With F.D. de T. von Zijlo's compliments.
The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.
INTRODUCTION

In presenting this thesis I have to admit, that the study, as far as the spinal anaesthesia part is concerned, was undertaken with frank scepticism. This I do not regret, for I was thereby able to view the effects of spinal anaesthesia most critically. The outcome was that by slow gradations scepticism had passed to real enthusiasm. This is not for spinal anaesthesia to be adopted in every case that needs to be anaesthetised, but for its use in carefully selected cases. This includes a very large proportion of all major operations below the diaphragm. It excludes all above this level. My statement can be criticised as being the outcome of a very limited experience. I can only say that the number was large enough to give me a thorough practical training in the subject and enable me with better understanding to appreciate the writings of those with greater experience. Besides, my deductions are in complete accord with those of men whose extensive experience and experimental and clinical investigations have qualified them to speak authoritatively.
As is implied by the title, something more than spinal anaesthesia is discussed. Experience as a general practitioner in a country district has impressed on my mind the dread the ordinary patient has of a general anaesthetic. Repeatedly I have seen operations postponed, or even altogether put off, for fear of the dreaded chloroform. How many children, undergoing a minor operation, have not been scarred for life by a suffocating anaesthetic mask, a never-to-be forgotten experience? Can we do anything to allay this fear? Can the same end not be reached by less unpleasant means?

This concerns the humane side chiefly. There is a very much stronger argument for the use of combined anaesthesia.

Crile’s researches have proved beyond doubt that the great importance of psychic shock. He has demonstrated how fear, like all other shock producing influences, produces marked pathological changes in the body, even to death. His method of avoiding shock in operations for exophthalmic goitre has become classical. That fear is a very potent shock-producing factor, becomes only too evident on a visit to some continental clinics, where local anaesthesia has been developed to a wonderful finale, but where inadequate premedication seems to be the rule, rather than the exception.
There is available today a large and efficient assortment of drugs, which are used as an aid to anaesthesia in the form of either preliminary medicaments or basal narcotics. My experience, apart from morphine and scopolamine, has mostly been with Avertin. For this reason Avertin will be chiefly referred to as the other important partner in the combination. This by no means reflects on the usefulness of drugs like nembutal, pernocton, etc. as basal anaesthetics. They are all favourably reported on.

It must be clearly understood, that I in no way wish to distract from the very great importance of Ether and other drugs as anaesthetic agents. They all have their proper place and use. I shall merely endeavour, in the light of my experience and by reviewing the current literature, to point out the usefulness of combined anaesthesia, and particularly refer to a combination of spinal anaesthesia and some narcotic or basal anaesthetic. I shall indicate that in a large number of cases this should be the method of choice. No attempt will be made to consider all matters pertaining to spinal or combined anaesthesia. I shall deal with such points as seem to be of practical importance to the operating surgeon and give my views on certain controversial subjects in the light of my clinical investigations and experience.
HISTORICAL

The use of inhalation narcosis dates from Sir Humphry Davy's experiment of inhaling nitrous oxide, as early as 1800. He then raised the question of the possible use of that substance as an anaesthetic agent. Yet it was not until 1844 that Wells first introduced the gas to dentistry. Ether was used in 1842 by Long of Georgia. It was not introduced to England, as a possible means of producing insensibility to pain, till 1846. Close on the heels of this innovation followed the use of Chloroform by Sir James Simpson of Edinburgh in 1847.

A new type of anaesthesia was made possible by the discovery of the properties of cocaine as a local anaesthetic, by Koller in 1884. This field of anaesthesia has been greatly developed, especially during the past twenty years, though cocaine itself is now discarded save for eye, throat, and nose operations. Less toxic and equally effective substitutes such as novacine, stovaine, novutox and many others are in daily use everywhere.

Local anaesthesia, as applied to the spinal nerve roots, that is spinal anaesthesia, was first suggested by Corning in 1885, but Bier of Bonn was the first to use it clinically in 1899. Characteristic it was of him to submit
his own body for the production of this new anaesthesia. Babcock was the first to take it up as his routine form of anaesthesia in 1904. The work of men such as Bier, Babcock and Barker has placed spinal anaesthesia on its present sound foundation. More recently Pitkin and Howard Jones have done pioneer work in the field of drugs with a specific gravity less than that of the cerebrospinal fluid. Spinal anaesthesia has emerged from the dangerous period, in which it was considered necessary merely to inject a solution of a local anaesthetic into the subarachnoid space and then to operate with little further attention to the patient. The need for a very precise technique to be gained only by considerable experience, and for the constant careful supervision of the anaesthetised patient, is now recognised.

The use of cocain and of rectal ether represented the first attempts at the evasion of the distressing process of induction by inhalation. Roux's attempt in 1846 with an aqueous solution of ether did little but injure the bowel, similarly Pirogoff's experiment with ether vapour in 1847. It was not until 1913 that rectal narcosis again merited attention, following Gwathmey's introduction of an ether-oil solution. Continuing research in the rectal administration of anaesