxygen using a Magill circuit, with spontaneous respiration throughout.

Post-operatively the patient regained consciousness rapidly but was violent in the theatre. Airway was clear, being thus maintained by having the tongue fixed by a suture. The patient died 3 hours post-operatively.

AUTOPSY:

Though autopsy was performed, no record of the report has been found.

COMMENT:

This patient appears to have died of cerebral injury resulting from gunshot wounds. Anaesthesia is not thought to have played any part in this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
19.1.1956	2	No comment	< 24	Hypertensive crisis. Phaeochromocytoma.	Yes.

Name: Frances Cato. Age: 33 Sex: F Race: C.

Disease: Severe re-eclamptic toxaemia. ?Malignant hypertension.

Operation: Anterior hysterotomy and sterilisation.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This patient, who was 26 weeks' pregnant, was in fair general condition. Her B.P., which was 260/140 mm.Hg, had been rising rapidly during recent weeks.

PREMEDICATION:

Morphine gr. ¼, Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether via a Magill's circuit, with spontaneous respiration. Gallamine, total dose 60 mg., was administered throughout the operation. Spontaneous respiration was permitted throughout most of the procedure, though for a short period IPPR was employed. No endotracheal intubation was performed.

During the operation - anterior hysterotomy and sterilisation - no untoward incident occurred. The duration of operation was 45 minutes. The patient recovered consciousness rapidly post-operatively. Respiration was normal. She died suddenly 14 hours post-operatively from what appeared clinically to be a pontine haemorrhage: she becams suddenly hyperpyrexial, developed pin-point pupils, became comatose and died.

AUTOPSY:

Mucous membranes and all internal viscera very pale. Signs of dehydration. Brain meninges and cerebral vasculature very pale, no evidence of pontine or intracerebral haemorrhage. Both lungs pale and collapsed. Heart and pericardium pale and contracted. Liver pale and soft. Spleen small and soft. Adrenals - right suprarenal phaeochromocytoma measuring 4 x 4 cm., weight 35 gm. Kidneys pale. Uterus - evidence of hysterotomy, all sutures secure.

COMMENT:

The anaesthetic is not implicated in this patient's death, which appears to have resulted from a hypertensive crisis, the result of a phaeochromocytoma.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
20.1.1956	1	Possibly	> 24	Cerebral anoxia (anoxic). Post-relaxant respiratory abnormality.	No

Name: Ellen Wagner. Age: 74. Sex: F Race: E.

Disease: Large ventral hernia. Operation: Repair of ventral hernia.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This patient was obese; though weighing 140 lb. she was only 5 ft. 2 ins. tall. The ventral hernia was large, she was old, had obvious arteriosclerosis and mild hypertension. B.P. 160/100 mm.Hg.

PREMEDICATION:

Morphine hydrochloride gr. $\frac{1}{6}$, Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. and inhalation of nitrous oxide, oxygen and ether vapour. Following quiet induction, oral intubation was performed. Thereafter an IPPR technique with nitrous oxide, oxygen (3 l. and 2 l/minute respectively) and ether was adopted, using a carbon dioxide circle absorber. Gallamine was used as the relaxant, 100 mg. being given in divided doses during the $1\frac{1}{2}$ hours of operation. Administration of ether vapour was continued throughout the major period of the procedure, a total of 4 fl.oz. being used.

The patient's B.P. - 160 mm.Hg systolic before anaesthetic - dropped progressively during induction of anaesthesia and, after 45 minutes, was 100 mm.Hg. The operating table was tilted slightly head-down. This caused no change in the level of the B.P. Administration of 10 mg. methyl amphetamine caused the B.P. to rise to 115 mm.Hg systolic. For the remainder of the operation it varied between this level and 100 mm.Hg systolic. During the operation blood loss was not severe and no blood was transfused.

At the conclusion of the operation, 2 hours after the start of anaesthesia, spontaneous respiration did not return. Following the administration of neostigmine 1 mg. preceded by Atropine 0.6 mg., gasping spontaneous respiration with a tracheal tug commenced. Intercostal muscles appeared active. The B.P. now rose to 120 mm.Hg systolic with a pulse rate of 110/minute. Consciousness was not regained but, thinking the patient was in a safe state, the anaesthetist allowed her to be returned to the ward. In the ward she is reputed to have regained consciousness 2 hours later, but manifested marked restlessness. Omnopon gr. 1/6 was administered. Two hours later she was found to be comatose and hypotensive - B.P. 80 mm.Hg systolic. Respiration still manifested a tracheal tug. On painful stimulation her jaw muscles tightened. Blood transfusion was now commenced. There was no change in her condition in the subsequent 2 hours. In the belief that Omnopon might be responsible for the unconsciousness, 10 mg.nalorphine was given intravenously. Respiratory rate and excursion improved but there was no change in the level of consciousness, It was noted now that urinary output sinee surgery was negligible.

The following day thelevel of consciousness improved slightly but this was not maintained. On the 3rd post-operative day death

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occurred, following respiratory failure. During this post-operative period a tentative diagnosis of a cerebrovascular accident had been made, but no localising signs were apparent.

AUTOPSY:

Regretably, due to circumstances not under the control of the anaesthetist, no autopsy was performed.

COMMENT:

Although she did not manifest motor-excitatory phenomena such as convulsions or athetosis, the post-anaesthetic course of this patient can be explained on the basis of irreversible cerebral damage - the result both of anoxic and ischaemic cerebral anoxia.

The untoward events which stand out in the course of the anaesthetic administered to this patient, and which would all cause just this result, are:-

(1) Hypotension following induction of anaesthesia;
(2) The persistence of curarised respiration at the conclusion of the anaesthetic, which involved the use of a relaxant drug and an IPPR technique. The dose of gallamine used (100 mg.) though not in itself excessive, must be considered in relation to the use of ether throughout the major period of anaesthesia, and in relation to the later observation that urinary excretion was negligible; in these circumstances the dose of neostigmine given (1 mg.) must be considered very modest;
(3) The additional reduction in respiratory excursion that may have been occasioned by the reduction into the abdomen of a large ventral hernia, present for a long period pre-operatively, would have worsened the state of under-ventilation;
(4) The delayed return of consciousness, followed by marked restlessness, are both recognised signs of cerebral anoxia;
(5) The administration of Omnopon at this time was followed by intractable coma, respiratory depression and circulatory collapse: the classical sequelae of opiate administration in the presence of cerebral anoxia;
(6) All these events occurred in the context of known arteriosclerosis in an aged patient.

This sequence of events gives adequate reason for the state of irreversible cerebral damage on both an anoxic and ischaemic basis. In that this sequence of events is directly related to the anaesthetic administration, this must be considered the major cause of this death - in the context of this patient's physical status and the ventilatory restriction imposed by the operation.

The major faults in the anaesthetic management which could have been corrected are:-

(a) Adequate IPPR should have continued post-operatively;
(b) A larger dose of neostigmine should have been tried;
(c) The post-operative supervision in the ward does not appear to have been adequate;
(d) The cause of restlessness following recovery of consciousness was not adequately evaluated before the exhibition of Omnopon;
(e) The administration of intravenous fluid during and post-operatively would have resulted in better urinary excretion, may have assisted intthe excretion of gallamine, and also may have helped to maintain a more stable circulatory state.

PREVENTABILITY:

In view of the correctibility of the faults enumerated, especially the failure to correct inadequate ventilation post-operatively, this death is regarded as "probably preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
21.1.1956	2	No comment	< 24	Basal atal-ectasis left lung.	Yes.

Name: Baby Keating. Age: 6 hours (neonate) Sex: M Race: C.

Disease: Exomphalos. Operation: Repair of exomphalos.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Healthy neonate.

PREMEDICATION: No premedication.

ANAESTHETIC:

Anaesthesia was induced and maintained by cyclopropane and oxygen, delivered through an infant face mask and modified T-piece circuit. Spontaneous respiration continued throughout.

Duration of operation - 45 minutes. Course uneventful. The patient recovered consciousness 5 minutes post-operatively and died suddenly 11 hours post-operatively.

AUTOPSY:

Midline abdominal surgical wound. Blood in pericardium. Partial atelectasis of lungs, especially left base. Meckel's diverticulum.

COMMENT:

This patient had an uneventful course of anaesthesia and recovered consciousness immediately post-operatively.

Atelectasis is a known complication of both anaesthesia and total surgical correction of exomphalos. There were no signs of aspirated secretions either during or after anaesthesia, from which recovery was rapid and complete. This atelectasis is thought to be related to the splinting effect on the diaphragm of the total surgical correction of the exomphalos, especially when considered in relation to the operative course of this patient. The lungs of a neonate may take many hours to expand completely following birth. The significance of the autopsy finding of blood in the pericardium is not known.

Anaesthesia per se is not regarded as contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY.
22.1.1956	2	No comment	< 24	Result of surgery: possibly herniation of the uncus.	Yes.

Name: Andrew Julims Age: 39 Sex: M Race: C.

Disease: Supratemporal haemangioma.

Operation: Craniotomy. Removal of supratemporal haemangioma. Induced hypothermia.

Anaesthetic risk: 2

PRE-OPERATIVE STATE:

The patient was in fair general condition and was conscious.

PREMEDICATION:

Pethedine 50 mg., Phenergan 50 mg., Largactil 50 mg., Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether, administered via a Magill circuit. Oral intubation was performed and the patient anaesthetised to second plane third stage with ether. The patient was immersed in an ice bath at 35°F for 20 minutes. Initial temperature of the patient was 99°F; in the bath this dropped to 90°F in 20 minutes. He was then removed from the bath and exhibited an after-drop down to 81°F. Temperature subsequently stabilised at 82°F. Anaesthesia was maintained on nitrous oxide and oxygen - flow rate 8 l. and 4 l./minute respectively. Respiration was depressed and for approximately 3-3½ hours of the operation, respiration was controlled using an IPPR technique, on Magill circuit. For long periods, oxygen alone was administered.

B.P. remained static at between 70 and 80 mm.Hg systolic throughout the operation. No hypotensive drugs were used. Marked cardiac irregularity occurred throughout the major part of the procedure and persisted until the patient was returned to the ward. In spite of this, B.P. was maintained at the level stated - 70 to 80 mm.Hg systolic - and no other treatment was adopted for this complication. Blood was transfused as lost, a total of 3 pints being given during the operation. Other than the cardiac irregularity, no untoward incidents occurred throughout the anaesthesia, which lasted 7 hours.

At the conclusion of the operation and anaesthetic, good spontaneous respiration of a normal tidal volume was re-established. The patient regained consciousness, even though the temperature at the end of operation was still 82°F. He still had cardiac irregularity with a pulse rate of 110/minute. The patient was returned to the ward, where slight shivering occurred. Largactil 50 mg. was given by intramuscular injection. The patient was conscious. He died 5 hours post-operatively from what appeared clinically to be an episode of medullary coning: loss of consciousness and central respiratory paralysis. The diagnosis of medullary coning was made by the neurosurgeon. Resuscitation was of no avail.

AUTOPSY:

Craniotomy; removal of tumour from right frontal lobe with flattening in the pituitary fossa. There was herniation of the uncus.

COMMENT / ...

COMMENT:

That this patient suffered from cardiac irregularity, probably ventricular extrasystoles, throughout the course of the anaesthetic resulted from the fact that the temperature was maintained at too low a level. However, the patient appears to have suffered no harm from this.

One may criticise the fact that the patient was returned to the ward at too low a temperature. Again, the patient appears to have suffered no harm from this.

The mode of the patient's death clinically and the autopsy findings of herniation of the uncus make this the most likely cause of death. Though the hypothermia may in some senses be regarded as suspect, one must conclude that this patient's demise was a result of surgery rather than of anaesthesia.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
23.1.1956	2	No comment	< 24	Meningo-encephalitis.	Yes.

Name; C. Visser. Age: 65 Sex: M Race: E.

Disease: ? Cerebral abscess. Operation: Carotid angiography.
Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Following an attack of otitis media, the patient had become stuperose. Marked papiloedema was present. In addition, he had pulmonary emphysema.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether, administered via a Magill circuit. The larynx was topically anaesthetised with Anethaine 1% and oral intubation was performed. Anaesthesia was maintained throughout with nitrous oxide, oxygen and ether administered through a Magill circuit. Spontaneous respiration throughout.

Duration of the investigation was 1 hour. The clinical course of anaesthesia was untoward. No cerebral abscess was demonstrated on angiography. The patient regained the same level of consciousness post-operatively as was present before, and died 23 hours after operation.

AUTOPSY:

Patchy chronic meningo-encephalitis with cerebral softening. Pulmonary emphysema. Infarct of left lung base. Hypertrophy and atheroma of pulmonary artery and hypertrophy of right ventricle. Passive congestion of liver.

COMMENT:

Anaesthesia per se appears to have played no part in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
24.1.1956	3	No comment	< 24	Cardiac failure & consolidation of lung.	Yes

Name: Doreen Johnson Age: 32 Sex: F Race: E.

Disease: Mitral stenosis. Operation: Mitral valvotomy.

Anaesthetic risk: 4

PRE-OPERATIVE STATE:

The patient was in an extremely poor pre-operative state. Peripheral circulation was poor, peripheral veins evident. She had auricular fibrillation, orthopnoea, a two finger hepatomegaly and was in severe cardiac failure. The patient was digitalised and had been treated with diuretics.

PREMEDICATION:

Pethidine 25 mg. Scopolomine gr. 1/200.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by inhalation of nitrous oxide, oxygen and ether. The larynx was sprayed with Anethaine 1% and oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether. dTC was administered and an IPPR technique used throughout via a carbon dioxide absorption circuit. Total dose of dTC - 20 mg.

Before and throughout operation the patient remained cyanosed. Following induction of anaesthesia the B.P. dropped and could not be recorded adequately, though at operation direct measurement of left ventricular pressure revealed this to be 90 mm.Hg systolic. Mitral valvotomy was performed. Following valvotomy, B.P. was recorded at 60 mm.Hg systolic. Blood loss of approximately 1 litre was replaced by transfusion. At the conclusion of the operation (total anaesthetic time 3 hours), there was respiratory depression. Atropine gr. 1/100 given, followed by two doses of neostigmine, 1 mg. each, but led to no improvement in respiration. The patient showed no other signs of persistent curarisation; she could move her arms with good power and bite strongly on the airway. Artificial ventilation with 100% oxygen led to no improvement in the cyanosis. Diaphragmatic movement was poor, especially on the left side. Bronchoscopy was performed and some mucous aspirated from the bronchi. The patient was returned to the ward and died 1 hour later.

AUTOPSY:

Bilateral pleural effusions. Pericardial effusion. Consolidation of left lung.

COMMENT:

That the induction of anaesthesia, accompanied by vasodilation, in this patient produced a marked drop in B.P. which persisted throughout the operation leads one to wonder why no vasopressor drugs were used in an effort to combat this, especially after the performance of a mitral valvotomy had failed to lead to any improvement. The possibility that there was some residual curarisation post-operatively is ruled out to some extent by the fact that the patient had good muscle power post-operatively, and no improvement followed the

administration ...

administration of neostigmine. Furthermore, the cyanosis evident post-operatively had been present pre-operatively as well as throughout the operation, and did not improve on the administration of oxygen by IPPR.

The extremely poor cardiac state, together with the autopsy finding of consolidation of the left lung and pleural effusion, tends to incriminate the patient's disease as the primary cause of death. However, one cannot entirely exculpate the anaesthetic, and it must be regarded necessarily as "contributing" to this patient's death. The pleural effusion should have been aspirated before anaesthesia. One must conjecture whether the maintenance of some form of IPPR and the performance of a tracheotomy post-operatively would have improved the patient's chances. One must question too the clinical decision to operate on this patient in the presence of consolidation of her left lung.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
25.1.1956	2	No comment	< 24	Extradural haematoma.	No.

Name: Robert Colin Wepener. Age: 21 Sex: M Race: E.

Disease: Extradural haematoma. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Extremely poor. The patient was unconscious, his pulse was irregular - rate 50-60/minute.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Topical anaesthesia of larynx with Anethaine 1%. Oral intubation was performed. Anaesthesia maintained with nitrous oxide and oxygen, delivered via Magill circuit. Spontaneous respiration throughout.

Just prior to craniotomy a trace of ether was added to the anaesthetic mixture.

The duration of the operation and anaesthesia was 3 hours. Blood was transfused as lost during operation and the patient died 12 hours post-operatively without regaining consciousness.

AUTOPSY:

No autopsy was performed.

COMMENT:

This patient died of cerebral injury. Anaesthesia was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
26.1.1956	3	No comment	< 24	Cardiac arrest (cardiac manipulation) Irreversible cerebral damage.	Yes.

Name: Daphne Lapari. Age: 39. Sex: F Race: C.

Disease: Aortic and mitral stenosis with incompetence. Operation: Mitral valvotomy.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Extremely poor. She suffered from gross rheumatic heart disease, having aortic and mitral stenosis with incompetence, cardiomegaly, auricular fibrillation, congestive cardiac failure. The patient was digitalised and had been treated with mersalyl.

PREMEDICATION:

Atropine gr. 1/100, pethidine 75 mg. 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with 150 mg. thiopentone, followed by inhalation of nitrous oxide, oxygen with gradually added ether vapour. Following topical analgesia of the larynx and trachea with Anethaine 1%, oral intubation was performed. Controlled respiration was instituted with nitrous oxide, oxygen and a trace of ether vapour, through a to-and-fro carbon dioxide absorber. dTC was used as the relaxant, a total dose of 12 mg. being used throughout.

Blood was replaced as lost during the operation. The performance of ventriculotomy for use of Tubb's dilator, produced cardiac arrest, followed by ventricular fibrillation. Cardiac massage was necessary for 20 minutes before the resumption of normal spontaneous cardiac rhythm. Following the commissurotomy, the pulse was 130/minute. B.P. 60 mm.Hg. At the end of the operation, spontaneous respiration was resumed. No antidote was necessary for the dTC. The patient failed to recover consciousness after operation and died 3 hours post-operatively.

AUTOPSY:

Gross rheumatic heart disease. Left sided organised pleurisy. Left haemothorax. Smell of ether in lung and brain. Lungs: right lung collapsed; left lung covered with old organised pleurisy, partially separated surgically; the lung was collapsed and showed bruising. Heart: enlarged, weighed 395 gm. Left ventricular hypertrophy. Mitral and aortic valves grossly thickened. Pulmonary and tricuspid valves normal. Small surgical tear in medial commissure of mitral valve. Left auricular appendage sutured and intact. Sutured surgical wound on anterior surface of left ventricle intact.

COMMENT:

This death resulted from the combined effects of the patient's cardiac lesion (which at this time - 1956 - was not completely correctable surgically), the surgical procedure and the undoubted cerebral damage which must have followed cardiac arrest due to surgical manipulation and subsequent prolonged severe hypotension. Though responsibility for the collapsed right lung found at autopsy may be implied, I do not feel that the anaesthetic played a significant part in the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
27.1.1956	3	No comment	< 24	Irreversible traumatic shock. Probable metabolic acidosis.	Yes.

Name: Charles Kapa. Age: 27 Sex: M Race: C

Disease: Stab wound of abdomen. Operation: Laparotomy. Repair of lacerated bladder and gut.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient was admitted to hospital many hours after injury, in extremely poor physical status. He was shocked, oligaemic and vasoconstricted. Gastric suction was instituted and 5 pints blood and 500 ml. of plasma were transfused before anaesthesia. Restoration of blood volume was considered adequate.

PREMEDICATION: Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide, oxygen and ether. Oral intubation was performed and anaesthesia maintained with nitrous oxide, oxygen and intermittent ether administered through a carbon dioxide absorption circuit. Spontaneous respiration which appeared adequate was permitted throughout. Following induction of anaesthesia and commencement of the operation, the B.P. which was 100 mm.Hg at the beginning of the procedure dropped to 75 mm.Hg systolic. Further administration of blood produced no response. A noradrenaline drip infusion was commenced. The B.P. remained at the level of 60 mm.Hg systolic. A consultant anaesthetist was called. The B.P. remained at this level throughout the procedure, which lasted 3 hours.

At the end of operation, the B.P. rose to 140 mm.Hg systolic. On recovery from anaesthesia, the patient vomited but pharyngeal aspiration was prompt and no inhalation ensued. The patient recovered consciousness and was restless post-operatively. For this, Omnopon gr. 1/6 was administered. However, the B.P. could not be maintained without the administration of noradrenaline, and the patient died 1½ hours post-operatively.

AUTOPSY:

Two sutured incised wounds in the lower abdomen. Wedge resection of mesentery, small intestinal resection and end-to-end anastomosis of small bowel. Sutured wound posterior surface of the bladder. Lungs: both lungs showed putrefactive changes; in the left lung there was an old caseous tuberculous focus 2 cc. indiameter, in the subapical region of the upper lobe.

COMMENT:

This death was due to irreversible or refractory oligaemic, traumatic shock, and the inevitable accompanying metabolic acidosis. The anaesthetic per se doubtless contributed "necessarily" to the precipitation of the peripheral vascular failure-like state which persisted throughout the operation and post-operatively.

The treatment of refractory shock with vasopressor drugs, though no longer accepted, was considered correct in 1956. Also in terms of present day standards, the failure to correct the metabolic acidosis is open to criticism.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
8.1.1956	2	No comment	< 24	Periton-itis.	No.

Name: David Coulson. Age: 65 Sex: M Race: C.

Disease: Post-gastrectomy intestinal obstruction with peritonitis.

Operation: Laparotomy. Resection of small gut.

Anaesthetic risk: 3 emergency

PRE-OPERATIVE STATE:

The pre-operative state of the patient was poor. A gastrectomy had been performed for carcinoma of the stomach 15 days previously. This had been followed within 24 hours by intestinal obstruction which had been treated by gastric aspiration and intravenous fluid replacement. The patient's temperature was 101.1°F. Respiration 22/minute. He had a patchy pulmonary atelectasis. B.P. 130/75 mm.Hg.

PREMEDICATION:

Morphine gr. 1/6, Atropine gr. 1/100 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced by inhalation of nitrous oxide and oxygen with gradually added ether. When the depth of anaesthesia was sufficient oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and ether, through a circle carbon dioxide absorption system. Gallamine was administered in two doses, 20 mg. and later 10 mg. An IPPR technique was used throughout. A total of 4 oz. ether was used throughout the operation.

At operation a volvulus of small bowel was found, and was already gangrenous. There was gross peritoneal soiling. This loop of small bowel was resected. During the operation the B.P. dropped to 70 mm.Hg systolic. The carbon dioxide absorber was switched off, for a period. Spontaneous respiration was allowed to recommence. The B.P. rose to 80 mm.Hg systolic. Throughout the operation, 1½ litres blood was transfused together with 600 ml. 5% dextrose in water. A noradrenaline drip infusion was commenced. The B.P. was subsequently maintained at between 110 and 130 mm.Hg systolic. At the conclusion of operation, which lasted 2½ hours, and discontinuance of anaesthetic, the patient rapidly regained consciousness. Spontaneous respiration was adequate. No antidote was necessary for the 50 mg. gallamine that had been given. Following return to the ward, the patient died 20 hours post-operatively.

AUTOPSY:

There was no autopsy.

COMMENT:

This patient died from gross peritonitis.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
29.1.1956	1	Possibly	ORD	Profound hypotension Ischaemic anoxia. Cardiac arrest.	Yes.

Name: P. Wolpowitz. Age: 69. Sex: M Race: E.

Disease: Adenoma of the prostate. Operation: Transvesical prostatectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient was in fair general condition. He suffered from essential hypertension, B.P. 190/100 mm.Hg, but otherwise his heart was clinically normal. Pulmonary system - some wheezing and emphysema; chest expansion reasonable. Other systems normal. Weight 246 lbs.

PREMEDICATION:

Morphine 1/8 gr. Atropine gr. 1/100, given 1 hour 20 minutes before operation.

ANAESTHETIC:

Prior to commencement of anaesthetic B.P. 190/100 mm.Hg. Anaesthesia induced with thiopentone 300 mg., in divided doses, followed by inhalation of nitrous oxide, oxygen with gradually added ether vapour, the patient breathing spontaneously. When anaesthesia was sufficiently deep, oral endotracheal intubation was performed. Subsequently 80 mg. gallamine was administered and an IPPR technique was adopted using a carbon dioxide absorber circuit. Anaesthesia was maintained with nitrous oxide, oxygen and ether. During induction of anaesthesia the blood pressure fell progressively. 20 minutes after injection of thiopentone, the B.P. had dropped to 80 mm.Hg systolic. Oral intubation produced a brief rise in B.P. to 120 mm.Hg systolic, but following the commencement of operation, the injection of gallamine and the institution of the IPPR technique, the B.P. again fell progressively. Within 15 minutes it had fallen to 80 mm. Hg systolic again. A 5^{0} dead-down tilt of the operating table had no effect.

Bleeding from the operation site was minimal. The intravenous infusion of a 5% solution of dextrose in water was commenced. After 20 minutes of hypotension the pulse - until then regular at 100/ minute - became irregular. Methyl amphetamine 10 mg. was injected intravenously and the administration of ether vapour was discontinued. The hypotension continued. By this time, re-suture of the operative site had been begun. Nitrous oxide was discontinued and IPPR performed with oxygen alone. Following the diagnosis of cardiac arrest, there was a delay of approximately 4 minutes before thoracotomy was performed and cardiac massage commenced. On thoracotomy, the heart was observed to be beating weakly and ineffectively. Cardiac massage was continued for 1 hour and two intraventricular injections of 1 ml. 1/1000 adrenaline, and an intraventricular injection of 10 ml. 10% $CaCl_2$, were administered. The heart beat was not restored, however.

AUTOPSY:

Gross obesity. Recent suprapubic prostatectomy and thoracotomy for cardiac massage. Hypertrophy of heart. Pleural cavities: right normal; left - moderate haemothorax. Lungs: partial collapse of both lungs. Heart and pericardium: left ventricular hypertrophy; heart weighed 600 gm. Minimal atheroma of aorta and coronaries.

Liver ...

Liver: fatty infiltration. Urinary system: prostate removed, capsule sutured, minimal bleeding.

COMMENT:

Cardiac arrest, when it ensued, must certainly have resulted from the decreased myocardial perfusion which will have followed the prolonged systemic hypotension. The minimal operative bleeding noted in the pre-operatively hypertensive patient is evidence of the extremely low cardiac output. This peripheral vascular failure-like state, with poor cardiac output, appears to be a direct result of anaesthesia, commencing with its induction.

Many factors could be responsible - thiopentone, ether anaesthesia initially deep enough to permit intubation, the type of IPPR pattern used, concomittant hyperventilation and respiratory alkalosis, and possibly, too, an element of hypovolaemia. The anaesthetic and its management in the context of the patient's essential hypertension was the major causative factor in this death.

PREVENTABILITY:

Throughout this case record one is struck by the tardiness with which resuscitative measures were adopted. The operating table was only in the slight Trendelenburg position, instead of in steep Trendelenburg position, following the initial onset of hypotension. A vasopressor drug (methyl amphetamine) was only administered after the patient had been severely hypotensive for 45 minutes. The administration of ether vapour was not discontinued early, when hypotension followed induction of anaesthesia. The observation of minimal operative bleeding in a patient who was formerly hypertensive, was not acted upon. No attempt was made to improve the venous return with expansion of the vascular compartment by blood or fluid. Once the diagnosis of cardiac arrest had been made, there was a 4 minute delay between diagnosis and the commencement of cardiac massage and cardiac resuscitative measures.

Prompter and more intensive resuscitative measures may possibly have averted this calamity. The death is thus regarded as "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
30.1.1956	1	Probably	ORD	Anoxic anoxia. Cardiac arrest.	Yes

Name: Arno Meyer. Age: 3 months. Sex: M Race: E.

Disease: Inhaled foreign body. Operation: Bronchoscopy for removal of inhaled foreign body.

Anaesthetic risk: 4,emergency.

PRE-OPERATIVE STATE:

This child had inhaled a foreign body. Pre-operative X-ray of the chest showed collapse of the upper and lower lobes of the right lung. The condition of the patient was poor. Pulse 120/minute. Peripheral vasoconstriction. Severe respiratory distress.

PREMEDICATION: No premedication was given.

ANAESTHETIC:

After an initial induction of anaesthesia with nitrous oxide, oxygen, an open drop ether technique was used, supplementary oxygen (500 ml. /minute) being added under the Schimmelbusch mask. After 10 minutes, the patient was deeply anaesthetised. Ether was discontinued and bronchoscopy commenced.

Two minutes after the start of bronchoscopy the pulse became feeble and respiration sporadic. Shortly afterwards the pulse became imperceptible but respirations continued sporadically, with long intervals between breaths, until the final cessation of respiration occurred. Two minutes after commencing bronchoscopy, the surgeon was requested to withdraw the bronchoscope, and immediately the anaesthetist applied intermittent bilateral pressure to the thoracic wall, (obviously in some attempt at artificial respiration). The surgeon passed an endotracheal tube and oxygen was administered by means of an IPPR technique. This was continued for a further 30 minutes, to no avail, at which time death was presumed.

AUTOPSY:

Upper respiratory tract passages contained a considerable quantity of clear, slightly frothy fluid, which extended into the air passages of the lungs. The stomach contents consisted of white milk curds, which bore no similarity in appearance to the contents of the respiratory passages. Both lungs showed evidence of partial collapse, especially marked in the upper and lower lobes of the right lung. No abnormal findings in the remaining organs.

COMMENT:

This death resulted from anoxic anoxia. This anoxia was the result of both the atelectasis, caused by inhalation of foreign body, and from the sequelae of anaesthesia and bronchoscopy. The omission of premedication with Atropine when ether was used as the main anaesthetic appears extremely serious in the light of the considerable quantity of frothy fluid found in the bronchi at autopsy. This must have added greatly to the existing pre-operative anoxia. No reason for this omission can be adduced.

The bronchoscopy itself added further embarassment of ventilatory function, and marked vagal stimulation, which is especially dangerous in the presence of anoxia.

When ...

When cardiac arrest was diagnosed, the resuscitative measures adopted were completely inadequate.

PREVENTABILITY:

This death is regarded as "probably preventable", in spite of the seriousness of this child's condition before the anaesthetic, because of the omission of Atropine as premedication and the completely inadequate resuscitative measures adopted once cardiac arrest had ensued.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
31.1.1956	2	No comment	< 24	Fulminating staphylo-coccal enteritis.	Yes

Name: James Warren. Age: 63 Sex: M Race: E.

Disease: ?Mesenteric thrombosis. Operation: No operation performed.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund. Pulse 100/minute, feeble. Respiration - tachypnoea, 46 /minute, extremely shallow. B.P. 90 mm.Hg systolic. Circulatory failure with cyanosis and difficulty in breathing because of extreme abdominal distension and splinting of diaphragm. ?Pleural effusion. The patient had had a transvesical prostatectomy 5 days previously. In the immediate post-operative phase 7 pints blood had been transfused. Gastric lavage had been performed. Two further pints blood given in theatre just before induction of anaesthesia. The diagnosis at that stage was mesenteric thrombosis, for which laparotomy was deemed necessary.

PREMEDICATION:

Atropine gr. 1/100, 1 hour before operation.

ANAESTHETIC:

On arrival in theatre the patient was moribund. However, on institution of noradrenaline drip infusion, the B.P. rose to 120 mm.Hg systolic and it was decided to proceed with anaesthesia.

Initially the patient received pure oxygen for 5 minutes, then anaesthesia was induced with oxygen and Cyclopropane. The B.P. dropped immediately. The patient was intubated following the administration of 40 mg. gallamine and IPPR was instituted with oxygen only. After some minutes the B.P. became recordable again and anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether vapour, administered via a carbon dioxide absorption circuit using an IPPR technique. The B.P. began falling again and ether was discontinued. Cyclopropane and oxygen were again administered. Marked peripheral failure was now evident. Gross cyanosis of the face, ears and fingers was present. Oxygen alone, by IPPR, failed to alter the cyanosis. Circulation time appeared grossly prolonged. In view of the gross peripheral circulatory failure, no operation was attempted. The patient was allowed to recover consciousness and was returned to the ward in much the same moribund condition as he had been in on arrival in theatre.

On return to the ward the patient's condition remained static for approximately 3 hours. Rapid deterioration thereupon followed, with death.

AUTOPSY:

Severe membraneous enteritis (fulminating staphylococcal enteritis). Marked dilatation of the small and large intestine. Indications of prostatic operation. Local haemorrhage among lesser curve of the stomach. Left ventricular hypertrophy. Subpleural and pericardial haemorrhages. Left and right sided pleural effusion. Severe aortic atherosclerosis. Pulmonary oedema.

COMMENT ...

COMMENT:

This moribund patient, who was diagnosed as suffering from a severe enteritis, but was in fact suffering from fulminating staphylococcal enteritis, recovered consciousness following the administration of this fruitless anaesthetic. On return to the ward, his condition was similar to that before anaesthesia. He died of the pre-existing disease, fulminating staphylococcal enteritis, toxaemia and endotoxic shock.

Anaesthesia is not regarded as significantly contributing to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
32.1.1956	2	No comment	< 24	Aspirational complications of tracheo-oesophageal fistula.	No

Name: Baby Beg Age: 2 days. Sex: M Race: C.

Disease: Tracheo-oesophageal fistula.

Operation: Thoracotomy. Repair of congenital tracheo-oesophageal fistula.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor. Patient cyanosed on admission, from inhalational complications of the tracheo-oesophageal fistula. Pulse rate 160/minute. Patient also had imperforate anus. Pre-operatively a lipiodol swallow had been performed and an intravenous infusion of 5% dextrose in water had been commenced.

PREMEDICATION:

ANAESTHETIC:

Anaesthesia was induced with Cyclopropane, oxygen and endotracheal intubation was performed. An IPPR technique was used. Succinylcholine, 4 doses of 5 mg., was administered during the operation, anaesthesia being maintained with nitrous oxide and oxygen, with a trace of ether vapour. Blood was replaced as lost.

At the conclusion of the anaesthetic, the child recovered consciousness. Pulmonary aspirational complications, from the tracheo-oesophageal fistula, present before operation, would appear to have been the cause of the child's death 12 hours after operation.

COMMENT:

This child appears to have died from pulmonary aspirational complications of a congenital tracheo-oesophageal fistula. Anaesthesia is regarded as non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
33.1.1956	2	No comment	< 24	Intra-cerebral haematoma	No

Name: Aletta Gous Age: 63 Sex: F Race: E.

Disease: Intracerebral haematoma.

Operation: Right carotid angiography. Burrhole craniotomy. Evacuation of intra-cerebral haematoma.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Semi-conscious. Respiration 22/minute. B.P. 110/60 mm.Hg. Pulse rate 88/minute.

PREMEDICATION:

Atropine gr. 1/100, 35 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and ether administered via a Magill semi-open circuit, the patient breathing spontaneously. The larynx was sprayed with Anethaine 1% and oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen, with minimal ether.

Right carotid angiography was performed, followed by right parietal burrholes and needle evacuation of an intracerebral haematoma. Anaesthesia was uneventful. Following the operation the patient was noted to have returned to the pre-operative level of consciousness within 5 minutes. She died 12 hours post-operatively apparently from the pre-existing disease.

AUTOPSY:

There was no autopsy.

COMMENT:

Death appears to have been due to the existing intracerebral haematoma. Anaesthesia is regarded as non-contributory to the fatal outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
34.1.1956	2	No comment	< 24	Cerebral abscess.	No.

Name: Leo Nero. Age: 19. Sex: M Race: E.

Disease: Mastoiditis and intracerebral abscess.

Operation: Mastoidectomy. Burrhole craniotomy to relieve mastoiditis with intracerebral abscess and transverse sinus thrombosis.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor - the patient was stuperose. He had had two previous operations; a mastoidectomy followed later by a burrhole craniotomy. This operation was for re-exploration of the mastoidectomy site, together with another burrhole for exploration for an intracerebral abscess. B.P. at commencement of operation was 120 mm.Hg systolic.

PREMEDICATION:

Atropine gr. 1/100, 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by inhalation of nitrous oxide, oxygen and ether, administered via a semi-open Magill circuit, with spontaneous respiration. Following induction of anaesthesia the larynx was sprayed with Anethaine 1% and oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen, with a trace of ether via the Magill circuit. The patient breathed spontaneously throughout.

The course of anaesthesia was uneventful. Following the cessation of anaesthesia, the patient was noted to return to the same level of consciousness as had existed pre-operatively. He died 22 hours post-operatively.

AUTOPSY:

No autopsy was performed.

COMMENT:

Death appears to have been due to the existing disease. Anaesthesia is not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
35.1.1956 .	3	No comment	ORD	Ventricular fibrilla-tion. Cardiac arrest, from manipulation	Yes.

Name: Henrietta J. Bonnington. Age: 53. Sex: F. Race: E.

Disease: Aortic stenosis. Operation: Percutaneous left ventricular puncture. Aortic valvotomy.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient suffered from aortic stenosis with incompetence. She was bedridden and in extremely poor condition. Orthopnoea, cardiac failure. She suffered from angina pectoris. B.P. 140/65 mm.Hg. Air entry was poor with poor pulmonary excursion. She had been digitalised. Medication included amnophyllin and mercurial diuretics.

PREMEDICATION:

Morphine gr. 1/6, Atropine gr. 1/100, 1½ hours before operation.

ANAESTHETIC:

The surgeon infiltrated procaine over the 5th left interspace prior to performing percutaneous ventricular puncture under local anaesthesia. Percutaneous puncture of the left ventricle was performed, followed immediately by the onset of ventricular fibrillation. IPPR with oxygen was instituted immediately, while the surgeon performed thoracotomy and commenced cardiac massage. The patient was then intubated orally and a carbon dioxide absorber circuit was incorporated in the IPPR technique.

Ventricular fibrillation persisted despite intracardiac injection of procaine amide, and direct electrical defibrillation of the heart. The surgeon decided to perform aortic valvotomy and requested than an anaesthetic be administered. A 50% mixture of nitrous oxide and oxygen, with a trace of ether vapour, was administered fora few minutes during intracardiac manipulations. Artificial respiration and cardiac massage were then resumed for a further 15 minutes, when electocardiographic monitoring indicated complete cessation of cardiac activity.

AUTOPSY:

Evidence of old rheumatic aortic and mitral valvulitis. Findings suggest mitral incompetence rather than aortic stenosis or incompetence. No mitral stenosis, the valve orifice admitting 3 fingers. No mural thrombi.

COMMENT:

This death resulted from ventricular fibrillation precipitated by the surgical manoeuvres.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
36.1.1956	2	No comment	< 24	Undetermined.	No.

Name: James Cook. Age: 68. Sex: M Race: C.

Disease: Post-operative volvulus. Operation: Laparotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Pulse rate 100/minute. Respiration 25/minute. B.P. 80 mm.Hg systolic. The patient had cor pulmonale. He had severe abdominal distension which reduced his respiratory excursion. Gross electrolyte disturbance had been corrected pre-operatively. Digoxin 0.5 mg. was administered pre-operatively. This patient had had an abdomino-perineal resection of rectum 9 days previously, and was now suffering from gross intestinal obstruction.

PREMEDICATION:

Atropine gr. 1/100, 30 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen. Oral intubation was performed. Cyclopropane and oxygen with an occasional trace of ether were administered by an IPPR technique, throughout the operation.

During the operation, the entire small gut was found to be included in a volvulus. Following decompression and unravelling of bowel, the B.P. fell from 80 mm.Hg to 30 mm.Hg systolic on three occasions, being restored to 80 mm.Hg by means of methyl amphetamine 6 mg. administered by intravenous injection each time. Following operation, a noradrenaline intravenous drip infusion was instituted. The patient regained consciousness within 5 minutes. He was apparently well for 7 hours post-operatively and died suddenly.

AUTOPSY:

No autopsy was done.

COMMENT:

Death was due to the existing disease in this patient. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
37.1.57	3	No comment	ORD	Massive aortic thrombosis. Cardiac failure Cardiac arrest	Yes

Name: Maud Bailey. Age: 66 Sex: F Race: C.

Disease: Aortic embolus-thrombosis. Operation: Transfemoral ilio-femoral thrombectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

In extremis. The patient presented with signs of complete aortic block, ?embolus ?thrombosis, in cardiac failure, with albuminuria 3+, liver 2 fingers enlarged. Permission for operation initially refused. Bilateral paravertebral block at L.2 and 3 performed with xylocaine 1.4%, using a total of 20 cc. The patient was given Digoxin 0.5 mg. and aminophyllin 250 mg. Six hours later a continuous caudal epidural was instituted with 0.5% procaine. Consent for operation was given 9 hours after admission.

PREMEDICATION: Atropine 0.6 mg.

ANAESTHETIC:

The patient presented in theatre with a B.P. of 80 mm.Hg systolic and a pulse rate of 110/minute. She was in extremis. Anaesthesia was induced with cyclopropane and oxygen administered via a CO_2 absorption circuit, with spontaneous respiration. After 12 minutes, cyclopropane was discontinued and anaesthesia continued with nitrous oxide and oxygen alone, with intermittent cyclopropane.

The femoral artery was incised and a thrombus removed from the femoral and iliac artery by means of catheter aspiration. After 15 minutes of anaesthesia the B.P. fell from 80 to 60 mm.Hg. At this stage methyl amphetamine 6 mg. was injected intravenously with no effect. Nitrous oxide was discontinued and oxygen alone administered. However, the B.P. continued to fall rapidly and cardiac arre st ensued after operation had been in progress for 25 minutes. No cardiac resuscitation was attempted because of the extent of this patient's disease.

AUTOPSY:

The aorta was blocked with thrombus and embolus from the level of T.12 down the entire length, including the internal and external iliac arteries. There was a saccular dilatation of the aorta just above the bifurcation. Gross atheroma of the aorta and coronary arteries. Heart was enlarged. No mural thrombus found. Liver and cardiac cirrhosis.

COMMENT:

Though anaesthesia must inevitably have contributed "necessarily" to this patient's death, the existing disease was of so gross a nature as to be considered the major cause of her death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
38.1.57	2	No comment.	< 24	Subdural haematoma and cerebral damage.	No.

Name: Isaac Hendricks. Age: 48. Sex: M Race: C.

Disease: Head injury. Operation: Carotid angiography. Burrhole craniotomy and evacuation of subdural haematoma.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Three days previously, a subdural haematoma had been evacuated through a burrhole craniotomy. Following this operation, initial improvement was not maintained. A left hemiplegia had developed, level of consciousness was markedly depressed and carotid angiography showed marked displacement of vessels.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 350 mg. in divided doses. The larynx was sprayed with Amethocaine and anaesthesia continued by inhalation of nitrous oxide, oxygen and ether. Following oral intubation, anaesthesia was maintained with nitrous oxide, oxygen and trichlorethylene, administered via a Magill circuit. Spontaneous respiration was maintained throughout.

An intravenous infusion of 5% dextrose in water was given throughout the operative procedure. The course of operation and anaesthetic was uneventful. At the conclusion of operation the patient returned to the state of consciousness in which he had been pre-operatively. He died 10 hours after operation.

AUTOPSY:

No autopsy was performed.

COMMENT:

It must be speculated why it seemed necessary to use thiopentone to induce anaesthesia in this patient. However, it would appear that anaesthesia played no part in the patient's demise which was due entirely to pre-existing cerebral damage.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
39.1.57	1	Possibly	< 24	Hypovolaemia. Hypotension. Cardiac arrest.	Yes.

Name: Nicolas Manuel. Age: 67 Sex: M Race: C.

Disease: Carcinoma of stomach. Operation: Gastrectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This patient had gastric carcinoma. He was in poor nutritional state and was cachectic. Weight 100 lb. Temperature 99°F. B.P. 110/70 mm.Hg. Respiratory system - emphysema. One pint blood had been transfused pre-operatively. No report on serum electrolytes.

PREMEDICATION:

Morphia gr.1/6, Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 15 mg. followed by inhalation of nitrous oxide, oxygen and ether. Larynx was sprayed with topical anaesthetic and oral intubation performed. Anaesthesia was continued with nitrous oxide, oxygen and a trace of ether, administered via a CO_2 absorbtion circuit. Gallamine was administered for relaxation, an initial dose of 60 mg. followed in the next 1¼ hours with two doses of 40 mg. each. Respiration was assisted. At no time was there complete apnoea.

After 2 hours' anaesthesia, the B.P. which had up to that stage been maintained at 105 mm.Hg systolic, pulse rate 120/minute, commenced to fall. Blood transfusion was then commenced. After a further ½ hour the B.P. had fallen to 80 mm.Hg, by when 1½ pints blood had been transfused. Cardiac arrest then occurred. Cardiac massage was instituted promptly, via the diaphragm, and the heart beat was restored within 2 minutes. The operation was complete at this stage and the patient's abdomen was resutured. Now, owing to haemorrhage through the abdominal drains, the abdomen was re-opened, the haemorrhage controlled and the abdomen was resutured. The total time of operation at this stage was 3 hours. The patient left theatre with a B.P. of 85 mm.Hg systolic, pulse rate 110/minute. Respiration was considered adequate; no atropine or neostigmine was given. The patient regained consciousness shortly after operation. He developed peripheral vascular failure 12 hours post-operatively and died.

AUTOPSY:

Recent surgery to the abdomen. Evidence of recent surgery to stomach with gastroduodenostomy suture lines all intact. No evidence of leakage of haemorrhage. Several hard glands along the lesser curvature of the stomach. Pancreas and duodenum fixed to vertebral column with large friable mass measuring 12 x 8 x 6 cm. Also enlarged glands and similar friable mass in para-oesophageal and paratracheal situations. No definitive cause of death demonstrated.

COMMENT:

Though the final cause of death is hard to determine, it is reasonable to relate it, in the absence of any other demonstrable features at autopsy, to the cardiac arrest during surgery. This appears to have been due to the anaesthetic technique.

Though ...

However, the patient's poor nutritional condition, probable pre-anaesthetic hypovolaemia and emphysema must be considered to be partly responsible for his demise.

The patient was not apnoeic in the presence of assisted ventilation following the administration of a relaxant (140 mg. gallamine in the first 75 minutes of operation in this cachectic patient must be considered to be a very adequate dose), indicating that the ventilation provided was not adequate - respiratory drive, probably from a high $PaCO_2$ persisted. In this context one must bear in mind the patient's emphysema, though this was not gross.

The operation was a palliative gastrectomy for carcinoma of the stomach, an operation usually associated with a fair degree of blood loss. In spite of this, no blood was replaced until the B.P. commenced falling after 2 hours of surgery. That the B.P. was recorded at 105 mm.Hg with a pulse rate of 120/minute during this time may well have been due to hypercarbia, masking the drop that may have ensued following blood loss. Blood transfusion was only commenced when the B.P. had fallen to 80 mm.Hg. This belated replacement of blood loss, in the probable presence of inadequate ventilation and hypercarbia, must be considered the cause of the cardiac arrest in this poor-risk patient.

Subsequent to the successful management of the cardiac arrest, the patient had to be re-opened for control of further haemorrhage. Blood transfusion was still not adequate. The patient was returned to the ward with a B.P. of 80 mm.Hg, which cannot be considered adequate.

Why was transfusion inadequate or, if adequate and the B.P. persisted at this low level, why was other supportive therapy (e.g. vasopressors) not resorted to? The anaesthetic management is considered to have been the major contributory factor to this patient's death, the poor state of the patient pre-operatively being a further contributory factor.

PREVENTABILITY:

One is unsure of the precise cause of death post-operatively. In that I am assuming that it is related to the cardiac arrest which occurred during operation, and that the anaesthetic technique is regarded as the major causative factor in this event, I regard this death as "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
40.1.57	2	No comment	< 24	Cardiac failure.	No

Name: Sophia Albrecht. Age: 53. Sex: F Race: E.

Disease: Intestinal obstruction. Strangulated umbilical hernia.

Operation: Laparotomy. Relief of obstruction. Excision and repair of umbilical hernia.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor condition. Pulse rate 140/minute. B.P. 150 mm.Hg systolic. Patient normally hypertensive, B.P. formerly 240/130 mm.Hg. Patient digitalised but still in cardiac failure. On admission, dehydrated and acidotic. Pre-operatively, 3 litres dextrose/saline and 250 cc. plasma were administered.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen with gradually added ether, following pre-oxygenation, via a semi-closed Magill circuit, the patient breathing spontaneously. After 3 minutes, gallamine 40 mg. was administered intravenously. An IPPR technique was instituted and endotracheal intubation performed. Using a semi-closed system with a total flow rate of 10 l/minute, 50% nitrous oxide, 50% oxygen, with minimal ether, an IPPR technique was used. Following the commencement of operation, a further 20 mg. gallamine was given.

The operation took 35 minutes. The course of the anaesthetic was uneventful. A total of 1½ oz. ether was administered. At the conclusion of the operation, Atropine gr.1/100 followed by neostigmine 0.5 mg. was given. Adequate spontaneous respiration was resumed. At conclusion of the anaesthetic, the patient rapidly regained consciousness in the theatre. B.P. was 120 mm.Hg systolic with a pulse rate of 120/minute. The patient was returned to the ward conscious and breathing adequately. She died in cardiac failure 23 hours post-operatively.

AUTOPSY:

No autopsy was performed.

COMMENT:

This patient's death resulted from pre-existing disease, hypertensive cardiac failure. Anaesthesia is not contributory. The use of an IPPR technique with a Magill semi-closed system is open to criticism even with a flow rate of 10 l./minute, but the amount of respiratory acidosis that would have followed in an operation of so short a duration is negligible.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
41.1.57	2	No comment.	< 24	Undetermined.	No

Name: Henry Hyde. Age: 87. Sex: M Race: C.

Disease: Post- sigmoid colectomy intestinal obstruction. Operation: Laparotomy. Freeing of adhesions.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor. Two weeks previously the patient had had sigmoid colectomy for carcinoma of the sigmoid. Three days previously intestinal obstruction had been relieved by caecostomy. This diversion had now become obstructed, necessitating the present laparotomy. The patient's condition was extremely poor. Pulse rate 100/minute, B.P. 160/100 mm.Hg. Continuous gastric suction with fluid and electrolyte replacement.

PREMEDICATION: Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxiee, oxygen with ether gradually added, administered via a Magill system, the patient breathing spontaneously. This was preceded by a period of 3 minutes' pre-oxygenation. Several minutes after the induction of anaesthesia, oral intubation with 10 cuffed endotracheal tube, was performed. Anaesthesia was continued with nitrous oxide, oxygen and ether, via the Magill circuit, the patient breathing spontaneously.

This was continued throughout the operation, which lasted 123 minutes. The course of anaesthetic and operation was relatively uneventful. At the conclusion of the operation the systolic B.P. was 150 mm.Hg, pulse rate 80/minute. The patient rapidly regained consciousness and respiration was adequate. The patient died 10 minutes short of 24 hours post-operatively.

AUTOPSY:

No autopsy was performed.

COMMENT:

Anaesthesia appears to have played no part in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
42.1.57	3	No comment	< 24	Cardiac failure Pulmonary oedema.	Yes

Name: Louisa Smith Age: 57 Sex: F Race: E.

Disease: Intestinal obstruction due to irreducible femoral hernia.

Operation: Laparotomy. Relief of obstructed femoral hernia. Repair of femoral hernia.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was in extremis. Together with intestinal obstruction, she suffered from a gross alcoholic cirrhosis of the liver with gross nutritional depletion, nutritional oedema and dehydration. She was anaemic - Hb 9 gm.% and had delirium tremens. Serum albumen 2 gm.%, serum globulin 2.5 gm.%. Pulse rate 120/minute and B.P. 60/30 mm.Hg on admission. No signs of congestive cardiac failure. Rehydration with 2½ litres 5% dextrose in water plus 20 cc. Pledex and 1 pint blood failed to restore her B.P. Wyamine sulphate 10 mg. intramuscularly 3 hourly was necessary to maintain the B.P. at approximately 100/60 mm.Hg. She weighed 160 lb.

PREMEDICATION: Nil.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide, oxygen and ether. Oral intubation was performed with a cuffed 9 endotracheal tube. Anaesthesia was maintained with nitrous oxide and oxygen, the patient breathing spontaneously. The patient was then positioned on her side and a lumbar epidural block was performed at L.3-4, using 25 cc. Xylocaine 1.2% with 1/100,000 adrenaline. This was preceded by methyl amphetamine 30 mg. given intramuscularly. In spite of this, with the onset of epidural anaesthesia, hypotension ensued. Divided doses of methyl amphetamine were given intravenously together with 1 pint blood. The patient was completely unresponsive to this treatment. Within the next ½hour, though 180 mg. methyl amphetamine was given intravenously, the B.P. fell from the pre-operative level of 100 to 60 mm.Hg systolic. At this stage a nor-adrenaline drip infusion was instituted and the B.P. was maintained at 100-110 mm.Hg.

Relaxation for operation was adequate and the laparotomy and relief of the obstructed femoral hernia was successfully performed. Duration of operation was 1¾ hours. Post-operatively the patient did not completely regain consciousness but recovered fairly rapidly to the stage of reacting to stimuli, and moving spontaneously. The noradrenaline drip had to be continued after operation and following her return to the ward, a further 1 pint blood and 2 litres dextrose in water with noradrenaline were given in increasing dosage. This was necessary to maintain the B.P.

Four hours post-operatively the patient began frothing at the mouth and diffuse moist sounds were auscultated in the chest. This was diagnosed as pulmonary oedema. The patient was given 500 mg. aminophyllin intravenously slowly, and morphia gr. 1/8 intramuscularly No improvement ensued and she died 4 hours after operation.

AUTOPSY ...

AUTOPSY:

Dependant oedema. Increased fluid in the pericardium and pleura. Congestion of upper respiratory tract mucous membranes. Small brown flabby heart with large patches of thickened epicardium, and small subendocardial haemorrhages in the left ventricle. Large fatty cirrhotic liver adherent to the diaphragm. Grossly dilated large bowel, dilatation ending abruptly in the sigmoid region opposite congested appendix. Shrunken grannular kidney. Flabby kidneys with marked diminution of superficial cortex. Submucous fibroid of uterus.

COMMENT:

From the description of this patient's pre-operative state it would appear that death was inevitable. Her condition was deteriorating rapidly before operation, and for this patient any form of anaesthesia would be hazardous, One may wonder at the choice of an epidural anaesthetic with its accompanying sympathectomy in the presence of an already hypotensive patient, who required a vasopressor to maintain the B.P. However, the anaesthetist would appear to have had good reasonfor this choice.

Because of the delirium tremens, some form of general anaesthesia was necessary. Because of the general nutritional state and the gross alcoholic cirrhosis of the liver, the anaesthetist considered the use of relaxants too hazardous. Because of the oedema of the abdominal wall, an abdominal block was considered too dangerous and likely to fail. Therefore the anaesthetist chose to achieve abdominal relaxation with the use of an epidural block, despite the previous hypotension, hoping to be able to maintain the B.P. by the use of a vasopressor. This was, to some extent, successfully achieved for a while, but the patient was so unreactive to vasopressors that massive doses had to be given. This would appear ultimately to have been cause of the final onset of pulmonary oedema. The failure to administer digitalis in these circumstances is open to criticism.

The recovery of consciousness post-operatively was difficult to assess, in view of the pre-operative delirium tremens. In view of the profound hypotension, cerebral ischaemia may well have occurred. Because of the patient's pre-operative general condition, this death is classified as 'inevitable', with anaesthesia necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
43.1.57	2	No comment	< 24	Undetermined.	Yes

Name: Betty Davis. Age: 3 months Sex: F Race: C.

Disease: Intestinal obstruction, shown at operation to be malrotation of midgut with constricting bands. Operation: Laparotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Pulse rate 200/minute. Temperature 97°F. Liver enlarged. No jaundice. Darrow's solution administered pre-operatively.

PREMEDICATION:

Atropine gr. 1/200.

ANAESTHETIC:

Anaesthesia was induced and maintained with cyclopropane and oxygen, administered via a semi-closed system, the patient breathing spontaneously.

The course of operation and anaesthetic was uneventful. The duration of operation and anaesthetic was 1½ hours. The child recovered consciousness immediately post-operatively and was breathing adequately. The patient died 4 hours after operation.

AUTOPSY:

No immediate cause of death could be determined. There was questionable pyloric stenosis.

COMMENT:

This death was due to causes unassociated with the anaesthetic.

CASE NO.	CLASSIFICATION GROUP	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
44.1.57	2	No comment	< 24	Irreversible traumatic shock. Pulmonary oedema.	Yes

Name: A.Rennie Age: 65 Sex: M Race: C.

Disease: Multiple injury. Bilateral traumatic amputation of both legs.

Operation: Debridement and bilateral amputation of legs.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

In extremis. The patient had been run over by a train. Admitted 3 hours pre-operatively in severe shock. 7 pints blood had failed to restore the B.P. Immediately pre-operatively the patient's B.P. was 50 mm.Hg systolic. The patient also had a small depressed linear fracture of the frontal bone.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide, oxygen with minimal ether for 10 minutes only, via a Magill semi-closed system, the patient breathing spontaneously. At the commencement of anaesthesia, methyl amphetamine 30 mg. was administered intravenously with no response. Thereafter an infusion of noradrenaline 1/250,000 was commenced, which succeeded in maintaining the B.P. at approximately 100 mm.Hg. To do this, increasing amounts of noradrenaline had to be administered.

During the course of operation, which lasted 2 hours 5 minutes, a further 2 pints blood were administered. The patient regained consciousness immediately on discontinuance of the anaesthetic. He was returned to the ward where he died 4 hours after operation, with what appeared clinically to be pulmonary oedema.

AUTOPSY:

Bilateral above knee amputations. Wound on right forehead with linear depressed fracture of left frontal region with contusion of underlying left frontal lobe of brain and surrounding subarachnoid haemorrhage. Collapse of both lungs. Numerous subendocardial haemorrhages in the posterior wall of the left ventricle and a fair amount of coronary atherosclerosis.

COMMENT:

This patient appears to have died from irreversible traumatic shock resulting from multiple injuries. On the whole, anaesthesia appears to have been non-contributory.

Pulmonary oedema is a common clinical end result of the treatment of "irreversible" traumatic shock with increasing amounts of vasopressor drugs, especially in the absence of the use of some means to increase left ventricular output, e.g. digitalis - or the more modern use of isoprenaline. The collapsed lungs found at autopsy were doubtless related to the existence of pulmonary oedema before death. The inhalation of vomitus or oral secretions was observed during the course of anaesthesia.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
45.1.57	2	No comment.	< 24	Undetermined.	Yes.

Name: Gava Joseph. Age: 28. Sex: F Race: C.

Disease: Septic incomplete abortion. Operation: Evacuation of uterus.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was in severe oligaemic shock, with a B.P. of 65/30 mm. Hg. Haemoglobin - 8 gm.%. Three pints of blood were transfused fairly rapidly and the patient's B.P. rose to 110/70 mm.Hg pre-operatively.

PREMEDICATION:

Morphia gr. 1/6, Atropine 1/100 gr., 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with 100 mg. thiopentone, continued with nitrous oxide and oxygen via a Magill semi-open system, the patient breathing spontaneously.

During evacuation of the uterus, a further 100 mg. thiopentone was administered. At the conclusion of operation, which lasted 10 minutes, the B.P. was 110 mm.Hg systolic and the patient rapidly regained consciousness, though was confused for a period post-operatively. In the next 10 hours, a further 2 pints blood was transfused. Ten hours post-operatively the patient collapsed manifesting a severe drop in B.P. A noradrenaline, 4 microgm.% drip infusion was instituted and rapidly raised the B.P. from 80 to 130 mm.Hg systolic. However, 30 minutes after commencing this infusion the patient had a cardiac arrest. Cardiac massage and artificial respiration were ineffective.

AUTOPSY:

Blood in the peritoneal cavity. Haematoma on vault of uterus but no perforation demonstrated.

COMMENT:

This death was due to causes unassociated with the anaesthetic, probably from gram negative septicaemia.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
46.1.57	1	Possibly	ORD	Cardiac arrest.	Yes

Name: W.Adonis. Age: 46 Sex: M Race: C.

Disease: Amoebic abscess right lobe of liver. Amoebic hepatitis. Right bronchopneumonia, lower lobe.

Operation: No operation. Intended operation: drainage of amoebic abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

In extremely poor state pre-operatively. Temperature 102°F. Pulse rate 120/minute. B.P. 100 mm.Hg Marked venous dilatation with profuse sweating and tachycardia. The patient had right lower lobe pneumonia with tachycardia. Pre-operatively continuous gastric suction had been instituted with administration of 1 l.normal saline and ½ litre 5% dextrose in water. The patient had been treated with emetine.

PREMEDICATION: Atropine gr. 1/100.

ANAESTHETIC:

After pre-oxygenation, anaesthesia was induced with inhalation of nitrous oxide and oxygen, with gradually added ether, administered via a Magill semi-closed system, the patient breathing spontaneously. Following induction of anaesthesia, the patient was positioned in the left lateral position and the anaesthetist assisted the patient's inspirationary efforts, though the patient was still permitted to breath spontaneously.

Surgery was commenced 30 minutes after the commencement of anaesthetic At this stage the anaesthetist diagnosed a cardiac arrest, by his inability to palpate the peripheral pulse. The patient was immediately turned supine and cardiac massage was commenced through a left thoracotomy. After 3 minutes of cardiac massage the heart recommenced beating, but thereafter it again stopped. Cardiac massage was continued and adrenaline 1/100,000, 1 cc., was injected intraventricularly. Thereafter the heart continued intermittently, stopping and being restarted with cardiac massage. Intraventricular injections of calcium chloride 10% to a total of 30 cc., and a further 5 cc. of 1/100,000 adrenaline, together with cardiac massage, failed to establish a sustained heart beat. Throughout this time IPPR with oxygen was continued. After 2 hours of cardiac massage there was no further tone in the heart. Resuscitative measures were abandoned.

AUTOPSY:

Right basal bronchopneumonia. Right lobe of liver completely replaced by amoebic abscess. Amoebic hepatitis.

COMMENT:

Although it can be accepted that this patient's death was in large measure due to his pre-existing disease and severe toxaemia, death was precipitated by the administration of an anaesthetic. In association with this existing disease, anaesthesia is regarded as the major cause contributory to the patient's death.

The ...

The precise cause of the cardiac arrest is not immediately apparent. Clinical monitoring and the recording thereof is so poor - the anaesthetist contenting himself with occasional digital recording of the pulse rather than accurate recording of the blood pressure and pulse rate - that it is also difficult to elucidate in retrospect. It is not known if the cardiac arrest was in fact so sudden, or if it was preceded by a period of hypotension or a progressive fall in blood pressure. The level of blood pressure was low before anaesthesia, and vasodilatation was evident, and the patient had received Emetine. The superimposition on this of the vasodilatation of ether anaesthesia may well have caused a further fall in systemic pressure, and so a decrease in myocardial perfusion in a heart already struggling under a burden of toxic myocarditis and the effects of Emetine, and so caused cardiac arrest.

In addition, the right basal pneumonia and subsequent positioning of the patient in the left lateral position must have caused some diminution and possibly inadequacy of pulmonary ventilation, and thus some degree of anoxic anoxia. Endotracheal intubation and the adoption of an IPPR technique, rather than the occasional assistance of respiration described, would have improved this aspect.

PREVENTABILITY:

Though it is difficult to diagnose the cause of this cardiac arrest, more meticulous clinical monitoring may have given warning of the impending disaster. The adoption of an endotracheal IPPR technique may have decreased the liability to anoxic anoxia. On these grounds, although the patient's existing disease is also a major factor, from the anaesthetic point of view this death is regarded as "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
47.1.57	1	Probably	> 24	Anoxic anoxia (Respiratory obstruction) Cerebral damage.	Yes

Name: Lena Bantom. Age: 50 Sex: F Race: C.

Disease: Uterine fibroids. Operation: Hysterectomy.

Anaesthetic risk: 1

PRE-OPERATIVE STATE:

Good. Nothing significant in the findings other than uterine fibroids.

PREMEDICATION:

Pethidine 100 mg., Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 300 mg. followed by inhalation of nitrous oxide, oxygen and ether, administered via a semi-open system, the patient breathing spontaneously. After topical analgesia of the larynx, oral intubation was performed and, at the commencement of the operation, gallamine 40 mg. was given. This produced respiratory paralysis and IPPR was instituted via a circle absorption system.

Following commencement of operation no thoracic expansion was observed on compression of the reservoir bag. The patient rapidly became cyanosed and pulseless. The oxygen bypass was turned on and more positive pressure was exerted without avail. The ether and nitrous oxide were turned off. The endotracheal tube was inspected and thought to be correctly inserted in the trachea. A senior anaesthetist was summoned from the adjacent theatre and confirmed that the position of the endotracheal tube in the trachea was correct, but suspecting it kinked, he removed and re-inserted it. Pulmonary ventilation could now be performed easily, with no further obstruction (it would appear that there had been a kink in the endotracheal tube, probably in the oropharynx). Cyanosis immediately disappeared, the pulse became palpable again.

The operation was commenced and completed, nitrous oxide and oxygen being administered via a circle absorption system by IPPR. Towards the end of operation ether was again administered, as the patient was moving. The subsequent course of anaesthesia during operation was uneventful. At the conclusion of the operation, which lasted 1 hour, the patient's respiration returned spontaneously and was adequate. No neostigmine was administered.

Following conclusion of the operation the patient did not regain consciousness and remained flaccid. Fluid therapy 2 l. daily was continued for two days, when it became obvious that the patient was suffering from cerebral oedema, and this was then stopped. Hypertonic 20% sucrose and magnesium sulphate were then administered to the patient, in an effort to cause dehydration. Throughout the post-operative period she was given continuous oxygen therapy. On the 4th post-operative day the patient became hyperpyrexial, her temperature reaching 105°F. This continued until the evening of the 6th post-operative day, when the patient died.

AUTOPSY ...

AUTOPSY:

Brain congested. No softening or haemorrhage. Trachea bruised. Lungs clear. Liver and kidneys congested. Cause of death undetermined - probably cerebral anoxia.

COMMENT:

This death resulted from the sequelae of profound cerebral anoxia, caused by an obstruction to the endotracheal tube - probably a kink at the level of the oropharynx that was not recognised and corrected quickly enough. The anaesthetist was inexperienced. This fault was corrected in time to prevent cardiac arrest.

A further aspect of this case open to grave criticism is the failure to anticipate the possibility of cerebral oedema, when the patient did not regain consciousness. Dehydration therapy and hypothermia should have been instituted at this stage, and not after a delay of two days.

PREVENTABILITY:

Failure to recognise the kinking of the endotracheal tube in this case was due to the inexperience of the anaesthetist. This mishap and consequent death of the patient are definitely preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
48.1.57	3	No comment	< 24	Profound hypotension. Circulatory failure.	Yes

Name: John William Cook. Age: 63 Sex: M Race: E.

Disease: Abdominal aortic aneurysm. Diffuse arteriosclerosis.

Operation: Resection and graft of abdominal aneurysm.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Large abdominal aortic aneurysm. Severe generalised arteriosclerosis. Pulmonary emphysema.

PREMEDICATION:

Omnopon gr. 1/3. Scopolomine gr. 1/150.

ANAESTHETIC:

Anaesthesia was induced with a thiopentone (300 mg.), succinylcholine (40 mg.) oxygenation, topical analgesia of the larynx, oral intubation sequence. Following return of spontaneous respiration, anaesthesia was continued with nitrous oxide, oxygen and trace of ether vapour administered via a carbon dioxide circle absorption by an IPPR technique. dTC was the relaxant used - initial dose 20 mg.

Surgery was commenced 40 minutes after induction of anaesthesia. The patient's general state and the B.P. were well maintained for the first hour of anaesthetic. With the commencement of aortic dissection approximately 20 minutes after the start of operation, a severe fall in B.P. occurred. This in all probability was precipitated by intermittent vena caval obstruction, necessitated by the fact that the inferior vena cava was tightly adherent to the abdominal aneurysm. The fall in B.P. to 60 mm.Hg systolic failed to respond to a blood transfusion, or later to clamping of the abdominal aorta. Infusion of noradrenaline gave temporary improvement, the B.P. rising to a level of 90-100 mm.Hg systolic. This was maintained by continuance of the noradrenaline infusion. The abdominal aneurysm was resected and grafted, blood being adequately replaced as lost. The operation lasted 6 hours. Spontaneous respiration commenced at conclusion of operation. Residual curarisation was reversed with neostigmine 2 mg. preceded by Atropine 1.2 mg. After the conclusion of operation the patient recovered consciousness to the extent of responding to stimuli, but did not fully regain consciousness. He died in a state of peripheral vascular failure, 6 hours after operation.

AUTOPSY:

Widespread arteriosclerosis with gross narrowing of the coronary ostia. Atrophic pulmonary emphysema. Gastric ulcer - previous gastroenterostomy. Granular kidneys.

COMMENT:

This patient died from circulatory failure due to gross arteriosclerosis. This virtually irreversible state was initially precipitated by a prolonged period of hypotension following the commencement of aortic dissection with intermittent vena caval

compression ...

compression and obstruction. The vena caval obstruction was unavoidable in that the inferior vena cava was closely adherent to the abdominal aortic aneurysm.

The state, which did not respond to blood transfusion and aortic clamping, responded to some extent to the infusion of noradrenaline.

The failure of the patient to fully regain consciousness must be assumed to have been due to cerebral ischaemia resulting from a period of severe hypotension in a patient with generalised arteriosclerosis. The anaesthesia may be considered necessarily contributory to some extent to the intractability of the hypotension, when it occurred. As this was predominantly a factor of the patient's severe generalised vascular disease, and the result of dissection of the aneurysm (with intermittent vena caval compression) no alteration in the anaesthetic technique may be considered likely to have changed the ultimate outcome.

No purpose is served in classifying this death as due to the anaesthetic. Anaesthesia may be considered necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
49.1.57	2	No comment	< 24	Subdural haematoma	Yes

Name: Adam Roberts. Age: 50 Sex: M Race: E.

Disease: Subdural haematoma. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

His condition was poor, the patient having suffered a head injury. He was comatose. Respiration was irregular with intermittent obstruction.

PREMEDICATION:

No premedication given.

ANAESTHETIC:

Following pharyngeal suction toilette, anaesthesia was induced with inhalation of nitrous oxide, oxygen and ether. Following topical analgesia of the larynx, oral intubation was performed. During angiography, anaesthesia was maintained with nitrous oxide and oxygen, the patient breathing spontaneously, using a Magill circuit. Intermittent doses of thiopentone sodium, totalling 300 mg., were given.

During the burrhole craniotomy the patient's condition was adequately maintained with a B.P. of 110 mm.Hg systolic and pulse rate varying between 80 and 90/minute. When spontaneous, respiration was irregular. Hyperventilation, by an IPPR technique, using a Magill circuit with nitrous oxide and oxygen (total flow 10 l./minute) was instituted. This was resorted to in an effort to reduce the markedly increased brain tension. At the end of operation, which lasted 4 hours, a tracheotomy was performed. The patient was returned to the ward, his level of consciousness being approximately what it had been before the start of anaesthesia. Three hours post-operatively the patient's respiration began to fail. IPPR was instituted, using a Pneuofore pump. The patient died 15 hours after operation.

AUTOPSY:

Contusion of left upper arm and right shoulder and base of neck. Lacerated wound over right parietal eminence. Fractured right clavicle. Surgical incisions on both sides of scalp overlying surgical openings in the skull. Fracture of right side of skull. Right subdural haematoma and mid-brain petechie. Tracheotomy wound.

COMMENT:

This death was due to cerebral trauma. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
50.1.57	2	No comment	< 24	Peritonitis.	Yes.

Name: Sophia Easton. Age: 22. Sex: F Race: C.

Disease: Intestinal obstruction. Volvulus. Pelvic abscess.

Operation: Laparotomy. Resection of volvulus. Ilio-ilial anastomoses. Drainage of tubo-ovarian abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor pre-operative state, having been on gastric aspiration and intravenous infusion for 2 weeks. Blood transfusion had been given. The patient was jaundiced. B.P. 110 mm.Hg systolic with tachycardia of 120/minute. Respiratory system - rhonchi audible all over lungs with basal crepitations. The patient was producing much sputum. Acetone was present in the urine.

PREMEDICATION :

Morphine gr. 1/6, Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and ether. Oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and minimal ether, via a circle absorption system. Gallamine, initial dose 40 mg. followed after 1½ hours operation by a further 20 mg., was used for relaxation. Initially respiration was assisted but during the latter 1½ hours of operation an IPPR technique was used.

During the first 1½ hours of operation the B.P. was maintained at a level of between 105-110 mm.Hg with a pulse rate varying from 132 to 156/minute. No blood was transfused initially, as there was little obvious blood loss. After 1½ hours surgery, the B.P. dropped to irrecordable levels and the pulse rate became almost uncountable. A pint of blood was rapidly transfused and a drip infusion of noradrenaline was commenced. This produced a rise in B.P. to the previous level. During the subsequent 1½ hours surgery a further 3 pints blood were given, totalling 4 pints transfused.

A tubo-ovarian abscess was revealed by laparotomy. The ilium was adherent to the abscess and had formed a volvulus. The gut involved was gangrenous. There was gross peritonitis. The gangrenous ilium was resected, the pelvic abscess drained and an ilio-ilial anastomosis performed. Spontaneous respiration resumed and residual curarisation was reversed with neostigmine 1 mg. preceded by Atropine 0.6 mg.

Following conclusion of the operation the patient rapidly regained consciousness and was returned to the ward with a B.P. of 125 mm.Hg systolic. She died 12 hours post-operatively

AUTOPSY:

Surgical incision in lower abdomen. Left tubo-ovarian abscess with adhesions to lower ilium. Ilio-ilial anastomosis had been performed because of volvulus of ilium, gangrene of the gut. Peritonitis was present, also jaundice.

COMMENT ...

COMMENT:

Although there was an episode of severe hypotension, which would appear to have resulted from inadequate blood replacement during the operation, this had been treated and controlled by adequate subsequent blood replacement. Death appears to have resulted from diffuse peritonitis. Anaesthesia in general is considered non-contributory tothis death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
51.1.57	2	No comment	< 24	Intracranial haemorrhage. Medullary coning.	Yes

Name: Phineas Sikundla. Age: 24. Sex: M Race: B.

Disease: Intracranial haemorrhage. Head injury. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was in a very poor pre-operative state: comatose, with a bradycardia of 56/minute and retention of bronchial secretions.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and ether by a semi-open method. Oral intubation was performed after topical analgesia of the larynx with Xylocaine 4%. Anaesthesia was continued with nitrous oxide, oxygen and ether, administered by a Magill circuit with the patient breathing spontaneously.

The course of anaesthesia throughout operation was uneventful. Carotid angiography was performed followed by occipital burrhole craniotomy and ventricular puncture. The operation lasted 2¼ hours. The patient died 5½ hours post-operatively without regaining consciousness.

AUTOPSY:

Surgical burrholes 1 cm. in diameter of both parietal occipital lobes. Brain congested and distended, gyria flattened. Marked medullary coning. No haemorrhages on surface except for two localised subarachnoid haemorrhages, approximately 2 cm. in diameter, each situated under the burrholes and extending into both lobes, with softening. The haemorrhage on the right was 4½ cm. in diameter and 5 cm. in diameter on the left. Small right-sided intracerebral haemorrhage in right internal capsule. Kidney, renal pelves and ureter congested.

COMMENT:

This death was due to cerebral oedema with medullary coning. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
52.1.57	1	Possibly	ORD	Hypotens-ion. Cardiac arrest.	Yes

Name: George Clelland. Age: 67 Sex: M Race: E.

Disease: Carcinoma of the prostate. Operation: Orchidectomy (proposed) No operation performed.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Apparently in a fair state but the patient suffered from arterio-sclerosis. He had an enlarged heart with a B.P. of 126/80 mm.Hg. He had complained on the previous night twice of praecordial pain. This pain was not radiating and was said to be similar as he had experienced with indigestion the previous year. The praecordial pain was not further investigated.

PREMEDICATION :

Pethidine 75 mg. Scopolomine 0.3 mg.

ANAESTHETIC:

A skin weal was raised over the sacral hiatus and a caudal puncture performed. Using Xylocaine 0.9% with 1/200,000 adrenaline, a test dose of 5 cc. was injected following aspiration tests, rotating the needle through 90°. Five minutes after injection there was no change in the patient's B.P. or leg muscular power. A further 25 ml. was then injected, repeat aspiration tests during the procedure revealing no blood or C.S.F. The caudal needle was removed and the patient was turned onto his back. He said he was feeling alright. A pillow was placed under his head.

At this stage, suddenly, the pulse became impalpable. Attempts to record the B.P. failed. The patient lost consciousness, ceased to respond to spoken enquiries, ceased breathing and became rapidly cyanosed. Oxygen was administered by IPPR and 50 mg. methyl amphetamine was immediately given intravenously. There was no response. The Trendelenburg position was adopted, a thoracotomy performed and cardiac massage commenced within approximately 4-5 minutes, of initial collapse. The heart was found to be in complete asystole. During cardiac massage, oral intubation was performed and IPPR with oxygen was continued by this means. Cardiac massage plus intraventricular injection of Adrenaline and subsequently of noradrenaline were of no avail. All resuscitative attempts were abandoned after 1 hour.

AUTOPSY:

Evidence of hypertensive heart disease. Nothing else significant. A specimen of C.S.F. taken post-mortem gave a questionable reaction in testing for Xylocaine.

COMMENT:

From the clinical point of view this patient appears to have died from acute myocardial ischaemia, which seems to have been precipitated by the administration of caudal epidural block. Acute hypotension was in all probability the mechanism responsible, and this may have resulted from:-

(1) rapid absorption or intravenous administration of the Xylocaine - tests revealed no venepuncture and the dose was not excessive; further, the Xylocaine was administered with 1/200,000 adrenaline; the patient manifested no signs of intravenous injection while the Xylocaine was being administered.

2 ...

(2) Over-dosage leading to excessively high level of sympathetic block - the total dose injected does not seem to have been excessive.

(3) Intrathecal injection, leading to a massive spinal anaesthesia - clinical testing revealed no thecal puncture and the test dose showed no effect. However, insspite of this, and the fact that subsequent chemical analysis of the C.S.F. revealed a questionable positive on test for Xylocaine, this appears to be the most likely explanation for the catastrophe.

The delay of up to 5 minutes in finally instituting cardiac massage must have all but guaranteed the fatal outcome. In this context one must bear in mind the patient's complaint of praecordial pain the previous evening, with a history of some former praecordial pain, together with the finding of cardiomegaly - but with a B.P. of 126/80 mm.Hg - all of which may well have indicated some episodes of myocardial ischaemia in the pre-operative period.

Ideally, an E.C.G. should have been performed before anaesthesia was administered.

This conclusion is further strengthened by the autopsy evidence of hypertensive heart disease, though no actual coronary occlusion was demonstrated. On balance, excluding the possibility of a coincidental coronary occlusion, one must accept that the administration of caudal epidural anaesthesia was the precipitating cause of the patient's death, on a background of pre-anaesthetic hypertensive heart disease with the possibility of myocardial ischaemia.

PREVENTABILITY:

In that the patient did complain of praecordial pain, had cardiomegaly with a suspiciously normal blood pressure, and no electrocardiogram was performed, and in that there was a delay of 5 minutes before the institution of cardiac massage, this death is considered "possibly preventable", although perhaps a harsh judgment.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
53.1.57	2	No comment	< 24	Peritonitis.	No.

Name: Alice Scholtz. Age: 51. Sex: F Race: C.

Disease: Peritonitis. Operation: Laparotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Temperature 101.4°F, pulse rate 150/minute. Respiratory system - extreme tachypnoea 44/minute. B.P. 240/160 mm.Hg. The patient had cardiomegaly but was not in cardiac failure. Bilateral basal crepitations. Trace of acetone in urine. Pre-operatively she had gastric aspiration and fluid and electrolyte replacement.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 50 mg. followed by inhalation of nitrous oxide, oxygen and ether, the patient breathing spontaneously. Following topical analgesia of the larynx with oral intubation, anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether vapour, administered by an IPPR technique via a carbon dioxide circle absorption system. Gallamine was used as the relaxant, a total dose of 120 mg. being used throughout the operation.

The patient's colour improved and the cyanosis disappeared. The further course of anaesthesia was untoward, and the operation lasted 105 minutes. During the procedure 200 cc. blood was transfused. At the conclusion of operation the patient's respiration was normal and adequate. Neostigmine 1 mg. preceded by Atropine 0.6 mg. was administered. Consciousness was rapidly regained following discontinuance of the anaesthetic. The patient was returned to the ward and died 9 hours post-operatively.

AUTOPSY:

No autopsy was performed.

COMMENT:

This death appears to have resulted from the patient's pre-existing disease (severe peritonitis) and anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
54.1.57	1	Probably.	< 24	Inadequate post-op. supervision. Respiratory obstruction. Anoxic anoxia.	Yes

Name: Sarah Lakay Age: 38 Sex: F Race: C.

Disease: Bronchiectasis. Operation: Bronchography.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Bilateral basal bronchiectasis following a previous middle lobe lobectomy for bronchiectasis in 1954.

PREMEDICATION:

Aminophyllin suppository 500 mg., Pethidine 50 mg., Atropine gr.1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 400 mg. followed by gallamine 60 mg., IPPR with oxygen and oral intubation. Anaesthesia was then maintained with nitrous oxide, oxygen and trichlorethylene administered by an IPPR technique via a Magill system.

Bronchography lasted 45 minutes. At the conclusion of this procedure, the bronchi were aspirated, residual curarisation was reversed by the administration of neostigmine 1 mg. preceded by Atropine 0.6 mg. The patient recovered consciousness in the X-ray department, responding to stimuli and was returned to the ward. 1½ hours later she was observed collapsed, cyanosed with no respiratory or cardiac action. Artificial respiration and cardiac massage were to no avail. No-one was aware how long the patient had been dead.

AUTOPSY:

Diffuse basal bronchiectasis.

COMMENT:

As this death was unobserved, the precise mechanism of death is unsure. It is apparent from this that post-operative supervision was very inadequate indeed. Salient points in the procedure she underwent were:

(1) General anaesthetic involving the use of a relaxant (gallamine) and an IPPR technique,
(2) Lipiodol was instilled into the bronchi of both lungs,
(3) The patient suffered from diffuse basal bronchiectasis.

Death probably resulted from anoxic anoxia. This must have arisen in one or, or a combination of, the following ways:-

1. Through curarisation, though apparently adequately reversed as judged by the volume of respiration and its pattern, the ability to cough may still be impaired. In the absence of a strong and effective cough, the patient's bronchi may have become rapidly occluded - both from her own profuse bronchial secretions and the lipiodol instilled. Such progressive bronchial occlusion will have led to progressive, ultimately fatal, anoxic anoxia.

2 ...

2. Simple upper respiratory tract obstruction from a falling back of the tongue may have occurred.

Of these two possibilities, the first is the more likely. In either event, anaesthesia and curarisation must be regarded as a most significant contributory factor in this patient's death.

PREVENTABILITY:

This fatality could almost certainly have been avoided by adequate post-operative supervision. As such, this death is regarded as "probably preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
55.1.57	2	No comment	< 24	Meningitis	Yes

Name: Daphne Minnaar. Age: 18 days Sex: F Race: C

Disease: Cephalhaematoma, Left side of head. Cerebral injury.

Operation: Burrhole craniotomy. Evacuation of cerebral abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Patient stuperose. Temperature 99°F, pulse rate 148/minute. Respiratory system - respirations 36/minute. Large cephalhaematoma on left side of the head.

PREMEDICATION:

No premedication.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered by a modified T-piece. Oral intubation was performed and anaesthesia was maintained with nitrous oxide oxygen and ether, administered via the T-piece, the patient breathing spontaneously.

A burrhole craniotomy was performed with evacuation of a cerebral abscess. The course of anaesthetic was untoward. Following the conclusion of anaesthetic the patient's level of consciousness returned to approximately what it had been before operation. The patient was returned to the ward and died 10 hours post-operatively.

AUTOPSY:

Recently evacuated cephalhaematoma. Extensive meningitis (K.pneumoniae). Haematoma of anterior abdominal wall.

COMMENT:

This death was due to the patient's pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
56.1.57	2	No comment	< 24	Cerebral injury. Medullary coning.	Yes

Name: Stephen Mayer. Age: 17 Sex: M Race: C.

Disease: Head injury. Stab wound of head, intracerebral haematoma. Depressed fracture of skull.

Operation: Elevation of depressed fracture of skull.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. The patient was semiconscious. Respiration 18/minute. Pulse rate 64/minute. B.P. 130/80 mm.Hg. Crepitations in the left lung field. Responded only to painful stimuli. Early papilloedema, pupils were dilated and fixed. Bilateral extensor plantar responses.

PREMEDICATION:

Atropine gr. 1/100, 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered via a Magill semi-closed system, the patient breathing spontaneously. Following topical analgesia of the larynx with 4% Xylocaine, oral intubation was performed. Anaesthesia was continued with nitrous oxide and oxygen with minimal ether.

The course of anaesthesia and operation was untoward. At the conclusion of the operation and discontinuance of anaesthesia the patient's level of consciousness returned to what it had been pre-operatively. He was returned to the ward and died 9 hours post-operatively.

AUTOPSY:

Knife wound penetrating anterior wall of middle cranial fossa as well as orbital tissues. The knife had also entered the anterior pole of the frontal lobe. Subdural haematoma present over the pons of the left occipital region. Coning and brain stem petechie were noted.

COMMENT:

This death was due to the patient's existing cerebral trauma and medullary coning. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
57.1.57	2	No comment.	< 24	Peritonitis	No.

Name: Mrs. E. Griffin. Age: 78 Sex: F Race: E.

Disease: Perforated pyloro-duodenal ulcer. Operation: Laparotomy. Suture of perforated ulcer.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient's pre-operative state was extremely poor. The peptic ulcer had perforated 28 hours previously. Respiration 28/minute. B.P. 70/55 mm.Hg. There was oedema of both ankles. No hepatomegaly. Bilateral basal atelectasis. Albuminuria and glycosuria but no ketonuria.

PREMEDICATION:

Soluble insulin, 30 units, intramuscularly 1 hour pre-operatively. Glucose 30 gm. An intravenous infusion of 1 litre 5% dextrose/normal saline was commenced. Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia induced with nitrous oxide, oxygen and ether administered via a semi-closed system. Oral intubation was performed and anaesthesia continued with nitrous oxide, oxygen and ether via semi-closed method with carbon dioxide absorption. Spontaneous respiration was permitted throughout. No relaxant was required or used.

At the commencement of anaesthesia the B.P. was 70/50 mm.Hg This gradually dropped to 60 mm.Hg. The pulse rate during operation varied between 116 and 140/minute. Respiratory rate was steady at 40/minute. Methyl amphetamine 9 mg. was given 15 minutes after the commencement of operation, intravenously, and this elevated the B.P. to 80 mm.Hg systolic. This was maintained throughout the operation. A total of 2½ oz. ether was used during the operation. At the conclusion of the procedure, which lasted 55 minutes, the patient regained consciousness promptly. Spontaneous respiration was adequate. A noradrenaline drip infusion was commenced before the patient was returned to the ward, where she died 12 hours post-operatively.

AUTOPSY:

There was no autopsy.

COMMENT:

From the nature of this patient's lesion, her age and the duration of the gastric perforation, the prognosis was very poor from the outset. Pre-operative fluid replacement does not appear to have been adequate. This is something over which the anaesthetist may not have had much control, this aspect of treatment falling into the province of the surgeon. However, to commence anaesthesia in a patient of this age with so low a level of blood pressure does seem a little misguided. However, as appears to be the case, the operation was undertaken in a measure of desperation from the surgical aspect, whereupon the anaesthetist must accept informed surgical opinion.

The anaesthetist refrained from using relaxant drugs for fear of the danger of the development of a post-relaxant apnoea - a not uncommon sequel in these clinical circumstances. Ether was used instead, the amount being moderate but nonetheless this appears to have further depressed the level of systolic blood pressure.

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It is possible that better results may have followed the use of blood transfusion, or some plasma expander, rather than vaso-pressor drugs, to combat this hypotension. The use of an IPPR technique may also have offered some advantages.

However, having regained consciousness promptly after anaesthesia, it is probable that this patient would have recovered from the major systemic effects of the anaesthetic well before she died 12 hours post-operatively. This patient died from the effects of severe peritonitis. Anaesthesia as such is not considered a significantly contributory factor.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
58.1.57	2	No comment.	< 24	Cerebral abscess.	No.

Name: Alice Poggenpoel. Age: 43. Sex: F. Race: C.

Disease: Cerebral abscess and mastoiditis.

Operation: Mastoidectomy. Burrhole craniotomy with drainage of cerebral abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient was extremely toxic. There was a history of previous cardiac failure. At this time though there were crepitations audible bilaterally, over the lung bases, there were no other signs of congestive cardiac failure. She was conscious. Pre-operatively the B.P. was 135/80 mm.Hg, pulse rate 94/minute, temperature 96.5°F.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 250 mg. followed by inhalation of nitrous oxide, oxygen and ether, administered via a Magill circuit, the patient breathing spontaneously. Following topical anaesthesia of the larynx, oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and ether, via a Magill circuit.

A mastoidectomy was performed followed by burrhole craniotomy and aspiration of a cerebral abscess. The course of anaesthesia throughout the operation was untoward, the B.P. remaining at 120 mm.Hg systolic with a pulse rate fluctuating between 100 and 120/minute. At the end of theprocedure, which lasted 2½ hours, the patient recovered consciousness rapidly on the discontinuance of anaesthetic, and was returned to the ward. She developed respiratory failure followed by cardiac arrest 4 hours after operation, and died.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of her pre-existing disease. Anaesthesia is not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
59.1.57	2	No comment	< 24	Cerebral thrombosis	No

Name: Magdalena Saayman. Age: 30 Sex: F Race: E.

Disease: cerebral thrombosis. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was in a poor pre-operative state - extremely confused, apathetic and somewhat dehydrated. No intravenous fluid therapy had been administered at the surgeon's request, for fear of raising the already raised intracranial pressure. The pulse rate was 160/minute and the B.P. 140/80 mm.Hg.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 125 mg. followed by nitrous oxide, oxygen and ether, administered by means of a semi-closed McGill system. The larynx was sprayed with 4% Xylocaine and oral intubation was performed. Anaesthesia was continued with nitrous oxide, oxygen and a trace of ether, via a McGill system, the patient breathing spontaneously.

Throughout the operation, which lasted 4 hours, the course of anaesthesia was untoward. At the end of operation and the discontinuance of anaesthetic, the patient recovered consciousness to the same level as had existed pre-operatively. She died 15 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This death resulted from the patient's pre-existing disease, anaesthesia not contributing.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
60.1.57	2	No comment	< 24	Bronchial carcinoma.	No.

Name: Emily Falotti. Age: 60. Sex: F Race: C.

Disease: Carcinoma of the oesophagus. Operation: Oesophagoscopy. Insertion of Soutar's tube.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Extremely poor. Emaciated and nearly in extremis. Pulse rate 120/minute. B.P. 130/60 mm.Hg. Effort tolerance nil. No overt cardiac decompensation. Respiratory system - complete consolidation of left lung. Vital capacity grossly reduced. Gross enlargement of mediastinal and deep cervical glands.

PREMEDICATION:

Dehydration was corrected and plasma transfused.
Omnopon gr. 1/6, Atropine gr. 1/100, 1½ hours before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether vapour. Following topical analgesia of the larynx, oral intubation was performed with a No. 8 Portex tube. Anaesthesia was continued with nitrous oxide and oxygen.

The course of anaesthesia was uneventful. The B.P. and pulse rate remained stable throughout. The operation of oesophagoscopy and insertion of Soutar's tube lasted 35 minutes. Following discontinuance of anaesthetic, the patient regained consciousness and was fully conscious and orientated on return to the ward. She died 23 hours after operation.

AUTOPSY:

No autopsy.

COMMENT:

This death was due to pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
61.1.57	2	No comment	< 24	Refractory irreversible oligaemic shock.	No

Name: Jacobus Oosthuizen. Age: 67 Sex: M Race: E.

Disease: Post-prostatectomy haemorrhage.

Operation: Laparotomy. Securing of bleeding points in prostatic bed.

Anaesthetic risk: 3/ emergency.

PRE-OPERATIVE STATE:

Extremely poor state. The patient was in severe oligaemic shock after haemorrhage from the prostatic bed following prostatectomy 8 hours previously. Impalpable pulse. Unrecordable B.P. Respiration 30/minute.

PREMEDICATION:

Immediately before operation 4 pints of blood were transfused. No other premedication.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane, nitrous oxide, oxygen and was subsequently maintained with nitrous oxide, oxygen and ether, administered via a circle absorption system, the patient breathing spontaneously. No relaxant drugs were used.

There was no response of the B.P. to the rapid pre-operative transfusion of blood but, following laparotomy and the securing of bleeding points in the prostatic bed, post-operative transfusion of blood produced a return of the B.P. to 100 mm.Hg systolic. The patient regained consciousness on discontinuance of the anaesthetic, which lasted 1 hour. The patient died 5 hours after operation.

AUTOPSY:

No autopsy.

COMMENTS:

This patient died of severe oligaemic shock due to massive post-operative haemorrhage. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
62.1.57	2	No comment	< 24	Broncho-pneumonia.	Yes

Name: Shiela Mayer Age: 7 weeks. Sex: F Race: C.

Disease; Aortic vascular ring with tracheal compression. Operation: Thoracotomy. Division of aortic ring.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was suffering from severe respiratory distress. Respiration 70/minute. Pulse rate 140/minute.

PREMEDICATION:

Atropine gr. 1/4oo.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen and was maintained after oral intubation with nitrous oxide, oxygen and ether, administered via a modified T-piece system. Succinylcholine was given and an IPPR technique instituted.

The course of anaesthesia was relatively untoward. The operation lasted 180 minutes. The patient regained consciousness after discontinuance of the anaesthetic but died 16 hours post-operatively.

AUTOPSY:

Bilateral severe bronchopneumonia with fibrinous exudates. Aortic ring compressing trachea. Diffuse fatty infiltration of the liver with lymphoid hyperplasia of the spleen.

COMMENT:

This death was due to bronchopneumonia aggravated by the effects of thoracotomy. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
63.1.57	1	No comment	< 24	Ventricular tachycardia Cardiac failure.	No

Name: Petrus Odendaal. Age: 72. Sex: M Race: E.

Disease: Carcinoma of rectum. Operation: Anterior resection of the rectum.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair. Debilitated, rather grossly arteriosclerotic patient. Not in frank cardiac failure but there were bilateral basal crepitations at the lung bases. B.P. 130/90 mm.Hg. There were fairly frequent extrasystoles.

PREMEDICATION:

Morphia gr. 1/6, Atropine gr. 1/100, 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by nitrous oxide and oxygen, with gradually added ether, the patient breathing spontaneously. Following topical analgesia of the larynx with 4% Xylocaine, oral intubation was performed. Anaesthesia was continued with nitrous oxide and oxygen with a trace of ether, administered via a circle absorption system. An initial dose of gallamine 40 mg. was followed later during operation by a further 20 mg. IPPR technique was instituted.

Induction of anaesthesia produced no change in the patient's condition. At the commencement of operation the Trendelenburg position was adopted. This caused an immediate drop in B.P. to 80 mm.Hg However this rose during the next 30 minutes to 120 mm.Hg. ½ hour later marked venous congestion developed, with a poor peripheral circulation as evidenced by the slow active capillary refill time. The B.P. dropped to unrecordable levels. Two doses of methyl amphetamine 6 mg. had no effect. The operating table was immediately levelled and digoxin 0.5 mg. was given intravenously. The B.P. rapidly recovered to a level of 80 mm.Hg and later to 110 mm.Hg, but at the same time the pulse rate suddenly accelerated from 120 to 180/minute. However, the patient's general condition was well maintained. As the operation had reached the point of no return, it was continued and no further treatment was adopted. 1 pint blood was transfused during operation, adequately replacing blood loss.

At the conclusion of operation, which lasted 2 hours, the patient rapidly recovered consciousness following discontinuance of the anaesthetic. Respiration was of normal volume and no reversal of curarisation was necessary. At this stage an ECG revealed the extreme tachycardia to be due to ventricular fibrillation. Repeated doses of Procainamide, to a total of 2,500 mg., failed to reverse this arrhythmia, only achieving some slowing of the pulse rate down to 140/minute for some time. Ventricular tachycardia persisted and the patient died in circulatory failure 12 hours after operation.

AUTOPSY:
No autopsy was performed.

COMMENT ...

COMMENT:

The cause of this patient's death was the onset of an intractable ventricular tachycardia, leading to death from cardiac failure. This appears to have been precipitated by the intravenous administration of 0.5 mg. digoxin during the operation. In that the patient displayed all the signs of congestive cardiac failure during the operation, treatment with digoxin must be considered justified. However, as it is known that rapid digitalisation of patients displaying extrasystoles may precipitate a ventricular tachycardia, digoxin should have been administered more slowly and in smaller doses.

Resort should perhaps have been had to some other vasopressor such as phenylephrine or noradrenaline in an attempt to raise this patient's blood pressure.

In that the onset of ventricular tachycardia was precipitated by the administration of digoxin by the anaesthetist during thecourse of the anaesthetic, the anaesthetic management is regarded as a significant contributory factor in this patient's ultimate death.

PREVENTABILITY:

It is known that intravenous digoxin therapy may precipitate ventricular tachycardia, as occurred in this case, especially when ventricular arrhythmias are present beforehand. In the clinical circumstances pertaining here, digitalisation appears to have been indicated. The dose of digoxin injected - approximately one third of the digitalising dose - was not excessive. No verdict is made as regards the aspect of preventability in this case.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
64.1.57	3	No comment	ORD	Cardiac arrest. Massive haemorrhage and air embolism.	Yes.

Name: Maria Plaatjies Age: 11 Sex: F Race: C

Disease: Fallot's tetralogy. Operation: Correction of tetralogy on cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

This patient was in poor pre-operative state, having a congenital tetralogy of Fallot. She was cyanosed. Her B.P. was 100/60 mm.Hg. Two years previously a pulmonary valvotomy had been performed. The patient was digitalised.

PREMEDICATION:

Pethidine 20 mg., Scopolamine 0.2 mg.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by cyclopropane and oxygen. Following the administration of succinylcholine 25 mg., the patient was ventilated with oxygen and oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen, with small doses of dTC, a total of 15 mg. being given during the operation. IPPR was instituted through a carbon dioxide circle absorber system.

The course of anaesthesia was relatively untoward. Surgical management was complicated by a severe haemorrhage because of the adhesions present from the previous operation. The heart-lung bypass was established but a severe haemorrhage occurred. The pump ran dry and the patient suffered a massive catastrophic air embolism. All this happened within 10 minutes of establishing cardiac bypass.

AUTOPSY:

Recent operation on thorax and heart. Longstanding enlargement of heart. Both lungs congested. Heart and pericardium: pericardial adhesions, especially at apex. Incision in right ventricle. Hypertrophy of right ventricular wall. Aorta and pulmonary artery over-riding a still patent intraventricular septum.

COMMENT:

This death resulted from technical difficulties experienced by the surgical team. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
65.1.57	2	No comment	< 24	Multiple lung abscesses. Bronchial pneumonia.	Yes.

Name: John Links. Age: 22 Sex: M Race: E.

Disease: Strangulated inguinal hernia. Operation: Right inguinal herniorrophy with reduction of strangulated hernia.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient was in a poor state. Suffering from a broncho-pneumonia, he had developed a strangulated inguinal hernia.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen and gradual addition of ether vapour, administered through a circle carbon dioxide absorption system, with spontaneous respiration. Following induction of anaesthesia, topical analgesia of the larynx and oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and ether, administered through a circle absorption system, the patient breathing spontaneously.

The anaesthetic and operation were untoward. The patient regained consciousness immediately after discontinuance of anaesthetic at the end of the procedure, which lasted 45 minutes. He was returned to the ward. There was a subsequent sudden deterioration in his condition, 20 hours post-operatively, and he died - apparently from rupture of one of multiple pulmonary abscesses and broncho-pneumonia.

AUTOPSY:

The significant findings were multiple lung abscesses and bronchopneumonia.

COMMENT:

This patient appears to have died of his pre-existing broncho-pneumonia and multiple lung abseesses. One may ask why some local anaesthetic technique was not used, in view of this patient's pre-existing bronchopneumonia with lung abscess. However, from the clinical account of the anaesthetic management, no untoward incidents occurred and anaesthesia was non-contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
66.1.57	2	No comment	< 24	Effects of intestinal obstruction.	No.

Name: Elizabeth Tubbs. Age: 70 Sex: F Race: E.

Disease: High intestinal obstruction. Volvulus of small bowel due to adhesions.

Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was moribund. Temperature 102°F. Pulse rate 120/minute. B.P. 100/50 mm.Hg. Gross peripheral circulatory failure. Gross dehydration. Bilateral hypostatic pneumonia. Anuria. Blood urea 118 mg.%. Serum potassium 3.7 mg.%. Serum sodium 134 m.Eq./l. Serum chloride 88 m.Eq./l. CO_2 combining power 39 vol.%.

PREMEDICATION:

Rehydration was attempted with 4 litres dextrose water and 3 litres dextrose in saline; 1 litre of plasma and 1 pint of blood were also transfused. No premedication was given.

ANAESTHETIC:

Anaesthesia, after pre-oxygenation, was induced with cyclopropane and oxygen administered via a closed circle absorption system. When anaesthesia was adequate, oral intubation was performed and anaesthesia was maintained with cyclopropane 10-20%, oxygen and nitrous oxide. Normal spontaneous respiration was continued throughout. No relaxants were given.

Before the operation the patient was stuperose. At the conclusion of the procedure, which lasted 1 hour, and on discontinuance of the anaesthetic the state of consciousness returned to the level at which it had been before the anaesthetic. On return to the ward, the patient was digitalised and hydrocortisone 100 mg. was given. However, she continued to deteriorate and died 14 hours after the operation.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of her pre-existing disease. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
67.1.57	2	No comment	< 24	Cerebral injury.	No

Name: Allan Cohen. Age: 6 Sex: M. Race: E.

Disease: Head injury. Cerebral laceration. Fractured skull.

Operation: Elevation of depressed fracture of skull.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was in a poor pre-operative state. He was comatose following a head injury with compound depressed fracture of the skull. The patient also had a fractured tibia and fibula.

PREMEDICATION:

No premedication.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen (50%). After topical analgesia of thelarynx, oral intubation was performed and anaesthesia was maintained with nitrous oxide and oxygen administered via a McGill semi-closed system, the patient breathing spontaneously.

The operation occupied 70 minutes. During this time there was no deterioration inthe patient's condition. At the end of the operation he did not regain consciousness and he died 17 hours later.

AUTOPSY:

Record lost.

COMMENT:

This death was due to the existing cerebral injury. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
68.1.57	1	Probably	> 24	Anoxic anoxia. Respiratory obstruction Cerebral damage.	Yes

Name: Philip Brond. Age: 61 Sex: M Race: E.

Disease: Staghorn calculus of kidney. Operation: Left nephrectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Considered a bad anaesthetic risk in that he had severe chronic respiratory disease, chronic purulent bronchitis, and severe cough. He had cor pulmonale and was in chronic congestive failure. He had hepatomegaly and some ascites. Before operation, he was digitalised treated with aminophyllin, physiotherapy and postural drainage.

PREMEDICATION:

ANAESTHETIC:

In view of the patient's pulmonary state, it was decided to attempt an epidural block. Xylocaine 1% with 1/200,000 adrenaline, 20 cc., was infiltrated into the epidural space from a lumbar approach. This failed. The anaesthetist then decided to use general anaesthesia, which was induced with thiopentone followed by nitrous oxide and oxygen with gradually added ether, the patient breathing spontaneously. A severe laryngospasm with bronchospasm occurred and oral intubation was performed under direct laryngoscopy, the tube being forced through the closed cords. The degree of general expiratory- and bronchospasm was so severe that it was impossible to ventilate the patient. The endotracheal tube was removed and inflation was attempted with a mask. Inflation proved impossible. The patient was then re-intubated and ventilation attempted as best as possible. Nitrous oxide, oxygen and ether were administered. Aminophyllin 10 cc. was given intravenously. However, despite the fact that bronchospasm and expiratory spasm passed off after about 10 minutes, the cyanosis which had occurred persisted for 30 minutes - in spite of apparently fair air entry into both lungs.

The operation now proceeded. A left nephrectomy was performed. The operation lasted 70 minutes and following discontinuation of the anaesthetic, the patient did not regain consciousness. Consciousness to the extent of return of coughing and swallowing occurred, and the pupils returned to their normal size, though the eyes were deviated to the right and upwards. Consciousness returned fully at no stage. The patient was given 40 cc. 50% sucrose 4 hourly and intravenous fluid administration was restricted, in an effort to combat cerebral oedema. He died 36 hours post-operatively.

AUTOPSY:

Sutured left nephrectomy wound. Left kidney removed. All sutures intact. Right kidney congested and showed some cortical scarring. Both lungs congested and purulent mucous present in the trachea. Heart showed gross coronary artery atherosclerosis and gross enlargement. It weighed 720 gm. Remaining viscera congested.

COMMENT ...

COMMENT:

This patient's death was the result of irreversible cerebral damage caused by anoxic anoxia occurring during the course of anaesthesia. The anaesthetic and its management must be identified as the major cause of this death.

Following the unsuccessful attempt to achieve lumbar epidural block, the anaesthetist resorted to general anaesthesia. Following this, the major difficulty which occurred was the complete inability of the anaesthetist to ventilate or oxygenate the patient after an incident of laryngospasm. This difficulty was due to bronchospasm together with gross spasm of the expiratory muscles. The administration of a muscle relaxant at this stage, if not earlier (when laryngospasm occurred) would have obviated this problem at least and allowed easier pulmonary ventilation.

Having decided on general anaesthesia, a thiopentone-succinylcholine-ventilation-intubation technique may well have obviated this sequence of events altogether. The difficulty with pulmonary ventilation which was experienced after intubation was misdiagnosed as an obstruction to the endotracheal tube - possibly a kind - and valuable time was wasted in removing and replacing the tube. Adequate bronchial toilette following intubation would also have permitted a better and easier pulmonary inflation.

PREVENTABILITY:

Initially having decided to perform a lumbar epidural block, the anaesthetist should perhaps have proceeded to do a spinal subarachnoid block when the former proved unsuccessful. Having resorted to general anaesthesia, a muscle relaxant should have been used to eliminate the gross expiratory muscle spasm as soon as this occurred, following endotracheal intubation. This would have been of even greater benefit if administered when the patient developed laryngospasm, even before intubation. Having performed endotracheal intubation, more efficient bronchial toilette should have been performed.

On these grounds, this death is regarded as "probably preventable" in spite of the patient's serious pulmonary and cardiac disease.

CASE No.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
69.1.58	2	No comment	< 24	Peritonitis Circulatory failure.	Yes.

Name: David Losper Age: 60 Sex: M Race: C.

Disease: Post-gastrectomy anastomotic leak. Peritonitis. Septicaemia.

Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

A moribund patient in extremely poor state. Pulse rate 160/minute. Respiration 32/minute. B.P. 110/60 mm.Hg. Plasma and blood had been transfused pre-operatively.

PREMEDICATION: Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered with the patient breathing spontaneously. Oral intubation was performed and carbon dioxide circle absorption started. Gallamine 80 mg. was given and IPPR instituted. Ether was discontinued at this stage.

The B.P. fell 15 minutes after induction of anaesthesia and could only be maintained with a noradrenaline drip infusion. Laparotomy was performed and drainage instituted for a diffuse peritonitis. At the end of operation normal respiration was resumed, no antidote for curarisation being considered necessary. On discontinuation of the anaesthetic consciousness was rapidly regained and the patient was returned to the ward. Respiration was adequate. He died in circulatory failure, apparently secondary to septicaemia, 4½ hours post-operatively.

AUTOPSY:

Generalised peritonitis. Atelectasis of basal segments of both lungs. Previous gastrectomy for carcinoma of the stomach.

COMMENT:

It is extremely difficult to assess the contribution of the anaesthetic to the patient's death in this case. Pre-operatively the patient's condition was very poor, if not moribund, with generalised peritonitis. The final rapid deterioration in his condition, as judged by the onset of circulatory failure - which responds with obvious tachyphylaxis to vasopressors - so often follows the administration of an anaesthetic, the institution of IPPR and the opening of the abdomen. Completion of the operation and discontinuance of the anaesthetic (though normal respiration is resumed and consciousness rapidly regained) fails to produce any change in the state of circulatory failure, which invariably ends in the patient's death within a few hours of operation.

Is the administration of anaesthetic really a contributory factor? Is the institution of IPPR with its probably resultant hypocapnia responsible for the sudden further drop in blood pressure, and the deterioration of the patient? Is metabolic acidosis the principle factor in a vicious cycle of circulatory failure-acidosis-circulatory failure? Or are both simply incidents in the course of a death which in fact is inevitable at that stage?

In that this death occurred some hours after recovery of consciousness with re-establishment of normal respiration, this death is regarded as due to peritonitis. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
70.1.58	1	Possibly	< 24	Hypotension Ischaemic anoxia. Cardiac arrest.	No.

Name; Nathan Diamond. Age: 58 Sex: M Race: E.

Disease: Intestinal obstruction. Operation: Laparotomy. Resection of two loops of gangrenous small intestine. Entero-anastomosis.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor. Pulse rate 120/minute, B.P. 80 mm.Hg systolic. In spite of transfusion of 3 pints blood and 4 litres replacement fluid, the patient still appeared to be dehydrated. However, in view of the diagnosis of a probably non-viable bowel, and the patient's worsening condition, it was decided to proceed with the operation.

PREMEDICATION:

Pethidine 50 mg., Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 375 mg. followed by nitrous oxide, oxygen and ether administered with spontaneous respiration via a closed circle absorption system. Oral intubation was performed and the B.P. remained at the pre-operative level of 80 mm.Hg systolic. Urethral catheterisation was performed and, 35 minutes after the induction of anaesthesia, laparotomy was commenced. At the start of laparotomy IPPR was instituted, though no relaxant was necessary or used. Three minutes later the B.P. dropped from 80 to 60 mm.Hg and in a further 3 minutes the pulse became impalpable. Cardiac massage was instituted within 1½ minutes. The heart started beating after a further 1½ minutes of massage. Noradrenaline 0.1 mg. was injected intraventricularly and the B.P. rapidly recovered to a level of 110 mm.Hg with a pulse rate of 125/minute. In view of the discovery of two obstructed gangrenous loops of small bowel, the operation had to be continued. These two loops were resected, entero-anastomosis was performed and the operation was completed in 70 minutes. During the last 10 minutes, the patient was allowed to breathe spontaneously.

At the conclusion of the operation, on discontinuation of anaesthesia, the patient recovered consciousness. The noradrenaline infusion was continued, digoxin and hydrocortisone being administered. The patient was returned to the ward to an oxygen tent. Later, noradrenaline failed to maintain the B.P. at a reasonable level and circulatory failure supervened, the patient dying 8 hours after operation.

AUTOPSY:

No autopsy was performed.

COMMENT:

This patient was in extremely poor condition before anaesthesia and surgery. He was hypotensive, in spite of fluid and blood replacement therapy. In these circumstances the use of thiopentone for the induction of anaesthesia must be considered misguided, though no further immediate deterioration occurred.

The anaesthetist took no immediate measures to elevate the blood pressure from its low level which, in a man of this age in this condition, must be considered fraught with danger.

The ...

The sudden deterioration in the patient's condition which followed the institution of IPPR and the opening of the abdomen could well have resulted from the mechanical effects of IPPR on the patient's already critical circulatory state, aided and abetted by the sudden hypocapnoea resultant on efficient hyperventilation through an absorber.

Cardiac arrest was caused, in all probability, from the results of ischaemic anoxia of the myocardium following the further fall in an aortic pressure already at a critically low level for some time. Treatment of the cardiac arrest when it occurred was prompt and effective. Vasopressor drugs were necessary subsequently to maintain an effective blood pressure. Although the patient regained consciousness rapidly after anaesthesia, intensive supportive measures failed to stave off the ultimate circulatory failure.

In spite of the seriousness of this patient's condition before anaesthesia, the management of the anaesthetic is considered a significant factor in the causation of cardiac arrest and thus in the ultimate demise of the patient.

PREVENTABILITY:

In view of the faults identified in the management of the anaesthetic, this death is regarded as "possibly preventable" in spite of the seriousness of the patient's condition pre-operatively, and the overall probable inevitability of his death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
71.1.58	1	Probably	ORD	Inhalation of regurgitated stomach content. Anoxic anoxia. Cardiac arrest.	Yes.

Name: Gavin Roos. Age: 6 Sex: M Race: E.

Disease: Tetralogy of Fallot. Operation: Relief of infundibular stenosis, with hypothermia and inflow stasis.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had a classical tetralogy of Fallot. He was cyanosed. There was no cardiac failure. B.P. 100/70 mm.Hg. Polycythaemia. Hb. 20 gm.%.

PREMEDICATION:

Pethidine 30 mg., Scopolomine 0.3 mg., 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg. followed by succinylcholine 20 mg. and orotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen and a trace of ether administered via a carbon dioxide absorption system with an IPPR technique. Occasional small doses of succinylcholine were given until the patient had settled. Prior to immersion in iced water, 10 mg. dTC was given. Cooling was rapid and uneventful, to 33°C, taking 25 minutes. A further 'drift' of 3°C occurred to 30°C and the temperature remained at this level for the rest of the procedure. Anaesthesia was discontinued at 32°C and the lungs were inflated with oxygen.

The operation was performed uneventfully and the period of total occlusion was 4½ minutes. On release of the tourniquets there was a fairly severe degree of cardiac dilatation but this responded well to massage and injection of calcium chloride into the left ventricle. Following this a B.P.of 70 mm.Hg systolic was obtainable. The patient was already making inspiration efforts as the chest was being closed. At completion of the operation the pupils were widely dilated and the skin cyanotic, but neither of these signs were regarded with apprehension as they are common accompaniments of hypothermia. Rewarming was carried out immediately after closure of the chest and proceded apparently uneventfully to 34°C. The heart rate then dropped from 90 to 40/minute and then ceased. The patient was immediately removed from the bath and the chest re-opened. All efforts to restart the heart were unsuccessful, as follows:-

1.22 cardiac massage commenced; 1.30 ½ cc. adrenalin intraventricularly without fibrillation; 1.32 intracardiac injection of calcium chloride; 1.53 noradrenaline drip infusion commenced; 1.57 ½ cc. adrenaline injected; 1.59 adrenaline injected; 2.17 calcium chloride injected; 2.36 calcium gluconate injected; 2.46 calcium gluconate injected; 2.50 D.C. defibrillator applied - 1 shock; 2.52 defibrillator applied - 1 shock; 3.00 efforts abandoned. From 1.45 an artificial pacemaker was used but this did not elicit a satisfactory beat.

AUTOPSY ...

AUTOPSY:

Sutured surgical wound anterior chest wall, 3rd left interspace. Sternum split exposing anterior mediastinum, sutured. Brain congested - smell of ether. Trachea contained a quantity of brownish mucus occluding both the main bronchi and extending well down into the lesser bronchi, with collapse of both lungs. Left sided pleural adhesions partially separated surgically. Small bilateral haemothoraces. Bruising of the visceral surfaces of the lungs. Heart weighed 170 gm. Heart: ventricular septal defect 1 x 1 cm. Atrial septal defect ½ cm. diameter. Hypertrophy of right auricle. Hypertrophy of right ventricle. Hypoplasia of pulmonary artery. Sutured surgical wound in anterior surface of right ventricle. Stomach contained quantity of brownish mucus.

COMMENT:

In this clinical record, four salient points stand out:-

1. At the conclusion of the operation, the skin was cyanotic and the pupils were widely dilated;
2. Cardiac arrest was preceded by rapidly progressive bradycardia;
3. When cardiac arrest occurred it proved quite refractory to treatment - the heart was completely unresponsive;
4. At autopsy (a) both lungs were collapsed,
   (b) both main and minor bronchi were obstructed on each side by brownish mucus,
   (c) the stomach contained similar brownish mucus.

Though, in the circumstances of hypothermia, polycythaemia and the incomplete correction of the cardiac lesion, the clinical observations stressed above are capable of interpretation other than indicating gross anoxic anoxia from respiratory obstruction, the autopsy findings indicate that this was in fact the probable cause of the patient's death.

At some stage in the operation, probably during or just after the stage of inflow stasis, fluid must have regurgitated from the stomach to the pharynx. This must then have passed down the trachea alongside the endotracheal tube (which was not cuffed), either at that time or when the patient commenced breathing spontaneously towards the end of operation.

In spite of all the general hazards peculiar to this form of anaesthesia and surgery, it seems that the ultimate cause of this death was simply the inhalation of silently regurgitated gastric content. The management of the anaesthetic, therefore, is the major causative factor in the patient's death.

PREVENTABILITY:

Awareness of the possibility of silent gastric regurgitation and adequate pharyngeal toilette would have prevented this death. Bronchial toilette at the conclusion of the operation may have averted the tragedy. On these grounds, this death is regarded as "probably preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
72.1.58	2	No comment	< 24	Haemorrhage	Yes

Name: Harriet Batchelor. Age: 30 Sex: F Race: C.

Disease: Right hydronephritis. Operation: Right nephrectomy.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Good other than for surgical condition of infected right hydronephrosis.

PREMEDICATION:

Morphine gr. ¼, Hyocine gr. 1/100.

ANAESTHETIC:

Lumbar epidural block was performed at a level of L.3-4 interspace, by loss of resistance technique, following a test dose of 5 ml. 1% Xylocaine, administering a main dose of 20 ml. 1% Xylocaine with 1/200,000 Adrenaline. The B.P., which initially was at a level of 130/70 mm.Hg, fell to 80 mm.Hg following positioning of the patient on the operating table. Administration of 6 mg. methyl amphetamine restored the B.P. to 110 mm.Hg.

Operation commenced 35 minutes after the epidural anaesthesia. Afterthe operation had been in progress for 2 hours, the patient became restless and coughed. This cough was difficult for the patient to control. Accordingly, general anaesthesia was induced with thiopentone 150 mg. followed by nitrous oxide, oxygen and ether administered via a McGill circuit with spontaneous respiration. Excessive haemorrhage resulted during the operation, from the operative site, and the inferior vena cava. The massive transfusion via two intravenous sites was necessary. Great difficulty was experienced in controlling the bleeding, which was never completely effectively controlled, due to the gross inflammatory process in the operative area. During the operation, lasting 3 hours, 8 pints blood were transfused.

At the conclusion of the operation anaesthetic was discontinued and the patient rapidly regained consciousness. However, continued blood transfusion was necessary because of continued haemorrhage, and difficulty was experienced in matching the rate of transfusion to that of blood loss. The patient became confused 18 hours post-operatively and pulled the intravenous infusion out. While feverish attempts were made to re-establish transfusion, the patient died.

AUTOPSY:

Extensive haemorrhage in the peritoneal cavity and into the nephrectomy bed and extraperitoneal tissues.

COMMENT:

This patient died of continuing haemorrhage from the operative site. The anaesthetic was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
73.1.58	2	No comment	< 24	Carcinoma of the oesophagus	No

Name: Ernest Tshanyela Age: 67 Sex: M Race: B

Disease: Carcinoma of the oesophagus. Operation: Oesophagoscopy and insertion of Soutar's tube.

Anaesthetic risk: 3.

PRE-OPERATIVE CONDITION:

The patient's condition was poor. He was emaciated.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 475 mg. followed by inhalation of nitrous oxide, oxygen and ether administered via a McGill's semi-open circuit. Oral intubation was performed.

Further course of the anaesthetic was untoward. Oesophagoscopy and insertion of Soutar's tube took 23 minutes. The patient recovered consciousness following discontinuance of anaesthesia and was returned to the ward. He died 23½ hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of his pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
74.1.58	2	No comment	< 24	Haemorrhage	Yes

Name: P.L. Hauptfleisch. Age: 64 Sex: M Race: E.

Disease: Haematemesis from duodenal ulcer. Operation: Partial gastrectomy.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

The patient was extremely shocked, B.P. 90 mm.Hg. Pulse rate 120/minute with continuing gastric bleeding. There was evidence that he had inhaled some blood. Pre-operatively 19 pints blood had been transfused and a total of 5 gm. calcium gluconate administered.

PREMEDICATION:

Morphine gr. ¼, 2 hours before operation. Atropine gr. 1/100 was given as further premedication.

ANAESTHETIC:

While oxygen was being administered in the theatre preparatory to induction of anaesthesia, there was a sudden catastrophic haemorrhage and haematemesis. The patient was intubated immediately and much blood aspirated from the trachea. A cuffed endotracheal tube was inserted and the cuff inflated. Anaesthesia was induced and maintained with nitrous oxide, and oxygen (flow rates 4 l. and 2 l/min. respectively) via a circle absorption system. Gallamine was given in divided doses, totalling 160 mg. throughout operation. An IPPR technique was used throughout.

At operation, an actively bleeding duodenal ulcer was found and a gastrectomy performed. During operation a further 8 pints blood was transfused which maintained the patient's B.P. at between 100 and 120 mm.Hg systolic throughout the major part of the procedure. 1 gm. calcium gluconate was given with every alternative pint of blood. Towards the end of the procedure, which lasted 3 hours, as the muscle relaxation was wearing off, cyclopropane and oxygen were used as the anaesthetic, to provide the relaxation for peritoneal closure. At the end of operation 2 mg. neostigmine preceded by Atropine gr. 1/100 was given to reverse the residual curarisation. Following discontinuance of the anaesthetic, the patient regained consciousness and had normal respiration. 35 minutes post-operatively he commenced further gastro-intestinal bleeding. This continued and the patient died approximately 1 hour post-operatively, despite transfusion.

AUTOPSY:

Autopsy revealed a massive gastro-intestinal haemorrhage, the gut being grossly distended with blood throughout its length. Gastrectomy had been performed but an actively bleeding duodenal ulcer with a large open vessel in its floor was found in the second part of the duodenum, distal to the site of duodenal closure. The spleen was enlarged and the splenic artery thrombosed. Gross pulmonary oedema was present.

COMMENT:

This case points the problem of correct estimation of blood loss in cases of gross haemorrhage. The patient was transfused with a total of 27 pints blood pre-operatively and during operation, with further blood post-operatively. This was in the face of continuing haemorrhage and was only sufficient to maintain the B.P. at a reasonable level. However the autopsy findings indicate the patient may have died of pulmonary oedema. One of the factors responsible may have been overtransfusion, as also other factors associated with massive transfusion, e.g. hypothermia. The anaesthetic per se is not considered to have contributed.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
75.1.58	2	No comment	< 24	Over-transfusion. Pulmonary oedema.	No

Name: Jacoba Horn. Age: 63 Sex: F Race: E

Disease: Hiatus hernia. Operation: Repair of hiatus hernia.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair.

PREMEDICATION:

Pethidine 75 mg., Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium and nitrous oxide, oxygen and ether were administered with spontaneous respiration. Following topical analgesia of the larynx, oral intubation was performed. Gallamine was given and an IPPR technique was instituted with the use of a circle absorber.

Throughout the operation, which lasted 2 hours 7 minutes, a total of 140 mg. gallamine was administered. The course of operation and anaesthetic was uneventful. At the conclusion of operation residual curarisation was reversed with neostigmine 2 mg. preceded by Atropine gr. 1/100. The patient rapidly regained consciousness on discontinuance of the anaesthetic and her condition was satisfactory.

Post-operatively, due to an error, the patient was inadvertently overtransfused, being given 2 litres 5% dextrose and water and 3 pints blood in the next 12 hours, without any indication for this. Atthis stage she developed pulmonary oedema and died shortly afterwards.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of pulmonary oedema due to the inadvertent overtransfusion. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
76.1.58	3	No comment	ORD	Elective cardiac arrest (cardiotomy, inflow stasis).	Yes

Name: Christina Granger Age: 3½ Sex: F Race: C.

Disease: Tetralogy of Fallot Operation: Pulmonary infundibular resection under hypothermia and inflow stasis.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Poor - the patient suffered from cyanotic congenital heart disease with convulsions. Three years old, she weighed bu 18 lbs.

PREMEDICATION:

Pethidine 10 mg. Scopolomine 1/600.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, ether being gradually introduced. The cyclopropane was subsequently discontinued anaesthesia being maintained with ether and oxygen administered via an infant circle absorption system. Oral intubation was performed The patient was given 6 mg. dTC and an IPPR technique instituted. She was placed in a bath of cold ice water and cooled to a temperature of 33°C. The patient was removed from the bath and placed on the operating table and operation commenced. An "after drop" of 5.5°C occurred during the procedure, bringing the temperature down to 28.5°C by the time cardiotomy was commenced.

The heart was arrested with an intracardiac injection of neostigmine. Inflow stasis was performed for a total of 6¾ minutes. Following cardiotomy, resection of the pulmonary obstruction was performed. A ventricular septal defect present was deemed too large to repair. The cardiotomy was closed, inflow stasis released but the heart failed to recover satisfactorily following release of the occlusion, in spite of intensive resuscitative measures including cardiac massage, intracardiac adrenaline, calcium chloride, calcium gluconate, the use of an electric defibrillator, and subsequently of an electrical pacemaker. The heart was rewarmed by pouring warm saline into the chest, restoring the myocardial temperature to 38°C. All these methods were of no avail, however, and the patient succumbed.

AUTOPSY:

Congenital heart disease. Pulmonary artery small, aorta over-rides both ventricles. Auricular and ventricular septal defects. Hypertrophy of right ventricle. Large subarachnoid haemorrhage over left parietal and medial surfaces of both lobes of brain.

COMMENT:

This patient's heart was arrested electively. Though hypothermia, as part of the anaesthetic technique, may have been a contributory cause to the refractoriness of the cardiac arrest, the anaesthetic management per se is not regarded as a significant factor in this fatality. Death basically was a result of the surgical procedure.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
77.1.58	2	No comment	< 24	Cerebral tumour	No

Name: Katie Bungo Age: 60 Sex: F Race: C.

Disease: Intracerebral tumour. Operation: Carotid angiography. Burrhole craniotomy. Brain biopsy.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Moribund. The patient was in a deep stupour, cyanosed, breathing stertorous. Respiratory rate 15/minute. Features of hypercarbia. B.P. 120/70 mm.Hg.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Following pharyngeal toilette, anaesthesia was induced with nitrous oxide, oxygen and gradually added ether. Oral intubation was performed and anaesthesia maintained with nitrous oxide, oxygen and minimal ether delivered via a McGill circuit, the patient breathing spontaneously.

The course of anaesthesia was uneventful during the operation, which lasted 2 hours. At the conclusion of the procedure the patient's level of consciousness returned to the level at which it had been pre-operatively. She died 10 hours after operation. The diagnosis of the lesion at operation was a malignant glioblastoma of the cerebrum.

AUTOPSY:

No autopsy.

COMMENT.

This patient died of her pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
78.1.58	3	No comment	< 24	Uncontrollable haemorrhage. Cardiac arrest. Cerebral anoxic damage.	Yes

Name: Hilda Jupp. Age: 50 Sex: F Race: C.

Disease: Mitral stenosis. Operation: Mitral valvotomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had tight mitral stenosis. B.P. 120/75 mm.Hg. She had a two finger hepatomegaly. She was digitalised and was not in cardiac failure.

PREMEDICATION:

Pethidine 75 mg. Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 225 mg. followed by dTC 20 mg. The patient was ventilated with oxygen; Xylocaine, for topical analgesia, was applied to the larynx, and endotracheal intubation performed. An IPPR technique was instituted using nitrous oxide, oxygen, via a circle absorption system. A trace of ether was administered until the thorax was opened.

The operation commenced and during the procedure two additional doses of dTC, 5 and 6 mg., were given. Anaesthetic and operation were uneventful until dilatation of the mitral valve was attempted. At this stage the auricular appendage tore, resulting in massive uncontrollable haemorrhage. The rapid exsanguination of the patient led to cardiac arrest. Cardiac massage was delayed for more than 5 minutes, as the surgeon was hampered by the open left atrium. After control of the left atrial appendage, cardiac massage was begun. This, together with intracardiac administration of adrenaline and calcium chloride, and the use of an electrical defibrillator on two occasions, finally restored a normal heart beat after ½ hour. Up to the time of the massive haemorrhage, 1 pint blood had been transfused. During the subsequent period, 6 pints blood and 1 pint plasma were transfused. At the conclusion of operation, which lasted 3¼ hours, residual curarisation was reversed with neostigmine 2 mg. preceded by Atropine 1.2 mg. Spontaneous respiration was resumed. The patient failed to regain consciousness, the pupils were widely dilated. B.P. was maintained at a level of 115-120 mm.Hg by means of an infusion of noradrenaline. However, the patient died 3 hours post-operatively.

AUTOPSY:

Haemorrhage following tear of excessively friable left auricle. Tear in left auricular appendage. Closed incision of left auricular appendage. Mitral valve stenotic with small lacerated wound where valve margins meet. Enlarged left auricle with lacerated wound 1 cm. in length. Bilateral pneumothorax, 500 ml. blood each side.

COMMENT...

COMMENT:

This death was obviously due to massive haemorrhage resulting in cardiac arrest and irreversible cerebral anoxia. Anaesthesia is not contributory but, as the patient suffered cardiac arrest while anaesthetised and subsequently failed to regain consciousness, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
79.1.58	2	No comment	< 24	Cerebral tumour involving vital centres.	Yes

Name: Sheida Abas Age: 2½ Sex: F Race: C.

Disease: Cerebellar tumour. operation: Posterior fossa craniotomy. Partial excision of tumour involving vital centres.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Poor. Very drowsy, signs of intracranial space-occupying lesion.

PREMEDICATION:

Atropine gr. 1/200.

ANAESTHETIC:

Pre-operatively ventricular drainage was established. Anaesthesia was induced with nitrous oxide, oxygen and ether. Oral intubation was performed and anaesthesia maintained with nitrous oxide and oxygen with minimal ether, delivered via an Ayre's T-piece, the patient breathing spontaneously.

Posterior fossa craniotomy was performed and a large cerebellar tumour was found. Dissection of the tumour was attempted but extensive dissection caused the patient to commence a gasping type of respiration. This indicated that the vital centres were involved by the tumour. The operation was abandoned. Normal respiration resumed. Blood loss of 350 ml. was replaced as lost. At the conclusion of the procedure the patient was breathing normally and she recovered consciousness to much the same level as had been present before surgery. The patient suffered a sudden respiratory arrest 13 hours post-operatively and died.

AUTOPSY:

Extensive cerebral tumour involving the midbrain. Death due to involvement of vital cerebral centres by tumour and surgical interference, with probably subsequent oedema.

COMMENT:

This death resulted from involvement of vital cerebral centres by a neoplasm, and post-operative cerebral oedema. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
80.1.58	2	No comment	< 24	Subarachnoid haemorrhage	No

Name: George Butler Age: 55 Sex: M Race: E.

Disease: Carcinoma of the tonsil (inoperable) Operation: Surgical section of cranial nerves 5 and 9.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient was suffering from inoperable carcinoma of the tonsil with a severe and intractable pain. The right nostril was occluded by growth, the patient was unable to open his mouth. He was also diabetic but this controlled on diet.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Xylocaine 4%, 4 cc., was injected transtracheally. The left nostril was sprayed with 5% cocaine. Following pre-oxygenation anaesthesia was induced with nitrous oxide and oxygen administered via a McGill circuit, the patient breathing spontaneously. Blind nasal intubation was performed with a cuffed tube. Anaesthesia was then maintained with nitrous oxide, oxygen and ether, the patient breathing spontaneously.

No difficulties were encountered. The operation, section of the trigeminal and glossopharyngeal nerves, was completed in 210 minutes. At the conclusion of the operation the patient rapidly recovered consciousness. Twelve hours later he suddenly collapsed and became comatose. A gross subarachnoid haemorrhage was diagnosed on lumbar puncture. The patient died within 5 minutes.

AUTOPSY:

No autopsy.

COMMENT:

This death was quite unrelated to the anaesthetic management.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
81.1.58	1	Probably	> 24	Cardiac arrest. Cerebral anoxic damage.	Yes

Name: Lionel Philander Age: 1¼ years Sex: M Race: C

Disease: Phimosis Operation: Circumcision.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Though thin, the child was not thought to show any obvious pathology. He was peaky and the eyes were sunken. It was discovered afterwards that he had been suffering from gastroenteritis. He was thought to be mildly dehydrated.

PREMEDICATION:

Atropine gr. 1/200.

ANAESTHETIC:

Anaesthesia was induced with open ethyl chloride followed by open drop ether, administered by means of a Schimmelbusch mask. When anaesthesia had been induced, it was maintained by a medical student under supervision, using open drop ether. No difficulty was encountered and anaesthesia of the depth of Guedel stage 3 plane 2 was maintained. Induction of anaesthesia took 15 minutes.

The operation took 10 minutes. The anaesthetist remarked that bleeding was very slight. There is no record of pulse rate and blood pressure. Anaesthesia was discontinued a few minutes before the end of the operation. At the conclusion of the procedure the patient was turned onto the right lateral position. Nothing untoward was observed at this stage, the airway was unobstructed. The anaesthetist turned to the basin, to wash his hands, leaving the medical student (who had administered the anaesthetic) to supervise the patient. Five minutes after conclusion of the operation attention was drawn to the child by the passage of a watery stool. It was then noticed that respiration had ceased. The child was immediately turned onto his back and IPPR with oxygen, via a face mask, was commenced. The chest was auscultated but a firm diagnosis of cardiac arrest was not made until 5 minutes later. At this stage a surgeon was called to perform cardiac massage. This resulted in a further 2 minute delay. Following incision of the skin, 7 minutes after the observation of the cessation of respiration, the surgeon noted that a degree of bleeding and the pink colour of the muscles did not bear out the diagnosis of cardiac arrest. Further time was wasted auscultating the chest. No cardiac beat wasaudible and thoracotomy was proceeded with. When thoracotomy was eventually completed, 10 minutes from the cessation of respiration, the heart was found arrested in diastole and was flabby. Cardiac massage was commended, it not being found necessary to open the pericardium. Methyl amphetamine 10 mg. intravenous with 10 mg. intramuscularly resulted in the return of a strong pulse. Respiration commenced shortly and was of a gasping nature. Endotracheal intubation was performed and IPPR ventilation maintained until the chest wall had been closed with underwater drainage.

The child was returned to the ward, the endotracheal tube left in situ. He failed to regain consciousness. In the ward the child's B.P. was 75 mm.Hg systolic, temperature 100°F, the eyeballs fixed and pupils dilated. It had also been observed on admission that the patient's haemoglobin was only 9 gm.% Some hours later the endotracheal tube was removed. As unconsciousness persisted, a

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tracheotomy was performed 24 hours later to maintain a clear airway and to allow tracheobronchial toilette. Unconsciousness persisted and the child died on the 6th post-operative day.

AUTOPSY:

Incised tracheotomy wound in neck. Incised chest wound for cardiac massage. Penis circumcised with sutures still in situ.

COMMENT:

The ultimate cause of this patient's death is clear. Irreversible cerebral anoxic damage followed cardiac arrest, recognition of which was tardy, taking 5 or more minutes. Effective cardiac massage was not instituted for another 2 to 3 minutes at least. Thus the minimum period of total cerebral anoxia must have been approximately 7 minutes, ensuring a bad prognosis.

Following cardiac massage and resuscitation, heart beat and respiration recommenced but, though the child lingered for 6 days, consciousness was regained at no time, confirming the anticipated presence of grave irreversible cerebral anoxic damage.

It is known that the child was: (1) malnourished, (2) had had gastroenteritis and was dehydrated, though pre-operatively this was not regarded as serious. Anaesthesia appears to have been untoward, though there is no record of the patient's blood pressure or pulse rate at the end of operation. However it was noted that during the operation bleeding was very slight. This may well have indicated severe hypotension, but not necessarily so.

Following the discontinuation of anaesthesia, the patient was positioned in the lateral position and was left under the supervision of a medical student, who observed that the airway was clear and there was no vomiting. The turning of the patient would have aggravated any existing hypotension. The passage of a watery stool 5 minutes later, which drew the anaesthetist's attention to the child, may well have indicated the onset of gross anoxia following cardiac arrest. It was only then that respiration was observed to have ceased. Inflation with oxygen was commenced but a firm diagnosis of cardiac arrest was only made 5 minutes later. Even then there was tardiness and further delay.

Judging from the clinical report, a reasonable hypothesis as to the cause of this cardiac arrest is that it resulted from a state of extreme hypotension, caused by the effect of the anaesthetic in a dehydrated and hypovolaemic child with probable electrolyte disturbance. In this light, the observation of extremely slight bleeding at the operative site is of great importance, especially in the absence of blood pressure and pulse recordings. Had this arrest resulted directly from an overdose of ethylchloride, a well-known condition, the arrest would have occurred during the induction of anaesthesia and not at its conclusion. That respiratory obstruction was the cause seems unlikely from the story.

Once cardiac arrest had occurred, both diagnosis and treatment were faultily tardy, ensuring the ultimate failure of resuscitation. On these grounds anaesthesia and its management must be inculpated as the cause of this death.

PREVENTABILITY:

On the grounds that (1) the pre-operatively noted dehydration was not taken seriously and treated, (2) observation of the patient while anaesthetised and during recovery was inadequate, and (3) the diagnosis and treatment of cardiac arrest, once it had occurred, was tardy in the extreme, this death must be regarded as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
82.1.58	2	No comment	< 24	Intra-cerebral tumour.	No

Name: Reuben Age: Unknown Sex: M Race: B

Disease: Intracerebral tumour. Operation: Craniotomy for removal of tumour.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient was in a poor pre-operative state, being deeply comatose, respiration 16/minute, B.P. 150/80 mm.Hg, with marked pooling of secretions in tracheobronchial tree.

PREMEDICATION:

None.

ANAESTHETIC:

Following oral and pharyngeal toilette, and pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen and gradually added ether. Following topical analgesia of the larynx, oral intubation was performed. Tracheobronchial toilette was performed and anaesthesia was maintained with nitrous oxide and oxygen, delivered via a McGill semi-open circuit, the patient breathing spontaneously.

The course of the anaesthetic, throughout the operation, was untoward. The operation lasted 4 hours 30 minutes and at its conclusion the patient's level of consciousness returned to the pre-operative level. He was returned to the ward with the endo-tracheal tube in situ. Frequent tracheobronchial toilette was necessary. The patient died 4½ hours post-operatively.

AUTOPSY:

No autopsy was performed.

COMMENT:

This death resulted from the patient's pre-existing disease and the results of surgery. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
83.1.58	2	No comment	< 24	Meningitis Septicaemia Lateral sinus thrombosis	No

Name: G. Jacobs Age: 16 Sex: M Race: C

Disease: Acute mastoiditis. Lateral thrombosis. Perisinus abscess. Meningitis. Septicaemia. Pyaemia.

Operation: Mastoidectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was in extremis, stuperose and 10% dehydrated. Temperature was 105°F, the pulse rate 120/minute, B.P. 110/60 mm.Hg, respiration 40/minute (acidotic in character). The patient was also jaundiced.

PREMEDICATION:

Atropine gr. 1/100 by intravenous injection.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered via a McGill circuit, the patient breathing spontaneously. Oral intubation was performed and anaesthesia maintained with nitrous oxide and oxygen, using minimal ether. During the anaesthetic, 1,000 ml. 5% dextrose in water was administered in view of the pre-operative clinical dehydration. There was considerable bleeding from the operative site during the procedure. This was replaced by transfusion immediately post-operatively.

The operation lasted 60 minutes and the patient returned to the pre-anaesthetic level of consciousness. He appeared in slightly better general condition than pre-operatively - the tongue was now moist, the respiratory rate 25/minute. The patient died 9 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of the pre-existing disease. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
84.1.58	1	Probably	< 24	Cardiac arrest. Cerebral anoxic damage.	Yes

Name: Allan Knowlden Age: 3 Sex: M Race: E.

Disease: Laceration of the ulnar nerve. Operation: Exploration and resuture of lacerated ulnar nerve.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Other than for the surgical lesion, the child's pre-operative state was quite normal. Temperature 99°F. An anaesthetic had been given to the patient 2 months previously, for exploration of the ulnar nerve; he had hiccoughed throughout the anaesthetic but otherwise this had been untoward.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, and oxygen with gradually added ether, administered via an infant's modified T-piece bag and mask. Induction of anaesthesia was smooth and trouble-free, with no episodes of respiratory obstruction. After 15 minutes, the method of anaesthesia was altered to open drop ether administration on a Schimmelbusch mask, the patient breathing spontaneously. Oxygen, at a flow rate of 1 litre/minute, was added under the Schimmelbusch mask by means of a rubber tube. An oropharyngeal airway was inserted. The pulse was noted to be normal and at no time were there signs of respiratory obstruction. The B.P. was not recorded. 20 minutes after induction of anaesthesia, surgery was commenced, and 25 minutes after this, the pulse was suddenly observed to be absent (having been present a few minutes previously). There were a few respiratory gasps and then apnoea ensued. Artificial ventilation with oxygen was immediately commenced by means of a face-mask, and the surgeon was informed. Two minutes after the observation of cardiac arrest, the surgeon was massaging the heart via the diaphragm, through an abdominal incision. After a further 3 minutes the heart started to beat irregularly. Nikethamide 2 ml. was injected into the heart. After a further 9 minutes the heart stopped again and a further injection of 1 ml. Nikethamide was administered into the heart. Massage was continued. Ten minutes later (25 minutes after the observation of cardiac arrest) ¼ ml. of a 1/1,000 solution of Adrenaline was injected into theheart. After 5 minutes the heart started beating regularly. At this stage an endotracheal tube was inserted and respiration was continued artificially by this means.

Following re-establishment of the heart beat, the abdomen was closed and the operation was abandoned. Spontaneous respirationdid not recommence and 1 hour after the conclusion of the operation the patient was transferred to a "Drinker cabinet" respirator. The patient died 4 hours later, without regaining consciousness.

AUTOPSY:

No significant findings; no discernable cause of death discovered. The brain was congested. The trachea contained frothy mucous. The lungs were pale. The thymus weighed 25 gm. The liver and kidneys were congested. The stomach contained some watery mucous. There was a smell of ether.

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COMMENT:

From the clinical description, this death clearly resulted from the administration of the anaesthetic. Pre-operatively the child was fit and surgery was not of a major nature. It appears that the first untoward event was what one might term "primary cardiac arrest" - the pulse was noted to cease, followed by cessation of respiration shortly afterwards, 45 minutes after the commencement of induction of anaesthesia and 25 minutes after the operative procedure was begun. The precise aetiology of this cardiac arrest is difficult to elucidate with any certainty. Possibilities are:

(1) Anoxic anoxia. There was no respiratory obstruction at any time. This is stated very definitely by the anaesthetist. However, it is known that - with open drop ether administration - anoxia may result from the high concentration of ether vapour under the mask, the vehicle being air, in the presence of the respiratory depression of deep anaesthesia. However, supplemental oxygen (which would tend to lessen this mechanism of anoxic anoxia) was administered in this case.

(2) Overdosage of ether. This is a definite possibility. The autopsy findings are typically non-specific but the smell of ether vapour in the brain is remarked. This must be remembered in relation to the fact that death occurred 6 hours after the cessation of anaesthesia, which period the infant had spent in a Drinker respirator - an observation which is of doubtful significance. The patient was an infant and deep anaesthesia is easily and rapidly achieved with open drop ether administered in small children. When questioned, the anaesthetist was unsure of the exact clinical depth of anaesthesia; the pupillary signs had not been sought, the B.P. was not recorded and the drop in B.P. resulting from deepening anaesthesia may not have been noticed by digital palpation of the pulse. But, against this, one must bear in mind that the anaesthetist (though a trainee) was reasonably experienced. Furthermore, overdosage with ether usually results in cardiac arrest secondary to respiratory failure, rather than in a primary cardiac arrest such as is described here. However, this is not invariable. Deep ether anaesthesia, besides having a direct depressant effect on the myocardium, may also result in some degree of ischaemic anoxia of the myocardium due to lowering of the B.P.

(3) Reflex cardiac arrest. There is a possibility arrest may have resulted from reflex vagal inhibition following stimulation of the ulnar nerve. This is considered unlikely in that, from the description of the quiet respiration of the patient, the depth of anaesthesia would not appear to have been light. However, this possibility cannot be totally excluded.

(4) Other causes. None of the other causes of cardiac arrest appear to have been operative in this case. The aetiological diagnosis of cardiac arrest may be regarded as uncertain, though overdosage of ether must still be considered a distinct possibility.

Once cardiac arrest had occurred, diagnosis and treatment appears to have been reasonably prompt, cardiac massage having been instituted within 2 minutes. However, one must question the efficiency of the abdominal route for massage and the efficacy of intracardiac nikethamide is also open to question.

It was obvious at the conclusion of the resuscitative procedures that the child had suffered from gross anoxic cerebral damage, a point which may further strengthen the diagnosis of anoxic anoxia preceding cardiac arrest (in view of the reasonably prompt institution of cardiac massage). This death was due to the anaesthetic management, but the precise cause remains uncertain.

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PREVENTABILITY%

In view of the lack of proper observation of the patient by the anaesthetist, and the lack of certainty as to the clinical depth of anaesthesia, together with the absence of records of B.P., this death must be regarded as probably preventable. Better clinical observation would almost certainly have avoided the outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
85. 1.58	2	No comment	< 24	Massive haema-temesis.	Yes

Name: William Adonis Age: 20 Sex: M Race: C

Disease: Lacerated axillary blood vessels. Operation: Ligation of severed axillary vessels.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

Poor: the patient had lacerated wounds of the left arm and axilla. He was "dead drunk", temperature sub-normal, respiration irregular. Pulse rate 120/minute. B.P. 40 mm.Hg systolic. He was in a state of severe circulatory collapse. The lungs were clear. It was considered essential to secure the lacerated axillary vessels to staunch the haemorrhage.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

A stomach tube was passed and the stomach contents emptied. Anaesthesia was induced with nitrous oxide and oxygen, and an endotracheal tube was passed. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether, administered via a semi-open anaesthetic circuit, the patient breathing spontaneously.

During the operation a total of 15 pints compatible blood was transfused and the patient's B.P. rose to a level of 120 mm.Hg systolic, with a pulse rate of 90/minute. At the conclusion of the procedure, which lasted 210 minutes, the patient rapidly regained consciousness and was returned to the ward. He was observed to be developing a paralytic ileus, 4 hours post-operatively, bowels sounds being completely absent. Gastric aspiration and intravenous fluid therapy was commenced. A sudden unexpected massive haematemesis developed 16 hours post-operatively, followed by gross melaena and death.

AUTOPSY:

Sutured wound in left arm and axilla. Brain congested; smell of ether. Lungs congested. Peritoneal cavity contained 200 ml. blood-stained fluid. Stomach distended, containing large amount of black fluid. Paralytic ileus associated with area of possible bruising of ileum; 20 cm. of ileum block and macroscopically bruised area well demarcated, congestion on either side. Histology: bruising of bowel. Carbide poisoning suspected.

COMMENT:

Death was due to a massive haemorrhage from the gastro-intestinal tract. It was not decided whether this was of traumatic or toxicological aetiology. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
86.1.58	2	No comment	< 24	Cerebral lacera9 tion.	No.

Name: Noel Vincent. Age: 19 Sex: M Race: E.

Disease: Head injury: gross cerebral damage. Operation: Craniotomy. Exploration and debridement of brain.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Deeply comatose, respiration sterterous. B.P. 140/80 mm.Hg. Respiratory rate 15/minute.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Topical analgesia of the larynx and pharynx was produced with a Xylocaine 4% spray. The patient was intubated and oxygen was administered. Subsequently nitrous oxide was given, when the patient responded by movement to stimulation. Anaesthesia had to be further deepened by the intermittent administration of minimal ether.

The respiratory rate, B.P. and pulse rate were maintained at a static level throughout the operation, which lasted 6 hours. At the operation the dura was found to be torn to shreds, the brain grossly contused and oozing from all areas. No extra- or subdural haematoma were found. Compatible blood was transfused throughout the procedure, as lost.

The patient ceased breathing 4 hours post-operatively. Artificial ventilation was instituted immediately and the patient was placed in a "Drinker cabinet" respirator. However there was progressive deterioration in his condition and the patient died 20 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient's death was due to severe cerebral laceration. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
87.1.58	2	No comment	< 24	Cerebral tumour	Yes

Name: Congile Zwelugani. Age: 40 Sex: M Race: B

Disease: Intracranial space-occupying lesion; ?Meningitis.

Operation: Carotid angiography.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor; the patient was comatose. He had bronchopneumonia. Temperature 104°F, B.P. 100/70 mm.Hg., pulse rate 76/minute. Signs of an intracranial space-occupying lesion were present.

PREMEDICATION:

No premedication. Angiography was attempted first without anaesthesia; however, as the patient moved slightly, it was decided that anaesthesia was necessary.

ANAESTHETIC:

Following pharyngeal toilette, topical analgesia of the pharynx and larynx was secured with a Xylocaine 4% spray. Oral intubation was performed. Anaesthesia was induced with nitrous oxide and oxygen administered via a semi-open circuit, the patient breathing spontaneously.

Following conclusion of angiography, which took 30 minutes, the patient was returned to the ward in much the same condition as he had been pre-operatively. A failure of respiration subsequently ensued, and cardiac arrest occurred. The patient was immediately brought back to theatre and artificial ventilation was instituted. Cardiac massage was commenced through the diaphragm via an abdominal incision. Little response was obtained although the heart gave a few desultory beats on two occasions, following intracardiac injections of 1 cc. 1/1,000 adrenaline into the left ventricle.

AUTOPSY:

Chronic subdural haematoma.

COMMENT:

This patient's death resulted from the effects of chronic subdural haematoma. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
88.1.58	3	No comment	< 24	Haemothorax Continuous haemorrhage. Anoxic anoxia.	Yes

Name: M.Nyamakazi. Age: 48 Sex: M Race: B.

Disease: Oesophagopulmonary fistula. Operation: Thoracotomy. Excision of oesophagopulmonary fistula and right basal lobectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient was in a poor state, wasted and suffering from severe pulmonary disease with marked right basal bronchiectasis. Haemoglobin 12.5 gm.% B.P. 120/80 mm.Hg. Temperature 98°F. Gastrostomy had been performed 3 months previously and gastrostomy feeds with high protein, vitamins and antibiotics had been given for the last 3 months. Physiotherapy had also been instituted. However, despite all this, the patient was in a poor condition pre-operatively.

PREMEDICATION:

Pethidine 75 mg. Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 400 mg. in divided doses followed by cyclopropane and oxygen, administered via a closed circuit, with spontaneous respiration. Following topical analgesia of the larynx with 4% Xylocaine, oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen, via a circle absorption system, using an IPPR technique. dTC, totaldose 39 mg., was given. Supplementary analgesia was achieved with Pethidine, in divided doses to a total of 75 mg. throughout the operation.

Initially, following commencement of surgery, control of respiration was difficult to establish but this was finally achieved with a combination of dTC and Pethidine. For the first 1½ hours of surgery the B.P. was maintained at a level of between 100 and 120 mm.Hg systolic. Throughout surgery, bleeding from the operative site was severe. B.P. was maintained by the adequate transfusion of compatible blood. After 2 hours' surgery, by which time 4 pints blood had been administered, the supply of compatible blood was exhausted and more had to be cross-matched. There was a delay of 30 minutes before more blood was obtained, during which time the B.P. fell progressively until it reached a level of 65 mm.Hg systolic. At this stage further blood was obtained and rapidly transfused. The B.P. rose first to 80 mm.Hg and subsequently, after a further 4 pints, to 120 mm.Hg systolic. The B.P. was maintained at this level until the end of operation by the transfusion of still more blood. During transfusion, a total of 40 ml. 10% solution of calcium gluconate was given in divided doses. Bleeding at the operative site did not appear to have been adequately controlled at the end of the operation, which lasted 5 hours. Curarization was reversed by the administration of 1 mg. neostigmine preceded by atropine gr. 1/50. Normal spontaneous respiration returned and appeared to be adequate. The patient did not recover consciousness and, 1½ hours following return to the ward, his condition deteriorated and he died.

AUTOPSY:

There was a right haemothorax in excess of 1 pint blood. The right lung was collapsed. The remaining part of the right lung and the left lung were severely diseased, with chronic fibroid pulmonary

tuberculosis ...

tuberculosis. There was very little functional lung tissue left.

COMMENT:

The cause of this patient's death appears to have been anoxia both anoxic and ischaemic, resulting from continued bleeding from the operative site, resulting in a haemothorax and in atelectasis of the lung, in the presence of little remaining lung tissue due to the presence of severe chronic fibroid tuberculosis. That he did not recover consciousness after receiving only nitrous oxide and Pethidine as anaesthetic agents points to a degree of cerebral anoxia during surgery.

The remark of the anaesthetist that respiration was difficult to control may be considered a sign of some degree of cerebral anoxia at the commencement of thoracotomy. Considering the autopsy findings, it will have been difficult to avoid some degree of anoxia during thoracotomy.

In this context, the episode of severe hypotension for approximately 30 minutes (resulting from unreplaced blood loss, when existing stocks of blood were temporarily exhausted) may well have resulted in cerebral ischaemic damage. This would have accounted for the post-operative unconsciousness.

Though the anaesthetic and its management are doubtless contributory to this death, taking into account the nature and extent oftthe pulmonary pathology, the difficulties imposed by the operative procedure on the maintenance of efficient pulmonary ventilation and the profound blood loss during the procedure - which continued post-operatively - the anaesthetic is regarded as an unavoidable, necessarily contributory factor.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
89.1.58	3	No comment	ORD	Myocardial ischaemia Cardiac arrest.	Yes

Name: Moosa Voterson Age: 58 Sex: M Race: C

Dissase: Dehiscence of abdominal wound (clinical diagnosis: massive haemorrhage from aortic graft).

Operation: Laparotomy. Resuture of abdominal wound.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient suffered from severe generalised atherosclerosis and had signs on ECG of myocardial ischaemia, together with frequent ventricular extrasystoles. Four days previously an abdominal aortic aneurysm had been resected and an aortic graft inserted. On this the 4th post-operative day, an extensive abdominal wound disruption had occurred and severe vascular collapse had ensued. The B.P. dropped to 80 mm.Hg systolic, the pulse rate rose to 120/minute. His extremities were cold. There was marked peripheral pallor and vasoconstriction and frequent ventricular extrasystoles. Respiration was irregular. A clinical diagnosis of a rupture of the aortic graft was made.

PREMEDICATION:

Atropine gr. 1/100 by intravenous injection.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen and oral intubation followed topical analgesia of the larynx. Anaesthesia was continued with nitrous oxide, oxygen and minimal ether administered via a carbon dioxide circle absorber by an IPPR technique. Relaxation was achieved by means of gallamine in divided doses, totalling 120 mg.; complete curarization was not achieved. At the commencement of induction of anaesthesia the B.P. was 80 mm.Hg systolic and during induction 1½ pints blood were rapidly transfused because of the diagnosis of a ruptured aortic graft with retroperitoneal haemorrhage. During induction the B.P. dropped to 60 mm.Hg systolic with no response to the rapid transfusion of blood.

Following laparotomy it was found that, in fact, no rupture of the aortic graft had occurred and there was indeed no intra-abdominal bleeding. Blood transfusion was discontinued promptly. The B.P. rose to 90 mm.Hg after 30 minutes and subsequently fluctuated between 90 and 60 mm.Hg systolic once the abdomen was closed. At the conclusion of the operation, 1 mg. neostigmine was administered preceded by Atropine gr. 1/100, but this produced little effect on the inadequate spontaneous respiration which was then present. At the immediate conclusion of the operation, cardiac arrest occurred. Cardiac massage was instituted immediately but was of no avail.

AUTOPSY:

Extensive myocardial fibrosis plus coronary atherosclerosis. No coronary thrombosis was demonstrated.

COMMENT:

This appears to have been a case of circulatory collapse accompanying the sudden disruption of an abdominal operative wound. The rapid transfusion of blood, pre- and during the induction of anaesthesia,

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was based on the incorrect clinical diagnosis of a massive haemorrhage from an aortic graft. The fact that no improvement in the condition resulted provided the first clue to the hypotensive state being probably primarily cardiac and not haemorrhagic in origin.

This death mainly resulted from myocardial ischaemia due to hypotension, precipitated by a wound disruption in a patient with severe ischaemic heart disease. Any contributory role played by the anaesthetic or its management is regarded as unavoidable in the circumstances.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
90.1.58	1	Possibly	ORD	Cardiac arrest.	Yes

Name: Mavis Quigley Age: 44 Sex: F Race: E.

Disease: Carcinoma of the splenic flexure with faecal fistula. Lung abscess.

Operation: Laparotomy and closure of faecal fistula.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient was in an extremely toxic, steadily deteriorating state pre-operatively. A few hours before the operation, the B.P. had dropped to a level of 80/60 mm.Hg. Transfusion of plasma and resuscitative measures with a vasopressor drug restored the B.P. to a level of 110 mm.Hg systolic just before the operation.

PREMEDICATION:

The patient was given Pethidine 100 mg. and Atropine gr. 1/100 2 hours pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone in divided doses, totalling 300 mg. This was followed by succinyl choline, ventilation with oxygen and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen delivered via a semi-closed circle absorption system, with minimal ether. Succinyl choline was administered in intermittent doses of 20 mg., as necessary, and an IPPR technique was instituted. Following induction of anaesthesia the BP remained stable at 110 mm.Hg systolic, and the pulse rate which 120/minute rose to 140/minute.

Surgery commenced 25 minutes after the induction of anaesthesia and an intravenous infusion of 5% dextrose and water was started. Blood loss during surgery was not great; however, after 30 minutes, a blood transfusion was commenced. The B.P. was still maintained at 110 mm.Hg. Sixty-five minutes after the induction of anaesthesia (40 minutes after commencement of operation) the pulse was observed to become feeble and the B.P. had dropped. Blood was now replaced rapidly, under pressure, despite the fact that the blood loss at the operative site was not great. At this stage a total of 1 pint blood had been transfused. After a further 2 minutes, the pulse became impalpable, the pupils were widely dilated and cardiac arrest was diagnosed. IPPR with pure oxygen was begun, the heart was palpated through the diaphragm and was completely asystolic, and cardiac massage was commenced. Within 1 minute theheart recommenced beating and the B.P. was recordable at 100 mm.Hg systolic. A marked tachycardia was present, the pulse rate was 160/minute The pupils became small. The operation had reached the point of no return and had to be continued. After a further 10 minutes cardiac arrest again occurred, the pupils once again being dilated. On this occasion a left thoracotomy was performed and direct cardiac massage commenced. The surgeon remarked on the presence of pericarditis. At this stage the heart was found to be in ventricular fibrillation. Electrical defibrillation was resorted to, and normal cardiac rhythm was restored. An injection of 10 cc. calcium chloride into the left ventricle produced effective cardiac action. An infusion of 1/500,000 noradrenaline was commenced. The pupils again became small and the operation was continued. After a further 10 minutes, spontaneous respiration recommenced. However, the B.P. dropped after a further 10 minutes to 60 mm.Hg systolic. Administration of vasopressors

failed / ...

failed to elevate this. The operation was continued and the patient was allowed to breathe spontaneously. After a further 15 minutes cardiac arrest occurred once again, with ventricular fibrillation, and again electrical defibrillation was resorted to. An effective beat could not be re-established. Calcium chloride 10 cc. was injected into the left ventricle, without effect. An electrical pacemaker was used in an attempt to restart the heart. All these resuscitative measures were of no avail and, after a further hour, resuscitative attempts were abandoned.

AUTOPSY:

Brain showed congestion. Trahhea and main bronchi contained purulent mucous. Left lung collapsed and left pleural cavity contained approximately 1 pint serosanguinous fluid. Abscess measuring 5 cm. in diameter in the base of the right upper lobe, well walled off, with surrounding consolidation of the upper and middle lobes and localised pleural adhesions. The heart showed flaccidmusculature with subendocardial haemorrhages in the posterior wall of the left ventricle. Transverse colon was exteriorised. Duodenum had been repaired surgically. Small bowel collapsed and very friable. Large intraperitoneal pelvic abscess and a smaller abscess at the base of the right paracolic gutter with matting of the bowel in these areas. The omentum was markedly thickened. Large fungating adenocarcinoma of 4 cm. diameter at the splenic flexure. Firm paracolic lymph nodes. Kidneys - bilateral pyelitis.

COMMENT:

It is notoriously difficult toimprove and maintain the general physical condition of patients with bowel fistulae. In addition, this patient was suffering from abscesses in both the lung and the pelvis. This would have rendered the task of improving and maintaining her condition doubly difficult. In spite of this, the wisdom of proceding with an operation that was not a dire emergency, in the face of the immediate pre-operative deterioration (for which in addition to the transfusion of plasma, vasopressor drugs were used) must be seriously questioned.

In these circumstances, the premedication ordered by the anaesthetist (Pethidine 100 mg.) must be regarded as foolishly heavy-handed. The same criticism applies to the use of thiopentone 300 mg. for the induction of anaesthesia. The fact that no obvious deterioration in the patient's B.P. occurred until after 1 hour of anaesthesia may possibly be regarded as exonerating both. However, this should be considered in relation to the administration of vasopressor drugs that had shortly preceded induction of anaesthesia.

A marked tachycardia was immediately apparent after induction of the anaesthesia. Though this may have been the result of toxic myocarditis, considered in conjunction with the patient's abdominal lesion and pre-operative vascular collapse, it more likely indicated a state of circulatory hypovolaemia. Blood transfusion was commenced after 30 minutes but was not pursued with any energy until marked deterioration had set in.

A further fault is evidenced in the management of this anaesthetic in relation to the adequacy of pulmonary ventilation. In addition to the right sided lung abscess, surrounding consolidation and left sided pleural effusion, autopsy showed the trachea and bronchi to contain purulent mucous. Yet no attempt was made by the anaesthetist to perform any form of bronchial toilette. This of itself must have led to some inadequacy of pulmonary ventilation, aside from any consideration of the manner in which IPPR was executed, and so have resulted in both anoxic anoxia and hypercarbia. The tachycardia observed may have been a reflection of this.

The probable under-estimation of blood loss at the operative site is another fault apparent in the anaesthetic technique. The nature of this patient's abdominal lesion, and the operative procedure

undertaken / ...

undertaken make it likely that the blood loss was considerably greater than that estimated and replaced by the anaesthetist (only 1 pint up to the time of cardiac arrest - most of this immediately prior to arrest, when the pulse had already become feeble).

The aetiology of the cardiac arrest appears to have been multiple:

(1) General toxicity,
(2) Hypovolaemia - before induction of anaesthesia and worsened by inadequate blood replacement during the operation,
(3) Anoxic anoxia from inadequate pulmonary ventilation.

Adequate blood replacement was still not instituted following the initial cardiac arrest, for which the treatment appears to have been correct and initially effective. The patient was later permitted to breathe spontaneously. In the presence of the pulmonary lesions, though oxygenation may have been adequate following the administration of oxygen only, respiratory acidosis will probably have resulted from inadequate volume of ventilation. This will have added to the effects of the metabolic acidosis which by now must have been gross.

Because of these criticisms, this anaesthetic and its management are regarded as significant contributory factors to the patient's death - in spite of the gravity of her condition before anaesthesia and the probable inevitability of her death.

PREVENTABILITY:

In view of the correctable faults identifiable in the anaesthetic management of this case, despite the gravity of the patient's pre-operative condition, this case is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
91.1.58	1	Possibly	< 24	Post-relaxant respiratory inadequacy. Acute haemorrhagic pancreatitis.	Yes

Name: Barend Malherbe Age: 55 Sex: M Race: E.

Disease: Acute abdomen ?Intestinal obstruction. Operation: Laparotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Extremely poor condition. There was gross abdominal distension. Mild dehydration which was treated by infusion of 1 litre 5% dextrose in water. B.P. 140/100 mm.Hg. Respiration 45/minute. Extremely dyspnoeic; atelectasis of the right lung base. Patient weighed 180 lbs.

PREMEDICATION:

Morphine gr. 1/6, Atropine 1/100. Gastric aspiration had been instituted 2 hours previously.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 500 mg. in divided doses, administered in a 15° head-up tilt, followed by 120 mg. gallamine. The patient was intubated orally and the anaesthesia was continued with nitrous oxide and oxygen administered via a circle absorption system, with intermittent minimal ether, by an IPPR technique. Induction of anaesthesia was uneventful. The gross distension of the abdomen resulted in an extremely low pulmonary compliance.

During opening of the abdomen, a supplementary dose of 40 mg. gallamine was given. The total dose was then 160 mg.

Laparotomy revealed an acute haemorrhagic pancreatitis with paralytic ileus. Further course of the anaesthetic was uneventful. The gut being extremely distended, the abdomen proved difficult to close. Closure resulted in gross abdominal tension. At the conclusion of operation, which took 90 minutes, neostigmine 2½ mg. was given preceded by Atropine 1.2 mg. Respiration, however, was inadequate with tracheal tug and IPPR was continued. Twenty-five minutes later respiration was still inadequate and a further 0.5 mg. neostigmine was administered, with little effect. IPPR was continued for a further 90 minutes. At this stage respiration was considered equal to the pre-operative level. Anaesthesia was discontinued. The patient regained consciousness and the endotracheal tube was removed. The patient was returned to the ward where he was seen 10 minutes later by the anaesthetist. It was noticed that the gross distension of the abdomen hampered respiration, but this appeared to be at the same level as pre-operatively and his colour was good. One hour after his return to the ward, the patient died suddenly.

AUTOPSY:

Bilateral basal pneumonia with bilateral pleural adhesions and small amounts of serosanguinous fluid in both pleural cavities. Acute haemorrhagic pancreatitis; the pancreas was enlarged, weighing 510 gm with numerous soft adhesions involving the under surface of the diaphragm, spleen, liver, pancreas and posterior abdominal walls. Peritoneal cavity contained serosanguinous fluid. Evidence of fat necrosis in mesentery and perinephric fat. Brain and meninges and cerebral vascular congestion. Smell of ether. Oesoohaguscontained yellowish mucous. Trachea and bronchi contained purulent mucus, also somewhat inflamed.

Heart ...

Heart and pericardial sac: heart enlarged, weight 545 gm. Slight amount of coronary atheroma. Liver pale and fatty. No obstruction to biliary tract.

COMMENT:

The probable immediate cause of this death was post-operative anoxic anoxia and hypercarbia , resulting from under-ventilation. The most obvious fault in the management of the anaesthetic was the failure of the anaesthetist to continue to provide, by means of IPPR, adequate pulmonary ventilation post-operatively. Though noting that the respiration was hampered by gross abdominal distension, the anaesthetist discontinued IPPR 90 minutes post-operatively. The lack of cyanosis is known to be a fallacious criterion in judging the adequacy of pulmonary ventilation in these circumstances.

The cause of this hypoventilation was twofold:-

(1) The gross abdominal distension together with bilateral basal pneumonia,

(2) The effects of curarisation or post-operative re-curarisation,
   (a) the metabolic acidosis known to accompany acute pancreatitis may have caused prolongation of the effects of the relaxant used,
   (b) poor excretion of gallamine due to low urinary output may have led to an element of re-curarisation.

The failure of the surgeon to effect surgical decompression of the gut is also open to criticism. The importance of the radical correction of metabolic acidosis with intravenous sodium bicarbonate was not as widely realised at this time (1958) as it is today.

PREVENTABILITY:

Whatever the cause of this patient's hypoventilation, the continuance of efficient IPPR would possibly have prevented his death.

NOTE:

It is interesting to note that two other patients in the group who died following post-relaxant respiratory inadequacy also suffered from acute pancreatitis. A profound metabolic acidosis is known to accompany this condition and this may well be responsible for the post-operative respiratory inadequacy. A third patient, suffering from acute pancreatitis, to whom no relaxant was administered also died of post-operative respiratory inadequacy. This patient, however, showed similar inadequacy before anaesthesia.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
92.1.58	2	No comment	< 24	Haemorrhage	Yes

Name: Stanley Muller. Age: 71. Sex, M Race: E

Disease: Gross melaena following anticoagulant therapy for myocardial infarction.

Operation: Partial gastrectomy.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

Following coronary thrombosis in 1954, and the extension of the infarct in 1958, the patient had been treated with Dindevan. Gross gastro-intestinal bleeding had now developed and the patient was in extremely poor condition. Pulse rate 120/minute, B.P. 95/50 mm.Hg; in cardiac failure. Basal crepitations at both lung bases. He had recently had acute bronchitis. 2 finger hepatomegaly was palpable; 2+ albuminuria was present.

PREMEDICATION:

10 pints of blood were transfused pre-operatively. Digoxin 1.25 mg. in divided doses and 40 ml. 10% solution of calcium gluconate but no atropine was administered.

ANAESTHETIC:

1 ml. 4% Xylocaine solution was injected trans-tracheally. Following pre-oxygenation, anaesthesia was induced with cyclopropane and oxygen. Oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide absorption system. A single dose of 80 mg. gallamine and the institution of an IPPR technique followed. This dose of relaxant was sufficient for the 100 minutes of the operation.

At the start of the operation there was an episode of severe hypo-tension due to (1) fresh haemorrhage, (2) opening the peritoneum, and (3) institution of IPPR. This was rapidly corrected by rapid transfusion of 2 pints blood. During the remainder of the operation 1 further pint of blood was given. A partial gastrectomy was performed. At the conclusion of operation, closure of the peritoneum was facilitated by the administration of cyclopropane. Curarization was reversed by the administration of 1 mg. neostigmine, preceded by atropine gr. 150. Following the conclusion of operation, the patient soon regained consciousness. Respiration was spontaneous and adequate. He died 7 hours post-operatively from what appeared to be continued haemorrhage.

AUTOPSY:

Partial gastrectomy had been performed, the lower third of the stomach was removed surgically and anastomosed to ileum, sutures intact. Stomach: full of blood clot and free blood. There were numerous bleeding erosions from the mucous membrane and also at the site of anastomosis. The gut contained much blood clot. Right sided basal pneumonic consolidation with haemopurulent mucus in trachea. Heart was enlarged weighing 700 gm. Gross left ventricular hypertrophy and coronary artery atherosclerosis. Viscera and mucous membranes not pale, except the kidneys.

COMMENT:

This patient died from his pre-existing disease. Cardiac failure due to severe ischaemic heart disease worsened by severe and probably continuing haemorrhage was the cause and the anaesthetic was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
93.1.58	2	No comment	< 24	Intra-cerebral haemorrhage	No

Name: Jean Simenoff Age: 51 Sex: F Race: E

Disease: Intracerebral haemorrhage.

Operation: Carotid angiography. Burrhole craniotomy and evacuation of intra-cerebral haematoma.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor general condition. B.P. 130/80 mm.Hg, pulse rate 140/minute. Respiration 40/minute. Diffuse rhonchi over both lungs. She was deeply comatose.

PREMEDICATION:

Atropine gr. 1/100 administered 45 minutes before the operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered via a semi-open system. Following topical analgesia of the larynx, oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and ether administered by the same method, with spontaneous respiration.

During the operation, the B.P. fell from 160 mm.Hg to 120 mm.Hg and the pulse rate settled from 140 to 110/minute, while the respiration dropped from 40/minute to 20/minute. At operation, which lasted 90 minutes, an intracerebral haematoma was evacuated. The patient was returned to the ward comatose and died in coma 13 hours after operation.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of intracerebral haemorrhage. Anaesthesia was not contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY.
94.1.58	2	No comment	< 24	Cardiac failure	No

Name: Maggie Sullavan. Age: 57 Sex: F Race: C

Disease: Duodenal obstruction, carcinoma of the third part of the duodenum.

Operation: Laparotomy; resection of duodenum.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor: the patient was cachectic; haemoglobin 10.5 gm.%. B.P. 100/85 mm.Hg with some degree of cardiomegaly. She had a chronic productive cough and, on examination, coarse rhonchi in both bases. She had lost much weight. One month previously she had had a cholecystectomy followed by a dehiscence of the abdominal wound. She was on gastric suction with intravenous fluid replacement.

PREMEDICATION:

Pethidine 100 mg., atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 350 mg., nitrous oxide, oxygen and ether administered via a circle absorption system. Gallamine 100 mg. was given, the trachea was sprayed with a topical anaesthetic and oral intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and minimal ether via a circle absorption system by an IPPR technique. There was some drop in the B.P. following induction of anaesthesia, but this was adequately controlled by the administration of 6mg. methyl amphetamine.

During the course of the operation, 2 pints blood were transfused. A further 20 mg. gallamine was given during the procedure. After the initial drop in B.P., the subsequent course of the anaesthetic and operation - which lasted 145 minutes - was untoward. At the conclusion of the operation 0.5 mg. neostigmine preceded by 1/100 gr. atropine restored the respiration to normal. However, following the administration of neostigmine, the pulse rate dropped to 60/minute. A further 1/100 gr. atropine was given and the pulse rate returned to its previous level of 120/minute. At the conclusion of the anaesthetic the patient rapidly regained consciousness, her B.P. at this stage being 100 mm.Hg systolic and the pulse rate 120/minute. The B.P. remained stable until 8 hours post-operatively, when it commenced falling. An intravenous infusion of noradrenaline was started and this restored the B.P. to 100 mm.Hg, where it was maintained for a further 2 hours, at which stage auricular fibrillation commenced. Digoxin 1.5 mg. was given but the patient died ½ hour after the onset of auricular fibrillation, 10 hours after the operation.

AUTOPSY:

No autopsy.

COMMENT:

This patient had recovered from the anaesthetic, was awake and conscious and breathing adequately before the final deterioration set in. Death appears to have been due to cardiac failure. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group.	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY.
95.1.58	2	No comment.	< 24	Cerebral lacera-tions.	Yes

Name: Pricilla Pearce. Age: 2 Sex: F Race: C

Disease: Compound depressed fracture of the skull; cerebral lacerations.

Operation: Elevation of compound depressed fracture; debridement of brain and wound.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Following a head injury, the patient was unconscious. Pulse rate 160/minute, B.P. 90/60 mm.Hg.

PREMEDICATION:

Atropine gr. 1/100

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether. During induction the patient vomited. This was adequately aspirated with a sucker and no vomitus was inhaled. Oral intubation was performed. Anaesthesia was maintained throughout the operation with nitrous oxide and oxygen, the patient breathing spontaneously, using a modified T-piece.

During operation, which lasted 75 minutes, the course of the anaesthetic was uneventful. Post-operatively, following the discontinuation of the anaesthetic, the state of consciousness was as it had been before operation. Respiratory failure ensued 3 hours post-operatively. The patient vomited, stopped breathing and died. The vomiting appears to have been terminal.

AUTOPSY:

Gross cerebral damage. The lungs were normal; there was no inhaled vomitus.

COMMENT:

This death was due to cerebral laceration. Anaesthesia was non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
96.1.58	2	No comment	< 24	Undetermined. ?Coronary insufficiency.	Yes

Name: Pieter Engelbrecht. Age: 52 Sex: M Race: E

Disease: Haematemesis; carcinoma of the stomach. Operation: Gastrectomy and splenectomy.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

The patient had had a severe haematemesis. On admission the haemoglobin concentration was 7.5 gm.%. 9 pints blood were transfused before operation and the patient's B.P. was 130/70 mm.Hg. There was no hepatomegaly but the patient was slightly jaundiced.

PREMEDICATION:

Pethidine 75 mg., atropine 0.6 mg.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 300 mg. followed by 120 mg. gallamine. While this took effect cyclopropane and oxygen were administered and the patient was then intubated with a No.10 cuffed endotracheal tube. An IPPR technique was instituted immediately with nitrous oxide and oxygen administered via a circle absorption system with minimal ether. This latter was later discontinued and pethidine 50 mg. was given in divided doses.

During the gastrectomy and splenectomy, blood loss was estimated at 1200 ml. which was replaced during the procedure by the transfusion of 1800 ml. compatible blood. During the operation, lasting 2¼ hours, a further 140 mg. gallamine was administered. The course of the operation and anaesthetic was untoward. B.P. remained at 120 mm.Hg systolic with a pulse rate varying around 100/minute. At the conclusion of the operation atropine 1.2 mg. was given followed by 2.5 mg. neostigmine in divided doses. Adequate spontaneous respiration returned and the patient regained consciousness promptly at the end of the anaesthetic. Three hours post-operatively he complained loudly of discomfort in the chest, asked to sit up and then suddenly stopped breathing. Artificial respiration and cardiac massage were performed, to no avail.

AUTOPSY:

Jaundice. Evidence of gastrectomy and gastroduodenostomy and splenectomy. 200 ml. dark blood in abdominal cavity. No evidence of leak from suture line.

COMMENT:

Although unproven by autopsy, this death appeared clinically rather like one resulting from coronary insufficiency. The patient was in a good condition throughout the anaesthesia and operation; at the conclusion of anaesthesia, the respiration was adequate and he recovered consciousness promptly. His condition was well maintained in the ward 3 hours after operation. The anaesthetic does not appear to be contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
97.1.58	1	Possibly	< 24	Inhalation of vomitus. Continuing haemorrhage.	Yes

Name: Walter Johannes. Age: 33 Sex: M Race: C

Disease: Traumatic laceration of liver. Operation: Laparotomy; suture of lacerated liver.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Fair. Pulse rate 110/minute, respiration 20/minute, B.P. 130/80 mm. Hg. To get the patient into a satisfactory state, 11 pints blood had been transfused. There was no abnormal tenderness, distension or vomiting.

PREMEDICATION:

Omnopon ⅓ gr. Scopolomine 1/150gr.

ANAESTHETIC:

Anaesthesia was induced with thiopentone. When 100 mg. had been given, the patient suddenly vomited copiously - about 4 pints black fluid. Oral intubation with a cuffed endotracheal tube was rapidly performed and a large stomach tube was passed through which a further 3 pints black liquid was aspirated from the stomach. At this stage the patient was moving and had to be restrained. He became cyanosed and this, with audible coarse rhonchi, indicated inhalation of some vomitus. This was confirmed by tracheal and bronchial toilette through the endotracheal tube. A fair amount of vomitus was removed by suction aspiration. Following bronchial toilette, the cyanosis disappeared and the patient's colour improved. B.P. was 140 mm.Hg systolic, pulse rate 120/minute and good air entry was audible in all areas of the chest, with no further moist sounds. No further vomitus could be aspirated from the trachea or bronchi. Anaesthesia was continued with nitrous oxide, oxygen and ether, administered via a circle absorption system. Abdominal relaxation was obtained with divided doses of gallamine, totalling 200 mg. An IPPR technique was instituted.

At laparotomy the liver was found to be severely lacerated with a large amount of blood in the peritoneal cavity. During resuture of the liver, the operation lasting 120 minutes, a further 3 pints blood were transfused. The patient's condition remained reasonably good during this time. However, at the conclusion of the procedure bleeding continued. Peritoneal drains were inserted and the abdomen was closed. Spontaneous respiration was restored adequately by the administration of 2.5 mg. neostigmine preceded by atropine 1/100 gr. Following discontinuance of the anaesthetic, the patient regained consciousness but rapidly became cyanosed. Air entry was auscultated to be poor over the left lung. The consultant anaesthetist was called and bronchoscopy was performed; a further 5 cc. vomitus was aspirated. Shortly thereafter, the patient developed mediastinal emphysema. An X-ray chest performed on the operating table showed mediastinal emphysema but no atelectasis of either lung. An endotracheal tube was inserted and the patient returned to the ward with this tube in situ. The patient was still bleeding from the abdominal drains, blood meantime being transfused. There was definite moderate respiratory difficulty. The patient wasplaced in an oxygen tent with humidification and "Alevaire". Frequent bronchial and tracheal toilette through the endotracheal tube continued, producing a few ccs. of aspirated vomitus each time.

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The surgical emphysema became steadily worse. One hour after return of the patient to the ward, a thoracic surgeon was consulted. While further X-rays were taken there was a sudden deterioration in the patient's condition and another bronchoscopy was rapidly performed. However, this revealed that the bronchi were clear. Marked respiratory difficulty ensued rapidly and mechanical artificial ventilation with oxygen was instituted. Within 5 minutes, cardiac arrest had occurred. Cardiac massage was of no avail and, after a further 2 minutes, resuscitative measures were abandoned.

AUTOPSY:

Mild mediastinal emphysema. No marked abnormality of the lungs, pleura or great vessels. A small amount of blood was found free in the peritoneal cavity. Large surgical drain in situ. Liver severely lacerated, the anterior surface sutured but lacerated posterior was not sutured.

COMMENT:

Although a good case can be made for this death having resulted from continued bleeding from a severely lacerated liver, the posterior surface of which was not sutured, respiratory difficulty for which little reason could be found at autopsy features prominently in the clinical post-operative course. This respiratory difficulty originated when the patient inhaled vomitus during the induction of anaesthesia, this episode resulting in a period of cyanosis at that time. Tracheo-bronchial toilette appeared sufficient at this stage, especially in view of the urgency of surgery. Later, however, signs of left bronchial obstruction indicated the need for bronchoscopy. Some mucosal damage must have resulted from this instrumentation, since mediastinal emphysema followed. Though subsequent bronchoscopy showed the bronchi to be clear, respiratory difficulty persisted and worsened. This means that there was some obstruction to the finer bronchioles, from the inhaled vomitus or from possible mucosal oedema, or as a result of bronchiolar spasm (Mendelsohn syndrome). That this was not revealed by autopsy may be due to the fact that (1) it was not assiduously sought, (2) death having followed shortly, insufficient time had elapsed for the formation of a frank atelectasis (the original obstruction had occurred when the patient was breathing air).

The respiratory difficulty was severe enough to result in cyanosis post-operatively, and even if death is ascribed to continuing haemorrhage, the inhalation of vomitus following induction of the anaesthesia must be considered a definite contributory factor.

The failure of the anaesthetist to have gastric lavage performed before operation is open to criticism, though the urgency of surgery may be considered an extenuating circumstance. The failure to put the patient in the Trendelenburg position following the vomiting is also open to criticism, as is the failure to perform bronchoscopy before the commencement of operation. However, with regard to the latter, the clinical observation of good air entry on both sides of the chest, together with the cessation of cyanosis following bronchial toilette through an endotracheal tube (when considered in relation to the urgency of surgery) may be regarded as extenuating circumstances.

A notable omission in treatment was that of the administration of hydrocortisone, usually found to be of help in cases of Mendelsohn type syndromes. The anaesthetist's management is regarded as a significant causative factor in this patient's death. Continuing haemorrhage from the lacerated liver was also a greatly contributory factor.

PREVENTABILITY:

In view of (1) the omission of gastric lavage pre-operatively, (2) the failure of the anaesthetist to put the patient in the Trendelenburg

position / ...

position following the vomiting episode, (3) the failure to perform a bronchoscopy immediately after the inhalation of vomitus, and (4) the failure to administer hydrocortisone, this death is regarded as possibly preventable - though haemorrhage from the lacerated liver also played a significant role in the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
98.1.58	3	No comment	ORD	Haemorrhage Cardiac arrest.	Yes

Name: Miriam Jacobs. Age: 13 Sex: F Race: C

Disease: Post-operative haemorrhage following repair of ventricular septal defect on cardiopulmonary bypass. Operation: Thoracotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was moribund. The previous day she had undergone the repair of a ventricular septal defect on cardiopulmonary bypass. Continuous intrathoracic haemorrhage occurred post-operatively, continuing throughout the night. In spite of apparently adequate blood replacement, the B.P. continued to fall. It was decided to perform a thoracotomy and explore for the source of the bleeding. At the commencement of the anaesthetic, the pulse rate was 160/minute, respiration 60/minute. The patient was markedly dyspnoeic, cyanosed and shocked. B.P. was unrecordable.

PREMEDICATION:

No premedication.

ANAESTHETIC:

Following pre-oxygenation, anaesthesia was induced by inhalation of cyclopropane and oxygen with gradually added ether. Oral intubation was performed. Shortly after this, cardiac arrest occurred. Thoracotomy was performed immediately and cardiac massage instituted. Within 2 minutes cardiac action returned. 1:100,000 solution of adrenaline was injected into the left ventricle and the heart continued to beat for 9 minutes, when cardiac arrest again occurred. Further massage resulted in the restoration of a heart beat within 1 minute, but this was shortly followed by ventricular fibrillation. Use of an electrical defibrillator, sodium bicarbonate, adrenaline, glucose, calcium chloride, and ultimately the use of the pacemaker, were all to no avail. Death was presumed 2 hours after the commencement of the anaesthetic.

AUTOPSY:

Operation on the heart with sutured surgical incisions of thorax and legs, removal of portion of the sternum and a few ribs. Incisions had been made in the chest for the insertion of drainage tubes. There was a small amount of bleeding in the lung sacs. Very large heart showing sutured surgical incisions. A small amount of bleeding into the left subdural space with clotted blood. No sign of head injury. Stomach empty.

COMMENT:

Although the cardiac arrest which preceded this patient's death occurred shortly after endotracheal intubation and before the commencement of surgery, and one may be tempted to inculpate the anaesthetic, intubation and vagal stimulation, this patient was all but moribund at the induction of anaesthesia and death must be attributed to the continued post-operative haemorrhage which had followed open heart surgery. It may well be that anaesthesia was in fact the last straw, but this cannot be considered culpably or avoidably contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
99.1.58	3	No comment	< 24	Post-valvotomy cerebral embolism.	Yes

Name: Daphne Ahlschlager Age: 27 Sex: F Race: E

Disease: Mitral stenosis. Operation: Mitral valvotomy.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Extremely poor operative risk. She suffered from a tight mitral stenosis, grade 4 disability. She was 5 months' pregnant and in persistent cardiac failure, in spite of adequate digitalisation and diuretic therapy. She had albuminuria. Because of her pregnancy and worsening cardiac state, in spite of adequate medical therapy, it had been decided to proceed with a mitral valvotomy.

PREMEDICATION:

Sodium seconal gr. 3, administered orally 3 hours before operation. Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

On arrival in theatre, immediately before anaesthesia was commenced, the B.P. was 100/60 mm.Hg. She had auricular fibrillation with a pulse rate of 120/minute. Anaesthesia was induced with thiopentone 100 mg. followed by cyclopropane and oxygen, administered via a closed circuit absorption system for 3 minutes. The larynx was anaesthetised topically and the patient was intubated orally. Following this the B.P. rose to 120 mm.Hg systolic and subsequently fell to 100 mm.Hg systolic. Anaesthesia was continued with nitrous oxide and oxygen (40%) with a trace of ether, via a circuit absorption system with an IPPR technique. At the commencement of operation the trace of ether was discontinued, anaesthesia being maintained with nitrous oxide and oxygen by IPPR via the same circle absorption system. An initial dose of 15 mg. dTc was administered as a relaxant. The soda lime used in the circle absorption system was fresh and ventilation was adequate and easily performed throughout.

Until the time of valvotomy, the B.P. was maintained at 100 mm.Hg systolic with a pulse rate of 120/minute, the rhythm being auricular fibrillation. Mitral valvotomy was performed 20 minutes after the start of operation and was well tolerated by the patient, the B.P. dropping only for the briefest moment and then rising to above the pre-valvotomy level, to 125 mm.Hg systolic, the pulse rate increasing to 140/minute. Blood loss throughout the procedure was minimal and it was only necessary to replace 400 ml. blood. Ten minutes after valvotomy (30 minutes after the initial 15 mg. dTc was given) the patient commenced respiratory efforts and a further 5 mg dTc was administered. Throughout the closing stages of the operation the B.P. remained at a level of 130 mm.Hg systolic, and the pulse rate at 140/minute, auricular fibrillation continuing. The patient's colour remained good at all times. Closure of thechest was completed 1¾ hours after the commencement of surgery and spontaneous respiration was allowed to return, occurring shortly but was of a gasping type with a marked tracheal tug. At this stage, neostigmine 2.5 mg preceded by atropine gr. 1/75 was administered in divided doses. This had no effect on the respiration, though at this stage the patient showed a lash reflex. Anaesthesia was discontinued and respiratory assistance with pure oxygen continued.

Ten minutes after the conclusion of the operation, while still on oxygen, the level of coma appeared to deepen, the pupils dilated unequally, the right being larger than the left, and at this stage a degree of bronchospasm became evident. This was little relieved by administration of atropine, aminophyllin 250 mg. and a neosynephrine spray down the endotracheal tube. To eliminate the possibility

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that the respiratory difficulty might be due to either hypo- or hypercarbia, the patient was first well over-ventilated with pure oxygen on the circle absorption system with the absorber in circuit, and when this did not help, she was ventilated for a while with pure oxygen on the circle absorption system with the absorber out of circuit. Neither manoeuvre made any difference to the respiration.

One hour after the conclusion of the operation, in an attempt to reduce the pulse rate of 140/minute, a dose of Cedilanid 0.2 mg. was administered. This had no effect on the pulse rate. An X-ray of the thorax taken at this stage revealed both lung fields to be clear, the lungs well expanded and the heart dilated. The patient was kept in theatre for 2 hours post-operatively but there was no change in her condition. Though the respiration was of a gasping type with a tracheal tug, it appeared to be of adequate volume and the patient maintained a good colour subsequently, on breathing air. By this time an attending physician had diagnosed cerebral embolism following mitral valvotomy. Two hours after the end of the operation the patient was returned to the ward and 10 minutes later she stopped breathing suddenly and died.

AUTOPSY:

Evidence of mitral valvotomy and cardiac failure. No cerebral embolism demonstrated.

COMMENT:

The evaluation of this case is very difficult. Though little fault can be found with the anaesthetic technique used, the patient failed to regain consciousness and did not breathe normally following its conclusion. To exonerate the anaesthetic as a significant contributory factor in this death, there should be lack of evidence of incidents in the conduct of the anaesthesia that could lead to this state, and there should be some other reasonable explanation to account for it. Factors in the anaesthetic management that might account for the state present at the end of the operation would be (1) over-dosage of anaesthetic drugs, (2) gross cerebral damage from anoxia, either anoxic or ischaemic, from circumstances under the control of the anaesthetist, (3) gross hypercarbia or metabolic acidosis, (4) prolonged curarization. From the clinical account there is no evidence of any of these.

The relevant facts are (1) the patient was in a very poor state before anaesthesia, having severe mitralstenosis and pulmonary hypertension; she was pregnant and in persistent cardiac failure with auricular fibrillation despite digitalisation and diuretic therapy; (2) induction of anaesthesia was well tolerated; (3) ventilation and carbon dioxide absorption appear to have been adequate throughout; the patient's colour was good at all times and there was no evidence of cyanosis; (4) throughout the procedure the anaesthesia was maintained with nitrous oxide and oxygen alone in mixtures of 40-50%, following the use of a small amount of ether administered for 5 minutes immediately preceding the commencement of surgery; (5) the dose of dTc was not excessive, the first dose of 15 mg. requiring supplementation after 30 minutes; this is usual; the supplementary dose of 5 mg. was small and preceded the end of operation by one hour; this total dose of 20 mg. in the 2 hours of the operation is rather smaller than the amount usually required for this type of surgery; (6) the B.P. was well maintained throughout surgery and recovered rapidly after valvotomy; (7) following the conclusion of surgery, the administration of neostigmine 2.5 mg. made no difference to the gasping respiration that returned spontaneously during the concluding stages of the operation; (8) though the lash reflex initially returned, the patient became rapidly and progressively more comatose while being ventilated with pure oxygen; (9) raising and lowering the pCO_2 by ventilating the patient both

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with and without carbon dioxide absorption had no effect on the character of the respiration, or on the state of consciousness; (10) following the sudden deepening of the patient's state of consciousness at the end of operation while being ventilated with oxygen, the pupils became unequally dilated.

Because of the above signs, the attending physician diagnosed the condition as one of cerebral embolism following valvotomy. As air embolism is most unlikely during mitfal valvotomy, because of the high left auricular pressure, it was presumed to be due to thrombo-embolism. The fact that this was not displayed at autopsy does not exclude this possibility: the clots may have been very small and mushy, and it may be extremely difficult to demonstrate adequately in the smaller cerebral vessels. For this reason it is considered that the anaesthetic itself was not a significant causal factor in this death, but that death resulted from cerebral embolism following mitral valvotomy.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
1001.58	1	No comment	<24	Ventricular tachycardia	Yes

Name: Dorothy Nelson Age: 51 Sex: F Race: E

Disease: Ureteric calculus. Operation: Cystoscopy and urethrotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient was grossly obese and suffered from diabetes mellitus controlled by diet. In the past she had had many operations: colostomy, repair of subsequent colostomy hernia, cholecystectomy, left nephrectomy for multiple kidney stones and pyelonephritis. No anaesthetic difficulty was experienced with any of these operations. For some years she had suffered from gross hypertension, B.P. 220/110 mm.Hg and concomittant cardiomegaly. One week previous to the present anaesthetic, she was admitted to hospital with a calculus in the right ureter and calculus anuria. During the week the right ureter was catheterised under general anaesthesia twice, at intervals of two days. On each occasion, though the stone was shifted, attempts to remove it were unsuccessful. On each occasion anuria recurred as soon as the ureteric catheter was removed 24 hours after cystoscopy. On the first occasion an epidural anaesthetic and on the second a general anaesthetic (thiopentone, nitrous oxide oxygen and ether) was administered without any untoward effect. After the second cystoscopy, blood urea was 72 mg.%, serum potassium 4.5 m.Eq./litre, Na 138 m.Eq/litre. Before this anaesthetic she again had been anuric for 36 hours.

PREMEDICATION:

Pethidine 100 mg., atropine gr. 1/100 .

ANAESTHETIC:

Anaesthesia was induced with thiopentone in divided doses up to total 300 mg. This produced no fall in B.P. Anaesthesia was then continued with nitrous oxide and oxygen via a Magill semi-open circuit, with the patient breathing spontaneously. Ether was gradually added to the mixture. The patient commenced hyperventilating. For the first 20 minutes following induction of anaesthesia, B.P. and pulse rate remained stable. The heart rate then suddenly increased to 200/minute which caused the B.P. to drop from 220 to 120 mm.Hg systolic. An ECG recorded at this stage showed the arrhythmia to be ventricular tachycardia, with grossly abnormal complexes, at a rate of 200/minute. The B.P. dropped to 80 mm.Hg systolic after a further 5 minutes. Procaine amide totalling 2 gm. at the rate of 100 mg. per minute was administered intravenously under ECG control. This slowed the pulse rate to 112/minute and the B.P. rose to 120 mm.Hg systolic. A noradrenaline infusion, 2 microgm./ml. was commenced.

Cystoscopy showed the stone impacted in the ureteric meatus. At the conclusion of the operation, which lasted 50 minutes in all, the anaesthetic was discontinued and the patient recovered consciousness. However, she was restless and 50 mg. pethidine was given intravenously; 10 minutes later a further 25 mg. pethidine was given as sedation. The patient died 5 hours post-operatively in severe cardiac failure, noradrenaline having been necessary to maintain the blood pressure throughout this time.

AUTOPSY:

Left kidney removed and right kidney enlarged and showed microscopic appearance of chronic nephritis plus hydronephrosis. The heart was enlarged to twice its normal size, coronary arteries being almost occluded by atherosclerosis. There was no evidence of degenerative changes in the liver.

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COMMENT:

Though no fault is immediately apparent in the anaesthetic technique, the anaesthetist noted the onset of tachycardia and hyperventilation during the induction of anaesthesia. The onset of ventricular tachycardia may well have been related to rapid changes in respiratory acid-base state following the induction of anaesthesia, in the presence of a probably raised serum potassium level because of anuria and ventricular irritability due to gross ischaemie heart disease. Ventilatory anoxia during the tachypnoea in this very obese patient is another possible factor that may have triggered the ventricular tachycardia.

Whatever the precise cause of this episode, the patient had had one other similar general anaesthetic in similar circumstances two days previously, without ill effects. One must conclude that the conditions produced by this general anaesthetic were a significant factor in causing this ultimately fatal ventricular arrhythmia. Though the patient regained consciousness after anaesthesia, the anaesthetic is regarded as a significant contributory factor to the death.

PREVENTABILITY:

As it is not possible to diagnose the precise cause of this ventricular arrhythmia, and no gross fault is immediately apparent in the anaesthetic management, no verdict can be arrived at with regard to the preventability of this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
101.1.58	2	No comment	< 24	Cerebellar abscess.	Yes

Name: Jennifer Lekeur Age: 15 Sex: F Race: C

Disease: Cerebellar abscess. Operation: Right occipital burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient had been admitted 4 days previously with depressed level of consciousness, neck stiffness and otitis media. A right radical mastoidectomy had been performed. No improvement had followed. The patient's level of consciousness had become deeper. She was now extremely drowsy and disorientated with marked neck stiffness. Temperature 99°F, B.P. 110/80 mm.Hg.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, delivered via a Magill circuit, with spontaneous breathing. Following topical analgesia of the larynx, oral intubation was performed and anaesthesia was maintained with the same agents, the patient breathing spontaneously. The course of the anaesthetic was entirely uneventful, the B.P. remaining steady at a level of 110/80 mm.Hg with a pulse rate of 96/minute.

Right occipital burrhole craniotomy was performed and approximately 7 cc. pus aspirated from the cerebellar abscess. The operation took 50 minutes. At the conclusion of the procedure and discontinuance of anaesthetic, the patient returned to the same level of consciousness as she had been previously. She died 20 hours post-operatively.

AUTOPSY:

Evidence of recent mastoidectomy. There was a right cerebellar abscess and marked oedema of the right cerebellar hemisphere with displacement of structures to the left.

COMMENT:

This death was due to the cerebellar abscess. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
102.1.58	3	No comment	ORD	Rupture of abdominal aortic aneurysm	Yes

Name: Harry Jagger Age: 65 Sex: M Race: E

Disease: Ruptured abdominal aortic aneurysm. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Very poor. The patient was severely collapsed. B.P. approximately 70 mm.Hg systolic, pulse rate 140/minute. On arrival in theatre the patient complained of a severe exacerbation of the pain previously felt, and simultaneously the B.P. became irrecordable. A rapid transfusion of 2 pints blood made no difference to the B.P. It was decided to induce anaesthesia and proceed with the operation, in an attempt to control haemorrhage from the aorta.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by 50 mg. succinylcholine. Oral intubation was performed and IPPR instituted through a circle absorption system, using nitrous oxide and oxygen, with minimal ether vapour. During preparation of the abdomen for operation, it was noticed that abdominal distension was increasing visibly. A total of 9 pints blood was given at maximum speed via 2 drips, but this did not appear to keep pace with the obvious internal bleeding. At laparotomy, gross aortic bleeding was uncovered, the circulation failed and the patient died. Cardiac massage via a thoracic incision was performed by a second surgeon while the first surgeon tried to control the aortic bleeding. This heroic effort was to no avail.

AUTOPSY:

Haemoperitoneum with a large ruptured aneurysm of the abdominal aorta (rupture 5 x 3 cm. in size). The aneurysm itself extended upwards for 5 inches from the bifurcation of the aorta. The whole of this aorta showed extensive atheroma. The aneurysm was surrounded by omentum and blood clots. All viscera very pale. There was a smell of ether in the body cavities. Stomach contained brownish fluid. Lungs collapsed. Heart showed a very slight degree of coronary artery atherosclerosis.

COMMENT:

Death was clearly due to massive uncontrollable haemorrhage from a ruptured abdominal aortic aneurysm. Death was clearly inevitable. In that death occurred while the patient was anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
103.1.58	2	No comment	< 24	Cerebellar abscess.	No

Name: Esther Amsterdam. Age: 50 Sex: F Race: C

Disease: Right cerebellar abscess. Operation: Posterior fossa burrhole craniotomy and drainage of abscess.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

This patient had had a right mastoidectomy 10 days previously. However, following the operation her condition did not improve and she developed signs of a cerebellar lesion. Her present condition was fair but was deteriorating. Pulse rate 108/minute, B.P. 140/80 mm.Hg. The patient was conscious.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 250 mg. followed by 50 mg. succinylcholine, using an IPPR technique. Topical analgesia of the larynx was followed by oral intubation. Anaesthesia was continued with nitrous oxide and oxygen with a trace of ether vapour, delivered via a Magill circuit. The patient was allowed to breathe spontaneously.

Throughout the operation the patient's condition was unchanged and the course of anaesthesia was completely uneventful. B.P. was maintained at 100mm.Hg systolic throughout. At the conclusion of theoperation, which took 105 minutes, anaesthesia was discontinued. The patient rapidly recovered consciousness. 5 hours after operation her pulse rate started rising sharply, respiration commenced failing and she died an hour later.

AUTOPSY:

No autopsy.

COMMENT:

This death was due to the patient's pre-existing cerebellar pathology, for which the operation was performed. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
104.1.58	1	Possibly	ORB	Respiratory obstruction Anoxic anoxia. Cardiac arrest.	Yes

Name: Caroline Engelbrecht. Age: 2½ Sex: F Race: C

Disease: Gross bronchiectasis. Pulmonary abscess. Operation: Bronchogram.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Extremely poor condition. This child had an empyema, consolidation of the lung, bronchiectasis and pulmonary abscesses on the right side. Previous to anaesthesia, she had a swinging temperature. On admission she had been grossly anaemic - haemoglobin 5 gm.%. In the days preceding the operation blood transfusion had been given and the haemoglobin was 10 gm.% at this stage. Though the patient's condition was poor, it was decided that a bronchogram was necessary for accurate pre-surgical anatomical diagnosis. In view of the child's age, a general anaesthetic was considered necessary.

PREMEDICATION:

Atropine gr. 1/150 was given 45 minutes before operation.

ANAESTHETIC:

Anaesthetic was administered via an infant's T-piece bag and mask. This was initially held well away from the face and slowly lowered onto the face, taking approximately 5 minutes. Halothane in a concentration increasing fairly rapidly from 0.5 to 2% was given with nitrous oxide and oxygen. Shortly after the mask had been placed on the patient's face, coughing commenced. The mask was immediately removed, a laryngoscope passed and the larynx inspected. This revealed a large amount of mucopus. This was removed with a sucker. The larynx was then sprayed with a topical analgesic and at this stage the patient became grossly cyanosed. An IPPR technique was instituted with pure oxygen, using a face mask, but this produced no improvement. All that occurred was that the stomach became distended by the oxygen. Complete respiratory obstruction was apparent. The patient rapidly became apnoeic. An endotracheal tube was passed and IPPR instituted. The pulse rate at this stage had slowed to 60/minute. On institution of IPPR moist sounds were audible. Bronchial toilette with a suction catheter passed down the endotracheal tube aspirated approximately 10 cc. thick mucopus. At this stage, 3 minutes after the commencement of coughing and 2 minutes after the onset of gross cyanosis, cardiac arrest occurred. In view of the grave pulmonary disease and the presence of gross anoxic anoxia, the surgeon felt that cardiac massage would be of no avail. This was thus not performed.

AUTOPSY:

The right lung was consolidated in toto. There were multiple pulmonary abscesses. The right pleural space was obliterated. The right bronchus was completely obstructed by mucopus. The left lung appeared normal.

COMMENT:

This patient died of cardiac arrest secondary to anoxic anoxia resulting from gross pulmonary disease together with bronchial and tracheal obstruction from pus from multiple lung abscesses. These events were precipitated by the induction of general anaesthesia.

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One might question the wisdom of submitting this child to general anaesthesia at all. However, it is difficult to see how bronchography could have been performed in so young a child without general anaesthesia.

Good pre-operative physiotherapy and postural drainage may have resulted in a happier outcome. The critical period of anoxia followed immediately after the anaesthetist, having aspirated mucopus from the pharynx, sprayed the larynx with 4% Xylocaine. At this stage he was unable to inflate the lungs because of complete respiratory obstruction. The completeness of this obstruction suggests something more than the mucopus subsequently aspirated from the trachea and bronchi by way of the endotracheal tube. The source of the obstruction at this stage may well have been laryngospasm, precipitated by stimulation of the sucker tip in the pharynx and the mechanical irritation of xylocaine sprayed on the larynx. Anaesthesia was light at this stage. It is known that mechanical stimulation of the larynx at this stage of anaesthesia, even with Halothane anaesthesia, can cause laryngospasm. The irritability of this child's larynx, the result of chronic respiratory tract infection, would have rendered this even more likely. The failure of the anaesthetist to notice gross laryngeal spasm at the time of intubation will have been due to either the commencing action of the xylocaine spray and/or the preterminal relaxation of the larynx that follows severe anoxia. In this child's poor pre-anaesthetic state, the period of anoxia that followed complete respiratory obstruction proved critical. Though suction aspiration of the pharynx was necessary at this stage, the spraying of the larynx in this light plane of anaesthesia was an error of judgment.

Also in view of the gravity of this child's pre-anaesthetic respiratory state, the administration of oxygen for a period before the induction of anaesthesia would have been a relevant safety factor. Its omission is considered faulty. Though this child posed a severe challenge to the anaesthetist, the anaesthetic management is considered a significant contributory factor to the death of the child.

PREVENTABILITY:

Because (1) the fatal anoxia is thought to have been precipitated inter alia by the manoeuvres of the anaesthetist, (2) pre-oxygenation was omitted, and (3) better physiotherapy and postural drainage may have resulted in a condition safer for anaesthesia, this death is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
105.1.58	2	No comment	< 24	Uncontrolled haemorrhage. Lacerated liver.	No.

Name: G. Adams Age: 21 Sex: M Race: C

Disease: Traumatic rupture of liver. Operation: Laparotomy. Suture of lacerated liver.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Following a car accident, this patient was in a state of severe oligaemic shock and moribund before operation. Pulse rate 110/minute. B.P. 90 mm.Hg systolic. The abdomen was distended with blood from internal haemorrhage. Auscultation of the lungs revealed scattered rhonchi on both sides of the chest. Four pints blood were transfused under pressure immediately before operation.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, preceded by a period of 3 minutes' pre-oxygenation. Succinylcholine 50 mg. was administered and oral intubation was performed with a No.10 cuffed endotracheal tube. Anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system by an IPPR technique, a trace of ether vapour being added to this mixture. A total of 140 mg. gallamine was given for relaxation during the operation.

During laparotomy it was found to be surgically impossible to stop the haemorrhage from the extremely lacerated liver, even after a right thoracotomy had been performed. Eventually attempts to stop the haemorrhage were abandoned and the abdomen and chest were closed after 185 minutes. At the conclusion of operation 2 mg. neostigmine was given preceded by atropine 1/100 gr. Normal respiration returned and the patient regained consciousness 30 minutes after the conclusion of the operation. The patient died 16 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This death resulted from uncontrollable haemorrhage. Anaesthesia was not contributory to the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
106.1.58	2	No comment.	< 24	Multiple injuries.	Yes

Name: Hilda Gamba Age: 5 Sex: F Race: C

Disease: Multiple injuries. Fractured skull, ruptured spleen, crushed pelvis and ruptured bladder.

Operation: Laparotomy. Splenectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund, comatose. The patient suffered a fractured skull, ruptured spleen, crushed pelvis and ruptured bladder. Her pulse rate was 140/minute, B.P. 80 mm.Hg systolic. Severe hypovolaemic shock was present. Immediately pre-operatively 100 cc. blood was rapidly transfused.

PREMEDICATION: Nil.

ANAESTHETIC:

The patient was intubated after topical analgesia of the larynx. Anaesthesia was induced with nitrous oxide and oxygen (50%) by means of a circle absorption system with an IPPR technique. No relaxant was used. Following laparotomy, a splenectomy was performed.

During the splenectomy, rapid transfusion of blood was continued. The B.P. at the conclusion of the operation, which lasted 60 minutes, was 100 mm.Hg systolic. During laparotomy, a large retroperitoneal haematoma from the crushed pelsiv and a ruptured bladder were noted. At the conclusion of the operation, lumbar puncture revealed heavily blood stained C.S.F., which led to the diagnosis of cerebral laceration. At the end of the anaesthetic the patient was in the same comatose state as pre-operatively. She died 3 hours post-operatively.

AUTOPSY:

Multiple abrasions on the body. Three sutured surgical wounds: (1) midline abdominal 18 cm. long, (2) horizontal in the left iliac fossa 10 cm. long, (3) curved incision inthe left iliac fossa from the vulva 18 cm. long, (4) a drain wound in the left lower quadrant of the abdomen. Fracture running through the coronal suture of the skull with underlying extradural haemorrhage, scattered sub-arachnoid haemorrhages of the brain. Fracture of left clavicle. Bruising of both lungs. Haemoperitoneum with lacerations on superior surfaces of the liver. Massive retroperitoneal and retro-pubic haematoma with fracture of right pubic ramus.

COMMENT:

This death resulted from multiple injuries. Anaesthesia was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
107.1.58	2	No comment	< 24	Cerebral abscess.	Yes

Name: Agnes Saayman. Age: 18 Sex: F Race: C

Disease: Mastoiditis; temporal lobe cerebral abscess.

Operation: Burrhole craniotomy. Right radical mastoidectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was in a poor state with a markedly depressed level of consciousness. Pulse rate 130/minute, respiration 16/minute. She had been admitted the previous day with chronic otorrhoea, 7 days' headache and vomiting with neck stiffness. The lumbar puncture revealed turbid fluid. Radical mastoidectomy was performed and revealed an intracerebral abscess. Burrhole craniotomy and drainage of the abscess had been performed. The condition continued to deteriorate. A more extensive burrhole craniotomy was considered necessary. Immediately pre-operatively the haemogolobin was 10.5 gm.%. An intravenous infusion of 5% dextrose in water had been administered pre-operatively.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 250 mg. in divided doses because of the patient's markedly confused state. This was followed by nitrous oxide and oxygen via a Magill circuit, the patient breathing spontaneously. After analgesia of the larynx, oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen, with spontaneous breathing. At times during the course of the operation, a trace of ether vapour was administered. In all, during the entire operation which lasted 2 hours, 1 oz. of ether was used. No relaxant drugs were used.

Throughout the operation the B.P. was maintained at about 100 mm.Hg systolic with little change. The pulse rate manifested a tachycardia throughout, being 130/minute. Towards the end of operation this slowed to 110/minute. During the operation 500 ml. blood was transfused. The course of the anaesthesia was untoward. At operation extradural pus with dural fistula and copious foul smelling from the middle cranial fossa and a temporal lobe abscess were found, At the conclusion of the operation the patient returned to approximately the same level of consciousness which had existed previously. Her lash reflex returned, pupils were small but she never fully regained consciousness. She developed respiratory failure and died. Respiratory failure occurred 1 hour post-operatively.

AUTOPSY:

Meningitis with large temporal lobe abscess on the left side.

COMMENT:

This death was due to the pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
108.1.58	2	No comment	< 24	Intra-cranial neoplasm.	No

Name: Antony Cohen Age: 4 months Race: E Sex: M

Disease: Papilloma of choroid plexus in lateral ventricle.

Operation: Craniotomy. Excision of lateral ventricular choroid plexus papilloma

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This infant was in a fair state pre-operatively. He weighed 12 lb. The previously raised intracranial pressure had been reduced by lumbar puncture.

PREMEDICATION:

Atropine gr. 1/300.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, delivered via a modified infant T-piece bag and mask, the patient breathing spontaneously. Oral intubation was performed and subsequently anaesthesia was maintained with nitrous oxide and oxygen, with minimal ether delivered via an Ayres T-piece, the patient breathing spontaneously. An intravenous infusion was established via a cutdown on the saphenous vein.

During surgical removal of the neoplasm, gross haemorrhage occurred. This loss was replaced by transfusion of compatible blood during the operation, to the extent of a volume equal to the child's blood volume: 500 ml. A further 100 ml. blood was transfused post-operatively. Together with the blood transfusion, 3 doses of 5 ml. calcium gluconate 10% were given. At the conclusion of the operation, which lasted 185 minutes, the patient was found to be hypothermic with a temperature of 90°F. In the theatre, at the conclusion of operation, the patient was warmed to a body temperature of 94°F. Following discontinuance of anaesthesia he rapidly regained consciousness and had normal respiration. 7 hours post-operatively the patient developed a marked tachypnoea and 2 hours later he died.

AUTOPSY:

No autopsy.

COMMENT:

The anaesthetic administered to this patient does not appear to have played in part in the causation of his death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
109.1.58	3	No comment	< 24	Cerebellar abscess.	Yes

Name: Philip Weber Age: 12 Sex: M Race: E.

Disease: Cerebellar abscess. Operation: Carotid angiography. Posterior fossa burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor. Admitted 2 days previously complaining of headache and a stiff neck, but in the last two days there had been a rapid deterioration and the level of consciousness had deepened. He was now stuperose. In addition the patient suffered from a tetralogy of Fallot and was grossly cyanosed. B.P. 100/60 mm.Hg, temperature 99°F.

PREMEDICATION:

Atropine gr. 1/150.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen delivered via a Magill circuit with the patient breathing spontaneously, and gradually added ether vapour. Within 3 minutes of commencement of the induction, respiration suddenly became totally diaphragmatic and almost immediately thereafter ceased. The patient was immediately intubated and IPPR with oxygen alone was instituted. He remained deeply unconscious. A carotid angiogram was performed and though this revealed no intracranial shift, the surgeon proceeded to perform a posterior fossa burrhole craniotomy. This revealed the brain to be under tension. No pus was found. At the conclusion of the operation, though the patient had been ventilated artificially, on oxygen alone, throughout the operation, he had still failed to regain consciousness, nor had respiration recommenced. He was placed in a Drinker respirator and remained there until he died 24 hours post-operatively.

AUTOPSY:

Cerebellar abscess 1 inch in diameter. Purulent meningitis. Tetralogy of Fallot with infundibular stenosis.

COMMENT:

Although respiratory failure occurred shortly after the commencement of anaesthetic, it is unlikely that this was precipitated by anaesthesia - although this cannot be categorically excluded. The gas mixture administered contained 36% oxygen, there had been no respiratory obstruction and respiration at this stage appeared tobe adequate. By the time respiratory arrest occurred, 3 minutes after commencing induction, little ether had been administered. There was in all probability a marked degree of cerebral anoxia. The effects of the cerebellar abscess and raised intracranial pressure would have been markedly worsened by the concomittant tetralogy of Fallot malformation. These were not circumstances over which the anaesthetist had any control. The level of consciousness had deteriorated rapidly pre-operatively, and immediate discontinuance of anaesthesia and the administration of oxygen alone by IPPR failed to reverse the coma and apnoea. It must be concluded that this death was probably inevitable. In that the terminal apnoea occurred while anaesthesia was being administered, anaesthesia must be considered unavoidably and necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
110.1.58	2	No comment.	< 24	Cerebral glio-blastoma.	No

Name: Katy Bungo Age: 59 Sex: F Race: B

Disease: Cerebral tumour. Operation: Carotid angiography. Burrhole craniotomy. Brain biopsy.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Grave - the patient was deeply stuperose with depressed shallow respiration, respiratory rate 15/minute. B.P. 120/70 mm.Hg.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Following pharyngeal toilette and pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen. The larynx was sprayed with topical analgesic and oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether, administered via a Magill circuit with spontaneous breathing.

During the operation the course of anaesthesia was uneventful. At the conclusion of the procedure the patient returned to the same state of consciousness as she had been in pre-operatively. She died 9 hours post-operatively without regaining consciousness. A tumour found at operation was a glioblastoma.

AUTOPSY:

No autopsy.

COMMENT:

The patient died of the effects of intracranial neoplasm. The anaesthetic played no part in this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
111.1.59	2	No comment	< 24	Peritonitis.	No.

Name; Barbara Benade Age: 62 Sex: F Race: E

Disease: Peritonitis. Operation: Laparotomy and drainage.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

One month previously the patient had had a gastrectomy and was discharged 11 days later. Three days after discharge she was re-admitted with pus oozing from a wound sinus, and 3 days later a subphrenic abscess was drained under general anaesthetic. Her condition was poor. Following drainage of the abscess her condition continued to deteriorate and signs of gross peritonitis became evident. At this stage laparotomy for drainage of the abdomen was decided on. Her condition was extremely poor and was graded by the anaesthetist as in extremis. Temperature 101°F, respiratory rate 24/minute, pulse rate 120/minute. She had a cold sweaty mottled skin with poor capillary refill time. Abdominal X-ray showed a raised fixed diaphragm. B.P. immediately pre-operatively was 115/80 mm.Hg. She was mentally confused and was on continuous drainage suction with intravenous replacement with added potassium. She was diabetic. This aspect was reasonably well controlled by soluble insulin.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 50 mg. followed by 25 mg succinylcholine. Endotracheal intubation was performed after preliminary IPPR with oxygen, and anaesthesia was continued with nitrous oxide and oxygen (50%) delivered via a circle absorption system. An IPPR technique was used. A trace of ether was added to the mixture. Just before the peritoneum was opened 10 mg. succinylcholine was given and IPPR was continued throughout the operation. A further dose of 25 mg. succinylcholine was necessary at the end of the procedure for closing the peritoneum.

Throughout the operation, which lasted 55 min., her condition was maintained fairly constant with a B.P. of 110 mm.Hg systolic, except for one period of 10 minutes when it fell to 85 mm.Hg systolic. The B.P. recovered after 10 minutes without any specific treatment. At the conclusion of the operation, the respiration returned to the pre-operative level and the patient regained consciousness. She died 9½ hours post-operatively from what appeared to be peripheral vascular failure secondary to peritonitis, possibly from septicaemia.

AUTOPSY: No autopsy.

COMMENT:

This patient died from gross peritonitis. Anaesthesia is not considered to have played any contributory role.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
112.1.59	1	Probably	>24	Respiratory obstruction Anoxic anoxia. Cardiac arrest.	Yes

Name: Willie Peterson. Age: 35 Sex: M Race: C

Disease: Gastric ulcer. Operation: Partial gastrectomy.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

This patient was entirely normal except for the gastric ulcer. B.P. 150/95 mm.Hg, pulse rate 80/minute.

PREMEDICATION:

Morphine gr. 1/6, atropine gr. 1/100, 1 hour before operation.

ANAESTHETIC:

Before the commencement of anaesthesia the B.P. was 150 mm.Hg systolic and the pulse rate 80/minute. Anaesthesia was induced with thiopentone 500 mg. followed by nitrous oxide and oxygen, with gradually added ether, via a circle absorption system, with spontaneous breathing. After 20 minutes the patient had been anaesthetised to the stage of moderate deep surgical anaesthesia (stage 3, plane 2-3). B.P. was 100 mm.Hg systolic with a pulse rate rise to 90/minute, and he was intubated orally, still breathing spontaneously. After a further 10 minutes the operation was commenced. Gallamine 80 mg. was given intravenously and an IPPR technique instituted. The B.P. rose to 180 mm.Hg systolic with a pulse rate of 100/minute. After a further 5 minutes attempts at respiration were made by the patient and a further 20 mg. gallamine was administered. The B.P. was now still 180 mm.Hg systolic and the pulse rate 100/minute. At this stage a blood transfusion was commenced. After a further 10 minutes (i.e. 25 minutes after the operation was begun) respiratory attempts were again noticed and a further 20 mg. gallamine was given intravenously. The B.P. was now 170 mm.Hg systolic, pulse rate 100/minute. After a further 10 minutes, during which time the patient's B.P. commenced dropping rapidly and reached a level of 100 mm.Hg systolic with a pulse rate slowing to 70/minute, the surgeon commented on the appearance of cyanosis in the operative area. The anaesthetist, who could not immediately see the reason for cyanosis, summoned help. A senior anaesthetist came immediately from an adjacent theatre and diagnosed an obstruction to the endotracheal tube, rapidly removed it and re-inserted another. As this was accomplished and IPPR with pure oxygen was instituted, cardiac arrest occurred. This was 3 minutes after the surgeon's comment on the presence of cyanosis. Immediate cardiac massage resulted in a prompt return of regular, forceful cardiac contractions. Cyanosis disappeared promptly and the B.P. returned to 170 mm.Hg systolic, but the pupils remained dilated. Within 8 minutes the B.P. had risen to 200 mm.Hg systolic and then to 210 mm.Hg systolic. The pulse rate was 140/minute. After a further 10minutes the B.P. settled back to 150 mm.Hg, pulse rate 100/minute.

The operation had reached the point of no return and was continued. The anaesthetist administered nitrous oxide and oxygen by IPPR. Just over 1½ hours later the operation was completed. Following discontinuance of anaesthetic and the administration of 1 mg. neostigmine preceded by atropine 1/100 gr., spontaneous respiration occurred.

However / ...

However, this was of a Cheyne-Stokes character. The patient was kept in theatre for a further ½ hour and another 1 mg. neostigmine was given. The respiration retained its periodic character though of apparently adequate volume. The patient maintained a normal colour. He did not regain consciousness, remaining deeply comatose with dilated pupils. 50 cc. of 50% dextrose was given for the anticipated cerebral oedema. 1½ hours after completion of operation the patient was returned to the ward. B.P. was 140 mm.Hg systolic, pulse rate 90/minute. He was deeply unconscious and still had Cheyne-Stokes respiration. Following return to the ward, the patient was treated for cerebral oedema by restriction of fluids for the first 48 hours and by administration of two further doses of 50 cc. 50% dextrose intravenously. However, it was soon apparent that the cerebral damage resultant on anoxia was gross and irreversible, and the patient remained deeply comatose for 4 days without showing any signs of improvement and then died.

AUTOPSY:

Cause of death undetermined but probably due to cerebral anoxia which arose during a general anaesthetic, causing irreversible brain damage. The operation was for partial gastrectomy. Both lungs were intensely congested and oedematous. Early bronchopneumonic changes. Brain meninges and cerebral vasculature appeared markedly congested. Histology was not performed.

COMMENT:

This is a frank anaesthetic death, due to irreversible cerebral damage resultant on anoxic anoxia, which also caused secondary cardiac arrest. This latter itself was treated successfully. The anoxic anoxia arose from an obstruction, probably incomplete, to the endotracheal tube - probably kinking in the oropharynx. Due to inexperience, the problem was not diagnosed by the anaesthetist.

The significance of the rising B.P. and pulse rate followed by a fall and slowing, and the constantly returning respiratory efforts on the patient's part, probably indicating anoxia, was not appreciated and was wrongly interpreted as an indication for the administration of more relaxant. Only when gross cyanosis became apparent and was commented on by the surgeon did the anaesthetist realise that all was not well, and summon help. While the endotracheal tube was removed and re-inserted, cardiac arrest occurred secondary to anoxic anoxia. This was promptly and correctly treated. The fact that an IPPR technique with muscle relaxant was used together with the fact that the obstruction was in all probability not complete, certainly at its onset, and the occurrence of cyanosis was not sudden, may have combined to distract the inexperienced anaesthetist from the true diagnosis.

PREVENTABILITY:

This death was obviously preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
113.1.59	3	No comment	ORD	Elective cardiac arrest.	Yes

Name: Janene Kench Age: 12 Sex: F Race: E

Disease: Pulmonary and aortic valve stenosis.

Operation: Pulmonary and aortic valvotomy; cardio-pulmonary bypass.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had congenital aortic and pulmonary stenosis and an auricular septal defect. B.P. 90/60 mm.Hg.

PREMEDICATION:

Seconal gr. 1½ orally 3 hours before operation. Atropine gr. 1/100 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 120 mg. followed by succinylcholine, IPPR with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued throughout the operation with nitrous oxide and oxygen administered via a circle absorption system with IPPR, dTc being used as the relaxant.

The courseof the operation and anaesthesia until bypass was started was relatively untoward. Cardiopulmonary bypass was established and the anaesthesia was maintained throughout with small supplemental doses of thiopentone and 3 mg. doses of dTc. During bypass the patient was cooled to a temperature of 33°C. The heart was electively arrested by means of potassium citrate. Following bypass adequate cardiac action could not be re-established. Ventricular fibrillation kept recurring. In spite of re-institution of bypass and the use of sodium bicarbonate, procaine amide and adrenaline, electrical defibrillation also being attempted, normal cardiac action could not be re-established.

AUTOPSY:

Congenital abnormalities in the heart. Evidence of operation on the heart. No cause of death was found. Death was presumed to have been due to functional arrest of the heart during the operation.

COMMENT:

This death was the result of open heart surgery with elective cardiac arrest. The anaesthetic was in no way contributory; in that death occurred while the patient was anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
114.1.59	2	No comment	< 24	Multiple injuries	Yes

Name: Adriana van der Linden. Age: 39 Sex: F Race: E

Disease: Multiple injuries. Operation: Laparotomy. Resection of colon.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was fully conscious and grossly shocked. Pulse rate 140/minute, B.P. 95/50 mm.Hg after 8 pints blood had been transfused. She had been completely pulseless on admission. Ribs on the left side of the thorax were fractured but there was no pneumo- or haemothorax. She had bilateral compound fractures of the femurs, evidence of a ruptured viscus and extensive lacerations.

PREMEDICATION: Nil.

ANAESTHETIC:

Following 5 minutes pre-oxygenation anaesthesia was induced with cyclopropane and oxygen. Succinylcholine was then administered and oral intubation performed. Following return of spontaneous respiration, 120 mg. gallamine was given and anaesthesia was maintained with nitrous oxide and oxygen via a circle absorption system, using an IPPR technique.

Laparotomy was performed and several extensive areas of gangrene were found in the small bowel with avulsion of the mesentary, and there were several perforations of the colon with gross faecal soiling of the peritoneum. The gangrenous areas of gut were resected with end to end anastomosis. Following the abdominal operation, 1 mg. neostigmine was given preceded by atropine 1/100 gr. Normal spontaneous respiration returned and anaesthesia was continued with nitrous oxide and oxygen, with spontaneous breathing, via a Magill system. The compound fractures of the femur were then attended to. During operation, her condition was well maintained and a further 3 pints blood were transfused together with 4 gm. calcium gluconate in divided doses. At the conclusion of the procedure, which lasted 4 hours, the patient's B.P. was 120 mm.Hg systolic with a pulse rate of 100/minute. On discontinuance of the anaesthetic the patient rapidly regained consciousness. She remained well post-operatively for 6 hours. Thereafter the B.P. commenced to decline and could not be properly maintained with further blood transfusions. The administration of noradrenaline and hydrocortisone also failed to stop the fall in B.P. Ultimately pulmonary oedema supervened and the patient died 12 hours after operation.

AUTOPSY:

Same findings as were noted at operation plus congestion of the lungs.

COMMENT:

This patient died as a result of multiple injuries. Anaesthesia is not considered to have played any part in the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
115.1.59	2	No comment	< 24	Intra-cerebral tumour; intracranial haemorrhage.	Yes

Name: Estelle Groenewald. Age: 5 months. Sex: F Race: E.

Disease: Hydrocephalus, intra-cerebral tumour. Operation: Ventriculocysternostomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair but the child was drowsy with signs of intracranial space-occupying lesion.

PREMEDICATION:

Atropine gr. 1/200.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane administered via a modified infant's T-piece bag and mask, with spontaneous breathing. Oral intubation was performed and anaesthesia continued with nitrous oxide and oxygen, and Halothane, 0.5 - 1% administered bia an Ayre's T-piece, the patient breathing spontaneously.

The anaesthesia was uneventful throughout the operation, a ventriculocysternostomy, which lasted 2½ hours. At the conclusion of operation the patient recovered consciousness promptly and was returned to the ward. She died 17 hours post-operatively.

AUTOPSY:

Cerebral tumour and intracerebral haemorrhage.

COMMENT:

This patient died of the pre-existing disease and as a result of surgery. Anaesthesia is not considered contributory to the outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
116.1.59	2	No comment	< 24	Neonatal tetanus.	No

Name: Daniel August. Age: 8 days. Sex: M Race: C

Disease: Tetanus Operation: Tracheotomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor. The patient had had frequent tetanic spasms. He was cyanosed and had a tachypnoea. Pulse rate 150/minute.

PREMEDICATION:

Atropine gr. 1/150 given 30 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, with spontaneous breathing. During induction there were tetanic spasms. These ceased once anaesthesia was achieved. Oral intubation was performed and tracheotomy was undertaken. The operation lasted 15 minutes.

The cyanosis which had been present improved immediately after oral intubation had been performed. Following discontinuance of the anaesthetic the patient returned to his previous level of consciousness. He died 12 hours after tracheotomy.

AUTOPSY:

No autopsy.

COMMENT:

It is considered that this patient died as a result of neonatal tetanus. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
117.1.59	2	No comment	< 24	Cirrhosis of liver. Portal hypertension. Haemorrhage.	No

Name: Hajiera Abrahams. Age: 29m Sex: F Race: C

Disease: Ruptured ectopic pregnancy; cirrhosis of liver; portal hypertension. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor - she was unconscious, having been admitted comatose. She had ascites, jaundice, cirrhosis of the liver, 2 months' amenorrhoea and vaginal bleeding. B.P. 50 mm.Hg systolic. She was thought to have a ruptured ectopic pregnancy.

PREMEDICATION:

Atropine gr. 1/100 given 40 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide and oxygen with the patient breathing spontaneously. A trace of ether was administered intermittently. A circle absorption system was used.

Blood transfusion was continued throughout the operation, which lasted 90 minutes. During this time there was some improvement in her condition. On laparotomy, gross haemorrhage from peritoneal surfaces was found. There was no ectopic pregnancy. After 12 hours generalised fits occurred and respiration became laboured. She died 22 hours after operation.

AUTOPSY:

Primary carcinoma of the liver.

COMMENT:

This patient is considered to have died as the result of the pre-existing disease. Anaesthesia did not play any significant part in the outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
118.1.59	2	No comment	< 24	Multiple injuries	Yes

Name: David Gerstner. Age: 41 Sex: M Race: E

Disease: Multiple injuries. Attempted suicide: multiple lacerations, ruptured urethra.

Operation: Amputation of arms. Laparotomy and colostomy. Repair of multiple injuries.

Anaesthetic risK: 4, emergency.

PRE-OPERATIVE STATE:

Having attempted suicide by throwing himself under a train, the patient was in extremely poor condition. Blood transfusion had failed to restore the B.P. above 40 mm.Hg systolic. An intravenous infusion of noradrenaline was commenced, which raised the B.P. to 90 mm.Hg systolic.

PREMEDICATION:

20 mg. pethidine. ½ hour before operation he was given atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg. Oral intubation was performed and anaesthesia continued with nitrous oxide, oxygen and a trace of ether administered via a circle absorption system. During operation, which lasted 237 minutes, gallamine 72 mg. was used as a relaxant. Anaesthesia was maintained with nitrous oxide and oxygen administered by an IPPR technique via a circle absorption system.

The B.P. could only be maintained throughout operation with massive blood transfusion and infusion of noradrenaline. The patient appeared to be in a state of irreversible shock. At the conclusion of operation, spontaneous respiration recurred and complete reversal of curarization was ensured by the administration of 0.5 mg. neostigmine preceded by atropine gr. 1/100. Following the conclusion of anaesthesia the patient regained consciousness. He died 7 hours post-operatively.

AUTOPSY:

Multiple injuries received on attempted suicide. Subarachnoid haemorrhage. Left arm amputated. Rupture of bladder. Lacerated perineum. Abdominal incisions and evidence of operation. Colostomy.

COMMENT:

This patient died from irreversible traumatic shock, the result of multiple injuries. Anaesthesia is not considered in any way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
119.1.59	2	No comment	< 24	Carcinoma of the oesophagus eroding and obstructing left main bronchus.	Yes

Name: Portrait Mnabalala. Age: 47 Sex: F Race: B

Disease: Post-oesophagectomy intestinal obstruction. Operation: Laparotomy and freeing of adhesions.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

She had carcinoma of the oesophagus. One week previously oesophagectomy had been commenced but following mobilization of the stomach it had been found that the carcinoma was irresectable. Now, one week later she developed intestinal obstruction. At this stage her condition was poor. She had rhonchi and fine crepitations at both lung bases with diffuse wheezing.

PREMEDICATION:

Pethidine 100 mg., atropine gr. 1/100.

ANAESTHETIC:

Following 5 minutes' pre-oxygenation, anaesthesia was induced with nitrous oxide oxygen and ether, and oral intubation was performed. During the operation, which lasted 60 minutes and consisted of a laparotomy and freeing of adhesions, anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether, administered via a circle absorption system using an IPPR technique. Gallamine was used asthe relaxant.

From the start of the anaesthetic it was observed that the patient had marked inspiratory difficultyand ventilation was difficult. At first it was thought that this inspiratory difficulty which was accompanied by wheezing, was due to bronchospasm. Aminophyllin 500 mg. was administered intravenously without effect. Though a dose of 70 mg. gallamine had been given throughout the operation, there was no residual curarization at the conclusion and no neostigmine was given. However, when the patient commenced breathing spontaneously, the marked respiratory difficulty appeared worse and she became cyanosed. Hydrocortisone 100 mg. was given in the belief that this was due to bronchospasm. This had no effect. 10minutes later a further 500 mg. amninophyllin was given, with no effect. Bronchoscopy was now performed. This revealed necrotic material obstructing the left main bronchus, with some in the right main bronchus. It was consequently concluded that the carcinoma was invading the left main bronchus and obstructing the lumen of the right main bronchus as well. A right endobronchial tube was inserted. The respiration became markedly better and the patient lost her cyanosis. Her condition continued to deteriorate post-operatively and 17 hours after operation, while attempts were being made to reposition the right endobronchial tube which no longer appeared satisfactory,the patient developed complete respiratory obstruction and died.

AUTOPSY:

Carcinoma of the middle and lower third of the oesophagus. Erosion and collapse of left main bronchus with collapse and oedema of the left lung. Secondary involvement of the hilar and coeliac glands with necrotic tumour. Ulceration of the larynx and main bronchus.

COMMENT:

The patient died of respiratory obstruction due to bronchial infiltration by an oesophageal carcinoma. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
120.1.59	2	No comment	< 24	Extradural haematoma	Yes

Name: Peter Taljaard. Age: 8 Sex: M Race: E.

Disease: Head injury; extradural haematoma. Operation: Carotid angiography. Craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was comatose but the cardiovascular status was adequate.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with Halothane 1%, nitrous oxide and oxygen administered via a Magill system with the patient breathing spontaneously. Oral intubation followed and anaesthesia was maintained with nitrous oxide, oxygen and ½% Halothane during carotid angiography. During the craniotomy, anaesthesia with Halothane was discontinued and a trace of ether administered in its place, with spontaneous breathing. The operation lasted 2½ hours and the patient's condition remained static. At the conclusion of the procedure the patient's state of consciousness was the same as pre-operatively. He died 7½ hours post-operatively.

AUTOPSY:

Fractured skull. Intracranial haemorrhage and cerebral lacerations. No evidence of foreign material in trachea, bronchi or bronchioles.

COMMENT:

This patient died as a result of cerebral lacerations. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
121.1.59	2	No comment	< 24	Cerebral abscess. Meningitis.	Yes

Name: Freddy Dirk Age: 9 Sex: M Race: C

Disease: Cerebral abscess. Operation: Burrhole craniotomy and ventricular drainage.

Anaesthetic risk: #, emergency.

PRE-OPERATIVE STATE:

Poor, comatose. Temperature 100°F, pulse rate 140/minute, respiration 26/minute, B.P. 110 mm.Hg systolic. Subacute bacterial endocarditis.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen administered via a Magill semi-open circuit. Orotracheal intubation was performed following topical analgesia of the larynx. Anaesthesia was maintained throughout the operation with nitrous oxide and oxygen.

At operation no cerebral abscess was found, but diffuse meningitis. Ventricular drainage was established. At the end of the operation the patient was in the same comatose state as pre-operatively. He died 4 hours post-operatively.

AUTOPSY.

Bacterial endocarditis on incompetent mitral valve (rheumatic). Splinter haemorrhages under nails. Flea-bitten kidney. Haemorrhages in the lungs. Diffuse meningitis.

COMMENT:

This death was due to the patient's pre-existing disease. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
122.1.59	1	Possibly	ORD	Cardiac arrest. (Thiopentone induced).	Yes

Name: April Snyders. Age: 10 months Sex: M Race: C

Disease: Congenital pulmonary atresia (cyanotic heart disease). Operation: Cardiac catheterisation.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This little patient suffered from congenital cyanotic heart disease. The lesion was suspected tobe congenital pulmonary atresia. The child had been in cardiac failure but this was now controlled with Digitalis. There was also a fistulous intracranial murmur. The patient's haemoglobin was 11 gm.%. Pulse rate 132/minute, temperature 99.5°F.

PREMEDICATION:

Atropine gr. 1/400.

ANAESTHETIC:

Rectal avertin, a dose of 100 mg./kg. body weight, was used. In this child the dose amounted to 700 mg. This was made up into 2.5% solution in 28 ml. water. The solution was run in through a rectal catheter with the baby lying on the right side. The patient dropped off to sleep in 5 minutes. After 15 minutes the patient was turned supine with the head turned to the side, and cut down for catheterisation was commenced on the right arm using local anaesthesia obtained with 5 ml. of 1% Xylocaine. From the anaesthetic point of view the fifst 2 hours of the investigation were uneventful, the baby breathing satisfactorily (room air) with the pulse rate steady and of good volume, at a rate of 120/minute. After 2 hours, when preparations were being made to take the main series of angiograms, the intravenous catheter had to be reinserted because of technical problems unrelated to anaesthesia. The disturbance caused the child to become restless. In an effort to control this the anaesthetist decided to supplement the avertin anaesthesia with thiopentone. Two successive doses of 25 mg. thiopentone were injected down the intravenous catheter in the right arm, (total dose 50 mg. thiopentone). Breathing continued satisfactorily but a bradycardia ensued immediately, the pulse rate dropping to 40/minute. The immediate administration of 1/200 gr. atropine intravenously caused the heart rate to accelerate immediately to 80/minute but this was not maintained and it just as quickly slowed to 40/minute. A further gr. 1/200 atropine was injected intravenously down the catheter but the bradycardia worsened. Artificial ventilation with pure oxygen was commenced but cardiac arrest occurred within 5 minutes of the administration of the dose of thiopentone. While artificial ventilation with oxygen was maintained, a thoracotomy was performed in the 4th left interspace and cardiac massage was commenced. After 3 minutes massage a slow heart beat commenced, however, this was ineffective and did not produce a peripheral pulse unless supplementary cardiac massage was maintained. Cardiac massage was maintained for an hour but at no stage could a spontaneous heart beat maintain a palpable peripheral pulse. During the first ½ hour some poor respiratory efforts were made and during this time endotracheal intubation was performed and IPPR with oxygen was continued manually by this means. From the period of cardiac arrest, the pupils were dilated and unresponsive to light. After an hour of cardiac massage, during which time 12 mg. methyl amphetamine had been given intravenously as well as 2 minims of 1:1,000 adrenaline, no effective response had been obtained from the myocardium, and resuscitative efforts were abandoned and death presumed.

AUTOPSY:

Enlarged heart due to congenital pulmonary atresia, accessory pulmonary lobes, pleural effusion and ascites, imperfect rotation of abdominal organs.

COMMENT:

Considering the gross congenital cardiac anomaly present in this child, it seems a little harsh to attribute this death to anaesthesia. However, the direct depressant effects of thiopentone on the myocardium are well known, especially on the functionally damaged myocardium. The 50 mg. thiopentone administered in this child via an intravenous catheter lying well up in the subclavian vein ensured a high concentration reaching the myocardium. With the bradycardia which immediately preceded asystole, following so closely on the heels of the administration of thiopentone, one is forced to conclude here that the precipitating factor in the cardiac arrest that caused this patient's death was the administration of thiopentone by the anaesthetist.

PREVENTABILITY:

More mature consideration of the position of the intravenous catheter and an awareness of the possibility of a "slug dose" effect of thiopentone administered by this route on the myocardium, should have led the anaesthetist to avoid this route of administration, or to modify the rate of administration. In this sense, the death was "possibly preventable".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
123.1.59	2	No comment	< 24	Cerebral laceration	Yes

Name: Patricia Siederman. Age: 6 Sex: F Race: C

Disease: Head injury, compound depressed fracture of the skull, brain herniation.

Operation: Craniectomy and elevation of depressed fractured skull.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor, comatose. There was a compound depressed fracture of the skull with herniation of the brain, gross haemorrhagic shock. On admission she was pulseless. Following blood transfusion with 2 pints blood, the pulse rate was 100/minute, B.P. 80 mm.Hg systolic, respiration 20/minute.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Following topical analgesia of the larynx, oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen delivered via an Ayres T-piece system. A trace of trichlorethylene vapour was administered at intervals. During operation which lasted 60 minutes, a further 1 pint blood was transfused. The patient's general condition remained much the same. At the conclusion of operation her state of consciousness was the same as before the operation. 12 hours post-operatively a tracheotomy was performed. The patient died 20 hours after operation.

AUTOPSY:

Fractured skull, left side. Tracheotomy wound. Lacerated brain. Cutdown wound in right ankle. Petechial haemorrhages in both lungs.

COMMENT:

This patient died of cerebral laceration. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
124.1.59	2	No comment	< 24	Peritonitis.	No

Name: Maria Sado Age: 74 Sex: F Race: E

Disease: Generalised peritonitis. ?Ruptured gall bladder. Operation: Laparotomy. Cholecystectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely grave. Patient was suffering from peritonitis, congestive cardiac failure and was deeply jaundiced. Pulse rate 140/minute with auricular fibrillation. Respiration 40/minute and very shallow. She was diabetic but this was adequately controlled by insulin. Three hours before operation she was given 0.25 mg. Digoxin and another 0.25 mg. Digoxin 1 hour pre-operatively.

PREMEDICATION:

Atropine gr. 1/100 1 hour before operation.

ANAESTHETIC:

Following 5 minutes of pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a Magill circuit with the patient breathing spontaneously. When the patient was adequately anaesthetised, the larynx was sprayed with topical analgesic and oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen, with ether, administered via a Magill circuit, with spontaneous breathing.

Throughout the operation, which lasted 1½ hours, the patient's condition remained static. At operation, diffuse peritonitis resulting from a rupture of the gall bladder was found. A cholecystectomy was performed. At the conclusion of the procedure and discontinuance of anaesthesia, the patient regained consciousness rapidly. She died 5 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of peritonitis. Anaesthesia is not considered to be a contributory factor.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY.
125.1.59	2	No comment	< 24	Undetermined.	No.

Name: Kenneth Sims Age: 52 Sex: M Race: C

Disease: Post-operative intestinal obstruction with dehiscence of abdominal wound.

Operation: Laparotomy. Resuture of dehisced abdomen.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Five days previously this patient underwent a resection of colon with loop colostomy for carcinoma of the colon. He now had intestinal obstruction and his abdominal operative wound had dehisced. His condition was poor. B.P. 120/80 mm.Hg, pulse rate 80/minute. Fluids had been adequately replaced.

PREMEDICATION:

Pethidine 75 mg., atropine gr. 1/100.

ANAESTHETIC:

Following pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen with gradually added ether. When anaesthesia was of sufficient depth, oral intubation was performed. Anaesthesia was maintained throughout with nitrous oxide and oxygen with a trace of ether, via a circle absorption system. Gallamine 100 mg. was administered and an IPPR technique was used. Throughout the operation, which lasted 95 minutes, the patient's condition was satisfactory on the whole, except for two occasions when drops in B.P. necessitated the administration of two doses of 10 mg. methyl amphetamine intravenously. During the operation, 500 ml. 5% dextrose in water was administered together with 500 ml. plasma.

At the conclusion of the operation, complete reversal of curarisation was achieved by the administration of 1.25 mg. neostigmine preceded by atropine gr. 1/100. When anaesthesia was discontinued, the patiently rapidly regained consciousness. On leaving the operating theatre his condition was satisfactory. Respiration was adequate and spontaneous. Following return to the ward the patient's condition gradually deteriorated and he died 8½ hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

Whatever the cause of this patient's death, it does not appear to have been related to any post-anaesthetic sequelae.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
126.1.59	2	No comment	< 24	Undetermined.	No

Name: Miriam Magin. Age: 75 Sex: F Race: C

Disease: Intestinal obstruction, small bowel volvulus.

Operation: Laparotomy. Resection of gangrenous loop of small bowel.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Very poor. Intestinal obstruction had been present for 48 hours. Pulse rate 98/minute, B.P. 100/70 mm.Hg. Fluid had been replaced and gastric suction established. One pint blood had been transfused.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a Magill circuit, with spontaneous breathing. During induction the patient vomited and regurgitated a copious amount of fluid. Laryngospasm ensued and the patient became cyanosed. She was immediately placed in a Trendelenburg position, the mouth and pharynx were aspirated clear with suction aspiration, and because of the suspicion that she may have inhaled some vomitus, an immediate bronchoscopy was performed. A very small amount of vomited fluid was found in the trachea but the major bronchi were clear. The cyanosis resolved within a minute. Anaesthesia was continued with nitrous oxide, oxygen and ether, with the patient breathing spontaneously. Auscultation of the chest at this time revealed clear breath sounds.

At operation a volvulus of jejunum was found with gangrenous loop of small bowel. This loop of small bowel was resected and end to end anastomosis performed. Throughout the operation anaesthesia was maintained with nitrous oxide and oxygen with ether administered via a Magill semi-open system with the patient breathing spontaneously. A total dose of 5½ oz. ether was administered. Her condition was static throughout operation, with a B.P. of 110 mm.Hg systolic and pulse rate 100/minute. Breathing appeared normal throughout. At the conclusion of the operation, which lasted 90 minutes, and on discontinuance of anaesthesia, the patient recovered consciousness within 10 minutes. Respiration was normal and auscultation of the chest at this time revealed clear breath sounds. Three hours after return to the ward the patient died. There appeared to be no respiratory difficulty prior to death. Breath sounds were normal; no rhonchi or rales were audible.

AUTOPSY:

No autopsy.

COMMENT:

Treatment of the episode of vomiting which occurred during induction of anaesthesia was correct and prompt. Bronchoscopy revealed little evidence of inhalation of vomitus. Subsequently no further signs of pulmonary aspiration of vomitus were evident. At the conclusion of operation the B.P. was at the same level as pre-operatively, and the patient regained consciousness 10 minutes after discontinuance of the anaesthetic. The respiration and breath sounds were normal on return to the ward. Her subsequent death did not appear to be associated with respiratory distress; there were no signs post-operatively of respiration rhonchi and breath sounds remained clear.

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It is not considered that the vomiting at the commencement of anaesthesia played any part in her subsequent death. The poor state of the patient pre-operatively, resulting from prolonged jejunal obstruction (48 hours) was of sufficient severity to account for the post-operative death. Though the fact that anaesthetic was perhaps contributory to this patient's death cannot be excluded, on the evidence, it is not considered that this was a significant contributory factor.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
127.1.59	2	No comment	< 24	Cerebral ischaemia. Cerebral softening.	Yes

Name: Petrus Windvogel Age: 35 Sex: M Race: C

Disease: Stab through left common carotid artery. Operation: Repair of left common carotid artery.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

General state was good. B.P. 130/80 mm.Hg, respiration 18/minute. However, there was distinct evidence of brain damage, the patient being paralysed on the right side. He was unconscious but responded to painful stimuli.

PREMEDICATION:

Atropine gr. 1/100 administered 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 225 mg. followed by 50 mg. succinylcholine. After 1 minute of artificial ventilation with oxygen, oral intubation was performed with a No. 10 cuffed endotracheal tube. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether vapour, delivered via a Magill semi-open circuit with the patient breathing spontaneously.

The course of anaesthesia was uneventful throughout operation and the B.P. and pulse rate remained steady at 130 mm.Hg systolic and 96/minute respectively. The operation lasted 110 minutes during which time the left common carotid was resutured. The patient breathed spontaneously throughout the procedure and at no time was there any change in the character of the respiration. On conclusion of the operation and anaesthetic, the level of consciousness was much as it had been before operation. One hour after his return to the ward the patient died.

AUTOPSY:

Sutured incised wound in neck. Lacerated wound of left sternomastoid and incised wound of left common carotid. Large area of cerebral softening 5 cm. in diameter on the left cerebral hemisphere.

COMMENT:

The cerebral ischaemia from left carotid occlusion accounts for this patient's death. Though anaesthesia is not considered a contributory cause, the administration of thiopentone as an induction agent to the patient when he was already unconscious may be criticised. In defence of its use, however, it may be said that the patient did respond to painful stimuli and the use of thiopentone expedited induction of anaesthesia when speed in proceeding with surgery was essential because of the nature of the patient's lesion. The administration of thiopentone caused no deterioration in the patient's condition.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
128.1.59	2	No comment	< 24	Cerebellar abscess.	Yes

Name: Martinus Stander Age: 42 Sex: M Race: E

Disease: Cerebellar abscess secondary to mastoiditis.

Operation: Craniotomy. Drainage of cerebral abscess.

Anaesthetic risk: 4,emergency.

PRE-OPERATIVE STATE:

Following acute mastoiditis for which a mastoidectomy had been performed, this patient developed a cerebellar abscess. During the previous week this had been successfully drained twice, through a posterior fossa burrhole craniotomy. However, his condition and level of consciousness continued to deteriorate. Another attempt at drainage was considered necessary. The patient was very ill and stuperose. Temperature 99°F, pulse rate 90/minute, B.P. 120/80 mm.Hg. A tracheostomy had been performed 2 days previously to facilitate bronchial toilette in the face of his depressed level of consciousness.

PREMEDICATION:

Atropine gr. 1/100 was administered 1 hour before surgery.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. and was subsequently maintained with nitrous oxide, oxygen and Halothane/ether azeotrope, delivered via a semi-open Magill circuit administered via the tracheostomy, with spontaneous breathing.

The operation consisted of re-opening of the posterior fossa burrhole on the right side, and drainage of a cerebellar abscess. Throughout the operation, which lasted 55 minutes, the course of anaesthesia was uneventful. During the anaesthetic, the drip infusion of 5% dextrose and water intravenously was established to correct the slight degree of dehydration that was clinically apparent pre-operatively. Following conclusion of operation and the discontinuance of the anaesthetic, the patient was slow to recover consciousness to the pre-operative level. The patient died 3½ hours post-operatively.

AUTOPSY:

Bilateral cerebellar abscesses, the one on the right having been drained surgically, that on the left being full of pus and undrained. Trachea and bronchi full of mucopus.

COMMENT:

This patient died of a cerebellar abscess. Anaesthesia is not considered to have played any part in this patient's death. Inefficient post-operative tracheobronchial toilette may have led to inadequate ventilation and so be a contributory factor.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
129.1.59	2	No comment	< 24	Neonatal tetanus.	No

Name: Myrtle Davids Age: 6 days Sex: F Race: C

Disease: Neonatal tetanus. Operation: Tracheotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was suffering from severe tetanic spasms.

PREMEDICATION:

Atropine gr. 1/400 was given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane, administered via an infant's T-piece bag and mask. Following induction, oral intubation was performed and anaesthesia was continued with nitrous oxide, oxygen and Halothane, the patient breathing spontaneously.

Tracheotomy was performed without difficulty, the operation lasting 20 minutes. At the conclusion of the operation and on discontinuance of anaesthesia, the patient regained consciousness. On return to the ward she wascurarized and treated with IPPR administered with a Radcliffe ventilator. The patient died 22 hours after the tracheotomy.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of tetanus. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
130.1.59	1	Probably	> 24	Drug induced cardiac arrest. Overdose of ether. Cerebral ischaemia.	Yes

Name: Isobel Hansen Age: 46 Sex: F Race: E

Disease: Fibromyomata of the uterus. Operation: Hysterectomy (abandoned).

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Other than for uterine fibromyomata, this patient was perfectly healthy.

PREMEDICATION:

Pethidine 100 mg., atropine gr. 1/100 administered 1 hour pre-operatively.

ANAESTHETIC:

Immediately pre-operatively the patient appeared a little apprehensive. Pulse rate was 160/minute, B.P. 100 mm.Hg systolic. This may have resulted to some extent from the premedicant atropine. Anaesthesia was induced with thiopentone 300 mg. followed by nitrous oxide and oxygen, administered via a Coxeter Mushin circle system with the carbon dioxide absorber turned off. Ether vapour was gradually added to the inhaled mixture in increasing concentrations from the Boyle bottle vaporizer on the fresh gas inflow. Twelve minutes after the start of induction of anaesthesia, the larynx was sprayed topically with 4% Xylocaine by direct laryngoscopy and the patient was orally intubated. At this time the anaesthetist turned on the carbon dioxide absorber, as he thought. Gallamine 40 mg. was administered and an IPPR technique instituted, the patient being turned into the Trendelenburg position, and the operation commenced. 11 minutes later it was noticed that no pulse was palpable. Exposure of the patient's face revealed gross cyanosis with widely dilated pupils. The surgeon was immediately notified and on feeling no pulsation in the aorta, diagnosed cardiac arrest. All anaesthetic agents were discontinued and IPPR with oxygen performed. Because of an inexplicable hesitation on the surgeon's part, a delay of 6 minutes followed before institution of cardiac massage. During this period the anaesthetist discovered that he had turned on the ether control knob on the Coxeter Mushin vaporizer to maximum, instead of turning on the carbon dioxide absorber control knob. This state of affairs he now rectified. Once cardiac massage had been instituted a good continuing heart beat was established within 3 minutes, 9 minutes after the diagnosis of cardiac arrest. An infusion of noradrenaline was commenced but the B.P. was maintained without this after a further 20 minutes. The thoracotomy wound was closed.

At the conclusion of operation the patient's B.P. was 200 mm.Hg systolic and the pulse rate 140/minute. Following the conclusion of the operation, she failed to regain consciousness and it was obvious that she had sustained severe brain damage. After return to the ward she was treated by hypothermia, temperature being reduced to 90°F, together with dehydration therapy, intravenous 20% sucrose and fluid restriction. She died on the 4th post-operative day without regaining consciousness.

AUTOPSY:

Heart was not enlarged, weighing 280 gm. and showed no abnormality. There was some atheroma of the aorta immediately above the aortic valve and atheroma of both coronary arteries. The rest of the organs appeared normal. Cause of death undetermined but not inconsistent with being due to the effects on the brain of anoxia, which occurred

as / ...

as a result of reflex cardiac arrest while the deceased was under a general anaesthetic. No histological examination of the brain was performed.

COMMENT:

This patient's death from the sequelae of gross cerebral anoxia resulted directly from the maladministration of a general anaesthetic. It illustrates tragically the grave results that may follow momentary inattention on the part of the anaesthetist.

Briefly, the sequence of events in this were as follows:- Induction of anaesthesia with thiopentone, nitrous oxide, oxygen, ether sequence, administered via a Coxeter Mushin circle system with the absorber turned off, the ether being administered via the Boyle bottle on the fresh gas in flow, the patient breathing spontaneously. Following oral intubation, a relaxant was administered, and IPPR instituted. The intention of the anaesthetist was at the same time to bring the carbon dioxide absorber cannister into the circuit. Inadvertently the control of the Coxeter Mushin ether vaporizer on the opposite side of the machine was turned on to maximum instead of the carbon dioxide absorber. This was not noticed. The Coxeter Mushin ether vaporizer is very efficient, especially within the circle circuit. Together with ether still administered on the fresh gas inflow, this will have resulted in a gross overdose of ether being administered to the patient, although the fresh gas inflow was high, 10 litre/minute. The paralysis resultant on the injection of a muscle relaxant must have rendered the early signs of such an overdose largely overt, though one wonders that no drop in B.P. was noticed. Only on the occurrence of a cardiac arrest 11 minutes later was it realised that anything was amiss. The fatal hesitancy of the surgeon in instituting cardiac massage resulted in an avoidable delay of 6 minutes from the time of diagnosis of cardiac arrest to the commencement of massage. This compounded the existing cerebral ischaemia and anoxia and virtually assured a fatal outcome. The surgeon's culpability, however, cannot be regarded as lessening the responsibility of the anaesthetist.

This case illustrates the greater clinical observation necessary with the use of relaxants in conjunction with other potent anaesthetic agents, such as ether. The use of relaxant drugs completely masks all the obvious signs of deepening anaesthesia, such as the changing character of respiration. The maladministration of anaesthesia is regarded as the major factor in the causation of this patient's death, though more prompt action on the part of the surgeon may have avoided the calamity.

PREVENTABILITY:

In view of the above, this death must be regarded as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
131.1.59	2	No comment	< 24	Meningitis Brain abscess.	Yes

Name: Edward Martin. Age: 12 Sex: M Race: C

Disease: Brain abscess. Operation: Burrhole craniotomy. Aspiration of abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Four days previously the patient had been admitted with acute mastoiditis. A right mastoidectomy had revealed a large collection of pus in the main mastoid antrum. Subsequent to the operation the patient developed signs of meningitis and a lumbar puncture revealed pus in the C.S.F. His condition continued to deteriorate. At this time he was critically ill with a temperature of 104°F and a pulse rate of 140/minute. B.P. 120 mm.Hg systolic.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with a steep dose of thiopentone, 100 mg., followed by nitrous oxide, oxygen and gradually added ether, administered via a Magill circuit with spontaneous breathing. When anaesthesia had been established and following topical laryngeal analgesia, oral endotracheal intubation was performed. Anaesthesia was maintained throughout the operation with nitrous oxide and oxygen with a trace of ether.

The operation of burrhole craniotomy and aspiration of a right cerebral abscess took 80 minutes. Throughout this time the course of anaesthesia was uneventful. At the end of the operation and on discontinuance of the anaesthetic, the patient regained consciousness rapidly. He died 9 hours post-operatively.

AUTOPSY:

Huge collection of pus subdurally over the right cerebral hemisphere and in the right posterior fossa.

COMMENT:

This patient died from the effects of a cerebral abscess. Anaesthesia played no part in this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
132.1.59	2	No comment	< 24	Haemorrhage from carcinoma of pancreas invading the colon.	Yes

Name: Johannes Slinger Age: 41 Sex: M Race: C

Disease: Haematemesis. (Carcinoma of pancreas invading stomach and colon). Operation: Laparotomy.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Fair physical status. He had suffered a haematemesis before admission but the blood loss had been replaced and his condition was now fair the B.P. being 120 mm.Hg systolic, pulse rate 96/minute. The provisional pre-operative diagnosis was haemorrhage from a gastric ulcer.

PREMEDICATION:

Omnopon gr. 1/3, scopolomine gr. 1/150, administered 1 hour pre-operatively.

ANAESTHETIC:

Following gastric aspiration, anaesthesia was induced with thiopentone 250 mg. followed by 40 mg. succinylcholine, an IPPR technique with oxygen being instituted, and oral intubation. Following return of spontaneous respiration, an IPPR technique was instituted with gallamine as relaxant. Nitrous oxide and oxygen were administered with a trade of ether vapour via a circle absorption system. A total of 80 mg. gallamine was administered throughout the operation.

The operation lasted 45 minutes, and the course of anaesthesia was uneventful throughout. B.P. and pulse rate remained stable at 120 mm.Hg systolic and 108/minute respectively. Laparotomy revealed an inoperable, irresectable carcinoma of the pancreas infiltrating the stomach and colon. No surgical treatment was possible. The abdomen was closed and the operation terminated. At the conclusion of the procedure, spontaneous respiration returned and complete reversal of curarization was ensured by the administration of 0.5 mg neostigmine preceded by atropine gr. 1/100. Following discontinuance of the anaesthetic, consciousness was regained within 5 minutes. The patient was returned to the ward. Following the patient's return to the ward, gross haemorrhage per rectum indicated bleeding from the carcinoma into the colon. The patient died 12 hours after operation, of haemorrhage.

AUTOPSY:

Carcinoma of the pancreas infiltrating the transverse colon, stomach, spleen, left adrenal and kidney. There were carcinomatous secondary deposits in the paratracheal glands and liver. Gross gastro-intestinal haemorrhage had resulted.

COMMENT:

This death was due to haemorrhage from an inoperable, irresectable carcinoma of the pancreas which had eroded the stomach and colon. Anaesthesia was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
133.1.59	3	No comment	ORD	Cardiac arrest, myocardial anoxia.	Yes

Name: James B. Conradie Age: 36 Sex: M Race: E

Disease: Aortic stenosis. Operation: Aortic valvotomy on cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

This patient was suffering from severe calcific aortic stenosis. He was not in cardiac failure at this time, but was digitalised.

PREMEDICATION:

Seconal gr. 3, orally, 3 hours pre-operatively. Atropine gr. 1/100 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone, succinylcholine, IPPR with oxygen, endotracheal intubation, nitrous oxide and oxygen sequence. Following induction, an IPPR technique with nitrous oxide and oxygen was used including carbon dioxide absorption with a circle absorber. Induction and maintenance of anaesthesia were uneventful until the time of establishment of cardiopulmonary bypass. No relaxant was used until the establishment of bypass, whereupon 9 mg. dTc was used and intermittent doses of 100 mg. thiopentone.

During cardiopulmonary bypass, ventricular fibrillation occurred and thereafter - despite intensive measures to defibrillate the heart - normal cardiac action did not return.

AUTOPSY:

Fresh surgical scars on chest and legs. Aortic stenosis with calcification of the valves and left ventricular hypertrophy. Right pleural cavity contained 1.6 litre blood. Left pleural cavity contained a small amount of blood. Right and left lungs were both collapsed.

COMMENT:

This death appears to have resulted from surgical myocardial damage during cardiopulmonary bypass. Anaesthesia is not contributory but, as death occurred while the patient was anaesthetised, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
134.1.59	3	No comment	ORD	Haemorrhage. Myocardial ischaemia. Cardiac arrest.	Yes

Name: Atillo Penso Age: 64 Sex: M Race: E

Disease: Abdominal aortic aneurysm. Operation: Resection of aortic aneurysm and grafting.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

An extremely poor operative and anaesthetic risk. The patient had a large aortic abdominal aneurysm and gross generalised atherosclerosis. One month previously he had suffered a coronary thrombosis. Subsequently he had suffered from congestive cardiac failure, which was controlled by the administration of digitalis and though the patient was still on this therapy, he was still markedly dyspnoeic on exertion. The presence of severe persistent abdominal pain now indicated the possible impending rupture of the abdominal aneurysm. In spite of the grave risks entailed, it was decided to resect the aneurysm. B.P. 100 mm.Hg systolic, pulse rate 96/minute.

PREMEDICATION:

Omnopon gr. 1/3, scopolomine gr. 1/150.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 250 mg. followed by 30 mg. succinylcholine, and IPPR with oxygen, topical analgesia of the larynx and oral intubation following. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether vapour, administered via a circle absorption system. Five minutes after the administration of succinylcholine, spontaneous respiration resumed. An initial dose of 60 mg. gallamine was given and an IPPR technique instituted. Induction of anaesthesia caused no change inthe patient's B.P. but provoked a shortlived drop in the pulse rate to 60/minute, which subsequently reverted to 96/minute.

The operation was prolonged and difficult, lasting 7 hours, and was punctuated throughout by marked oscillations in B.P. though the average pressure for the greater part of the time was approximately 130 mm.Hg systolic. These oscillations were caused successively by intermittent vena caval compression during resection of the aneurysm, which was adherent to the inferior vena cava, and subsequently to alternating blood loss and replacement of blood, and to clamping and subsequent release of the aorta. Following an initial clamping of the aorta of 70 minutes, preceding which 150 m. heparin was injected into the distal aorta, a further period of 30 minutes' clamping was necessitated by the need to tailor the aortic graft, which was found to be too long. This shortening of the graft and the subsequent unclamping of the aorta was followed by severe haemorrhage and a steady deterioration in the patient's condition, in spite of what was thought to be adequate blood transfusion, a total of 10 pints together with which 2 gm. calcium gluconate having been given. During the entire operation a total of 360 mg. gallamine had been administered. During resuturing of the abdomen, normal spontaneous respiration recommenced and at the end of the procedure, though the respiration was adequate, a dose of 1 mg. neostigmine preceded by gr. 1/100 atropine was given for complete reversal of curarization. The B.P. was now 100 mm.Hg systolic. The patient appeared very pale. This was diagnosed as being due to further loss of blood and another 4 pints blood was transfused over the next hour, with no effect on the B.P., bringing the total of blood

transfused / ...

transfused to 14 pints. Despite this, the B.P. continued to fall and rapidly became unrecordable post-operatively, though the femoral and brachial pulses could be palpated. An infusion of noradrenaline was commenced and 100 mg. hydrocortisone administered. Within ½ hour of the conclusion of the operation the patient's pupils became widely dilated. An ECG taken at this stage showed marked ischaemic changes over and above those shown on the pre-operative ECG. The patient failed to regain consciousness, though ventilating pure oxygen, and all anaesthetic agents had been discontinued at the end of the operation.

One hour after the conclusion of surgery (75 minutes after the recommencement of normal spontaneous respiration), the respiration commenced failing and became gasping. IPPR was recommenced with pure oxygen. However, the deterioration continued and 3 hours after the end of operation resuscitative measures were abandoned in view of the prolonged period during which there had been no recordable B.P., dilated pupils and lack of return of consciousness. Cardiac arrest ensued 15 minutes later. No cardiac massage was attempted.

AUTOPSY:

Heart enlarged, left ventricle dilated. Heart weighed 700 gm. Marked atherosclerosis of both coronary arteries. Subendocardial fibrosis. Marked atherosclerosis of aorta and large blood vessels. Peritoneal cavity contained 1,500 ml. blood. Large retroperitoneal haemorrhage.

COMMENT:

This death appears to have resulted primarily from the effects of massive and continuing haemorrhage, massive transfusion, probable metabolic acidosis and possibly inadvertent hypothermia. By today's standards, the anaesthetic management may be criticised on the failure to correct the metabolic acidosis that must inevitably have been present. At this date the importance of this aspect was not fully realised, nor were the facilities for rapid biochemical diagnosis and monitoring available. Also by present day standards, the administration of an infusion of adrenaline orisoprenaline would be considered more efficacious than the vasopressor noradrenaline in maintaining the failing cardiac output. Furthermore, the dose of calcium gluconate administered might be considered too small to effectively neutralise the effects of the amount of citrate included in the 14 pints of blood transfused. However, the basic cause of this death was the massive blood loss and, though the omissions detailed may be considered as contributory to the final outcome, the anaesthetic technique is not considered to have played a significant contributory role in causing the death of this patient but rather to have been "necessarily contributory".

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
135.1.59	1	Probably	< 24	Inhalation of vomitus Anoxic anoxia. Circulatory failure.	Yes

Name: Anna de Gourville Age: 74 Sex: F Race: E

Disease: Fractured neck of femur. Operation: Insertion of Smith Peterson pin and plate.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Though classified by the surgeons as in a fair state, the anaesthetist did not share this opinion. The patient was old and frail and appeared mildly dehydrated. She had atherosclerosis and was hypertensive, B.P. 190 mm.Hg systolic. Haemoglobin 12.5 gm.%. She appeared drowsy and answered questions rather hazily. The anaesthetist attributed this to senility. Though not known to the anaesthetist, at this time, it subsequently came to light that the patient had been vomiting since admission 4 days previously, and more severely on the day preceding surgery. Because of this the nursing staff had stopped her feeding this day. Strangely enough, the medical staff concerned were unaware of this.

PREMEDICATION:

Pethidine 25 mg., phenergan 25 mg., atropine gr. 1/100 administered 1 hour pre-operatively.

ANAESTHETIC:

On arrival intheatre, the B.P. was 120 mm.Hg systolic, and pulse rate 108/minute. At this time the anaesthetist attributed this drop from the ward level (190 mm.Hg systolic) to the administration of phenergan and pethidine, albeit in small doses, as premedication. Anaesthesia was induced with thiopentone while the patient lay on the trolley on which she had arrived in theatre, a dose of 50 mg. being given. This did not induce sleep and a further 50 mg. was given, inducing a light anaesthetic sleep. The patient was moved onto the Spiker table and responded to pain at this stage by facial wincing. On the table, the B.P. was 110 mm.Hg systolic and pulse rate was 108/minute. Nitrous oxide and oxygen were administered via a Magill circuit, with spontaneous breathing. Ether vapour was gradually added but resulted in laryngospasm and the anaesthetic gases were discontinued and only oxygen administered. Laryngospasm relaxed. Administration of nitrous oxide and oxygen was recommenced and ether vapour was again gradually added. The patient was now being secured and positioned on the Spiker operating table. A further episode of laryngospasm ensued due either to the recommencing addition of ether, or perhaps to pain from manipulation of the hip whilst the patient was inadequately anaesthetised. Anaesthetic gases were again discontinued and oxygen only administered. Laryngospasm again relaxed but immediately thereafter regurgitation of a cupful of black, printers-ink-like fluid gastric content occurred. The operating table to which the patient was now secured was non-tipping. The patient's head and shoulders were rotated off the table to one side, as much as possible. Regurgitated material was aspirated from the mouth and pharynx with a sucker. No sooner had the patient been returned to the supine position that another large quantity of black fluid, in excess of a cupful, regurgitated. Her head and shoulders were again turned off the table and the liquid was aspirated from the mouth and pharynx with a sucker. It was now suspected that some of the vomitus had been aspirated into the bronchi and lungs. The patient became cyanosed for about 1 minute at this stage. A no. 9 endotracheal tube was passed immediately and the bronchi were

aspirated / ...

aspirated through this tube. Gastric content was obtained. After this, breath sounds were clear on auscultation. A bronchoscopy was planned but a bronchoscope was not immediately available. The bronchi were again aspirated by means of a suction catheter passed down the endotracheal tube. The chest was now clear to auscultation, the patient breathing adequately and spontaneously, and with no central cyanosis. This entire episode had lasted approximately 5 minutes, during which time pure oxygen was administered. After 5 minutes a trace of ether vapour was added. The B.P. was now 80 mm.Hg systolic, pulse rate 108/minute. Marked peripheral cyanosis was present. Capillary refill time was slow. She was observed for a further 5 minutes but as the B.P. remained at the low level, 2 doses of 6 mg. methyl amphetamine were administered intravenously. The B.P. slowly rose to 100 mm.Hg systolic.

It was decided to continue with the operation as quickly as possible in spite of the patient's parlous state and this was done 15 minutes after this episode of vomiting and regurgitation. In the absence of a bronchoscope, bronchoscopy was deferred until the end of the operation. The operation took 50 minutes. For the initial 15 minutes anaesthesia was maintained with oxygen and ether, with spontaneous respiration. For the remainder of the operation, anaesthesia was maintained with 50% nitrous oxide and oxygen. Blood was replaced as lost, a total of 200 ml. blood being transfused. The B.P. remained at a level of 100 mm.Hg systolic, pulse rate of 108/minute. At the conclusion of the operation, while the wound was being stitched, anaesthesia was discontinued and bronchoscopy was performed. The bronchi were clear. At the end of the operation the B.P. again fell, to a level of 60 mm.Hg systolic this time. Methyl amphetamine 6 mg. intravenously followed by 100 mg. cortisone was administered. The B.P. rose to 90 mm.Hg systolic. An infusion of noradrenaline 4 microgm. per cc. was commenced and the B.P. rose to 110 mm.Hg systolic. The patient was returned to the ward breathing spontaneously and adequately. However, peripheral cyanosis with slow capillary refill time was present. Consciousness returned to a level where she responded to painful stimuli but was not fully regained. In the ward the patient was nursed with a head-down tilt, and noradrenaline infusion was continued. Cyanosis with slow capillary refill time persisted. In spite of resuscitative measures, the patient's condition deteriorated and she died 6½ hours post-operatively.

AUTOPSY:

Fractured femur with prosthesis in situ. Thickening and calcification of the aortic valves. Atheroma of aorta and coronary arteries, although not apparently encroaching on the lumen of the coronary vessels. Three dark stained patches in the stomach, which contained 200 ml. of dark fluid content. Intestines contained more of the same sort of fluid. Lungs were clear. Cause of death undetermined.

COMMENT:

Failure to recognise that this patient was suffering from ileus was the outstanding error in the management of this case. What followed stems largely from this. No fluid and electrolyte replacement were undertaken. No gastric aspiration was instituted. Immediately before anaesthesia, the patient was doubtless suffering from fluid and electrolyte depletion and had a full stomach as well. Clinical signs of this were present but were overlooked or misinterpreted.

The precipitation of vomiting and/or gastric regurgitation by laryngospasm in the presence of a full stomach is well known. The second and most serious episode of regurgitation followed laryngospasm provoked by the painful stimulus of manipulation of the fractured femur, in all probability, which was permitted by the anaesthetist while anaesthesia was too light. It may, however, have been related to the introduction of ether vapour into the inhaled gases. Once regurgitation occurred, treatment was correct but in spite of this the patient inhaled gastric content. Although this appears to have been cleared

adequately / ...

adequately, the patient definitely suffered a period of anoxicanoxia at this time. Peripheral circulatory failure ensued. With conditions present that could be productive of both anoxic and stagnant anoxia, subsequent interpretation of the cyanosis was difficult. No bronchospasm followed the inhalation of vomitus. In retrospect, in view of her probable hypovolaemia, the treatment of choice for the circulatory failure with vasopressor drugs was also incorrect.

Post-operatively it is possible that tracheostomy and augmented ventilation with oxygen may have improved this patient's chances of survival. The failure to regain consciousness indicates that between the initial anoxic anoxia and the later ischaemic anoxia of prolonged hypotension, marked cerebral damage may have occurred.

Though exonerated to some extent by the inadequate pre-operative surgical preparation, the anaesthetic management is considered a significant contributory factor in the patient's death.

PREVENTABILITY:

The errors in the surgical and anaesthetic management enumerated above are all correctable. Though the ultimate prognosis following fractures of the femur in frail old patients such as this one is not good, this death is regarded - certainly in terms of immediate operative survival - as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
136.1.59	2	No comment	< 24	Multiple injuries. Ruptured liver. Haemothorax.	Yes

Name: Fanie Ngobo. Age: 30 Sex: M Race: B

Disease: Multiple injuries: fractured ribs, lacerated thigh. Operation: Suture of lacerated thigh.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Fair. Blood transfusion had been administered. The B.P. was 115/75 mm.Hg, pulse rate 80/minute. There were fractures of the 7th, 8th and 9th ribs on the right side but no flail chest. There was no pneumothorax or haemothorax.

PREMEDICATION:

Atropine gr. 1/100 administered immediately preceding the induction of anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with thiopentone, totalling 225 mg., in divided doses. Nitrous oxide and oxygen was then administered via a Magill semi-open circuit with the patient breathing spontaneously. Ether vapour was gradually added. Following induction of anaesthesia there was a drop in the B.P. from 120 mm.Hg systolic to 90 mm.Hg systolic. Methyl amphetamine was administered intravenously, which raised the B.P. to 120 mm.Hg systolic.

Throughout the operation, blood was transfused as lost. The operation, which took 65 minutes, consisted of debridement and suture of the lacerated thigh injury. At the conclusion of the procedure and on discontinuance of the anaesthetic, the patient regained consciousness and was breathing normally. The patient died 4 hours post-operatively.

AUTOPSY:

Fractured ribs on the right side. Haemothorax of 1 litre blood on the right side. Ruptured liver with haemoperitoneum of 500 ml. blood. Sutured wound of right thigh.

COMMENT:

This patient obviously died as a result of multiple injuries. From the autopsy findings, a large haemothorax on the right side with a haemoperitoneum from a ruptured liver, both of which appear to have been undiagnosed ante-mortem, one wonders what part continuing haemorrhage played in this patient's death. Anaesthesia appears not to have played any contributory role in the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
132.1.59	2	No comment	< 24	Myocardial infarction Cardiac arrest.	Yes

Name: William Donaldson Age: 65 Sex: M Race: E

Disease: Post-operative disruption of abdominal wound following cholecystectomy.

Operation: Resuture of disrupted abdominal wound.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient had had a cholecystectomy 10 days previously. His history at that time revealed a previous episode of coronary infarction and an ECG taken before that operation showed evidence of ischaemic damage to the left ventricle. He was moderately emphysematous and markedly atherosclerotic. The first post-operative week was complicated inter alia by purulent bronchitis. On the 9th day the patient suffered from a severe cardiovascular collapse; though the aetiology of this was not finally decided, it was possibly due to sodium depletion. On the 10th post-operative day he ruptured his abdominal wound, becoming rapidly markedly shocked with gross peripheral vasoconstriction. His condition deteriorated rapidly. Immediately pre-anaesthetic his condition was poor; pulse rate 96/minute, B.P. 130 mm.Hg systolic (it had been 170 mm.Hg systolic 1 hour previously), and he appeared shocked and pale with severe peripheral vasoconstriction. He had purulent bronchitis with profuse secretion.

PREMEDICATION:

Atropine gr. 1/100 administered 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen administered via a closed circle absorption system. After 3 minutes, 25 mg. succinylcholine was administered and oral intubation performed. IPPR was performed for a further 5 minutes until the return of spontaneous respiration. Anaesthesia was then continued with nitrous oxide and oxygen. Bronchial toilette was performed via the endotracheal tube by suction aspiration before the operation commenced. This was repeated during the operation at intervals. At the commencement of operation, a further 25 mg. succinylcholine was administered and IPPR continued. Induction of anaesthesia caused no drop in the B.P. from the pre-operative level.

The operation, which consisted of rapid exploratory laparotomy and resuture of the dehisced abdominal wound, lasted 35 minutes. During this time the B.P. oscillated between 110 and 130 mm.Hg systolic. Pulse rate 96/minute. The patient remained peripherally very vasoconstricted. At the conclusion of operation good spontaneous respiration returned. Bronchial and pharyngeal toilette were performed, and the patient extubated, after a stomach tube had been passed and 100 ml. fluid aspirated. The patient rapidly regained consciousness on discontinuance of anaesthesia and was removed from the operating table onto his bed in theatre, and the bed wheeled out into the corridor. Here, 26 minutes post-operatively, he suddenly gave two gasps and stopped breathing. Cardiac arrest was immediately diagnosed. Artificial respiration with oxygen and subsequently oral intubation were performed. Cardiac massage was commenced via a left thoracotomy within 3 minutes. Cardiac massage was of no avail. The heart was flabby and the injection of 2 doses adrenaline 0.5 mg. into the left ventricle had no effect at all.

AUTOPSY:

Surgical abdominal and left thoracotomy wounds. Two drains from gall bladder and adjacent area. Mucopurulent discharge from bronchioles.

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Coronary atherosclerosis. The cause of death was undetermined but not incompatible with that of coronary atherosclerosis with occlusion. The lumen of the left coronary artery was obliterated 1 cm. from the aorta. The right coronary artery was markedly atherosclerotic. The myocardium had two large scars on the left ventricle wall, 1 cm. in diameter each. Marked atherosclerosis of the ascending aorta.

COMMENT:

Cardiac arrest in this patient would appear to have resulted from myocardial ischaemia, superimposed on a previous myocardial infarction. It is probable that a state of shock following the dehiscence of the abdominal wound caused further myocardial ischaemia which resulted in his death. Anaesthesia per se does not appear to have resulted in any worsening of his condition; the B.P. was stable throughout operation and pulmonary ventilation good during the IPPR technique used. The spontaneous respiration which returned at the conclusion of anaesthesia was normal and of adequate volume. The bronchial toilette performed during the anaesthesia and at its conclusion would in fact have resulted in better pulmonary ventilation post-operatively than was possible before. With his death occurring in such close juxtaposition to the termination of the anaesthetic, it might be argued that anaesthesia should be considered to have been necessarily contributory to this death. However, this is an academic point. As the patient's condition was reasonably static during the anaesthetic, the patient rapidly regaining consciousness afterwards, this death is regarded as resulting from the patient's pre-existing myocardial infarction with added ischaemia resulting from shock of dehiscence of the abdominal wound.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
138.1.59	2	No comment	< 24	Acute pancreat-itis.	No

Name: Janey Brown Age: 18 Sex: F Race: C

Disease: Acute pancreatitis. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was considered moribund by the anaesthetist. In the ward the B.P. was unrecordable, though immediately pre-anaesthetic it was recorded at 90 mm.Hg systolic. There was gross toxaemic shock. The patient was anuric and stuperose. Pre-operatively 1 litre plasma and 3 pints blood were administered.

PREMEDICATION: Nil.

ANAESTHETIC:

1 cc. 4% Lignocaine was administered transtracheally. Following 3 minutes of pre-oxygenation, nitrous oxide and oxygen were administered via a Magill semi-open circuit with the patient breathing spontaneously. After 2 minutes, oral intubation was performed, using a No.9 cuffed endotracheal tube. Thereafter, until the end of operation - which lasted 100 minutes - anaesthesia was maintained with nitrous oxide and oxygen administered via a semi-closed system with the patient breathing spontaneously.

During the operation 2 pints blood were transfused together with the administration of 100 mg. hydrocortisone and 1 gm. calcium gluconate. During the procedure the patient's B.P. rose gradually from 90 to 140 mm.Hg systolic at the conclusion. Laparotomy revealed an exceedingly gross acute pancreatitis. At the end of operation a tracheostomy was performed. The patient's level of consciousness after discontinuance of anaesthesia was the same as it had been pre-operatively. The patient died 7½ hours later.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of her pre-existing disease. Anaesthesia is not considered to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
139.1.59	2	No comment	< 24	Haemorrhage from carcinoma of the stomach.	No

Name: Johannes Hendricks Age: 63 Sex: M Race: C

Disease: Haematemesis Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

In extremis. The patient had had severe haematemesis and 8 pints blood had been transfused in the 1½ hours immediately pre-operatively. At this stage his B.P. was 90 mm.Hg systolic with a pulse rate of 130/minute. He still appeared to be bleeding. Immediately before induction of anaesthesia he vomited a further 800 ml. bright red blood. A further disability from which he suffered was pulmonary emphysema.

PREMEDICATION: Nil.

ANAESTHETIC:

Following the transtracheal injection of 1 ml. 4% Xylocaine, anaesthesia was induced with nitrous oxide and oxygen administered via a circle absorption system. After 1 minute, oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen with 10-20% cyclopropane, administered via a circle absorption system, with spontaneous respiration throughout.

During the operation a further 3 pints blood and 20 ml. 10% calcium gluconate were administered. At laparotomy a completely inoperable carcinoma of the stomach was found, with gross invasion of the liver. No further surgical treatment was attempted and the abdomen was closed. The operation lasted 40 minutes. At the conclusion of operation and on discontinuance of anaesthesia, the B.P. was 100/60 mm.Hg and the patient rapidly regained consciousness. Respiration was normal. He died 5 hours post-operatively following further haemorrhage.

AUTOPSY:

No autopsy.

COMMENT:

This patient died as a result of continued haemorrhage from an inoperable carcinoma of the stomach. Anaesthesia was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
140.1.59	1	Possibly	< 24	Anoxic anoxia. Post-relaxant respiratory abnormality. Acute pancreatitis.	Yes

Name: Nicolas Boshoff Age: 41 Sex: M Race: E

Disease: Acute pancreatitis. Pancreatic abscess. Peritonitis.

Operation: Laparotomy. Drainage of peritonitis and pancreatic abscess.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was very ill with gross toxaemia. B.P. 100/80 mm.Hg, pulse rate 108/minute. On X-ray of the right diaphragm, this was shown to be raised - apparently by intra-abdominal pressure. Pre-operatively one pint blood and 2 pints plasma had been transfused.

PREMEDICATION:

Omnopon 1/3 gr., atropine gr. 1/100, administered 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. in divided doses followed by 25 mg. succinylcholine. Ventilation with oxygen, topical analgesia of the larynx and oral intubation were performed. Anaesthesia was continued with nitrous oxide and oxygen with minimal ether, by IPPR via a circle absorption system until the return of spontaneous respiration, the latter being allowed to persist until the peritoneum was opened, whereupon 80 mg. gallamine was administered and an IPPR technique instituted. Induction of anaesthesia was untoward and caused no change in the patient's condition.

At laparotomy 3 pints pus were aspirated from the peritoneal cavity. Underlying this an extremely gross acute pancreatitis was found. Abdominal drainage was instituted and the abdomen was closed. The operation lasted 75 minutes during which time the patient's condition remained static. Following closure of the abdomen the patient remained apnoeic. Respiration recommenced after the administration of 5% carbon dioxide in the inhaled anaesthetic mixture for some minutes, but with a marked tracheal tug. Atropine gr. 1/100 was now administered followed by 1 mg. neostigmine. The tracheal tug became somewhat less but was still definitely present. A further 2 doses of neostigmine 0.5 mg. were administered but made no difference to the tracheal tug. On conclusion of the operation anaesthesia was discontinued and the patient regained consciousness within a few minutes. Extubation was performed. At this stage he was wide awake and could fully open his eyes. Though a tracheal tug was still present, his respiration appeared of somewhat adequate tidal volume and he was permitted to return to the ward. Throughout operation there had been a tachycardia of between 120 and 140/minute. During operation 1 pint of blood had been transfused. 30 minutes after his return to the ward the patient was noticed to be apnoeic and pulseless. Immediately cardiac massage and artificial respiration were commenced, but to no avail.

AUTOPSY:

Gross pancreatitis with purulent peritonitis. Pericarditis with fibrinous exudate.

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COMMENT:

There was more than sufficient cause for this patient's death in the existing disease. However, one is faced with the fact that before operation, though extremely ill, his respiration was normal. Following operation and anaesthesia, the conduct of which cannot be faulted and during which circulatory homeostasis was adequate, his respiration was abnormal in that it manifested a tracheal tug, though apparently of adequate volume. Within 30 minutes of his return to the ward, he was dead.

The return of the patient to the ward while he manifested an abnormal pattern of respiration is open to criticism, especially as post-operative supervision in the ward does not appear to have been adequate. Reversal of curarisation may not have been adequate, or some degree of recurarisation may have occurred. In these circumstances, hypoventilation and anoxic anoxia may have resulted from persistent neuromuscular block or from pharyngeal respiratory obstruction which the patient, because of muscular weakness, was unable to overcome.

In the circumstances, the provision of continued IPPR until the respiratory pattern was normal, or even of oxygen, may have prevented death at this time. In retrospect, the probable reason for the persistence of abnormal respiratory pattern and probable hypoventilation was gross metabolic acidosis. At the period in question, however, the importance of the rapid diagnosis and correction of this condition was not fully realised as it is today, nor were rapid diagnostic facilities available. Another possible reason for the persistence of curarisation was the use of gallamine in a patient with poor urinary excretion. This is less likely than the former reason.

Because this patient was returned to the ward while still exhibiting an abnormal respiratory pattern after general anaesthesia and the use of a muscle relaxant, the anaesthetic management is regarded as contributory to this death, in spite of the gravity of the ultimate prognosis.

PREVENTABILITY:

In consideration of the shortcomings in the management of the immediate post-anaesthetic period, this patient's death is regarded as possibly preventable in terms of immediate operative survival.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
141.1.59	2	No comment	< 24	Subarachnoid haemorrhage. Cerebral oedema.	Yes.

Name: Geoffrey Peterson. Age: 24 Sex: M Race: E

Disease: Cerebral injury. Subdural haematoma.

Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Comatose. Pulse rate 54/minute, B.P. 150/70 mm.Hg, respiration 20/minute Cheye-Stokes in character. Besides head injury, the patient also had bilateral fractures of the radius and ulna.

PREMEDICATION:

Atropine gr. 1/100 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered via a Magill semi-open system, the patient breathing spontaneously. Following topical analgesia of the larynx, oral intubation was performed and anaesthesia maintained with nitrous oxide, oxygen and ether via the Magill circuit, with spontaneous breathing.

The course of anaesthesia was uneventful. At operation, which lasted 120 minutes, a subdural haematoma was evacuated. At the conclusion of the operation the patient's level of consciousness was the same as pre-operatively. The patient died 21 hours post-operatively.

AUTOPSY:

Bilateral subarachnoid haemorrhages with a small subdural haemorrhage and there was marked cerebral oedema.

COMMENT:

This patient died of an existing cerebral injury and subarachnoid haemorrhage. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
142.1.59	2	No comment	< 24	Subdural haemorrhage	Yes

Name: Stoffel Klein Age: 65 Sex: M Race: C

Disease: Subdural haematoma. Operation: Carotid angiography. Burrhole craniotomy and evacuation of subdural haematoma.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Comatose. The patient had signs of intracranial haemorrhage and a right hemiplegia. Breathing was shallow, 50/minute. Temperature 102°F, pulse rate 130/minute, and B.P. 90 mm.Hg systolic.

PREMEDICATION:

Atropine gr. 1/100 administered 45 minutes pre-operatively.

ANAESTHETIC:

Following induction of anaesthesia with nitrous oxide and oxygen with gradually added ether, administered via a Magill semi-open system, with spontaneous breathing, the larynx was topically anaesthetised and oral intubation performed. Anaesthesia was maintained with nitrous oxide, oxygen and trace of ether, using the same method.

A fall in B.P. from the pre-anaesthetic level of 90 mm.Hg systolic to 60 mm.Hg systolic followed induction of anaesthesia. Administration of two doses methyl amphetamine, 10 mg. each, intravenously, elevated the B.P. to 90 mm.Hg systolic again. Subsequently, a drip infusion of noradrenaline 4 microgm./cc. was commenced and was continued throughout the operation. The B.P. remained at a level of 90 mm.Hg systolic. A large subdural haematoma was found on the left side at operation, and this was evacuated. Following discontinuance of anaesthesia, which lasted110 minutes, the level of the patient's consciousness was the same as it had been before operation. The patient died 22 hours post-operatively.

AUTOPSY:

Remains of a large subdural haematoma which had been surgically evacuated on the left side of the brain. This had caused gross compression on the left side of the brain. The brain was atrophic and small in size relative to the capacity of the skull.

COMMENT:

Though circulatory collapse followed the administration of an anaesthetic to this patient, the reason seems to be more than adequately supplied by the nature and extent of the disease rather than by the anaesthetic. The circulatory collapse was treated by effective means. The patient survived into the post-operative period well beyond the stage at which the inhalation anaesthetic he had received would have had any further effect. Death was the result of the existing cerebral lesion.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
143.1.59	2	No comment	< 24	Periton-itis.	No

Name: David Vermaak Age: 78 Sex: M Race: C

Disease: Acute appendix; rupture of appendix. Peritonitis.

Operation: Laparotomy. Deflating enterostomy and drainage of peritoneal cavity.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was extremely ill and toxic. Temperature 97°F, pulse rate 130/minute, B.P. 110 mm.Hg systolic. A Ryles tube was passed pre-operatively and gastric lavage performed.

PREMEDICATION:

Atropine gr. 1/100 55 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen administered via a Magill semi-open circuit with gradually added ether. During induction of anaesthesia there was an episode of vomiting; this was slight. The pharynx and mouth were sucked clear and no vomitus was inhaled. Following induction of anaesthesia, oral intubation was performed and anaesthesia maintained throughout operation with nitrous oxide and oxygen with ether using the Magill semi-open circuit, the patient breathing spontaneously. The respiratory rate which initially was 36/minute, slowed following the induction of anaesthesia, to 28/minute. The B.P. remained reasonably constant at between 105 and 110 mm.Hg systolic. During the operation the pulse rate, which initially was 130/minute, slowed progressively to a final level of 96/minute and this was maintained.

Laparotomy revealed gross diffuse peritonitis following the rupture of an appendix abscess. A deflating enterostomy was performed and drainage of theperitoneal cavity instituted. The operation lasted 85 minutes. At the conclusion of the procedure, following the discontinuance of anaesthesia, the patient regained consciousness. Subsequently, in the ward, the B.P. reached a level of 140/90 mm.Hg. The patient died 9 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient appears to have died as a result of his diffuse peritonitis. Anaesthesia does not appear to have played any part in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
144.1.59	2	No comment	< 24	Cerebral neoplasm. Malignant astra-cytoma.	No

Name: Jacobus Smit Age: 65 Sex: M Race: E

Disease: Intracranial space-occupying lesion.

Operation: Carotid angiography. Burrhole craniotomy and biopsy of intracranial tumour.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was in a poor condition. He was semi-comatose having previously showed signs of an intracranial space-occupying lesion. Pulse rate 68/minute, B.P. 130/60 mm.Hg. Respiration 22/minute.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes pre-operatively.

ANAESTHETIC:

Following 4 minutes pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen with gradually added ether administered via a Magill semi-open circuit, with spontaneous breathing. After topical analgesia of the larynx, oral intubation was performed anaesthesia subsequently being maintained with nitrous oxide and oxygen with a trace of ether. Spontaneous respiration was allowed throughout. Anaesthesia was uneventful.

Carotid angiography was performed and revealed a large space-occupying lesion. Burrhole craniotomy revealed a tumour, probably a malignant astracytoma. A biopsy was taken. Following the conclusion of the operation, which lasted 90 minutes, and the discontinuance of the anaesthetic, the level of the patient's consciousness was the same as pre-operatively. The patient died 6½ hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient's death resulted from the intracerebral neoplasm. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
145.1.59	2	No comment	< 24	Haema-temesis. ?Adrenal failure.	No

Name: Elizabeth van der Spuy Age: 38 Sex: F Race: E

Disease: Gastric ulcer with catastrophic haematemesis. Operation: Laparotomy. Partial gastrectomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

4 months previously the patient had had a bilateral total adrenalectomy for Cushing's syndrome. She had subsequently had a daily maintenance dose of cortisone. A gastric ulcer developed and she was now suffering from a gross haematemesis from this ulcer. Two hours preceding admission, she had vomited 12 pints blood and intestinal content, and was admitted in a state of extreme collapse. Pulse rate 140/minute, B.P. 80 mm.Hg systolic, respiration 30/minute. A rapid transfusion of 8 pints blood via 2 intravenous routes with 15 gauge needles was given pre-operatively. Restoration of the B.P. to 100 mm.Hg systolic was followed on three further occasions by massive haematemesis. 125 mg. hydrocortisone was administered intravenously on admission. It was decided to proceed with operation in view of the continuing severe haemorrhage.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by 30 mg. succinylcholine and oxygenation. Oral intubation followed and anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system, using a trace of ether. An IPPR technique was used. An initial dose of 80 mg. gallamine was administered followed by 2 subsequent doses of 20 mg. each, the total throughout the operation being 120 mg.

A rapid laparotomy was performed and control of the bleeding point achieved. Rapid improvement of the circulatory condition was then obtained and maintained with rapid transfusion, the B.P. rising to 140 mm.Hg systolic. 100 mg. hydrocortisone was given during the procedure. After securing the bleeding point, a partial gastrectomy was performed; the operation lasted 140 minutes. At the conclusion of operation atropine gr. 1/100 was administered followed by 1½ mg. neostigmine. Normal spontaneous respiration returned and after the conclusion of anaesthetic the patient rapidly regained consciousness. She left the operating theatre with a B.P. of 120 mm.Hg systolic, pulse rate 120/minute, respiration 25/minute. Pari passu with the administration of blood during the operation, a total of 60 cc. calcium gluconate 10% was given. The patient's B.P. fell from 140 mm.Hg systolic to 100 mm.Hg systolic 4 hours post-operatively, and was restored by rapid transfusion of 150 cc. blood, but at this time a tachycardia of 160/minute was recorded. There was a sudden cardiovascular collapse 6 hours post-operatively and the patient died.

AUTOPSY:

No autopsy.

COMMENT:

The ultimate cause of death is open to surmise. In spite of the large doses of cortisone, a not unreasonable suggestion is that it was

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the result of hypo-adrenalism following the severe stress of the massive haematemesis followed by surgery in an adrenalectomised patient.

Anaesthesia is not considered a contributory factor in the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
146.1.59	3	No comment	ORD	Cardiac arrest. Cardiac manipulation.	Yes

Name: Salega Jacobs. Age: 40 Sex: F Race: C

Disease: Mitral stenosis. Saddle embolus of the aorta. Operation: Mitral valvotomy (planned aortic embolectomy, not performed).

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

In extremis - the patient suffered from tight mitral stenosis. Pulse rate 130/minute with auricular fibrillation. B.P. 140/90 mm.Hg. Haemoglobin 11.5 gm.%. Temperature 100°F. She had poor chest expansion and was extremely vasoconstricted. There was an occlusion of both iliac arteries following the impaction of a saddle embolus in the aorta. She was also severely diabetic and on admission had 3+ glycosuria and ketonuria. Mitral valvotomy to be followed by aortic embolectomy was planned. She was given 40 units of soluble insulin intravenously together with 1 mg. digoxin.

PREMEDICATION:

Morphine gr. 1/6, atropine gr. 1/100 one hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. administered over 3 minutes, followed by nitrous oxide and oxygen administered via a circle absorption system. Oral intubation was performed after 25 mg. succinylcholine had been administered and topical analgesia of the larynx performed. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether administered via a circle absorption system with IPPR.

Thoracotomy was performed and following atriotomy and evacuation of thrombus from the auricle, cardiac arrest ensued. Cardiac massage together with the use of adrenaline, hydrocortisone, glucose, sodium bicarbonate, calcium chloride and eventually the electrical pacemaker, all failed to restore an effective cardiac action.

AUTOPSY:

Brain: atrophy of left temporal lobe in region of insula. Weight 1005 gm. Chest: Incision left chest. Haemothorax, pleural effusion. Pericardial sac open. Left auricular appendage incised with proximal ligature in situ. Mitral valve had 2 opposed lacerations which when approximated showed valve to have been stenosed. Several slight epicardial haemorrhages.

COMMENT:

Clearly the main cause of this patient's death was her pre-existing disease and the cardiac surgery undertaken. In that cardiac arrest and death occurred whilst she was anaesthetised, anaesthesia is regarded as necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
147.1.59	3	No comment	ORD	Cardiac arrest. Incompatible blood transfusion. (Lab./Ward error).	Yes

Name: Alida Crous Age: 45 Sex: F Race: E

Disease: Cerebral neoplasm (Glyoblastoma multiforme). Operation: Craniotomy. Excision of glyoblastoma.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair although the patient was aphasic. She was classed by the anaesthetist as a grade 2 risk. B.P. 140/80 mm.Hg. Haemoglobin 11 gm.% Pulse rate 84/minute. Respiration 20/minute. Circulatory and respiratory systems were normal. Pre-operatively 2 pints blood had been transfused.

PREMEDICATION:

Pethidine 50 mg., phenergan 50 mg., atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone sodium 350 mg. followed by succinylcholine 50 mg, ventilation with oxygen, topical analgesia of the larynx and oral intubation. IPPR was continued with nitrous oxide, oxygen, via a Magill semi-open system until spontaneous respiration resumed within 5 minutes. Anaesthesia was then maintained with nitrous oxide and a trace of ether, with spontaneous breathing. Induction of anaesthesia was untoward except for a slight drop in B.P. from the pre-anaesthetic level to 120 mm.Hg systolic; pulse rate 120/minute. At the commencement of craniotomy the transfusion of the 1st pint blood was begun. When approximately 300 ml. blood had been transfused the B.P. dropped progressively to 80 mm.Hg systolic, pulse rate 92/minute. Blood transfusion was continued as the operation proceded. Within the next 15 minutes signs of peripheral circulatory stagnation became apparent. After a further 20 minutes the B.P. rose for a while to 110 mm.Hg systolic, the pulse rate still being 96/minute. At this stage, transfusion of a 2nd pint blood was begun. The B.P. again started to fall and was very soon 80 mm.Hg systolic again, the pulse rate being 110/minute. Blood transfusion was continued but the B.P. fell progressively reaching a level of 60 mm.Hg systolic after a further 15 minutes. Signs of peripheral circulatory stagnation became worse. No reason for this sudden deterioration could be seen. Blood transfusion was continued. After a further 25 minutes (approximately 1½ hours after the induction of anaesthesia) methylamphetamine in divided doses up to a total of 30 mg. was administered without response. An infusion of noradrenaline was then commenced, initially a concentration of 4 microgm./cc. being used but when this produced no response the concentration was increased to 12 microgm./cc. This also failed to produce any response. After a further 45 minutes 200 mg. cortisone was administered intravenously. Respiration began to fail. IPPR was commenced approximately 2½ hours after the induction of anaesthesia. There was now gross peripheral stagnant anoxia (with cyanosis) for which no good reason could be seen. Ventilation was normal. The correct gases were being used. No operative cause could be ascertained either for this sudden deterioration. After a further 40 minutes, cardiac arrest ensued. Cardiac massage was of no avail.

Subsequent investigation revealed a most unfortunate error in the cross matching of the patient's blood for blood transfusion. This

patient's / ...

patient's bed and place in the ward had been interchanged with that of another without the relevant folders being changed as well. Because of this, specimens of blood taken from these two patients had been incorrectly labelled, each getting the other's name. This error in identification of specimens sent to the blood transfusion laboratory had resulted in the patient receiving incompatible blood. This was doubtless the cause of her demise.

AUTOPSY:

Operation on left side of brain. Evidence of previous tappings. Signs of atherosclerosis of coronary arteries and around coronary ostia.

COMMENT:

This patient appears to have died from an acute anaphalactic reaction to incompatible blood transfusion. Because this error of wrong identification occurred at laboratory level, the blood sent to theatre for the patient was in fact labelled with her name. It was not possible at this time for the anaesthetist to be aware that the blood was incompatible. This was only revealed by subsequent investigation. Although blood transfusion comes into the sphere of responsibility of the anaesthetist, the error that resulted in this patient's death occurred at ward level and was not the anaesthetist's responsibility. This death is thus not classified as one due to the anaesthetic nor to matters over which the anaesthetist must assume responsibility. As death occurred while the patient was anaesthetised, the case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSEOF DEATH	AUTOPSY
148.1.59	2	No comment.	< 24	Intra-cerebral secondary carcinoma from primary in lung.	Yes

Name: Elizabeth Pheko Age: 35 Sex: F Race: B

Disease: Intracerebral neoplasm. Operation: Craniotomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor. The patient was stuperose and deteriorating rapidly. B.P. 145/95 mm.Hg. Respiration was occasionally periodic. Three days pre-operatively carotid angiography had been performed and ventricular drainage established.

PREMEDICATION: Nil.

ANAESTHETIC:

After 3 minutes pre-oxygenation, anaesthesia was induced with nitrous oxide, oxygen and gradually added ether vapour, administered via a Magill semi-open system with the patient breathing spontaneously. After topical analgesia of the larynx, oral intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen. Later, oxygen alone was administered. At one stage, respiration deteriorated to Cheyne-Stokes type and later became gasping. Triple strength plasma, 1 pint, was given intravenously with dramatic improvement in the respiration, which returned to normal spontaneous respiration. The patient started responding to painful stimuli and nitrous oxide was again administered with the oxygen. At operation, a bone flap was raised but the dura was not opened because it was was extremely tense. The brain was needled in the left parietal region and soft necrotic tumour was obtained. Opening the ventricular drainage catheter did not improve operative conditions. After the operation, which lasted 3 hours, the patient's condition was much as it had been pre-operatively. She died 12 hours post-operatively.

AUTOPSY:

A single large secondary carcinoma was found in the region of the angular gyrus of the left cerebral hemisphere. Marked oedema of the left side of the brain. The primary carcinoma was found in the apex of the right lung.

COMMENT:

This patient died as a result of the intracerebral secondary carcinoma. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
149.1.59	2	No comment	< 24	Multiple injuries. Extradural haematoma.	Yes

Name: Nico Marais. Age: 25 Sex: M Race: E

Disease: Multiple injuries; extradural haematoma. Left haemopneumothorax.

Operation: Carotid angiography. Left frontotemporal craniotomy. Evacuation of extradural haematoma.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Unconscious, the patient's left pupil was widely dilated and there was bilateral extensor plantar response. B.P. 190/110 mm.Hg, pulse rate 120/minute, respiration 40/minute. There was a left pneumohaemothorax.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

After topical analgesia of the larynx, oral intubation was performed. Oxygen only was administered for most of the operation. At times, when there appeared to be some movement in response to pain stimuli, nitrous oxide was administered with the oxygen as a 50% mixture. The patient breathed spontaneously throughout.

A left frontotemporal craniotomy was performed and a large extradural haematoma evacuated. At the conclusion of operation, which lasted 2½ hours, a Foley's catheter and underwater drainage of the left chest was established. The patient died 5 hours after operation without regaining consciousness.

AUTOPSY:

Abrasions and bruises of face and body. Evidence of surgical procedure on head. Ruptured lung with haemothorax and fractured ribs. Death due to multiple injuries.

COMMENT:

This patient died of the existing cerebral injury. Anaesthesia is not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
150.1.59	3	No comment	ORD	Uncontrollable haemorrhage. Cardiac arrest.	Yes

Name: Lionel Forman. Age: 30 Sex: M Race: E

Disease: Mitral stenosis and incompetence; tricuspid incompetence; aortic incompetence.

Operation: Mitral valvuloplasty on cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

He had been in chronic congestive cardiac failure for many years, as a result of rheumatic heart disease. B.P. 130/80 mm.Hg and he had auricular fibrillation. 5 finger hepatomegaly was present. Intensive medical therapywith digitalis and diuretics had been administered.

PREMEDICATION:

Seconal gr. 1, orally 3 hours before induction of anaesthesia; atropine gr. 1/100 intramuscularly 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine, oxygenation, topical analgesia of the larynx, and oral intubation. IPPR was continued with nitrous oxide and oxygen until spontaneous respiration had been resumed after 5 minutes. Thereafter, once the patient had been taken into the theatre and positioned on the table, anaesthesia was maintained with nitrous oxide and oxygen via a circle absorption system by an IPPR technique. dTc in doses of 3 mg. was used as therelaxant, totalling 12 mg. dTc in all. The course of the anaesthetic was relatively untoward until the commencement of cardiopulmonary bypass. During bypass anaesthesia was maintained with 5 mg. doses of thiopentone and 3 mg. doses of dTc following an initial dose of 9 mg. dTc at the commencement of bypass. No difficulties were experienced with the anaesthesia.

During bypass a mitral annuloplasty was performed. Difficulty was experienced with closure of a rent in the pulmonary vein and the right auricle, leading to gross uncontrollable haemorrhage following a very prolonged bypass. Death followed from exsanguination at the conclusion of the bypass.

AUTOPSY:

Chronic rheumatic heart disease with grossly dilated left auricle and thickening of the myocardium of the left ventricle, plus incompetence of mitral, aortic and tricuspid valves. Evidence of surgical procedures on the heart. Right pleural cavity contained 700 ml. blood. Liver and spleen showed chronic congestion. Cause of death undetermined but consistent with leakage of blood from sutured incision in heart wall.

COMMENT:

This death resulted from uncontrollable haemorrhage due to a surgical mishap. Anaesthesia was in no way contributory. As death occurred while the patient was anaesthetised, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
151.1.59	3	No comment.	ORD	Uncontrollable haemorrhage. Cardiac arrest.	Yes

Name: Eunice Karamacher Age: 49 Sex: F Race: E

Disease: Mitral stenosis and incompetence.

Operation: Mitral valvotomy and annuloplasty on cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient had been in chronic cardiac failure for many years and this was no longer adequately controlled by digitalis. Rheumatic heart disease had caused mitral stenosis and incompetence. She had basal crepitations, dependent oedema and a 3 finger hepatomegaly. B.P. 110/70 mm.Hg. She was on treatment with digitalis and diuretics.

PREMEDICATION:

Sodium seconal gr. 3, 2 hours before operation and atropine gr. 1/100 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with a thiopentone, succinylcholine, IPPR oxygenation, topical analgesia of larynx and oral intubation sequence. IPPR with nitrous oxide and oxygen was then continued until spontaneous respiration returned after 5 minutes. From the commencement of operation, anaesthesia was maintained with nitrous oxide and oxygen administered by an IPPR technique via a circle absorption system with dTc being used as the relaxant. dTc was given in small incremental doses of 3 mg, a total of 12 mg. being used in the time up to the establishment of bypass. The course of the anaesthesia was relatively untoward until then. During bypass anaesthesia was maintained with small 5 mg. doses of thiopentone administered into the venous well with 3 mg. doses of dTc following an initial dose of 9 mg. at the commencement of bypass.

During mitral valvotomy and annuloplasty, a tear was made in the atrioventricular ring. This proved impossible to repair adequately and resulted in gross uncontrollable haemorrhage, leading to the patient's death.

AUTOPSY:

Chronic rheumatic heart disease. Evidence of surgical procedures on the heart. Death consistent with bleeding from the heart due to tearing of the ventricle and left auricle following instrumental dilatation of the mitral valve. Pleural cavities: right contained 500 ml. blood and left, 200 ml. blood. Both lungs partially collapsed.

COMMENT:

This death resulted from uncontrollable haemorrhage due to a surgical mishap. Anaesthesia appears to have been in no way contributory. As death occurred during anaesthesia, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY.
152.1.59	2	No comment	< 24	Cerebral oligoden-droglioma.	Yes

Name: Tsola Ghangusie Age: 30 Sex: M Race: B

Disease: Cerebral tumour Operation: Craniotomy. Partial excision of oligodendroglioma.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair. There were signs of a cerebral space-occupying lesion. B.P. 160/100 mm.Hg. He was completely blind from secondary optic atrophy due to the cerebral tumour and he had bilateral nerve palsies. Pre-operatively ventricular drainage had been established.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succincholine 50 mg., oxygenation, oral intubation. Thereafter anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether, administered via a non-rebreathing circuit, the patient breathing spontaneously. From the anaesthetic point of view the course of operation was uneventful. However, at operation, a large tumour was found extending into the third ventricle and basal ganglia. Much tumour was sucked away but only partial removal could be effected. The operation lasted 4 hours. At the end of operation the patient failed to regain consciousness and exhibited midbrain type convulsions. His condition deteriorated and he died 17 hours post-operatively.

AUTOPSY:

In addition to the evidence of surgery there was medullary and cuncall herniation. The tumour was an oligodendroglioma.

COMMENT:

This patient died from the results of attempted removal of a large oligodendroglioma, resulting in midbrain damage and ultimately in medullary and uncal herniation. Anaesthesia was not contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
153.1.59	2	No comment	< 24	Cerebral laceration Subdural haematoma.	Yes

Name: Elizabeth Moodie Age: 67 Sex: F Race: E

Disease: Head injury, fractured skull, subdural haematoma.

Operation: Carotid angiography. Left parietal craniotomy Evacuation of subdural haematoma.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had suffered a head injury resulting in a ffactured skull and cerebral damage. She was comatose and in extremely poor general state. Pulse rate 160/minute, B.P. 80 mm.Hg systolic. Her condition appeared to be deteriorating rapidly.

PREMEDICATION:

Atropine gr. 1/100, administered 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a Magill circuit with the patient breathing spontaneously. Topical analgesia of the larynx was followed by oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen (50%). After induction of anaesthesia the patient's condition did not change. B.P. remained 80 mm.Hg systolic and the pulse rate 160/minute. This showed no response to the administration of the vasopressor methylamphetamine.

Blood transfusion was commenced and 1 pint was transfused, though blood loss at operation was minimal. At operation a very small subdural haematoma was found and evacuated. At the conclusion of operation and on discontinuance of anaesthesia, the patient's level of consciousness appeared the same as pre-operatively. A drip infusion of noradrenaline 4 microgm./cc. elevated the B.P. to 100 mm.Hg systolic post-operatively and it remained at this level until the patient died, 17 hours post-operatively. During this time there was no change in the comatosed state of the patient.

AUTOPSY:

Fractured base of skull damaging pituitary gland. Bilateral subdural and subarachnoid haemorrhages with numerous petechae in brain and pons. Arteriosclerosis of cerebral arteries and aorta. Partial collapse of the left lung.

COMMENT:

This death appears to have been the result of traumatic cerebral damage. Anaesthesia is considered non-contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
154.1.59	2	No comment	< 24	Toxaemia, dehydration from intestinal obstruction.	Yes

Name: Ruth Brock Age: 70 Sex: F Race: E

Disease: Intestinal obstruction. Operation: Laparotomy. Freeing of band causing ileal obstruction.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. She showed signs of peripheral circulatory failure from dehydration. B.P. 70 mm.Hg systolic, pulse rate 100/minute. She had marked hyperpnoea. Gastric suction was instituted and intravenous fluid therapy commenced pre-operatively.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively

ANAESTHETIC:

On arrival in theatre, the B.P. was still 70 mm.Hg systolic and had not responded to rehydration therapy. Before the induction of anaesthesia, 6 mg. methylamphetamine was administered intravenously and this raised the B.P. to 80mm.Hg systolic. Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a Magill semi-open system, with spontaneous breathing. When anaesthesia had been induced, oral intubation was performed and anaesthesia was then continued with nitrous oxide and oxygen via a Magill semi-open system with spontaneous breathing. A further dose of 6 mg. methylamphetamine was given following the induction of anaesthesia, but the effect (as after the previous dose) lasted only 10 minutes. Thereafter the B.P. commenced falling again. A drip infusion of noradrenaline 4 microgm./cc. was commenced and the B.P. was maintained at a level of 100 mm.Hg systolic. The peripheral circulation improved and anaesthesia was continued without event.

At operation, ileal obstruction was found to be caused by a peritoneal band. This was freed. Succinylcholine 25 mg. was given to produce relaxation for peritoneal closure and an IPPR technique instituted for the period of paralysis. At the end of the operation, which lasted 35 minutes, and on discontinuance of the anaesthetic, the patient regained consciousness rapidly and was talking when leaving theatre. Post-operatively it was found necessary to continue the noradrenaline infusion. The patient died 5½ hours post-operatively.

AUTOPSY:

Bilateral bronchopneumonia. Healed gastric ulcer. Duodenal stenosis. Rheumatoid arthritis. Marked autolysis of and haemoglobin staining of internal organs. ?Septicaemia.

COMMENT:

This patient's poor condition does not appear to have been worsened by the administration of anaesthesia. At the conclusion of the operation, she recovered consciousness adequately. The patient appears to have died as a result of the surgical lesion, toxic absorption and possible septicaemia and dehydration. Anaesthesia does not appear to have been contributory to the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
155.1.59	2	No comment	< 24	Undetermined.	No

Name: Ethel Mayne Age: 83 Sex: F Race: E

Disease: Pathological fracture of femur from secondary carcinoma.

Operation: Insertion of a Kunchner nail.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor: the patient had secondary carcinomatous deposits in her bones and in both lungs. It was a secondary carcinoma deposit in the femur which caused a pathological fracture for which insertion of a Kunchner nail was now deemed necessary. B.P. was 150/80 mm.Hg, pulse rate 90/minute, respiration 24/minute.

PREMEDICATION:

Atropine gr. 1/100 and pethidine 50 mg. administered 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a Magill semi-open system the patient breathing spontaneously. Anaesthesia was maintained similarly.

The operation lasted 60 minutes, during which time the patient's condition remained stable, the B.P. 150 mm.Hg systolic and the pulse rate between 90 and 100/minute. 1 pint blood was transfused during the procedure. The patient was awake 15 minutes after the conslusion of the operation and discontinuance of anaesthesia. She died suddenly 5 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

Whatever the cause of this patient's death, it does not appear to be related to the anaesthetic or its management.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
156.1.59	2	No comment	< 24	Myocardial ischaemia	No

Name: C. Kramer Age: 75 Sex: M Race: E

Disease; Ruptured aortic aneurysm. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient presented with a ruptured aortic aneurysm. He was severely collapsed although he had been resuscitated to some extent by blood transfusion. Immediately before surgery the B.P. was 100 mm.Hg systolic with a pulse rate of 140/minute, respiration 30/minute. There was severe peripheral vasoconstriction. The urinary output was satisfactory.

PREMEDICATION:

Pethidine 50 mg., atropine gr. 1/100 administered 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by succincholine 25 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether, administered via a circle absorption system, using an IPPR technique. Gallamine 60 mg. was administered as a relaxant.

At laparotomy a ruptured aortic aneurysm was found to involve the renal vessels. It was decided to proceed no further and the abdomen was closed. To provide relaxation for closure of the abdomen, cyclopropane was administered for a short period at this stage of the procedure. At the end of the operation the patient regained consciousness rapidly. He died 22 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

The patient died of myocardial ischaemia. Anaesthesia was in no way contributory tothis death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
157.1.59	1	No verdict.	< 24	Post-relaxant respiratory abnormality Peritonitis	Yes

Name: Ernest Engelke. Age: 61 Sex: M Race: E

Disease: Post-gastrectomy dehiscence of abdominal wound. Peritonitis.

Operation: Resuture of dehisced abdominal wound.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient had had a partial gastrectomy for peptic ulcer 8 days previously. The post-operative course had been stormy and now, on the 8th day, he dehisced the abdominal wound. Condition was poor. He had a tachycardia of 160/minute and B.P. 150/90 mm.Hg (his normal B.P. was 200 mm.Hg systolic), respiration 30/minute and of a grunting character. There was poor urinary output and a raised blood urea. He was on continuous gastric aspiration and intravenous fluid replacement therapy which had been instituted for intestinal ileus before the wound dehisced.

PREMEDICATION:

Morphine gr. 1/4, atropine gr. 1/100, administered 45 minutes before the operation.

ANAESTHETIC:

On arrival in theatre the B.P. was 150 mm.Hg systolic, pulse rate 160/minute. Anaesthesia was induced with thiopentone 200 mg. followed by succinylcholine 50 mg., IPPR with oxygen and oral intubation. IPPR with nitrous oxide, oxygen and a trace of ether was continued until spontaneous respiration returned. Anaesthesia was then maintained with nitrous oxide and oxygen with minimal ether, administered via a circle absorption system. Gallamine 40 mg. was given and IPPR continued.

At laparotomy, diffuse peritonitis with a pelvic abscess was found. Following induction of anaesthesia the B.P. dropped from 150 mm.Hg systolic to 110, and subsequently rose again to 120 mm.Hg systolic. Throughout this time the pulse rate remained around 160/minute. During operation 50 ml. dextrose/saline was infused. After laparotomy the wound was resutured. The operation lasted 36 minutes. At the end of operation and on discontinuance of the anaesthetic, the patient failed to regain consciousness. Spontaneous respiration returned but was of a gaspingnature with a pronounced tracheal tug. Neostigmine 2 mg. in divided doses preceded by atropine 1.2 mg. failed to improve the respiration or alter the state of consciousness. At no time during the operation did the patient appear anoxic or cyanosed.

25 minutes after the conclusion of operation the patient had still failed to regain consciousness, though he did react to painful stimuli. Respiration was still of a gasping nature with a tracheal tug, but was of apparently adequate volume. The administration of 5 mg. nikethamide made no difference to the patient's state of consciousness or respiration. The patient was returned to the ward 25 minutes post-operatively. To exclude the possibility of metabolic acidosis 10 ml. 4% sodium bicarbonate solution was administered intravenously without effect. For the next 6 hours post-operatively his condition remained in status quo. He failed to regain consciousness but reacted to painful stimuli. The respiration was of a gasping character with a tracheal tug, but apparently of adequate volume. He retained a good colour and the B.P. remained at approximately 130 mm.Hg systolic. However, 6 hours after conclusion of the operation the B.P. commenced falling. An intravenous noradrenaline drip infusion

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was commenced. Initially this caused a response and the B.P. rose to its former level, but this state lasted but 1 hour whereupon the infusion had no effect and the patient died.

AUTOPSY:

Partial collapse of left lung with 16 ozs. pleural effusion, oedema and congestion of both lungs. Numerous fibrous adhesions below left lung and a few at the right lung apex. Diffuse healed pericarditis. Enlarged liver and enlarged heart. Generalised peritonitis. Bilateral hydronephrosis with lobulated kidney.

COMMENT:

In outline, the problem here is that of a patient who albeit in poor condition with peritonitis, pleural effusion and a renal lesion, is conscious and breathing adequately though with a "grunt", and who fails to regain both consciousness and a normal pattern of respiration following a short general anaesthetic involving the use of a muscle relaxant for resuture of a dehisced abdominal wound. There is no single positive episode in the conduct of the anaesthetic or the surgical management that would result in such an outcome. One is forced to approach the diagnosis by elimination.

The patient's condition at the conclusion of operation may have resulted from:

(1) Cerebral damage resulting from an episode of anoxia during anaesthesia. The anaesthetist was sure that there was no dramatic episode of anoxia during the anaesthesia ("the patient maintained a good colour throughout") and the patient was at no time cyanosed. However, the autopsy revealed a partially collapsed left lung with a pleural effusion. This will have resulted in a ventilation perfusion disturbance with venous shunting which would have produced some degree of anoxia. In the absence of cyanosis, the degree of anoxia produced is unlikely to have caused cerebral damage of great moment. However, one cannot exclude anoxia as a possible factor in this case.

(2) Cerebral damage resulting from cerebral ischaemia. Although the B.P. did drop following induction of anaesthesia, the level was maintained for the most part at 120 mm.Hg systolic - which should be adequate for cerebral perfusion. Though we do know that this patient's normal B.P. was of the order of 200 mm.Hg systolic, this probably points to the existence of some degree of vascular disease.

(3) Overdosage with cerebral depressant drug. The dose of morphine given to this patient as premedicant (gr. 1/4) was certainly larger than was wise or necessary. Furthermore, in view of his poor preoperative state, one might also criticise the use of a dose of 200 mg. thiopentone for induction of anaesthesia. However, the type of respiration that returned spontaneously post-operatively was not the pattern that pertains to pharmacological depression of the respiratory centre, but rather that of some form of cerebral damage or neuromuscular block. The administration of nekethamide made no difference to the respiration or to the state of consciousness, as one would anticipate if drug depression of the central nervous system was the cause of the patient's post-operative state.

(4) (a) A mixed neuromuscular block, or (b) so-called neostigmine resistant curarisation. The former is unlikely in that spontaneous respiration was restored after administration of succinylcholine for intubation, before the administration of gallamine. With regard to the latter, though the dose of gallamine was modest indeed, its administration must be considered in relation to the renal lesion, the poor urinary output and the probable metabolic acidosis.

(5) Cerebral vascular accident. This is probably excluded by (a) the lack of localising signs and (b) the failure to demonstrate such lesion at autopsy - though again, neither of these reasons is conclusive.

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(6) Metabolic acidosis. This is a strong possibility. The amount of sodium bicarbonate given was homeopathic. At this time the facilities for the rapid biochemical diagnosis of acidosis were not available.

(7) Toxaemia and possibly septicaemia from diffuse peritonitis. The post-operative condition of the patient could well have been an extension of his steadily worsening pre-operative condition, as a result of generalised peritonitis and the resulting toxaemia. It is difficult to form a firm conclusion in this regard.

Because of (1) the possibility of anoxia, which cannot be excluded, (2) the dose of morphine administered for premedication which was excessive, and (3) the possibility of so-called neostigmine resistant curarisation, (4) the treatment of metabolic acidosis (if it played a part in this patient's condition) was inadequate, and (5) the patient failed to regain consciousness - anaesthesia is regarded as definitely contributory to this patient's death, although generalised peritonitis and toxaemia are also major factors.

PREVENTABILITY:

In view of the uncertainty as to the precise cause of this patient's death, no verdict can be offered as to preventability. One must criticise (1) the excessive dose of morphine given as premedication, (2) the completely inadequate treatment of the probable metabolic acidosis, and (3) in view of the above described abnormal pattern of respiration, some form of oxygen therapy would have been appropriate.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
158.1.59	2	No comment	< 24	Cerebral tumour. Glioblastoma multiforme.	No

Name: Lucy Brand. Age: 42 Sex: F Race: E

Disease: Cerebral tumour. Operation: Carotid angiography. Burrhole craniotomy and brain biopsy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Deeply comatose and somewhat dehydrated patient. Right hemiparesis and bilateral plantar extensor responses. B.P. 110 mm.Hg systolic pulse rate 120/minute. There was albuminuria and pus cells in the urine. The fluid balance had been maintained pre-operatively by fluid administration by way of a Ryle's tube.

PREMEDICATION:

Atropine gr. 1/75 administered 45 minutes pre-operatively.

ANAESTHETIC:

A left carotid angiogram was performed under local anaesthetic, and showed an indication for craniotomy. An endotracheal tube was inserted under topical analgesia of the larynx. The burrhole craniotomy was commenced on oxygen alone, the patient breathing spontaneously, but subsequently (when she moved following the stimulus of pain) 50% nitrous oxide was added to the inhaled mixture.

At operation, a cerebral tumour was found which was considered to be inoperable. Biopsy of the tumour revealed this to be a glioblastoma multiforme. After brain biopsy the operation was concluded. In all, it took 30 minutes. At the conclusion of operation and discontinuance of the anaesthetic, the patient's level of consciousness appeared, if anything, a little lighter than pre-operatively. She died 7½ hours after operation without regaining consciousness.

AUTOPSY:

No autopsy.

COMMENT:

The patient died of the results of the pre-existing disease - an intracerebral tumour: glioblastoma multiforme. Anaesthesia was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
159.1.59	3	No comment	< 24	Post-relaxant apnoea. Circulatory failure. Acute haemorrhagic pancreatitis	Yes

Name: Sophia Krynauw Age: 45 Sex: F Race: E

Disease: Acute haemorrhagic pancreatitis. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

There was a diffuse peritonitis from a suspected rupture of the gall bladder; the patient had a previous history of cholecystitis and cholelithiasis. Her condition was very poor; she was grossly vaso-constricted. B.P. 110 mm.Hg systolic, pulse rate 120/minute, respiration 48/minute with a gasping type of respiration marked by a tracheal tug. On admission she was dehydrated. Pre-operatively gastric aspiration had been performed and rehydration therapy commenced. She was considered an extremely poor anaesthetic risk.

PREMEDICATION:

Pethidine 100 mg., atropine gr. 1/100 given 3 hours before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg. followed by nitrous oxide and oxygen with gradually added ether, administered via a circle absorption system with spontaneous breathing. When anaesthesis of sufficient depth had been induced, the patient was orally intubated. Anaesthesia was then maintained with nitrous oxide and oxygen with minimal ether. After induction of anaesthesia the B.P. dropped to 100 mm.Hg systolic, pulse rate remaining approximately 120/minute throughout the operation.

On commencement of the laparotomy, 23 minutes after the induction of anaesthesia, gallamine 60 mg. was administered and an IPPR technique instituted. Laparotomy revealed a severe acute haemorrhagic pancreatitis. Abdominal drains were inserted and the abdomen was closed. The operation took 80 minutes. Throughout the procedure the patient's condition remained relatively static, B.P varying between 80 and 100 mm.Hg systolic with a pulse rate of 120/minute. However, at the conclusion of the procedure, normal respiration did not return nor did the patient regain consciousness. Neostigmine 1.5 mg preceded by atropine 100 mg. produced no response. Artificial ventilation with pure oxygen through a circle absorber was continued. 25 minutes after the conclusion of the operation, nikethamide 5 cc. was administered intravenously without response. Artificial ventilation was continued with oxygen. After another 25 minutes deptazole 30 mg. was administered intravenously with a further 30 mg. 5 minutes later. Neither injection produced any response. After a further 10 minutes artificial ventilation with oxygen, nalorphine 3 mg. was given by intravenous injection. This too produced no response. Artificial ventilation was continued. After another 5 minutes 1 gm. calcium gluconate (10 cc. of 10% solution) was given intravenously. Immediately after this the B.P. fell progressively until it reached 40 mm.Hg systolic with a pronounced bradycardia. Methylamphetamine 6 mg. was then injected intravenously without effect. A further 15 minutes later atropine gr. 1/100 was administered intravenously and the B.P. rose to 80 mm.Hg systolic, the pulse rate being noted to rise too. 20 minutes later hydrocortisone 100 mg. was administered. Throughout this time artificial ventilation with oxygen was continued. After another

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40 minutes 50 cc. of 50% sucrose was administered intravenously without effect. Three hours after the conclusion of operation the patient was transferred from the theatre to a Drinker respirator. The endotracheal tube was left in situ. She died 2½ hours later.

AUTOPSY:

Acute pulmonary oedema and congestion of the right lung with congestion and oedema and pleural adhesions of left lung. Early atherosclerosis of the aorta and coronary arteries. Acute haemorrhagic pancreatitis with necrosis and gangrene of approximately 90% of the pancreas. Extensive digestion and fat necrosis of omentum, mesentary and peritoneal cavity. 500 ml. haemorrhagic fluid in abdominal cavity. The liver was enlarged, weighing 2,200 gm. and showed extensive fatty changes. Chronic cholecystitis and gall stones.

COMMENT:

The patient's condition before anaesthesia could be described as in extremis. There is little if any fault apparent in the conduct of the anaesthetic. As such, this death may be regarded as inevitable; although one is faced with the fact that apnoea followed anaesthesia involving the use of a relaxant drug, it must be noted that respiration was gasping and manifested a tracheal tug before the operation and the administration of anaesthesia.

One aspect open to criticism is the size of the dose of pethidine given to control the patient's severe pain 3 hours before the induction of anaesthesia. In view of her poor condition, this dose was unnecessarily large.

Post-anaesthetic, there was no response at all to the narcotic antagonist drugs, or respiratory stimulants. While the patient was apnoeic, efficient mechanical pulmonary ventilation was maintained. A major aetiological factor in the patient's lack of response to resuscitative measures and her death was probably severe metabolic acidosis. However, at this time, facilities for the rapid diagnosis and treatment of this condition were not available, nor was its importance as clearly realised as it is today. In its main essentials, this case is similar to Case No. 138.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
160.1.60	2	No comment	< 24	Undetermined.	No

Name: Alice Nkanlu. Age: 44 Sex: F Race: B

Disease: Aneurysm of the descending thoracic aorta.

Operation: Resection and graft of thoracic aortic aneurysm, under hypothermia.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Fair. The aneurysm of the descending thoracic aorta all but completely obstructed the left main bronchus. B.P. 125/50 mm.Hg.

PREMEDICATION:

Seconal gr. 3 orally 3 hours pre-operatively, atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 275 mg. followed by succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued with nitrous oxide oxygen and ether, administered via a circle absorption system, the patient breathing spontaneously, while preparations were made for the operation. dTc was administered and an IPPR technique was instituted. Cooling was begun by means of a cooling blanket. Before operation the patient's temperature was reduced to 28°C and during this, administration of ether was stopped and anaesthesia continued with nitrous oxide and oxygen, via a circle absorption system by an IPPR technique with intermittent doses of dTc as a relaxant. Throughout operation, continuous EEG and ECG monitoring was maintained.

During resection of the aneurysm and grafting of the aorta, the aorta being clamped off, the patient was heparinised and an aorta-femoral bypass established. At the conclusion of the operation, which took 7 hours, and on discontinuance of the anaesthetic, the patient rapidly regained consciousness and normal respiration was established. Neostigmine 1 mg. was administered preceded by atropine gr. 1/100, to ensure reversal of curarisation. At the conclusion of operation the patient's temperature was 30°C and slow rewarming was instituted.

Throughout the procedure, blood had been replaced as lost, and the replacement was considered adequate. On return to the ward, she was placed in an oxygen tent. Subsequently the B.P. fell to 70 mm. Hg systolic, and a noradrenaline drip infusion was instituted which maintained the B.P. at 120 mm.Hg systolic. However, the patient died 13 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

The course of anaesthesia and recovery therefrom was uneventful. Anaesthesia is not considered to have played any part contributing to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
161.1.60	3	No comment	ORD	Cardiac arrest. Ventricular fibrillation.	Yes

Name: Melville Warren. Age: 48 Sex: M Race: E

Disease: Calcific aortic stenosis. Operation: Aortic valvotomy on cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient had severe calcific aortic stenosis with a low cardiac output. B.P. 90/70 mm.Hg in the arms, left ventricular pressure was 170/5 mm.Hg and there was severe left ventricular hypertrophy. He had pulmonary hypertension.

PREMEDICATION:

Seconal gr. 3 orally, 3 hours before operation. Atropine gr. 1/100 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by a sequence of succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx, oral intubation, IPPR with nitrous oxide and oxygen with a trace of ether, going on to spontaneous respiration on a circle absorption system. At the commencement of operation, ether was discontinued, dTc administered and the IPPR technique, via a circle absorption system, instituted. The total dose of dTc throughout operation was 30 mg.

In the pre-bypass stage the anaesthesia was uneventful. During bypass, during which elective cardiac arrest was employed, anaesthesia was maintained by the administration of intermittent small doses of dTc together with thiopentone (respectively, totalling 24 mg. and 90 mg.). On conclusion of cardiopulmonary bypass, a normal heart beat wasestablished but a low cardiac output state resulted. The B.P. was extremely low and an infusion of adrenaline 1:100,000 was commenced which maintained the B.P. at 100 mm.Hg systolic. However, shortly after discontinuance of bypass, ventricular fibrillation ensued and the complete flattening of the EEG record indicated the onset of cerebral anoxia. Despite intensive measures, electrical defibrillation, calcium chloride, sodium bicarbonate, the ventricular fibrillation proved refractory to treatment.

AUTOPSY:

Aortic incompetence with left ventricular enlargement. Both lungs partially collapsed with small bilateral haemothorax. Sutured surgical wound of aorta and right auricle. Heart enlarged, weighing 930 gm. Gross ventricular enlargement due to aortic incompetence, with distortion and calcification of valves. An artificial valve cusp had been sutured in position. Stomach empty. Spleen enlarged.

COMMENT:

This death in all probability resulted from the effects of the surgical procedure, on an already badly damaged heart, together with myocardial anoxia during the period of aortic valvotomy and elective cardiac arrest. Anaesthesia is not considered contributory to this death. As death occurred during anaesthesia, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
162.1.60	2	No comment	< 24	Intra-cranial tumour.	Yes

Name: Jan Meyer Age: 19 Sex: M Race: C

Disease: Intracerebral tumour, posterior fossa. Operation: Posterior fossa craniotomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Signs of an intracranial space-occupying lesion, but the patient was otherwise normal. Drowsy, a markedly raised intracranial pressure. B.P. 110 mm.Hg systolic, respiration 20/minute. Ventricular drainage had been established pre-operatively and a ventriculogram and carotid angiogram had been performed.

PREMEDICATION:

Atropine gr.1/100 given intravenously 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by succinylcholine 40 mg., IPPR with oxygen, topical analgesia of the larynx, and oral intubation. Anaesthesia was then continued with ether and air, administered from an E.M.O. ether vapourizer, with spontaneous breathing. Towards the end of the operation, 30% oxygen and 70% nitrous oxide were passed through the E.M.O. as a vehicle for the vapour. The operation lasted 3½ hours and the course of anaesthesia was uneventful.

At the conclusion of the operation and on discontinuance of the anaesthetic, the patient rapidly regained consciousness. 9 hours after surgery his respiration suddenly dropped to 8/minute, his level of consciousness deteriorated and the B.P. rose from 150 mm.Hg to 200 mm.Hg systolic. Within 30 minutes the patient was dead.

AUTOPSY:

Meningioma (histologically proven) occupying the right cerebellar fossa and attached to cerebellum. Dilatation of both lateral veins. Haemorrhage over base of brain. Some congestion and oedema of both lungs.

COMMENT:

This death appears to have been due to post-operative intracranial haemorrhage occurring 9 hours after the operation. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
163.1.60	2	No comment	< 24	Myocardial infarction	No

Name: George Edwards Age: 66 Sex: M Race: E

Disease: Intestinal obstruction, volvulus of small bowel, bowel gangrenous.

Operation: Laparotomy. Small bowel resection.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. The patient was collapsed and sweating profusely with moist, cold extremities. B.P. 80 mm.Hg systolic with a pulse rate of 140/minute. Gastric aspiration was instituted and rehydration therapy commenced.

PREMEDICATION:

Pethidine 50 mg., atropine gr. 1/100, given 1 hour before operation.

ANAESTHETIC:

On arrival in theatre, the B.P. was 80 mm.Hg. Anaesthesia was induced with thiopentone 300 mg. followed by nitrous oxide, oxygen and ether administered via a non-rebreathing system, using a Reuben valve, the patient breathing spontaneously. When light anaesthesia had been induced, the larynx was topically anaesthetised and oral intubation performed. Gallamine in divided doses of 20 mg. each, totalling 100 mg. throughout operation, was given and an IPPR technique instituted with the same system as before. During the first hour of surgery repeated doses of methylamphetamine were given to maintain the B.P. at a satisfactory level. For the last hour of operation, which lasted 2 hours in all, the B.P. was stabilised at 120 mm.Hg systolic. A total of 48 mg. methylamphetamine was given in the first hour.

At operation, a volvulus of the small bowel withgangrenous loop of jejunum was found and was resected, jejuno-jejunal anastomosis being performed. At the conclusion of operation and discontinuance of anaesthesia, normal respiration returned and was considered adquate in volume. No antidote for the gallamine was considered necessary. The patient rapidly regained consciousness 5 minutes after discontinuance of the anaesthetic. The initial post-operative phase was satisfactory but 10 hours post-operatively severe circulatory collapse followed the patient's complaint of pain in the chest. An ECG revealed myocardial infarction. The patient died 12 hours after the operation.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of a proven post-operative myocardial infarction. The part played by the hypotension in the pre- and early operative phases inthe aetiology of this coronary thrombosis must of course be considered. Consequently, the administration of a dose of thiopentone as large as 300 mg. must be considered in relation to this hypotension. However, the patient was hypotensive before anaesthesia and this did not worsen following the administration of thiopentone. Further, the clinical onset of the myocardial infarction followed the operation at a stage when he had recovered from the anaesthetic. Anaesthesia is not considered contributory to this patient's death.

CASE NO.	CLASSIFICATION GROUP	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
164.1.60	1	Possibly	ORD	Hypovolaemia. Hypotension. Cardiac arrest.	Yes

Name: Miriam Matthews Age: 32 Sex: F Race: C

Disease: Malignant hypertension, renal artery stenosis. Operation: Renal artery and aortic endarterectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient suffered from malignant hypertension secondary to stenosis of the right renal artery. On admission she was in mild cardiac failure. B.P. 220/180 mm.Hg. There was marked left ventricular hypertrophy. She had been treated with the hypotensive drug Dorenthin and with chlorthiazide. Cardiac failure responded to bed rest. Dorenthin was discontinued before operation, causing the B.P. to rise to 260/180 mm.Hg immediately before the operation.

PREMEDICATION:

Pethidine 100 mg., phenergan 50 mg., atropine gr. 1/100, given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by the sequence succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued with nitrous oxide and oxygen administered via a circle absorption system by an IPPR technique. dTc was given in divided doses, an initial dose of 25 mg. followed by one later of 5 mg. and another of 7.5 mg., totalling 37.5 mg. throughout operation. The initial course of anaesthesia was untoward.

The operation lasted 4 hours 25 minutes and consisted of extensive dissection of the aorta, from the bifurcation to the diaphragm, an operative aortogram, pressure recordings in the aorta and renal arteries, and finally an aortic and renal endarterectomy. During the period of dissection, the B.P. fell below 90 mm.Hg systolic on three occasions, but recovered rapidly once pressure on the inferior vena cava was relieved, and presumably was of mechanical origin. Finally, when the endarterectomies were performed, the aorta was clamped above the renal arteries for 20 minutes. Up to this time, 2 pints blood had been transfused. After release of the aortic clamp the B.P. fell markedly. 30 mg. methylamphetamine was given without response and within 5 minutes cardiac arrest occurred. Cardiac massage was commenced immediately, the heart being found to have arrested in asystole, and the heart filled poorly. After 2 minutes of massage, ventricular fibrillation commenced. Repeated attempts at electrical defibrillation after intraventricular injection of adrenaline were unsuccessful. After cardiac arrest a further pint blood was rapidly transfused.

AUTOPSY:

Cause of death undetermined. Evidence of heart and renal disease. Pulmonary tuberculosis. Surgical procedures of heart and aorta. Enlarged heart, collapsed left lung. Assmann tuberculous focus in apical region of left lung. Abnormal kidneys. Blocked right renal artery. Atherosclerosis of the aorta. Coronary arteries normal. Marked atheroma of abdominal aorta with plaque obstructing right renal artery. Incised wound of aorta below right renal artery sutured. Liver showed fine cirrhosis. Hypertensive kidney.

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COMMENT:

From the clinical description, it appears that cardiac arrest resulted from myocardial ischaemia which in turn resulted from the hypotension that followed release of the aortic clamp. The administration of methylamphetamine had no effect in elevating the B.P. after release of the aortic clamp. One must wonder whether the rapid administration of blood by the anaesthetist, as the clamp was removed, together with the administration of a vasopressor drug, might not have averted this calamity. Overall, only 2 pints blood had been transfused up to the time that the aortic clamp was released; at this time the operation had been in progress for $3\frac{1}{2}$ hours. One wonders if, for an operation of this nature, such blood replacement was indeed adequate. The calamitous drop in blood pressure that followed release of the clamp is just what one would expect to occur if the patient was hypovolaemic.

On cardiac massage, the observation that ventricular filling was poor is probably another indication of hypovolaemia secondary to inadequate blood replacement.

Because blood replacement is considered inadequate, the anaesthetic management in this case is considered a significant contributory factor to the patient's death.

PREVENTABILITY:

As the adequacy of blood replacement is doubted, this death is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
165.1.60	3	No comment	ORD	Uncontrollable haemorrhage. Cardiac arrest.	Yes

Name: Maureen Bedwell Age: 15 Sex: F Race: C

Disease: Patent ductus arteriosus. Operation: Ligation of patent ductus arteriosus.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Satisfactory other than for the surgical lesion, which had resulted in some cardiac enlargement and pulmonary plethora. B.P. 140/10 mm.Hg Pulse rate 160/minute.

PREMEDICATION:

Omnopon gr. 1/6, scopolomine gr. 1/200 administered 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by inhalation of nitrous oxide and oxygen with gradually added ether, via a circle absorption system, with spontaneous breathing. After the induction of light anaesthesia, topical analgesia of the larynx and oral intubation were performed. Anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system using an IPPR technique. dTc was given in divided doses, totalling 18 mg. throughout the operation. Anaesthesia was relatively untoward until, during suture of the ductus, the pulmonary artery was torn and a severe uncontrollable haemorrhage resulted. Massive blood transfusion was resorted to but cardiac arrest ensued. Cardiac massage and electrical defibrillation started the heart on three occasions, but it re-arrested in ventricular fibrillation. Ultimately, 3 routes were used to transfuse blood (2 intravenous and 1 into the aorta), a total of 14 pints blood being transfused in all. This was to no avail.

AUTOPSY:

520 ml. in left pleural cavity. Surgical wound of left thorax. Sutured wounds of inferior side of aortic arch and superior side of pulmonary artery.

COMMENT:

This death resulted from massive operative uncontrollable haemorrhage due to a surgical mishap. Anaesthesia is not considered contributory. As death occurred while the patient was anaesthetised, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
166.1.60	2	No comment	< 24	Haemor-rhage.	Yes

Name: Mrs. M. Kennet Age: 58 Sex: F Race: E

Disease: Intrahepatic abscess (diagnosed as a subphrenic abscess)

Operation: Laparotomy. Drainage of abscess. Thoracotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor. Pulse rate 100/minute, B.P. 105/70 mm.Hg. She appeared toxic. There was dullness of the right posterior lung base.

PREMEDICATION:

Morphine gr. 1/8, atropine 1/100 gr., given 1 hour before surgery.

ANAESTHETIC:

anaesthesia was induced with thiopentone 200 mg., succinylcholine 25 mg., followed by IPPR with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued with nitrous oxide, oxygen and ether administered via a non-rebreathing technique with IPPR. No further relaxant was used other than the initial succinyl-choline. Induction and maintenance of anaesthesia were initially untoward.

When laparotomy was performed and the surgeon approached the abscess, gross haemorrhage occurred from the liver. This soon became massive and could not be stopped, despite all measures. Massive transfusion of blood was resorted to but the B.P. fell markedly, rising occasion-ally when blood replacement caught up with blood loss, and subsequently falling again when the loss exceeded the amount replaced. Control of the haemorrhage was impossible from the abdominal approach used, and thoracotomy was performed. No great success was achieved in stopping the bleeding by this route either. A total of 21 pints blood was transfused during the operation, which lasted 4 hours. At one stage during surgery the B.P. and pulse rate became unrecordable but recovered after further blood transfusion. At the conclusion of the operation and on discontinuance of the anaesthetic, the patient rapidly regained consciousness. However, bleeding continued and some 8 hours later the patient was extremely distressed due to a right haemothorax. An underwater drain was inserted under local anaesthesia in the ward. Haemorrhage continued and ultimately further transfusion was to no avail. The patient died 23 hours post-operatively.

AUTOPSY:

Large intrahepatic abscess. Large parahepatic cyst of unknown nature. Pulmonary oedema. Laryngeal oedema. Fibroids, hydrosalpinx and right ovarian cyst. Right lower lobe atelectasis.

COMMENT:

This patient obviously died from gross uncontrollable haemorrhage from an abscess cavity in the liver, and associated pathology. Anaesthesia is not considered contributory to the death. The laryngeal oedema found at autopsy was not clinically apparent before death; this may have resulted from intubation during operation.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
167.1.60	3	No comment	ORD	Ventricular fibrillation. Cardiac arrest.	Yes

Name: C.R. Weitz. Age: 30 Sex: M Race: E

Disease: Rupture of sinus of Valsalva into the right ventricle.

Operation: Repair of ruptured sinus of Valsalva on cardio-pulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

This patient was in chronic cardiac failure and was digitalised. B.P. 170/50 mm.Hg, pulse rate 100/minute. There was a 3 finger hepatomegaly.

PREMEDICATION:

Sodium seconal gr. 3, orally, 3 hours pre-operatively and atropine gr. 1/100 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone, succinylcholine, IPPR with oxygen, topical analgesia of the larynx, and oral intubation, followed by nitrous oxide, oxygen and a trace of ether administered via a carbon dioxide circle absorption system. After the return of spontaneous respiration, anaesthesia was maintained with nitrous oxide and oxygen administered via an IPPR technique via a circle absorption system with intermittent doses of dTc. Until the start of bypass, a total of 27 mg. dTc had been administered. Induction and maintenance of anaesthesia was uneventful until the start of bypass. EEG, ECG and arterial blood pressure monitoring via an internal mammary artery cannula were used throughout.

After commencement of bypass, hypothermia down to 32°C was induced. During bypass anaesthesia was maintained with small intermittent doses of thiopentone and dTc. Elective cardiac arrest by anoxia was induced. At the conclusion of surgery, ventricular fibrillation supervened when bypass was discontinued. This was refractive to treatment by electrical defibrillation, adrenaline, calcium chloride, sodium bicarbonate and glucose.

AUTOPSY:

Gross enlargement of the heart. Aortic incompetence repaired. Intraventricular septal defect. Evidence of surgical procedure to heart. Cause of death undetermined. Evidence of heart disease.

COMMENT:

Death followed the failure of the heart to defibrillate following elective anoxic arrest of the heart. Anaesthesia is not considered contributory. As death occurred during anaesthesia, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
168.1.60	3	No comment	< 24 c	Haemo-pericardium Irreversible shock. Cardiac arrest.	Yes

Name: George Makagoner Age: Unknown Sex: M Race: B

Disease: Ruptured spleen, traumatic. Operation: Laparotomy. Splenectomy. Thoracotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund; there was no palpable pulse and no recordable B.P. Respiration 20/minute. The abdomen was grossly distended. 3 pints blood were rapidly transfused pre-operatively and gastric lavage was performed.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Endotracheal intubation was performed without anaesthesia. Nitrous oxide, oxygen and a trace of ether was then administered via a Magill system, with spontaneous breathing. Seven minutes after induction of anaesthesia, cardiac arrest was diagnosed. Nitrous oxide and ether were discontinued and IPPR performed with oxygen alone via a circle absorption system. Thoracotomy was performed and cardiac massage instituted immediately. A large haemopericardium was found. After cardiac massage and the injection of 1 ml. adrenaline 1:10,000 into the left ventricle, the heart was restarted after 1 minute. The B.P. was recorded at 100 mm.Hg systolic. The source of the haemo-pericardium was not found. Thoracotomy was closed and a laparotomy performed. This revealed a ruptured spleen. During this time the B.P. gradually fell again to 65 mm.Hg. Throughout this period blood was transfused.

During operation, which took 4 hours, 9 pints blood were transfused. An infusion of noradrenaline was commenced, which effectively maintained the B.P. for a time, but then the B.P. continued to fall and the strength of the infusion had to be increased. Splenectomy was performed and the abdomen closed. At the end of the operation the B.P. was 75 mm.Hg systolic. The patient was returned to the ward without regaining consciousness and he died 3 hours after surgery.

AUTOPSY:

Evidence of surgical removal of spleen and 4th rib. Contused myo-cardium, abdominal wall, scalp and hip. Haematomata of intestine and mesentery. Haemoperitoneum and cirrhosis of the liver.

COMMENT:

The patient was moribund without a recordable B.P. or pulse before the induction of anaesthesia. His condition had been like this since admission, some hours before operation. To what extent this was due to blood loss from the ruptured spleen, or to the haemopericardium with cardiac tamponade, is difficult to say. That the intubation of the patient and subsequent anaesthesia was the final straw in the causation of the ultimate cardiac arrest is possible, though in a patient in this condition such a cardiac arrest was inevitable, as was his subsequent death. Anaesthesia is not considered avoidably contributory to the outcome. As cardiac arrest occurred with the induction of anaesthesia, the case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
169.1.60	1	Possibly	< 24	Post-relaxant respiratory inadequacy. ?Adrenal insufficiency. Circulatory failure.	Yes

Name: Louisa Els Age: 48 Sex: F Race: E

Disease: Diffuse secondary carcinomatosis from carcinoma of the breast.

Operation: Bilateral adrenalectomy. Bilateral loin approach.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This patient had disseminated secondaries from carcinoma of the left breast. A radical mastectomy had been performed previously and subsequently a bilateral oopherectomy, the latter giving some relief of the symptoms but these had recurred and it had now been decided that bilateral adrenalectomy might offer some further relief. She also had chronic bronchitis; she was somewhat obese, had a systolic cardiac murmur and complained of some shortness of breath. It was decided, after consultation with her physician, that no cardiac failure was present. In view of her physical status, however, and the magnitude of the operation, she was considered a poor operative risk. Adrenal corticoid replacement therapy was commenced before operation.

PREMEDICATION:

Hydrocortisone 100 mg., pethidine 50 mg., phenergan 25 mg. and atropine 0.6 mg. were given 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with a sleep dose of thiopentone 250 mg. and continued with inhalation of nitrous oxide, oxygen and gradually added ether, with spontaneous breathing. A carbon dioxide circle absorption system was used. After 8 minutes the patient's larynx and trachea were topically anaesthetised and after a further 2 minutes of inhalational anaesthesia, an endotracheal tube was introduced. The patient was positioned in the lateral position with the kidney rest elevated. At the beginning of operation, an initial dose of 60 mg. gallamine followed by subsequent intermittent doses of 40 mg., was administered as the muscle relaxant. An IPPR technique was instituted via a carbon dioxide circle absorption system with nitrous oxide and oxygen, with minimal ether. Throughout the operation, a total of 300 mg. gallamine was administered.

At operation a bilateral loin approach was used with elevation of a kidney rest. Blood transfusion was commenced and blood replaced as lost. The course of the anaesthetic was comparatively uneventful except for a fall in the systolic B.P. from the pre-anaesthetic level of 160 to an average of 120 mm.Hg. This varied being up to 150-180 mm.Hg systolic on occasion, synchronous with manipulation of the adrenal gland. Tracheobronchial toilette was performed during the change-over from the right lateral to the left lateral position. During the procedure 1,500 ml. compatible blood was transfused. 4 ozs. ether and 300 mg. gallamine was used by the conclusion of the operation. While the second operative loin wound was being sutured, spontaneous respiration commenced. At the conclusion of operation, 1 mg. neostigmine preceded by 0.6 mg. atropine was given and

spontaneous / ...

spontaneous respiration of adequate volume returned. Anaesthesia was discontinued and tracheobronchial toilette performed via the endotracheal tube, using suction aspiration. The tube was removed and the patient returned to her bed. She regained consciousness and appeared to be alert. At the same time she developed a severe attack of hiccoughs. Respiratory inadequacy developed, and the patient became distressed. Oxygen was administered and a further 1 mg. neostigmine given. Though she could now fully open her eyes and had full muscle power in her jaws, the respiratory inadequacy persisted. Manual assistance of respiration with oxygen was continued. The patient re-intubated, further tracheo-bronchial toilette was performed and IPPR with oxygen, using a Bennet ventilator, was commenced. After 15 minutes the pump was discontinued, the patient breathing adequately. After a further 10 minutes, the tracheal tube was removed. Very soon afterwards she developed hiccoughs again, with respiratory inadequacy, and became very distressed again. Intubation and tracheo-bronchial toilette were again performed and respiratory assistance with oxygen and the Bennet ventilator resumed. Her condition improved once more. An X-ray chest taken at this stage revealed basal atelectasis of the left lung with patchy atelectasis of the right lung. It was now decided that provision would have to be made for possible prolonged respiratory assistance and adequate continued tracheo-bronchial toilette. A tracheotomy was performed, for which the patient was re-anaesthetised with nitrous oxide and oxygen. Preceding this operation, a further 100 mg. hydrocortisone was given. After this operation, on discontinuance of anaesthetic, the patient regained consciousness and respiratory assistance was continued with the Bennet ventilator. Pethilorfan 50 mg. was given for analgesia. After a further 30 minutes, the respiration again appeared adequate. Assistance with the Bennet ventilator was stopped and thepatient was allowed to breathe spontaneously. Further X-rays of the chest at this time revealed the lungs to be clearer than formerly. Her condition continued to be satisfactory for 12 hours and she again developed respiratory distress and the B.P. commenced to fall. She appeared to develop circulatory failure. IPPR with the Bennet ventilator was immediately commenced and an infusion of noradrenaline was started intravenously. However, in spite of these resuscitative measures, her condition continued to deteriorate and she died 3 hours later - 16½ hours post-operatively in all.

AUTOPSY:

Diffuse carcinomatosis with secondaries in (1) right frontal lobe of the brain, (2) pathological fracture of the right humerus, (3) secondaries in the vertebrae, ribs and sternum, liver. Nothing abnormal found in the lungs. Kidney: small unilocular cyst of the left kidney. Evidence of previous bilateral oopherectomy and recent bilateral adrenalectomy. Tracheotomy had been performed. Cause of death - diffuse carcinomatosis.

COMMENT:

Following bilateral adrenalectomy, the patient had respiratory inadequacy post-operatively. She died 16½ hours after operation in circulatory failure, which might have been due to adrenal insufficiency. Although 200 mg. cortisone had been given on the day of operation, this could well have been inadequate. The exact cause of respiratory inadequacy is difficult to determine. The first possibility that may be considered is that or residual curarisation together with the additional possibility in this case of the myasthenic syndrome of carcinomatous neuropathy. The total dose used of the relaxant gallamine was large, when it is remembered that it was used in association with ether. With regard to the former possibility, the patient did not appear clinically curarised following the administration of 2 mg. neostigmine at the end of the anaesthetic; she opened her eyes well, had good muscle power in her jaws and appeared alert. The latter possibility, too, is regarded as unlikely. This condition is rare and is usually related to bronchial carcinoma, whereas this patient had diffuse carcinomatous secondary deposits from the breast, with no pulmonary lesions. Clinically, a suggestive fact in favour

of / ...

of such a diagnosis was the continued fatiguing of the respiratory effort which was improved by periods of IPPR treatment. No further neostigmine was given after the initial 2 mg. and no attempt was made to explore this diagnosis electromiographically. The presence of a cerebral secondary brings to mind the possibility of cerebral vital centre failure, because of the presence of a space-occupying lesion. However, this secondary was in the frontal lobe and was not very large.

The patient was obese and had chronic bronchitis. The basal atelectasis of the left lung, and patchy atelectasis of the right, demonstrated on X-ray, was probably the result of inadequate tracheobronchial toilette during the operation, and to splinting of the lung bases by the elevated kidney rest. Not only would this have caused ventilation/perfusion disturbances, but would also have caused a marked decrease in pulmonary compliance. This would have accentuated the effect of any muscle weakness of residual curarisation on the volume of pulmonary ventilation. However, the post-operative bronchial toilette led to radiological improvement. It is strange that no gross pulmonary changes were found at autopsy. Whatever the reason for the post-operative respiratory inadequate function and ultimate circulatory failure, it appears to have been precipitated by operation and anaesthesia.

Anaesthesia must be regarded as a significant contributory factor though doubtless the patient's disease and the effects of bilateral adrenalectomy itself played an equally important part.

PREVENTABILITY:

Although the precise cause of this death is not clear, correctable faults are evident in the management of the anaesthetic:-

(1) The dose of gallamine used is unnecessarily large when it is considered that this was used in association with ether.

(2) In a patient known to have chronic bronchitis, tracheobronchial toilette during operation was not adequate.

(3) The anaesthetist was not alert to the splinting effect on the dependant lung base of a raised kidney rest, especially inan obese patient such as this. Ventilating volumes used during IPPR were probably inadequate to prevent this.

Though this patient's death was in large measure the result of her existing disease, because of the above described faults in the management of anaesthetic, this death is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
170.1.60	2	No comment	< 24	?Secretional bronchial obstruction. Respiratory failure. Gross emphysema.	No

Name: John Milton Kemp Age: 77 Sex: M Race: E

Disease: Haematemesis from gastric ulcer. Operation: Partial gastrectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Grave condition. The patient was a known alcoholic and had a severe haematemesis. In spite of blood transfusion, the haemoglobin was now 8 gm.%. There was gross emphysema with chronic bronchitis. There were coarse crepitations in both lung fields with very poor air entry, and numerous rhonchi throughout the chest. Gastric suction had been instituted.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour before surgery.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, administered via a circle absorption system. When the patient was adequately anaesthetised, topical analgesia of the larynx and oral intubation followed. Anaesthesia was continued with nitrous oxide and oxygen with a trace of ether, via a carbon dioxide circle absorption system, using an IPPR technique. Gallamine in divided doses, totalling 80 mg., was administered throughout the operation.

Partial gastrectomy was performed, taking 1 hour. Throughout, the course of the anaesthetic was untoward. 4 pints blood were transfused during the operation and 10 cc. 10% calcium gluconate. At the end of the surgical procedure and on discontinuance of the anaesthetic, respiration was found to be inadequate in that the patient became cyanosed, although the respiratory excursions appeared of a reasonable magnitude.

This was thought to be due to (a) a minor degree of residual curarisation, (b) pre-existing pulmonary disease, (c) the possibility of overloading of the circulation with the 4 pints transfused blood. IPPR was therefore continued while the following treatment was instituted: (a) venesection and removal of 1 pint blood, (b) digitalisation of the patient with two doses of Digoxin 0.25 mg., (c) 500 mg. aminophyllin, and (d) 2.5 mg. neostigmine preceded by 1.2 mg. atropine. This treatment was performed over a period of 2 hours after operation, IPPR being continued during this time. By then the patient was awake moving and responding to verbal stimuli. The respiratory excursions were considered adequate and the patient maintained a good colour with spontaneous respiration, breathing air. He was returned to the ward with the endotracheal tube in situ. The anaesthetist subsequently suggested that a tracheotomy be performed for tracheo-bronchial toilette during the post-operative phase, but the surgeon did not agree to this. Four hours after surgery the endotracheal tube was removed and the patient's respiration appeared adequate. He developed severe respiratory failure 20 hours post-operatively and died 22 hours after operation.

AUTOPSY:

No autopsy.

COMMENT:

This death resulted from the patient's existing disease functionally ...

worsened by the known effects of upper abdominal surgery. The initial difficulties in restoring normal spontaneous respiration following operation and anaesthesia had been dealt with well by the anaesthetist. The final respiratory difficulty was probably due to secretional obstruction of the bronchi. The patient might have had a better chance of survival in the post-operative period had an elective tracheotomy been performed for tracheo-bronchial toilette in the post-operative period. This was suggested to the surgeon who did not concur.

Anaesthesia is not regarded as contributory to this patient's demise when it occurred.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
171.1.60	2	No comment	< 24	Extensive pontine haemorrhage	Yes

Name: Patrick Peat Age: 48 Sex: M Race: E

Disease: Subarachnoid haemorrhage, anterior cerebral artery aneurysm with internal carotid thrombosis.

Operation: Carotid angiography. Craniotomy and clipping of aneurysm under hypothermia/hypotension.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had had 4 recent episodes of subarachnoid haemorrhage. He was comatose and had a B.P. of 140/75 mm.Hg, pulse rate 100/minute. Numerous rhonchi were audible in both lung fields with marked expiratory wheezing. Pre-operatively, antibiotic therapy had been instituted to control the pulmonary infection.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen followed by 100 mg. thiopentone, succinylcholine 50 mg., ventilation with oxygen topical analgesia of the larynx and oral intubation. Spontaneous respiration was allowed to recommence and anaesthesia was dontinued during angiography with nitrous oxide and oxygen with Halothane in concentrations of 15 - 18%, administered via a carbon dioxide circle absorption system, with spontaneous breathing. The patient was placed in cooling blankets for hypothermia, during which time an IPPR technique was instituted through a carbon dioxide circle absorption system, the administration of Halothane being discontinued, nitrous oxide and oxygen being used. In 1½ hours the temperature was reduced to 85°F.

When dissection and clipping of the anterior cerebral artery aneurysm was performed, hypotension had been induced with the combined use of Halothane 1.5% in oxygen, and an Arfonad drip infusion. Systolic pressure was reduced to 60 mm.Hg. After 60 minutes of hypotension the clipping of the aneurysm was completed and the B.P. was allowed to rise. During the hypotensive phase the temperature was 85°F. Thereafter the Arfonad and Halothane were discontinued and anaesthesia was maintained with nitrous oxide and oxygen with continued IPPR until the end of the operation. The temperature was allowed to rise and when this reached 91°F spontaneous respiration returned. At the conclusion of the procedure, after 5 hours of anaesthetic, the patient's temperature was 94°F and the B.P. 140 mm.Hg systolic. In all, 700 ml blood had been transfused during surgery, being the estimated loss. The level of consciousness at the conclusion of the operation and on discontinuance of anaesthetic was, as pre-operatively, grossly depressed. Two hours after the operation a tracheotomy was performed. Tracheo-bronchial toilette produced much purulent secretion from the bronchi. One hour later there was a distinct deepening of the level of coma and another hour later the administration of Daptazole 30 mg. intravenously was performed because of periodic respiration. This was ineffective. The patient died after a further 5 hours, 13 hours post-operatively.

AUTOPSY:

Very extensive haemorrhage into the hypothalamus and pons.

COMMENT:

This patient died of hypothalamic and pontine haemorrhage as a result of the surgical procedure. Anaesthesia is not considered contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
172.1.60	2	No comment	< 24	Mitral regurgitation. Pulmonary oedema.	Yes

Name: Sargent Sandelo. Age: 32 Sex: M Race: B

Disease; Mitral stenosis, pulmonary hypertension, tricuspid incompetence. Operation: Mitral valvotomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had rheumatic heart disease with mitral stenosis, tricuspid incompetence and pulmonary hypertension. He had been in cardiac failure which was now controlled with digitalis. Before operation the B.P. was 120/80 mm.Hg, pulse rate 56/minute.

PREMEDICATION:

Omnopon gr. 1/3, scolpolamine gr. 1/150 administered 1½ hours before operation.

ANAESTHETIC:

Following 5 minutes of pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen. dTc 30 mg. was then administered and IPPR instituted. Oral intubation was performed and anaesthesia maintained with nitrous oxide and oxygen using an IPPR technique via a circle absorption system. The course of anaesthesia during the operation was relatively untoward.

At the conclusion of operation, which took 2 hours 20 minutes, 2½ mg. neostigmine preceded by atropine 1/50 gr. was given. When the anaesthetic was discontinued the patient rapidly regained consciousness and had adequate spontaneous respiration. During the post-operative period the patient's condition deteriorated and he developed a pulmonary oedema. In all probability this was due to some degree of mitral regurgitation following mitral valvotomy. He died 20 hours post-operatively.

AUTOPSY:

The patient's heart was enlarged with mitral stenosis (which had been dilated). Evidence of surgical procedure on heart. Pulmonary oedema. Cause of death undetermined. Evidence of cardiac disease and surgical procedures on the heart.

COMMENT:

This patient appears to have died from the effects of mitral regurgitation following, or worsened by, the operation of mitral valvotomy. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
173.1.60	3	No comment	ORD	Exsanguination. Uncontrollable haemorrhage. Cardiac arrest.	Yes

Name: Ruby Buckley Age: 32 Sex: F Race: C

Disease: Mitral stenosis. Operation: Mitral valvotomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had had a mitral valvotomy performed 5 years previously with some improvement but the valve had re-stenosed and a further valvotomy was felt necessary. Chronic cardial failure had been present, was now controlled by digitalisation. There was slow auricular fibrillation and right bundle branch block. B.P. 130/80 mm.Hg.

PREMEDICATION:

Pethidine 75 mg., atropine 1/100 gr. 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 300 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued with nitrous oxide and oxygen with a trace of ether, via a circle absorption system. At the commencement of operation, ether was discontinued. dTc was given in divided doses, totalling 12 mg. during the operation, and an IPPR technique was used. The course of anaesthesia was relatively untoward until the opening of the left atrium.

The lung was adherent to the left parieties and the pericardium to the heart and lung. The left auricular appendage was absent. A pursestring suture was placed in the left auricular wall which was incised and the mitral valve found to be very tightly stenosed on palpation. The left auricle was large. Digital palpation of the valve caused slowing of the heart rate to 50/minute. Blood replacement at this time had been 3/4 pint, which was the estimated loss. An attempt to dilate the valve via a transventricular approach, with a Tubb's dilator, caused multiple ventricular extrasystoles and severe blood loss. Blood replacement was speeded up under pressure via two infusion systems. Dilatation of the valve was unsuccessful and marked blood loss occurred via the ventriculotomy. Blood transfusion was given as rapidly as possible, 6 pints being given in a very short time. However, ventricular fibrillation supervened. Electrical defibrillation, intracardiac adrenaline and calcium chloride and cardiac massage were to no avail.

AUTOPSY:

Severe mitral stenosis. Enlarged heart. Surgical incision in the heart, unsutured (because of sudden death during operation). 800 ml. blood in left pleural cavity. Liver cirrhosis. Spleen enlarged. Cause of death: heart failure due to mitral stenosis, death being precipitated by surgical procedure.

COMMENT:

Death resulted from ventricular fibrillation precipitated by exsanguination from a gross uncontrollable haemorrhage from ventriculotomy, and surgical dilatation of the mitral valve. Anaesthesia was not contributory. As death occurred during anaesthetic, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
174.1.60	2	No comment	< 24	Mitral incompetence.	Yes

Name: Alfred de Reuck Age: 47 Sex: M Race: E

Disease: Auricular septal defect (with cleft mitral valve). Operation: Repair of atrial septal defect on cardio-pulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Dyspnoea on exertion and palpitations. The patient's jugular venous pressure was elevated to 1.2 cm. above the clavicles and he had numerous extrasystoles. B.P. 130/80 mm.Hg. He was not digitalised.

PREMEDICATION:

Seconal gr. 3, orally, 3 hours before operation, atropine gr. 1/100 1 hour pre-operatively.

ANÆSTHETIC:

Anaesthesia was induced with thiopentone, succinylcholine, IPPR with oxygen, topical analgesia of the larynx and oral intubation. dTc was administered and anaesthesia maintained with nitrous oxide and oxygen, via a circle absorption system with an IPPR technique. Induction of anaesthesia and its maintenance were uneventful. At the conclusion of the operation, which took 8 hours 21 minutes, 1 mg. neostigmine preceded by atropine gr. 1/100 was administered. On the discontinuance of anaesthetic, the patient awoke rapidly. Respiration was spontaneous and adequate. Following his return to the ward the patient rapidly developed severe cardiac failure and died 5 hours after surgery.

AUTOPSY:

Atrial septal defect with defect in the foramen ovale repaired. Split mitral valve cusp. Heart very enlarged, weighing 1000 gm. Tuberculous pericarditis with calcification and caseation. Lungs congested and oedematous.

COMMENT:

Death resulted from mitral regurgitation due to a cleft mitral valve. The closure of the atrial septal defect precipitated overfilling of the left atrium. This is a surgical death. Anaesthetic management is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
175.1.60	2	No comment	< 24	Undetermined.	No

Name: Jan Martens Age: 67 Sex: M Race: E

Disease: Chromophobe adenoma of the pituitary. Operation: Craniotomy. Excision of pituitary ademoma.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Good. Other than for the symptoms of the pituitary lesion, the patient was fit. There were occasional ventricular extrasystoles but otherwise the ECG was normal. Pre-operatively he was given 100 mg. hydrocortisone.

PREMEDICATION:

Omnopon gr. 1/6, atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone, succinylcholine, ventilation with oxygen, topical analgesia of the larynx and oral intubation. IPPR was continued until spontaneous respiration commenced 4 minutes later. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a Magill semi-open system with intermittent addition of ether vapour. During the 3½ hours of the operation, the course of anaesthesia was uneventful.

At the conclusion of the operation, on discontinuance of the anaesthetic, the patient rapidly recovered consciousness. His respiration was normal and the B.P. returned to the pre-operative level. For a while post-operatively he was apparently quite well and fully conscious. Twelve hours after operation he had a nose bleed, became restless and pulled off the dressing. 10 mg. Physeptone was given as sedation. After 3 hours he again became restless and again pulled the dressing off, and 30 minutes later he died suddenly.

AUTOPSY:

No autopsy.

COMMENT:

Whatever the cause of this patient's death, in all probability a post-operative cerebral haemorrhage, it appears to have been related to the effects of surgery and not to the anaesthetic.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
176.1.60	1	Probably	< 24	Incompatible blood transfusion (anaesthetist error)	Yes

Name: Daniel van Barlin Age: 67 Sex: M Race: C

Disease: Prostatic adenoma Operation: Transvesical prostatectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had pulmonary emphysema. B.P. 160/90 mm.Hg. Haemoglobin 12 gm.%. One week previously he had presented with acute retention of urine, which had been treated conservatively with catheter drainage of the bladder.

PREMEDICATION:

Phenergan 25 mg., pethidine 25 mg., atropine gr. 1/100 given 45 minutes before the operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone followed by inhalation of nitrous oxide and oxygen, via a circle absorption system. After oral intubation, anaesthesia was maintained with an IPPR technique with nitrous oxide and oxygen and a trace of ether, administered via a circle absorption system. Gallamine in divided doses, totalling 80 mg. during the operation, was used as the relaxant. The course of operation and anaesthesia were uneventful. 1 pint of blood was transfused towards the end of the operation. At the end of the procedure the patient's condition was satisfactory. Neostigmine 0.5 mg. preceded by atropine gr. 1/100 was given and normal adequate spontaneous ventilation recommenced. Consciousness was rapidly regained after discontinuance of the anaesthesia. On return to the ward, however, the patient was shivering severely and was cyanosed. The pulse was not palpable. It was now discovered that, due to the anaesthetist's error, the pint of blood administered had been incompatible. This patient was a Group O rhesus positive and had received Group A rhesus positive blood.

Fifteen minutes after his return to the ward the patient had severe rigors, tachypnoea and marked peripheral cyanosis. The B.P. was now 180/150 mm.Hg. The senior anaesthetist, physician and pathologist were called in consultation and recommended the following treatment: (1) 2 pints compatible blood, (2) alkalization of the urine with sodium bicarbonate 100 gm. orally over 6 hour periods, (3) hydrocortisone 100 mg. to be followed by 50 mg. 6 hourly. This treatment was carried out and the patient's condition remained fairly well until 5 hours after operation, when sudden circulatory collapse developed, followed shortly by respiratory failure. Artificial respiration was commenced together with the administration of nikethamide intravenously, but to no avail and he died 5 hours post-operatively.

AUTOPSY:

Evidence of prostatectomy and vasectomy. Coronary arteriosclerosis. History of incompatible blood transfusion.

COMMENT:

Death appears to have resulted from the transfusion of 1 pint incompatible blood. The patient was a Group O rhesus positive and

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received 1 pint of incompatible Group A rhesus positive blood during the operation. In that this unfortunate accident was due to an error on the part of the anaesthetist, who failed to check the name and number on the ordering slip for blood against the patient's folder, and that the administration of blood during operation falls into the anaesthetists' sphere of responsibility, this death must be regarded as one due to the management of the anaesthetic.

PREVENTABILITY:

In that this error occurred because of failure on the anaesthetist's part to check the name on the slip for ordering blood against that on the patient's folder, an accepted elementary precaution before the administration of blood, this death must be regarded as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
177.1.60	2	No comment	< 24	?Cerebral oedema.	Yes

Name: Robert MacWilliams Age: 60 Sex: M Race: E

Disease: Post-leucotomy meningo-cephalitis. Operation: Cerebral drainage through existing burrhole.

Anaesthetic risk: 3,emergency.

PRE-OPERATIVE STATE:

Eleven days previously this patient underwent a bilateral pre-frontal leucotomy. In the post-operative period, infection had supervened and the patient was now semiconscious. B.P. 110 mm.Hg systolic, pulse rate 85/minute, and respiration only 12/minute. Pre-operatively had received fluid requirements intravenously. The B.P. had dropped pre-operatively and wyamine sulphate 10 mg. had been administered intramuscularly.

PREMEDICATION:

Atropine gr. 1/100 given 30 minutes before operation.

ANAESTHETIC:

Following pharyngeal toilette, anaesthesia was induced with thiopentone 250 mg., followed by topical analgesia of the larynx and oral intubation 3 minutes later, during which time nitrous oxide and oxygen were administered via a Magill semi-open system. During the operation, anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether vapour, via a Magill semi-open system, with spontaneous breathing.

The operation lasted 25 minutes and the patient's general condition remained static. Following the conclusion of the operation and on discontinuance of the anaesthesia, the patient returned to much the same level of consciousness as he had been at before surgery. He died 22 hours after the operation.

AUTOPSY:

Intense oedema and congestion of brain, lungs, liver and kidneys. Small atrophic spleen. Mild fatty infiltration of the liver.

COMMENT:

Although one must question the administration of 250 mg. thiopentone to a patient already semiconscious, this death does not appear to be related to the administration of an anaesthetic, but rather to the patient's existing disease. This itself was the result of previous surgery.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
178.1.60	2	No comment	< 24	Coronary thrombosis	No

Name: Elizabeth Duncan Age: 83 Sex: F Race: E

Disease: Acute cholecystitis, carcinoma of the gall bladder and emphysema of gall bladder.

Operation: Cholecystectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Fair, considering her age. B.P. 150/85 mm.Hg, pulse rate 80/minute. The patient had kyphosis and a poor respiratory excursion with senile emphysema. Chronic bronchitis. She was not jaundiced and there was no enlargement of the liver. Haemoglobin 12 gm.%.

PREMEDICATION:

Pethilorfan 75 mg., atropine gr. 1/100, given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg, succinylcholine 25 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system, using an IPPR technique. Gallamine, initially 60 mg. followed 30 minutes later by another 20 mg., was used as the relaxant. During the operation two doses 10 mg. each Pethidine were administered. The course of anaesthetic and operation were uneventful and the procedure lasted 1 hour 25 minutes. Blood loss during surgery was approximately 100 ml. During the operation, 200 ml. 5% dextrose in water was administered.

At the conclusion of the operation curarisation was reversed with 1.5 mg. neostigmine preceded by atropine gr. 1/100. B.P. at this stage was 150 mm.Hg systolic, pulse rate being 110/minute. Respiration was full and adequate. Consciousness was rapidly regained and the patient returned to the ward.

Four hours post-operatively her condition was satisfactory but the patient complained of abdominal pain. Pethilorfan 50 mg. was administered. After 12 hours a poor pulse rate and B.P. were noted the B.P. being 80 mm.Hgsystolic. 1 pint blood was transfused quite rapidly but failed to change the B.P. An ECG taken at this time showed left bundle branch block with marked ischaemic changes. Congestive cardiac failure supervened. An infusion of noradrenaline failed to produce any rise in the B.P. and the patient died 14 hours after operation.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of post-operative myocardial infarction and congestive cardiac failure. Anaesthesia itself does not appear to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
179.1.60	3	No comment	ORD	Cardiac arrest. (Ventricular fibrillation)	Yes

Name: Rosina Pikeur Age: 23 Sex: F Race: C

Disease: Mitral stenosis. Operation ; Mitral valvotomy

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had mitral stenosis with grade 3 dyspnoea on exertion. She had pulmonary hypertension and had been in chronic cardiac failure, which had been controlled by digitalisation and diuretics. She still had a palpable hepatomegaly and also splenomegaly.

PREMEDICATION:

Pethidine 100 mg., scolpolomine gr. 1/150, given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with a sleep dose of thiopentone 200 mg., followed by inhalation of nitrous oxide, oxygen and gradually added ether; via a circle absorption system, with spontaneous breathing. After the induction of light surgical anaesthesia and topical analgesia of the larynx, oral intubation was performed. The administration of ether was then discontinued and, after positioning of the patient, anaesthesia was maintained with nitrous oxide, and oxygen, administered via a circle absorption system by an IPPR technique. dTc was used as the relaxant, only 6 mg. being given. On this dose, respiration was easily controlled. The operation and course of anaesthesia were untoward until the mitral valvotomy was performed, 1 hour after the start of operation. After mitral valvotomy, the B.P. - which had momentarily dropped - recovered reasonably rapidly to a level of between 90 and 100 mm.Hg systolic, the pulse rate being 75/ minute. Until this stage blood loss had been replaced as lost, by transfusion. 5 minutes after completing the mitral valvotomy, while the atrial incision was being closed, ventricular fibrillation supervened suddenly. Cardiad massage and electrical defibriklation were resorted to. For the next 10 minutes the ventricular fibriklation proved refractory and despite repeated shocks it kept recurring. Adrenaline 1:10,000 was injected into the left ventricle and, after massage and electrical defibrillation, the heart beat returned but cardiac astion was inadequate, the B.P. being merely 55 mm.Hg systolic. Shortly thereafter there was a massive haemorrhage and the patient died.

AUTOPSY:

Evidence of heart disease. Dilated right and left auricles. Enlarged right ventricle. Thickened mitral cusps. Mitral valvotomy had been performed. Collapsedleft lung. Blood in left pleural cavity.

COMMENT:

The precise cause of the cardiac arrest cannot be accurately diagnosed. For the purpose of this survey it is important to evaluate any possible contributory role played by the anaesthetic. Ventricular fibrillation supervened shortly after mitral valvotomy, soon after placement of the atrial clamp preparatory to resuture of the atrial appendage. Damage to, or occlusion of the left coronary artery by the clamp is a known danger at this point of the operation. Furthermore, the refractoriness of the ventricular fibrillation to electrical defibrillation (often indicating ischaemia of the myocardium) would support such a diagnosis. Until this stage

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course of the anaesthesia per se had been untoward, and nothing in its management had altered.

In view of the smooth course until this time, it is difficult to see what factors in the anaesthetic management could have resulted in cardiac arrest at this stage.

The final haemorrhage that resulted in death must have been caused by the dislodgement of the atrial clamp, or dehiscence of the atrial sutures caused by the handling of the heart during cardiac massage and electrical defibrillation. Overall, this ventricular fibrillation and ultimate haemorrhage appear to have been related to surgical manoeuvres and to mishap rather than to any incident in the anaesthetic management. Therefore this death is classified as one due to surgery but, as it occurred while the patient was anaesthetised, the case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
180.1.60	2	No comment	< 24	Massive intestinal infarction	Yes

Name: James Poole Age: 45 Sex: M Race: C

Disease: Mesenteric thrombosis. Operation: Laparotomy. Cholectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Very poor; having suffered from malignant hypertension for some time the patient's B.P. was now only 140/80 mm.Hg, and pulse rate was 120/minute with auricular fibrillation. Gastric aspiration had been instituted, estimated blood loss replaced and fluids administered intravenously.

PREMEDICATION:

Atropine gr. 1/100, given immediately pre-operatively.

ANAESTHETIC:

Local analgesia of the larynx and trachea was followed by anaesthesia with inhalation of nitrous oxide and oxygen with gradually added ether, via a circle absorption system, with spontaneous breathing. Once consciousness was lost, oral intubation was performed with a No. 10 cuffed endotracheal tube. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether via a circle absorption system, using an IPPR technique. Gallamine in divided doses, totalling 210 mg. throughout the 3¾ hours of operation, was used as the relaxant.

At operation an inferior mesenteric artery thrombosis was found with massive infarction of the large bowel. The entire colon was resected. Blood was replaced as lost during the procedure, a total of 1,200 ml. being transfused. Circulatory homeostasis was well maintained. At the end of operation, 3.5 mg neostigmine was given preceded by gr. 1/50 atropine, and spontaneous adequate respiration was soon established. On discontinuance of the anaesthetic, the patient regained consciousness rapidly and was returned to the ward. He died 12 hours post-operatively.

AUTOPSY:

Excess of blood stained fluid in the peritoneal cavity. Peritonitis. A cholectomy had been performed. No large bowel was present. Small gut perforation was imminent on the serosal aspect which showed evidence of infarction; small gut was distended. The entire gastro-intestinal tract mucosa, commencing at the cardiac sphincter, was intensely congested and haemorrhagic, and covered by a greyish, flaky false membrane, maximal at the mucosal crest. Extreme oedema of the gut wall. Stomach and small bowel contained 1 pint blood. The anastomotic sites were intact. Venous and arterial hepatic infarction. Small infarct of 1 adrenal gland. Reactive splenomegaly. Intense renal congestion. Free fluid in both pleural cavities. Subpleural petechiae. Intense pulmonary congestion and oedema. Hydropericardium. Right auricular and right ventricular dilatation. Left ventricular hypertrophy. Moderate coronary atheroma (not occlusive). Calcified hila lymph node.

COMMENT:

This death was due to massive infarction on the large and small bowel. Anaesthesia was in no way contributory to the patient's demise.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
181.1.60	2	No comment	< 24	Undetermined.	Yes

Name: Mogamat Sait Age: 40 Sex: M Race: C

Disease: Tetralogy of Fallot Operation: Correction of Fallot's tetralogy on cardio-pulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

The patient was not in cardiac failure and was notdigitalised. B.P.120/50 mm.Hg, pulse rate 108/minute.

PREMEDICATION:

Seconal gr. 3, orally, 2 hours before operation and atropine gr. 1/100 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 175 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system using an IPPR technique. dTc was given as the relaxant, a total of 22 mg. being used throughout the operation which lasted 13 hours. Induction and maintenance of anaesthesia was satisfactory and relatively untoward. During bypass, anaesthesia was maintained with intermittent doses of thiopentone and dTc. At this period of the operation, extracorporeal hypothermia down to 14°C was induced. The bypass lasted 176 minutes. The heart defibrillated spontaneously on rewarming. At the conclusion of the operation, thoracic haemorrhage occurred and the patient was immediately re-anaesthetised and thoracotomy performed to secure haemostasis. This took 4½ hours. At the conclusion of the operation 1 mg. neostigmine preceded by atropine gr. 1/100 was given and normal spontaneous respiration returned. On discontinuance of the anaesthetic, the patient rapidly regained consciousness and was in apparently satisfactory condition. The B.P. was 90 mm.Hg systolic and the pulse rate 110/minute. However, he died 9 hours post-operatively.

AUTOPSY:

Tetralogy of Fallot. Evidence of surgery on the heart. Sub-arachnoid haemorrhage over the left and right frontal regions and the right posterior pole. No intracerebral disease evident. One litre blood in thepleural cavities.

COMMENT:

This death appears to have resulted from the effects of surgery and continuing haemorrhage. Anaesthesia is not considered to have been contributory in any way.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
182.1.60	1	No verdict	ORD	Cardiac arrest.	Yes

Name: Antony Appel Age: 64 Sex: M Race: C

Disease: Prostatic urinary obstruction. Operation: Transurethral resection by the punch technique.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient was admitted 2 days before operation, suffering from urinary obstruction with overflow incontinence and retention. Uraemic, the blood urea was 220 %. Temperature 103°F, probably from cystitis. B.P. 180/80 mm.Hg and there was cardiomegaly. Pulmonary emphysema, though not gross, was present. There was no jaundice or visceromegaly. The bladder was drained by means of a Foley's catheter for 24 hours pre-operatively, but this had been removed for 24 hours at the time of surgery. At this time the temperature was 101°F but this had risen to 103°F again on the day of surgery.

PREMEDICATION:

Morphine gr. 1/8, atropine gr. 1/100, given 1¼ hours pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg, followed by the inhalation ofnitrous oxide and oxygen with gradually added ether, via a Magill semi-open system, with spontaneous breathing. After topical analgesia of the larynx, oral intubation was performed. As diathermy was to be used during operation, ether was discontinued and trichlorethylene substituted, with nitrous oxide and oxygen, for the maintenance of anaesthesia, via a Magill semi-open circuit with spontaneous breathing. In all, during the 1 hour used, 15 ml. trichlorethylene was used. The course of anaesthesia was uneventful throughout the 1¼ hours of operation. Initially 140 mm.Hg systolic, the B.P. fell to 100 mm.Hg systolic on induction, but subsequently rose to 110 mm.Hg systolic, where it remained for the major part of the operation. Commencing at 86/minute, the pulse rate rose during the first half of operation to 100/minute and then fell to 96/minute, where it remained throughout the remainder of the procedure. The pulse was steady with no irregularities. Blood loss was apparently not great, estimated by the anaesthetist as less than 1 pint and no blood transfusion was instituted.

After 1¼ hours of operation, the transurethral resection was complete and the surgeon was checking the prostatic bed for adequate haemostasis when there was a sudden drop in B.P. and the pulse was found to be impalpable. Respiration ceased and cardiac arrest was diagnosed. IPPR with oxygen was commenced. There now followed a period of heistancy and internal cardiac massage was only commenced after a delay of 5 minutes from the time cardiad arrest had been diagnosed. The heart was flabby and in asystole on palpation. Cardiac massage was continued for 26 minutes, when 0.5 ml. adrenaline 1:1,000 was injected into the left ventricle, without response. After a further 8 minutes, 10 ml. 10% calcium chloride was injected into the left ventricle, without response either. After another 10 minutes, atropine gr. 1/100 was administered intravenously, followed in another 10 minutes by adrenaline 1:100,000 intravenously. Throughout this time cardiac massage was continued, without response. After a further 15 minutes another 0.5 ml. adrenaline was injected (1:1,000) into the left ventricle, followed 1 minute later by 10 ml. calcium chloride. None of these measures availed. After another 10 minutes 0.5 ml. isoprenaline 1:5,000 was injected intravenously followed after a further 5 minutes by another 0.5 ml. isoprenaline. There was no response whatsoever.

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AUTOPSY:

Disease of the heart, which was enlarged. Marked coronary atherosclerosis. Urogenital disease. Dilatation and thickening of bladder with dilatation of ureters and renal pelves. Evidence of surgery to the prostate.

COMMENT:

It is difficult to form any conclusive ideas as to the precise cause of cardiac arrest, although many of the possible causes could have been present in this case.

1. Anoxia. Gross anoxia of a grade sufficient to cause cyanosis was certainly not present. However, the patient had a pyrexia of 103°F, he was toxic and had a higher than normal oxygen demand. That the inspired mixture contained 30% oxygen would probably ensure the absence of anoxia, even though he had emphysema and was permitted to breathe spontaneously while anaesthetised.

2. Hypercarbia. The fact that he had emphysema and was permitted to breathe spontaneously while anaesthetised may have resulted in some degree of hypercarbia. The constancy of the B.P. and pulse rate probably belie this.

3. Anaesthetic drugs. Though the use of trichlorethylene is known to be associated - especially in excessive doses - with cardiac arrhythmiasand even with cardiac arrest, the dosage used here was not excessive. Further, the pulse and B.P. had been in a very steady state for approximately 1 hour of trichlorethylene administration, with no arrhythmia at all.

4. Ischaemia. It is possible that the anaesthetist misjudged the degree of blood loss, as this was mixed with the bladder washings. However, if this had been really severe one could well expect the blood loss to have been reflected by some change in the pulse rate and/or B.P.; there was no such change.

5. Water intoxication. Gross haemodilution and water intoxication from absorption of bladder washing fluid from venous sinuses and the bladder mucosa, with overloading of the circulation, has been described as occurring during transurethral resection. Should this have been the case, one would have expected some change in the pulse rate and a rise in venous pressure, though the latter could have been missed by the anaesthetist. The fluid used here was distilled water.

6. Toxicity from infection, pyrexia and uraemia. The patient suffered from all these conditions, any of which would increase the liability to cardiac arrest from other causes . He was so pyrexial that one must wonder why he was submitted to an operation of this magnitude. Infected material may well have been infused directly into the blood stream through cut prostate venous sinuses by the bladder washing fluid, and this would have led to acute pyaemia.

7. Vagal reflex from stimulation of the bladder neck and prostate bed during light anaesthesia. This is another possible cause of cardiac arrest. However, if so, one is left wondering why the stimulation of the same area in the prededing hour had produced no cardiac slowing or arrhythmias.

Whatever the cause of cardiac arrest in this case, the institution of cardiac massage after a delay of 5 minutes was excessively tardy and must virtually have guaranteed an unsuccessful outcome. The injection of adrenaline was also tardy and the dose of isoprenaline finally used was excessive. Though at the time this latter was used it doubtless made no difference. One is left wondering why transurethral resection could not have been postponed until the patient's temperature was normal and the blood urea at a lower level.

Whatever ...

Whatever the cause of cardiac arrest in this case, one is forced to conclude that the anaesthetic played a significant contributory role. Doubtless the general condition of the patient also played a markedly contributory part.

PREVENTABILITY:

In view of the overall uncertainty of the precise causation of the cardiac arrest in this case, even though one may conclude that the anaesthetic was significantly contributory, one cannot draw any firm conclusions as to the preventability of the outcome. The decision to perform a transurethal resection on a patient in this severely pyrexial toxaemic state is open to criticism, but this was a surgical decision.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
183.1.60	2	No comment	< 24	Cerebral laceration. Head injury.	Yes

Name: Elizabeth Bezuidenhout. Age: 1½ Sex: F Race: E

Disease: Severe head injury. Cerebral laceration with subarachnoid haemorrhage.

Operation: Pneumo-encephalogram.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This small patient had suffered a severe head injury and was in extremis. There was an irregular pulse and irregular respiration. She was already in a state of decerebrate rigidity. Pneumo-encephalogram was undertaken to exclude any operable condition.

PREMEDICATION Nil.

ANAESTHETIC:

Nitrous oxide and oxygen (50%) were administered. Pharyngeal toilette was performed followed by orotracheal intubation. Much blood-stained froth was aspirated from the endotracheal tube. Thereafter, anaesthesia was continued with oxygen alone administered via the Ayre's T-piece. Respiration, which was irregular at the start of the investigation, took on a Cheyne-Stokes character after 20 minutes. Pneumo-encephalogram revealed no operable condition. The patient died 1½ hours after return to the ward.

AUTOPSY:

Bilateral subdural haematomata. Infra- and supratentorial subarachnoid staining. Very marked cerebral oedema.

COMMENT:

The patient obviously died of the results of her head injury. Anaesthesia is in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
184.1.60	2	No comment	< 24	Haemorrhage from inoperable carcinoma of the stomach.	No

Name: Attie Leukes Age: 55 Sex: M Race: C

Disease: Haematemesis. Carcinoma of the stomach. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had had a severe haematemesis and was extremely shocked. Pulse rate 120/minute, B.P. 120/75 mm.Hg. although his normal B.P. was 230/105 mm.Hg. Crepitations were audible at both lung bases. Blood transfusion of 3 pints had been given but bleeding had continued profusely.

PREMEDICATION:

Atropine grl/100 given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced by the inhalation of nitrous oxide and oxygen, administered via a circle absorption system with gradually added ether. When surgical anaesthesia was present, orotracheal intubation was performed. Anaesthesia was maintained by the same means, with spontaneous breathing.

Laparotomy revealed a carcinoma of the gastric cardia, which was bleeding copiously, but was quite inoperable. When the nature of the lesion was discovered, restorative therapy by way of blood transfusion was discontinued, the operation being abandoned and the abdomen closed. At the conclusion of operation, the B.P. had fallen to a level of 80 mm.Hg systolic. The patient died 7 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient died from haemorrhage as a result of an inoperable gastric carcinoma. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
185.1.60	2	No comment	> 24	Cerebral ischaemia Arterial occlusion	Yes

Name: Ali Reinink Age: 42 Sex: F Race: E.

Disease: Carotid body tumour on left carotid artery.

Operation: Excision of carotid body tumour and vein graft of carotid artery.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Except for the presence of a tumour of the neck, this patient was physically fit. B.P. 140/90 mm.Hg, haemoglobin concentration was 13 gm.%. She had a slight dorsal kyphosis.

PREMEDICATION:

Atropine gr. 1/100.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Spontaneous respiration returned after 5 minutes and anaesthesia was then maintained with nitrous oxide and oxygen, with spontaneous breathing. Intermittent supplemental doses of thiopentone were administered. Throughout the 8½ hours of operation, a total of 650 mg. thiopentone was used. The course of the anaesthetic was uneventful.

After 4½ hours dissection, the left carotid artery was clamped and a tube bypass established. The carotid body tumour andleft carotid artery were excised and a vein graft inserted. The initial operation was concluded at the end of 6 hours. At the discontinuance of anaesthetic, the patient became restless and a right-sided hemiplegia became obvious. The patient was immediately re-anaesthetised with nitrous oxide and oxygen, with ether, and left carotid angiogram was performed. This revealed a block in the vein graft which had been inserted in the carotid artery, and the graft was thus taken down and re-performed. Subsequently a second angiogram showed the graft to be patent. The second grafting operation concluded 8½ hours after the commencement of the original procedure. However, when anaesthesia was again discontinued, the patient commenced struggling and became very restless, never regaining normal consciousness. She developed right sided epileptiform fits 24 hours after the conclusion of the operation and was immediately sedated on hypothermia to a level of 90°F. However, the fits continued - occurring approximately every 30-45 minutes - and the patient died on the 7th post-operative day.

AUTOPSY:

Excision of bifurcation of carotid artery on left for carotid body tumour. Thrombosis with occlusion at the upper anastomosis of vein graft to internal carotid artery. Total excision of left external carotid artery.

COMMENT:

This patient died of cerebral ischaemia which resulted from left carotid occlusion occurring during and after the excision of a carotid body tumour. Anaesthesia is not considered contributory to the patient's demise.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
186.1.60	2	No comment	<24	Multiple injuries	Yes

Name: Annie Julies Age: 21 Sex: F Race: C

Disease: Multiple injuries with visceral haemorrhage. Operation: Laparotomy. Splenectomy. Packing of lacerated liver.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor - the patient was shocked, had a rapid, grunting respiration. Pulse rate 160/minute. B.P. 150 mm.Hg systolic. There was poor air entry on the left side of the chest. 5 pints blood had been transfused pre-operatively. Passage of a Ryle's tube revealed the presence of blood in the stomach.

PREMEDICATION:

Atropine gr. 1/100, given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg., succinylcholine 35 mg., ventilation with oxygen and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen with a trace of ether, administered via a circle absorption system by an IPPR technique. Gallamine, totalling 80 mg., was used as the relaxant.

At operation a ruptured spleen and lacerated liver were found. A splenectomy was performed and lacerations in the liver were sutured. During the operation a further 3 pints blood were transfused. The procedure lasted 90 minutes. At the conclusion, the patient's condition was much improved, although she still had a tachycardia. Aspiration via the endotracheal tube yielded no blood. After discontinuance of the anaesthetic, she recovered consciousness and had normal spontaneous respiration. It was not found necessary to give any antidote to the gallamine used, as the patient had spontaneous and adequate respiration of normal character at the conclusion of the procedure. Her condition remained much the same throughout the night. The next morning, 8 hours after operation, she died.

AUTOPSY:

Multiple injuries with severe generalised bruising of the lungs. Apical tuberculosis of the left lung. A splenectomy had been performed. Laceration of the liver, stitched. Haemorrhage around the left kidney. Slight subarachnoid haemorrhage.

COMMENT:

The actual cause of this patient's death is not clear, although doubtless associated with the original multiple injuries. It is improbable that the anaesthetic was in any way contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
187.1.60	2	No comment	< 24	Periton-itis.	No

Name: Paul de Villiers Age: 64 Sex: M Race: E

Diaease: Peritonitis (?perforation of diverticulitis). Operation: Laparotomy. Colostomy. Tracheotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund. Besides evidence of peritonitis, he had bronchitis and very gross emphysema, with marked respiratory inadequacy, and cor pulmonale. Temperature 101°F. Pulse rate 108/minute. B.P. 110 mm. Hg systolic. Slight cyanosis was present with marked congestion of the neck veins during expiration.

PREMEDICATION:

Aminophyllin 250 mg., atropine gr. 1/100, given immediately before anaesthesia.

ANAESTHETIC:

After transtracheal injection with 2 cc. 4% Xylocaine, anaesthesia was induced with cyclopropane, oxygen, administered via a closed circle absorption system. After induction of anaesthesia, oral intubation was performed, followed immediately by tracheobronchial toilette. Anaesthesia was continued with nitrous oxide and oxygen with ether vapour, with spontaneous respiration. Following the introduction of ether vapour there was a marked drop in B.P. 6 mg. methylamphetamine was administered and ether administration was discontinued. Respiration was assisted and controlled at times. On the whole, the B.P. remained around 110-120 mm.Hg systolic, but oscillated rather violently at times. When there was a drop in B.P. methylamphetamine was administered in 6 mg. doses.

The findings at laparotomy were gross faecal peritonitis from a perforated diverticulitis. A transverse colostomy was performed and an abdominal drain inserted. For closure of the peritoneum succinylcholine 25 mg., and subsdquently a further 10 mg., was administered and IPPR performed. Spontaneous respiration returned 7 minutes after the last dose of succinylcholine. The patient regained consciousness on the operating table while the dressings were being applied. Tracheotomy was performed immediately after operation. The patient remained in status quo for approximately 10 hours, whereupon there was sudden deterioration and he died.

AUTOPSY:

No autopsy.

COMMENT:

This patient died from gross respiratory inadequacy due to emphysema and cor pulmonale, together with the effects of severe toxaemia from faecal peritonitis. Anaesthesia is not considered contributory to the death, when it occurred.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
188.1.60	2	No comment	< 24	Myocardial ischaemia.	No

Name: Nicolas Basson Age: 71 Sex: M Race: E

Disease: Arteriosclerotic vascular occlusion of right leg. operation: Above knee amputation of right leg.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Very poor. The patient suffered from congestive cardiac failure and had been digitalised on admission. He had grave emphysema and the B.P. was a mere 85 mm.Hg systolic.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes before operation.

ANAESTHETIC:

On arrival in theatre the patient was given 2 doses of 6 mg. each methylamphetamine, causing the B.P. to rise to a level of 95-100 mm.Hg systolic. Following 4 minutes' pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen, with gradually added ether vapour, via a Magill circuit. Induction of anaesthesia was quiet but the patient sweated profusely. The B.P. rose to 120 mm. Hg systolic, with a pulse rate of 108/minute. Operation was then commenced.

During the procedure methylamphetamine was given in small doses, maintaining the B.P. at a level of 120 mm.Hg systolic. The patient continued to sweat profusely during operation. During the last 15 minutes of the procedure, which altogether lasted 45 minutes, anaesthesia was maintained with nitrous oxide and oxygen only. The total amount of ether used to this stage was 2 oz. While the skin was being stitched, the B.P. dropped to unrecordable levels for 5 minutes but recovered again to 80 mm.Hg systolic. Throughout the operation, the total dose of methylamphetamine used was 30 mg. Phenylephrine 0.5 mg. was administered at the conclusion of the procedure. The patient regained consciousness immediately on discontinuation of anaesthesia, while the dressings were being applied, and was returned to the ward with a B.P. of 80 mm.Hg systolic. An ECG taken at this stage showed subendocardial ischaemia. Myamine 10 mg. was injected intramuscularly in the ward and the patient appeared to improve slightly. However, 17 hours after operation, he suddenly became cold, clammy, pulseless, cyanosed, restless and died.

AUTOPSY:

No autopsy.

COMMENT:

In all probability, this death was due to arteriosclerotic myocardial ischaemia. Anaesthesia is not considered to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
189.1.60	2	No comment	< 24	?Myocardialc infarction	No

Name: Joan Alder Age: 72 Sex: F Race: E

Disease: Strangulated femoral hernia. Operation: Reduction and repair of femoral hernia.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Very poor - the patient was suffering from intestinal obstruction due to a strangulated hernia. She was in cardiac failure and there was a previous history of two attacks of myocardial infarction. Pleural effusion of the right lung base and a 2 finger hepatomegaly were present. Pre-operatively the pleural effusion was aspirated and the patient was digitalised.

PREMEDICATION:

Phenergan 50 mg., by intramuscular injection 1 hour pre-operatively.

ANAESTHETIC:

Spinal analgesia was induced with 1 cc. heavy Nupercaine to a level of 10th thoracic dermatome. A slight head-down tilt was induced and oxygen was administered throughout the operation, the patient breathing spontaneously. Immediately before the administration of the spinal analgesia, methylamphetamine 12 mg. was given intravenously and another 12 mg. 35 minutes later. At the commencement of the spinal analgesia the B.P. was 140 mm.Hg systolic, and this dropped to 120 mm.Hg systolic, where it was maintained throughout the operation, which lasted 60 minutes.

The course of the procedure was untoward, as was the anaesthetic. The patient was in good condition on return to the ward. Twelve hours later, in a manner suggestive of myocardial infarction, the patient died suddenly.

AUTOPSY:

No autopsy.

COMMENT:

This death does not seem related in any way to the anaesthetic administered. The B.P. did not drop below 120 mm.Hg systolic at any time during the operation and in the immediately post-operative period. The clinical diagnosis of myocardial infarction after operation is in keeping with the previous history. Anaesthesia is not regarded as contributory to this death when it occurred.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
190.1.60	2	No comment	< 24	Pulmonary oedema.	Yes

Name: Trooi Jonas Age: 22 Sex: F Race: C

Disease: Ruptured ectopic pregnancy. Operation: Right salpingectomy.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Just before admission to hospital, this patient had had a transfusion reaction to a unit of Group O Rhesus negative blood. After admission she was severely anaemic and 4 pints compatible blood were transfused during the 12 hours pre-operatively. Immediately before surgery the B.P. was 120 mm.Hg systolic, pulse rate 90/minute. There was also a history of haematemesis and melaena.

PREMEDICATION:

Atropine gr. 1/100 given 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen, via a Magill semi-open system, with gradually added ether, with spontaneous breathing. Oral intubation was then performed and anaesthesia maintained by the same method. After commencement of operation, 20 mg. gallamine was given and for the next 10 minutes, respiration was assisted. The patient was subsequently allowed to breathe spontaneously and ventilation was of adequate volume.

Laparotomy was performed and revealed a ruptured ectopic pregnancy. Right salpingectomy was performed. During the procedure a further 1½ pints blood were transfused. The course of anaesthesia and operation were untoward, though the B.P. fell initially to 100 mm.Hg systolic and subsequently rose to 110 mm.Hg systolic, where it stabilised. The pulse rate, which was 96/minute at the commencement of anaesthesia, rose to 120/minute on administration of gallamine, where it remained for 30 minutes, subsequently falling to 100/minute towards the end of the procedure.

The operation took 60 minutes and at the end, on discontinuance of anaesthetic, the patient recovered consciousness rapidly and had normal respiration. She died 10 hours later in the ward.

AUTOPSY:

Lungs congested and acutely oedematous with respiratory tract full of frothy fluid. Haemorrhage into inner half of uterine wall. Right fallopian tube surgically removed. Organs congested. Sutured laparotomy wound.

COMMENT:

The patient recovered satisfactorily from the general anaesthetic. The cause of death appears, from autopsy, to have been pulmonary oedema. This may have been related to over-transfusion post-operatively. An incompatible transfusion reaction was possibly also contributory. Anaesthesia does not appear to have been contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
191.1.60	1	Possibly	< 24	Hypotension Cerebral ischaemia.	Yes

Name: Barbara Malan Age: 55 Sex: F Race: E

Disease: ?Left cerebellar tumour. Operation: Posterior fossa exploratory craniotomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Good. B.P. 160/100 mm.Hg. Haemoglobin 15 gm.%. Other than for hypertension and some symptoms of a space-occupying lesion, she was normal. Abnormal gait, nystagmus, defective sensation left side face. C.S.F., EEG and Skul X-ray all normal.

PREMEDICATION:

Atropine gr. 1/100 and phenergan 50 mg., 65 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg., succinylcholine 25 mg., ventilation with oxygen, followed by oral intubation. After 4 minutes of IPPR, spontaneous breathing returned and anaesthesia was continued by inhalation of ether and air with supplemental oxygen, using an E.M.O. ether vaporizer, via a non-rebreathing system, the patient breathing spontaneously. She was now positioned in the ereet sitting position with the head acutely fixed. During the first hour of anaesthesia there was a steady, progressive fall in B.P. from the pre-operative level of 160 mm.Hg systolic to an ultimate level of 80 mm.Hg systolic. The administration of ether was accordingly reduced to a mere trace, and anaesthesia continued with nitrous oxide and oxygen using the same non-rebreathing system. After 10 minutes when the B.P. was 80 mm.Hg systolic, it commenced rising and within 15 minutes was 120 mm.Hg systolic, where it remained throughout the rest of the procedure.

Posterior fossa craniotomy revealed nothing abnormal and the operation was concluded. The total time of anaesthesia was 2 hours 45 minutes. When anaesthesia was concluded the patient failed to regain consciousness through she did show swallowing reflexes. She remained unconscious post-operatively, developing myotonic jerks later for which 6 ml. paraldehyde was given 16 hours post-operatively. She died 17 hours after operation.

AUTOPSY:

Surgical wound left side head. Bilateral flattening of cerebral convolutions and softening of mid brain and pons. Unusual amount of extradural staining associated with operation site and small amount about left vertebral artery.

COMMENT:

Progressive hypotension following induction of anaesthesia was the only notable event in the course of this anaesthetic. Nothing abnormal was found in the posterior cranial fossa at operation. At the end of the operation the patient failed to regain consciousness and she subsequently died, 17 hours post-operatively, without regaining consciousness. Autopsy showed evidence of cerebral ischaemia. The evidence strongly suggests that, with the patient in the erect sitting position, the neck being acutely flexed, complete or relative occlusion of the cerebral blood supply occurred during the period of hypotension. The level of systolic blood pressure, 80 mm.Hg, was recorded by observing the oscillations produced from a brachial cuff on an anaeroid sphygmomanometer. At low levels of blood pressure

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this method is inaccurate and the actual level may have been even lower.

Hypotension probably followed the vasodilatation of anaesthesia in a patient in an erect posture. The phenothiazine drug phenergan (promethazine) was administered as pre-anaesthetic medication. This drug is known to produce some vasomotor instability occasionally. Thiopentone, also known to cause vasomotor depression, had been given to induce anaesthesia, though the dose used was small. To what extent 'retractor anaemia' during operation was contributory is a matter of surmise.

The drop in systolic B.P. following the induction of anaesthesia was not sudden, and was already present before exploration of the posterior fossa was under way. It is strange that in the presence of cerebral ischaemia at this time, no change was apparent in the pattern of respiration. It is fair to conclude that the period of hypotension following the induction of anaesthesia, in the erect posture, probably caused the cerebral ischaemia which ultimately resulted in the patient's death. The anaesthetic and its management are therefore considered a factor significantly contributing to the patient's death.

PREVENTABILITY:

The drug phenergan administered as pre-anaesthetic medication is one of the possibly causes of the hypotensive episode following the induction of anaesthesia. Once hypotension occurred, the anaesthetist adopted an expectant policy. This seems justified to some extent in that no respiratory signs of cerebral ischaemia were apparent. Tothe extent that in this posture, in a known hypertensive, this level of systolic pressure may be considered dangerous, especially when inaccuracies in its measurement are taken into account, more active measures to elevate the blood pressure are indicated. To this extent, this death is regarded as possibly preventable - perhaps a harsh judgement in the circumstances.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
192.1.60	2	No comment	< 24	Myocardial infarction.	No

Name: Arthur Milner Age: 65 Sex: M Race: E

Disease: Aorto-iliac vascular occlusion. Operation: Aorto-ilio-femoral endarterectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Good except for generalised vascular disease. The patient had essential hypertension with a B.P. of 220/120 mm.Hg.

PREMEDICATION:

Pethidine 50 mg., phenergan 50 mg., atropine gr. 1/100 given 1 hour before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 300 mg., succinylcholine 100 mg., ventilation with oxygen, topical analgesia of the larynx, and oral intubation. IPPR with nitrous oxide and oxygen, with ether, was continued until the return of spontaneous respiration. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether, via a circle absorption system, using an IPPR technique. Gallamine was used as the relaxant throughout the operation, which lasted 7 hours 40 minutes, a total of 260 mg. being used. The course of anaesthesia was uneventful, except for two occasions when the B.P. fell - first when the aortic clamp was released and then again, a little later, when the peritoneum was being closed. On both occasions methylamphetamine 3. mg. was administered, and the B.P. restored to its former level. Blood was replaced as lost, 4 pints being transfused during the procedure, together with 20 cc. 10% calcium gluconate. At the condlusion of the operation, 4 mg. neostigmine was administered preceded by atropine gr. 1/50. Normal spontaneous respiration returned. At the end of the procedure and on discontinuance of anaesthesia, the patient rapidly regained consciousness and was returned to the ward. Ten hours post-operatively there was a sudden deterioration in his condition. An ECG taken at this time revealed myocardial infarction, and he died shortly afterwards.

AUTOPSY:

No autopsy.

COMMENT:

Death resulted from a post-operative myocardial infarction. Anaesthesia was not considered contributory at this stage.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
193.1.60	3	No comment	ORD	Cardiac arrest. Ventricular fibrillation	Yes

Name: Christina Peterson Age: 18 Sex: F Race: C

Disease: Mitral stenosis (misdiagnosis, lesion being a ventricular septal defect). Operation: Mitral valvotomy

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Reasonably satisfactory. Some degree of dyspnoea on exertion and she had been in cardiac failure but this had been controlled with digitalisation. B.P. 120/70 mm.Hg, pulse rate 74/minute.

PREMEDICATION:

Omnopon gr. 1/3, scopolomine gr. 1/150, 1½ hours pre-operatively.

ANAESTHETIC:

After 5 minutes' pre-oxygenation, anaesthesia was induced by inhalation of nitrous oxide and oxygen, via a circle absorption system, with spontaneous breathing. dTc 24 mg. was then injected and IPPR commenced as apnoea occurred. Topical analgesia of the larynx and oral intubation followed. Anaesthesia was maintained with nitrous oxide and oxygen, via the same system, with IPPR. All was well until, after 1¼ hours of anaesthesia, a clamp was applied to the left atrial appendage, immediately resulting in the onset of ventricular fibrillation. Cardiac massage and defibrillation were successful and the B.P. was immediately restored to 100 mm.Hg systolic, with a regular pulse rate. Her condition appeared good and digital exploration of the mitral valve wasperformed. No mitral lesion could be found. On removal of the surgeon's finger, the atrial clamp was replaced and ventricular fibrillation again occurred. Cardiac massage was again performed and electrical defibrillation attempted but on this occasion the ventricular fibrillation proved refractory. Adrenaline 10 cc. 1:10,000 was given intravenously in divided doses during cardiac massage. Calcium chloride 10 cc. of a 10% solution was injected into the left ventricle and eventually an extremely weak spontaneous beat was obtained. An infusion of adrenaline 1:75,000 was instituted but failed to increase the efficiency of the cardiac action. Despite all resuscitative attempts cardiac arrest occurred again and was now totally refractory. During the operation, blood was replaced as lost, 1 pint being transfused in all, which appeared more than adequate.

AUTOPSY:

Fresh sutured wounds left chest wall and left atrium. Left lung partially collapsed. Pericardium was opened. No evidence of rheumatic heart disease. Mitral valve showed no stenosis but congenital absence of posterior cusp. Patchy areas of fibrosis at apex of myocardium and inner aspect. Left and right ventricular hypertrophy. Large ventricular septal defect. Uterus contained 6 week foetus.

COMMENT:

The incidents in this case are very similar to those occurring in Case No. 178, except that here the patient in fact had a ventricular septal defect misdiagnosed as mitral stenosis. The anaesthetic management appears good. Ventricular fibrillation was triggered off by the application of a clamp on the left atrial appendage; the heart did not appear to be irritable before this. Ventricular fibrillation can occur as a result of such handling of the heart, especially if there is anoxia. The anaesthetist was at pains to ensure that the patient

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was not anoxic from any cause over which he had control. Ventricular fibrillation may also follow damage to, or occlusion of, the left coronary artery by the atrial clamp. Some change in the shunt through the ventricular septal defect occasioned by digital exploration of the mitral valve may have caused some momentary cardiac anoxia. Whatever the cause, it seems closely related to surgical manoeuvres and it does not appear that the anaesthetic administration, which was smooth and untowærd until this stage, was implicated. As death occurred while the patient was anaesthetised, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
194.1.60	3	No comment	ORD	Myocardial ischaemia. Cardiac arrest.	Yes

Name: George Thomson Age: 31 Sex: M Race: E

Disease: Aortic stenosis. Operation: Aortic valvuloplasty on cardiopulmonary bypass with hypothermia.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had severe aortic stenosis. Previous congestive cardiac failure had been controlled by digitalisation and diuretic therapy. Before anaesthesia the B.P. was 95/60 mm.Hg, and the pulse rate 60/minute and regular.

PREMEDICATION:

Seconal gr. 3, orally, 2 hours before surgery; atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx, oral intubation. Thereafter anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system using an IPPR technique. dTc was used as the relaxant, 30mg. being used in the pre-bypass phase. During cardiopulmonary bypass, anaesthesia was maintained with intermittent doses of thiopentone, totalling 100 mg., with dTc 15 mg. to maintain apnoea. Deep hypothermia, to a temperature of 14°C was induced and elective hypothermic cardiac arrest induced. The aorta was cross clamped. No coronary artery perfusion was used.

After 1 hour of bypass, rewarming was commenced. On rewarming, ventricular fibrillation ensued which proved completely refractory to all attempts at defibrillation.

AUTOPSY:

Evidence of recent surgery to the heart. Lungs slightly congested.

COMMENT:

Death was due to refractory ventricular fibrillation following prolonged myocardial ischaemia. Anaesthesia was not contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
195.1.60	2	No comment	< 24	Undetermined.	Yes

Name: Arthur Claasen Age: 3 Sex: M Race: C

Disease: Tuberculous upper dorsal spondylitis with paraplegia and paravertebral abscess.

Operation: Anterolateral decompression of the spine.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Satisfactory other than for his surgical lesion. B.P. 110/60 mm.Hg. Haemoglobin 13.5 gm.%. Poorly developed chest.

PREMEDICATION:

Pethidine 25 mg., scopolomine 0.2 mg., administered 53 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane administered via a T-piece system, the patient breathing spontaneously. After induction of anaesthesia, oral intubation was performed. Anaesthesia was then maintained by the same method. No difficulty was experienced during the operation. Blood loss was adequately replaced.

At the conclusion of the procedure, which took 2½ hours, and on discontinuance of the anaesthetic, the patient rapidly recovered consciousness and was in a satisfactory state. Eight hours after operation the child was restless and pale, and within 15 minutes he was dead.

AUTOPSY:

Collapsed tuberculous focus in lung. Slight compression of cord with inflammatory tissues. Possible cerebral oedema. No convincing cause of death was demonstrated.

COMMENT:

Whatever the cause of this child's death, it does not appear to be related in any way to the anaesthetic administered 8 hours before he died.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
196.1.60	2	No comment	< 24	Multiple injuries	Yes

Name: Fani M'tunga Age: 36 Sex: M Race: B

Disease: Multiple injuries, fractured skull, multiple fractured ribs, intra-abdominal haemorrhage with rupture of the liver, spleen pancreas, stomach.

Operation: Laparotomy. Repair of stomach, splenectomy. Suture of pancreas and liver. Tracheotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor - the patient had gross multiple injuries. Respiration 30/minute, B.P. approximately 100 mm.Hg systolic following massive blood transfusion; 6 pints blood had been transfused pre-operatively. The multiple fractured ribs caused some flail movement of the chest and paradoxical respiration. A wide-bore stomach tube was passed before operation, and a tracheotomy was performed.

PREMEDICATION:

Omnopon gr. 1/6, and atropine gr. 1/100, given just before operation.

ANAESTHETIC:

Anaesthesia was induced by inhalation of nitrous oxide and oxygen, with minimal ether, administered via the tracheostomy. IPPR was used for most of the operation. No relaxant was used. During the procedure, blood was administered via two intravenous infusion sites, in the arm and through the left jugular vein, and a further 6 pints blood were transfused with 3 gm. calcium gluconate.

At operation, a splenectomy was performed, the rent in the stomach repaired and lacerations in the liver and pancreas sutured. At the end of the procedure, which took 4 hours, the B.P. was 180 mm.Hg systolic and the pulse rate 120/minute. Respiration was 28/minute, and appeared of adequate volume. Flail movement of the chest was not very obvious and the patient maintained a good colour in spite of the fractured ribs. Following discontinuance of the anaesthetic, the patient regained consciousness to the same level as had been present pre-operatively. He died 6 hours post-operatively. During the post-operative period a further 3 pints blood had been transfused and a noradrenaline infusion instituted.

AUTOPSY:

Multiple injuries with suture of liver and pancreas, fractured ribs and 500 ml. blood in the right pleural cavity. Numerous head wounds and small subdural haematomata over both hemispheres. Evidence of recent surgery. Tracheotomy.

COMMENT:

This patient died of the results of multiple injuries. Anaesthesia did not seem in any way contributory. The respiration apparently was adequate at the conclusion of surgery; one wonders why IPPR was not instituted. The 500 ml. right haemothorax would have impaired respiratory excursion and should have been drained.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
197.1.60	2	No comment	< 24	?Cardiac failure.	No

Name: Katherine Thys Age: 88 Sex: F Race: C

Disease: Haematemesis Operation: Laparotomy. "Blind" partial gastrectomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Poor - she had had a haematemesis and, on admission, her haemoglobin was 4 gm.%. In spite of this she was in early congestive cardiac failure. She was digitalised and blood transfusion commenced. After receiving 6 pints blood, the B.P. was 160/100 mm.Hg with a pulse rate of 120/minute.

PREMEDICATION:

Atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Following 4 minutes of pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen delivered via a circle absorption system with spontaneous respiration. The patient was in the head-up position. Immediately she lost consciousness, succinylcholine 40 mg. was given and oral intubation performed. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether. An IPPR technique was instituted. Gallamine was used as the relaxant, a total dose of 100 mg. being given during the course of the operation.

At laparotomy, no active bleeding point or peptic ulcer was found. A "blind" partial gastrectomy was performed. During the procedure 1 pint of blood was transfused and a second pint commenced. Although the patient's haemoglobin was only 6 gm.% at the commencement of surgery, the existence of congestive cardiac failure led to some caution on the part of the anaesthetist with regard to blood transfusion. Throughout the operation, the B.P. remained at a level of 150 mm.Hg systolic, the pulse rate varying between 100 and 120/minute. No difficulties were experienced with the anaesthetic and at the conclusion of the operation, which lasted 120 minutes, and on discontinuance of anaesthetic, the patient regained consciousness rapidly. Neostigmine 1 mg. preceded by atropine gr. 1/100 was administered at the conclusion of operation, normal spontaneous adequate respiration returning when IPPR was terminated. She died 22 hours post-operatively.

AUTOPSY.

No autopsy.

COMMENT:

In spite of her poor pre-operative condition, this patient tolerated anaesthesia and operation well. She recovered consciousness rapidly subsequent to anaesthesia. Her death, in all probability, was due to causes related to her original disease. Anaesthesia does not appear to have been contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH.	AUTOPSY
198.1.60	3	No comment	ORD	Cardiac arrest. (Ventricular fibrillation) Constrictive pericarditis	Yes

Name: Regina Komani Age: 59m Sex: F Race: B

Disease: Constrictive pericarditis. Operation: Cardiac catheterisation. Pericardiectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted for investigation of constrictive pericarditis. During the performance of cardiac catheterisation, cardiac arrest occurred. Thoracotomy and cardiac massage were performed immediately and led to re-establishment of cardiac action with a B.P. of 55 mm.Hg systolic. The patient regained consciousness. A surgeon and a physician decided that immediate pericardiectomy offered her the only chance of survival.

PREMEDICATION:

No premedication.

ANAESTHETIC:

Oral intubation was performed while the patient was conscious and IPPR instituted with nitrous oxide and oxygen, administered via a circle absorption system. Thiopentone 50 mg. was administered during induction of anaesthesia. Respiratory control was effected by gallamine 40 mg.. Blood transfusion was instituted and 1 mg. phenylephrine was administered, repeated after 15 minutes. The B.P. initially was maintained at 90 mm.Hg systolic. While pericardiectomy was being performed, the cardiac muscle lost all its tone and cardiac arrest ensued once more. Cardiac massage and intraventricular injection of adrenaline were of no avail. When the ECG showed no further complexes, further resuscitative measures were abandoned.

AUTOPSY:

Constrictive tuberculous pericarditis with constriction of heart; small heart, death being precipitated by urgent life-saving measures. Pleural adhesions and small effusion in the left pleural cavity. Partial collapse of atrophic lungs.

COMMENT:

This death resulted from the patient's disease, constrictive pericarditis, the cardiad arrest ensuing on cardiac catheterisation and subsequent pericardiectomy. Anaesthesia was not contributory. As death occurred during anaesthesia, this case is classed for uniformity in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
199.1.60	2	No comment	< 24	Undetermined.	No

Name: Nathan Trakman Age: 80 Sex: M Race: E

Disease: Intestinal obstruction. Operation: Laparotomy. Resection and anastomosis of gangrenous small bowel.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Fair operative risk, though this may have been an over-optimistic assessment of his condition. He had been ill for 2 weeks, vomiting for 1 week, more seriously for the last 24 hours. There was gross arteriosclerosis and he had a slow auricular fibrillation. B.P. was 120/80 mm.Hg. Intravenous fluid replacement therapy had been instituted before operation and gastric drainage had been performed.

PREMEDICATION:

Morp hine gr. 1/6, atropine gr. 1/100, given 66 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced by inhalation of nitrous oxide and oxygen, via a carbon dioxide circle absorption system. As the patient was losing consciousness, he hiccoughed and brown fluid was seen in the mouth. Anaesthesia was immediately discontinued and a stomach tube passed, though the patient was only semi-conscious, coughing and biting. Fluid gushed out of the stomach tube and the stomach was emptied as well as possible before induction of anaesthesia was continued. Anaesthesia was continued with the large stomach tube in situ. Ether vapour was gradually added to the inhaled gases. When the patient was adequately anaesthetised, oral endotracheal intubation was performed. Tracheobronchial toilette produced about 2 ml. brownish mucopurulent fluid. Auscultation of the lungs at this stage revealed clear breath sounds. Gallamine 40 mg. was administered and an IPPR technique used. The remainder of the anaesthetic was uneventful.

At operation an internal hernia with incarcerated small bowel was found. This was gangrenous, was resected and an end to end anastomosis performed. The operation lasted 84 minutes. At the conclusion of the procedure, tracheobronchial toilette produced no further bronchial secretions or any inhaled gastric content; breath sounds were clear. No antidote was necessary for the gallamine and the patient resumed normal spontaneous respiration of adequate volume. Following discontinuance of the anaesthetic, the patient regained consciousness approximately 15 minutes after operation. Twelve hours post-operatively thepatient collapsed and died.

AUTOPSY:

No autopsy.

COMMENT:

The patient vomited during the induction of anaesthesia at a stage when he still had intact laryngeal and pharyngeal reflexes. The stomach was emptied at this time and induction of anaesthesia was a little stormy. Subsequent tracheobronchial toilette produced a little evidence of aspiration of stomach content in that there was some brown staining of the mucopurulent secretion. However, auscultation of the lungs at this time and later revealed clear breath sounds. The course of anaesthesia was subsequently uneventful. The patient's death 12 hours post-operatively did not appear to be related to the anaesthetic per se; it was possibly related to his cardiac disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
200.1.60	2	No comment	< 24	?Fat embolism	Yes

Name: Sarel Vosloo Age: 32 Sex: M Race: E

Disease: Multiple injuries. Comminuted fracture of left knee. Laceration of right thigh with torn femoral artery and fracture of left radius and ulna.

Operation: Excision of patella. Veingraft to femoral artery. Repair of lacerated thigh.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Though shocked, this patient was in a fair state. B.P. was 110/80 mm.Hg and the pulse rate 120/minute. Altogether 9 pints blood had been transfused pre-operatively. Gastric aspiration was commenced.

PREMEDICATION:

Atropine gr. 1/100 was given 1 hour 15 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. followed by nitrous oxide, oxygen and gradually added ether vapour, administered via a Magill semi-open system, with spontaneous breathing. Following induction of surgical anaesthesia, and topical analgesia of the larynx and trachea, oral intubation was performed. Anaesthesia was maintained with the patient breathing spontaneously, and was uneventful throughout the operation. A further 5 pints blood were transfused during the procedure with 6 gm. calcium gluconate. The operation lasted 4 hours. Following discontinuance of the anaesthetic, the patient rapidly regained consciousness and had normal, adequate respiration. All was well for the first 12 hours post-operatively but subsequently the patient became increasingly drowsy. He died 23 hours after the operation.

AUTOPSY:

Evidence of surgery to right thigh with suture of femoral artery. Fractures of left forearm and leg. Abrasions of head and right hand. No lung embolism.

COMMENT:

This death appeared clinically to have resulted from fat embolism, though this was not demonstrated at autopsy, but special histological examination of the brain and kidney was not undertaken. Whatever the cause of the patient's death, it does not appear to be directly related in any way to the anaesthetic or its management.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
201.I.60	3	No comment	< 24	Multiple injuries. Haemorrhagic shock. Inadvertent hypothermia.	Yes

Name: John Philander Age: Approx.23 Sex: M Race: C

Disease: Multiple injuries: Pelvic and abdominal.

Operation: Laparotomy. Securing of bleeding points and resection of traumatised small bowel.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had been run over by a bus. On admission to hospital he had no recordable B.P. His injuries were abdominal and pelvic together with fractured femurs. Following transfusion of 20 pints blood, the systolic B.P. was recordable as 85 mm.Hg, and the pulse rate was 60/minute. He was mentally confused and there was an overlay of alcoholic intoxication. Laparotomy was necessitated by evidence of continued internal haemorrhage.

PREMEDICATION:

Atropine gr. 1/100 given intravenously immediately before induction of anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen with gradually added ether, administered via a non-rebreathing system with a Ruben's valve. Following induction of anaesthesia, oral intubation was performed. Thereafter the B.P. fell to unrecordable levels. With a rapid transfusion of blood, it rose to 120/80 mm.Hg. Anaesthesia was maintained with nitrous oxide and oxygen with a trace of ether, administered via a non-rebreathing system. The patient was allowed to breathe spontaneously for most of the operation but an IPPR technique was used intermittently. After commencement of laparotomy the B.P. again fell to unrecordable levels, and remained there for a long period. As nitrous oxide and oxygen alone were insufficient for anaesthesia, a trace of ether was administered intermittently. There were periods of hypoventilation during which IPPR was used. During laparotomy a further 13 pints blood were transfused, with a total of 8 gm. calcium gluconate. There was a bradycardia throughout the operation, though the pulse rate increased temporarily following each injection of calcium gluconate. Laparotomy revealed a crushed pelvis with uncontrollable oozing from pelvic and upper thigh tissues, massive extraperitoneal and mesenteric haematomata, lacerated bowel of which 12 inches of small bowel were resected, lacerated right kidney which was sutured. During operation and massive blood transfusion, the patient's temperature dropped from 88°F at the outset of the procedure to 83°F at the conclusion. The operation lasted 3 hours 15 minutes. At the conclusion of the operation and on discontinuance of anaesthetic, the patient had no recordable B.P., though he had adequate spontaneous respiration. After discontinuance of anaesthetic, the patient did not fully regain consciousness but recovered to a stuperose state. He died 1 hour post-operatively.

AUTOPSY:

Multiple injuries including bilateral fractured pelvis. End to end anastomosis of ruptured ileum, 12 inches from ileocaecal valve. Lacerations of ileum 6 inches from ileocaecal valve.

COMMENT:

This patient was in a severe state of haemorrhagic traumatic shock which was never controlled. Death was inevitable. In that anaesthesia itself, by causing a vasodilation, must have reduced his powers of compensatory vasoconstriction, it could be said to be necessarily

contributory. The patient's failure to regain consciousness post-operatively is probably related to the long period of gross hypotension during operation. However, it may also be related to the marked degree of inadvertent hypothermia which was induced by the massive blood transfusion of unwarmed blood. His temperature at the conclusion of the procedure was 83°F (28.2°C). The bradycardia during anaesthesia was also probably related to this hypothermia. The induction of hypothermia in such a case is difficult to avoid; this patient received in all a total of 33 pints blood.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
202.1.60	2	No comment	< 24	Respiratory failure.	Yes

Name: Robert Hickson Age: 83 Sex: M Race: E

Disease: Oesophageal stricture Operation: Oesophagoscopy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This old patient was in a poor state. He suffered from arteriosclerosis and had a B.P. of 170/90 mm.Hg. There was gross pulmonary emphysema and chronic bronchitis. He had an oesophageal stricture and an oesophagoscopy was necessary for diagnostic purposes.

PREMEDICATION:

Pethidine 50 mg., atropine gr. 1/100, given 70 minutes pre-operatively.

ANAESTHETIC:

Following premedication, the patient was very drowsy and showed respiratory depression. Anaesthesia was induced with the sequence thiopentone 200 mg. (in divided doses), succinylcholine 40 mg., IPPR with oxygen, topical analgesia of the larynx and oral intubation. IPPR with nitrous oxide and oxygen was then instituted with the Magill semi-open circuit. Oesophagoscopy was completed within 15 minutes. No further succincholine was necessary. Following the procedure, apnoea persisted due to respiratory depression. No muscle paralysis was evident, the patient could move legs and could cough but did not breathe adequately. IPPR was continued. After 10 minutes, 30 mg. daptazole was administered followed after 5 minutes by a further 30 mg. Spontaneous respiration then recommenced and the patient was extubated and regain consciousness, but respiration remained depressed. Nikethamide 2 ml. was administered after a further 20 minutes. The patient was kept under observation for 2 hours post-operatively. At this stage he was wide awake and had normal respiration of adequate volume. On return to the ward, the patient was wide awake. Four hours post-operatively the patient was able to sit up in bed and eat supper. Six hours post-operatively he became restless. In spite of instructions from the anaesthetist to the contrary, due to an error, a nurse administered pethidine 75 mg. which had previously been ordered by the house surgeon. The patient thereupon fell into a deep sleep. Three hours later he was seen to be deeply asleep. Four and 5 hours later, he was still deeply asleep (13 hours post-operatively). After another 2 hours (14 hours post-operatively) the patient was found dead.

AUTOPSY:

Senile keratosis. Papillomatous wart in abdominal wall. Infected epiglottis. Purulent bronchopneumonia and pulmonary oedema (yellow mucopurulent material blocking bronchioles). Coronary arteriosclerosis, atheroma of aorta. Perilobular fibrosis of liver. Slight enteritis. Interstitial hyperaemia of kidney. Glomerular hyalinosis of kidney.

COMMENT:

This patient manifested marked sensitivity to the respiratory depressant drugs pethidine and thiopentone. Following recovery from anaesthesia he was given 75 mg. pethidine in error. His death ensued, probably from respiratory depression, in the deep sleep that followed. The anaesthetic he received did not directly contribute to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
203.1.60	2	No comment	< 24	Cerebral abscess.	Yes

Name: Martin Bretman Age: 28 Sex: M Race: C

Disease: Duodenal fistula Operation: Laparotomy. Closure of duodenal fistula.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This patient had a duodenal fistula which had resulted from perforation of a duodenal ulcer. His B.P. was 115/60 mm.Hg and the pulse rate was 120/minute. He was grossly emaciated but pre-operatively had normal serum electrolyte values. A blood transfusion had been given before operation, increasing the haemoglobin concentration from 12 to 13.5 gm.%½

PREMEDICATION:

Pethidine 25 mg., phenergan 25 mg., atropine gr. 1/100 were administered 70 minutes before operation.

ANAESTHETIC:

Before induction of anaesthesia, the B.P. was 100 mm.Hg systolic. Anaesthesia was induced with thiopentone 250 mg. followed by gallamine 80 mg., IPPR with nitrous oxide and oxygen for 2 minutes, topical analgesia of the larynx, and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen, administered via a carbon dioxide circle absorption system, using minimal ether. An IPPR technique was used. No further gallamine was administered.

The duodenal fistula was closed, the operation lasting 65 minutes. Following conclusion of the procedure and discontinuance of the anaesthetic, the patient regained consciousness rapidly and had normal spontaneous respiration. No antidote was considered necessary for the relaxant administered. Seven hours post-operatively the patient commenced having Jacksonian type epileptic fits, and was sedated, but he died 4 hours later.

AUTOPSY:

The only abnormal finding at autopsy was a cerebral abscess, which had been undiagnosed pre-operatively.

COMMENT:

This death is not considered in any way to have been due to the anaesthesia. It would appear to have resulted from an undiagnosed cerebral abscess, which subsequently cause Jacksonian epilepsy.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
204.1.60	3	No comment	< 24	Pontine and inter-intraventricular cerebral haemorrhages.	Yes

Name: Leah Shneier Age: 56 Sex: F Race: E

Disease: Aortic aneurysm, non-functioning left kidney. Hypertension.

Operation: Laparotomy. Aorto-iliac endarterectomy. Left nephrectomy. Aortic aneurysmorrhaphy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had suffered from angina pectoris for some years, occasionally at rest. ECG showed left ventricular hypertrophy. She had hypertension; the B.P. was 180/110 mm.Hg. There was evidence of a non-functioning left kidney. She had marked peripheral vascular disease and 1 year previously had had a left lumbar sympathectomy. She now had an aortic aneurysm.

PREMEDICATION:

Omnopon gr. 1/6, atropine gr. 1/100 were given 75 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg., succinylcholine 30 mg., ventilation with oxygen and oral intubation. Subsequently anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Gallamine was used as the relaxant, a total dose of 140 mg. being used throughout the operation. Between 20 and 30 minutes after induction of anaesthesia, coincidental with exploration of the abdomen and preliminary freeing of the left kidney, prior to removal, an episode of severe hypertension occurred, the B.P. rising rapidly to a level of 300 mm.Hg systolic, the pulse rate rising to 150/minute. The cause of this hypertension, which gradually subsided, was not established. Pethidine (2 doses of 20 mg.) was given intravenously and over the next 30 minutes the B.P. fell to a level of 150 mm.Hg systolic, the pulse rate slowing to 120/minute. The patient did not appear to be anoxic at this stage, the soda lime was fresh, and the volume of ventilation appeared adequate. A further 15 minutes after this episode, when the aorta was cross clamped following heparinisation, the B.P. again rose to a peak of 260 mm.Hg systolic, subsiding after 10 minutes to 180 mm.Hg systolic, where it remained for 30 minutes, subsequently falling to 160 mm.Hg systolic. After removal of the aortic clamp, 45 minutes after initial clamping, the B.P. - after an initial short drop to 150 mm.Hg systolic - rose once more to 180 mm.Hg. At the conclusion of the procedure, which lasted 2½ hours, and on discontinuance of anaesthetic, the patient appeared partially curarised. She initially responded to neostigmine 3.5 mg. preceded by atropine gr. 1/75. However, "tracheal tug" persisted through volume of ventilation appeared adequate, and the patient failed to recover consciousness. Four hours post-operatively one pupil was observed to dilate and a lumbar puncture at this time revealed a subarachnoid haemorrhage. The patient died 11 hours after operation.

AUTOPSY:

Haemorrhages into brain and pons as a result of arteriosclerosis of cerebral arteries with calcification. Heart was enlarged with a greatly thickened left ventricular wall, as a result of hypertension. The one remaining kidney was slightly enlarged with grannular surface, capsule adherent.

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COMMENT:

Autopsy clearly reveals the cause of this patient's death to have been cerebral haemorrhage. This occurred in all probability as a result of one of the occasions during surgery when the blood pressure peaked viciously upwards. The first time this occurred was when the operation was commenced. To what extent this was due to surgical stimulus in light (perhaps too light) anaesthesia, and to what extent it could have been prevented by deeper anaesthesia is difficult to evaluate. Certainly this response is extreme. The anaesthetist did take action in an attempt to increase the analgesia by injection of pethidine and the blood pressure dropped again. The second occasion on which the blood pressure peaked was when the aorta was clamped; on this occasion the patient was heparinised. The response was brisk but the blood pressure soon returned to its resting pre-operative level of 180 mm.Hg systolic. Whether in fact the anaesthetist should have taken measures to avoid the rise in blood pressure, which could have been anticipated, is difficult to say. A rise usually occurs when the aorta is clamped, is usually not excessive and does no harm. On the other hand, blood pressure occasionally drops when the aorta is clamped. An expectant attitude can be justified. However, on whichever of these occasions the cerebral haemorrhage occurred, its effects would have been worsened by the subsequent heparinisation of the patient prior to clamping of the aorta.

Because of doubts as to the responsibility, anaesthetic or surgical, for the cerebral haemorrhage, this death is classified as due to the patient's existing disease and surgery, but with anaesthesia necessarily contributory. One may infer from this case that before the aorta is clamped in patients with hypertension, the prior administration of some vasodilator drug, such as Halothane, may be indicated to prevent too high a rise in the systolic blood pressure.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
205.1.60	3	No comment	> 24	Carotid occlusion. Cerebral ischaemia.	Yes

Name: Elizabeth Maile Age: 58 Sex: F Race: E

Disease: Left carotid thrombosis. Right carotid stenosis. Operation: Right carotid endarterectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor operative risk. Four months previously she had suffered thrombosis of the left carotid artery, resulting in a right-sided hemiplegia with aphasia. The right carotid artery was stenosed and weakness in the left hand now indicated a worsening circulatory insufficiency. It was decided to perform a right carotid endarterectomy under general anaesthesia with hypothermia. B.P. was 140 mm.Hg systolic the temperature was 100°F and the pulse rate 96/minute. She was conscious, aphasic and hemiplegic.

PREMEDICATION:

Phenergan 50 mg, atropine gr. 1/100 given 100 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg. (mixed with 6 mg. methylamphetamine in order to maintain a systolic blood pressure), succinylcholine 40 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was continued with nitrous oxide and oxygen with gradually added ether, administered via a circle absorption system with spontaneous breathing. Surface cooling was induced with a cooling blanket set at 40°F. The ECG was monitored throughout the cooling and operation, but EEG monitoring was not possible. The patient's temperature was 100°F. The rate of cooling was extremely slow. In spite of the use of ether, muscle relaxant, IPPR and ultimately chlorpromazine, it took 3½ hours to cool the patient to 91°F. During cooling the patient's B.P. dropped from 140 mm.Hg to 100 mm.Hg systolic. When the right carotid artery was exposed, no pulsation was visible beyond the region of stenosis. Following the raising of the patient's B.P. to 120 mm.Hg systolic with 0.5 mg. phenylephrine, small pulsations were visible distal to the stenosis. Endarterectomy was now performed, the artery being occluded for 10 minutes. (The planned use of a femoro-carotid bypass was abandoned because of technical difficulties). At the conclusion of the operation and on rewarming of the patient, to 94°F, respiration recommenced but the patient failed to regain consciousness. The level of consciousness lightened to a level of response to stimuli with movement but unconsciousness with conjugate deviation of the eyes persisted. Urovert 70 mg. was administered and caused a marked diuresis, but no change in the level of consciousness occurred. 36 hours post-operatively, while preparations for tracheotomy were being made, the patient died.

AUTOPSY:

Gross arteriosclerosis of cerebral arteries, carotid, vertebral and coronary arteries. Areas of softening in the brain and pons. Death due to arteriosclerosis of the cerebral arteries with pontine haemorrhage.

COMMENT:

Death was due to irreversible brain damage following prolonged cerebral ischaemia.

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During endarterectomy, the right carotid artery was occluded for a period of greater length (10 minutes) than considered safe at the temperature prevailing (91°F). As the patient's B.P. dropped during the surface cooling and it is possible that some circulatory insufficiency occurred before surgical occlusion of the right carotid artery, the anaesthetic is regarded as necessarily and unavoidably contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
206.1.60	3	No comment	ORD	Air embolism. Cardiac arrest.	Yes

Name: Sina van Wyk Age: 36 Sex: F Race: C

Disease: Sterility. Operation: Tubal insufflation; uterine dilatation and curettage.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

The patient was in excellent health. Her sole complaint was sterility.

PREMEDICATION:

Pethidine 50 mg., phenergan 50 mg., atropine gr. 1/100 given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 250 mg. and maintained with nitrous oxide and oxygen, administered via a Magill semi-open system with spontaneous breathing. During the operation a further 250 mg. thiopentone was given in divided doses. An apparently uneventful diagnostic tubal insufflation was done, followed by cervical dilatation and uterine curettage. When anaesthetic was discontinued, the patient's legs were lowered preparatory to transferring her to a trolley. A sudden gasping apnoea occurred withloss of consciousness. Cardiac arrest was diagnosed. Oral intubation was performed and artificial ventilation with oxygen instituted. The patient was returned to the operating table and a thoracotomy performed, cardiac massage being instituted. At this stage 50 mg. methylamphetamine was administered intravenously. The surgeon performing cardiac massage palpated gas in the right ventricle. Cardiac massage was of no avail.

AUTOPSY:

Gas escaped from the right ventricle on underwater dissection. Right ventricle contained approximately 15-18 ml. gas, as far as could be estimated. 5th rib was resected. Pericardium was torn. Left lung was partially collapsed. Uterus contained fibroids. Right fallopian tube and one fibroid contained subperitoneal gas.

COMMENT:

The patient died after cardiac arrest due to a gas embolism following a tubal insufflation. The gas which had entered the blood-stream during insufflation must have remained in the pelvic veins until the legs were lowered, preparatory to the patient's transfer from the operating table to the trolley, whereupon the gas shifted and went to the heart, resulting in cardiac arrest. That this occurrence should follow the use of carbon dioxide is strange. Anaesthesia is not considered contributory to this death. As cardiac arrest occurred immediately on conclusion of anaesthesia, this case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
207.1.60	2	No comment	>24	Haemorrhage. Prolonged cerebral ischaemia. Aortic occlusion.	Yes

Name: Wilson Dyani. Age: 59 Sex: M Race: B

Disease: Rupture of aortic carotid graft. Operation: Thoracotomy. Suture of aortic arch. Removal of aortic carotid graft.

Anaesthetic risk: P 4, emergency.

PRE-OPERATIVE STATE:

3 weeks previously this patient had had a very large right carotid aneurysm excised and a prosthetic teflon graft inserted, from the aortic arch to the remnant of the right carotid artery. The operation was performed under profound hypothermia and cardiopulmonary bypass, and was successful but subsequently wound sepsis occurred, the graft thrombosed and finally the anastomosis to the aortic arch commenced breaking down, leading to gross haemorrhage. This necessitated the present operation. There was gross emphysema and chronic bronchitis. The patient was severely shocked and the B.P. was 90 mm.Hg systolic, pulse rate being 96/minute. Two hours pre-operatively he had had a full supper.

PREMEDICATION:

Atropine gr. 1/100 administered immediately before operation.

ANAESTHETIC:

While anaesthesia was being induced, with cyclopropane and oxygen, the patient vomited profusely. Thus straining caused the trickle of blood from the previous wound sinus to enlarge into a gushing torrent from the aortic arch. Thiopentone 100 mg. was given immediately, the patient was intubated, succinylcholine 50 mg. given and IPPR instituted with nitrous oxide and oxygen via a carbon dioxide circle absorption system. While this was being done a large pad was held tightly down on the aortic wound tract by an assistant. No food was inhaled. Intermittent doses of gallamine, totalling 120 mg. during the operation, were administered. Right thoracotomy was performed and great trouble was experienced isolating the ruptured aortic arch because of dense adhesions and gross haemorrhage. This was finally achieved. To facilitate suture of the nectoric arch of the aorta, the aorta was clamped for prolonged periods. During most of the procedure the B.P. was maintained at a level of between 90 and 110 mm.Hg systolic, but for 1 hour no B.P. or pulse could be recorded. During this stage irreparable cerebral ischaemic damage must have ensued. Finally, after a prolonged struggle, the septic carotid-aortic graft was removed and the necrotic hole in the aortic arch wall sutured. During the operation, 12 pints blood were transfused with 6 gm. calcium gluconate. The thoracotomy was closed and spontaneous respiration allowed to recur. Neostigmine 1 mg. preceded by atropine gr. 1/100 was given, to ensure reversal of curarisation. The patient failed to regain consciousness and died 36 hours after the operation.

AUTOPSY:

Oedema of scalp. Absence of innominate artery. Narrowing of superior vena cava by ligature. Thrombosis of right common carotid artery. Cardiomegaly. Old apical tuberculosis. Right pleural adhesions. Bruising of right lung and tissues in neck.

COMMENT:

Severe and irreparable ischaemic cerebral damage was incurred during the period of operation when (1) no blood pressure or pulse could be recorded for 1 hour, and (2) during prolonged periods of aortic clamping. Anaesthesia is not considered contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
1.2.63	2	No comment	< 24	Myocardial ischaemia	No

Name: Sidney Wood Age: 72 Sex: M Race: E

Disease: Intestinal obstruction with left ureteric obstruction. Operation: Laparotomy. Resection of sigmoid colon

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Extremely poor condition. The patient had suffered a myocardial infarct 14 years previously and had an extremely poor effort tolerance. Gross emphysema and chronic bronchitis were present. Secondary carcinomatous deposits in the liver had caused a hepatomegaly. Intravenous pyelogram revealed a left hydronephrosis. Intensive physiotherapy had been undertaken pre-operatively.

PREMEDICATION:

Morphine 10 mg., atropine 0.65 mg., given 1 hour pre-operatively with satisfactory results.

ANAESTHETIC.

Anaesthesia was induced with thiopentone 150 mg., gallamine 120 mg., IPPR with oxygen, topical analgesia of the larynx and trachea followed, after 2 minutes of IPPR with oxygen, by endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen via a circle absorption system by an IPPR technique. Further doses of gallamine were administered as indicated, the total dosage for the whole operation being 200 mg. gallamine. The last dose of 40 mg. gallamine was administered 60 minutes before the end of the operation.

The operation lasted 2 hours 39 minutes and an advanced carcinoma of the sigmoid was discovered with large secondary deposits in the liver. A total sigmoidectomy was performed. Blood was replaced as lost during the procedure. The course of anaesthesia was untoward. At the conclusion of the operation, curarization was easily and adequately reversed by 1 mg. neostigmine administered after 0.6 mg. atropine. At the conclusion of anaesthetic, the patient recovered consciousness within minutes, the respiration being normal and of adequate volume. Following his return to the ward, his condition appeared satisfactory for a little over 1 hour post-operatively. He developed a sudden cardiovascular collapse 1½ hours after the operation and died. Clinically, death appeared to have resulted from myocardial infarction. The only drug administered in the immediate post-operative period was Reverin, which was given intravenously 1 hour post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This patient had emphysema and chronic bronchitis pre-operatively. As an IPPR relaxant technique was used, and 200 mg. gallamine had been administered during the operation, one must be sure that post-operative respiratory inadequacy played no part in this death. From the clinical account, the course of anaesthesia was uneventful. 1 mg. neostigmine produced adequate reversal of what residual curarisation remained at the end of the procedure. The patient recovered consciousness rapidly post-operatively, and in the ward thereafter respiration appeared adequate and he could move normally. His sudden death was not preceded by respiratory inadequacy and appeared clinically to have resulted from myocardial ischaemia following an episode of hypotension. A history of previous myocardial infarction adds strength

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to this diagnosis. Intravenous Reverin, known to produce hypotension, was administered shortly before death. In the circumstances, anaesthesia was not apparently contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
2.2.63	3	No comment	ORD	Left carotid/ vertebral thrombosis	Yes

name: David Cloete Age: 28 Sex: M Race: C

Disease: Multiple injuries. Brachial plexus injury and injury to left shoulder, fractured ribs, fractured scapula. Operation: No operation performed.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

Fair. Besides a fractured scapular, he had fractures of the 1st and 2nd ribs on the left, but there was no flail injury. His respiration was adequate although the fractured ribs caused some pain and distress. On arrival in theatre, he was not shocked. Systolic blood pressure was 150 mm.Hg, pulse rate being 100/minute. Haemoglobin concentration was 11 gm.%. There was no cyanosis.

PREMEDICATION:

Pethilorfan 100 mg., atropine 0.6 mg., administered 30 minutes before operation.

ANAESTHETIC:

Because of the pain in his shoulder and from the fractured ribs, the patient was brought to theatre propper up on a trolley at an angle of 20° with pillows. Anaesthesia was induced in this position, using thiopentone 300 mg., succinylcholine 50 mg., IPPR with oxygen for 1 minute, topical analgesia of the larynx, pharynx and trachea, and endotracheal intubation. IPPR was continued with nitrous oxide and oxygen with gradually added ether, via a Magill circuit. IPPR was continued for 5 minutes, when spontaneous respiration was resumed. The patient was moved to the operating table. While he was positioned, marked pulsations were noted on the left side of the neck. Within 5 minutes the respiration, which until this stage had been normal, became gasping. Having checked the endotracheal tube for correct placement and the thorax for air entry, and having excluded the possibility of a tension pneumothorax, the anaesthetist immediately commenced IPPR. A marked bradycardia was now apparent and was followed within minutes by a cardiac arrest. While IPPR with oxygen was continued, the chest was thumped three times without effect. An immediate thoracotomy was performed and cardiac massage commenced. The heart was in a state of ventricular fibrillation. Electrical defibrillation was attempted but the heart re-fibrillated. All attempts at resuscitation failed.

AUTOPSY:

Marked contusion of the left subclavian artery with a thrombosis extending up the left common carotid artery and left vertebral artery. Fractures of the first two ribs on the left side and the scapular. There was no tension pneumothorax.

COMMENT:

As revealed by autopsy, this death was caused by acute cerebral ischaemia from vascular occlusion of the left carotid and vertebral arteries. There was, in allprobability, thrombus present in the contused left subclavian artery pre-operatively. The retrograde extension of this ti involved the carotid and vertebral arteries appears to have been precipitated in some way by the positioning of the patient on the operating table. However, this is surmise. The anaesthetic per se and the anaesthetic teqhnique do not appear to have been significantly contributory. Because the patient died while anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
3.2.63	2	No comment	< 24	Liver failure.	Yes

Name: Cecil Salzman Age: 54 Sex: M Race: E

Disease: Portal hypertension, oesophageal varices. Operation: Porto-caval anastomosis.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Extremely poor condition. There was gross cirrhosis of the liver with portal hypertension and splenomegaly. He had oesophageal varices from which he had suffered haematemesis for 12 hours pre-operatively. 8 pints blood were transfused pre-operatively.

PREMEDICATION:

Atropine 0.65 mg. given 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen followed immediately on loss of consciousness by succinylcholine 50 mg. and endotracheal intubation. No vomiting or regurgitation of blood occurred during induction of anaesthesia. Anaesthesia was maintained throughout the procedure with the nitrous oxide and oxygen, administered via a carbon dioxide absorption system using an IPPR technique. Gallamine, in divided doses, totalling 200 mg. throughout the procedure, was used as the relaxant.

The operation took 4 hours to complete. During this time blood loss was replaced, a total of 6 pints blood being transfused. The course of anaesthesia was uneventful. At the conclusion of the operation residual curarisation was adequately reversed with 2 mg. neostigmine preceded by atropine 1.2 mg. The patient regained consciousness rapidly. Post-operatively he developed a rapidly progressive increasing jaundice. He became comatose from what appeared clinically to be liver failure, about 14 hours post-operatively, and died 24 hours after the operation.

AUTOPSY:

Severe cirrhosis of the liver, ruptured oesophageal varices. Established porto-caval shunt. Cyst of the liver. Acute peptic ulceration. Vesicular pulmonary emphysema and early bronchial pneumonia.

COMMENT:

Thispatient died of liver failure as a result both of his disease and of the operation performed. The anaesthetic is not considered contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
4.2.63	2	No comment	< 24	Cerebral abscess.	Yes

Name: Walter Deacon Age: 38 Sex: M Race: E

Disease: Intracranial tumour Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was unconscious on presentation in the theatre. He had been markedly restless on admission; paraldehyde had been administered. All the signs of an intracranial space occupying lesion, with marked neck stiffness, were present. Both pupils were widely dilated and fixed, the right being larger. In addition, he had Fallot's tetralogy. Pulse rate was 130/minute, B.P. 140 mm.Hg. systolic.

PREMEDICATION:

Atropine 0.65 mg., given 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. followed by succinylcholine 50 mg., ventilation with oxygen for 1 minute, topical analgesia of the larynx and trachea, and endotracheal intubation. IPPR with nitrous oxide, oxygen and minimal ether was continued via a carbon dioxide circle absorption system, until the return of spontaneous respiration. Subsequently the patient was allowed to breathe spontaneously. Carotid angiography was performed followed by burrhole craniotomy which revealed the presence of a cerebral abscess which was drained. The operation lasted 90 minutes. The course of anaesthesia was uneventful. After return to the ward, the patient did not regain consciousness. There was no respiratory inadequacy. The patient died 12 hours post-operatively without regaining consciousness.

AUTOPSY:

Abscess of right cerebral hemisphere. Acute meningitis. Haemorrhage around carotid artery following angiography. Tetralogy of Fallot.

COMMENT:

Death was due to the patient's pre-existing disease. Anaesthesia is not considered contributory. However, one must criticise the use of thiopentone sodium here, even though the dose was small, as the patient was comatose and it appears to have been unnecessary.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
5.2.63	2	No comment	< 24	Intra-cerebral haematoma	No

Name: Paul Afrikaner Age: 45 Sex: M Race: C

Disease: Intracerebral haematoma. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: A 3.

PRE-OPERATIVE STATE:

Stuperose pre-operatively with a right hemiplegia and signs of an intracranial space occupying lesion. B.P. 140/80 mm.Hg. Pulse rate 56/minute. The pre-operative neurological diagnosis was that of subdural haematoma.

PREMEDICATION:

Atropine 0.5 mg, was given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and gradually added ether, the patient breathing spontaneously. Oral endotracheal intubation was performed following topical analgesia of the larynx. During carotid angiography, the anaesthesia was maintained with nitrous oxide, oxygen and ether, delivered via a Magill circuit, with spontaneous breathing. During the burrhole craniotomy, at which an intracerebral haematoma was discovered, anaesthesia was maintained with nitrous oxide, oxygen and Halothane, administered via a Magill circuit with spontaneous breathing.

The operation took 2 hours. During this time the patient's physical state remained static and the course of anaesthesia was untoward. At the conclusion of the procedure, on discontinuance of the anaesthetic, the patient did not recover consciousness. There was no respiratory inadequacy. Six hours post-operatively, a tracheostomy was performed in the ward because of his continued coma. He died 8 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

Anaesthesia is not considered to have been contributory to this patient's death, which appears to have resulted from an intra-cerebral haematoma and neuronal injury.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
6.2.63	3	No comment	ORD	Cardiac arrest. Surgical cardiac trauma.	Yes

Name: James Bently. Age: 60 Sex: M Race: E

Disease: Carcinoma of the oesophagus. Operation: Oesophagectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Besides carcinoma of the oesophagus, thispatient had severe alcoholic cirrhosis of the liver with palmar erythema, ascites, spider nevae and atrophic testes. He had a mild emphysema and dyspnoea on exertion. He had been admitted to hospital 6 years previously with nutritional heart failure on an alcoholic cirrhotic basis,and had subsequently continued to show left bundle branch block on ECG. At this time his pulse rate was 100/minute and systolic B.P. 160 mm.Hg.

PREMEDICATION:

Pethidine 50 mg., atropine 0.65 mg., given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 275 mg., succinylcholine 30 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. After the return of spontaneous respiration, anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorptionsystem by an IPPR technique. Halothane was added to this mixture in concentrations varying from time to time but averaging 0.5%. dTc was used as the relaxant and was administered intermittently, as needed, a total dose of 50 mg. being used throughout the operation which lasted 6½ hours, the last dose being given approximately 1 hour before the end of the procedure. The first part of the operation, mobilization of the stomach through a midline abdominal incision, passed off uneventfully. Abdominal gastric mobilization took 2 hours. The patient was turned into the right lateral position and the operation continued through a right thoracotomy. After turning the patient, his neck and head became cyanosed. There was no evidence of cyanosis in other parts of the body, and this presumably was due to compression of the venous drainage of the head and neck at the level of the thoracic inlet. At thoracotomy a large carcinoma of the oesophagus was found infiltrating the right lower lobe of the lung and the pericardium. Right lower lobectomy was performed initially, during which there was quite marked blood loss, replaced by transfusion. Subsequently, while attempting to cut the carcinoma from the pericardium, a hole was made in the left atrium, causing fairly severe blood loss. During attempts to control this haemorrhage, the heart was handled and manipulated quite severely. At this stage, the B.P. which had been steadily maintained previously (the operation had been in progress for 5 hours) commenced a steady and progressive fall. This fall continued in spite of subsequent attempts at resuscitation. Blood was replaced as lost and calcium gluconate was administered at the rate of 1 gm. for every 2 pints transfused. When this failed to arrest the progressive fall in B.P., small doses of phenylephrine were administered intravenously without effect. In view of possible metabolic acidosis which was now developing, sodium bicarbonate was administered in a dose of 2 m.Eq./kg. body weight. Digitalisation was also commenced. The mixture of anaesthetic gases was changed to nitrous oxide and oxygen alone, 50%. Hydrocortisone 100 mg. was administered intravenously and ultimately a noradrenaline drip infusion of 4 microgm./cc was commenced. However, none of these measures appeared to have the slightest effect on the very steady and progressive cardiac failure that ensued, and after 2 hours of fall, the B.P. was 60 mm.Hg systolic. At this stage, 6½

hours after commencement of the operation, cardiac arrest ensued in complete asystole. Until this stage a total of 7 pints blood had been transfused. Cardiac massage was immediately instituted and this, together with intraventricular injection of adrenaline 1:10,000 8 cc. failed to resuscitate the patient.

AUTOPSY:

Left paramedian incision resutured. Right thoracotomy incision from scapula extending around to sternum. 7th rib removed. Two diverticuli at the lower third of the oesophagus, 10 cm. apart. A large carcinoma extending entirely the length between the two diverticuli. Heart was enlarged, weight 580 gm. Left ventricle was 2 cm. thick. Right ventricle was 0.5 cm. thick. There was a wound approximately 2 cm. long in the left auricle, which had been sutured. There was a patent atrial septal defect. The right lower lobe of the lung had been removed. The stomach had been mobilised.

COMMENT:

Surgically this was a poorly selected case. On thoracotomy, following mobilisation of the stomach, the carcinoma (which had infiltrated the lung and pericardium) should have been adjudged irresectable. However, resection was attempted. This necessitated a right lower lobe lobectomy and, during resection of the growth from the pericardium, a hole was made in the left atrium. This resulted in grave blood loss and much handling and manipulation of the heart. This, in the presence of what seems to have been an undiagnosed atrial septal defect, resulted in progressive cardiac failure, ending ultimately in asystole. Doubtless, inadvertent hypothermia following prolonged exposure and massive transfusion also played a significant part in the ultimate progressive cardiac failure. Though possible inadequacy of ventilation was a contributory cause, the conditions imposed by the surgical procedure rendered the task of the anaesthetist to maintain life all but impossible. On these grounds, this case is classified in group 3 - an all but inevitable death with anaesthesia necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
7.2.63	1	Possibly	< 24	Prolonged hypotension. Myocardial failure.	No

Name: F. Buckingham Age: 74 Sex: F Race: E

Disease: ?Appendix abscess. Operation: Laparotomy and drainage.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor. The patient had been admitted to hospital 14 days previously with a hectic fever varying between 100 and 102°F, with a diagnosis of chronic appendix abscess. Laparotomy was performed 4 days after admission and the patient did not do well after this. The hectic fever persisted, she remained toxic and a mass reappeared in the iliac fossa. It was decided to perform another laparotomy. At this time temperature was 101.2°F and, suffering as she did from some aortic stenosis, a mild degree of cardiac failure was also suspected. There were bilateral basal crepitations over both lung bases. B.P. 130/80 mm.Hg, pulse rate immediately before operation was 95/minute. She was not digitalised.

PREMEDICATION:

Pethilorfan 50 mg., atropine 0.65 mg., administered 1¼ hours before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg., succinylcholine 50 mg. and ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen delivered by an IPPR technique via a circle absorption system. Minimal ether vapour was added to the anaesthetic mixture. Gallamine was used for relaxation, an initial dose of 60 mg. being followed by subsequent doses of 40 mg. and 20 mg., to a dose totalling 160 mg. throughout the operation which lasted 2 hours 10 minutes. Immediately following induction of anaesthesia, the patient's condition commenced deteriorating. The B.P. fell steadily and progressively to 100 mm.Hg systolic at the end of 45 minutes and to 60 mm.Hg systolic after a further 15 minutes. During this time the pulse rate rose progressively from 96/minute to 150/minute.

At laparotomy dense adhesions were found but no abscess. Blood loss was recorded at this time to be 600 ml. Having initially accepted an intravenous infusion set with a small bore needle, established pre-operatively (suitable for aqueous solutions only) the anaesthetist found difficulty in replacing blood at the rate lost. As all the peripheral veins were now collapsed, the anaesthetist could not establish a venepuncture with a large bore needle. When the B.P. had fallen to 60 mm.Hg systolic, vasopressor drug phenylephrine was resorted to and with the help of intermittent 0.5 mg. doses of this, the B.P. was maintained at a level varying from 80 - 100 mm.Hg systolic for the remainder of the procedure. Throughout this time, the pulse rate persisted at 150/minute. By the end of operation, 600 ml. blood had been transfused. At the end of the procedure residual curarisation was adequately reversed with neostigmine 1.5 mg. preceded by atropine 0.75 mg. and respiration was adequate. Consciousness was regained rapidly but the patient's circulatory state continued to be unsatisfactory. Following the administration of 100 mg. hydrocortisone, an intravenous infusion of a vasopressor drug Wyamine was necessary to maintain the B.P. at a level of between 80 and 100 mm.Hg systolic. Her condition continued to deteriorate and the patient died from myocardial failure 15 hours post-operatively.

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AUTOPSY:

No autopsy.

COMMENT:

The circulatory condition of this very ill and toxic patient commenced deteriorating immediately she was anaesthetised. In such a case, the use of thiopentone sodium for the induction of anaesthesia must be seriously questioned. Pre-operative hypovolaemia may have been a factor, as may be the circulatory changes produced by the IPPR technique. This latter aspect is difficult to judge clinically in retrospect. An important fault in the anaesthetic technique was the acceptance of an intravenous infusion set-up with a bore of needle too small to permit rapid transfusion of blood. The probable inadequacy of blood replacement while it was being lost is borne out by the collapsed state of the peripheral veins, which resulted in the anaesthetist's later failure to establishe a more adequate venepuncture. The more rapid fall in B.P. once the level of the systolic pressure had reached 100 mm.Hg was probably due to the falling off at this level of coronary perfusion. This patient had aortic stenosis. Cardiac output was reduced because of a deficiency in cardiac pump mechanism. At the same time, the very rapid pulse rate would have further hampered the cardiac pump action by the marked reduction in ventricular filling time. In view of the pre-operative diagnosis of myocardial failure, one wonders why this patient was not digitalised.

Once gravely disturbed, the patient's circulatory homeostasis was never restored. Though the poor physical status of the patient pre-operatively must have been a major contributory factor to her subsequent death, factors concerned with the anaesthetic administration - which appear to have precipitated the progressive circulatory failure - cannot be ignored. Anaesthesia must be considered significantly contributory to this death.

PREVENTABILITY:

Though, because of the patient's physical status, the prognosis for ultimate recovery may have been slight, correctible faults are identifiable in the anaesthetic technique used in this case. These were:-

(1) the use of thiopentone in such an ill and toxic patient;

(2) failure to establish an adequate intravenous infusion set-up;

(3) failure to digitalise a patient in whom cardiac failure had been diagnosed.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
9.2.63	2	No comment	< 24	Pneumonia. Cardiac failure.	No

Name: Van de Walt Age: 64 Sex: M Race: E

Disease: Acute vascular occlusion of the left leg.

Operation: Left iliac and femoral embolectomy. Post-operative tracheotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Virtually moribund. The patient was in gross cardiac failure from cor pulmonale, which had resulted from severe chronic bronchitis and emphysema. At the same time he had bronchopneumonia. He was severe arteriosclerotic and had recently suffered acute vascular occlusion of the left leg. He was extremely toxic. The patient was digitalised.

PREMEDICATION:

Atropine 0.65 mg.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen with the patient breathing spontaneously on a carbon dioxide circle absorption system. Oral intubation was performed and anaesthesia maintained with nitrous oxide and oxygen with 10% cyclopropane added, using the same system. The patient was permitted to breathe spontaneously. No relaxant drugs were used. Operation and anaesthesia were uneventful.

At operation, which lasted 2 hours, a large thrombus which had originated on an atheromatous plaque was removed from the femoral and iliac arteries. Immediately post-operatively an elective tracheotomy was performed. The patient revived immediately on discontinuance of anaesthetic. On return to the ward, he was treated with nasopharyngeal oxygen inhalation. He died 3 hours after the operation.

AUTOPSY:

no autopsy

COMMENT:

This death was the result of the patient's pre-existing disease. Anaesthesia is not considered contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
9.2.63	3	No comment	ORD	Prolonged haemorrhagic hypotension.	No

Name: Kathleen Storbridge Age: 84 Sex: F Race: E

Disease: Ruptured abdominal aortic aneurysm

Operation: Resection of aneurysm and graft of abdominal aorta.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

On presentation in theatre, the patient wasmoribund. Pulse rate was 120/minute, respirationwas rapid - 30/minute - and B.P. was 60 mm.Hg systolic. She was in a state of severe hypovolaemic shock.

PREMEDICATION:

Atropine 0.3 mg. by intravenous injection 10 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen administered via a carbon dioxide circle absorption system, using a closed circuit technique. Oral intubation was performed after 3 minutes and anaesthesia then maintained with nitrous oxide, oxygen and occasional mimimal traces of ether, via the circle absorption system using an IPPR technique. dTc was used as the relaxant, 20 mg. total being used in two divided doses throughout the operation which lasted 1¾ hours, until the patient's death. ECG monitoring was used throughout. Following a rapid laparotomy, the diagnosis was confirmed and the aorta was speedily clamped above the site of the rupture. There was gross retroperitoneal haemorrhage around the aorta. Immediately before clamping the aorta, the patient was heparinised. After clamping the aorta the patient remained pulseless and the B.P. remained just recordable at a level of 60 mm.Hg systolic. This persisted in spite of rapid transfusion of 6 pints blood, the patient remaining pale and vasoconstricted. The aorta was rapidly resected and a graft inserted; this took only 30 minutes. The removal of the aortic clamp precipitated a further severe drop in B.P. and cardiac arrest ensued in spite of B.P. cuffs inflated around the legs to prevent excessive run-off. In view of the patient's age and the prolonged hypotension, no cardiac massage was attempted.

AUTOPSY:

No autopsy.

COMMENT:

This was an inevitable death. The patient was moribund at the commencement of surgery and the level of the systolic B.P. (60 mm.Hg) did not even rise when the aortawas clamped. Rapid blood replacement failed to produce any change in her condition either. The removal of the clamp from the aorta after resection and grafting was sufficient to produce a final drop in B.P. and ischaemia of the myocardium which resulted in cardiac arrest. If one can fault the anaesthetist's technique at all, it is on the omission of the administration of sodium bicarbonate to this patient to correct the inevitable metabolic acidosis from which she must have suffered. This was an inevitable death to which anaesthesia was necessarily and unavoidably contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
10.2.63	2	No comment	< 24	Undetermined.	No

Name: Jackson Gadlela Age: 41 Sex: M Race: B

Disease: Carcinoma of the oesophagus Operation: Oesophagectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Except for the evidence of a marked loss of weight, the patient was in fair condition. B.P. 110/80 mm.Hg. Fluid balance had been adequately restored.

PREMEDICATION:

Pethidine 100 mg., atropine 0.6 mg., administered 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 300 mg., succinylcholine 50 mg. ventilation with oxygen, topical analgesia of the larynx and oral intubation. After the return of spontaneous respiration, anaesthesia was maintained with nitrous oxide and oxygen with intermittent minimal traces of ether administered via a non-rebreathing circuit, using an IPPR technique. Gallamine was used as the relaxant. To control hiccoughs that occurred on laparotomy, 2 doses of pethidine 20 mg. were administered. In all, a total dose of gallamine 220 mg. was used throughout the operation, which lasted 6 hours, the last dose being administered 81 minutes before the end of the procedure. Operation and anaesthesia were uneventful, adequate transfusion for replacement of blood loss being performed. At the conclusion of the operation, residual curarisation was adequately reversed, 1 mg. neostigmine preceded by atropine 0.6 mg. being administered. At the end of the procedure, the patient rapidly regained consciousness and there was no respiratory inadequacy. He was returned to the ward. No post-anaesthetic complications were identifiable during the immediate post-operative period. The patient died 13 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

The reason for this patient's death is uncertain. However, on enquiry, it does not appear to be related in any way to the anaesthetic and so anaesthesia is not regarded as contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
11.2.63	2	No comment	< 24	Cerebro-vascular accident	No

Name: N. Warrington Age: 79 Sex: F Race: E

Disease: Subtrochanteric fracture of femur. Operation; Insertion of Smith Peterson pin and plate.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Not good - the patient had been admitted 1 week before surgery. She had aortic nodular sclerosis and had been in cardiac failure on admission, which had now been adequately controlled by digitalisation. B.P. 180 mm.Hg systolic and the pulse rate was 100/minute. In addition she suffered from Parkinsonism. Haemoglobin concentration before operation was 9 gm.%, which was not "topped up" immediately pre-operatively for fear of precipitating cardiac failure.

PREMEDICATION:

Atropine 0.65 mg. given 65 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 75 mg., succinylcholine 30 mg. ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was then maintained with nitrous oxide, oxygen and minimal ether vapour, administered via a carbon dioxide circle absorption system. The patient was permitted to breathe spontaneously. Though blood loss at operation waa minimal, blood was transfused from the commencement of the operation being continued throughout and subsequently for a short time in the ward. In all, 600 ml. blood was transfused during the operative and immediate post-operative periods. Throughout the procedure, which lasted 85 minutes, the course of anaesthesia was quite uneventful. At the conclusion of operation and on discontinuance of anaesthesia, she rapidly regained consciousness and there was no respiratory inadequacy. Some 12 hours after anaesthesia, the patient became drowsy, then comatose and she died shortly afterwards. Death appeared clinically to have resulted from a cerebrovascular accident.

AUTOPSY:

No autopsy.

COMMENT:

This patient appeared to recover satisfactorily from anaesthesia. Death seems to have been due to a cerebrovascular accident and anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
12.2.63	1	Probably	ORD	Anoxic anoxia. Respiratory obstruction	Yes

Name: Mrs. de Freitas Age: 68 Sex: F Race: E

Disease: Diabetic gangrene of left leg. Operation: None performed. Intended amputation of left leg.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Gravely ill patient presenting with diabetic gangrene of the left leg. It had taken 24 hours of rehydration and insulin therapy to control the state of ketosis in which she presented. In addition, she suffered diabetic nephrotic syndrome, with albuminuria 4+. She was in cardiac failure and had been digitalised. Bilateral pleural effusions were present as was a degree of pulmonary oedema. There was a 3 finger hepatomegaly. Fasting blood sugar shortly before surgery was 244 mg.% and the blood urea was 62 mg.%.

PREMEDICATION:

Atropine 0.65 mg. administered 30 minutes pre-operatively.

ANAESTHETIC:

On arrival in theatre, the patient displayed cyanosis of the face, lips and tongue and was breathing with a moderate tachypnoea of 30/minute. Anaesthesia was induced with cyclopropane and oxygen, delivered via a circle absorption system (closed) with spontaneous respiration. Anaesthesia was induced in 5 minutes following a stormy delirium stage. Great difficulty was experienced in maintaining a clear airway. Succinylcholine was then administered, 25 mg., and after a short period of IPPR with oxygen, oral intubation was performed. The patient immediately developed marked respiratory muscle spasm and bronchospasm. This was of such a degree that the resistance to inflation was so great that ventilation of the lungs became impossible. The cyanosis worsened. Suspecting that the endotracheal tube might have been kinked, the anaesthetist immediately removed this and attempted pulmonary inflation by means of a face piece and oropharyneal airway. Inflation was still impossible, laryngospasm now seemed to have added to the anaesthetist's difficulties. Cyanosis was now gross. The trachea was re-intubated and inflation now was possible. By this time, however, the brachial pulse was impalpable and cardiac arrest was diagnosed. The surgeon was informed and immediately performed a thoracotomy. A large amount of pleural effusion was found. Ventricular fibrillation was present. Cardiac massage was commenced immediately and after 3 minutes, electrical defibrillation was attempted. After one shock of 110 volts for 0.1 second, ventricular fibrillation recurred. Following a second shock, complete asystole supervened. This was refractory to all further treatment.

AUTOPSY:

Enlarged and scarred heart with gross atheroma of the coronary arteries. Markedly enlarged kidneys. Surgical wound of left chest. Septic lesions of both big toes.

COMMENT:

This patient was anoxic before anaesthesia was induced. This state was worsened following the induction of anaesthesia by difficulties experienced by the anaesthetist in maintaining a clear airway. The profound difficulty experienced in ventilating the patient following intubation was probably due to bronchospasm, and expiratory muscle spasm, known to follow tracheal intubation under light cyclopropane

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anaesthesia. No topical analgesia of the larynx was used. The dose of succinylcholine (25 mg.) was apparently too small to control this muscular spasm. That a larger dose of muscle relaxant may have permitted adequate pulmonary ventilation, by paralysing the muscles of respiration, is apparent from the fact that there was no further resistance to ventilation after cardiac arrest had occurred.

Cardiac arrest was probably due to anoxic anoxia. But, vagal reflexes at the time of intubation - in the presence of light cyclopropane anaesthesia and anoxia - may well have played parts. The presence of ischaemic heart disease undoubtedly increased the liability to cardiac arrest in the presence of anoxia. The fault here lies with the attempted intubation of the patient in a light cyclopropane anaesthesia, in the absence of adequate analgesia of the larynx, without adequate muscular relaxation.

A further fault in the conduct of this case is the ommission before the induction of anaesthesia of the tapping of pleural effusions known to be present. In spite of the seriousness of this patient's condition before the induction of anaesthesia, the conduct of anaesthesia is regarded as the major immediate cause of the patient's death.

PREVENTABILITY:

Because the faults in the conduct of this anaesthetic, identified in the commentary, are considered to have been correctable, this death is considered probably preventable. This case is so classified in spite of the fact that the patient may well have died of her pre-existing disease even had she survived the anaesthetic.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
13.2.63	1	Probably	ORD	Cardiac arrest due to inadvertant hypothermia and ischaemic anoxia.	Yes

Name: Nonfundo Bala Age: 3 Sex: F Race: B

Disease: Extradural haematoma Operation: Carotid angiography. Parietal craniotomy.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

This child had fallen from a 1st floor balcony on the previous evening and had been admitted to hospital for observation. X-ray skull revealed large linea fractures in the left parietal area. During the night the patient became stuperose and developed a right hemiplegia. The left pupil became dilated. Immediately before anaesthesia the temperature and respiration were normal. B.P. was 160 mm.Hg systolic and the pulse rate 140/minute. The child weighed 35 lb. There had been no other pre-operative treatment.

PREMEDICATION:

Atropine 0.65 mg. administered 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane via an infant T-piece system (Cape Town system). After topical analgesia of the larynx, oral intubation was performed. Anaesthesia was then maintained with inhalation of nitrous oxide and oxygen with intermittent ether vapour, delivered via an Ayre's T-piece. Spontaneous respiration was permitted throughout the operation. Carotid angiography took approximately 1 hour to complete and revealed a large left extradural haematoma. Tachycardia, present before anaesthesia was induced, persisted throughout the course of the anaesthetic. At the conclusion of angiography, the B.P. was 160 mm. Hg systolic and the pulse rate 150/minute. Though cross-matched blood was not available at this time, it was decided to proceed with craniotomy. Blood had been ordered while angiography was in progress and was cross-matched while the operation was commenced. Blood loss was fairly marked from the outset of the operation, although not all was fresh bleeding. The extradural haematoma itself contained approximately 50 ml. clotted blood. Compatible blood arrived in the theatre 25 minutes after the start of the operation by which time the estimated blood loss, including that in the haematoma, was approximately 1,400 ml. The B.P. had begun to fall and was at a systolic level of 90 mm.Hg when blood transfusion was instituted. Blood, cold as it had arrived straight from storage, was administered rapidly. After 150 ml. had been transfused, a brief period of bradycardia preceded cardiac arrest. IPPR with oxygen was commenced and a thoracotomy performed immediately. This revealed a flabby heart in asystole. Cardiac massage was commenced immediately; cardiac filling was poor. Intracardiac injections of adrenaline 1:20,000 in doses of 1, 2 and 4 ml., and calcium chloride 400 mg. followed by repeated cardiac massage were of no avail, except for the occasional isolated heart beat. Resuscitative measures were eventually abandoned 45 minutes after cardiac arrest, 80 minutes after the start of the craniotomy.

AUTOPSY:

Fractures of skullwith left surgical defect and scalp flap wound. Surgical wound of chest. Abrasion of face. Lacerations of scalp. Brain showed flattening of left side and flattening of convolutions,

and narrowing of sulci on the right side, but no other signs of cerebral compression or damage.

COMMENT:

Two possible causes for the cardiac arrest in this case are apparent. The child had lost something over 10% of her estimated blood volume, most of this fairly rapidly, before blood transfusion was commenced. When transfused, blood that was very cold (probably at a temperature of between 4-10°C) was given rapidly. The occurrence of cardiac arrest immediately following the transfusion of 150 ml. of this blood, an amount which should have largely replaced the deficit in blood volume, indicates the probability that gross cooling of the heart - added to the effects of the initial hypovolaemia - caused cardiac arrest. The child may have already cooled somewhat before transfusion, simply from a period of anaesthesia of 1 hour in a cold theatre environment. The complete failure of resuscitative measures to elicit any response adds strength to the diagnosis of hypothermic cardiac arrest.

Two faults are identifiable in the management of this anaesthetic:-

(1) The commencement of the operation before compatible blood was available in the theatre,

(2) The rapid transfusion of very cold blood,

While the failure to make some attempt at warming the heart by pouring warm saline into the thoracic cavity, once thoracotomy had been performed, may be considered a fault in the resuscitative technique too.

PREVENTABILITY:

In view of the obvious correctability of the faults ennumerated above, this death is considered probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
14.2.63	2	No comment	< 24	Multiple injuries	Yes

Name: Alska Stainson Age: 29 Sex: F Race: E

Disease: Multiple injuries, ruptured spleen, lacerated liver, ruptured caecum. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient, who suffered from depressive psychosis, had attempted suicide by jumping off a building. A laparotomy and splenectomy had been performed immediately after admission to hospital. Lacerations in the caecum had been repaired and lacerations in the liver sutured and packed. At the concludion of this operation, tracheotomy had been performed. However, following this operation, haemorrhage into the abdomen continued and another laparotomy was considered necessary 12 hours after the first. At this stage the patient was gravely ill. Pulse rate was 120/minute and B.P. fluctuated between 70 and 120 mm. Hg systolic. Massive blood transfusion had been given during and after the first operation.

PREMEDICATION:

Atropine 0.65 mg. administered 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide and oxygen administered via the tracheostome by an IPPR technique with carbon dioxide absorption. Succinylcholine chloride, administered in intermittent divided doses, used as the relaxant, total dose being 100 mg. during the 2½ hour operation during which the liver was resutured and packed. At the conclusion of the second operation the patient regained consciousness and respiration appeared adequate. There were no apparent sequelae from the administration of succinylcholine. The patient died 7 hours after this second operation.

AUTOPSY:

Bruising of eyelids. Sutured lacerated wounds of lower lip and chin. Fractured lower jaw. Acute oedema of glottis, vocal chords and larynx. Tracheotomy. Sutured surgical wound of abdominal wall. Acute pulmonary oedema with severe congestion of the lungs. Blood-stained fluid in pleural and abdominal cavities. Sutured lacerated wounds of inferior surface of liver and numerous small haemorrhages in liver substance. Spleen had been removed surgically. Haemorrhages into right suprarenal gland which, on histology, showed areas of necrosis and haemorrhage.

COMMENT:

This patient died as a result of the grave multiple injuries present. Anaesthesia is considered not contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
15.2.63	3	No comment	ORD	Acute cardiac failure.	Yes

Name: Wase Lause Age: 46 Sex: M Race: B

Disease: Post-pericardiectomy vascular collapse. ?Cardiac tamponade.

Operation: Thoracotomy, Left.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had suffered from severe constrictive pericarditis for which a pericardiectomy had been performed 2 days previously. Sudden cardiovascular collapse had ensued leading to a diagnosis of possible cardiac tamponade. Immediate thoracotomy was indicated. Immediately before anaesthesia the patient was moribund with no palpable pulse and no recordable B.P. A small pleural effusion was known to be present on the right side.

PREMEDICATION:

None.

ANAESTHETIC:

ECG monitoring was used throughout the anaesthetic and operative procedures. Following pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen, succinylcholine 50 mg. being given then and oral intubation performed. Anaesthesia was maintained with nitrous oxide and oxygen administered using an IPPR technique, via a carbon dioxide circle absorption system. One further dose of succinylcholine was given.

Left thoracotomy showed no cardiac tamponade and no reason for the patient's condition. The heart was beating, but ineffectively, and cardiac massage was instituted immediately. An infusion of noradrenaline 1:450,000 was commenced but without effect on the heart action. Sodium bicarbonate 50 ml. 4% solution was administered with equal lack of effect. During cardiac massage the feeble heart beat gradually petered out over 30 minutes.

AUTOPSY:

Right pleural effusion. No pulmonary embolus. Leg veins normal. Left haemothorax. Evidence of recent pericardiectomy.

COMMENT:

The reason for this patient's collapse after pericardiectomy is not immediately apparent. At the time of this anaesthetic he was moribund and the death must, in effect, be regarded as inevitable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
16.2.63	3	No comment	ORD	Haemorrhage.	Yes

Name: James Taylor Age: 70 Sex: M Race: E

Disease: Abdominal aortic aneurysm. Operation: Resection of aneurysm and graft of abdominal aorta.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Although he had a large abdominal aortic aneurysm, the patient was in a fair condition. Circulatory and respiratory systems were normal except for the presence of gross arteriosclerosis. B.P. was 150 mm. Hg systolic.

PREMEDICATION:

Omnopon gr. 1/3, atropine gr. 1/100, given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 200 mg., succinylcholine 50 mg., topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether vapour, administered via a circle carbon dioxide system usingan IPPR technique. dTc was used as the relaxant. The course of anaesthesia was uneventful until full exposure of the aneurysm. At this stage, surgical technical difficulties were encountered resulting in severe uncontrolled haemorrhage. Massive transfusion was resorted to, but after transfusion of 38 pints blood, bleeding still continued. Hypothermia and the effects of massive transfusion were now added to those of massive haemorrhage and the patient died.

AUTOPSY:

The heart was flabby. There was severe atherosclerosis of the coronary arteries with near total occlusion of their ostia. Severe atherosclerosis of the entire aorta with an abdominal aortic aneurysm extending into the right iliac artery. A teflon graft was in place. The inferior vena cava had sutured wounds in several places. There was a large retroperitoneal haemorrhage as well, 300 ml. blood being contained in the abdominal cavity.

COMMENT:

Death in this case resulted from the combined effects of massive haemorrhage and massive transfusion with resultant hypothermia. Anaesthesia is not considered contributory. In that the patient died while anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
17.2.63	2	No comment.	< 24	Mesenteric thrombosis Infarction of bowel.	No

Name: Mrs. A. Petrucci Age: 76 Sex: F Race: E

Disease: Haematemesis (caused by infarction of small bowel - found at operation)

Operation: Laparotomy

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient presented with haematemesis from what was thought to be a bleeding peptic ulcer. Despite massive transfusion, her condition continued to deteriorate. Immediate laparotomy was indicated. Immediately before anaesthetic was induced, the patient was in extremis. Respiration was laboured and gasping, B.P. was 80 mm.Hg systolic, and pulse rate was 96/minute. She appeared grossly shocked. In addition she suffered from pulmonary emphysema and two toes on the left foot were gangrenous.

PREMEDICATION:

Atropine 0.65 mg. given 5 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 25 mg., and oral intubation. Anaesthesia was continued with nitrous oxide and oxygen administered via a circle absorption system with an IPPR technique. No further relaxant was used except a further dose of succinylcholine 10 mg. to facilitate the closure of the abdomen. Following induction of anaesthesia, the B.P. rose to 100 mm.Hg systolic and at the conclusion of the operation was still at this level.

Laparotomy revealed superior mesenteric artery thrombosis with infarction of the jejunum and ileum. Other than the deflation of the bowel, to facilitate closure of the abdomen, no further operative procedure was attempted. At the conclusion of the operation and on discontinuance of the anaesthetic, which in all had lasted 55 minutes, the patient regained consciousness to the same state as pre-operatively, and respiration returned to pre-anaesthetic state. She died shortly after her return to the ward.

AUTOPSY:

No autopsy.

COMMENT:

The nature of this patient's pathology meant that death was inevitable. In fact, she was moribund before the administration of the anaesthetic. Anaesthesia is not considered contributory to the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
18.2.63	3	No comment	ORD	Pulmonary embolus.	Yes

Name: Mrs. M. Kearney Age: 39 Sex: F Race: E

Disease: Bilateral fracture dislocation of hips. Recovering head injury.

Operation: Manipulation and reduction of dislocated hips.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had been involved in a motor accident 2 weeks previously and had suffered a head injury. She had been deeply unconscious for some days and had now recovered consciousness and was a little confused. Circulatory and respiratory systems were normal. There was a bilateral fracture dislocation of her hips. Haemoglobin was 11 gm.%.

PREMEDICATION:

Atropine 0.6 mg. and pethilorfan 50 mg., given 100 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 100 mg. and maintained with nitrous oxide and oxygen. During manipulation of the hips, relaxation was achieved by the administration of succinylcholine 50 mg. and an IPPR technique was used. Spontaneous respiration was allowed to recommence while the hip was being stabilised. While this was being done, 50 minutes after the start of anaesthesia, cyanosis suddenly became apparent, the pupils dilated and cardiac arrest was diagnosed. IPPR with oxygen and immediate external cardiac massage produced an immediate resumption of normal heart beat, the pupils constricted and cyanosis disappeared. However, within 5 minutes, cardiac arrest recurred. External cardiac massage was now ineffective. Immediately, thoracotomy was performed and the heart was found to be in asystole. Cardiac massage was attempted but ventricular fibrillation, which was refractory to all attempts to defibrillate it electrically, supervened.

AUTOPSY:

A large antemortem thrombosis was found occluding both pulmonary arteries. Extensive bruising in the pelvis. Old-standing signs of diffuse head injury.

COMMENT:

This death was obviously due to a massive pulmonary embolus. Anaesthesia is not considered contributory but, in that death occurred while the patient was anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
19.2.63	3	No comment	ORD	Uncontrollable haemorrhage. Massive transfusion. Ruptured aortic aneurysm	Yes

Name: William Bohls Age: 67 Sex: M Race: E

Disease: Rupture of abdominal aortic aneurysm

Operation: Laparotomy. Resection of aneurysm and graft of abdominal aorta.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor - the patient was obviously shocked and vasoconstricted. B.P. 120 mm.Hg systolic, whereas it was known to be normally 210 mm. Hg systolic. In addition, on ECG, there was evidence of myocardial ischaemia and a right bundle branch block.

PREMEDICATION:

Atropine 0.65 mg., given 2 minutes before induction of anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 175 mg., succinylcholine, topical analgesia of the larynx and endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen with minimal ether vapour, administered via a circle absorption system, by an IPPR technique. Relaxation was achieved with intermittent doses of dTc, a total of 45 mg. being used during the 3¾ hours of the operation. With the induction of anaesthesia, there was an initial fall of B.P. to a level of 80 mm.Hg systolic, which recovered to its former level following the administration of phenylephrine 0.5 mg. Subsequently, throughout the operation, the B.P. fluctuated between 100 and 160 mm.Hg systolic, depending on the balance between blood loss and replacement by transfusion.

At laparotomy a large abdominal aneurysm, which had ruptured, was found to involve the lower aorta and both iliac arteries. After heparinisation, the aorta was cross clamped, the aneurysm resected and a graft inserted. Blood loss was severe and was replaced as lost. For 3 hours the patient's condition was maintained in a reasonable state. However, after about 20 pints blood had been transfused, a steady deterioration set in in his condition. At about the time the aortic clamp was removed, the B.P. wa s 80 mm.Hg systolic. Further blood transfusion and the administration of vasopressor drug, phenylephrine, in divided doses, and also of atropine, failed to elicit any response. The patient was cold. The B.P. continued to fall progressivelyand, while the 28th pint blood was being transfused, he died. No cardiac massage was attempted.

AUTOPSY:

Surgical replacement of lower portion of abdominal aorta and iliac arteries by artificial teflon graft. Gross arteriosclerosis of abdominal and thoracic aorta. Gross arteriosclerosis of coronary arteries with calcification and narrowing, and an old thrombus. Heart very much enlarged, weighing 750 gm. Left ventricular wall very much thickened, 4 cm. thick. Cirrhosis of the liver. Smell of ether.

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COMMENT:

This death resulted from massive haemorrhage and subsequent massive blood transfusion. One of the most important factors ultimately responsible for cardiac arrest in patients in these circumstances is the resultant hypothermia. One other complicating factor, besides the lack of clotting due to heparinisation, is the derangement in clotting mechanism which results from massive transfusion. This patient, in addition, had grave coronary atherosclerosis which, in the presence of periods of hypotension, must have resulted in myocardial ischaemia. In that this patient died while anaesthetised, the case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
20.2.63	2	No comment	< 24	Mitral incompetence Pulmonary oedema.	Yes

Name: Nomakepe Mafuguana Age: 16 Sex: F Race: B

Disease: Mitral stenosis. Operation: Mitral valvotomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient suffered from a tight mitral stenosis and was in poor condition. Cardiac failure was poorly controlled with digitalis. Immediately before surgery the pulse rate was 120/minute and the B.P. 110/60 mm.Hg. Respiratory system was normal.

PREMEDICATION:

Atropine 0.06 mg., phenergan 25 mg., pethilorfan 25 mg., given 1 hour pre-operatively.

ANAESTHETIC:

ECG monitoring was conducted throughout. Anaesthesia was induced with inhalation of nitrous oxide and oxygen. dTc 20 mg. was injected and orotracheal intubation was performed. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system, using an IPPR technique. Circulatory collapse followed the induction of anaesthesia. Injection of small doses of phenylephrine 0.5 mg. elicited poor response. The infusion of adrenaline 1:100,000 restored a more adequate cardiac action and B.P. At cardiotomy, the mitral valve was found to be grossly stenosed and structurally badly damaged. Following dilatation of the mitral valve with a Tubb's dilator, stenosis was relieved but the valve was now incompetent. A total dose of dTc 30 mg. had been given during the procedure which lasted 2 hours. At the conclusion of the operation residual curarisation was adequately reversed with neostigmine 20 mg. preceded by atropine 1.25 mg. When anaesthetia was discontinued, the patient rapidly regained consciousness and breathed adequately. However, within 10 minutes, she developed gross pulmonary oedema. Tracheotomy was performed immediately and IPPR with a mechanical pulmonary ventilator was instituted. In spite of energetic supportive and resuscitative measures, the patient died in cardiac failure 6 hours post-operatively.

AUTOPSY:

Bilateral cardiac ventricular dilatation accompanied tricuspid incompetence, manifesting its chronicity in gross cardiac cirrhosis of the liver. Gross pulmonary oedema, the left lung weighing 530 gm. as did the right. Surgical commissural relief of mitral stenosis had been satisfactorily achieved with a Tubb's dilator.

COMMENT:

The cause of this patient's death was the acute mitral incompetence which followed attempts at closed mitral valvotomy. Though initially the administration of anaesthetic to this patient did cause circulatory collapse, this was adequately treated in the circumstances. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
21.2.63	2	No comment	< 24	Peritonitis.	Yes

Name: Leo Morkel Age: 40 Sex: M Race: C

Disease: Intestinal obstruction following stab wound in abdomen.

Operation: Laparotomy. Drainage of peritonitis.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted intoxicated following an abdominal stab wound, with symptoms suggestive of intestinal obstruction. After rehydration fluid therapy, with 2 litres fluid, the B.P. rose from a level of 150 mm.Hg to 200 mm.Hg systolic. However, immediately before operation the B.P. dropped dramatically to 80 mm.Hg systolic with a pulse rate of 90/minute. The patient appeared toxic.

PREMEDICATION:

Pethidine 75 mg., atropine 0.65 mg., administered 40 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 50 mg., and orotracheal intubation. Immediately after induction of anaesthesia, no B.P. was recordable. An immediate left thoracotomy was performed but the heart was found to be beating. After rapid transfusion of 4 pints reconstituted plasma and 1 litre 5% dextrose in water, and 2 pints blood (4 litres fluid in all) the B.P. rose steadily to a level of 125 mm.Hg systolic, where it remained throughout the remainder of the operation, which lasted 2 hours 5 minutes. Throughout the procedure anaesthesia was maintained with nitrous oxide and oxygen, with intermittent addition of ether vapour, administered by an IPPR technique via a carbon dioxide circle absorption system. At operation the small bowel was found to have been perforated by a stab wound and there was gross generalised peritonitis, with 5 pints pus in the peritoneal cavity. No intestinal obstruction was evident. At the conclusion of the operation and on discontinuance of the anaesthetic, the patient rapidly regained consciousness. Respiration was normal. After return to the ward, the patient died, 5 hours post-operatively.

AUTOPSY:

Gross generalised peritonitis with sutured laceration in the small bowel.

COMMENT:

This patient died of gross peritonitis and septicaemia. His death appears to be in no way related to the anaesthetic or its administration.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
22.2.63	2	No comment	<24	Haema-temesis.	Yes

Name: Aletta Milner Age: 37 Sex: F Race: E

Disease: ?Haemoptysis Operation: Bronchoscopy

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor. The patient presented with gross haemoptysis and by the time she came to theatre, 2 pints blood had been transfused. At this time the B.P. was 90 mm.Hg systolic. She had generalised rhonchi over both lung fields. The left pulmonary base was dull and showed a pneumonic lesion on X-ray.

PREMEDICATION:

Atropine 0.6 mg. given just before the operation.

ANAESTHETIC:

Anaesthesia was induced by inhalation of nitrous oxide and oxygen, with gradually added Halothane, the patient breathing spontaneously. When light anaesthesia had been induced, topical analgesia of the larynx was followed by bronchoscopy. During this procedure, an apnoeic anaesthetic technique was adopted following the injection of succinylcholine 50 mg. and thereafter intermittent ventilation with nitrous oxide and oxygen was given via the bronchoscope. At bronchoscopy no cause was found for the haemorrhage. At the conclusion of the procedure, which lasted 45 minutes, the patient regained consciousness and respiration was adequate. After her return to the ward, the patient developed a further massive bleed, still thought to be haemoptysis, and she died 4 hours after the operation.

AUTOPSY:

Other than some increased bronchial secretion and inhalation pneumonia in the left lower lobe, there was nothing in the lungs to explain the haemoptysis. At the lower end of the oesophagus was an area of oesophagitis with an ulcer overlying a large artery.

COMMENT:

This patient died of a massive blood loss from an ulcer in the oesophagus, which was undiagnosed, eroding an artery. This was misdiagnosed as an haemoptysis. Anaesthesia is not considered to have been contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
23.2.63	1	Possibly.	ORD	Hypotension Ischaemic anoxia. Cardiac arrest.	Yes

Name: H. Mantel Age: 78 Sex: M Race: E

Disease: Urethral calculus Operation: Cystoscopy

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor. The patient had had a myocardial infarct 4 years before, since when he had suffered cardiac failure, controlled with digitalisation. Though fully digitalised, he now had dependent oedema and little or no exercise tolerance. Gross cardiomegaly was present with ECG evidence of left ventricular damage. At this time the B.P. was 160/100 mm.Hg. In addition, chronic bronchitis and emphysema were present. A urethral calculus necessitated bystoscopy.

PREMEDICATION:

Atropine 0.65 mg., given 3 minutes before the start of the operation.

ANAESTHETIC:

In view of the patient's poor cardiac status, the anaesthetist had decided to administer a caudal saddle block. He did not adhere to this decision and, changing his mind, proceeded to administer a general anaesthetic. Anaesthesia was induced with thiopentone 150 mg. followed by inhalation of nitrous oxide, oxygen and Halothane via a Magill semi-open circuit, with spontaneous breathing. Halothane concentration was increased during induction, to 1.5%, and after 10 minutes, reduced to 0.5%. A further 25 mg. thiopentone was given when cystoscopy commenced, 10 minutes after induction of anaesthesia. With induction, the B.P. dropped from 160 mm.Hg to 100 mm.Hg systolic while the pulse rate rose from 84 to 96/minute. 10 minutes after the start of cystoscopy, 20 minutes after commencement of the anaesthetic, cardiac arrest occurred without any apparent warning. While external cardiac massage was undertaken, immediate orotracheal intubation was performed and IPPR with oxygen instituted. External cardiac massage proved ineffective and after 3 minutes thoracotomy was performed and internal cardiac massage resorted to. The heart was found to be asystolic. A noradrenaline drip infusion, 4 microgm./cc., was started. After some minutes of cardiac massage, 5 cc. adrenaline 1:10,000 was injected into the left ventricle. Cardiac massage was resumed. Ventricular fibrillation now occurred. In spite of repeated attempts at electrical defibrillation, this proved completely refractory, recurring each time after the initial shock had arrested the heart. Resuscitative measures were abandoned after 45 minutes.

AUTOPSY:

Myocardial hypertrophy. Heart weighed 470 gm. Severe myocardial fibrosis. Severe atherosclerosis of both coronary arteries with extremely narrow ostia. Cerebral arteriosclerosis.

COMMENT:

This cardiac arrest followed fairly soon after a fall in the B.P. from the pre-operative level, after induction of anaesthesia. In view of the diseased state of the patient's myocardium and coronary arteries, one may suggest that the drop in pressure was sufficient to cause functional myocardial ischaemia from poor coronary perfusion. Although immediately cardiac arrest, the B.P. was recorded as 100 mm.Hg

systolic / ...

systolic, normally an adequate level, this patient's B.P. before anaesthesia had been 160 mm.Hg, and this may well have already been lower than his previous "normal" level because of myocardial failure.

The refractoriness of the ventricular fibrillation, once started, so often a feature of an ischaemic myocardium, would bear out this proposition. There is little else positive in the clinical record to account for the cardiac arrest. One may postulate that the respiratory depression known to occur with Halothane anaesthesia, especially when taken in conjunction with the emphysema from which the patient was known to suffer, may have been an added factor. But the anaesthetist does not remark on this aspect. The possibility that any drop in B.P. might cause myocardial ischaemia obviously occurred to the anaesthetist as he planned initially to use a caudal saddle block anaesthetic technique. Having changed his mind, one must question his use of Halothane, a drug known to cause hypotension, especially in such an atherosclerotic patient.

Even if the mechanism proposed as the explanation of cardiac arrest is not correct, the administration of the anaesthetic was obviously a major causative factor in this patient's death in the context of his gross myocardial disease.

PREVENTABILITY.

Having anticipated the possible sequelae of a general anaesthetic in this patient, the anaesthetist failed to adhere to his original decision to use a local anaesthetic technique. Having embarked on a general anaesthetic, one must question his use of Halothane, a drug known to produce hypotension. Because adherence to his original decision - or the abstention from the use of Halothane - may have resulted in the avoidance of hypotension, this death is regarded as possibly preventable. Considering the patient's serious myocardial disease, this is perhaps a harsh judgment.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
24.2.63.	2	No comment	< 24	Cardiac failure.	Yes

Name: Steven Nomkelde Age: 46 Sex: M Race: B

Disease: Tuberculous constrictive pericarditis. Operation: Pericardiectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Chronic cardiac failure from constrictive pericarditis. The patient had severe dyspnoea on exertion. There were crepitations at both lung bases and there was a 3 finger hepatomegaly. B.P. 135/100 mm.Hg Pulse rate 70/minute. The patient was fully digitalised and on anti-tuberculous therapy.

PREMEDICATION:

Pethidine 100 mg., atropine 0.6 mg., administered 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 150 mg., succinylcholine 50 mg. ventilation with oxygen, and oral intubation, followed by IPPR with nitrous oxide and oxygen until spontaneous respiration returned. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system using an IPPR technique. dTc was used as the relaxant, 27 mg. being used throughout the operation, which lasted 3¼ hours. The course of anaesthesia was uneventful. At the conclusion of the operation residual curarisation was reversed with neostigmine 2 mg., in divided doses, preceded by atropine 1.2 mg. At the conclusion of the procedure and discontinuance of anaesthesia, the patient rapidly regained consciousness and there was no respiratory inadequacy. The patient died 17 hours post-operatively from causes unrelated to any complication of anaesthesia.

AUTOPSY:

Constrictive pericarditis with evidence of recent pericardiectomy. Old right-sided empyema with massive fibrosis of basal pleura. Chronic congestion of the liver.

COMMENT:

Anaesthesia is not regarded as contributory to this patient's death which, on enquiry, appears to have resulted from cardiac failure.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
25.2.63	3	No comment	ORD	Coronary thrombosis	Yes

Name: Elizabeth Melamba · Age: 48 Sex: F Race: B

Disease: Inoperable Stage 4 carcinoma of breast with secondary, disseminated carcinomatosis.

Operation: Bilateral oophorectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This obese patient, who weighed 200 lbs., was critically ill. She had Stage 4 carcinoma of the right breast with widely disseminated secondary carcinomatosis. Lymphoedema was present in the right arm and adjacent part of the trunk. There was an extensive right pleural effusion and she was dyspnoeic. There was no cardiac failure. B.P. was 140 mm.Hg systolic, pulse rate was 120/minute. Pre-operatively the pleural effusion was tapped, 1 litre being removed.

PREMEDICATION:

Atropine 0.6 mg., given 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 50 mg., and oral intubation. During the operation, which lasted only 30 minutes, anaesthesia was maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. Two further doses of succinylcholine 25 mg. each were given to maintain relaxation. The course of anaesthesia was completely uneventful,however, when the anaesthetic was discontinued at the end of the procedure and the patient was allowed to breathe spontaneously, she rapidly developed respiratory inadequacy due to profuse pulmonary oedema as she regained consciousness. She was re-intubated and IPPR with oxygen was recommenced. Digoxin 1 mg. was given intravenously and shortly after, 25 m.Eq./l. sodium bicarbonate solution was injected intravenously. However, the patient's cardiac action became progressively worse and she died 30 minutes after regaining consciousness.

AUTOPSY:

Generalised carcinomatosis. Fresh thrombosis of left coronary artery. Gross atherosclerosis. Small aneurysm of left ventricular wall. Pulmonary oedema.

COMMENT:

The terminal pulmonary oedema that occurred immediately after termination of the anaesthetic would appear from autopsy to have been the result of acute myocardial failure from coronary thrombosis. From the account of the smooth and uneventful course of the anaesthetic, it is impossible to decide when this occurred. Furthermore, it is not possible to decide if the anaesthetic management played any contributory role in this occurrence. No episodes of hypotension were recorded, although the B.P. and pulse rate were meticulously monitored during anaesthesia. The occurrence of coronary thrombosis in the Bantu is rare. While in persons in any racial group, coronary thrombosis during anaesthesia in itself is uncommon. From the autopsy evidence of a small left ventricular aneurysm, it would appear that the patient had had a previous episode of coronary occlusion. As death occurred in the theatre immediately following anaesthesia and before her return to the ward, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
26.2.63	2	No comment c	< 24	?Myocardial infarction.	No

Name: Emma Grootien Age: 79 Sex: F Race: E

Disease: Acute vascular occlusion of lowerlimbs, saddle embolus of aorta.

Operation: Aorto-iliac embolectomy and endarterectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient presented with acute vascular occlusion of both lower limbs. She was a known diabetic and suffered from severe hypertension. Although on treatment with Rautrax, on admission her B.P. was 210/110 mm.Hg. She suffered auricular fibrillation and had been in cardiac failure, but was now fully digitalised. The diabetes had been controlled by the oral hyperglycaemic diabenese.

PREMEDICATION:

Atropine 0.6 mg. was given 70 minutes pre-operatively.

ANAESTHETIC:

Following induction of anaesthesia with thiopentone 200 mg., succinylcholine and oral intubation, anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique via a carbon dioxide circle absorption system. dTc was used as the relaxant, a total dose of 18 mg. being used throughout the 100 minutes of the procedure. The course of anaesthesia was punctuated by some violent oscillations in B.P., the result of profuse bleeding from the operative site, followed by equally rapid blood replacement. However, at no stage did the B.P. fall below 170 mm.Hg. At operation, an embolus was found at the bifurcation of the aorta. This was removed and an endarterectomy was performed on both iliac arteries. At the conclusion of the operation, residual curarisation was adequately reversed with neostigmine 1 mg. preceded by atropine 0.6 mg. The patient regained consciousness immediately after discontinuance of anaesthesia. After her return to the ward, she died suddenly 17 hours after the operation, from what appeared clinically to be myocardial ischaemia.

AUTOPSY:

No autopsy.

COMMENT:

Anaesthesia is not considered as contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
27.2.63	2	No comment	< 24	Cerebral laceration	Yes

Name: Richard Weber Age: 31 Sex: M Race: C

Disease: Head injury. Fractured skull. Intracranial haemorrhage.

Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was unconscious and showed signs of intracranial compression. The B.P. was 160 mm.Hg systolic and there was a bradycardia of 64/minute. Breathing was stertorous with coarse rhonchi audible over both lung fields.

PREMEDICATION:

Atropine 0.6 mg. given intravenously 5 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide and oxygen. Succinylcholine 25 mg. was given and topical analgesia of the larynx performed for orotracheal intubation. Bronchial toilette was then performed. During angiography, anaesthesia was maintained with inhalation of nitrous oxide, oxygen and a trace of trichlorethylene, with spontaneous respiration. During the burrhole craniotomy an IPPR technique with nitrous oxide and oxygen, with carbon dioxide absorption, was used following the administration of dTc 9 mg. The course of anaesthesia was untoward.

Craniotomy revealed a large subdural and intracerebral haematoma with brain laceration. At the end of the procedure, which took 130 minutes, all residual curarisation was adequately reversed with neostigmine 1.5 mg. preceded by atropine 1.2 mg. The patient did not recover consciousness. A tracheotomy was performed immediatedly post-operatively. The patient died 9 hours post-operatively.

AUTOPSY:

Several lacerated wounds of head, face and legs. Linear fracture of the skull from occiput to temporal bone. Gross epidural, subdural and subarachnoid bleeding. Severe brain laceration with intra-cerebral pontine haemorrhages. Severe contusion of pelvic viscera with retroperitoneal haemorrhage.

COMMENT:

Anaesthesia is not considered contributory at all to this death, which resulted from severe cerebral laceration.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
28.2.63	2	No comment	< 24	Prolonged hypotension. Metabolic acidosis.	No

Name: Jacobus Philips Age: 64 Sex: M Race: E

Disease: Ruptured abdominal aortic aneurysm. Operation: Laparotomy. Resection of aneurysm and grafting of abdominal aorta.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted with a frank rupture of an abdominal aortic aneurysm, in gross oligaemic shock, with severe peripheral vaso-constriction. Pulse rate 160/minute, B.P. 100 mm.Hg systolic. Blood transfusion was immediately commenced and the patient was taken direct to the operating theatre.

PREMEDICATION:

Atropine 0.6 mg. by intravenous injection immediately before operation.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 100 mg, succinyl-choline 50 mg., IPPR with oxygen, and oral intubation. During the operation, anaesthesia was maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system, a trace of ether vapour being used throughout. dTc was used as the relaxant, a total of 40 mg. being administered during the course of the operation, which lasted 5 hours. After clamping of the aorta, rapid transfusion was resorted to and the B.P. was elevated to between 120-140 mm.Hg systolic, where it was maintained. Aortic resection was extremely difficult and blood loss throughout the procedure was severe. In all, 22 pints blood (warmed in a water bath) were transfused. In spite of this, the patient's temperature dropped to 32°C during the operation. To counteract the sodium citrate contained in the large volume of blood transfused, 6 gm. calcium gluconate was given during the course of transfusion. To correct the metabolic acidosis, 300 m./Eq. per litre sodium bicarbonate was also administered. The patient rapidly regained consciousness at the end of the operation on discontinuance of anaesthesia. Residual curarisation was reversed with 1 mg. neo-stigmine preceded by atropine 1.2 mg. 30 minutes after the patient was returned to the ward, an episode of profound hypotension occurred because of haemorrhage. Concomittant with this, the respiratory excursion became diminished. Rapid blood transfusion and IPPR with oxygen for 30 minutes restored circulatory homeostasis at this stage. Four hours post-operatively the blood pH was found to be 7.08. The patient died ultimately 8 hours after the operation.

AUTOPSY:

No autopsy.

COMMENT:

The cause of this death was almost certainly metabolic acidosis due to gross haemorrhage and the sequelae of massive blood trans-fusion in a cold, shocked patient. Anaesthesia was not contributory to the death.

CASE NO:	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
29.2.63	3	No comment	> 24	Air embolism. ?Hypokalaemia.	Yes

Name: B. Lavis Age: 53 Sex: F Race: E

Disease: Mitral and tricuspid incompetence.

Operation: Insertion of mitral prosthesis and tricuspid annuloplasty, using cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Poor - the patient had been in chronic cardiac failure for some time. She had been digitalised for years and intensively treated with diuretics. There was still evidence of congestive cardiac failure, crepitations at both lung bases and a pleural effusion. This pleural effusion was tapped pre-operatively. There was 3 finger hepatomegaly.

PREMEDICATION:

Sodium seconal gr. 1½ orally 3 hours before anaesthesia; atropine 0.6 mg. by intramuscular injection 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx, oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered vy an IPPR technique with minimal Halothane. A non-rebreathing circuit was utilized employing a cyclator ventilator. dTc 30 mg. in all was administered in the pre-bypass phase. Anaesthesia duringbypass was maintained with Halothane 1.5% administered via the bubble oxygenator. Post-bypass anaesthesia was continued, trouble-free, with nitrous oxide and oxygen alone. Two hours cardiopulmonary bypass was necessary. At the conclusion of operation, the restoration of normal respiration was rapid and the EEG was normal. While an immediate post-operative chest X-ray was being taken, the patient was moved and a sudden cessation of respiration and cardiovascular collapse ensued, with widely dilated pupils. The clinical diagnosis at this time was one of massive air embolism. The patient was immediately treated with IPPR and oxygen. Tracheostomy was performed and IPPR was continued by this means, using a Bird ventilator. An intravenous infusion of adrenaline 1:100,000 was commenced. The patient failed to regain consciousness post-operatively and failed to resume normal respiration after this episode. 30 hours after the operation, cardiac arrhythmia and then cardiac arrest occurred.

AUTOPSY:

Other than repaired cardiac lesions, nothing significant was found.

COMMENT:

Clinically, the episode at the conclusion of this operation appears to have been a massive air embolism. Spontaneous respiration never resumed post-operatively. Subsequent cardiac arrest, 30 hours after the procedure, may have been related to this or to the hypokalaemia which is now known to be a feature of long cardiopulmonary bypass procedures in this type of case. In that the sudden apnoea and circulatory collapse occurred while the patient was still anaesthetised, this case is included in group 3, although anaesthesia is not regarded as contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
30.2.63	3	No comment	ORD	Cardiac arrest. ?Hypokalaemia	Yes

Name: R. Visser Age: 32 Sex: F Race: E

Disease: Mitral and aortic incompetence.

Operation: Insertion of mitral and aortic valve prosthesis, using cardiopulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

Poor - the patient was in chronic congestive cardiac failure with mitral and aortic valve regurgitation. She had auricular fibrillation. B.P. 100/60 mm.Hg. There was dependent oedema and a 4 finger hepatomegaly. She had been digitalised and had intensive treatment with diuretics.

PREMEDICATION:

Atropine 0.6 mg. given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 100 mg., succinylcholine, ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen, with minimal Halothane (intermittent 0.5%) via a carbon dioxide circle absorption system by an IPPR technique. dTc 20 mg. was used as the relaxant. Operation and anaesthesia continued without untoward complications until the end of the operation, during closure of the skin. At this time there was sudden cardiac arrest, following the onset of multiple ventricular extrasystoles. Cardiac arrest proved totally refractory to treatment.

AUTOPSY:

Other than evidence of open heart surgery and the replacement of the mitral and aortic valves with prosthetic valves, there was nil of note.

COMMENT:

The reason for the refractory cardiac arrest and the previous irritability of the myocardium was not correctly diagnosed at this time. In the light of subsequent experience, it is thought to have been due to hypokalaemia, now known to be a feature of prolonged bypass surgery in this type of case. Anaesthesia is not considered contributory to this death, but as the cardiac arrest occurred while the patient was anaesthetised, the case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
31.2.63	2	No comment	< 24	?Myocardial infarction.	No

Name: Maggie Wyngaard Age: 62 Sex: F Race: C

Disease: Carcinoma of the ascending colon. Operation: Laparotomy. Entero-colostomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

On admission to hospital, the patient had a large mass in the right iliac fossa and was anaemic. Over the week before surgery he received a total of 5 pints blood by transfusion. Haemoglobin concentration was 14 gm.%. B.P. was 150/95 mm.Hg. All other systems were normal.

PREMEDICATION:

Morphine gr. 1/6, atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx and oral intubation. Throughout operation anaesthesia was maintained with nitrous oxide and oxygen with minimal ether, administered with an IPPR technique via a carbon dioxide circle absorption circuit. Gallamine, total dose 120 mg., was used as the relaxant during the operation, which lasted 1 hour 55 minutes.

At operation, the carcinoma of the ascending colon was found to be irresectable and an enterocolostomy was performed. Blood loss was estimated at between 700 - 1,000 ml. Two pints blood were transfused and 1 gm. calcium gluconate given concomittantly. At the conclusion of the operation, residual curarisation was reversed with 2 mg. neostigmine preceded by atropine 1.2 mg. Spontaneous respiration returned and was adequate. The patient regained consciousness rapidly after discontinuance of anaesthesia. Post-operative course was quite normal but the patient died suddenly 10 hours after operation.

AUTOPSY:

No autopsy.

COMMENT:

Although the exact cause of this death is not known (possibly myocardial infarction), it does not appear to have been related to the anaesthetic management.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
32.2.63	2	No comment	< 24	?Myocardial ischaemia. Haemorrhage	No

Name: F. Rees Age: 68 Sex: M Race: E

Disease: Prostatic obstruction. Prostatic adenoma. OperatioN: Transurethral resection of prostate.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Besides the prostatic disease, this patient had hypertension and arteriosclerosis. There was a history of three episodes of cerebro-vascular accidents and he was now hemiplegic. B.P. was now 210/120 mm.Hg. There was also pulmonary emphysema.

PREMEDICATION:

Pethidine 50 mg., atropine 0.6 mg. given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with thiopentone 125 mg., followed by inhalation of nitrous oxide, oxygen and trichlorethylene. After topical analgesia of the larynx, oral intubation was performed. Anaesthesia was then maintained with nitrous oxide, oxygen and trichlorethylene administered via a non-rebreathing circuit by an IPPR technique. Gallamine in doses of 40 mg. and subsequently 20 mg. was administered during operation. The course of anaesthesia was untoward, the B.P. being maintained at the pre-operative level throughout, and the pulse rate at 110/minute. At the end of operation, spontaneous respiration was satisfactory but neostigmine 1 mg. preceded by atropine 1.2 mg. was given to ensure reversal of curarisation. At the conclusion of the anaesthetic, which lasted 1½ hours, the patient rapidly regained consciousness. Five hours post-operatively he was found to be sweating, with a B.P. which had dropped from the previous level of 210 mm.Hg to 120 mm.Hg systolic. Shortly thereafter he died.

AUTOPSY

No autopsy

COMMENT:

This death was due either to myocardial infarction or to ischaemia as a result of hypotension following post-operative haemorrhage, in the presence of pre-existing coronary disease. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
33.2.63	2	No comment	< 24	Mediastinitis.	No

Name: Jacoba Bingham Age: 60 Sex: F Race: C

Disease: Traumatic perforation of oesophagus. Operation: Thoracotomy. Repair of tear in oesophagus.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had been submitted to gastroscopy the previous day, during which procedure the oesophagus was perforated. She was now in severe oligaemic shock and was moribund. Temperature 101°F, B.P. 80 mm.Hg systolic, pulse rate 140/minute, in spite of blood transfusion which was thought to be adequate. A left pneumothorax was present. In addition she suffered albuminuria and bilirubinaemia and had a 3 finger hepatomegaly with jaundice.

PREMEDICATION:

Atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

On arrival in the operating theatre, the B.P. was being maintained at a level of 80 mm.Hg systolic by an intravenous infusion of Aramine. Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 50 mg., and oral intubation. Anaesthesia was maintained during the operation with nitrous oxide and oxygen administered by a carbon dioxide circle absorption system with an IPPR technique. Gallamine 100 mg. in divided doses was administered throughout the procedure, which lasted 1¾ hours.

During operation, when a large haemothorax was found together with a tear in the oesophagus - which was repaired - 4 pints blood were transfused. Initially the B.P. was maintained with small 0.5 mg. doses of phenylephrine. After adequate blood transfusion the B.P. stabilised at 100 mm.Hg systolic. At the conclusion of operation, residual curarisation was reversed with neostigmine 1.5 mg. preceded by 1.2 mg. atropine. Adequate spontaneous respiration returned and the patient rapidly regained consciousness on discontinuance of the anaesthetic. She died 12 hours post-operatively from causes related to the original disease.

AUTOPSY:

No autopsy.

COMMENT:

This death resulted from the patient's original disease, the complications of a perforation of the oesophagus which was not treated timeously. These were mediastinitis and the effects of prolonged profound oligaemic shock. The death does not appear to have been related to the anaesthetic management.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
34.2.63	2	No comment	< 24	Cerebral laceration	Yes

Name: Leo Neilson Age ; 54 Sex: M Race: E

Disease: Bullet wound of skull with cerebral laceration. Operation: Exploratory craniotomy and debridement of brain.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had suffered a bullet wound in the skull. He was stuperose and had C.S.F. leaking into the pharynx. B.P. 140/70 mm.Hg pulse rate 110/minute, respiration 28/minute. Apart from pulmonary emphysema, he had no other abnormalities.

PREMEDICATION:

Atropine gr. 1/100, given 1 hour pre-operatively.

ANAESTHETIC:

Bronchial toilette was performed following oral intubation, after topical analgesia of the larynx. Anaesthesia was then induced and maintained with nitrous oxide and oxygen administered via a circle absorption system using an IPPR technique. Gallamine 70 mg. was given in divided doses throughout the operation, which took 2 hours 5 minutes. During the operation, blood was transfused as lost, a total of 3½ pints being given. B.P. and pulse rate were maintained at the pre-anaesthetic level throughout. At the conclusion of operation, residual curarisation was reversed with 2 mg. neostigmine preceded by atropine 1.2 mg. Normal spontaneous respiration returned and was adequate. Bronchial toilette was performed at the conclusion of anaesthesia. The endotracheal tube was left in situ and the patient was returned to the ward. He failed to regain consciousness after the operation and died from the effects of the cerebral laceration, 8 hours post-operatively.

AUTOPSY:

Sutured right frontal wound, 3 cm. inlength, 4 cm. above the right outer eyebrow. Right frontal craniotomy for debridement and haemostasis of brain. Bilateral subarachnoid cerebral haemorrhage with extensive laceration and pulverisation of frontal lobes of the brain. Bullet tip was found in right fronto-parietal area.

COMMENT:

Death was due to extensive cerebral laceration. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
35.2.63	2	No comment	< 24	Cerebral laceration	Yes

Name: B. Wanzi Age: Unknown Sex: M Race: B

Disease: Subdural haematoma. Cerebral laceration. Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted to hospital deeply unconscious having been found thus many hours previously, the victim of an assault. Tracheostomy was performed shortly after admission because of retained bronchial secretions. Before operation B.P. was 110/60 mm.Hg pulse rate 100/minute. The respiration was irregular at 20/minute.

PREMEDICATION:

Atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Following bronchial toilette performed via the tracheostomy tube, anaesthesia was induced and maintained with nitrous oxide and oxygen with intermittent ether vapour, administered via a carbon dioxide circle absorption system using an IPPR technique. No relaxant was used.

At operation, which took 3 hours, a large subdural haematoma was evacuated. At the conclusion of the procedure spontaneous respiration was resumed. The patient died 15 hours post-operatively without regaining consciousness.

AUTOPSY

Diffuse neuronal damage. Multiple contused areas and petecheal haemorrhages in the brain. Bilateral subarachnoid haemorrhages.

COMMENT:

Anaesthesia is not considered contributory to the death, which resulted from diffuse neuronal injury.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
36.2.63	2	No comment	<24	Coronary thrombosis	Yes

Name: J. Rycklief Age: 82 Sex: M Race: C

Disease: Intestinal obstruction. Operation: Laparotomy. Reduction of volvulus of small bowel.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor. Besides the small bowel obstruction for which he was admitted, the patient had gross pulmonary emphysema and X-ray evidence of chronic bilateral fibroid tuberculosis of both upper and mid-zones of the lungs. Fluid replacement before operation was adequate. B.P. was 130/80 mm.Hg shortly after admission and had risen to 180/100 mm.Hg with a pulse rate of 100/minute just before operation.

PREMEDICATION:

Atropine 0.6 mg. given 1½ hours pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen, administered via a carbon dioxide absorption system by an IPPR technique. dTc 20 mg. was administered in divided doses during the course of operation, which lasted 1¾ hours. The B.P. was labile, though maintained at 120 mm.Hg systolic for the major part of the procedure, it fell on three occasions to a level of 80 mm. Hg systolic. On the first occasion this was restored by the adoption of a head-down tilt on the operating table. Increasing the rate of blood replacement restored the B.P. to its former level on the second occasion, while on the third, a dose of 0.5 mg. phenylephrine produced a rise in B.P. to 140 mm.Hg systolic, where it remained for the rest of the operation. Two attacks of bronchospasm occurred during the course of the anaesthetic; on each occasion these were adequately controlled by 250 mg. aminophyllin.

At operation, a volvulus of the small bowel round adhesions wasfound, and was reduced. At the conclusion of the procedure, spontaneous respiration returned. Residual curarisation was reversed with 2 mg. neostigmine preceded by atropine 1.2 mg., and spontaneous respiration was adequate. The patient rapidly regained consciousness on discontinuance of the anaesthetic. He died suddenly 3 hours post-operatively.

AUTOPSY:

Arteriosclerosis of the coronary arteries with fresh blood clot. Tuberculosis of lungs with gross emphysema. Arterionephrosclerosis of only kidney. Adhesions of small intestine which had caused the obstruction.

COMMENT:

Autopsy revealed a fresh coronary thrombosis. In the light of present knowledge, it is impossible to evaluate the part played by the effects of anaesthesia and the labile fluctuations in B.P. in the causation of this event. The anaesthetic management was no obvious faults and is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
37.2.63	2	No comment	< 24	Cerebral haematoma	Yes

Name: Dennis Vegotine Age: 26 Sex: M Race: C

Disease: Intracerebral haematoma (traumatic). Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was stuperose and had left hemiplegia and evidence of meningitis. Temperature 103°F, pulse rate 120/minute, B.P. 160/110 mm.Hg. Respiration was adequate but diffuse rhonchi and crepitations on auscultation of the chest revealed retained bronchial secretions.

PREMEDICATION:

Atropine 0.6 mg.given 40 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 100 mg., succinylcholine 40 mg., pre-oxygenation, topical analgesia of the larynx and oral intubation, followed by bronchial toilette. During angiography anaesthesia was maintained with nitrous oxide and oxygen alone, delivered via a carbon dioxide circle absorption system by an IPPR technique. Gallamine, total dose 100 mg., was administered during the procedure, which lasted 4 hours in all. The course of anaesthesia was untoward.

At operation, a large intracranial haematoma was evacuated. Blood was transfused as lost, 1.5 litres being required. At the conclusion of the operation, tracheostomy was performed. Normal spontaneous respiration resumed at the end of the procedure, and reversal of residual curarisation was ensured by administration of 1 mg. neostigmine preceded by atropine 1.2 mg. The patient died 3 hours post-operatively without regaining consciousness.

AUTOPSY:

Diffuse subarachnoid haemorrhage. Right sided fronto-parietal medullary haematoma which had been surgically evacuated. Diffuse bilateral parietal pyogenic meningitis.

COMMENT:

This patient died of existing cerebral damage. Anaesthesia is not considered to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
38.2.64	2	No comment	< 24	Cerebral haemorrhage	Yes

Name: Doortjie Moos Age: 32 Sex: M Race: C

Disease: Head injury and cerebral laceration. Right subdural haematoma.

Operation: Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATUS:

Stuperose, but the patient responded to painful stimuli. Pyrexial: temperature 102°F. Pulse rate 84/minute, B.P. 150/90 mm.Hg. He appeared somewhat dehydrated. Respiration appeared adequate but diffuse rhonchi on auscultation of the lungs evidenced gross retention of bronchial secretions.

PREMEDICATION:

Atropine 0.6 mg. given 40 minutes pre-operatively.

ANAESTHETIC:

Following pharyngeal and oral toilette, anaesthesia was induced with a thiopentone 200 mg., succinylcholine 40 mg., oxygenation, oral intubation sequence. Following this, bronchial toilette was performed and a large amount of purulent bronchial secretion removed by suction aspiration. Anaesthesia was maintained with nitrous oxide, oxygen and Halothane delivered via a Magill semi-open circuit with spontaneous breathing. The course of anaesthesia during the operation, which lasted 3 hours, was entirely untoward. The patient died 14 hours post-operatively without regaining consciousness.

AUTOPSY:

Three burrholes in skull. Diffuse cerebral haemorrhages. Diffuse cerebral damage. Bilateral haematoma of neck.

COMMENT:

This death was the result of diffuse traumatic neuronal injury. Anaesthesia is not considered to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
39.2.64	1	Possibly	< 24	Post-relaxant respiratory abnormality. Anoxic anoxia.	No

Name: Harry Lambert Age: 67 Sex: M Race: C

Disease: Strangulated left inguinal hernia. Operation: Left inguinal herniorrhaphy. Appendicectomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient had had a hernia for 20 years. He presented with a 5 day history that it had become painful and tender; he had been vomiting. He was extremely ill and toxic, and was dehydrated. B.P. 85/65 mm.Hg There was an enormous left indirect inguinal hernia which hung down to his knees; it was oedematous and tender and contained bowel. There was generalised abdominal tenderness. Adequate fluid replacement therapy was instituted before operation.

PREMEDICATION:

Atropine 0.6 mg. given 70 minutes pre-operatively.

ANAESTHETIC:

After pre-oxygenation, anaesthesia was induced with thiopentone, 150 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. Gallamine, initial dose 60 mg., and two further doses of 30 mg. each (total dose 120 mg. during the operation), was used as the relaxant. The operation lasted 3 hours.

At operation the hernial sac was found to contain half the small bowel together with caecum and a gangrenous appendix; pus was also present. The appendix was removed, the bowel cleaned and returned with great difficulty to the abdomen, the hernia was repaired. 1 pint blood was transfused during the procedure and the B.P. remained fairly steady at a level of 100 mm.Hg systolic, where it had been pre-anaesthesia. Pulse rate was maintained at 120/minute throughout the major part of the operation. On one occasion, for approximately 5 minutes, the B.P. dropped to 80 mm.Hg, when visceral traction occurred. The last dose of gallamine was administered 1 hour 20 minutes before the end of the operation. At the conclusion, spontaneous respiration returned but was inadequate. After administration of neostigmine 2 mg. preceded by 1.2 mg atropine, intercostal activity appeared normal but, because of gross abdominal distension, it was doubtful if respiration was in fact adequate. On discontinuance of the anaesthetic, the patient recovered consciousness and was returned to the ward. Twenty minutes later intercostal activity was observed to have virtually ceased. Respiration was abdominal and quite inadequate. Cyanosis was evident. The anaesthetist was called to the ward to re-intubate the patient to establish IPPR. However, the patient died before this could be effected.

AUTOPSY:

No autopsy.

COMMENT:

The immediate cause of death was quite clearly anoxic anoxia, the result of underventilation. The factors probably responsible were:

(1) gross abdominal distension,

/ ...

(2) Incomplete reversal of curarisation, or possibly post-operative recurarisation.

Whatever the cause, as the hypoventilation was obvious immediately at the conclusion of anaesthesia, this patient should not have been returned to the ward before adequate IPPR had been established, either through an endotracheal tube or a tracheotomy. In view of the gross abdominal distension described, and the difficulty experienced by the surgeon in returning the bowel to the abdominal cavity, the failure of the surgeon to surgically decompress the bowel must also be faulted.

As the primary fault was the failure of the anaesthetist to provide adequate pulmonary ventilation immediately after the conclusion of the anaesthetic, the anaesthetic management is regarded as the major causative factor in this patient's death.

PREVENTABILITY:

The failure to provide adequate pulmonary ventilation immediately post-operatively was easily correctable. In spite of the patient's serious surgical disease, this death therefore must be regarded, from the point of view of the anaesthetic management, as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
40.2.64	2	No comment	< 24	Infarction of bowel.	No

Name: Brigid Doidge Age: 56 Sex: F Race: E

Disease: Mesenteric thrombosis. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund. Besides the abdominal lesion, this patient had gross myocardial disease - ischaemic changes evident on ECG, with bundle branch block and multiple ventricular extrasystoles. She was not in congestive cardiac failure. There was both respiratory and metabolic acidosis, the former the result of gross abdominal distension leading to some hypoventilation. Standard bicarbonate shortly after admission was 16.5 m.Eq./litre. Before operation, 140 m.Eq. Sodium bicarbonate was administered. Gastric aspiration and fluid replacement therapy had been instituted.

PREMEDICATION:

Atropine 0.4 mg. given 2 hours pre-operatively.

ANAESTHETIC:

After pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen, succinylcholine 40 mg. being administered and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen, with intermittent trace of ether vapour, administered via a circle absorption system by an IPPR technique. A further dose of succinylcholine 20 mg. was given to facilitate closure of the abdomen at the end of laparotomy. The operation lasted 40 minutes, and the course of anaesthesia was untoward.

Laparotomy revealed multiple areas of infarcted bowel, both small and large, due to multiple embolisation. Nothing could be done for this condition. At the conclusion of the anaesthetic, the patient recovered consciousness rapidly. She died 12 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

Death was due to existing disease and anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
41.2.64	2	No comment	< 24	Multiple injuries. Cerebral damage.	Yes

Name: George Hector Age: Unknown Sex: M Race: C

Disease: Multiple injuries. Fractured skull. Subarachnoid haemorrhage. Fractured left tibia and fibula. Fractured right tibia.

Operation: Carotid angiography. Craniotomy, for compound depressed fractured skull. Fixation of fractures of legs.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Comatose. There was blood in the pharynx. B.P. 180/75 mm.Hg. Diffuse rhonchi on auscultation of the lungs. Two pints blood had been transfused pre-operatively.

PREMEDICATION:

Nil.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone, succinylcholine, oxygenation, topical analgesia of the larynx, and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. The course of anaesthesia was untoward during the operation. Blood was transfused as lost.

At the conclusion of the operation, which lasted 3 hours, spontaneous respiration recommenced. The patient died 8 hours post-operatively without regaining consciousness.

AUTOPSY

Fracture of the left parieto-occipital area of the skull. Left subdural haematoma, left subarachnoid haemorrhage, left intracerebral tempero-parietal haematoma. Diffuse bilateral frontocortical contusion. Left temporocortical contusion. Fractures of right tibia and fibula, and left Pott's fracture.

COMMENT:

This patient died of diffuse neuronal injury. Though one may question the use of thiopentone for induction of anaesthesia in a patient already comatose, the anaesthetic management does not appear to have played any part in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
42.2.64	3	No comment	<24	Haemorrhage. Cardiac arrest. Cerebral anoxia.	Yes

Name: Elizabeth Majekwa Age: 1¾ yrs. Sex: F Race: B

Disease: Left Wilm's tumour Operation: Nephrectomy and excision of Wilm's tumour.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

After she had received a course of deep radiotherapy for a very large Wilm's tumour, this child was presented for nephrectomy and excision of this tumour. She was thin,in poor general condition, weighing 19 lbs. Preoperatively the B.P. was 100/60 mm.Hg.

PREMEDICATION:

Atropine 0.2 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen, and Halothane. After topical analgesia of the larynx, oral intubation was performed. Anaesthesia was then maintained with nitrous oxide and oxygen with 0.5% Halothane, by an IPPR technique via a modified T-piece system. Surgical difficulties were encountered because of the size of the tumour, its vascularity and the results of the previous deep X-ray therapy. These resulted in gross haemorrhage which at times was uncontrollable. Grave difficulties were experienced by the anaesthetist in replacing blood as it was lost, by rapid transfusion. Attempts to replace blood with pressure transfusion resulted in rupture of veins and new infusion sites were established with difficulty. Blood replacement lagged behind loss, and after the operation had been in progress for 2 hours, cardiac arrest occurred. After cardiac massage via the diaphragm, the heart resumed beating; adrenaline 1 ml. 1:10,000 and 5 ml. 10% calcium gluconate had also been injected. During completion of the operation, 5 m.Eq. sodium bicarbonate was administered with 10 ml. 50% dextrose in water. All anaesthetic agents were discontinued and oxygen only was administered by IPPR from the time of the cardiac arrest. A total of 800 ml. blood (approximately equal to the child's total blood volume) was transfused during the procedure.

At the conclusion of surgery, spontaneous respiration resumed. The patient failed to recover consciousness post-operatively and she died 7 hours later, after a further haemorrhage.

AUTOPSY:

Left nephrectomy performed. Cervical mediastinal and mesenteric lymph glands enlarged. Heart showed puncture mark (needle).

COMMENT:

The cause of cardiac arrest during operation was ischaemic anoxia from acute hypovolaemia and possibly from hypothermia as a result of massive transfusion of cold blood, though this lagged behind the actual blood loss. In the absence of temperature monitoring, the latter is supposition. The cerebral ischaemia that occurred at the time of cardiac arrest, and probably for a period before this event, appears to have caused severe and probably irreversible cerebral damage. The immediate cause of death seems to have been post-operative haemorrhage.

/ ...

The rate at which blood was lost during operation was beyond that at which the anaesthetist could reasonably be expected to replace adequately and timeously by ordinary means. The anaesthetic management can perhaps be faulted in that, when it became apparent that it was impossible to replace blood adequately by ordinary means, the surgeon should have been requested to transfuse blood directly into the inferior vena cava.

This death basically is a surgical death, but to this extent the anaesthetic management is regarded as necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
43.2.64	2	No comment	<24	Removal of cerebral angioma. Haemorrhage	No

Name: Martha Crowley Age: 52 Sex: F Race: E

Disease: Haemorrhage following surgical removal of cerebral frontal angioma.

Operation: Re-opening of craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

On the previous day, craniotomy had been performed and a cerebral angioma had been removed. Severe bleeding had commenced post-operatively from the operative sites, necessitating the re-opening of the craniotomy and exploration of the site. Blood transfusion had been instituted but, on arrival in the theatre, the patient was in a state of severe circulatory collapse with vasoconstriction. B.P. 60 mm.Hg systolic, respiration exhibited a tracheal tug and the patient was deeply unconscious.

PREMEDICATION:

Nil.

ANAESTHETIC:

Oral intubation was performed following topical analgesia of the larynx, and anaesthesia was induced and maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. During operation, 6 pints blood were transfused together with 2 mg. phenylephrine in divided doses. Initially the B.P. rose to 100 mm.Hg systolic and after 1½ hours of operation it rose to a level of 130 mm.Hg systolic, where it stabilised for the remainder of the procedure. The operation lasted 4 hours. At the conclusion of the operation, spontaneous respiration was resumed. The patient failed to regain consciousness and died 21 hours post-operatively.

AUTOPSY:

No autopsy.

COMMENT:

This death resulted from the effects of the surgical procedure. Anaesthetic management is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
44.2.64	2	No comment	<24	Intra-cranial haemorrhage	No

Name: N. Mwanda Age: 37 Sex: M Race: B

Disease: Intracranial haemorrhage (traumatic) Operation: Ventriculogram and burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was deeply comatose. B.P. 130/90 mm.Hg. Respiration was spontaneous and normal. Temperature 99°F.

PREMEDICATION:

Atroping 0.6 mg. given intravenously 5 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen, injection of succinylcholine and oral intubation, following topical analgesia of the larynx. Throughout the operation, which lasted 85 minutes, anaesthesia was maintained with nitrous oxide and oxygen delivered via a Magill semi-open circuit, with spontaneous breathing. The course of anaesthesia was untoward.

At the conclusion of the operation, tracheostomy was performed. The patient failed to recover consciousness post-operatively and died after 12 hours.

AUTOPSY

No autopsy

COMMENT:

Death was due to the pre-existing cerebral injury. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
45.2.64	2	No comment	< 24	Undetermined.	Yes.

Name: Maria Nyeni Age: 67 Sex: F Race: B

Disease: Pelvic carcinomatosis. Operation: Laparotomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Grossly emaciated; abdomen distended with ascites. B.P. was 120/80 mm.Hg systolic.

PREMEDICATION:

Atropine 0.6 mg. given just prior to the induction of anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen administered in closed circuit. dTc 10 mg. was given and oral intubation performed. A period of IPPR followed and then anaesthesia was continued with nitrous oxide and oxygen, via a carbon dioxide circle absorption system by an IPPR technique. One further dose of dTc 10 mg. was given during the operation, which lasted 50 minutes. During the procedure the B.P. was extremely labile, being between 120 and 80 mm.Hg systolic. After 2 doses of 0.5 mg. each phenylephrine, the B.P. stabilised at a level of 110 mm.Hg systolic.

At operation the pelvis was found to be completely filled with matted carcinomatous tissue and omentum. The operation was terminated after biopsy of the omentum. At the conclusion of the anaesthetic, spontaneous respiration resumed. Residual curarisation was reversed withneostigmine 2 mg. preceded by atropine 1.2 mg. The patient recovered consciousness rapidly post-operatively and respiration was spontaneous and of adequate volume. She died suddenly 4 hours post-operatively; the cause was not diagnosed but did not appear to be related to the anaesthetic.

AUTOPSY

Carcinoma of the body of the uterus. Sedondaries involving large bowel and peritoneum. 500 ml. of ascitic fluid in the abdominal cavity. No immediate cause for death was found.

COMMENT:

Although the cause of this patient's death was not determined, it does not appear to have been related to the anaesthetic she had received 4 hours previously. Consciousness had been rapidly regained and maintained after anaesthesia, and respiration was spontaneous and of normal, adequate volume. Of interest is the fact that although at the conclusion of laparotomy the abdominal cavity was 'dry', autopsy revealed 500 ml. ascitic fluid in the abdominal cavity, which must have accumulated there between the time of operation and her death 4 hours later.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
46.2.64	2	No comment	< 24	Diffuse neuronal injury.	Yes

Name: Michael Reynolds. Age: 22 Sex: M Race: E

Disease: Head injury, fractured skull, intracranial haemorrhage. Operation: Carotid angiography.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund. Deeply stuperose. Extensor spasms following severe head injury. Tachypnoea - 35/minute with diffuse coarse rhonchi audible over both lung fields. B.P. 140/90 mm.Hg.

PREMEDICATION: Nil

ANAESTHETIC:

Following pharyngeal toilette, oral intubation was performed. This was followed by bronchial toilette. Anaesthesia was induced and maintained with nitrous oxide and oxygen via a Magill semi-open circuit with spontaneous breathing. Following angiography, no operation was performed.

At the conclusion of the anaesthetic, the endotracheal tube was left in situ and the patient returned to the ward, still comatose, as he had been before anaesthesia. Death occurred 12 hours post-operatively.

AUTOPSY:

Extensive fractures of vault of skull. Fractures of base of skull extending forward into frontal bone. Bilateral thin extradural haematomata. Extensive lacerations of both frontal lobes of the brain.

COMMENT:

This death resulted from extensive traumatic brain damgge. The anaesthetic and its management were not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
47.2.64	2	No comment	< 24	Cerebral injury.	Yes

Name: Edward Mkolo Age: Unknown Sex: M Race: B

Disease: Head injury, fractured skull. Operation: Carotid angiography.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Extremely poor - comatose, pyrexial (temperature 103°F). Pulse rate 150/minute. B.P. was 110 mm.Hg systolic. The patient had tachypnoea and coarse rhonchi were audible bilaterally over all lung fields. Tracheotomy had been performed and tracheo-bronchial toilette revealed large amounts of purulent bronchial secretions.

PREMEDICATION:

Atropine 0.6 mg. given 45 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced and maintained with inhalation of nitrous oxide and oxygen, administered via a tracheostome by means of a Magill semi-open circuit, with spontaneous breathing. The course of anaesthesia was untoward.

After discontinuance of anaesthetic, when the angiogram had been completed, the patient remained comatose, as he had been before the operation. Respiration was rapid but appeared to be adequate. There was no cyanosis. Following angiography, the pyrexia worsened to a temperature of 104°F and the patient died 19 hours after angiography.

AUTOPSY

Fracture of base of skull. Bilateral subdural subarachnoid haemorrhages.

COMMENT:

Death was the result of the pre-existing cerebral injury. Anaesthesia is not considered to have been contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
48.2.64	3	No comment	ORD	Mesenteric thrombosis Infarction and gangrene of bowel.	Yes

Name: Isaac Minting Age: 75 Sex: M Race: C

Disease: Intestinal obstruction. Mesenteric thrombosis. Operation: Exploratory laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This old man had had a myocardial infarction 13 years previously. He now presented with large bowel obstruction of some 36 hours' duration. He appeared to be moribund immediately before anaesthesia. An infusion of the vasopressor Aramine was necessary to maintain the B.P. at a level of 100 mm.Hg systolic. Because of a rare blood group, difficulty was experienced in obtaining compatible blood for transfusion. 1 litre plasma and 1 litre "Travert" were transfused pre-operatively. A laparotomy was considered necessary immediately in the face of his deteriorating condition.

PREMEDICATION.

Atropine 0.6 mg. administered 5 minutes pre-operatively.

ANAESTHETIC:

Following induction of anaesthesia with cyclopropane and oxygen, administered via a closed carbon dioxide circle absorption system, succinylcholine 20 mg. was given and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen alone via the same system by an IPPR technique. No further relaxant was administered. Shortly after the induction of anaesthesia, the B.P. fell to 70 mm.Hg systolic. Soon after this the patient s condition deteriorated further, with a further fall in B.P. and 30 minutes after the start of the anaesthetic, cardiac arrest occurred. Meanwhile laparotomy had revealed extensive infarction of both large and small bowel, following mesenteric thrombosis. Because of this finding, no attempts were undertaken to resuscitate the patient.

AUTOPSY

The entire large bowel was gangrenous and there was a perforation at the splenic flexure with approximately 200 ml. of bloody faeculent fluid in the peritoneal cavity. The cause of the gangrene was not demonstrated but in all probability was due to mesenteric thrombosis. Severe arteriosclerosis was present in both coronary arteries. There was evidence of an old myocardial infarction.

COMMENT:

The nature of this patient's lesion and his condition pre-operatively rendered death inevitable. If at all contributory, anaesthesia is considered to have been necessarily and unavoidably contributory.

CASE NO.	CLASSIFICATION Group	pREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
49.2.64	2	No comment	< 24	Myocardial ischaemia	No

Name: Jack Hardwick Age: 65 Sex: M Race: E

Disease: Acute vascular occlusion of right leg.

Operation: Right iliofemoral and popliteal thrombo-endarterectomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This patient, who had ECG evidence of previous myocardial infarction, developed acute vascular occlusion of the right leg 5 days after an emergency appendicectomy. 1 year previously he had had a right sided stroke, of which sequelae were evident. Some pulmonary emphysema was present. Heparin 125 mg. had been administered 3 hours before operation. Immediately before anaesthetic, B.P. was 130/90 mm.Hg.

PREMEDICATION:

Atropine 0.6 mg. given 50 minutes pre-operatively.

ANAESTHETIC:

After pre-oxygenation, anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 60 mg., oxygenation, topical analgesia of the larynx and oral intubation. On resumption of spontaneous respiration, dTc 20 mg. initial dose was administered and anaesthesia maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. During the course of the operation, which lasted 165 minutes, two further doses of dTc 10 mg. each were administered, the last being given 90 minutes before the end of the operation. During the procedure a further 50 mg. heparine was given. The course of anaesthesia was untoward.

At operation, iliofemoral embolectomy was performed followed by a thrombectomy of the posterior tibial artery. Blood was replaced as lost, by transfusion, a total of 1,200 ml. being transfused. At the conclusion of the operation, an infusion of Rheomacrodex was commenced. On discontinuance of anaesthetic, normal spontaneous respiration resumed and the patient rapidly regained consciousness and was completely rational. No antidote was considered necessary for the dTc given. One hour after return to the ward, the patient appeared to develop myocardial ischaemia and died suddenly.

AUTOPSY

No autopsy

COMMENT:

The only aspect of the anaesthetic management which requires comment was the omission of an antidote for curarisation. The total dose of dTc used was moderate for the length of the procedure and the last dose preceded the end of surgery by 90 minutes. Adequate spontaneous respiration resumed immediately atthe end of anaesthesia, and at no time before death was there any respiratory inadequacy. The anaesthetic and its management does not appear to have played any significant role in this patient's demise.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
50.2.64	3	No comment	ORD	Rupture of thoracic aortic aneurysm.	Yes

Name: Mentor Abrahams Age: 59 Sex: M Race: C

Disease: Dissecting thoracic aortic aneurysm. Initial diagnosis was small bowel obstruction) Operation: Laparotomy

Anaesthetic risk: 2, emergency

PRE-OPERATIVE STATE:

The patient presented with small bowel obstruction, vomiting, constipation and haematuria. Inaddition there was consolidation of the lower lobe of the left lung. Treatment was conservative - rehydration and gastric aspiration - for 12 hours. During this period the patient's B.P. remained at a level of 120/80 mm.Hg. At the end of this period in the absence of improvement, laparotomy was decided upon.

PREMEDICATION:

Morphine 10 mg., atropine 0.6 mg. given 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 400 mg., succinylcholine 50 mg., oxygenation and oral intubation. Anaesthesia was then maintained with nitrous oxide, oxygen and Halothane 0.5-1% administered via a carbon dioxide circle absorption system, by an IPPR technique. Gallamine 100 mg. was given as the relaxant. Laparotomy, lasting 45 minutes, was negative. The only observation of note made by the anaesthetist during the operation was that the B.P. remained elevated at a level of 200 mm.Hg systolic with a noticeably marked praecordial systolic thrust. At the conclusion of operation and anaesthetic, residual curarisation was adequately reversed with neostigmine 1.5 mg preceded by atropine 0.6 mg. Normal spontaneous respiration resumed and the patient regained consciousness rapidly. Immediately after being wheeled from the theatre, the patient suddenly became acutely distressed and cyanosed. He was immediately returned to theatre, re-intubated and IPPR with oxygen commenced. Cardiac arrest occurred. External cardiac massage was commenced immediately. The pupils remained small but, after 10 minutes, as spontaneous cardiac action had not returned, an open thoracotomy was performed to facilitate internal cardiac massage. On thoracotomy, a large amount of blood gushed out and exploration revealed rupture of a dissecting aneurysm of the thoracic aorta. Further resuscitative efforts were abandoned.

AUTOPSY

300 ml. blood in the left pleural cavity. Ruptured dissecting aneurysm of the thoracic aorta.

COMMENT:

Rupture of an aneurysm of the thoracic aorta caused this death. For the sake of uniformity, as death occurred immediately after the anaesthetic, while the patient was still in the operating theatre, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
51.2.64	3	No comment	< 24	Haemorrhage from oesophageal varices.	No

Name: Colin McGrath Age: 43 Sex: M Race: E

Disease: Haematemesis (from oesophageal varices)

Operation: Laparotomy. Gastric transection and splenectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient presented with grave haematemesis. In addition, there was gross distension of the abdomen from ascites and an enlarged, grossly cirrhotic liver. The profuseness of the haematemesis, the source of which was thought to be either a gastric ulcer or oesophageal varices, rendered it impossible to improve the patient's generally poor condition pre-operatively. He was in a state of marked oligaemic shock, the B.P. being 80 mm.Hg systolic, pulse rate 108/minute and he suffered respiratory distress because of marked abdominal distension. 6 pints blood had been transfused pre-operatively without improvement.

PREMEDICATION:

Atropine 0.6 mg. given 5 minutes pre-operatively.

ANAESTHETIC:

During induction of anaesthesia with cyclopropane and oxygen, profuse haematemesis occurred. The patient was immediately paralysed with succinylcholine 50 mg., the operating table was tipped head-down, the mouth and pharynx were aspirated clear of blood and oral intubation was performed. Bronchial toilette was performed immediately, no blood being obtained; thus no inhalation of blood had occurred. Auscultation of the lungs revealed clear breath sounds at this stage. Following intubation, blood continued to regurgitate up the oesophagus in torrents. This continued torrential bleed from the oesophagus identified the source of bleeding as from the varices. A balloon, cuffed tube was inserted down the oesophagus and was inflated, but was not effective in stemming the haemorrhage. dTc was administered (total dose throughout the operation being 30 mg.) and anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Laparotomy revealed a gross cirrhosis of the liver with ascites. Splenectomy and gastric transection were performed. Operative difficulties caused by adhesions and profuse haemorrhage from all operative surfaces, were immense. A clotting defect was apparent. Vitamin K and fibrinogen were administered with little effect. 14 pints blood, together with 8 gm. calcium gluconate were given during the operation. A dose of 1 mg./kg. body weight sodium bicarbonate was given to correct the possible metabolic acidosis.

Throughout operation, which lasted 4 hours, the B.P. remained at a level of between 80 and 100 mm.Hg systolic with a pulse rate of approximately 100/minute. At the conclusion of the procedure the patient was hypothermic, oesophageal temperature being 30°C. Following discontinuance of the anaesthetic, spontaneous respiration of a gasping character commenced and this was not improved by the administration of neostigmine 2 mg. preceded by atropine 1.2 mg. Though the respiration was of the type that would be seen with cerebral ischaemia, the pupils remained small. On removal of the endotracheal tube, there was evidence of some return of consciousness, the patient phonating, but profuse oesophageal bleeding continued. The patient was immediately re-intubated and IPPR instituted. On return to the ward, IPPR with air was provided by means of a Radcliffe ventilator.

Besides / ...

Besides the oesophageal bleeding, haemorrhage appeared to be continuing from the intra-abdominal surfaces as well, and 500 ml. blood exuded from the abdominal drain. Shortly after return to the ward, the patient's pupils commenced dilating and in spite of continued transfusionand IPPR, he died 2 hours post-operatively without fully regaining consciousness.

AUTOPSY:

No autopsy.

COMMENT:

This patient died of gross continuing haemorrhage, both from oesophageal varices and from the intra-abdominal operative site and adhesions. This continued bleeding was probably partly due to a clotting defect and high portal pressure, from gross cirrhosis of the liver. Anaesthesia is not considered significantly contributory to this death but, as the patient failed to regain consciousness before dying, and IPPR was necessary post-operatively, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
52.2.64	2	No comment	< 24	Post-operative ventricular fibrillation	Yes

Name: M. Swanepoel Age: 44 Sex: F Race: E

Disease: Aortic stenosis with mitral stenosis.

Operation: Aortic valve replacement with prosthesis and mitral valvotomy, on cardio-pulmonary bypass.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

This patient, who suffered stenosis of mitral and aortic valves, had been in congestive cardiac failure for some time. She had had intensive medical treatment and had undergone previous aortic valvotomy. Auricular fibrillation was present. B.P. 110/70 mm.Hg.

PREMEDICATION:

Seconal gr. 1½ orally, 2½ hours pre-operatively, atropine 0.6 mg. given 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinyl-choline 50 mg., oxygenation, topical analgesia of the larynx and oro-tracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen with Halothane 0.5-0.7%, administered by an IPPR technique via a carbon dioxide circle absorption system. No further relaxant was used. Pre-bypass the course of anaesthesia was relatively uneventful. During bypass, anaesthesia was maintained with Halothane 0.5% administered on the oxygen inflow of the bubble oxygenator. Bypass was conducted with a moderate hypothermia and coronary perfusion was used. At the conclusion of cardiopulmonary bypass, the heart was easily defibrillated. Post-bypass, anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique. Cardiac output and B.P. were maintained with an infusion of adrenaline 1:100,000.

At the conclusion of the anaesthetic, the patient rapidly regained consciousness and spontaneous respiration resumed. She died 4 hours post-operatively from ventricular fibrillation.

AUTOPSY:

Cardiotomy and aortotomy had been performed. Aortic valve prosthesis in situ. Valvotomy of mitral valve.

COMMENT.

The anaesthetic management is not considered to have been contributory to this death. In retrospect, death may have been due to hypokalaemia which is now known to follow prolonged bypass procedures in this type of case.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
53.2.64	2	No comment	< 24	Post-operative cerebral haemorrhage	Yes

Name: Nogolide Gagaza Age: 31 Sex: M Race: B

Disease: Posterior fossa tumour (Tuberuloma of vermis). Operation: Exploration of the posterior fossa.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had bilateral 6th nerve palsies and gross papilloedema. There were signs of cerebella dysfunction, especially on the left, but the patient was conscious. He was in extremely poor physical condition being extremely emaciated. B.P. was 130/90 mm.Hg and pulse rate 90/minute.

PREMEDICATION:

Atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., topical analgesia of the larynx and endo-tracheal intubation. Anaesthesia was then maintained with nitrous oxide, oxygen and ether for 15 minutes and subsequently Halothane, administered via a Magill semi-open circuit with spontaneous respiration. Operation was performed in the sitting position. ECG monitoring was conducted throughout. Blood was replaced by transfusion as lost, a total of 2 pints being given during the operation, which lasted 180 minutes. Cardiovascular homeostasis was normal throughout the procedure, the B.P. remaining at a level of between 110 and 130 mm.Hg systolic and the pulse rate at between 90 and 100/minute. During operation, the respiratory rate rose from 24/minute to 40/minute just before the end of the procedure.

At operation, a tuberculoma of the vermis and cerebellum was found. At the conclusion of surgery on discontinuance of the anaesthetic, the patient regained consciousness but was drowsy. There was no respiratory inadequacy. He died 24 hours post-operatively.

AUTOPSY:

Extensive tuberculoma of the vermis and cerebellum extending into subthalamic region. Post-operative haemorrhage into operative area. Extensive generalised abdominal tuberculosis.

COMMENT:

This patient died from post-operative haemorrhage at the operative site. Anaesthesia is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
54.2.64	2	No comment	< 24	Aneurysm of circle of Willis. Intra-cranial haemorrhage.	Yes

Name: Makhepmoe Xapa Age: 40 Sex: M Race: B

Disease: Subarachnoid haemorrhage. (Ruptured aneurysm of circle of Willis).

Operation: Carotid and vertebral angiography. Burrhole craniotomy. Insertion of Crutchfield clamp.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Poor. The patient had been comatose for 36 hours. Tracheostomy had been performed to enable tracheobronchial toilette to beperformed. Despite this, there was a gross amount of retained bronchial secretions and early bronchopneumonia was present. Rehydration and antibiotic therapy had been instituted pre-operatively. Immediately before anaesthesia the B.P. was 140/90 mm.Hg, pulse rate 110/minute and respiration 40/minute.

PREMEDICATION:

Atropine 0.6 mg. given 45 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide, oxygen and Halothane administered via the tracheostome from a Magill semi-open circuit with spontaneous breathing. During operation, the course of anaesthesia was punctuated by the occurrence of convulsions but cardiovascular and respiratory conditions remained static. At the conclusion of operation the patient remained comatose. Spontaneous respiration returned and was adequate. The patient died 1 hour after return to the ward.

AUTOPSY

Crutchfield clamp on right common carotid artery.. Widespread sub-arachnoid and intracerebral bleeding over the basal right temporal areas of the brain. Subdural haemorrhage around the circle of Willis. Aneurysm between internal carotid and middle cerebral arteries.

COMMENT:

This patient died as the result of subarachnoid haemorrhage from an aneurysm of the circle of Willis. The convulsions which occurred during anaesthesia were probably from a cerebral ischaemia which may have occurred during the application of the Crutchfield clamp. Anaesthesia is not regarded as contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
55.2.64	2	No comment	< 24	Haemorrhage	Yes

Name: Eliza Daniels Age: 61 Sex: F Race: C

Disease: Oesophageal varices. Cirrhosis of liver. Operation: Splenectomy (abandoned splenorenal shunt).

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

There was gross cirrhosis of the liver and oesophageal varices but the patient's general condition was fair. B.P. was 120 mm.Hg systolic. The heart and lungs were normal. Blood urea 92 mg.%, bilirubin 4.2 mg.%, alkaline phosphatase 15.5 units. One pint blood had been transfused 3 days pre-operatively. Haemoglobin 14.5 gm.%.

PREMEDICATION

Omnopon 10 mg., atropine 0.6 mg. given 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 250 mg., succinylcholine 25 mg., oxygenation, topical analgesia of the larynx, and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen with a trace of ether vapour, administered by an IPPR technique via a non-rebreathing circuit, using a Cyclator ventilator. dTc was given in divided doses, totalling 50 mg. during the 3¾ hours of the operation.

At operation, great difficulty was encountered technically with a gross amount of haemorrhage. Blood was replaced by transfusion as lost, 8 pints being administered. The B.P. remained stable throughout the procedure at a level of 110 and 140 mm.Hg systolic and the pulse rate at 90/minute. The operation originally intended - splenorenal shunt - was abandoned and splenectomy performed. At the conclusion of the operation residual curarisation was reversed with 2.5 mg neostigmine preceded by atropine 1.2 mg. Normal spontaneous and adequate respiration returned following discontinuance of anaesthesia and the patient rapidly regained consciousness. Grave intra-abdominal haemorrhage occurred post-operatively, 10 pints blood being transfused in the first 12 hours post-operatively. The patient died 22 hours after operation.

AUTOPSY

Gross cirrhosis of the liver. Oesophageal varices. Large intraperitoneal haemorrhage from splenic bed.

COMMENT:

This patient's death appears to be the result of continued haemorrhage from the operative site. Anaesthesia is not considered to have been contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
56.2.64	3	No comment	< 24	Peritonitis	No

Name: A. Nordien Age: 59 Sex: M Race: C

Disease: Post-gastrectomy. Anastomotic leak. Peritonitis. Operation: Laparotomy. Drainage of peritonitis.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Poor and apparently toxic. The patient had had a palliative gastrectomy for carcinoma of the stomach 6 days previously and had developed a leak from the gastric anastomosis. He now had peritonitis. Gross abdominal distension was present, causing embarrassment of respiration. B.P. 100/60 mm.Hg, pulse rate 108/minute. Rehydration therapy pre-operatively was thought to be adequate though urinary output for 12 hours pre-operatively had been poor.

PREMEDICATION:

Atropine 0.6 mg. administered 30 minutes pre-operatively.

ANAESTHETIC:

Following induction of anaesthesia with cyclopropane and oxygen, oral intubation was performed after administration of succinylcholine 50 mg. Anaesthesia was then maintained with nitrous oxide and oxygen, by an IPPR technique via a non-return system utilizing a cyclator ventilator. dTc 15 mg. was administered. The course of anaesthesia was untoward. At the conclusion of operation, which lasted 80 minutes, spontaneous respiration returned, manifesting a tracheal tug. The administration of neostigmine 2.5 mg. preceded by atropine 1.2 mg. led to no improvement in the respiratory pattern. The minute volume of respiration, measured as 4 litres/minute was considered inadequate. A tracheostomy was performed and IPPR with 30% oxygen and air was continued, using a mechanical ventilator, during the post-operative period. The patient rapidly regained consciousness after discontinuance of the anaesthetic. Circulatory collapse occurred and the patient died, 15 hoursafter the operation.

AUTOPSY

No autopsy

COMMENT:

Assessment of the contributory role of anaesthesia in this patient's death is difficult. There was respiratory inadequate pre- and post-operatively. The dose of curare used was moderate for the length of the operation. If we conclude that this post-operative respiratory inadequacy was due to the use of a relaxant drug with anaesthesia, the subsequent treatment was correct and effective. Furthermore, as the patient survived 15 hours post-operatively, it is likely that the dose of curare used would have been metabolised by then. The gross peritonitis from which the patient suffered would provide adequate reason for his death.

What may be criticised by today's standards was the omission of correction of the probable metabolic acidosis, from which the patient must have suffered post-operatively. Peritonitis is regarded as the cause of death but, as respiratory inadequacy followed anaesthesia - though correctly and efficiently treated - anaesthesia is regarded as having been necessarily and unavoidably contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
57.2.64	2	No comment	< 24	Cerebral oedema.	No

Name: Richard Fanshaw Age: 12 Sex: M Race: C

Disease: Acute mastoiditis. Subdural and ventricular empyema. Operation: Mastoidectomy. Carotid angiography. Burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Stuperose and pyrexial, temperature 104°F. B.P. 120/70 mm.Hg. Pulse rate 100/minute. Haemoglobin 12 gm.%.; this had been lower 2 days previously and 1 pint blood had been transfused.

PREMEDICATION:

Atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane administered via a Magill circuit with spontaneous breathing. Oral intubation was performed after topical analgesia of the larynx. Halothane was discontinued and a trace of ether vapour given in its place. After 12 minutes' anaesthesia, respiration suddenly became shallow and rapid. At the same time the B.P. rose from 120 to 160 mm.Hg systolic, and the pulse rate accelerated sharply to 180/minute. One half minute after the onset of shallow respiration, IPPR was instituted via a circle absorption system, and continued for the remainder of the operation. Anaesthesia was now maintained with nitrous oxide and oxygen alone. After 20 minutes the B.P. had subsided to 110 mm.Hg systolic and the pulse rate to 120/minute, and both remained at about this level during the remainder of the procedure. After mastoidectomy it was noticed that the left pupil was larger than the right. Carotid angiograp hy was now performed followed by burrhole craniotomy, which revealed a large subdural and ventricular empyema with marked oedema of the brain. Blood was transfused as lost during the operation, a total of 300 ml. being given.

Following operation, which lasted 4¾ hours, spontaneous respiration did not recommence. IPPR with air was provided by a Radcliffe ventilator, via the endotracheal tube, which was left in situ. The patient died 17 hours post-operatively without regaining consciousness.

AUTOPSY

No autopsy

COMMENT:

Existing cerebral pathology caused this death. Although respiratory failure occurred while the patient was anaesthetised, the failure is thought to have been primarily due to the cerebral oedema, which may have been worsened by anaesthesia. However, no episode of anoxia or hypercarbia were apparent during anaesthesia and, in the circumstances, anaesthesia is not considered to have been a significant factor contributing to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
58.2.64	2	No comment	< 24	?Cerebral oedema.	No

Name: H. Page Age: 40 Sex: M Race: C

Disease: Left temporal lobe tumour.

Operation: Craniotomy. Temporal lobectomy (tumour histologically astrocytoma).

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Confused, restless and with marked clouding of consciousness. The patient was aphasic and had gross papilloedema. General physical state was good except for essential hypertension. B.P. 190/100 mm.Hg.

PREMEDICATION

Atropine 0.6 mg. given 45 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 400 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. A Pulmomat ventilator was used. dTc was administered in divided doses, totalling 65 mg. during the 3½ hours for which the operation lasted. Following induction of anaesthesia, before the commencement of surgery, 90 gm. urovert was administered by intravenous injection, causing a rise in B.P. from the pre-operative level of 180 mm.Hg systolic to 200 mm.Hg systolic. The B.P. returned to the pre-anaesthetic level after 40 minutes. During operation, blood was transfused as lost (approximately 3 pints). The general course of anaesthesia was untoward.

At the conclusion of the operation residual curarisation was reversed with neostigmine 2.5 mg. preceded by atropine 1.2 mg. and normal and adequate spontaneous respiration resumed. Consciousness was not fully regained on discontinuance of the anaesthetic. Approximately 12 hours post-operatively, respiration became sterterous and the level of consciousness became progressively deeper. The patient died 16 hours after operation.

AUTOPSY

No autopsy.

COMMENT:

Anaesthesia does not seem to have been contributory to this death which resulted from the effects of the operative procedure.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
59.2.64	2	No comment	<24	Subarachnoid haemorrhage. Intracerebral haematoma.	Yes

Name: Herman Nipper Age: 58 Sex: M Race: E

Disease: Intracerebral and sub-arachnoid haemorrhage. Operation: Craniotomy. Evacuation of haematoma.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund. Temperature 102°F. Respiration was stertorous. B.P. was maintained at a level of 140/75mm.Hg by means of a noradrenaline drip infusion. Coarse rales were audible over both lung fields. A tracheostomy had been performed to facilitate tracheobronchial toilette. Blood urea was 358 mg.%.

PREMEDICATION:

Atropine 0.6 mg. administered 40 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide and oxygen inhalation, administered via the tracheostome from a Magill semi-open circuit with spontaneous breathing. The patient's condition remained unchanged throughout craniotomy, which lasted 55 minutes. He died 7 hours post-operatively without regaining consciousness.

AUTOPSY

There was a ruptured aneurysm of anterior cerebral artery with subarachnoid and intracerebral haematoma.

COMMENT:

Death was the result of the patient's pre-existing disease.

CASE NO.	CLASSIFICATION Group	BREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
60.2.64	2	No comment	< 24	Myocardial infarction	No

Name: George Manusi Age: 61 Sex: M Race: E

Disease: Perforated duodenal ulcer. Operation: Laparotomy. Resuture of perforated duodenal ulcer.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Very ill patient with vasoconstriction. There was a tachypnoea of 36/minute. B.P. 120/80 mm.Hg. Pulse rate 92/minute. Adequate rehydration therapy had been performed. A 2 finger hepatomegaly was palpable. The patient had had a myocardial infarction 1 year previously and was currently on anticoagulant therapy (Dindevan).

PREMEDICATION:

Hyoscine 0.3 mg., given 55 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 50 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was then maintained throughout the operation with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. A single dose of dTc, 15 mg., was given. The course of anaesthesia was untoward. At the conclusion of operation, which lasted 35 minutes, residual curarisation was reversed with neostigmine 1.5 mg preceded by atropine 1.2 mg. Normal and adequate spontaneous respiration resumed. On discontinuance of the anaesthetic, the patient rapidly regained consciousness. The post-operative course was uneventful until 12 hours after surgery, when the patient appeared to suffer a myocardial infarction and died.

AUTOPSY

No autopsy

COMMENT:

Although there is no proof, by autopsy, this death appears to have been, clinically, the result of a myocardial infarction. The history of a previous infarct is a point in favour of such a diagnosis. Anaesthesia per se does not appear to have been contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
61.2.64	1	Possibly	> 24	Cardiac arrest. Anoxic anoxia. Pneumonia.	No

Name: Emily Singsing Age: 15 months Sex: F Race: C

Disease: Acute mastoiditis. Operation: Mastoidectomy (abandoned).

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Extremely poor. In addition to acute chronic mastoiditis (pyogenic or tuberculous) there was a right middle lobe lung abscess, probably tuberculous, with consolidation of the surrounding lung. Temperature 101°F. Pulse rate 110/minute, respiration 30/minute. She was malnourished and anaemic, haemoglobin being 8.5 gm.%. No pre-operative blood transfuions or rehydration therapy had been undertaken as difficulty was experienced in establishing an intravenous infusion route. It was thought that this might be better done while the patient was anaesthetised.

PREMEDICATION:

Atropine 0.3 mg., given 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane up to a concentration of 2%, administered via an infant T-piece circuit with face piece, the patient breathing spontaneously. After topical analgesia of the larynx, oral intubation was performed, and anaesthesia maintained with nitrous oxide and oxygen, with 1% Halothane, via an Ayre's T-piece, the patient breathing spontaneously. B.P. was not monitored but the femoral pulse was palpated, and was of good volume and the colour of the mucous membranes was normal during induction of anaesthesia, which lasted 10 minutes. An intravenous drip infusion was established. When operation commenced, cyanosis of the blood was noticed in the operative wound. No pulse was palpable and respiration was observed to have ceased. Cardiac arrest was diagnosed. IPPR with oxygen was immediately instituted and thoracotomy confirmed the presence of cardiac arrest in asystole. Cardiac massage through the pericardium was commenced immediately and, after 10 minutes of this, normal spontaneous heart beat was restored. After closure of the thorax, normal spontaneous respiration resumed and the pupils, which had dilated, became small. During thoracotomy, 125 ml. compatible blood was transfused. The operation of mastoidectomy was abandoned. The patient did not regain consciousness and convulsions occurred in the post-operative period. During these, cyanosis occurred. Six hours post-operatively, cardiac arrest occurred during a convulsion. External cardiac massage and IPPR restored spontaneous cardiac rhythm again. Some 6 hours later, a second cardiac arrest occurred, in similar circumstances, and again external cardiac massage and IPPR restored the spontaneous cardiac rhythm and spontaneous respiration. A tracheotomy was now performed, to permit efficient tracheobronchial toilette. The patient appeared to regain consciousness 24 hours after this episode. The pneumonic consolidation in the right lung worsened and bronchopneumonic changes commenced in the left lung. The further course was one of worsening bronchopneumonia with profuse bronchial secretions. Humidification of the inspired air was not efficient and inspiration and crusting of the secretions rendered efficient removal difficult. No further surgical attack was contemplated on the mastoid, as the patient's general condition did not appear to warrant this. Furthermore, the mastoiditis did not worsen, appearing to improve somewhat on antibiotic therapy. The patient died 12 days after operation. The immediate cause of death was pneumonia and secretional bronchial obstruction, resulting in anoxic anoxia.

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AUTOPSY

No autopsy

COMMENT:

Although this baby's death ultimated resulted from pneumonia and secretional bronchial obstruction, the post-operative sequence of events followed cardiac arrestwhich occurred shortly after the induction of a general anaesthetic. In adjudging the contributory factors to this death, one must examine the part played by the anaesthetic management in this original cardiac arrest.

This child was an extremely poor operative and anaesthetic risk. The failure to rehydrate and correct the anaemia of this sick child before surgery and anaesthesia must be faulted. A venous cut-down should have been performed following failure of attempts to establish veneclysis by a push-in method.

Although the anaesthetist reports no difficulty during the actual induction of anaesthesia, no efficient tracheobronchial toilette was performed following intubation, despite the known pulmonary pathology. That the precise moment of cardiac arrest seems to have escaped the observation of the anaesthetist must also be regarded as a fault. During the moments immediately before commencement of operation, the anaesthetist's attention was engaged by his attempts to establish intravenous infusion. The first untoward sign noticed was cyanosis of the blood in the operative wound - and only then were the concomittant signs of cardiac arrest, absence of pulse and cessation of respiration observed. The precise cause of the cardiac arrest is unknown. In view of the pulmonary pathology, the fact that no tracheo-bronchial toilette was undertaken, and the known respiratory depression produced by Halothane anaesthesia, it is probable that anoxic anoxia from respiratory depression and/or secretional bronchial obstruction was the main causative factor. In addition, the patient was anaemic and dehydrated. Both the anaemic anoxia of the former and the decreased cardiac output of the latter, especially in the presence of Halothane anaesthesia, would have further decreased the "available oxygen" in the presence of an increased oxygen demand, because of pyrexia.

For these reasons, despite the grave illness of the child before anaesthesia, the anaesthetic management is regarded as a significant factor in the causation of this cardiac arrest. Once diagnosed, the treatment of the cardiac arrest was prompt, conventional and effective. The cardiac arrest which occurred in the post-operative period was doubtless the result of anoxia, both because of the convulsions - themselves a sequel of the cerebral ischaemia during the operative period of cardiac arrest - and probably secretional bronchial obstruction. The subsequent worsening of the pneumonia and the difficulties with bronchial obstruction from inspirated secretions followed. Some of these difficulties may have been the result of poor and inconsistent humidification of the inspired air breathed by the patient through a tracheostome.

PREVENTABILITY:

Although the precise cause of the original cardiac arrest is not known, three preventable errors in the management are apparent:

(1) lack of pre-operative rehydration therapy and transfusion;

(2) lack of tracheo-bronchial toilette following intubation in the fact of known pulmonary pathology;

(3) lack of clinical observation of the patient following induction of anaesthesia.

This death is thus regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
62.2.64	2	No comment	< 24	Carcinoma of the oesophagus. Pneumonia.	No

Name: Nolast Pendu Age: 58 Sex: F Race: B

Disease: Carcinoma of the oesophagus.

Operation: Oesophagoscopy. Gastrostomy. Insertion of Celestine tube.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient was in extremis. A large carcinoma of the oesophagus had eroded into the bronchi, causing a broncho-oesophageal fistula. B.P. was 80/60 mm.Hg and pulse rate 100/minute. Rehydration therapy had been undertaken before anaesthesia.

PREMEDICATION:

Atropine 0.6 mg. given 45 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 100 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx, and oral intubation, and was then maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. Intermittent doses of succinylcholine were given up to a total dosage of 70 mg. during the operation, which lasted 1 hour. The course of anaesthesia was uneventful.

At the conclusion of the operation, on discontinuance of anaesthesia, the patient rapidly regained consciousness, and normal spontaneous adequate respiration was resumed. She died 22½ hours post-operatively from causes unrelated to the anaesthetic.

AUTOPSY

No autopsy.

COMMENT:

This death was due to the patient's pre-existing disease. Anaesthesia is not considered to have been contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
63.2.64	3	No comment	> 24	Peritonitis. Cardiac arrest.	No

Name: A. Jacobs Age: 18 Sex: M Race: C

Disease: Perforated peptic ulcer with generalised peritonitis. Operation: Laparotomy. Closure of perforation and drainage of peritonitis.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted to hospital 48 hours after perforation of a peptic ulcer, gravely ill if not moribund, from generalised peritonitis and dehydration. Gastric aspiration and rehydration with replacement fluid and plasma was undertaken. After 1½ litres plasma and 1 litre fluid had been administered, serum electrolytes concentrations were potassium 5.1 m.Eq./l., sodium 130 m.Eq./l., chloride 84 m.Eq./l. and bicarbonate 24.2 m.Eq./l. The abdomen was distended and on X-ray the diaphragm, under which was air, was elevated and there was compression of the lung fields. Immediately before anaesthesia, cyanosis with tachypnoea and markedly reduced respiratory excursion was present. There was a persistent tachycardia of 140/minute. B.P. was 120 mm.Hg systolic. Intravenous therapy with the antibiotics Reverin and chloromycetin had been commenced.

PREMEDICATION:

Atropine 0.6 mg. given intravenously immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 30 mg. was injected and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. A single dose of dTc 10 mg. was administered. Throughout the operation, which lasted 1 hour, B.P. was maintained at a level of 110 mm.Hg systolic. Pulse rate, initially 140/minute, slowed to 120/minute and stabilised at this level.

Laparotomy revealed a large duodenal perforation and 4 pints bile-stained pus in the peritoneal cavity, which was aspirated, and the perforation sutured. At the conclusion of the operation, 2 mg. neostigmine preceded by atropine 0.6 mg. was given to reverse any residual curarisation. Spontaneous respiration returned but was of inadequate volume, tidal volume measuring 150-200 ml. The patient became cyanosed on breathing air. Colour improved slightly on breathing oxygen and the patient regained consciousness. Nikethamide 2.5 ml. administered intravenously produced some improvement of the tidal volume, to 250 ml., which was considered inadequate and IPPR with oxygen was instituted, via a nasal endotracheal tube. After 30 minutes, spontaneous respiration was again permitted. The patient had good muscle power all this time and could move both legs and arms. 90 minutes post-operatively, 30 ml. 4% sodium bicarbonate solution was administered to commence correction of possible metabolic acidosis. While this was being given, cardiac arrest occurred. IPPR was commenced immediately and spontaneous heart beat resumed following external cardiac massage. After commencement of the heart beat, the pupils which had dilated rapidly became small again. IPPR with air and oxygen was continued, using a Cyclator ventilator. Biochemical investigation revealed a metabolic acidosis, with base deficit of 10 m.Eq./l., which was corrected by the administration of further sodium bicarbonate until further estimation revealed the reduction of the base deficit to 2 m.Eq./l.

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4½ hours post-operatively the patient breathed adequately with a tidal volume of 450 ml. and the B.P. was steadily maintained at 110 mm.Hg systolic. After a further 45 minutes' observation in the operating theatre, the heart arrested a second time. On this occasion, external cardiac massage did not appear adequate. Left thoracotomy and internal cardiac massage were performed. The heart (asystolic on thoracotomy) commenced beating after some 5 minutes of cardiac massage, but the patient failed to regain consciousness or resume spontaneous respiration. IPPR was maintained by means of the Cyclator ventilator, initially via the endotracheal tube and later via a tracheostomy. The patient died 1 week post-operatively from the result of cerebral anoxic damage and bronchopneumonia.

AUTOPSY

No autopsy.

COMMENT:

This patient was gravely ill, if not moribund, from peritonitis before anaesthesia. Surgical opinion is that submitting this patient to formal laparotomy and suture of the perforated ulcer while in this critical state, so long after the original perforation, was an error of judgement. A peritoneal drain, inserted through a small incision under local anaesthesia, would have sufficed.

Respiratory inadequacy, present before, was also present after the anaesthesia. This inadequate respiration was correctly treated and ultimately improved. It is unlikely that it was related solely to the administration of a relaxant drug during anaesthesia.

At the time when the first cardiac arrest occurred, 100 minutes post-operatively, only 15 m.Eq. of sodium bicarbonate had been given. This first arrestmay well have been due to metabolic acidosis. Treatment of the arrest was prompt and effective. The metabolic acidosis was subsequently rapidly corrected. The cause of the second cardiac arrest is difficult to assess. During this arrest, irreversible anoxic cerebral damage occurred which, ultimately, caused the patient's death.

On balance, this death basically is due to the patient's disease, his moribund condition pre-operatively rendering death almost inevitable. Anaesthesia may be regarded as having been unavoidably necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
64.2.64	3	No comment	ORD	Haemorrhage.	Yes

Name: Albertus Nieuwort Age: 1½ Sex: M Race: E

Disease: Occipital tumour (melanotic adamantinoma of skull). Operation: Craniotomy. Excision of tumour.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This child, who weighed 20 lb., had a large vascular tumour the size of a man's fist, on the occipital region of the skull. Except for this, the child was well.

PREMEDICATION:

Atropine 0.2 mg. intramuscularly 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide, oxygen and Halothane, administered via an infant T-piece system. After topical analgesia of the larynx, oral intubation was performed, and anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique via the T-piece system. Intravenous infusion was established at two sites, in anticipation of severe blood loss. Despite this, blood transfusion could not keep pace with blood loss during resection of the tumour. The transverse sinus was opened. Rapid haemorrhage of approximately 300 ml. blood (almost 40% of the child's estimated total blood volume) caused cardiac arrest. External cardiac massage was commenced, with poor effect. After 7 minutes, a left thoracotomy was performed for internal cardiac massage. Cardiac filling was poor and thus 100 ml. blood was administered directly into the left ventricle. All resuscitative measures were of no avail.

AUTOPSY:

Scalp, skull and dura mata had been excised from an oval area, 4 x 2½ inches, on the left side of the occipital region. Remains of a greyish-black tumour were still present in the dura mata. The brain and cerebellum were flattened on the left side. A 3½ inch surgical wound was present in the left side of the thorax. Left lung collapsed. There was extensive haemorrhage in the wall of the left ventricle.

COMMENT:

Cardiac arrest and death followed uncontrollable haemorrhage during the surgical procedure. Blood could not be replaced rapidly enough by transfusion, despite the use of two infusion sites. As death occurred while the patient was anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
65.2.64	2	No comment	< 24	?Myocardial ischaemia.	Yes

Name: Ben Johannes Age: 84 Sex: M Race: C

Disease: Prostatic adenoma. Urinary obstruction. Operation: Transurethral resection of prostate.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Besides prostatic urinary obstruction, this patient had marked arteriosclerosis and cirrhosis of the liver. His general condition was considered fair.

PREMEDICATION:

Atropine 0.6 mg. given by intramuscular injection 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation, and was then maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. A single dose of dTc 10 mg. was administered. The course of operation and anaesthesia were trouble-free.

Residual curarisation was reversed at the conclusion of operation by administration of neostigmine 2 mg. preceded by atropine 0.6 mg. Normal spontaneous respiration resumed and the patient regained consciousness. He died suddenly 23 hours post-operatively from what appeared clinically to be myocardial ischaemia.

AUTOPSY

Small retroperitoneal haemorrhage in the pelvis. Partial removal of prostate. Both lungs showed evidence of tuberculous lesions in the upper lobes. Marked left ventricular hypertrophy of the heart, with widespread atherosclerosis of coronary arteries and the aorta. Small and cirrhotic liver. Small spleen. Both kidneys showed signs of glomerular nephritis, were small and wrinkled with capsules which stripped with difficulty.

COMMENT:

Anaesthesia was not contributory to this death. In view of the severe coronary artery disease, one may surmise that the myocardial ischaemia was the result of hypotension, perhaps through haemorrhage. This type of death is not uncommon shortly after surgery in patients with ischaemic heart disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
66.2.64	2	No comment	< 24	Peritonitis. Bronchopneumonia	Yes

Name: Archibald Dziba Age: 43 Sex: M Race: B

Disease: Dehiscence of abdominal wound. Generalised peritonitis.

Operation: Laparotomy. Resuture of abdominal wound.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This obese patient, weighing 250 lb., had been admitted 5 days before with a penetrating stab wound of the abdomen, sustained 12 hours before admission. There was a history of unconsciousness and vomiting before admission. He was a diabetic and, on admission, had gross glycosuria and ketonuria. After rehydration and insulin therapy, laparotomy was performed under general anaesthesia. There was no damage to bowel but local peritonitis was present and the abdomen was sutured, drainage being instituted. The course of anaesthesia was uneventful and there was no vomiting. Breath sounds were normal post-operatively. After operation a severe bronchopneumonia with klebsiella and other mixed organisms developed. Gross glycosuria persisted despite 20 units insulin 3 times daily, though the ketonuria was controlled. Bronchopneumonia worsened as did the signs of peritonitis. On the 5th post-operative day the abdominal wound dehisced. At this time the patient was in extremely poor condition, very restless and disorientated. Gross respiratory distress was present, probably from bronchopneumonia, and splinting of the diaphragm because of abdominal distension. A tracheal tug was present and the patient was cyanosed on breathing air.

PREMEDICATION:

Omnopon gr. 1/3 and atropine 0.6 mg. administered 2 hours pre-operatively.

ANAESTHETIC:

Thirty minutes before anaesthesia, severe circulatory collapse occurred. The B.P. became unrecordable. 500 ml. plasma, 20 units insulin and 30 gm. glucose were rapidly administered and the B.P. rose to 95 mm.Hg systolic with a marked tachycardia of 150/minute.

Anaesthesia was induced with cyclopropane and oxygen and oral intubation performed. After tracheo-bronchial toilette, anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique via a circle carbon dioxide absorption system. A single dose of gallamine 80 mg. was given. After induction of anaesthesia the B.P. became unrecordable. ECG monitoring continued to show normal sinus rhythm, but with tachycardia. There was little effect on administration of phenylephrine in divided doses, totalling 2 mg. 200 mg. cortisone was given together with rapid transfusion of plasma. The B.P. recovered to 100 mm.Hg systolic but the tachycardia remained at a level of 140/minute.

At laparotomy, pus was found in the peritoneal cavity and was drained. At the conclusion of the operation, which lasted 75 minutes, spontaneous respiration resumed. Neostigmine 2.5 mg preceded by atropine 1.2 mg. was administered to reverse any residual curarisation. Respiration of similar tidal volume and pattern to that existing pre-operatively resumed. On breathing air, cyanosis occurred. Marked abdominal tension persisted. A tracheostomy was performed and a patient-triggered mechanical pulmonary ventilator with 40% oxygen in air, was instituted using a Bird ventilator, at a tidal volume of 400 ml. and a minute volume of 13 litre/minute. The patient recovered consciousness and maintained his condition for some time post-operatively, when the B.P. again commenced to fall. The patient died 7 hours after operation.

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AUTOPSY

Diffuse peritonitis. Bilateral bronchopneumonia.

COMMENT:

Death resulted from the effects of generalised peritonitis and bronchopneumonia. Although there was respiratory inadequacy after the second operation and anaesthetic, this was similar to that present before anaesthesia and the administration of a relaxant drug. The correct treatment was adopted. The anaesthetic at the second operation is not regarded as contributory to the patient's death. However, one must question the possible part played by the anaesthetic at the first operation in the causation of the bronchopneumonia which developed post-operatively. If there is any association, this is not obvious from the clinical records. The lungs were clear on auscultation, both before and after the first anaesthetic, during which there was no vomiting or apparent regurgitation. Although it is possible that it may have been contributory, there is no concrete evidence to indicate any contributory role of the anaesthetic management during the first operation in the development of the bronchopneumonia.

The patient's death resulted from the existing disease and the anaesthetic he received is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
67.2.64	2	No comment	<24	Peritonitis.	No

Name: Ntembe Stubi Age: 22 Sex: M Race: B

Disease: Fulminating peritonitis. Lobar pneumonia. Operation: Laparotomy. Drainage of peritonitis.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund and semi-comatose following intestinal obstruction and ileus of long duration. Signs of gross peritonitis. In addition there was a left lobar pneumonia; respiration 88/minute. A ventricular septal defect was present. After rehydration therapy, biochemical investigation (Astrup technique) showed a gross metabolic acidosis, the base excess being 12 m.Eq./litre, which was corrected pre-operatively with 240 m.Eq. sodium bicarbonate. After this treatment the respiration dropped to 60/minute. Antibiotic therapy was instituted intravenously. B.P. immediately pre-anaesthetic was 120 mm.Hg systolic.

PREMEDICATION:

Atropine 0.6 mg. intravenously immediately before operation.

ANAESTHETIC:

Anaesthesia was induced and maintained throughout the procedure with cyclopropane and oxygen, administered by a closed circuit technique. Succinylcholine 50 mg. was administered and oral intubation performed. A short period of IPPR was instituted. Spontaneous respiration was permitted throughout the laparotomy but IPPR was adopted during the closure of the abdomen, when a further dose of succinylcholine 50 mg. was given.

Laparotomy revealed gross peritonitis which was drained. The course of anaesthesia was uneventful during the procedure, which lasted 1 hour. At the conclusion of the anaesthetic, respiration was spontaneous and adequate, but still fast - 40/minute. The colour of mucous membrane was good. The patient recovered consciousness to the same level as had been present pre-operatively. He died 10 hours post-operatively.

AUTOPSY

No autopsy.

COMMENT:

This patient died of the effects of fulminating peritonitis. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
68.2.64	3	No comment	ORD	Cardiac arrest, (cardiac handling).	Yes.

Name: Gloria Jacobs Age: 26 Sex: F Race: C

Disease: Mitral stenosis Operation: Mitral valvotomy

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was known to have severe mitral stenosis and suffered a cardiac arrest while attending the hospital Out-Patient Department. External cardiac massage was not successful so left thoracotomy and internal cardiac massage were performed. IPPR, initially by expired air ventilation, was subsequently applied via an endotracheal tube by an inflating bellows. Spontaneous heart beat resumed in auricular fibrillation. The pupils which had dilated became small again. Some signs of returning consciousness was apparent. Inview of the severity of the mitral stenosis, it was decided to perform an emergency mitral valvotomy.

PREMEDICATION: Nil.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. A single dose of dTc 15 mg. was given. At this stage the B.P. was 110 mm.Hg systolic. ECG monitoring was instituted, which showed auricular fibrillation and signs of full digitalisation. At the outset of operation, 48 m.Eq. sodium bicarbonate and 50 gm. glucose were administered by intravenous infusion, followed by 100 ml. 10% Mannitol. Transfusion of compatible blood was commenced just prior to atriotomy. Multifocal ventricular extrasystoles with short runs of ventricular tachycardia followed handling of the heart. Pronethalol 10 mg. (0.2 mg./kg body weight) was administered in an effort to control this. When the mitral valve was split, with a Tubb's dilator, cardiac arrest in asystole occurred. Cardiac massage was commenced immediately but asystole persisted. Rapid intravenous infusion of adrenaline 1:100,000 caused ventricular fibrillation. Electrical defibrillation resulted in asystole which proved refractory to the further administration of adrenaline, isoprenaline, calcium chloride and sodium bicarbonate. The myocardium remained flabby and toneless. After 55 minutes of cardiac massage the patient's pupils commenced dilating, indicating severe cerebral anoxia. Cardiac massage was then abandoned.

AUTOPSY

Severe tight mitral stenosis.

COMMENT:

This death was primarily due to the patient's disease and the surgical procedure undertaken in rather desperate circumstances. In retrospect, one wonders if the ultimate completely refractory state of the myocardium was not due, in part, to the administration of Pronethalol. Perhaps the administration of potassium mayhave been wiser. However, this is hypothesis. Anaesthesia is considered to have been no more than necessarily contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
69.2.64	2	No comment	< 24	Cerebral infarction	No

Name: Gasant Miller Age: 50 Sex: M Race: C

Disease: Cerebral infarction. Operation: Craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Unconscious. The patient was rousable by very painful stimuli. Carotid angiography was performed, without anaesthetic, and showed what was thought to be an intracranial tumour.

PREMEDICATION:

Atropine 0.6 mg. administered 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced and maintained with nitrous oxide and oxygen administered via a Magill semi-open circuit. Succinylcholine 50 mg. was given and oral intubation performed, followed by tracheo-bronchial toilette, and a period of IPPR. Subsequently the patient breathed spontaneously.

There was no change in the patient's general condition during the craniotomy which revealed a cerebral infarct. Following operation the patient failed to regain consciousness and died 6 hours later.

AUTOPSY

No autopsy.

COMMENT:

Death was due to cerebral infarction. The anaesthetic is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
70.2.64	1	Possibly	< 24	Post-relaxant respiratory inadequacy. Circulatory failure.	Yes

Name: Eloise Knaggs Age: 69 Sex: F Race: E

Disease: Acute pancreatitis (diagnosed as intestinal obstruction). Operation: Laparotomy.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

The patient presented with what appeared to be small bowel obstruction. After gastric aspiration and rehydration therapy, using 1 litre plasma and 1 litre normal saline, her general condition was good. B.P. was 130/90 mm.Hg. Respiration was normal. The only disquieting sign was a tachycardia of 120/minute. The anaesthetist considered her a 'fair' anaesthetic risk.

PREMEDICATION:

Atropine 0.3 mg., given immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen and oral intubation was performed after injection of succinylcholine 40 mg. and a period of IPPR. Anaesthesia was maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide absorption system. An initial dose of dTc 20 mg. was given, followed by further doses of 4 mg. and 2 mg. (total 26 mg.) during the procedure, which lasted 75 minutes.

Laparotomy revealed acute pancreatitis. The course of anaesthesia was uneventful but, at the conclusion, normal spontaneous respiration did not return. Neostigmine 2.5 mg preceded by atropine 1.2 mg. produced inadequate reversal of curarisation; some respiratory activity returned but was inadequate, characterised by a tracheal tug. Further doses of neostigmine, totalling 1.5 mg., produced no improvement. Suspecting that metabolic acidosis might be responsible, the anaesthetist administered 35 m.Eq. sodium bicarbonate. Although there was some muscular activity, the patient did not fully regain consciousness. The endotracheal tube was left in situ and IPPR continued. One hour after conclusion of the operation, the patient was returned to the ward, where IPPR with 30% air in oxygen was provided by means of a Cyclator ventilator. The patient was permitted to trigger the ventilator. This resulted in unsatisfactory pulmonary ventilation with marked lack of synchrony between ventilator and patient, the appearance described as 'the patient fighting the pump'. After these conditions had persisted for a while, circulatory failure supervened and the patient died, 12 hours post-operatively.

AUTOPSY

Widespread arteriosclerosis in main blood vessels. Fat necrosis in abdomen and signs of acute pancreatitis.

COMMENT:

This patient had normal respiration before anaesthesia. Following what appears to have been an uneventful anaesthetic, involving a moderate dose of dTc, this patient presented post-relaxant respiratory inadequacy which did not respond to the administration of neostigmine ('neostigmine resistant curarisation'). Many reasons have been advanced for this state. Suspecting rightly, in the presence of acute pancreatitis, that a possible reason was metabolic acidosis, the

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anaesthetist administered sodium bicarbonate in a dose which was totally inadequate. The degree of metabolic acidosis that occurs in these circumstances, which is sufficient to cause respiratory inadequacy or to prolong the effects of curare, is usually gross. The amount of sodium bicarbonate administered here would only have provided for the neutralisation of base excess of -1.5 m.Eq/l., a homeopathic dose in the circumstances.

The correct treatment of post-relaxant respiratory inadequacy is the continued provision of adequate IPPR. The type of ventilator used for this purpose in this case has properties which make it unsuitable for patient-triggering. The response time is slow and triggering mechanism not sensitive enough, and the inflow rate of gas cannot be altered. This causes (especially in patients with tachpnoea) a type of paradoxical respiration between the ventilator and the patient, the former lagging behind the patient's demand. The condition most adequately described as 'the patient fighting the pump' is produced. The only way to overcome this, especially with a ventilator of this type, is not to permit patient-triggering. This may be achieved by hyperventilation and depression of the patient's respiratory centre with opiates until adequate control of ventilation is achieved. Continuation of this 'paradoxical' type of respiration causes exhaustion of the patient, inadequate ventilation and grave disturbance of circulatory dynamics. Such is what, one assumes, occurred in this case.

General anaesthesia and the use of a relaxant drug and its sequelae are regarded as significant contributory factors to this patient's death.

It is of interest to note that, of five cases in the 1st period survey who displayed post-anaesthetic respiratory inadequacy, two suffered acute pancreatitis.

PREVENTABILITY:

Because of the faulty application of mechanical pulmonary ventilation and the inadequate treatment of the probably severe metabolic acidosis this death is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
71.2.64	2	No comment	< 24	Circulatory failure.	No

Name: Pieter Pienaar Age: 70 Sex: M Race: C

Disease: Haematemesis from bleeding gastric ulcer. Operation: Partial gastrectomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient presented with severe haematemesis and suffered, in addition, congestive cardiac failure from cor pulmonale, gross pulmonary fibrosis of the right lung and diabetes - there being both glycosuria and ketonuria. He was digitalised and blood transfusion was commenced. The administration of 50 units of insulin caused the disappearance of ketonuria, but glycosuria persisted. Immediately before anaesthesia, B.P. was 100 mm.Hg systolic, and pulse rate 84/minute.

PREMEDICATION:

Atropine 0.6 mg. given immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen, succinylcholine 50 mg. being given and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen, with cyclopropane 10-20%, by an IPPR technique via a carbondioxide circle absorption system. No further relaxant drug was given until the conclusion of the procedure, when a further 30 mg. succinylcholine was given to facilitate closure of the abdomen. This produced a marked bradycardia of 40/minute which returned to 84/minute following the administration of 0.6 mg. atropine. During the 2½ hour operation, 2 pints blood were administered. The B.P. and pulse rate remained at a level of 100-150 mm.Hg systolic and 80-90/minute respectively throughout the procedure.

Because of the gross pulmonary fibrosis, tracheostomy was performed immediately post-operatively. The patient regained consciousness rapidly and breathed adequately. Before return to the ward the B.P. was 100 mm.Hg systolic. Tidal volume, initially 350 ml., rose to 500 ml. 30 minutes after the operation and his colour remained good. 30 minutes later, 1 hour post-operatively, the B.P. fell to a level of 100 mm.Hg systolic. A slow infusion of the vasopressor Aramine was instituted and the B.P. rose to 120 mm.Hg systolic. Shortly after this, the patient died suddenly.

AUTOPSY

No autopsy

COMMENT:

This patient's death appears to be related to the pre-existing disease. Anaesthesia, from which he recovered rapidly and adequately, does not appear to be contributory. In view of the patient's gross pulmonary fibrosis, the question does arise as to whether the patient's treatment may not have been improved by oxygen therapy, or perhaps even by IPPR.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
72.2.64	1	Possibly	< 24	Subarachnoid haemorrhage Rupture of berry aneurysm.	Yes

Name: Lily Jakomo Age: 34 Sex: F Race: C

Disease: Intracranial aneurysm of circle of Willis. Operation: Craniotomy. Intended ligation of aneurysm.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Except for an intracranial aneurysm of the circle of Willis, the patient was fit. The left internal carotid artery had been occluded with a Crutchfield clamp. B.P. immediately before anaesthesia was 140/85 mm.Hg and the pulse rate 72/minute.

PREMEDICATION:

Omnopon 20 mg., atropine 0.6 mg. given 1 hour pre-anaesthesia.

ANAESTHETIC:

After induction of anaesthesia with the sequence thiopentone 300 mg., succinylcholine 100 mg., oxygenation, topical analgesia of the larynx and endotracheal intubation, anaesthesia was maintained with nitrous oxide and oxygen by an IPPR technique, via a carbon dioxide circle absorption system, IPPR being provided with a Pulmomat ventilator. A single dose of dTc 20 mg. was given. Immediately following induction of anaesthesia, 90 gm. urovert in 300 ml. water were infused intravenously to produce diuresis and to reduce brain tension and bulk. 30 minutes after commencing this infusion the B.P. rose rapidly from the previous level to 250 mm.Hg systolic. When the B.P. reached its peak, from the previous level of 80/minute, a tachycardia of 130/minute occurred. Craniotomy revealed a tense brain and a large fresh subarachnoid haemorrhage. The operation was abandoned. At the conclusion of operation and anaesthesia, the patient failed to regain consciousness. Spontaneous respiration resumed but was of a Cheyne-Stokes character. Residual curarisation was reversed with neostigmine 2 mg. preceded by atropine 1.2 mg. The patient died 4 hours post-operatively without regaining consciousness.

AUTOPSY

Ruptured Berry aneurysm of circle of Willis. Extensive subarachnoid and subdural haemorrhage. Extravasation of blood in neck muscles.

COMMENT:

The sequence of events here strongly suggests that the rapid rise in B.P. following administration of urovert caused a rupture of the intracranial aneurysm, which was to be ligated following craniotomy. The resultant subarachnoid haemorrhage caused abandonment of the operation and the subsequent death of the patient.

The diuretic urovert is known to cause an increase in blood volume and B.P. before the onset of diuresis. What was a little unexpected in this case was the magnitude of the rise in B.P. If the use of urovert is considered as a standard ancillary to the anaesthetic technique in certain neurosurgical conditions, then the anaesthetic technique in this case must be considered - in the context of the intracranial pathology - contributory to the patient's death.

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The lesson that emerges from this case is that where urovert is used in the presence of an intracranial aneurysm, the rise in blood pressure that may follow should be anticipated and prevented, either by the use of an agent such as Halothane or a ganglophegic drug.

PREVENTABILITY:

The rise in blood pressure that follows the administration of the diuretic urovert is well known. Intelligent anticipation could have prevented the sudden rise in blood pressure which was the probable cause of the rupture of this intracranialaneurysm. This death is regarded thus as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
73.2.64	2	No comment	< 24	Cerebral laceration	Yes

Name: Fritz Matthee Age: 36 Sex: M Race: E

Disease: Multiple injuries. Fractured left femur, left tibia, ?ruptured kidney, fractured skull, subdural haematoma.

Operation: Right carotid angiography. Right parietal temporal burrhole craniotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had sustained multiple injuries. Besides severe head injuries, he had a fractured left femur and tibia, and possibly ruptured kidney. He was comatose, severely shocked on admission to hospital. After transfusion of 5 pints blood, the B.P. rose to 120 mm.Hg systolic.

PREMEDICATION Nil

ANAESTHETIC:

Oral intubation was performed after topical analgesia of the larynx and trachea. Anaesthesia was induced and maintained with nitrous oxide and oxygen. An IPPR technique was used for the early part of the operation, the patient later being allowed to breathe spontaneously. During operation, 1 pint blood and 100 ml. 5% dextrose in water was administered.

Craniotomy revealed severe cerebral laceration. At the conclusion of operation, which lasted 95 minutes, a tracheostomy was performed. The patient died without regaining consciousness, 20 hours post-operatively.

AUTOPSY:

Abrasions on both legs, back and face. Contused wound over the occipital region of skull. Fracture of left occipital bone with subdural haemorrhage covering right frontal parietal and occipital regions. Fractures of right femur and tibia. Rupture of spleen with 500 ml. blood in abdominal cavity.

COMMENT:

Death was due to cerebral laceration. Haemorrhage from a ruptured spleen may have contributed. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
74.2.64	1	Probably	ORD	Anoxic anoxia. Respiratory obstruction. Inadequate post-operative supervision. Cardiac arrest.	Yes

Name: Doreen Slinger Age: 24 Sex: F Race: C

Disease: Prolonged labour. Pre-eclamptic toxaemia. Operation: Lower segment caesarean section.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

This obese (weight 190 lb.) primipara, who suffered from pre-eclamptic toxaemia, had been in labour for 40 hours. Prolonged labour, pre-eclamptic toxaemia and the onset of foetal distress were the indications for caesarean section. Albuminuria ++ and gross ketonuria were present and she appeared acidotic and was drowsy. After administration of 1 litre 10% dextrose in water, the B.P. was 120/70 mm.Hg and pulse rate 120/minute. The probable metabolic acidosis was not corrected or investigated pre-operatively by biochemistry. It is doubtful if rehydration was adequate.

PREMEDICATION:

Atropine 0.6 mg. given immediately pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence nitrous oxide and oxygen inhalation, succinylcholine 50 mg., oxygenation, and oral intubation, and was then maintained with nitrous oxide and oxygen administered by an IPPR technique via a carbon dioxide circle absorption system. dTc 20 mg. was administered via the intravenous infusion drip. When it was noted that this drip had come out of the vein and solution had run into the tissues, a new intravenous infusion drip was established and a further 20 mg. dTc was administered. Little of note occurred during caesarean section, which took 75 minutes in all. At the conclusion of the procedure, 2.5 mg. neostigmine preceded by atropine 1.2 mg appeared to produce adequate reversal of residual curarisation. On discontinuance of the anaesthetic, the patient appeared to be regaining consciousness. Respiration was spontaneous and appeared adequate. B.P. was 130 mm.Hg systolic and the pulse rate 96/minute. The patient was transferred from the operating table to the theatre trolley, lying on her back, where she was left unattended and unobserved for approximately 3 minutes. After this she was seen to be cyanosed with 'heaving and gasping' respiration. The anaesthetist immediately re-intubated the patient and commenced IPPR with oxygen, but cardiac arrest occurred and the pupils became dilated. External cardiac massage was commenced immediately. No peripheral pulse was palpable. Adrenaline 1:1,000 solution, 1 ml., was injected into the left ventricle and external cardiac massage was continued, but still no peripheral pulse was palpable. The pupils remained dilated. Only after 8 minutes of inefficient external cardiac massage was left thoracotomy performed and internal cardiac massage commenced, 10 minutes after cardiac arrest. The heart was dilated and atonic. Adrenaline solution1:1,000, 1 ml, was injected intraventricularly without effect. Sodium bicarbonate 55 m.Eq. was rapidly administered intravenously. After 4 minutes of cardiac massate, heart beat recommenced, extremely feebly. 1 gm. calcium chloride was injected intraventricularly and cardiac massage continued. Weak ventricular fibrillation followed, with little improvement in the cardiac tone. Repeated electrical defibrillation produced asystole followed immediately by weak ventricular fibrillation. After 30 minutes all resuscitative efforts were abandoned.

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AUTOPSY

16 cm. thoracotomy (left) wound. 5 cm. incision in pericardium. Sutured wound of lower segment caesarean section. Both kidneys enlarged and pale. Fatty infiltration of the liver.

COMMENT:

From the description of events following immediately on conclusion of anaesthesia, it is very likely that, while unobserved on the theatre trolley, this obese patient suffered pharyngeal respiratory obstruction - probably from the falling back of the tongue. She was obese, short-necked and was lying on her back. She had been curarised (total dose 40 mg.), approximately one half of the dose administered subcutaneously. It is probable that the reversal of curarisation was incomplete, leaving little reserve of muscle power to cope with the slightest respiratory obstruction.

From the account, it is difficult to conclude if cardiac arrest preceded or coincided with re-intubation. It is most likely that it preceded re-intubation. If it coincided, a case could be made for vagal cardiac arrest in the presence of gross anoxia. However, as the endotracheal tube only recently removed from the trachea had been lubricated with a jelly containing 2% xylocaine, this is unlikely.

The persistence with external cardiac massage when no peripheral pulsation could be felt, and the pupils remained dilated, was a major fault in the conduct of this case. The delay of 10 minutes before instituting effective cardiac massage, after external massage had proved ineffective, must have ensured the fatal outcome.

Another major fault in the conduct of this case was the failure to diagnose the extent of, and to treat, the metabolic acidosis that must certainly have been present. Inter alia, gross ketonuria was noted, the patient had pre-eclamptic toxaemia and had been in prolonged labour. This metabolic acidosis may in part have been responsible, if not for the cardiac arrest, certainly for the refractory asystole.

The anaesthetic management is a significant contributory factor to this patient's death.

PREVENTABILITY:

In view of the correctable faults evident in the conduct of this case:

(1) failure to treat acidosis pre-operatively,

(2) the positioning of an obese patient on her back on the theatre trolley,

(3) inadequate supervision in the immediate post-operative period, especially in view of

(4) relatively large dose of curare, part of which was administered subcutaneously, and

(5) the failure to produce effective cardiac massage within 3 minutes of cardiac arrest,

this death is regarded as probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
75.2.64	2	No comment	< 24	Peritonitis	Yes

Name: Linda Price Age: 60 Sex: F Race: E

Disease: Large bowel obstruction

Operation: Laparotomy. Colostomy. Drainage of peritonitis.

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

The patient was admitted with large bowel obstruction. Following rehydration therapy, her condition was fair except for the presenting condition. All other systems were normal.

PREMEDICATION

Atropine gr. 1/100 given immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 20^{0} mg., succinylcholine 50 mg., oxygenation and oral intubation, and was then maintained with nitrous oxide and oxygen, with 10-20% cyclopropane, administered via a carbon dioxide absorption system by an IPPR technique.

Laparotomy revealed a carcinoma of the sigmoid colon with perforation of the bowel and faecal peritonitis. A transverse colostomy was performed and the abdomen was closed with drainage. The course of anaesthesia was untoward. The operation lasted 60 minutes. Succinylcholine 25 mg. was given to facilitate closure of the abdomen. At the conclusion of anaesthesia, the patient breathed spontaneously and adequately. She recovered consciousness rapidly post-operatively but died 18 hours later.

AUTOPSY

Carcinoma of the sigmoid colon. Perforation of the bowel above the carcinoma. Faecal peritonitis. Hypertrophy of large bowel. Old Polya type gastrectomy with splenic adhesions. Bilateral basal pulmonary collapse. Ventricular hypertrophy. Atheroma of the aorta. Transverse colostomy.

COMMENT:

Death was probably due to the effects of peritonitis. The anaesthetic is not considered contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
76.2.64	2	No comment	< 24	Massive intra-cerebral haemorrhage	Yes

Name: C.H. Loubser Age: 51 Sex: M Race: E

Disease: Intracranial haemorrhage. Operation: Carotid angiography.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Comatose, breathing spontaneously but with Cheyne-Stokes type respiration. On admission an endotracheal tube was passed and IPPR with air was commenced, using an Ambu bag. B.P. 130 mm.Hg systolic. Pulse rate 130/minute.

PREMEDICATION:

Atropine 0.6 mg. given 45 minutes pre-operatively.

ANAESTHETIC:

Aleft carotid angiogram was commenced without anaesthesia, the patient being ventilated with oxygen only. He responded to carotid puncture with gross muscular movements. Anaesthesia was then induced with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Little dye entered the cerebral circulation on carotid injection. No further operatige procedure was undertaken. At the conclusion of angiography, apnoea and coma persisted. IPPR with oxygen was continued. One hour later the neurosurgeon decided that the patient's prognosis was hopeless and IPPR was discontinued. The patient died.

AUTOPSY

Massive intracranial and intracerebral haemorrhage.

COMMENT:

This death was the result of the existing disease and the anaesthetic administered was in no way contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
77.2.64	3	No comment	ORD	Massive haemorrhage. Massive transfusion Hypothermic cardiac arrest.	Yes

Name: Helen Miller Age: 54 Sex: F Race: E

Disease: Abdominal aortic aneurysm. Operation: Resection of aneurysm and graft of aorta.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

There was a large abdominal aortic aneurysm. B.P. was 150/100 mm.Hg and the haemoglobin concentration was 15 gm.%. Urea clearance was impaired.

PREMEDICATION:

Pethilorfan 100 mg., atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation, and was then maintained with nitrous oxide and oxygen administered via a non-rebreathing circuit by means of a Cyclator ventilator. ECG monitoring was instituted immediately following induction of anaesthesia. dTc was given in divided doses, total dose being 39 mg. during the operation. After induction of anaesthesia and administration of a water load of 800 ml. 5% dextrose in water, 20 gm. Mannitol was given, followed by 100 m.Eq. sodium bicarbonate.

During dissection the aneurysm ruptured causing gross blood loss. This was combatted by massive blood transfusion. During the first hour after this event, 9.5 litres blood were transfused maintaining the B.P. at a level of about 140 mm.Hg systolic. Before the aorta was cross clamped, 150 mg. heparin was administered. Massive transfusion of cold blood caused hypothermia, the patient's temperature dropping to 28°C. At this stage ventricular extrasystoles occurred and were soon followed by the onset of ventricular fibrillation. Attempts at electrical defibrillation were vain, as were attempts to raise the patient's temperature with peritoneal lavage with warm saline.

AUTOPSY

30 cm. long paramedian abdominal incision. An open aneurysm of the abdominal aorta with a partially sutured aortic graft in situ. The coronary arteries displayed gross arteriosclerosis. Both lungs were congested and oedematous.

COMMENT:

Massive transfusion of cold blood, necessitated by severe haemorrhage, led to hypothermia. This resulted in hypothermic ventricular fibrillation which proved refractory to treatment, as attempts to warm the patient failed. The anaesthetic management is necessarily and unavoidably contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
78.2 64	3	No comment	ORD	Haemorrhage.	Yes

Name: Janice & Jennifer Schonraad Age: 2 months Sex: F Race: E

Disease: Craniopagus twins. Operation: Separation of craniopagus twins.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

These craniopagus twins were joined at the occipital region of the skull. In their two months of life, the one twin had suffered repeated episodes of cardiorespiratory failure.

PREMEDICATION: Nil.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen and halothane 0.5 - 1% inhalation. Intubation was extremely difficult but was successfully achieved. During carotid angiography, anaesthesia was maintained with nitrous oxide, oxygen and halothane 0.5%, with the twins breathing spontaneously. The B.P. of one child fell repeatedly during attempts at carotid puncture. Before craniotomy, a femoral venous cutdown was performed on each of the babies and ECG monitoring was instituted. During craniotomy, anaesthesia was maintained with nitrous oxide and oxygen alone, administered by an IPPR technique using T-piece circuits. dTc 2 mg. was administered.

The operation produced marked blood loss, to such an extent that after the first hour the femoral vein cannulae were replaced by larger ones. At operation, the twins were found to have a common posterior cranial fossa with large communicating venous sinuses. One twin was sacrificed by cerebral evisceration. Gross haemorrhage continued and difficulty was experienced in replacing blood as rapidly as it was lost. After 6 pints blood had been transfused, the remaining twin suffered cardiac arrest.

AUTOPSY

(Janice Schonraad): The heart, lungs and abdominal organs were normal. There was a 15 cm. long operative wound across the skull. Under this incision a large portion of the skull was absent. There was a large space between the brain and the skin incision. The brain in the skull was normal.

COMMENT:

In retrospect, the anatomy of twins with a common posterior cranial fossa rendered successful separation of these craniopagus twins impossible. The final cause of death was uncontrollable haemorrhage from large venous sinuses. Anaesthesia is not considered contributory to death but, as death occurred while the patients were anaesthetised, the case is classifiedin group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
79.2.64	3	No comment	ORD	Massive haemorrhage. Massive transfusion, Hypothermic cardiac arrest.	Yes

Name: Marjory Wyngaard Age: 49 Sex: F Race: C

Disease: Carcinoma of the cervix. Operation: Wertheim hysterectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

In additionto carcinoma of the cervix, this patient had essential hypertension, B.P. 210/120 mm.Hg. This was not treated. She was obese and weighed 180 lbs.

PREMEDICATION

Omnopon 20 mg., atropine 0.65 mg. given 40 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained then with nitrous oxide and oxygen, and for the early part of the operation a trace of ether vapour, administered via a carbon dioxide circle absorption system by an IPPR technique. Following an hour of uneventful anaesthesia, excessive venous haemorrhage occurred from the operative site. Within the next hour, 10 pints cold blood were transfused rapidly. The B.P. dropped to 130 mm.Hg systolic during this period. Two doses of 0.5 mg. each phenylephrine were administered and, with blood transfusion, 4 gm. calcium gluconate was given. The massive transfusion of cold blood caused the patient's temperature to drop and, though not measured, she felt extremely cold. After giving the 10th pint of blood - although on estimate, blood replacement was not more than 1-2 pints behind loss - cardiac arrest occurred. Following a period of 1 minute, when external cardiac massage was performed, a left thoracotomy was performed and internal cardiac massage commenced. The heart was flabby and completely unresponsive. Massage was continued for 30 minutes without effect, 25 m.Eq. sodium bicarbonate being given and an infusion of adrenaline 1:75,000 commenced, without effect. There was no response whatever to resuscitative measures and cardiac massage was abandoned after 30 minutes.

AUTOPSY

Surgical incision in lower abdomen and left chest. Uterus had been excised.

COMMENT:

The probable cause of this cardiac arrest was hypothermia resulting from massive transfusion of cold blood, necessitated by an equally massive haemorrhage from the operative site. No method of warming blood rapidly was available to avoid this. Perhaps some attempt should have been made to warm the patient after cardiac arrest. However, in a patient of this size, this is unlikely to have been successful. The anaesthetic management per se is not regarded as more than necessarily and unavoidably contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
80.2.64	1	Probably	<24	Inadequate post-operative supervision. Anoxic anoxia, Respiratory obstruction. Partial curarisation. Cardiac arrest.	Yes

Name: Abbas Hattas Age: 44 Sex: M Race: C

Diseaae: Chronic gastric ulcer. Operation: Partial gastrectomy.

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

The patient had a chronic gastric ulcer which had been the cause of haematemesis 1½ months before this admission to hospital. The other systems appeared normal. B.P. was 130/90 mm.Hg and haemoglobin concentration 12.5 gm.%.

PREMEDICATION

Omnopon 10 mg., atropine 0.6 mg. given 70 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 350 mg., succinylcholine 100 mg., oxygenation, topical analgesia of the larynx, oral intubation, and spontaneous respiration resumed within minutes of the injection of succinylcholine. An initial dose of dTc 30 mg. was administered and anaesthesia was then maintained with nitrous oxide and oxygen, with minimal etherfor the early part of the procedure, administered via a carbon dioxide circle absorption system by an IPPR technique. Further small doses of dTc were administered dufing the operation, up tothe total of 45 mg., the last dose of 10 mg. being given 30 minutes before the end of the procedure. The course of anaesthesia during operation, which lasted 95 minutes, was untoward. Blood loss was estimated at 400 ml. and 400 ml. of 5% dextrose in water was infused during operation. At the conclusion of the procedure, after some respiratory activity had returned, 3 mg. neostigmine preceded by atropine 1.2 mg. was administered. The respiration became of adequate volume but the pattern of mild residual curarisation persisted; there was slight tosis on eye-opening and a mild tracheal tug with respiration. The patient regained consciousness rapidly after discontinuance of the anaesthetic and, as the respiratory tidal volume appeared adequate, although there were signs of residual curarisation, the patient was returned to the ward in the care of a nurse. No pharyngeal airway was inserted. Before the journey to the ward, the patient was positioned on the theatre trolley on his back. The nurse who accompanied the patient to the ward subsequently described the patient as making a snoring or sterterous noise when breathing en route to the ward. She did not realise the importance of this observation, nor did she do anything about it. The journey from the operating theatre to the ward involved a 1 floor journey in a lift. On arrival in the ward, the patient was handed over to another nurse who, hearing the peculiar snoring noises, summoned the help of the ward house-surgeon. When he arrived at the patient's bedside, the patient was cyanosed and a radial pulse just palpable when the initial examination was made rapidly became impalpable. He diagnosed the onset of cardiac arrest and commenced external cardiac massage. Endotracheal intubation wasperformed and IPPR commenced. All resuscitative efforts, including intracardiac injection of adrenaline, were in vain.

AUTOPSY

Multiple adhesions in the pleural cavity. Early constrictive pericarditis. A Polya gastrectomy had been performed. The right kidney

was severely pyelonephritic with a blocked right ureter. Pulmonary oedema.

COMMENT:

It seems reasonably clear that, at the conclusion of this general anaesthetic involving the use of a muscle relaxant, the patient - though conscious - was still partially curarised. From the account he appears to have suffered respiratory obstruction, probably of the oropharynx due to the tongue falling back while he was lying on his back, en route to the ward. Nothing was done about this and the patient had insufficient muscle power to overcome it himself. On arrival in the ward, thenurse into whose care the patie nt was given also heard the snoring breathing, and also did nothing herself to relieve this, but summoned help from the ward house-surgeon. From his description, it is obvious that the patient suffered a cardiac arrest from anoxic anoxia at just about the time he reached him. This death was the result of anoxic anoxia due to respiratory obstruction occurring immediately post-operatively in a patient who though initially conscious, was still partially curarised and was inadequately supervised.

The anaesthetic management is the sole cause of this patient's death. It is doubtful if the other lesions discovered at autopsy were of any contributory significance.

PREVENTABILITY

More complete reversal of curarisation and/or adequate post-operative supervision would have prevented this patient's death, which is thus considered probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
81.2.64	3	No comment	ORD	Massive haemorrhage Massive transfusion Hypothermic cardiac arrest.	Yes

Name; Beatrice Meyer Age: 27 Sex: F Race: C

Disease: Primary carcinoma of the vagina. Operation: Abdomino-perineal resection and Wertheim hysterectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Besides the surgical lesion, the patient had an atrial septal defect. She was not in cardiac failure and had a fair exercise tolerance. B.P. was 115/70 mm.Hg and the other systems were normal. She weighed 100 lbs.

PREMEDICATION

Omnopon 10 mg., atropine 0.65 mg. given 1 hour pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 300 mg., succinylcholine 40 mg., oxygenation, topical analgesia of the larynx, and oral intubation. ECG monitoring was instituted. Anaesthesia was maintained with nitrous oxide, oxygen and Halothane administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was given in divided doses, total dose being 35 mg. throughout the operation. The Trendelenburg postion was used. Halothane concentration, 0.5% for the first 30 minutes of the procedure, was then reduced to 0.25% and was discontinued after 45 minutes. Pelvic dissection resulted in a rapid blood loss which was replaced by rapid transfusion. Blood replacement kept pace with the loss but, after 3½ pints cold blood had been transfused, quite rapidly, the oesophageal temperature had dropped to 31°C. In spite of what was felt to be adequate blood replacement, the B.P. fell progressively and showed little response to intravenous injection of two doses of 0.5 mg. phenylephrine. At this time the procedure had been in progress for 60 minutes. Simultaneously with the progressive fall in B.PA., bradycardia with ventricular extrasystoles became apparent on the ECG and within 10 minutes this had progressed to cardiac arrest, in ventricular fibrillation. IPPR was continued with oxygen only and the operating table was levelled. A left thoracotomy was performed immediately and internal cardiac massage commenced. This was not very effective. Vigorous massage appeared to distend the right atrium and right ventricle grossly, and also the liver, more than producing an effective pulse. Cardiac muscle tone felt poor. Adrenaline 1:100,000 was infused intravenously to improve the cardiac tone and 50 m.Eq. sodium bicarbonate was rapidly given. The heart was defibrillated electrically and sinus rhythm restored, lasting but 2 minutes, after which ventricular fibrillation recurred. After another attempt at electrical defibrillation, ventricular fibrillation again recurred. The oesophageal temperature had now dropped to 30.5°C. Attempts were made to raise the patient's temperature by lavage of the abdominal and pleural cavities with warm saline. 10 ml. 10% calcium gluconate was administered and further attempts made at defibrillation, without effect. The pupils were now widely dilated. Resuscitative measures were abandoned 40 minutes after cardiac arrest. A total of 4 pints blood had been transfused.

AUTOPSY

24 cm. thoracotomy incision. 20 cm. long left paramedian abdominal incision. The vulva, vagina, uterus and parametria had been removed. The liver was large and extremely congested. There was an atrial septal defect measuring 4 cm. in diameter.

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COMMENT:

From the description of events, two possible causes of the cardiac arrest are evident: (1) hypovolaemia from blood loss, and (2) hypothermia from the rapid transfusion of cold blood.

The anaesthetist was fairly sure that blood replacement did match blood loss quite closely. The more likely possibility from the clinical description is that of hypothermic cardiac arrest. It is surprising that the oesophageal temperature should be so depressed by a volume of cold blood not very large, even though the patient weighed only 100 lb. and the blood was transfused very rapidly. A suggested hypothesis of the mechanism by which this could occur is based on the fact that the patient had a large atrial septal defect. Although the major shunt is left to right, there is always a small right to left component. The raised right atrial pressure resulting from the Trendelenburg position together with the rapid pressure of transfusion of blood via a brachial vein, may have increased the volume of the right to left atrial shunt through the large septal defect. Thus, a volume of cold blood would have been introduced directly into the left atrium and ventricle without the heat-buffering effect of passage through the pulmonary circuit. Thus aortic blood, coronary blood and the myocardium would be more rapidly cooled - reflected by the rapid drop in oesophageal temperature.

Another aspect of this case which merits discussion is the ineffective cardiac massage. Vigorous cardiac massage caused gross distension of the right atrium and liver, but this may have been due to themanual squeezing of the left ventricle causing some dilatation and distortion of the mitral valve ring and mitral valve. Any regurgitant blood would then have passed through across the large atrial septal defect into the right atrium, distending this and, retrograde, the liver and central venous system. Once cardiac arrest occurred, although cardiac massage was undertaken immediately, its ineffectiveness for the above reasons was the coup de grace.

This death, then, was the result of operative haemorrhage and the effects of brisk replacement of cold blood in the presence of a large atrial septal defect. The anaesthetic management is regarded as nedessarily and unavoidably contributory to this death. Means for the rapid heating of stored blood were not available at this time.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
82.2.64	2	No comment	< 24	Haemorrhage Gross sepsis.	Yes

Name: Samuel Abrahams Age: 46 Sex: M Race: C

Disease: Obstructive jaundice. Cholelithiasis. Operation: Cholecystenterostomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had chronic pancreatitis and had had many operations including splenectomy, excision of tail of pancreas for retrograde gastric drainage of pancreas. 1 month before this operation he was admitted with an attack of acute pancreatitis. This had settled but the patient developed progressive obstructive jaundice. It was thought to be due to impaction of a gallstone in the cystic duct. Serum bilirubin was 28 mg.%. The liver was enlarged and was palpable 3 fingers below the costal margin. There was consolidation of the right lung base. A swinging temperature had been present; immediately before anaesthesia, the temperature was 99°F. The patient appeared very toxic.

PREMEDICATION:

Atropine 0.6 mg. given 1 hour pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide and oxygen. As soon as the patient lost consciousness, oral intubation was performed after injection of succinylcholine 30 mg. and a period of IPPR. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Abdominal relaxation was maintained with intermittent doses of succinylcholine, using a total of 275 mg. during the 3½ hours of operation. Repeated doses of succinylcholine were administered only when spontaneous respiration resumed. At operation, gross adhesions were found around the gall bladder and the liver tract. Dissection of these resulted in quite severe blood loss, which, replaced by transfusion, involved the administration of 6 pints blood during the operation. This was considered adequate replacement. Cholecystitis and cholelithiasis were present but the precise obstruction of the biliary tract could not be identified. The cystic duct was difficult to identify amidst the gross adhesions. A cholecystenterostomy was performed. During the operation, 100 m.Eq. sodium bicarbonate was infused intravenously. At the conclusion of the operation, on discontinuance of anaesthesia, the patient regained consciousness rapidly but respiration did not return to normal. The tidal volume of the spontaneous respiration was only 200 ml. and a distinct tracheal tug was evident. He could move his arms and legs adequately, however. Facial movements and eye-opening were normal. A nasal endotracheal tube was inserted and the respiratory volume augmented by means of a Bird ventilator. The action of the ventilator was patient-triggered. There was no impairment of consciousness and he could communicate adequately by means of written messages. Post-operatively, haemorrhage from the intra-abdominal adhesions continued manifested by the abdominal drains. Blood transfusion was continued and 5 pints administered within the next 12 hours. The patient's condition continued to deteriorate, however, and he died 12 hours post-operatively.

AUTOPSY

The body was grossly jaundiced. There was a 22 cm. midline abdominal incision. A rubber drain was inserted through the upper end of this incision. There was 1,000 ml. blood in the abdominal cavity. The gall bladder, though anastomosed to the duodenum, still contained gall stones and purulent mucus. The cystic duct was not identifiable.

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There was biliary cirrhosis of the liver with wide dilatation, of the bile ducts. The bile ducts contained purulent mucus. The ampulla of Vater was markedly oedematous and a gallstone was packed therein. There was gross pancreatitis.

COMMENT

The patient's existing disease and continued intra-abdominal haemorrhage were doubtless the cause of his death. The one aspect of the anaesthetic management which is open to criticism was the use of intermittent doses of succinylcholine as the muscle relaxant in a patient with known liver disease. Repeated doses were only given when spontaneous respiration returned, this being taken as an indication that the previous dose had been adequately metabolised. The total dose used was very moderate.

Spontaneous respiration was inadequate post-operatively although muscle power and facial muscles appeared to be normal. The treatment of this respiratory inadequacy was correct and this does not appear to have been a factor contributing significantly to the patient's death.

It is interesting to note the association of pancreatitis with the onset of abnormal post-anaesthetic respiration. This has been apparent in several other cases in this survey, although in this case a depolarizing relaxant was used whereas in the others non-depolarizing relaxants were used.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
83.2.64	3	No comment	ORD	Haemorrhage Haemophilia	No

Name: Gamat Saban Age: 31 Sex: M Race: C

Disease: Pleural and pulmonary bleeding; haemophilia. Operation: Left thoracotomy.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

The patient had haemophilia. He presented 2 days before operation with gross haemoptysis from pulmonary haemorrhage. Increasing dullness of the left thorax indicated intrapleural bleeding as well. In spite of continued blood transfusion, his condition deteriorated steadily and the present operation was decided on as a desperate measure to stem the haemorrhage. Shortly before operation he appeared to have an attack of pulmonary oedema, which was treated by the bleeding of ¾ pint blood, despite the continued bleeding by haemoptysis and intrapleural haemorrhage. Immediately before anaesthesia he appeared moribund. Respiration was gasping and signs of pulmonary oedema were still present. B.P. 150 mm.Hg systolic the pulse rate being 140/ minute and there was marked generalised vasoconstriction.

PREMEDICATION: Nil.

ANAESTHETIC:

After topical analgesia of the larynx and pharynx, oral intubation was performed with the patient awake. Thereafter, frothy fluid and blood was aspirated from the trachea and bronchi. Anaesthesia was induced and maintained with nitrous oxide and oxygen, with a trace of ether vapour, administered by a carbon dioxide circle absorption system by an IPPR technique. IPPR was easily performed and revealed a large haemothorax. During this time, the patient's condition steadily deteriorated. Blood was rapidly transfused but, despite this, shortly after thoracotomy, cardiac arrest occurred. In view of the apparently hopeless condition of the patient, no cardiac massage or resuscitative measures were undertaken.

AUTOPSY

No autopsy

COMMENT

Haemorrhage and ultimately cardiac failure doubtless caused the death of this haemophiliac patient. The treatment of the pulmonary oedema by blood-letting, in the face of continued haemorrhage, is questionable. Perhaps temporary measures, such as the application of limb tourniquets, may have been more practical and have served the purpose of conserving blood that was to be greatly needed shortly thereafter.

Although the anaesthetic management per se is not considered a significantly contributory factor, as this death occurred while the patient was anaesthetised, it is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
84.2.64	1	Probably	ORD	Relaxant associated death. Anoxic anoxia. Respiratory acidosis. Neostigmine cardiac arrest.	Yes

Name: Ingvald Rusthoi Age: 82 Sex: M Race: E

Disease: Obstructive jaundice; carcinoma of the head of the pancreas. Operation: Cholecystenterostomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This obese patient (weight 200 lb.) was admitted to hospital 6 days before this operation, suffering from obstructive jaundice. On investigation, carcinoma of the head of the pancreas was diagnosed. Before surgery, signs of peritonitis developed and his condition deteriorated. Immediately pre-anaesthesia he was pyrexial, temperature 99.2°F with a tachypnoea of 30/minute with laboured breathing. The B.P. was 140/80 mm.Hg and pulse rate 130/minute. The cardiovascular system appeared normal.

PREMEDICATION:

Pethilorfan 50 mg., atropine 0.6 mg. given 1 hour pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Gallamine was used to achieve muscle relaxation, total dose being 150 mg. (initial dose 100 mg., then 30 mg. and then 20 mg.) throughout the operation which lasted 85 minutes.

Laparotomy revealed a carcinoma of the head of the pancreas with biliary peritonitis. A cholecystenterostomy was performed. During closure of the abdomen, spontaneous respiration resumed, characterised by a marked tracheal tug and the tidal volume was inadequate. This respiration was allowed to continue for some 15 minutes until the operation was complete. Atropine 1.2 mg. was given followed by 1 mg. neostigmine. Within 1 minute of the injection of neostigmine, respiration suddenly ceased. IPPR was resumed. It was not noticed that no pulse was palpable. The anaesthetist failed to diagnose cardiac arrest immediately. Not believing that this had in fact occurred he administered 0.5 mg. phenylephrine and whenthis had no effect, infused noradrenaline in a concentration of 4 micr.gm./ml., only diagnosing cardiac arrest after a delay of some minutes. The pupils by now were widely dilated. External cardiac massage was commenced. This was completely ineffective. Because a delay in excess of 5 minutes had occurred since the probable occurrence of cardiac arrest, and irreversible ischaemic changes had doubtless occurred, and because of the ultimately hopeless prognosis of the patient's condition, no further resuscitative measures were undertaken. Internal cardiac massage was not performed.

AUTOPSY

The body was markedly jaundiced. 20 cm. long leftparamedian incision. Cholecystenterostomy had been performed. Carcinoma of the head of the pancreas. Drainage tubes present in the abdomen.

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COMMENT:

In this case, cardiac arrest appears to have followed immediately after intravenous injection of 1 mg. neostigmine, even though this was preceded by an adequate dose of atropine. The dose of neostigmine was not large. The question of whether neostigmine injected intravenously for the reversal of curarisation can produce cardiac arrest when preceded by an adequate dose of atropine is a vexed one. Cases have been described. The cardiac arrest in this case does certainly appear to have been related to the intravenous injection of neostigmine, in time. What is certain is that, for a period of at least 15 minutes before administration of atropine and neostigmine, this patient was permitted to breathe spontaneously with a definitely inadequate tidal volume. During this time he would have developed respiratory acidosis and would probably have been anoxic as well. This state of itself could cause cardiac arrest in poor risk patients. If neostigmine is to be cited as related to the cardiac arrest, these conditions of respiratory acidosis and anoxia are the very conditions which its occurrence is described in.

Whatever the precise aetiology of the cardiac arrest, its diagnosis and treatment were quite inadequate. Although the clinical evidence was there, the anaesthetist frittered away vital minutes, administering vasopressor drugs. Although cardiac arrest occurred in the presence of the anaesthetist, cardiac massage was commenced only several minutes later. And again, only after some minutes had been wasted in ineffective external cardiac massage (perhaps ineffective because of the patient's obesity, or because of inexperience of the surgeon) was the thought of internal cardiac massage entertained - only to be rejected, as irreversible changes would have occurred by then.

The anaesthetic management is considered significantly contributory to this patient's death.

PREVENTABILITY

Because (1) this poor risk patient was allowed to develop respiratory acidosis and anoxia unnecessarily at the end of the operation, and (2) the diagnosis and treatment of cardiac arrest, when it occurred, were completely inadequate and faulty, this death is regarded from the point of view of the anaesthetic management as having been probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
85.2.64	2	No comment	< 24	Haemorrhage from ruptured aortic aneurysm	Yes

Name: Herman Hirsch Age: 65 Sex: M Race: E

Disease: Ruptured abdominal aortic aneurysm

Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted with a leaking abdominal aortic aneurysm and was taken straight to the operating theatre. Considering the lesion present, his condition was not extreme. B.P. 120 mm.Hg systolic and pulse rate 100/minute. He was grossly atherosclerotic and had pulmonary emphysema. Slow blood transfusion was commenced immediately.

PREMEDICATION

Atropine 0.6 mg. given immediately before anaesthesia, intravenously.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a circle absorption system by an IPPR technique. dTc was used for muscle relaxation, a total of 30 mg. being given during the operation, which lasted 180 minutes. Laparotomy revealed a marked retroperitoneal haemorrhage from a leaking abdominal aortic aneurysm. The aneurysm was extensive reaching right up to the diaphragm. It was quite inoperable , and no operative treatment was undertaken. At the conclusion of the operation and anaesthetic, 2.5 mg. neostigmine preceded by 1.2 mg atropine was administered to reverse residual curarisation. Normal spontaneous respiration resumed and the patient rapidly regained consciousness. He died 2 hours post-operatively.

AUTOPSY

Right paramedian abdominal incision. Extensive abdominal aortic aneurysm. Extensive retroperitoneal haemorrhage.

COMMENT:

This death was due to rupture of an extensive, inoperable abdominal aortic aneurysm.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
86.2.65	2	No comment	<24	Cerebellar haematoma.	Yes

Name: Michael Colson Age: 8 Sex: M Race: C

Disease: ?Extradural haemorrhage. Operation: Carotid angiography

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Carotid angiography under general anaesthesia had been performed earlier in the day on this child, who was unconscious. At this time, athetoid movements were evident and the child's breathing was sterterous. The anaesthetic (nitrous oxide, oxygen and 0.5% halothane) was uneventful. After carotid angiography, repeated generalised convulsions occurred. Further angiography was felt necessary. Before this second anaesthetic, the patient was unconscious and was having frequent convulsions.

PREMEDICATION: Nil.

ANAESTHETIC:

Oral intubation was performed after topical analgesia of the larynx. Nitrous oxide and oxygen were then administered with the patient breathing spontaneously. Following angiography, the anaesthetic was discontinued and convulsions returned. Hyperpyrexia then followed and the child died 3 hours after these last angiograms.

AUTOPSY

Cavernous malformation of the cerebellum. Haemorrhage and ploughing up of cerebellum.

COMMENT:

This child died of the pre-existing disease and anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
87.2.65	3	No comment	ORD	Haemorrhage	Yes

Name: Henry Malgas Age: 45 Sex: M Race: C

Disease: Multiple injuries. Ruptured liver, ruptured diaphragm.

Operation: Laparotomy. Suture of ruptured liver.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted suffering from a ruptured liver and diaphragm and other injuries. Blood transfusion was commenced; response was poor and there was evidence of continuing intra-abdominal haemorrhage. Although his general condition was poor, the B.P.being 90 mm.Hg systolic, laparotomy was considered urgent. There was some impairment of respiratory excursion.

PREMEDICATION:

Atropine 0.6 mg. by intramuscular injection 45 minutes pre-operatively.

ANAESTHETIC :

Anaesthesia was induced with inhalation of nitrous oxide and oxygen. As soon as consciousness was lost, oral intubation was performed after injection of succinylcholine 50 mg. Profuse vomiting ensued. Pharyngeal aspiration and, once the endotracheal tube was in position with the cuff inflated, tracheo-bronchial toilette were performed. No vomitus was found in the trachea or bronchi. Anaesthesia was then maintained with nitrous oxide and oxygenadministered via a carbon dioxide circle absorption system by an IPPR technique. dTc 20 mg. was given for relaxation. Laparotomy revealed a grossly lacerated liver, haemoperitoneum and a ruptured diaphragm. Blood was transfused under pressure. During exploration of the liver, the B.P. dropped to unrecordable levels for a period; this was thought to be due to vena caval obstruction. The B.P. recovered after 10 minutes. Thoracotomy was performed, to permit access to the lacerated posterior aspect of the liver, and to permit repair of the diaphragm. The posterior surface of the liver was packed with a large swab after vain attempts to suture the lacerations. Shortly after this, the B.P. again dropped and was unrecordable. Cardiac arrest occurred and internal cardiac massage was commenced immediately. The heart was empty and flabby. Resuscitative attempts were without success. Up to the time of cardiac arrest, 6 pints blood had been transfused.

AUTOPSY

Grossly lacerated liver. Fracture of ribs.

COMMENT:

Death was probably due to oligaemia with a blood loss inadequately replaced before and during surgery. Inferior vena caval obstruction due to swab packing behind the liver, to stop haemorrhage, may have been a factor finally precipitating cardiac arrest. Anaesthesia is regarded as necessarily and unavoidably contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
88.2.65	3	No comment	< 24	Hypotension (traction on cervical cord) Fracture dislocation of cervical spine.	Yes

Name: Daniel Jantjies Age: 16 Sex: M Race: C

Disease: Neurofibroma of cervical cord. Operation: Cervical laminectomy and excision of tumour.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

Pethidine 50 mg., hyocine 0.4 mg. 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. After resumption of spontaneous respiration, dTc 15 mg. was administered and anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system, by an IPPR technique, using a Pulmomat ventilator. One further dose of dTc 10 mg. was necessary during the operation. The patient was positioned sitting up with the neck acutely flexed; initial positioning caused a transient fall in B.P. The subcutaneous tissues and the neck muscles were infiltrated with a 1:300,000 solution of adrenaline. Blood loss during laminectomy was not great. The lamina of the 3rd to the 6th cervical vertebrae were removed, exposing the tumour. After the operation had been in progress for 45 minutes, manipulation of the tumour and traction on a cervical portion of the spinal cord caused a marked drop in B.P. and a bradycardia of 40/minute. 300 ml. blood was transfused and atropine 0.6 mg. injected. The B.P. rose to 80 mm.Hg systolic but the pulse rate remained at 40/minute. Surgical dissection continued and the administration of 0.1 mg. phenylephrine produced a rapid response, the B.P. rising precipitantly to 180 mm.Hg systolic and the pulse rate to 120/minute. ECG monitoring was instituted at this stage. The B.P. settled at a level of 140 mm.Hg systolic and the pulse rate at 120/minute for the following 30 minutes, althoughthere were transient falls. More blood was transfused during this period. The surgical manipulation of tumour and cervical cord again resulted in sudden rapid fall in pulse rate, to 100/minute, while the B.P. also dropped, the exact level could not be exactly recorded. 0.1 mg. phenylephrine was again injected but the B.P. remained unrecordable. Ventricular extrasystoles occurred shortly after and these persisted for 10 minutes, after which ventricular fibrillation supervened. External cardiac massage was performed while the patient was taken from the sitting position and laid supine. Left thoracotomy was performed and internal cardiac massage commenced. After intraventricular injection of 0.5 mg. adrenaline and further cardiac massage, a spontaneous heart beat returned. A drip infusion of aramine was instituted. More blood was transfused and the operation was completed with the patient in the lateral position. A total of 5½ pints blood were transfused during the operation. The B.P. was maintained at a level of 120 mm.Hg systolic with a pulse rate of 120/minute during the remainder of the procedure. At the conclusion of the operation, which lasted 4 hours, and on conclusion of anaesthetic, normal spontaneous respiration resumed. No antidote was considered necessary for reversal of curarisation. The patient failed to regain consciousness but the pupils were small. Immediately post-operatively 100 m.Eq. sodium bicarbonate was administered, and also 20 gm. Mannitol, and the infusion of aramine was continued. He died 20 hours after operation, without regaining consciousness.

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AUTOPSY

Laminectomy operation had been performed on the neck. There was a fractured disloaation between the 4th and 5th cervical vertebrae with laceration of the spinal cord in this region. Left thoracotomy.

COMMENT:

The episodes of hypotension that occurred during this operation was associated in each case with the slowing of the heart rate, and doubtless were the result of surgical autonomic stimulation from traction on the cervical portion of the spinal cord during dissection of the tumour.

Inadequate replacement of blood loss may have been an added factor but close examination of this aspect does not really support this.

The fact that the ventricular extrasystoles proceeding to cardiac arrest followed injection of a small dose of phenylephrine may implicate this drug in their causation. However, at the same time, systolic blood pressure was unrecordable for 10 minutes and it is more than likely that myocardial ischaemia was the cause. Prolonged hypotension in the sitting position, with acute flexion of the neck, before cardiac massage was commenced must have caused irreversible cerebral ischaemia damage, later manifested by the failure to regain consciousness.

The autopsy finding of a fractured disloaation of the cervical spine between C4 and 5, with disruption of the spinal cord at this level, is interesting and poses the problem of identifying the time at which this occurred. To fix this is impossible. It may have occurred at laminectomy, or while external cardiac massage was applied to the seated patient, or during the time the patient was removed from this position and placed supine for internal cardiac massage. Its occurrence must have ensured the fatal outcome of this case.

IN general, the surgical manoeuvres are thought to be the principal cause of the hypotension and ultimate cardiac arrest. Inadequate replacement of blood loss is a possible secondary factor. The prolonged period of gross hypotension in the sitting position which preceded cardiac massage, and the fractured dislocation of the cervical spine, both ensured the fatal outcome.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
89.2.65	2	No comment	< 24	Subdural and intra-cerebral haemorrhage.	Yes

Name: Gideon Gaza Age: Unknown Sex: M Race: B

Disease: Multiple injuries. Subdural haematoma.

Operation: Carotid angiography. Drainage of subdural haematoma. Repair of fractures.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

This patient was unconscious and had sustained multiple injuries. He had a fractured skull, subdural haematoma and compound fractures of the tibia and fibula. 3 pints blood were transfused before operation. B.P. 120/80 mm.Hg, pulse rate 100/minute and the temperature was 99°F.

PREMEDICATION:

Atropine 0.6 mg., given intravenously immediately before anaesthesia.

ANAESTHETIC:

Following inhalation of nitrous oxide and oxygen, oral intubation was performed after the injection of succinylcholine 50 mg. Tracheo-bronchial toilette was performed. Anaesthesia was then maintained with nitrous oxide and oxygen 50% administered via a Magill semi-open circuit with spontaneous breathing.

Burrhole craniotomy, drainage of the subdural haematoma and repair of compound leg fractures were undertaken. 5 pints blood were transfused during the operation, which took 4½ hours in all. At the conclusion of the procedure, tracheotomy was performed. The patient failed to regain consciousness and died 14 hours after operation.

AUTOPSY

Signs of operation on skull. Multiple lacerations of the face. Depressed fracture of right temporal region of skull. Subdural and intracerebral haemorrhage. Fractures of left tibia and fibula and right olechranon. Tracheotomy wound.

COMMENT:

Death was due to traumatic cerebral injury and the anaesthetic was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
90.2.65	2	No comment	< 24	Gas gangrene of abdominal wall.	No

Name: Alfred de Bruin Age: 63 Sex: M Race: C

Disease: Extravasation of urine, gas gangrene of abdominal wall.

Operation: Suprapubic cystostomy and incision of abdominal wall.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

Extremely ill - there was gas gangrene of the abdominal wall which had followed extravasation of urine. The patient was pyrexial, temperature 100°F, and very toxic. Gross metabolic acidosis with a base deficit of 13 m.Eq./l. was present and this was corrected pre-operatively by the infusion of 273 m.Eq. sodium bicarbonate. 2 pints blood were transfused. Before anaesthesia the B.P. was 150/70 mm.Hg and haemoglobin concentration was 10 gm.%. Albuminuria was present.

PREMEDICATION:

Atropine 0.6 mg. 45 minutes pre-anaesthesia by intramuscular injection.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 125 mg., succinylcholine 50 mg., ventilation with oxygen, and oral intubation. Anaesthesia was maintained with cyclopropane and oxygen via a carbon dioxide circle absorption system by an IPPR technique. The course of anaesthesia was uneventful and the operation lasted 60 minutes. At the conclusion of the procedure, on discontinuance of anaesthesia, normal spontaneous respiration resumed. The patient regained consciousness. A marked deterioration occurred in his condition 12 hours post-operatively and he died 19 hours after surgery.

AUTOPSY

No autopsy

COMMENT:

This patient died as a result of the existing disease. The anaesthetic used was not contributory to death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
91.2.65	2	No comment	< 24	Cerebral trauma.	Yes

Name: Leni Martin Age: 30 Sex: F Race: C

Disease: Head injury. Intracerebral haematoma. Operation: Carotid angiography.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had sustained a head injury and was comatose. B.P. 100/60 mm.Hg, respiration 30/minute.

PREMEDICATION:

Atropine 0.6 mg. by intramuscular injection 45 minutes pre-operatively

ANAESTHETIC:

Oral intubation was performed after inhalation of nitrous oxide and oxygen for 3 minutes, and the injection of succinylcholine 50 mg. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a Magill semi-open circuit with spontaneous respiration. The course of anaesthesia was untoward. At the conclusion of the procedure, the failure of the patient to regain consciousness was followed by death 10 hours post-operatively.

AUTOPSY

Diffuse intracerebral haemorrhage with gross cerebral lacerations.

COMMENT:

This patient died of diffuse neuronal injury. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
92.2.65	3	No comment	ORD	Mitral stenosis. Cardiac arrest.	Yes

Name: Laura Peterson Age: 53 Sex: F Race: C

Disease: Mitral stenosis. Operation: Mitral valvotomy

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

She had severe mitral stenosis, with some calcification of the valve visible on chest X-ray. Cardiac failure was poorly controlled by digitalisation. Congestion of the lungs and orthopnoea were still present. She had been hypertensive, and this had been treated with Aldomet, which had been discontinued 7 days pre-operatively. The B.P. remained at the level it had been at before discontinuance of Aldomet, 130/90 mm.Hg. Pulse rate was 100/minute with auricular fibrillation.

PREMEDICATION:

Sodium seconal gr. 1½ orally 2 hours pre-anaesthesia. Atropine 0.6 mg. 45 minutes before operation by intramuscular injection.

ANAESTHETIC:

Anaesthesia was induced with th sequence thiopentone 150 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx andoral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide absorption system by an IPPR technique. dTc 20 mg. was administered. The B.P. stabilised at a level of 100 mm.Hg systolic. Palpation of the mitral valve via a left atriotomy revealed this to be densely calcified all round. The surgeon decided it was completely unsuitable for commissurotomy and closure of the atriotomy was commenced. After atriotomy and palpation of the mitral valve, there had been a progressive fall in B.P. due to poor ventricular filling and contraction, and poor cardiac output. Blood loss had not been marked and had been replaced by transfusion. Injection of phenylephrine and infusion of adrenaline solution of 1:300,000 produced no response. While closure of the atriotomy progressed, the B.P. fell to unrecordable levels. Cardiac massage was commenced when closure of the cardiotomy was complete, when the heart was still beating but ineffectively. Cardiac massage in the face of severe mitral stenosis was useless: the left ventricle would not fill while the left atrium became more distended and the cardiac musculature rapidly lost its tone. Adrenaline was given intraventricularly, to produce ventricular fibrillation, cardiac massage was continued and electrical defibrillation attempted. This resulted in complete asystole. Mitral commissurotomy as an emergency measure was reconsidered at this stage, but was rejected by the surgeon because of the dense calcification of the valve.

AUTOPSY

Surgical incision on left anterolateral side of chest. Gross tight mitral stenosis with heavy calcification around the complete circumference of the mitral valve. The mitral orifice was a pin-hole. Hypertrophy of the left atrium and dilatation of the right atrium. Emphysema of upper lobes of both lungs.

COMMENT:

Death resulted from low cardiac output following surgical exploration of a densely calcified, tightly stenosed mitral valve. Without commissurotomy the heart could not recover from the fall in cardiac output which resulted and subsequent cardiac massage was ineffective because of the severity of mitral stenosis present. Anaesthesia was perhaps necessarily and unavoidably contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
93.2.65	3	No comment	ORD	Haemorrhage Inadvertent hypothermia. Cardiac arrest.	Yes

Name: Hendrik Schreuder Age: 52 Sex: M Race: E

Disease: Cirrhosis of liver; portal hypertension. Bleeding oesophageal varices.

Operation: Oesophago-gastrectomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient was suffering from bleeding oesophageal varices because of portal hypertension. In the past he had had a splenectomy and a gastric transection for the same complaint. The oesophageal varices had persisted and bleeding from them was again severe. Pre-operative P.I? was 97%. Haemoglobin concentration 11.5 gm.%. B.P. 100/85 mm.Hg and the cardiovascular and pulmonary systems were normal.

PREMEDICATION:

Atropine 0.6 gm., pethilorfan 40 mg., 60 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 350 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was given for relaxation, total dose being 45 mg. throughout the operation, which lasted 5 hours in all. The operation was technically extremely difficult because of the adhesions present and was marked by profuse haemorrhage throughout its course. Though blood replacement kept pace with loss for the first few hours, it slowly fell behind. Blood loss was truly massive - measured in the suction bottles together with an estimate of that lost on swabs totalled 23,000 ml. at the end of operation, when cardiac arrest occurred. By this time 21,600 ml. blood and plasma had been transfused, with 12 gm. calcium gluconate. The patient's temperature had dropped to 32°C. When 9 litres blood had been lost and replaced, the blood clotting mechanism appeared to be impaired. Vitamin K was administered without effect. After 14 litres blood had been lost and replaced, biochemical investigation revealed a marked metabolic acidosis with a base deficit of 14. m.Eq./l. Correction with sodium bicarbonate was commenced and by the time cardiac arrest occurred, 150 m.Eq. had been infused. The operation had been completed and the abdomen resutured, when arrest occurred, and the B.P. had been maintained reasonably until the last hour of surgery, when it fell to near unrecordable levels. Because of this period of hypotension and the intractability of the haemorrhage which continued unabated via the abdominal drains, no cardiac massage was undertaken.

AUTOPSY

Oesophago-gastrectomy had been performed. There were 200 ml. blood in the abdominal cavity. The spleen was missing. There was gross cirrhosis of the liver.

COMMENT:

This death followed truly massive and protracted haemorrhage, and the effects of equally massive transfusion. Anaesthesia is not considered primarily contributory to death but could be considered necessarily and unavoidably contributory. Of the effects of massive blood loss and replacement, inadvertent hypothermia is probably an important contributory cause to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
94.2.65	2	No comment	< 24	Multiple injuries	Yes

Name: Violet Etheridge. Age: 9 Sex: F Race: E

Disease: Multiple injuries, ?ruptured liver. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Unconscious and in oligaemic shock with multiple injuries. Blood transfusion improved her condition but had to be continued or the B.P. fell again. It was assumed that the patient was bleeding from a ruptured liver. Although she had also suffered a head injury, the ruptured liver was considered more urgent.

PREMEDICATION:

Atropine 0.6 mg. given 35 minutes before operation, intramuscularly.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide and oxygen with 0.5% halothane. Oral intubation followed the injection of succinylcholine 25 mg. Anaesthesia was then maintained with nitrous oxide and oxygen, with ether vapour, administered via a Magill semi-open circuit with spontaneous breathing.

Laparotomy failed to reveal any laceration of the liver. The operation took 75 minutes. The course of anaesthesia was untoward. At the conclusion of the procedures, the patient failed to regain consciousness and she died 15 hours post-operatively.

AUTOPSY

Subdural haemorrhage. Subpleural haemorrhage. Bleeding into the left adrenal gland.

COMMENT:

This death was the result of multiple injuries sustained by this patient. Of particular interest is the autopsy finding of haemorrhage into the left adrenal gland as well as subdural bleeding. The anaesthetic administered was not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
95.2.65	2	No comment	< 24	Traumatic cerebral injury.	Yes

Name: Charles Maart Age: Unknown Sex: M Race: C

Disease: Severe head injury. Operation: Carotid angiography. Traeheostomy.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

This patient, who was unconscious, had signs of aspiration pneumonia and was pyrexial (temperature 100°F). Immediately before anaesthesia the B.P. was 140 mm.Hg systolic, respiration 36/minute.

PREMEDICATION:

Nil.

ANAESTHETIC:

After pharyngeal toilette and topical analgesia of the larynx, oral intubation was performed. Anaesthesia was induced and maintained with a 50% mixture of nitrous oxide and oxygen, administered via a Magill semi-open circuit with spontaneous respiration. At the conclusion of angiography, tracheostomy was performed. The patient died 12 hours post-operatively without regaining consciousness.

AUTOPSY

Subdural haemorrhage over the left temporal lobe of the brain. Haemorrhage into the pons. Intraventricular haemorrhage and intracerebral haemorrhage in the frontal lobe of the brain. Tracheostomy wound in neck.

COMMENT:

Death was the result of gross traumatic cerebral injury. Anaesthesia was certainly not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
96.2.65	2	No comment	< 24	Rupture of cardiac aneurysm	Yes

Name: M. Lauridsen Age: 77 Sex: F Race: E

Disease: Cholelithiasis Operation: Cholecystectomy and exploration of common bile duct.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This patient suffered cholelithiasis and had had a previous coronary thrombosis. Signs of left ventricular damage were apparent on ECG. Ventricular extrasystoles were present. B.P. 145/80 mm.Hg, pulse rate was 84/minute. She was not in congestive cardiac failure. On admission to hospital the haemoglobin concentration was 9.5 gm.%. Transfusion of 1 pint blood had raised this to 11 gm.%.

PREMEDICATION:

Atropine 0.6 mg., omnopon 10 mg., given 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 40 mg., oxygenation, topical analgesia of the larynx and oral intubation, and was then maintained with nitrous oxide and oxygen administered via a carbon dioxide absorption system by an IPPR technique. dTc was given as the relaxant, to a total dose of 35 mg. throughout the operation, which lasted 135 minutes. The course of operation and anaesthesia was untoward.

At the conclusion of the procedure, neostigmine 2.5 mg. preceded by atropine 1.2 mg. was given for reversal of residual curarisation. Normal spontaneous respiration resumed and consciousness was rapidly regained. Initially the post-operative course was quite uneventful. The patient was found dead during the night, 7 hours post-operatively.

AUTOPSY

A large haemopericardium. Rupture of cardiac aneurysm formed on the basis of a previous myocardial infarct.

COMMENT:

The anaesthetic administered to this patient was not contributory to her death. This was due to pre-existing myocardial disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
97.2.65	3	No comment	ORD	Peritonitis Cardiac arrest.	Yes

Name: Hendrik Jaoobs Age: 25 Sex: M Race: C

Disease: Multiple injuries. Ruptured viscera. Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was admitted to hospital 34 hours after being hit by a car. He had a grossly distended abdomen, free fluid in the abdominal cavity and gas under the diaphragm. He was grossly shocked. B.P. 90 mm.Hg systolic, pulse rate 135/minute and he was acidotic. Pyelogram showed no function of left kidney. Pre-operatively 2 litres blood, 2 litres plasma, 1 litre replacement fluid and 150 m.Eq. sodium bicarbonate were given. With this, the B.P. rose briefly to 110 mm.Hg systolic but, on arrival in the operating theatre, it had fallen again to 90 mm.Hg systolic and the patient appeared moribund.

PREMEDICATION:

Atropine 0.3 mg., given intravenously immediately before anaesthesia.

ANAESTHETIC:

After topical analgesia of the larynx, oral intubation was performed with the patient awake. Immediately thereafter anaesthesia was induced with thiopentone 125 mg., followed by inhalation of nitrous oxide and oxygen, with minimal ether, with spontaneous breathing. Ether was discontinued after 5 minutes. dTc 15 mg. was given anaesthesia then being maintained with nitrous oxide and oxygen delivered via a non-rebreathing circuit by an IPPR technique, using a Cyclator ventilator. During induction of anaesthesia, the B.P. dropped to 60 mm.Hg systolic and after 10 minutes became unrecordable, remaining so throughout the remainder of the procedure. Energetic attempts at resuscitation were made, to no avail, including serially: rapid transfusion of 2 pints blood, injection of phenylephrine 0.5 mg., calcium gluconate 1 gm., hydrocortisone 500 mg., Mannitol 20 gm., 1 litre replacement fluid, digoxin 0.5 mg., and 150 m.Eq. sodium bicarbonate. Although no B.P. was recordable, the wound edges bled and the blood was pink.

Laparotomy revealed rupture of the colon at the splenic flexure with generalised peritonitis, with collections of pus in the pouch of Douglas, Morrison's pouch and suprahepatic space on the right and subphrenic space on the left. A loop colostomy was performed, multiple drains inserted and the abdomen closed. This took 120 minutes. After the first 45 minutes of operation, the pupils commenced dilating. For the last 45 minutes of operation, oxygen only was administered. After 1 hour the oesophageal temperature dropped to 89°F and by the end of operation was 87°F. As the operation was being concluded, capillary refill ceased and the ECG showed complete asystole. No cardiac massage was undertaken.

AUTOPSY

Haemorrhage into the left side of the neck. Contusion of both lungs with approximately 200 ml. blood in both cavities. Subcapsular haematomas of the liver and spleen. Intestine grossly congested. Gross diffuse peritonitis. Haemorrhage into the left adrenal gland and retroperitoneal haemorrhage around the left kidney. Subcapsular haemorrhage in hilus of left kidney.

COMMENT:

This patient was moribund when anaesthesia was induced. Although induction appears to have precipitated the final gross hypotension, anaesthesia is regarded as only necessarily and unavoidably

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contributory to a death which was inevitable. The administration of thiopentone, even in the small dose given, is perhaps open to criticism in this case. A valuable drug for stimulation of cardiac output which was not used is isoprenaline. Inadvertent hypothermia was a probable contributory factor in the unresponsiveness of this patient to resuscitative measures and the ultimate cardiac arrest.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
98.2.65	3	No comment	ORD	Haemorrhage	Yes

Name: Kathrina Louw Age: 77 Sex: F Race: C

Disease: Tumour of liver. Operation: Laparotomy. Biopsy of liver.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

This patient had a large liver mass which was rapidly increasing in size, and was also in congestive cardiac failure in spite of energetic medical treatment with digitalis, diuretics and bed-rest. As there was no further improvement in her cardiac condition, despite prolonged medical treatment, and the liver mass was increasing in size, laparotomy was decided on.

PREMEDICATION:

Morphine 8 mg., atropine 0.3 mg. intramuscularly 60 minutes pre-operativel

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 100 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation, and was then maintained with nitrous oxide and oxygen, administered via a circle absorption system by an IPPR technique. A trace of ether vapour was administered intermittently. dTc was used for relaxation, total dose 30 mg. In addition to the B.P. and pulse rate, the ECG and venous pressure were monitored throughout the operation. The course of operation and anaesthesia were untoward until attempts were made to biopsy the large friable liver mass, revealed by laparotomy. This precipitated a massive haemorrhage which was ultimately uncontrollable. 6 pints blood were transfused rapidly within the following 40 minutes. The B.P. and venous pressure dropped during haemorrhage and, after transfusion, the venous pressure rose rapidly to 20 cm.water although the B.P. remained at a level of 70 mm.Hg systolic. Infusion of a solution of isoprenaline 1:300,000 was commenced without effect. The oesophageal temperature was now 31.8°C and cardiac arrest ensued. No cardiac massage was instituted.

AUTOPSY

Large diffuse carcinoma of the liver. Gross intraperitoneal haemorrhage. Body tissues pale and exsanguinated.

COMMENT:

This death was the result of massive haemorrhage and the effect of massive, rapid transfusion of blood. Inadvertent hypothermia from the latter was doubtless a factor contributing to the final acute cardiac failure.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
99.2.65	2	No comment	<24	Cerebral injury	Yes

Name: Wilson Nobola Age: 26 Sex: M Race: B

Disease: Head injury. Cerebral laceration. Operation: Burrhole craniotomy. Evacuation of subdural haematoma.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Comatose. Cardiovascular and respiratory systems appeared normal.

PREMEDICATION:

Atropine 0.6 mg. intravenously immediately before anaesthesia.

ANAESTHETIC:

After pharyngeal toilette and topical analgesia of the larynx, oral intubation was performed. Anaesthesia was then induced and maintained with nitrous oxide and oxygen administered via a Magill semi-open circuit, with spontaneous breathing. The course of anaesthesia was untoward. At the conclusion of craniotomy. a tracheostomy was performed.

The patient died 12 hours post-operatively without regaining consciousness.

AUTOPSY

Multiple sutured scalp wounds. Surgical burrholes of skull. Extensive cerebral contusion and bilateral subdural haemorrhages. Bruise of right upper arm. Extensive bruising of scalp.

COMMENT:

This patient died of extensive traumatic cerebral injuries. The anaesthetic administered was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
100.2.65	2	No comment	< 24	Haemorrhage	Yes

Name: Elizabeth Matthews Age: 54 Sex: F Race: C

Disease: Renal tumour Operation: Laparotomy. Right nephrectomy.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient presented with fever and a tender mass in the right hypochondrium. There was evidence of chronic renal disease. She had a history of asthma and scattered wheezing rhonchi were audible on auscultation.

PREMEDICATION:

Aminophyllin suppository 7½ gr., and pethidine 50 mg.,, atropine 0.6 mg. by intramuscular injection 1 hour pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Gallamine was used as the relaxant. An initial dose of 80 mg. was found to have been injected extravenously into the tissues. Hyalase was injected to ensure rapid absorption and a subsequent dose of 80 mg. was given. At laparotomy the mass was found to be a pyelonephritic kidney with an infected perirenal haematoma adherent to the under-surface of the liver. Right nephrectomy resulted in massive haemorrhage. The replacement with warmed blood kept pace with haemorrhage, as far as could be ascertained, a total of 12 pints blood being lost and replaced. The course of anaesthesia, especially in the early phase, was punctuated by intermittent falls in B.P. This was due at times to inferior vena caval compression. On occasion it was due to sudden spurts of haemorrhage with temporary lag in replacement. Great difficulty was experienced in controlling the haemorrhage from the under-surface of the liver. Finally, recourse was had to packing with surgical gauze. Towards the end of operation, biochemical investigation (Astrup method) showed a mild metabolic acidosis. This was corrected with infusion of sodium bicarbonate. At the conclusion of operation, which lasted 3 hours, neostigmine 2.5 mg. preceded by atropine 1.2 mg. was given to reverse any residual curarisation. Spontaneous respiration resumed which, though initially inadequate, soon became normal. The patient rapidly regained consciousness on discontinuance of anaesthetic, and had normal muscle power. 2 hours post-operatively, blood loss of some volume was evident from the surgical drains. Blood was transfused. However, the patient's condition continued to deteriorate and she died 6 hours post-operatively.

AUTOPSY

200 ml. blood in peritoneal cavity. Right kidney had been removed. Calcified cyst in liver. Operative incision in liver, stitched and packed with gauze.

COMMENT:

The probable cause of this patient's death was continued haemorrhage from a large raw area on theliver, to which the pyelonephritic kidney had been adherent. Anaesthesia was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
101.2.65	2	No comment	< 24	Diffuse neuronal injury.	Yes

Name: Hennie Bester Age: 20 Sex: M Race: E

Disease: Head injury. Operation: Carotid angiography. Tracheostomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient was deeply comatose following a head injury. He had also sustained a fracture of the right femur. B.P. 90/60 mm.Hg and pulse rate 130/minute.

PREMEDICATION:

Atropine 0.6 mg. by intramuscular injection, 75 minutes pre-anaesthesia.

ANAESTHETIC:

Following pharyngeal toilette and topical analgesia of the larynx, oral intubation was performed. Thereafter anaesthesia was maintained with nitrous oxide and oxygen delivered via a carbon dioxide circle absorption system, by an IPPR technique. No relaxant drug was used. The B.P. remained steady at the pre-anaesthetic level throughout.

At the conclusion of angiography, tracheostomy was performed. Normal spontaneous respiration resumed. The patient died 8 hours post-operatively without regaining consciousness.

AUTOPSY

Small scattered haemorrhages in brain and cerebellum. Brain convolutions flattened. Tracheostomy wound. Fracture of right femur. Haemorrhage into tissues of right orbit and over the right mandible and right side of neck. Bruise over sternum. No fractures of skull. Subcutaneous haemorrhage over whole scalp. Congestion of the lungs.

COMMENT:

The patient died of diffuse neuronal injury. The anaesthetic he received was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
102.2.65	2	No comment	< 24	Post-operative intra-cerebral haemorrhage	Yes

Name: Leslie Smith Age: 32 Sex: M Race: C

Disease: Posterior fossa tumour Operation: Posterior fossa craniotomy.

Anaesthetic risk: 1

PRE-OPERATIVE STATE:

Except for the symptoms and signs of a right posterior fossa tumour, this patient's condition was normal.

PREMEDICATION:

Atropine 0.6 mg. given 60 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 375 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx, and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique, using a Pulmomat ventilator. A minute volume of ventilation of 12 l/minute was maintained throughout the operation. dTc was given in divided doses, a total of 40 mg. being used throughout the operation, which lasted 4½ hours. The patient was positioned sitting with the head flexed acutely forward. Blood was replaced as lost, a total of 600 ml. being transfused. The only untoward incident in the course of anaesthesia and operation was the transient bradycardia and hypotension which followed manipulation of the brain stem during surgery. Cardiovascular homeostasis reverted to normal immediately manipulation ceased. At the conclusion of the procedure, residual curarisation was reversed with neostigmine 1.5 mg preceded by atropine 1.2 mg. Normal spontaneous respiration resumed and the patient rapidly regained consciousness. He lost consciousness rapidly 12 hours after operation and died 15 hours post-operatively. Histologically, the excised tumour was a haemangioblastoma.

AUTOPSY

Haemorrhage into the neck muscles. Extensive haemorrhage into the cerebellum pons and mid-brain. Reverse tentorial coning.

COMMENT:

This death resulted from intracranial haemorrhage from the operative site, with reverse coning. Anaesthesia was not contributory to the death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
103.2.65	2	No comment	< 24	Intra-cranial haemorrhage	Yes

Name: Unknown. Age: Unknown. Sex: M Race: C

Disease: Multiple injuries; compound fractures left tibia and fibula. Fractured skull. Subdural haematoma.

Operation: Carotid angiography. Burrhole craniotomy. Reduction of fractures.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had sustained multiple injuries and was unconscious. Oligaemic shock was present on admission to hospital. 3 pints blood were transfused before operation. B.P. 90 mm.Hg systolic at this stage. Tracheostomy was performed before operation.

PREMEDICATION: Nil.

ANAESTHETIC:

Carotid angiography was performed without anaesthetic. Anaesthesia for the burrhole craniotomy for evacuation of subdural haematoma was induced with nitrous oxide and oxygen with 0.5% halothane, administered via the tracheostome, using a Magill semi-open circuit with spontaneous respiration.

The compound fractures were debrided, manipulated and set. General cardiovascular condition remained static throughout the operation. The patient died 1 hour post-operatively without regaining consciousness.

AUTOPSY

Fracture of left parietal region of skull. Two burrholes in the left parietal bone. Fractures of left and right tibia and fibula. Tracheotomy. Intracerebral haemorrhage in left cerebral hemisphere and gross haemorrhage into pons.

COMMENT:

Death was due to cerebral haemorrhage and to the multiple injuries. The anaesthetic administered to this patient was not contributory.

CASE NO.	CLASSIFICATION Group	pREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
1o4.2.65	3	No comment	ORD	Coronary thrombosis	Yes

Name: James Sawyer Age: 75 Sex: M Race: C

Disease: Carcinoma of prostate. Operation: Transurethral resection of prostate.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had urinary obstruction because of carcinoma of the prostate. His physical status was fair for his age. There was mild pulmonary emphysema. There was atherosclerosis and, though there was no history of ischaemic heart disease, occasional extrasystoles were palpable in the pulse. B.P. 120/80 mm.Hg. No ECG was performed.

PREMEDICATION:

Aminophyllin gr. 7½ suppository 1½ hours pre-operatively. Atropine 0.6 mg. by intramuscular injection 1½ hours pre-operatively.

ANAESTHETIC:

Anaesthesia was indueed with the sequence thiopentone 150 mg., succinylcholine 40 mg., ventilation with oxygen, analgesia of the larynx and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was given, total dose 30 mg. during the operation. Blood loss did not appear to be great during surgery; 150 ml. 5% dextrose in water was infused and the B.P. remained steady at between 120 and 140 mm.Hg systolic, the pulse rate remaining between 80 and 90/minute. When operation had been in progress for 1 hour and was all but complete, cardiac arrest occurred suddenly, after the B.P. had been recorded as 120 mm.Hg systolic only3 minutes before. External cardiac massage was commenced immediately while IPPR with oxygen only was continued. This did not produce a palpable pulse and the pupils remained dilated. After 2 minutes, thoracotomy was performed and internal cardiac massage commenced. In the process, a laceration was made in the left atrium. Cardiac massage was continued intermittently while attempts were made to suture this incision. At the same time, 2 pints blood were rapidly transfused and 1 mg. phenylephrine, 2 gm. calcium gluconate and 100 m.Eq. sodium bicarbonate were administered. Adrenaline solution, 1 ml. of 1:20,000 was injected intraventricularly. The heart was completely unresponsive and all resuscitative efforts were abandoned after 25 minutes.

AUTOPSY

Left haemothorax of 1,200 ml. Collapse of left lung. Contusion of pericardium. Coronary atherosclerosis with fresh obstruction of left coronary artery. Heart was soft and flabby.

COMMENT:

No adequate explanation for this death emerged from examination of the clinical records. The autopsy provided the answer, although why occlusion of the coronary artery should occur at this time is unknown. No episodes of hypotension occurred during the procedure. A hazard peculiar to transurethral resection of the prostate is haemodilution from absorption of water used for irrigation of the bladder; it is difficult to see how this could have contributed to this coronary thrombosis, however. Efforts at resuscitation were markedly hampered by the inadvertent incision of the left atrial wall. As cardiac arrest occurred while the patient was anaesthetised, the death is classed in group 3, although no contributory role was played by the anaesthetic as far as can be identified.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
105.2.65	2	No comment	< 24	Respiratory depression. Cardiac arrest.	Yes

Name: Arthur Peterson Age: 47 Sex: M Race: C

Disease: Pott's disease of the spine. Operation: Anterolateral decompression of the spinal cord.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This small patient (weight 80 lb.) who had Pott's disease of the spine required an anterolateral decompression of the spinal cord because of compression. The severe thoracic kyphosis had caused marked deformity of the chest and reduction in vital capacity.

PREMEDICATION:

Atropine 0.6 mg., pethidine 25 mg. intramuscularly 35 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system, by an IPPR technique. dTc was given in divided doses, total dose being 34 mg. during the 165 minutes of operation. The course of operation and anaesthesia were untoward. At the conclusion of the procedure, residual curarisation was reversed by the administration of neostigmine 2.5 mg. after atropine 1.2 mg. had been given. Normal spontaneous respiration resumed and the patient rapidly regained consciousness. The post-operative course was uneventful until medication was considered necessary for post-operative pain, 3 hours after surgery. The house-surgeon ordered 100 mg. pethidine. Within minutes of its administration, the patient ceased breathing. Cardiac arrest ensued. External cardiac massage was commenced shortly after but in vain.

AUTOPSY

Healed spinal tuberculosis with gibbus. Anterolateral decompression had been performed. Two small cerebellar cysts. Markedly reduced vital capacity.

COMMENT:

Death was due to respiratory depression and failure following administration of pethidine for post-operative pain. The dose given to so small a patient, especially with reduced vital capacity, was unduly large. The rapidity with which respiratory failure followed injection of the pethidine suggests that inadvertent intravenous injection may have occurred. This mishap was not directly concerned with the anaesthetic administered to the patient.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
106.2.65	2	No comment	< 24	Cerebral abscess	No

Name; Hendrik van Wyk Age: 23 Sex: M Race: C

Disease: Cerebral abscess. Operation: Carotid angiography. Burrhole craniotomy and aspiration of right temporal lobe abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient had recently had acute mastoiditis and mastoidectomy. He was now suffering from cerebral abscess and appearedtoxic. There had been no difficulty with the general anaesthetic administered for mastoidectomy. Cardiovascular and respiratory systems appeared normal. B.P. 120/80 mm.Hg, pulse rate 120/minute.

PREMEDICATION

Atropine 0.6 mg. intravenously, immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 125 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation, and thereafter was continued with nitrous oxide and oxygen with 0.5% halothane, administered via a Magill circuit (semi-open) with spontaneous breathing.

Carotid angiography was followed by a burrhole craniotomy and evacuation of the temporal lobe abscess. There was underlying cerebral softening. The course of operation and anaesthesia was uneventful. At the conclusion of the procedure, which lasted 150 minutes, the patient regained consciousness and had normal spontaneous respiration. He died 9 hours post-operatively.

AUTOPSY

No autopsy

COMMENT:

The cause of this patient's death, although not known for certain, was probably related to the existing disease. Anaesthesia is not considered to have been contributory to death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
107.2.65	2	No comment	< 24	Intra-cerebral secondary carcinomatous deposits.	Yes

Name: C. Blom Age: 45 Sex: M Race: E

Disease: Left hemiplegia, ?cerebral thrombosis. Operation: Right frontal burrhole craniotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient was stuperose, restless and had a left hemiplegia. His state of consciousness was steadily worsening. Cardiovascular and respiratory systems were normal. B.P. 110/70 mm.Hg and pulse rate 100/minute. Carotid angiography had been performed previously

PREMEDICATION:

Atropine 0.6 mg. given 30 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. dTc 20mg. was administered as relaxant.

A right parietal burrhole craniotomy did not reveal intracranial haemorrhage, as had been suspected. The course of anaesthesia was uneventful. At the conclusion of the procedure, which lasted 50 minutes, neostigmine 2.5 mg. preceded by atropine 1.2 mg. was given for adequte reversal of residual curarisation. Normal spontaneous respiration resumed and the patient recovered consciousness to the same stuperose state as had been present pre-operatively. He died 16 hours after operation.

AUTOPSY

Craniotomy wound. Large cell undifferentiated carcinoma arising from the left upper main bronchus. Diffuse cerebral metastases Cerebral oedema. Tumour metastases to pleura and left upper lobe of lung.

COMMENT:

Death resulted from intracranial secondary carcinomatous deposits. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
108.2.65	2	No comment	< 24	Mesenteric thrombosis	Yes

Name: Mrs. A.O'Kennedy Age: 68 Sex: F Race: E

Disease: Mesenteric thrombosis. Operation: Laparotomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient presented in poor physical state with severe peritonitis from what was thought to be perforation of diverticulitis. A known hypertensive (normal B.P. 220/120 mm.Hg) the present B.P. was only 120/60 mm.Hg. Despite good respiratory excursion and the lungs appeared normal, there was marked peripheral cyanosis. A known diabetic, on admission both glycosuria and ketonuria were present. Gastric aspiration and rehydration therapy were instituted and insulin therapy was commenced. The patient was submitted to laparotomy.

PREMEDICATION:

Atropine 0.6 mg. given 30 minutes pre-operatively.

ANAESTHETIC:

Immediately before anaesthesia, the B.P. was 120/60 mm.Hg and peripheral cyanosis was still present. Anaesthesia was induced with nitrous oxide and oxygen with 10-15% cyclopropane, administered via a carbon dioxide circle absorption system with spontaneous breathing. As soon as consciousness was lost, succinylcholine 50 mg. was injected and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen with 10-20% cyclopropane by an IPPR technique. During operation, which lasted 65 minutes, 1 pint blood and 1 litre Rheomacrodex were infused. The B.P. rose steadily, reaching a level of 160 mm.Hg systolic by the end of the procedure.

Laparotomy revealed mesenteric thrombosis with patchy areas of infarction throughout the large and small bowel. The condition was quite inoperable. During closure of the abdomen, succinylcholine 40 mg. was administered. On conclusion of the anaesthetic, spontaneous respiration resumed. The patient regained consciousness rapidly but died 12 hours post-operatively.

AUTOPSY

Extensive patchy infarction of both small and large intestines. Acute peritonitis. Atheromatous plaques in superior mesenteric artery but no occlusive lesions demonstrated; stenosis of the ostium of the coeliac axis but little atheroma beyond this point. Left ventricular hypertrophy and dilatation due to long-standing essential hypertension. Recent subcapsular infarct involving right and left lobes of the liver. Recent laparotomy wound.

COMMENT:

Anaesthesia was not contributory to this death, which resulted from diffuse infarction of the gut.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSEOF DEATH	AUTOPSY
109.2.65	2	No comment	< 24	Cerebral embolisa-tion.	Yes

Name: Arthur Cockerell Age: 21 Sex: M Race: E

Disease: Post mitral valvotomy intrathoracic bleeding. Operation: Left thoracotomy

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

The previous day this patient had undergone mitral valvotomy for mitral stenosis. Extensive embolisation had occurred and the patient failed to regain consciousness. Intrathoracic haemorrhage from the operative wound now necessitated thoracotomy to control this. Before anaesthetic, although the B.P. was maintained at a level of 120 mm.Hg systolic by blood transfusion, the pulse rate was 110/minute and the patient appeared shocked; tachypnoea of 30/minute was present.

PREMEDICATION

Atropine 0.6 mg. given just before anaesthesia.

ANAESTHETIC:

After pre-oxygenation and injection of succinylcholine, and oxygenation with IPPR, oral endotracheal intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and 0.5% halothane, via a carbon dioxide circle absorption system, by an IPPR technique. dTc 20 mg. was given at the time of thoracotomy. Clots were evacuated from the left pleural cavity and the source of bleeding was found and secured. The course of anaesthetic was untoward. At the conclusion of the procedure, which lasted 90 minutes, normal spontaneous respiration was resumed after reversal of residual curarisation by the administration of neostigmine 2.5 mg. preceded by atropine 0.6 mg. At the end of the anaesthetic the patient's condition was unchanged from the pre-operative state and he died 20 hours post-operatively.

AUTOPSY

Thrombus in left carotid artery. Mitral valvotomy with old thrombus over operation wound, part of which appeared to have been dislodged. Right renal infarction. Duodenal ileus.

COMMENT:

The patient died from cerebral emboli dislodged during mitral valvotomy. Neither of the general anaesthetics administered before death played any contributory role in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
110.2.65	1	Possibly	ORD	Anoxic anoxia. (Atmospheric anoxia)	Yes

Name: Peter van der Westhuizen Age: 73 Sex: M Race; E

Disease: Dehiscence of abdominal wound. Pelvic abscess. Operation: Resuture of dehisced abdominal wound.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

The patient had had a laparotomy 2 weeks previously, when a right psoas abscess was drained. After operation severe chest infection developed with copious purulent sputum. During a bout of coughing the abdominal wound disrupted 2 weeks post-operatively. He immediately became grossly shocked. B.P. 100/70 mm.Hg, pulse rate 120/minute. Severe peripheral vasoconstriction and cyanosis were present. Urinary output for the 24 hours before operation was 700 ml. and the blood urea had risen to 54 mg.%. Immediately pre-anaesthesia the B.P. became unrecordable. 1 pint blood was transfused. This produced no change in the B.P.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection, 60 minutes pre-operatively.

ANAESTHETIC:

On commencement of induction of anaesthesia, the B.P. was 100 mm.Hg systolic. Using a carbon dioxide circle absorption system, the anaesthetist administered pure oxygen for 2 minutes. Anaesthesia was then induced with cyclopropane and oxygen via the same system, with spontaneous breathing. After 2-3 minutes the patient appeared to lose consciousness. Succinylcholine 40 mg. was injected and when paralysis supervened, and IPPR was attempted, this was found not possible. Due to failure of the anaesthetist to check the anaesthetic machine properly the fact that it was incorrectly connected had escaped notice. The fresh gas flow and oxygen was connected through the semi-open circuit, whereas the anaesthetist was using the carbon dioxide circle absorption circuit. This mistake was rapidly rectified, however, when the patient was thought to be inhaling oxygen and later cyclopropane and oxygen, he in fact was merely rebreathing his own exhalations. This probably resulted in anoxia. On correction of this error, IPPR with oxygen was instituted for 1 minute and oral intubation performed. Anaesthesia was then maintained with nitrous oxide and oxygen, with a trace of ether, administered via the carbon dioxide circle absorption system by an IPPR technique. After this induction of anaesthesia, the B.P. had fallen to 90 mm.Hg systolic and the pulse rate had risen to 130/minute. Blood transfusion was commenced and 0.5 mg. phenylephrine was given, with 0.5 mg. digoxin, and then 100 m.Eq. sodium bicarbonate was infused. Operation was commenced. The pulse rate slowed to 100/minute and the B.P. continued to fall and, within a further 5 minutes, had reached the level of 60 mm.Hg systolic. Normal sinus rhythm was still apparent on the ECG. Two units plasma (500 ml.) were rapidly infused, with 1 mg. phenylephrine. The B.P. continued falling, however, and infusion of 1:200,000 solution of adrenaline was commenced. The B.P. was not accurately recordable, but the carotid pulses were palpable when ventricular extrasystoles commended. These proceeded to ventricular fibrillation after 10 minutes. The operation had been inprogress for 30 minutes at this time. The pupils were widely dilated. External cardiac massage was instituted and a carotid pulse could be felt, but the pupils remained dilated and the periphery grey and vasoconstricted. 15 m.Eq. sodium bicarbonate was infused and 5 minutes after the onset of ventricular fibrillation, left thoracotomy was performed and internal cardiac massage commenced. After intraventricular injection of 5 ml. 1:200,000 adrenaline solution, electrical defibrillation was attempted. This and several further attempts were in vain and all resuscitative measures were abandoned after 30 minutes.

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AUTOPSY

Surgical wound in abdomen and left thoracotomy. 600 ml. blood in left pleural cavity. Oedema of lungs. Purulent chronic bronchitis.

COMMENT:

This case is difficult to assess. There are good reasons for classifying it as an inevitable death in a patient moribund before induction of anaesthesia. However, the clinical error during the induction in this very sick patient must have rendered him anoxic. From the time of induction onward, the cardiac output fell steadily and this state proved refractory to all treatment.

Because there was a technical error during the induction of the anaesthetic, which probably resulted in the patient's becoming anoxic and may have caused the subsequent events which led to death, the management of the anaesthetic is regarded as significantly contributory to this patient's death. This is regarded thus even though the patient was in all probability moribund before the commencement of induction of anaesthesia. The ultimate cause of ventricular fibrillation was doubtless prolonged hypotension and subsequent myocardial ischaemia.

PREVENTABILITY

As the clinical error during the induction of anaesthesia could have been avoided by the anaesthetist checking his equipment before administering the anaesthetic, this death is regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
111.2.65	1	Possibly	< 24	Rupture of berry aneurysm. Subarachnoid haemorrhage. Cerebral ischaemia.	Yes

Name: John Elverson Age: 32 Sex: M Race: E

Disease: Intracranial berry aneurysm. Operation: Craniotomy. Clipping of berry aneurysm (abandoned).

Anaesthetic risk: 1.

PRE-OPERATIVE STATE:

Normal except for the intracranial lesion, which was quiescent. Signs and symptoms of a previous leak from the berry aneurysm had cleared. B.P. before anaesthesia was 130/75 mm.Hg, pulse rate 52/minute.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and endotracheal intubation. Anaesthesia was maintained with nitrous oxide and ether, and oxygen, administered via a carbon dioxide circle absorption system by an IPPR technique, while surface cooling was induced, with a water circulating mattress. While cooling was in progress, 90 gm. Urovert was infused intravenously. About 15 minutes after commencing this infusion, the B.P. climbed rapidly to 230 mm.Hg systolic and the pulse rate slowed to 40/minute. This state was maintained for about 5 minutes, when the B.P. commenced to fall and slowly returned to 130 mm.Hg systolic, the pulse rising to 56/minute. A good diuresis ensued. The temperature was reduced to 30°C and cooling was stopped, the operation commencing. The administration of ether was discontinued. dTc 20 mg. was given and then anaesthesia was maintained with nitrous oxide and oxygen only. An afterdrop of 2°C, down to 28°C occurred. Except for the dramatic rise in B.P. after administration of Urovert, the cardiovascular homeostasis was normal and the B.P. remained stable. Craniotomy revealed a brain so tense that the operative procedure was abandoned. It was assumed that the aneurysm had ruptured. After closure of the craniotomy, rewarming to 35°C was performed. Neostigmine 2.5 mg. preceded by atropine 1.2 mg. was administered for reversal of residual curarisation, and normal spontaneous respiration resumed. The patient failed to regain consciousness after the operation and died 5 hours later.

AUTOPSY

Berry aneurysm of the right anterior cerebral artery with rupture through the right frontal lobe into the ventricular system. Large intracerebral haematoma of the right frontal lobe.

COMMENT:

Berry aneurysms which have leaked pre-operatively causing subarachnoid haemorrhage are liable to rupture. This is especially the case if any incident during administration of anaesthetic causes the B.P. to rise (e.g. coughing). The administration of Urovert for reduction of brain bulk and tension is known to temporarily increase the blood volume and cause a rise in B.P., occasionally quite severe. This was the case here. Nothing was done to prevent the rise in B.P. The aneurysm

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ruptured some time after the induction of anaesthesia and craniotomy. It is reasonable to suppose that this happened at the time the B.P. rose so severely, when marked bradycardia was evident.

The administration of Urovert is now a standard part of the anaesthetic technique for certain neurosurgical procedures and the rise in B.P. which so often occurs is well recognised, and is possibly avoidable. The anaesthetic management in this case is thus considered the major factor contributing to the patient's death.

PREVENTABILITY:

A berry aneurysm is liable to rupture. It is known that the administration of Urovert may cause a systemic B.P. rise. If anticipated, the rise in B.P. could be prevented or modified by the administration of vasodilator drugs, such as halothane or by ganglioplegic drugs. This was not done in this case and so the death must be considered to have been possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
112.2.65	2	No comment	< 24	Undetermined.	No

Name: Leonora Phillips Age: 21 Sex: F Race: C

Disease: Cerebral abscess. Operation: Carotid angiography. Burrhole craniotomy. Evacuation of cerebral abscess.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient had had acute mastoiditis for which mastoidectomy had been performed. Subsequently her condition deteriorated and she had now become stuperose. She was pyrexial and appeared toxic. A cerebral abscess was diagnosed.

PREMEDICATION

Atropine 0.6 mg. by intravenous injection immediately pre-anaesthetic.

ANAESTHETIC:

Anaesthesia was induced with the sequence nitrous oxide and oxygen inhalation, succinylcholine 50 mg., IPPR with oxygen, topical analgesia of the larynx, oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. dTc 15 mg. was administered. Following carotid angiography, a temporal burrhole craniotomy was performed and a large temporal lobe abscess was found and 41 ml. pus evacuated. The course of anaesthesia and operation were untoward.

On conclusion of the procedure, which lasted 90 minutes, residual curarisation was reversed with 2 mg. neostigmine preceded by atropine 1.2 mg. Normal spontaneous respiration resumed. The patient's level of consciousness returned to the pre-operative level. Very suddenly, 7 hours post-operatively, the level of consciousness deepened and she died 12 hours after surgery.

AUTOPSY

No autopsy

COMMENT

Death does not appear to be related to the anaesthetic administered to this patient. The question was posed as to whether this death was possibly related to adrenal insufficiency because of previous steroid therapy, about which neither the surgeon nor the anaesthetist was informed, and for which no cover therapy was provided. This is possible, but haemorrhage into the large abscess cavity is more likely to have caused death. Without autopsy this cannot be decided.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
113.2.65	2	No comment	< 24	Continued haemorrhage	No

Name: Joseph Neillson Age: 63 Sex: M Race: E

Disease: Haematemesis. Duodenal ulcer. Operation: Partial gastrectomy

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

The patient had had a repeat haematemesis from a bleeding peptic ulcer. Before anaesthesia, blood had been adequately replaced and he was in fair physical conditon. B.P. 120 mm.Hg systolic, pulse rate 96/minute.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 60 minutes pre-operatively.

ANAESTHETIC

Anaesthesia was induced with the sequence thus: pre-oxygenation, thiopentone 150 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide absorption circuit by an IPPR technique. dTc was given for relaxation, the total dose being 30 mg. during operation, which lasted 2 hours. No ulcer was found in the stomach at operation. It was thought to have been transected in the duodenum during the operation of partial gastrectomy. The course of anaesthesia was untoward.

On conclusion of the procedure, residual curarisation was reversed with neostigmine 2 mg. preceded by atropine 0.6 mg. and normal spontaneous respiration resumed. As the patient regained consciousness, a large haematemesis occurred. The endotracheal tube was left in situ to prevent inhalation of blood. Four further haematemeses occurred. The patient was kept in the operating theatre under observation, the endotracheal tube in situ, for 1½ hours post-operatively. After this, the haematemesis appeared to have ceased. The post-operative course was then uncomplicated until 7 hours after operation, when the patient had a further massive haematemesis and drowned with inhaled blood before anything could be done to prevent this.

AUTOPSY

No autopsy

COMMENT:

Death was the result of continued bleeding from a duodenal ulcer. The accumulation of so vast a quantity of blood in the stomach must have been caused by inefficient gastric drainage, perhaps due to blockage of the nasogastric aspiration tube.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
114.2.65	2	No comment	< 24	Periton-itis.	No

Name: Jackson Plaatjies AgeL 50 Sex: M Race: B

Disease: Carcinoma of the oesophagus. Blockage of Celestine tube. Operation: Oesophagoscopy

Anaesthetic risk: 3,emergency.

PRE-OPERATIVE STATE:

The patient had a carcinoma of the oesophagus for which a Celestine tube had been inserted previously. This had penetrated the stomach wall. The patient had peritonitis and the Celestine tube had become blocked by food. Oesophagoscopy was necessary to clear the tube and to pass a Ryle's tube, to drain the stomach. The patient was dehydrated and moribund. B.P. was 80 mm.Hg systolic.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 45 minutes pre-operatively.

ANAESTHETIC:

Immediately before anaesthesia, 500 ml. plasma was rapidly infused. Anaesthesia was then induced with inhalation of nitrous oxide and oxygen with 1% halothane. Oral intubation was performed. Induction of anaesthesia produced a marked drop in B.P. to 60 mm.Hg systolic, but this recovered to 80 mm.Hg systolic.

During oesophagoscopy, anaesthesia was maintained with nitrous oxide and oxygen only, administered via a Magill semi-open circuit by an IPPR technique. At the conclusion of the procedure, which lasted 30 minutes, the patient regained consciousness rapidly. He died 7 hours later.

AUTOPSY

No autopsy

COMMENT:

Anaesthesia is not considered contributory to this death, which was caused by perforation of the stomach by a Celestine tube, with resultant peritonitis.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
115.2.65	2	No comment	< 24	Fungal arteritis. Fungal lympho-adenitis. Infarction of small bowel. Tubular necrosis of kidney.	Yes

Name: Gilbert Qiqa Age: 29 Sex: M Race: B

Disease: Liver abscess. Subphrenic abscess. Generalised peritonitis. Ruptured jejunum. Operation: Laparotomy

Anaesthetic risk: 3, emergency

PRE-OPERATIVE STATE:

Thispatient, who suffered chronicrenal failure (blood urea 200 mg.%) presented with what was diagnosed as a liver abscess. Attempts to aspirate this had been unsuccessful. After admission to hospital his general condition deteriorated and he developed obvious clinical evidence of generalised peritonitis. A right pleural effusion was present. Haemoglobin concentration 8 gm.%. B.P. 120 mm.Hg systolic with a pulse rate of 120/minute. He appeared grossly toxic. 2 pints packed cells were transfused before operation.

PREMEDICATION: Nil

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide and oxygen. Succinylcholine 35 mg. was given and oral endotracheal intubation performed. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Succinylcholine was used for relaxation, in divided doses, a total of 125 mg. being used during the procedure, which lasted 50 minutes. Atlaparotomy, generalised peritonitis was found with some loculated pus above the dome of the diaphragm, but no intrahepatic abscess was palpable. He also had a ruptured jejunum just beyond the duodenojejunal flexure, which was sutured. Multiple drains were inserted and the abdomen was closed. 500 ml. 10% invert sugar and 600 ml. blood were administered during the operation. Thecourse of anaesthesia was untoward. On conclusion of the procedure, normal spontaneous respiration of adequate volume returned and the patient regained consciousness rapidly. He died 20 hours post-operatively.

AUTOPSY

Gross jaundice. Generalised peritonitis. Liver abscess. Tubular necrosis of kidney. Bilateral hydrothorax. Infarction of almost the entire small bowel. Fungal arteritis. Fungal lymphadenitis.

COMMENT:

The anaesthetic was not contributory to this patient's death, which resulted from his original disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
116.2.65	1	Probably	>24	Post-relaxant respiratory abnormality Anoxic anoxia	Yes

Name: Hendrik Julius Age: 55 Sex: M Race: C

Disease: Obstructed inguinal hernia. Operation: Laparotomy. Repair of inguinal hernia.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

The patient presented with small bowel obstruction due to an irreducible inguinal hernia. In addition, he had severe bronchiectasis with chronic obstructive airway disease. He was cyanosed breathing air. Dehydration and metabolic acidosis were corrected before operation. B.P. 155/95 mm.Hg, pulse rate 110/minute.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 60 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with the sequence oxygen inhalation for 4 minutes, thiopentone 350 mg., succinylcholine 50 mg., oxygenation, oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. dTc was used as the relaxant, a total dose of 27 mg. being given during the operation, which lasted 100 minutes. The course of operation was uncomplicated. The patient's pulmonary compliance was noted to be extremely low. Frequent tracheo-bronchial toilette was performed via the endotracheal tube. At the conclusion of the operation, spontaneous respiration resumed but was grossly inadequate. Neostigmine 3.5 mg. preceded by atropine 1.2 mg., and a further dose of neostigmine 1.5 mg. preceded by 0.6 mg. atropine did not improve the volume of respiration. While assessing the need for post-operative mechanical pulmonary ventilation, the anaesthetist permitted the patient to breathe spontaneously - although this respiration was obviously inadequate in volume. Within a few minutes, cardiac arrest occurred. IPPR with oxygen was commenced and external cardiac massage begun. Spontaneous heart beat soon returned. 100 m.Eq. sodium bicarbonate was rapidly infused. Tracheotomy was performed and IPPR with 40% oxygen and air, by means of a Bird ventilator, was commenced. The patient did not regain consciousness. The provision of adequate pulmonary ventilation with the ventilator presented grave problems because of the poor pulmonary compliance and the profuse bronchiectasis. The patient developed increasingly severe bronchopneumonia post-operatively and died 5 days after surgery, without regaining consciousness.

AUTOPSY

Tracheotomy wound. Paramediam abdominal incision. Right groin wound (surgical). Diffuse bronchopneumonia. Enlarged heart weighing 550 gm.

COMMENT:

The patient suffered a cardiac arrest because of anoxic anoxia due to hypoventilation. Because of the grave pulmonary disease, the anaesthetist anticipated the possibility of post-operative respiratory inadequacy. Poor pulmonary compliance and residual curarisation may have been contributory to this. It must be remembered that the degree

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of bronchiectasis and chronic airway obstruction were severe enough to cause cyanosis when the patient breathed air pre-operatively.

It was while assessing the respiratory inadequacy and the need for post-operative IPPR that the anaesthetist misjudged the seriousness of the situation, allowing the patient to breathe inadequately spontaneously for too long. The subsequent handling of the situation was correct and adequate. However, as is so often the case, when cardiac arrest is due to anoxic anoxia, cerebral damage is irreversible by the time that the heart arrests. This patient suffered irreversible cerebral damage and did not regain consciousness. In the circumstances, it was inevitable that the pulmonary lesion would proceed to the bronchopneumonia which ultimately cause his death.

The management of the anaesthetic is regarded as significantly contributory to this patient's death, although this may be a harsh judgement in the presence of the severe pulmonary lesion which led to the pre-anaesthetic cyanosis.

PREVENTABILITY

In view of the severe pulmonary lesion and the anticipated post-operative respiratory inadequacy, elective post-operative tracheotomy and IPPR should have been performed. The time for which inadequate spontaneous ventilation was permitted, while assessing the situation, constituted a misjudgement. Inthese terms, this death - certainly immediately post-operatively - was probably preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
117.2.65	3	No comment	ORD	Ruptured aortic aneurysm Exsanguination.	Yes

Name: Charles Ross Age: 81 Sex: M Race: E

Disease: rupture of aortic aneurysm

Operation: Laparotomy. Resection and graft of abdominal aneurysm.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

The patient presented with a leaking abdominal aortic aneurysm. His condition was good immediately pre-operatively, especially considering the lesion. B.P. 130/80 mm.Hg. Pulse rate 110/minute.

PREMEDICATION:

Atropine 0.6 mg given 45 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with cyclopropane and oxygen. Succinylcholine 50 mg. was injected and oral endotracheal intubation performed. The anaesthetic was continued with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. dTc was used as the relaxant, total dose being 50 mg. throughout the 6 hours of operation. The ECG was monitored during the procedure. The course of anaesthesia was untoward until the operation had been in progress for 3 hours, when massive haemorrhage occurred from the operative site. In spite of heroic efforts, the anaesthetist could not replace blood as rapidly as it was lost. When 13 pints blood (which had been warmed) and 4½ pints plasma had been transfused, cardiac arrest occurred. The surgeon was unable to control the massive haemorrhage and cardiac massage was of no avail.

AUTOPSY

Long paramedian abdominal incision. Teflon graft inserted into the abdominal aorta. Blood clots in peritoneal cavity. Atherosclerosis of coronary arteries. Hypertrophy of left ventricle of heart. Congestion of lungs.

COMMENT

Death resulted from massive and continuing haemorrhage which occurred during resection of a leaking abdominal aortic aneurysm, it not being possible to keep pace with the haemorrhage by blood transfusion.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
118.2.65	3	No comment	ORD	Massive haemorrhage. Exsanguination.	Yes

Name: Koos Adams Age: 14 Sex: M Race: C

Disease: Right haemothorax Operation: Right thoracotomy

Anaesthetic risk; 4, emergency

PRE-OPERATIVE STATE:

The patient had presented with a stab wound in the 2nd right intercostal space, near the edge of the sternum, 1 week previously. After suture, he was discharged. There was now active secondary intrathoracic haemorrhage and an increasing right haemothorax. 9 pints blood and 1 gm. calcium gluconate during the 5 hours pre-anaesthetic failed to halt deterioration in his condition. Massive increasing right haemothorax, or possibly cardiac tamponade from intrapericardial bleeding, was felt to be present. The patient was moribund immediately before operation: no pulse palpable, B.P. unrecordable. The heart was displaced far to the left and was seen on ECG to be beating. He was conscious and very dyspnoeic with obvious hypoventilation. The right chest did not move at all on respiration. 75 m.Eq. sodium bicarbonate was infused.

PREMEDICATION

Atropine 0.6 mg. given 40 minutes before operation.

ANAESTHETIC

ECG monitoring was instituted. This and the fact that the patient was conscious was the only means to ascertain that the heart was beating and the patient was alive. After 5 minutes of pre-oxygenation, anaesthesia was induced with nitrous oxide and oxygen with 0.5% halothane via a carbon dioxide circle absorption system with spontaneous breathing. After 3 minutes, oral intubation was performed. IPPR was started and anaesthesia commenced with nitrous oxide and oxygen only. 30 seconds after inbutation the heart rate slowed for a short period, from 110 to 40/minute. Atropine 0.6 mg. intravenously was ineffective. Although electrical complexes were still visible on ECG, this state was regarded as virtual cardiac arrest. The table was tilted to the head-down position and IPPR with oxygen only continued. Right thoracotomy was performed rapidly and revealed a truly massive right haemothorax. A large quantity of blood had to be evacuated before the surgeon could reach the heart for cardiac massage. There was no cardiac tamponade. Blood was transfused under pressure and cardiac massage commenced. An effective carotid pulse was produced and gross haemorrhage from the right internal mammary artery continued. The heart was flabby and toneless. Injection of 0.25 mg. adrenaline in 10 ml. saline into the left ventricle was ineffective. Subsequent infusion of 1:360,000 isoprenaline likewise had no effect, the ventricular muscle remaining completely asystolic and toneless. After 30 minutes of cardiac massage, unsuccessful attempts were made to pace the heart electrically. The pupils were now widely dilated and haemorrhage from the internal mammary artery was still uncontrolled. Within the 45 minutes following cardiac arrest, 7 pints blood, with 75 m.Eq. sodium bicarbonate, had been given. Resuscitation was abandoned at this stage.

AUTOPSY

Right thoracotomy wound. Haemothorax of 500 ml. blood. Collapse of right lung. Bruising of heart. Pallor of all abdominal organs.

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COMMENT

The patient was clearly dying from blood loss and hypoventilation because of massive right haemothorax when he was anaesthetised. The slowing of the pulse rate preceding virtual cardiac arrest so soon after intubation leads one to consider if the vagal stimulation resulting from intubation was contributory. This bradycardia didnot respond to atropine, and it is more likely that it was but a sign of a 'dying heart' and the association with intubation was incidental.

The primary cause of death in this case was the delay in performing thoracotomy after admission of the patient to hospital. At the time he presented in the operating theatre, the situation was already hopeless.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
119.2.65	3	No comment	ORD	Massive haemorrhage Ruptured liver.	Yes

Name: Lily Human Age: 22 Sex: F Race: E

Disease: Multiple injuries. Intra-peritoneal haemorrhage. Ruptured liver.

Operation: Laparotomy

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

Virtually moribund. Severe oligaemic shock from rupture of liver with intraperitoneal haemorrhage. In spite of massive blood transfusion, her condition had deteriorated steadily and for ¾ hour before operation no B.P. had been recordable. She was cold from transfusion of cold blood. Pulmonary contusion had occurred and traumatic pulmonary oedema was visible on chest X-ray, although not clinically apparent. The urine was blood-stained. 150 m.Eq. sodium bicarbonate was infused.

PREMEDICATION: Nil

ANAESTHETIC

Immediately before anaesthetic, a further 2 pints blood were rapidly transfused. B.P. rose to 70 mm.Hg systolic. ECG monitoring was commenced. Anaesthesia was induced with cyclopropane and oxygen and succinylcholine 25 mg. was given. Oral endotracheal intubation was performed and anaesthesia was maintained with nitrous oxide and oxygen via a circle absorption system by an IPPR technique. dTc 15 mg. was given. After 20 minutes, IPPR with oxygen only was administered. Laparotomy revealed multiple lacerations of both lobes of the liver, a large retroperitoneal haematoma, laceration of the left kidney, laceration of the spleen and injury to the tail of the pancreas, with gross intraperitoneal haemorrhage. Gross oozing occurred from all lacerated surfaces, and a clotting defect was apparent. Trasylol 20,000 units and 4 units fresh frozen plasma were infused without much effect. During operation, 14 pints warmed blood were transfused. After the operation had been in progress for 40 minutes, cardiac arrest occurred. After 10 minutes of external cardiac massage, resuscitative measures were abandoned.

AUTOPSY

Contused and lacerated wounds all over body. Ruptured liver and spleen. Intraperitoneal bleeding. Both surfaces of liver ruptured with severe laceration of posterior surface. Large retroperitoneal haematoma, posterior to the right kidney.

COMMENT

The cause of death was massive haemorrhage with which blood replacement could not keep pace. Besides the extensive internal injuries from which haemorrhage took place, bleeding was aggravated by a bleeding tendency which followed massive transfusion. It is interesting to surmise if pancreatic injury caused any elevation of fibrinolytic factors. As death occurred while the patient was anaesthetised, this case is classified in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
120.2.65	2	No comment	< 24	Cerebral infarction	No

Name: Ranje Lallo Age: 30 Sex: M Race: C

Disease: Cerebral abscess Operation: Carotid angiography. Burrhole craniotomy

Anaesthetic risk: 3, emergency

PRE-OPERATIVE STATE:

Poor. The patient was confused, drowsy and had generalised flaccidity. CSF pressure was over 300 mm.water. He was pyrexial, temperature 101°F. Pulse rate was 116/minute, B.P. 110/70 mm.Hg

PREMEDICATION

Atropine 0.6 mg given 45 minutes before operation.

ANAESTHETIC

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx and oral endotracheal intubation. During angiography anaesthesia was maintained with nitrous oxide and oxygen, with spontaneous respiration. Thereafter dtc 15 mg. was given and subsequently anaesthesia was continued with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. A left parietal burrhole craniotomy revealed ischaemia and infarction of the temporal lobe. The course of anaesthesia was untoward. At the conclusion of the procedure, neostigmine 2.5 mg. preceded by atropine 1.2 mg. adequately reversed residual curarisation. The patient's level of consciousness returned to the level present before anaesthesia and he died 7 hours post-operatively, following respiratory failure.

AUTOPSY

No autopsy

COMMENT

The anaesthetic was not contributory to this patient's death, which resulted from the existing cerebral disease.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
121.2.65	3	No comment	ORD	Common carotid artery thrombosis Cerebral softening Myocardial ischaemia Cardiac arrest.	Yes

Name: Margo Zinober c Age: 60 Sex: F Race: E

Disease: Hemiplegia. Total occlusion of right common carotid artery. Operation: Carotid angiography

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Angiography was necessary to investigate the state of the patient's carotid arteries, to assess the possibly value of carotidartery surgery for hemiplegia. A known hypertensive, she had had coronary thrombosis in the past. B.P. at this time was 170/70 mm.Hg. Pulse rate 120/minute. Gross arteriosclerosis was present.

PREMEDICATION

Atropine 0.6 mg. 60 minutes pre-operatively.

ANAESTHETIC

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 30 mg., ventilation with oxygen, topical analgesia of the larynx and oral intubation. After resumption of spontaneous respiration, anaesthesia was continued with nitrous oxide, oxygen and 0.5% halothane via a Magill semi-open circuit with spontaneous breathing. ECG monitoring was instituted. The course of anaesthesia was untoward until the radio-opaque dye was injected into the carotid artery. Profound hypotension followed and cardiac arrest soon occurred. IPPR with oxygenonly was commenced. External cardiac massage was started, and was effective. A peripheral pulse was palpable and the pupils, which had dilated, became small. After 5 minutes, ventricular fibrillation occurred. External defibrillation with a DC defibrillator restarted normal sinus rhythm. The circulation was unsatisfactory and the B.P. remained low. External cardiac massage was therefore continued. Within a few minutes ventricular fibrillation recurred. External defibrillation resulted in complete asystole, refractory to treatment. Thoracotomy and internal cardiac massage were not attempted. The carotid angiogram taken showed complete obstruction of the right common carotid artery.

AUTOPSY

Gross coronary atherosclerosis. Partial collapse of the left lung. Subdural haemorrhage. Congestion of liver and kidneys. Thrombosis of the right common carotid artery. Cerebral softening in the region of internal capsule and basal ganglia. Hypertrophy of the left ventricle of the heart.

COMMENT

This patient was an extremely poor anaesthetic risk. The episode of gross hypotension following the injection of dye intra-arterially, and preceding cardiac arrest, did not follow induction of anaesthesia. Hypotension is known to follow the injection of dye intra-arterially and is usually transient. However, in this case, with coronary atherosclerosis, hypotension was sufficient to cause cardiac arrest - probably on the basis of myocardial ischaemia. Resuscitative measures

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appear to have been prompt and adequate. Success was vitiated by the poor state of the myocardium. Thrombosis of the right common carotid artery with demonstrable cerebral softening were probably also contributory to the death.

Anaesthesia is possibly necessarily and unavoidably contributory to this death. The finding of partially collapsed lung at the autopsy was not anticipated. It is thought that this may have occurred during the resuscitative procedures; however, it was not of sufficient magnitude to have caused death.

CASE NO.	CLASSIFICATTON Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
122.2.65	2	No comment	< 24	Traumatic cerebral damage	Yes

Name: Nicholas Windvoegel Age: 40 Sex: M Race: C

Disease: Head injury. Intra-cerebral haemorrhage.

Operation: Burrhole craniotomy. Evacuation of haematoma.

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

Extremely poor condition. He was comatose, had bilateral extensor rigidity with superimposed extensor spasms. Gross papilloedema was present. B.P. before anaesthesia was 290/120 mm.Hg, and the pulse rate 60/minute.

PREMEDICATION

Nil.

ANAESTHETIC

After oxygenation, oral endotracheal intubation was performed following injection of succinylcholine 75 mg. During angiography, oxygen only was administered by an IPPR technique. Intermittent small doses of succinylcholine were given to facilitate IPPR. During the burrhole craniotomy, a total dose of 25 mg. dTc was given for relaxation, anaesthesia being maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. The operation lasted 130 minutes. Craniotomy revealed a midparietal intracerebral haematoma. During the procedure the B.P. remained at between 250 and 300mm.Hg systolic and the pulse rate slowly rose to 84/minute. Blood loss was small, estimated at only 120 ml. 200 ml. 5% dextrose in water was infused. At the end of operation, neostigmine 2.5 mg preceded by atropine 1.2 mg. was given to reverse residual curarisation, and spontaneous respiration resumed at a rate of 40/minute, the tidal volume being normal. The endotracheal tube was left in situ. The patient failed to regain consciousness and died 1½ hours post-operatively.

AUTOPSY

Severe brain oedema. Large midparietal cerebral haemorrhage with considerable dissolution of surrounding brain.

COMMENT:

Death was the result of traumatic cerebral damage and the anaesthetic was not contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
123.2.65	3	No comment	ORD	Massive haemorrhage Inadvertent hypothermia Cardiac arrest.	Yes

Name: Anna Silvester Age: 62 Sex: F Race: C

Disease: Sarcoma of the uterus and cervix, Stage 3. Operation: Total abdominal hysterectomy.

Anaesthetic risk: 1

PRE-OPERATIVE STATE:

In spite of the gynaecological lesion, this patient was generally fit. Haemoglobin concentration was 13 gm.%. B.P. 140/80 mm.Hg and pulse rate 84/minute.

PREMEDICATION

Omnopon 10 mg., atropine 0.6 mg. given 75 minutes pre-operatively

ANAESTHETIC

Anaesthesia was induced with the sequence thiopentone 250 mg., succinylcholine 50 mg., oxygenation, topical analgesia of the larynx, oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. dTc was given for relaxation, a total of 33 mg. being used throughout the 220 minute operation. The course of anaesthesia was uneventful for the first two hours of operation. Blood was replaced as lost, by transfusion. During this time, 5 pints blood were transfused with 50 m.Eq. sodium bicarbonate. At this stage, gross uncontrollable haemorrhage from the iliac vessels ensued. Blood was rapidly transfused under pressure, a further 7 pints being given in the next hour, when a further 50 m.Eq. sodium bicarbonate was given. The B.P. had now fallen to 90 mm.Hg systolic. Two doses 0.5 mg. phenylephrine were administered without response. The B.P. continued falling. A further 1 mg. phenylephrine was ineffective. Haemorrhage was still not controlled. Blood was transfused even more rapidly, a further 4 pints and 5 units (250 ml. each) plasma being rapidly transfused during the next 30 minutes. The latter units of blood were not warmed because of lack of time, but were administered at the storage temperature of 4°C. The patient's oesophageal temperature, until this time having fallen to 33°C, now rapidly fell to 29°C. Cardiac arrest occurred. The anaesthetist estimated that 16 pints blood and 2½ pints plasma given had been adequate replacement of loss and that the cardiac arrest was probably due to inadvertent hypothermia. When arrest occurred, as no success had been achieved in controlling the haemorrhage from the operative site, no cardiac massage or further resuscitation was undertaken.

AUTOPSY

Congestion and oedema of both lungs. Atherosclerosis of large blood vessels and coronary arteries. Fatty infiltration of liver. Uterus and adnexae had been removed.

COMMENT

Massive haemorrhage and the effects of equally massive transfusion, notably inadvertent hypothermia, were the cause of this cardiac arrest and death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
124.2.65	2	No comment	< 24	Mesenteric thrombosis	No

Name: Bennet Williams Age; 51 Sex: M Race: C

Disease: Mesenteric thrombosis. Operation: Laparotomy

Anaesthetic risk: 3, emergency

PRE-OPERATIVE STATE:

A known hypertensive, the patient had aortic incompetence and had been in and out of congestive cardiac failure for the last 4 years. In addition, he had pulmonary emphysema. He now had an acute abdomen with gross abdominal distension, and mesenteric thrombosis was diagnosed. After rehydration therapy, he appeared extremely ill. B.P. was 130/80 mm.Hg although it was known normally to be 200/80 mm.Hg.

PREMEDICATION

Atropine 0.6 mg. given 60 minutes before operation.

ANAESTHETIC:

Anaesthesia was induced with nitrous oxide and oxygen inhalation. Oral intubation was performed after injection of succinylcholine 50 mg. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system, by an IPPR technique. Succinylcholine was used intermittently for abdominal relaxation. The course of anaesthesia was relatively uneventful. Laparotomy revealed virtually all the small bowel and much of the large bowel to be infarcted. Nothing further was done and the abdomen was closed. The patient regained normal respiration post-operatively. Though abdominal distension may have caused some inadequacy, the volume of respiration was no worse than pre-operatively. He regained consciousness immediately on discontinuance of the anaesthetic. No further resuscitative measures or active treatment was undertaken and the patient died 1 hour after the operation.

AUTOPSY

No autopsy

COMMENT

Death was due to the effects of massive mesenteric thrombosis with bowel infarction, which was surgically inoperable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
125.2.65	3	No comment	ORD	Massive haemorrhage Massive transfusion Inadvertent hypothermia Cardiac arrest	Yes

Name: Moira Butler Age: 51 Sex: F Race: E

Disease: Carcinoma of the uterus. Operation: Pelvic exenteration

Anaesthetic risk: 1

PRE-OPERATIVE STATE:

Except for carcinoma of the uterus, the patient was otherwise normal, though mildly hypertensive. B.P. 160/95 mm.Hg

PREMEDICATION

Omnopon 15 mg., atropine 0.6 mg., given 75 minutes pre-anaesthesia.

ANAESTHETIC

A warming mattress was placed beneath the patient on the operating table. ECG monitoring was instituted. Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. During the early part of the operation, halothane 0.5% was used. A total of 60 mg. dTc wasused for relaxation during the operation, which lasted 300 minutes. The course of anaesthesia was uneventful for the first 3 hours. Blood loss was replaced by transfusion, 10 pints being given during this time and the B.P. was maintained at 120 mm.Hg systolic. Although the blood was warmed and the patient was on a warming mattress, the oesophageal temperature dropped to 35.2°C at this time. A large haemorrhage occurred at the operative site and, during the next hour, the systolic B.P. dropped briefly to 75 mm.Hg on two occasions. Small doses of phenylephrine and continued rapid transfusion of a further 6 pints blood restored this to a level of 120 mm.Hg. Calcium gluconate 2 gm. had also been given. The oesophageal temperature dropped to 30.5°C by now. 150 m.Eq. sodium bicarbonate was infused. The haemorrhage now worsened. In spite of transfusion of a further 8 pints blood, no longer warmed, the administration of 0.5 mg. digoxin and small repeated doses of phenylephrine, the B.P. droppedprogressively. When the oesophageal temperature reached 30°C, cardiac arrest occurred. Left thoracotomy and internal cardiac massage were performed immediately. This and other resuscitative measures were ineffectual. In all, a total of 25 pints blood had been transfused.

AUTOPSY

Midline abdominal incision from umbilicus to pubis. Left thoracotomy incision. 800 ml. blood in abdominal cavity. All tissues appeared anaemic. Uterus and bladder had been removed.

COMMENT

Cardiac arrest and death was the result of massive haemorrhage and the effects of massive transfusion, notably inadvertent hypothermia. The anaesthetic per se is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
126.2.65	2	No comment	ORD	Pulmonary embolism	Yes

Name: Fanele Mnguni Age; 40 Sex: M Race: B

Disease: Dehiscence of abdominal wound. Operation: Resuture of dehisced abdominal wound.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Five days before this operation, the patient had undergone laparotomy and splenectomy, with reduction of compound fractures of the tibia and fibula, for multiple injuries suffered in a motor accident. Small bowel ileus had followed operation. The abdominal wound had now dehisced. His condition was extremely poor although B.P. was 110/80 mm.Hg, the pulse rate was 120/minute and haemoglobin concentration 14 gm.%. Peripheral vasoconstriction and cyanosis were present. He appeared dehydrated, was mentally confused and uncooperative. Neither serum electrolytes nor acid-base investigations had been done. Attempts to pass a nasogastric tube had failed, only resulting in copious vomiting and straining, with further dehiscence of the wound and protrusion of gut. Immediately before anaesthesia, 1000 ml. plasma was transfused.

PREMEDICATION: Nil

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 100 mg., ventilation with oxygen, and oral endotracheal intubation. The latter caused a bradycardia of 56/minute with a fall in B.P. to 70 mm.Hg systolic. The rapid administration of atropine 0.6 mg. and phenylephrine 0.25 caused a rise in pulse rate to 108/minute and in B.P. to 120 mm.Hg systolic, and subsequently to 140 mm.Hg systolic Anaesthesia was maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. Succinylcholine was used intermittently for relaxation, total dose being 100 mg. during the 60 minute operation. The B.P. and pulse rate remained steady during the procedure. At the conclusion of the operation, normal spontaneous respiration resumed. As the patient was being moved from the operating table to his bed in the theatre, respiration ceased and cyanosis ensued. Cardiac arrest followed immediately and he was returned to the operating table and re-intubated. IPPR was commenced and external cardiac massage started. After 1 minute, as an effective pulse could not be palpated, and the pupils which had dilated remained large, left thoracotomy was performed and internal massage commenced. Adrenaline 1:200,000 solution, 10 ml. was injected intraventricularly and cardiac massage continued, 100 m.Eq. sodium bicarbonate and 1 mg. phenylephrine being given. Spontaneous heart beat resumed after 5 minutes. The B.P. was now recorded at 100 mm.Hg systolic. The thoracotomy wound was sutured and spontaneous respiration resumed, byt the patient did not recover consciousness. Though respiratory effort appeared adequate, he remained lightly cyanosed. The endotracheal tube was left in situ and the patient was kept under observation in the theatre. IPPR with oxygen was continued, though for short periods spontaneous respiration on oxygen was permitted, to assess the adequacy of his pulmonary ventilation. He remained lightly cyanosed, although no good cause (e.g. pneumothorax) could be found. After 30 minutes, respiratory failure was again followed by cardiac arrest and this time external cardiac massage was to no avail. No further resuscitation was undertaken as irreversible cerebral damage appeared to have followed the first cardiac arrest.

AUTOPSY

Fracture of left tibia and fibula, and left femur. Sutured left thoracotomy wound. Sutured wound on forehead and left foreleg. Scattered bruising of small bowel. Absence of spleen. Multiple small pulmonary artery emboli. Histology of lung showed haemorrhage into alveoli.

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COMMENT:

The respiratory arrest and cyanosis that occurred when this patient was moved from the operating table onto his bed, immediately post-operatively, and the subsequent persistent cyanosis preceding final cardiac arrest, are explained by the finding at autopsy of multiple pulmonary emboli. These in all probability originated in the leg which had been fractured 5 days previously, and were dislodged by the movement of the patient from the operating table.

Interestingly enough, the autopsy finding exculpates the anaesthetist and the management of the anaesthetic from the responsibility for the post-anaesthetic respiratory inadequacy.

This case, as does Case No. 18.2, illustrates the importance the autopsy may have in excluding anaesthesia as a cause of death. The anaesthetic and its management would perhaps have been held responsible for the post-operative respiratory inadequacy in this case, in the absence of autopsy.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
127.2.65	3	No comment	ORD	Haemorrhage and embolisation. Cardiac arrest (Left ventriculotomy).	Yes

Name: Job Riddles Age: 30 Sex: M Race: B

Disease: Mitral stenosis. Operation: Mitral valvotomy

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had severe mitral stenosis and atrial fibrillation. Cardiac output was poor and there was vasoconstriction of the extremities. A pleural effusion was present at the left lung base. He was digitalised. He weighed 160 lbs.

PREMEDICATION

Omnopon 10 mg., atropine 0.6 mg. given by intramuscular injection 60 minutes before anaesthesia.

ANAESTHETIC

The ECG was monitored throughout. Anaesthesia was induced with the sequence thiopentone 300 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen, administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was given in divided doses, total dose being 29 mg. during the procedure. The heart was very irritable, surgical handling producing multiple ventricular extrasystoles. A large adherent thrombus was found in the left atrium. After commissurotomy with a Tubb's dilator, the B.P. fell to unrecordable levels. An infusion of isoprenaline solution 1:360,000 produced improvement and the B.P. subsequently varied between 80 and 100 mm.Hg systolic. 100 m.Eq. sodium bicarbonate was infused. At the end of operation spontaneous respiration resumed. Ventricular fibrillation supervened and, at the same time, the left pupil was noticed to be large while the right was small. External cardiac massage was begun. The right carotid pulse was palpable but the left was now not palpable. The thorax was re-opened and internal cardiac massage instituted, and subsequently electrical defibrillation restored spontaneous heart beat, although the B.P. was still unrecordable. The surgeon thought he could feel a large thrombus, formerly in the left atrium, in the left ventricle. Within 7 minutes, ventricular fibrillation recurred. Left ventriculotomy was rapidly performed to evacuate the thrombus, and this resulted in rapid haemorrhage of at least 2 pints. Both pupils were now widely dilated. Further resuscitative measures were abandoned.

AUTOPSY

Brain congested. 300 ml. blood in left pleural cavity. Lungs congested. Sutured wounds in left atrium, apex of left ventricle and anterior wall of left ventricle. Left atrium large and dilated. Fibrous clot adherent to wall of atrium. Loose friable thrombus loosely attached to atrial wall. Mitral valve - fibrotic cusps adequately split along anterior and posterior commissures, but only partially split. No mitral incompetence. Nothrombus in left ventricle. Aortic valve normal. Right ventricle and left atrium dilated and enlarged.

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COMMENT:

This death resulted from what appeared to have been the dislodgement of a large adherent thrombus from the left atrium, and the subsequent manoeuvres adopted to remove this. Anaesthesia per se is not thought to have been a significant contributory factor in this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
128.2.65	2	No comment	< 24	Results of removal of menin-gioma.	Yes

Name: Solomon Nyimbinya Age: 30 Sex: M Race: B

Disease: Cerebral tumour Operation: Left parieto-occipital craniotomy and excision of tumour.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had a cerebral tumour and was stuperose and pyrexial. Temperature 100°F. The cardiovascular system was normal.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection, 40 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 40 mg., ventilation with oxygen, topical analgesia of the larynx and oral endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen via a carbon dioxide circle absorptionsystem, by an IPPR technique. dTc, total dose 20 mg., was given during the course of operation for relaxation. The course of anaesthesia was uneventful.

A large tumour, histologically a meningioma, was removed from the left temporal region. Blood was replaced by transfusion as lost. At the conclusion of the procedure, neostigmine 1.5 mg. preceded by atropine 1.2 mg. was given to reverse residual curarisation. Normal adequate spontaneous respiration resumed. The level of consciousness returned to what it had been pre-anaesthesia. The patient died 17 hours post-operatively.

AUTOPSY

Removal of left temporal lobe and left cerebellar hemisphere. Right lateral ventricle filled with blood. Remaining cerebral surfaces ragged. No sign of residual tumour. Haemorrhage present in pons and medulla. Histologically, surgical specimen removed was meningioma.

COMMENT:

Death was the result of the effects of surgical removal of a large meningioma. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
129.2.65	2	No comment.	< 24	Cardiac failure. Water intoxication.	No

Name: J. Windvogel Age: 82 Sex: M c Race: C

Disease: Prostatic adenoma Operation: Transurethral resection of adenoma by punch technique

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

The patient had benign prostatic urinary obstruction. Except for mild pulmonary emphysema, he was fit for his age. B.P. 130/70 mm.Hg

PREMEDICATION

Atropine 0.6 mg. intramuscularly, 90 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 250 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral endotracheal intubation. When spontaneous respiration resumed anaesthesia was maintained for 15 minutes with nitrous oxide and oxygen, with halothane 0.5%, via a carbon dioxide circle absorption system with spontaneous breathing. dTc 10 mg. was then given and an IPPR technique instituted. Two further small doses of dTc were given subsequently, a total of 25 mg. being used during the prolonged operation, which lasted 2 hours. During the 1st 90 minutes of the procedure, there was a steady progressive rise in B.P. from 140 mm.Hg systolic at the beginning of anaesthesia, to 220 mm.Hg systolic. The pulse rate remained at 100/minute during this time. Oozing was observed from the operative site, increasing during this time, and 1 pint blood being transfused to replace this. Over the next 30 minutes the B.P. fell to 100 mm.Hg systolic and finally rose again to reach 140 mm.Hg systolic at the end of the operation. Residual curarisation was reversed with neostigmine 1.5 mg. preceded by atropine 1.2 mg., and normal adequate spontaneous respiration resumed. The patient rapidly regained consciousness but was confused, disorientated and restless. Assuming that this was due to water intoxication from intravenous absorption of the bladder-irrigating fluid, hypertonic sodium chloride and 10% Mannitol were administered post-operatively. However, 4 hours later the patient's condition suddenly deteriorated and he died. Histology of the tumour resected from the prostate showed benign prostatic adenoma.

AUTOPSY

No autopsy.

COMMENT:

One of the known complicationsof transurethral resection of the prostate, especially when the operation is prolonged and resection is radical, with exposure of venous sinuses, is intravenous absorption of large quantities of bladder-irrigating fluid. This causes water intoxication, overloading of the circulation, hyponatraemia from haemodilution and perhaps even intravascular haemolysis. Events during this operation point to this having occurred. The B.P. rose to high levels, oozing from the operative site increased and finally, when the patient regained consciousness, he was confused, disorientated and restless. His sudden death 4 hours later was probably due to the effects of the water intoxication. The management of the anaesthetic is not contributory to death, although the administration of 1 pint blood should, in the circumstances, have been omitted.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
130.2.65	2	No comment	< 24	Haemorrhage from thoracic stab wound.	Yes

Name: Douglas Mlembo Age: 42 Sex: M Race: B

Disease: Penetrating stab wound of chest. Operation: Thoracotomy

Anaesthetic risk: 4, emergency

PRE-OPERATIVE STATE:

The patient had been stabbed in the chest and had a right haemothorax. He was in a state of extreme oligaemic shock and had shown no response to the transfusion of 3 pints blood, transfused rapidly under pressure. B.P. and pulse rate were unrecordable. It was thought at this stage that the stab wound might have penetrated the heart.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection, 60 minutes pre-anaesthesia.

ANAESTHETIC

Anaesthesia was induced with inhalation of nitrous oxide and oxygen, injection of succinylcholine 100 mg., and oral intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique, after administration of dTc 10 mg.

Thoracotomy revealed that the haemorrhage was from a severed right internal mammary artery. Once this had been secured and more blood transfused, the B.P. rose to 100 mm.Hg systolic and was maintained at this level. The operation took 110 minutes. Calcium gluconate 1 gm. and 50 m.Eq. sodium bicarbonate were given during the operation. At the end of the procedure, residual curarisation was reversed with neostigmine 1 mg. preceded by atropine 0.6 mg. Normal spontaneous respiration resumed and the patient regained consciousness. Post-operatively, he continued to bleed into his chest and he died 2 hours after surgery.

AUTOPSY

Sutured right thoracotomy wound. Wounds for intra-thoracic drains, between ribs 7 and 8. Gross anaemia of all abdominal organs and of brain. Contused wound on face.

COMMENT:

This patient died of continued haemorrhage from a thoracic stab wound. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
131.2.65	2	No comment	< 24	Urinary obstruction Extravasation of urine. Gas gangrene of scrotum and abdominal wall.	No

Name: A. Garcia Age: 40 Sex: M Race: E

Disease: Urinary obstruction. Extravasation of urine. Gangrene of scrotum and abdominal wall. Septicaemia.

Operation: Suprapubic cystostomy. Multiple incisions for drainage of scrotum and abdominal wall.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

After urethral urinary obstruction with extravasation of urine, this patient had developed gas gangrene of the scrotum and spreading cellulitis of the abdominal wall. He was gravely toxic and moribund. B.P. 100 mm.Hg systolic, pulse rate 140/minute. Tachypnoea - 30/minute. 1 litre plasma was transfused before anaesthesia.

PREMEDICATION: Nil

ANAESTHETIC:

Anaesthesia was induced and maintained with inhalation of nitrous oxide and oxygen, with minimal ether bapour, administered via a carbon dioxide circle absorption system, with spontaneous breathing. 1 pint blood was administered during the operation, which lasted 60 minutes. The patient regained consciousness immediately on discontinuance of the anaesthetic. He died 9 hours post-operatively.

AUTOPSY

No autopsy

COMMENT:

The patient died of gas gangrene and spreading cellulitis of the abdominal wall, with septicaemia. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
132.2.65	2	No comment	< 24	Post-meningitic hydro-cephalus	No

Name: Johannes Potgieter Age: 3 months Sex: M Race: E

Disease: Post-meningitic hydrocephalus.

Operation: Bilateral burrhole craniotomy and ventricular drainage.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had gross post-meningitic hydrocephalus. The cardiovascular and respiratory systems were normal. B.P. 100 mm.Hg systolic and pulse rate 132/minute.

PREMEDICATION:

Atropine 0.2 mg. by intramuscular injection, 20 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide, oxygen and halothane. After topical analgesia of the larynx, oral endotracheal intubation was performed. Anaesthesia was maintained with nitrous oxide, oxygen and halothane delivered via a T-piece circuit with spontaneous breathing. During the operation, which lasted 65 minutes, 90 ml. blood was transfused. At the conclusion of the procedure, the patient regained consciousness and breathed adequately. Later, the patient developed fits and he died 7 hours post-operatively

AUTOPSY

No autopsy

COMMENT

This patient died from the effects of his existing disease. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
133.2.65	2	No comment	< 24	Haemorrhage from carotid artery.	No

Name: Cornelius Jansen Age: 48 Sex: M Race: C

Disease: Carcinoma of nose and maxillary antrum, secondary haemorrhage from carotid artery.

Operation: Repair of carotid artery.

Anaesthetic risk: 4, emergency.

pRE-OPERATIVE STATE:

The patient had a fungating carcinoma of the maxillary antrum and nose. One month previously this had been perfused with cytotoxic drugs, via the carotid artery. He now presented with gross secondary haemorrhage from the carotid artery; there was also bleeding from the nose and some blood had been aspirated into the lungs. Blood transfusion had maintained the B.P. at a level of 110 mm.Hg systolic.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 45 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide, oxygen and halothane. Endotracheal intubation was performed and was difficult because of the displacement of the trachea and larynx. Tracheobronchial toilette was performed. Halothane was then discontinued and anaesthesia maintained with nitrous oxide and oxygen with minimal ether vapour. The B.P. remained steady at a level of 120 mm.Hg systolic while the carotid artery was resutured. Two pints blood were transfused. At the conclusion of the operation the patient regained consciousness and respiration was normal and adequate. However, when in transit down thetheatre corridor, en route to the ward, respiratory obstruction occurred - probably due to anatomical distortion produced by the lesion, and cardiac arrest promptly ensued. External cardiac massage was performed immediately and he was re-intubated. IPPR with oxygenwas instituted. Spontaneous heart beat returned and he regained consciousness. He was then returned to the ward with the endotracheal tube in situ. No further respiratory obstruction occurred. Haemorrhage from the carotid artery recurred 5 hours post-operatively, and he rapidly bled to death.

AUTOPSY

No autopsy

COMMENT

The cardiac arrest that occurred immediately post-operatively was due to respiratory obstruction. Because of the distorted anatomy, the anaesthetist should have left the endotracheal tube in situ in the first place, as he did subsequently. However, this arrest was promptly and correctly treated. Anaesthesia was not contributory to the patient's death when it finally occurred.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSEOF DEATH	AUTOPSY
134.2.65	3	No comment	ORD	Cardiac arrest. (Massive haemorrhage)	Yes

Name: Mymona Daniels Age: 24 Sex: F Race: C

Disease: Mitral stenosis and incompetence; aortic incompetence. Operation: Mitral valvotomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

The patient had developed re-stenosis of the mitral valve following previous mitral valvotomy 5 years before. There was pulmonary hypertension and mild aortic incompetence. B.P. 130/90 mm.Hg. She was digitalised and had been treated with diuretics.

PREMEDICATION

Atropine 0.6 mg., pethidine 75 mg. intramuscularly 60 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia and oral endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. For the first 30 minutes, 0.5% halothane was administered. A total dose of dTc 27 mg. was used during the procedure. The B.P. remained constant until atriotomy was attempted, at a level of 120 mm.Hg systolic. The anatomy was much distorted by the previous operative procedure - there was no left atrial appendage. While dissection of the left atrium from adhesions was in progress, an incision was made in the left ventricle. Gross haemorrhage was followed by cardiac arrest and asystole. 4 pints blood were rapidly transfused under pressure while the incision in the ventricular wall was resutured so that cardiac massage could be commenced. This, and the presence of mitral stenosis, hampered resuscitative measures. Isoprenaline solution, intracardiac adrenaline (20 ml. 1:10,000 solution) and calcium chloride were administered, in vain.

AUTOPSY

23 cm. left thoracotomy incision. 5th rib disengaged at costochondrial junction. 600 ml. blood in left pleural cavity. Incision into left ventricle closed with 6 interrupted catgut sutures. Severe mitral stenosis, the valve not permitting introduction of the tip of the small finger.

COMMENT:

Death was the result of a surgical mishap. Anaesthesia was not contributory to this patient's demise.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
135.2.65	2	No comment	< 24	Myocardial ischaemia	Yes

Name: A. Abrahams Age: 67 Sex: F Race: C

Disease: Diverticulitis. Generalised peritonitis.

Operation: Laparotomy. Closure of ruptured diverticulum. Colostomy.

Anaesthetic risk: 3, emergency.

PRE-OPERATIVE STATE:

This grossly obese patient presented with a 12 hour history of constant severe abdominal pain, with colicky exacerbations. The abdomen was distended and red. X-ray abdomen showed free gas in the peritoneal cavity. B.P. was 200/90 mm.Hg, pulse rate 120/minute. She was not shocked. Aortic incompetence was present. Rehydration with fluid and plasma was commenced.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 60 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 200 mg. succinylcholine 50 mg., ventilation with oxygen, and oral endotracheal intubation. dTc 30 mg. wasgiven and anaesthesia was continued with nitrous oxide and oxygen, administered via a carbon dioxide absorption system by an IPPR technique. Halothane 0.5% was used intermittently. The course of anaesthesia was untoward. Laparotomy revealed generalised peritonitis, free gas in the peritoneal cavity, perforated diverticulum of the sigmoid colon. The perforation was closed and a defunctioning colostomy performed.

At the conclusion of the procedure, residual curarisation was reversed with neostigmine 2.5 mg preceded by atropine 1.2 mg. Normal spontaneous respiration resumed and the patient rapidly regained consciousness. She died suddenly 16 hours post-operatively.

AUTOPSY

Signs of abdominal operation involving colostomy. Gross generalised atherosclerosis with marked atheroma of coronary arteries. Aortic incompetence.

COMMENT:

This patient had both coronary atherosclerosis and aortic incompetence. It is possible that acute myocardial ischaemia was the cause of death. Anaesthesia is not considered contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
136.2.65	1	Possibly	< 24	Pulmonary oedema (Over-transfusion)	Yes

Name: Doreen Pipers Age: 26 Sex: F Race: C

Disease: Ruptured ectopic pregnancy. Operation: Left salpingectomy

Anaesthetic risk: 2, emergency.

PRE-OPERATIVE STATE:

The patient had suffered rupture of an ectopic pregnancy 48 hours previously. She was not shocked. Pulse rate 85/minute, B.P. was 120 mm.Hg systolic. Shifting dullness was apparent on examination of the abdomen. Haemoglobin concentration 7.5 mg.%.

PREMEDICATION:

Atropine 0.6 mg. intramuscularly, 45 minutes pre-operatively.

ANAESTHETIC:

Anaesthesia was induced with inhalation of nitrous oxide and oxygen, injection of succinylcholine 100 mg., and oral endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. A single dose of dTc 25 mg. was given. The B.P. remained at a level of between 120-130 mm.Hg systolic throughout the operation and the pulse rate, except for a brief rise to110/minute at the end of the operation, remained between 90 -100/minute. This rise was probably due to administration of ether vapour, referred to later.

Laparotomy revealed a ruptured ectopic pregnancy. Approximately 1,350 ml. free blood in the peritoneal cavity was aspirated. A left salpingectomy was performed. After infusion of 500 ml. Ringer's lactate solution, the anaesthetist transfused 2 pints blood during the next 30 minutes to replace the blood lost into the peritoneal cavity. During abdominal closure, relaxation did not appear adequate. Ether vapour was added to the anaesthetic gases, a total of 1 fl.oz. being vaporised during this procedure. The operation lasted 45 minutes in all. At the end of the operation, residual curarisation was reversed with neostigmine 2.5 mg. preceded by atropine 1.2 mg. Normal spontaneous respiration resumed and, when the anaesthetic was discontinued, the patient rapidly regained consciousness, responding to stimulation, but was drowsy. As her condition was good and the level of unconsciousness light, on return to the ward she was only watched intermittently by the nursing staff. About 50 minutes after return to the ward, her level of consciousness was seen to be markedly depressed, the pupils were dilated and reacted to light only sluggishly. Respiration was stertorous and coarse crepitations were audible over both lungs. The pulse rate was regular. The house-surgeon saw the patient at this stage and elevated the foot of the bed. The stertorous breathing became gasping; coarse rhonchi and crepitations continued to be audible over both lungs, but cyanosis was not observed. The pupils ceased reacting to light. The B.P. soon commenced falling, the pulse rate rose and the patient died 1¾ hours after operation.

AUTOPSY

Lower abdominal midline surgical incision. Free blood in peritoneal cavity. Left salpingectomy. Gross pulmonary oedema.

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COMMENT:

The gross pulmonary oedema found at autopsy seemed obvious from the clinical description. It is strange that more active treatment was not undertaken. The reason for the pulmonary oedema is apparent when the description of the anaesthetic management is examined.

By the time the patient presented for laparotomy, 48 hours had elapsed since rupture of the ectopic pregnancy. She did not appear shocked, having compensated by haemodilution for the blood loss. Haemoglobin concentration was only 7.5 gm.% and the circulatory blood volume was probably normal. The 1,350 ml. free blood found in the peritoneal cavity was doubtless old blood. In replacing this as rapidly as he did, the anaesthetist grossly overloaded the patient's circulation. At the time, the vasodilation from anaesthesia and the use of an IPPR technique will have protected against the development of pulmonary oedema until such time as the patient was permitted to breathe spontaneously.

It is strange, however, that the systolic blood pressure did not rise at all at this stage.

Two other conditions that must also be considered in the differential diagnosis are (1) recurarisation or inadequate reversal of residual curarisation, and (2) regurgitation and inhalation of gastric content producing a Mendelsohn-like syndrome. The former is not considered likely in the circumstances. Though the dose of curare was more than moderate, it was felt necessary to administer ether vapour at the end of the operation, as relaxation did not appear adequate. The dose of neostigmine was adequate and, though respiratory inadequacy may have arisen, inadequate reversal of curarisation would not produce pulmonary oedema. The second possibility bears more serious consideration. The patient was poorly observed post-operatively and gastric regurgitation and inhalation could have occurred - she was not completely conscious and an endotracheal tube lubricated with 2% xylocaine jelly had been in situ for the 45 minutes of the operation. Very acid gastric content may have produced pulmonary oedema such as this, but one would expect too an element of broncho-spasm, which was not evident. There was no suggestion of inhaled material at autopsy, although this alone would not exclude the possibility.

The management of the anaesthetic is considered a significant contributory factor to this patient's death. The post-operative care and treatment was also quite inadequate.

PREVENTABILITY

This tragedy came about as a result of misjudgement of the blood loss and the state of the patient's blood volume by the anaesthetist. In the circumstances, the blood loss should have been replaced by packed cells and these should not have been rapidly administered. This death is thus regarded as possibly preventable.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
137.2.65	2	No comment	< 24	Undetermined.	Yes

Name: John McPherson Age: 38 Sex: M Race: C

Disease: Acute intestinal obstruction. Operation: Laparotomy. Appendicectomy. Drainage of pelvic abscess.

Anaesthetic risk: 2, emergency

PRE-OPERATIVE STATE:

The patient had had a previous right pneumonectomy for tuberculosis presented with signs of acute intestinal obstruction. The mediastinum was shifted over to the right, due toprevious surgery. The left lung appeared normal on clinical examination, although X-ray chest showed a small area of basal atelectasis. This was probably due to abdominal distension, and vital capacity did not appear grossly reduced. Adequate rehydration therapy was administered pre-operatively.

PREMEDICATION

Atropine 0.6 mg. intramuscularly, 45 minutes before anaesthesia.

ANAESTHETIC:

After 4 minutes of oxygen inhalation, anaesthesia was induced with the sequence thiopentone 200 mg., succinylcholine 50 mg., and oral endotracheal intubation. Anaesthesia was maintained with nitrous oxide and oxygen via a circle absorption system by an IPPR technique. dTc 20 mg. was given. Laparotomy revealed an acute appendicitis and a pelvic abscess. Appendicectomy was performed and the abscess drained. Bowel distension was relieved by a deflating enterostomy. The course of anaesthesia was quite uneventful. At the conclusion of the operation which lasted 60 minutes, residual curarsiation was reversed with neostigmine 2.5 mg preceded by atropine 1.2 mg. Normal adequate spontaneous respiration resumed and the patient regained consciousness rapidly. The post-operative course was uneventful until 12 hours later when the patient suffered acute respiratory distress. Vigorous physiotherapy was sufficient to relieve this. The need for tracheostomy was considered but, as there were no signs of a large amount of bronchial secretion, the idea was rejected. Six hours later (18 hours postoperatively) the patient suffered a similar attack of acute respiratory distress and died.

AUTOPSY

Old pneumonectomy scar on the right. Recent right paramedian wound with adjacent drainage sites. Pneumonectomy on right with deviation of heart and trachea to the right and dense fibrous adhesions. Congestion and oedema of left lung with an old healed tuberculous lesion in the upper lobe. Organising pelvic peritonitis involving several loops of small bowel with loculated abscesses and proximally dilated jejunum. Appendicectomy. Oedema of liver and kidneys. Right hydrocoele.

COMMENT

On clinical grounds, one would suppose this death to have been caused by secretional bronchial obstruction with massive collapse of the patient's only lung. Autopsy failed to demonstrate this, or any convincing cause for death. The pulmonary oedema was not marked and may have been the result of the ultimate cardiac failure. Whatever the cause of death, anaesthesia is not considered directly contributory.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
138.2.65	2	No comment	< 24	Cerebral laceration	Yes

Name: Carel Kampher Age: 25 Sex: M Race: C

Disease: Compound fracture of left parietal region of skull. Penetrating wound of abdomen.

Operation: Craniectomy for depressed fracture of skull. Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Unconscious. After transfusion with 4 pints blood, the B.P. was 140 mm.Hg systolic and the pulse rate 100/minute.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection, 45 minutes pre-anaesthesia.

ANAESTHETIC:

After topical analgesia of the larynx, oral intubation was performed. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was used for muscle relaxation, in two doses of 10 mg.

During the operation, at which multiple small gut perforations were sutured and a craniectomy was performed for a depressed skull fracture, 600 ml. blood was transfused. The course of anaesthesia, lasting 2 hours, was untoward. At the conclusion of the operation, adequate reversal of curarisation was achieved with neostigmine 1.5 mg. preceded by atropine 1.2 mg. Normal spontaneous and adequate respiration resumed. 10 gm. Mannitol 10% was given immediately post-operatively. The patient failed to regain consciousness and died 2 hours post-operatively.

AUTOPSY

Signs of operation on abdomen with resuturing of bowel. Operation on the head. Large area of brain laceration in the left parietal region. Haemorrhage into and necrosis of the pons.

COMMENT

This death was the result of existing cerebral injury. Anaesthesia was not contributory to this patient's demise.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
139.2.65	2	No comment	< 24	Uncontrolled haemorrhage	Yes

Name: Charles Ziervogel Age: 55 Sex: M Race: C

Disease: Profuse melaena Operation: Laparotomy.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Moribund, the patient had suffered profuse rectal haemorrhage. After transfusion of 11 pints blood, he was still in a state of severe oligaemic shock. B.P. was 60/40 mm.Hg, and pulse rate 100/minute. 50 m.Eq. sodium bicarbonate and 1 gm. calcium gluconate were given.

PREMEDICATION

Atropine 0.6 mg., intramuscularly, 45 minutes pre-operatively.

ANAESTHETIC:

After pre-oxygenation for 4 minutes, anaesthesia was induced with nitrous oxide, thiopentone 25 mg., succinylcholine 50 mg., followed by oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen delivered via a carbon dioxide circle absorption system by an IPPR technique. dTc was used in divided doses for relaxation, a total of 28 mg. being given throughout the operation, which lasted 150 minutes. During the procedure, a further 7 pints blood (warmed) was transfused together with 150 m.Eq. sodium bicarbonate. Calcium gluconate 2 gm. and 20 gm. Mannitol were also given. The B.P. was maintained at its original level of 80 mm.Hg systolic until the last hour of anaesthesia, when it rose to 110 mm.Hg systolic. At operation, no source of bleeding was found, though large and small bowel were widely explored with a sigmoidoscope. Eventually the rectum was packed. At the conclusion of the procedure residual curarisation was reversed with neostigmine 2.5 mg. preceded by atropine 1.2 mg. and normal spontaneous respiration resumed, the patient rapidly regaining consciousness at the conclusion of the anaesthetic. Post-operatively the haemorrhage from the rectum continued unabated. In spite of massive transfusion, the patient died 5 hours post-operatively. 24 pints blood had been transfused.

AUTOPSY

Large bowel filled with blood, fresh and old. Superficial inflammation of colon, with ulceration. Terminal ileitis. Leiomyosarcoma of the prostate communicating with the rectum, from which bleeding occurred.

COMMENT:

This death was due to haemorrhage from a leiomyosarcoma of the prostate. Anaesthesia was not contributory to the patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
140.2.65	3	No comment	ORD	Haemorrhage. Cardiac arrest.	Yes

Name: Iris Coutrier Age: 49 Sex: F Race: C

Disease: Mitral stenosis. Operation: Mitral valvotomy

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

This patient, who had severe mitral stenosis, presented for mitral valvotomy still in congestive cardiac failure, in spite of intensive medical treatment with digitalis and diuretics. She had recently had an attack of pulmonary oedema. The venous pressure was markedly raised. There was hepatomegaly and ascites. Before anaesthesia, B.P. was 120/80 mm.Hg and the pulse rate 140/minute. She was on anticoagulant therapy, the prothrombin index being 50%.

PREMEDICATION

Sodium seconal 90 mg., orally 2 hours before anaesthesia, atropine 0.6 mg. intramuscularly 45 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 250 mg., succinylcholine 80 mg., ventilation with oxygen, analgesia of the larynx, and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen delivered by an IPPR technique via a carbon dioxide circle absorption system. dTc was given for relaxation, the total dose being 30mg. The course of anaesthesia was untoward until during valvotomy, when a large tear was made in the left atrium, from the auricular appendage to the atrioventricular ring. Gross haemorrhage and cardiac arrest occurred. Rapid transfusion of 1,500 ml. warmed blood, cardiac massage and other resuscitative measures were of no avail.

AUTOPSY

Signs of a heart operation. Chronic congestive changes in the liver, lungs and kidneys. Thickening and narrowing of the mitral valve. Blood in the left pleural cavity.

COMMENT:

Death resulted from a surgical mishap which caused gross haemorrhage and cardiac arrest. Anaesthesia is not regarded as contributory to this death but, as this occurred while the patient was anaesthetised, the case is classed in group 3.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
141.2.65	2	No comment	< 24	Cerebral lobe glioma.	No

Name: Rae Rosenberg Age: 59 Sex: F Race: E

Disease: Cerebral tumour: temporal lobe glioma. Operation: Craniotomy. Partial excision of temporal lobe glioma.

Anaesthetic risk: 4.

PRE-OPERATIVE STATE:

1 week prior to this operation, the patient had a brain biopsy performed. Thereafter her condition deterioted progressively due to steadily increasing intracranial tension. This craniotomy was proposed as a last vain hope of salvage. She was deeply stuperose. Marked papilloedema was present. Scattered coarse rhonchi were audible over all lung fields. B.P. was 160/100 mm.Hg and pulse rate 90/minute. Haemoglobin concentration 14 gm.%.

PREMEDICATION

Atropine 0.6 mg. intramuscularly, 60 minutes pre-operatively.

ANAESTHETIC

After topical analgesia of the larynx, oral endotracheal intubation was performed. Anaesthesia was then induced and maintained with nitrous oxide and oxygen, 50%, delivered by a non-return system by an IPPR technique. dTc 50 mg. was administered for relaxation. During operation, 500 ml. 10% Mannitol was infused to reduce the intracranial tension. Blood was replaced as lost, 500 ml. being transfused in all. Throughout the procedure the B.P. was maintained at a level of 140 mm.Hg systolic.

At operation, the temporal lobe glioma was partially excised. At the conclusion of the procedure, residual curarisation was adequately reversed with neostigmine 2.5 mg preceded by atropine 1.2 mg. The patient did not regain consciousness and died 13 hours post-operatively.

AUTOPSY

No autopsy

COMMENT:

This death was caused by a large cerebral tumour and the effects of surgical removal. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
142.2.65	2	No comment	< 24	Cerebral laceration	Yes

Name: Graham Camphor Age: 19 Sex: M Race: C

Disease: Head injury. Operation: Craniotomy. Drainage of subdural haemorrhage.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Deeply unconscious. Respiration was deep, sighing and irregular - this was not due to metabolic acidosis, pH being 7.5, pCO_2 31 mm.Hg The pupils were fixed, the right being more dilated than the left. No corneal reflex waspresent. Before anaesthesia, the B.P. was 150/70 mm.Hg and the pulse rate 110/minute.

PREMEDICATION

Atropine 0.6 mg. intravenously immediately pre-anaesthesia.

ANAESTHETIC:

After transtracheal injectionof xylocaine 4%, oral endotracheal intubation was performed. Anaesthesia was then induced and maintained with nitrous oxide and oxygen, 50%, delivered via a carbon dioxide circle absorptionsystem by an IPPR technique. Halothane 0.5% was administered intermittently. Blood loss during operation was extensive and was replaced by blood transfusion, with 4 pints blood. At operation an extensive subdural haematoma and large intracerebral haematoma were drained. At the conclusion of the procedure, adequate spontaneous respiration was resumed but the patient failed to regain consciousness. He died 9 hours post-operatively.

AUTOPSY

Multiple bruises and lacerations over the body. Operation wound in right temperoparietal region of skull. Craniotomy wound and fracture of right parietal region of skull. Softening and laceration of brain. Bilateral bromchopneumonia.

COMMENT:

Death was due to severe cerebral injury. Anaesthesia was not contributory to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
143.2.65	3	No comment	ORD	Myocardial ischaemia. Cardiac arrest.	Yes

Name: Edward Seale Age: 71 Sex: M Race: C

Disease: Acute vascular occlusion of right iliac artery. Operation: Right iliac embolectomy

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Near moribund, in the opinions of the surgeon and anaesthetist. He had vascular occlusion of the right iliac artery, was orthopnoeic and suffered from congestive cardiac failure and atrial fibrillation due to previous myocardial infarction and cor pulmonale. Since discharge from hospital 2 years previously, he had been maintained on digitalis and diuretic medication. B.P. varied between 110 and 140 mm.Hg systolic. There was gross pulmonary emphysema and chronic bronchitis, with profuse amounts of purulent sputum. He was also pyrexial - temperature 102°F.

PREMEDICATION:

Nil. Pethidine had been given for pain 3 hours before operation.

ANAESTHETIC

A right unilateral spinal subarachnoid block was performed using 1.5 ml. heavy nupercaine solution injecged between L.3 and 4. vertebrae. After induction of spinal analgesia, the B.P. fell to 105 mm.Hg systolic, where it remained stable for the next hour. No reduction in the volume of pulmonary ventilation occurred. Operation was commenced 30 minutes after the induction of subarachnoid block, and 30 minutes later the B.P. suddenly fell precipitously. This was not due to profuse blood loss. Rapid infusion of 250 ml. plasma and 2 doses of 0.25 mg. phenylephrine had no effect. Cardiac arrest occurred within 10 minutes. External cardiac massage and IPPR with oxygen was commenced immediately without effect. In view of the patient's pre-operative condition, nothing further was done.

AUTOPSY

Large emphysematous bullae at right lung apex and many smaller emphysematous bullae in both lungs. Partial collapse of upper lobes of both lungs. Heart enlarged, weighing 600 gm. Aortic valve calcified, mitral valve stenosed and slightly incompetent. Aorta, both thoracic and abdominal, narrowed and calcified, as also femoral arteries. Coronary atherosclerosis. Retroperitoneal haemorrhage in region of right iliac artery.

COMMENT:

Before anaesthesia and operation, this patient was moribund. To some extent one must criticise the wisdom of operating on this patient at all, although it could be said to have offered him what little chance there was of survival. One must also question the wisdom of the induction of spinal analgesia, albeit unilateral, with the probability of sympathetic block and hypotension in a patient with coronary insufficiency. However, the drop in B.P. which followed induction of spinal analgesia was mild.

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The patient's gross pulmonary emphysema and productive chronic bronchitis was perhaps the main factor which decided the anaesthetist in favour of spinal analgesia. Here too, one may propose that endotracheal intubation and thorough bronchial toilette followed by IPPR, even with the difficulties in this type of patient, may have been better. On the other hand, the presence of emphysematous bullae discovered at autopsy may well have led to difficulties with this form of anaesthesia. It is probable that this patient would have died whatever the technique of anaesthesia.

The ultimate cause of death was probably myocardial ischaemia. Perhaps in the face of the calcified aortic valve and coronary arteries, the mild drop in B.P. which followed induction of spinal analgesia may be regarded as a contributory factor to his death. However, taken on balance, the anaesthetic is regarded as a necessarily and unavoidably contributory factor to this patient's death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
144.2.65	2	No comment	< 24	Haemorrhage.	Yes

Name: Natalia Mobongwana Age: 35 Sex: F Race: B

Disease: Arteriovenous fistula of splenic artery. Operation: Splenectomy and excision of arteriovenous fistula.

Anaesthetic risk: 2.

PRE-OPERATIVE STATE:

This patient had hypertension, B.P. 175/120 mm.Hg, and cardiomegaly. Other than this, her condition was good.

PREMEDICATION

Omnopon 10 mg., atropine 0.6 mg. by intramuscular injection 60 minutes before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 350 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen, via a carbon dioxide circle absorption system by an IPPR technique. Halothane 0.5% was used intermittently. dTc 25 mg. was given during the operation, which lasted 75 minutes, for relaxation. The course of anaesthesia was untoward. At the conclusion of the procedure, residual curarisation was reversed adequately with neostigmine 2.5 mg. preceded by atropine 1.2 mg. The patient rapidly regained consciousness. Two hours later there was a sudden gush of blood through the abdominal drain, and the patient suffered a cardiac arrest. External cardiac massage was performed, but to no avail.

AUTOPSY

Signs of previous splenectomy. 1100 cc. free blood in the peritoneal cavity. All organs pale and anaemic.

COMMENT

Death followed profuse post-operative haemorrhage, apparently from a slipped ligature, and was unrelated to the anaesthetic.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
145.2.65	2	No comment	< 24	Cerebro-vascular accident	No

Name: Jacobus Smith Age: 57 Sex: M Race: E

Disease: Intracerebral haemorrhage.

Operation: Carotid angiography. Burrhole craniotomy. Tracheostomy.

Anaesthetic risk: 3.

PRE-OPERATIVE STATE:

Comatose. He had a left hemiplegia, was hypertensive (B.P. 235/130 mm.Hg) and was a known porphyric.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection 45 minutes before anaesthesia.

ANAESTHETIC

After topical analgesia of the larynx, oral endotracheal intubation was performed. Anaesthesia was then induced and maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. Carotid angiography followed by burrhole craniotomy, revealed a large intracerebral haematoma. Hydrocephalus and oedema of the brain were also present. At the end of operation, elective tracheostomy was performed. Spontaneous respiration of adequate volume resumed. The patient failed to recover consciousness and died 7 hours after operation.

AUTOPSY

No autopsy

COMMENT:

This patient died as a result of a cerebro-vascular accident.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
146.2.65	2	No comment	< 24	Haemor-rhage.	Yes

Name: Antony Chambers Age: 65 Sex: M Race: E

Disease: Ruptured abdominal aortic aneurysm Operation: Resection and graft of abdominal aorta.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Following rupture of an abdominal aortic aneurysm, the patient was in a state of severe oligaemic shock. B.P. 100 mm.Hg systolic. Pulse rate 120/minute. Respiration was panting. 1 pint blood was transfused.

PREMEDICATION:

Atropine 0.6 mg. intravenously, immediately before anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen via a carbon dioxide circle absorption system by an IPPR technique. dTc was used, total dose 35 mg., for relaxation during the operation, which lasted 3 hours. Massive blood loss occurred during surgery and was replaced by equally massive transfusion; 14 pints warmed blood were given and 150 m.Eq. sodium bicarbonate was also infused. Grave difficulty was experienced in the operation with anastomosis of the aortic graft, because of the extreme softness and degeneracy of the aortic wall. The B.P. was maintained at a level of between 100 and 120 mm.Hg systolic throughout the procedure. At the conclusion of the operation, residual curarisation was adequately reversed with neostigmine 2.0 mg. preceded by atropine 1.2 mg. The patient rapidly regained consciousness. However, post-operatively, haemorrhage continued and the patient died 3 hours later.

AUTOPSY

Recent abdominal operation. Resection of lower end of aorta and replacement with Teflon prosthesis. Gross generalised atherosclerosis. Lumena of coronary arteries appeared completely blocked. Basilar artery also appeared blocked. Enlarged left ventricle.

COMMENT:

This patient died of continued haemorrhage in the post-operative period. Anaesthesia was not contributory to this death.

CASE NO.	CLASSIFICATION Group	PREVENTABILITY	TIME OF DEATH	CAUSE OF DEATH	AUTOPSY
147.2.65	2	No comment	<24	?Myocardial ischaemia	Yes

Name: C. Cronje Age: 58 Sex: M Race: E

Disease: Ruptured abdominal aortic aneurysm. Operation: Resection and graft of abdominal aorta.

Anaesthetic risk: 4, emergency.

PRE-OPERATIVE STATE:

Following rupture of abdominal aortic aneurysm, he was in a state of gross oligaemic shock. B.P. 80 mm.Hg systolic, pulse rate 100/minute. Myocardial ischaemia was evident on ECG and multiple extrasystoles were present. He also had gross chronic bronchitis. He had had a myocardial infarct 8 years previously. 1 litre Ringer's lactate solution had been infused pre-operatively.

PREMEDICATION

Atropine 0.6 mg. by intramuscular injection, 45 minutes pre-anaesthesia.

ANAESTHETIC:

Anaesthesia was induced with the sequence thiopentone 150 mg., succinylcholine 50 mg., ventilation with oxygen, topical analgesia of the larynx and oral endotracheal intubation. Anaesthesia was then maintained with nitrous oxide and oxygen administered via a carbon dioxide circle absorption system by an IPPR technique. dTc was used for the relaxant, total dose being 50 mg. during the 6 hours of operation. Profuse haemorrhage occurred when resection and graft of the abdominal aorta was being performed, requiring massive blood transfusion, 20 pints warmed blood being given. A base deficit of -13 m.Eq./l. during operation was reduced by the infusion of 170 m.Eq. sodium bicarbonate to -3 m.Eq./l. by the end of the procedure. At the conclusion of the operation, residual curarisation was reversed with neostigmine 3mg. preceded by atropine 1.2 mg. Spontaneous respiration was resumed but was of inadequate volume. A further 2 mg. neostigmine was given and produced no change in the volume of respiration. Tracheostomy was performed and respiration assisted with a Bird ventilator post-operatively. The patient regained consciousness rapidly on discontinuance of anaesthesia, and was returned to the ward. B.P. was maintained at a level of 100 mm.Hg systolic for some hours post-operatively but 10 hours after operation he died suddenly. Throughout the operation the patient had been anuric and this anuria continued throughout the post-operative period.

AUTOPSY

Teflon graft replacing lower end and bifurcation of aorta. Congestion of liver and kidneys. Enlarged heart with gross coronary artery atherosclerosis.

COMMENT:

This patient's death - when it occurred - did not appear to be related to the anaesthetic administered. Clinically, death appeared to result from myocardial ischaemia.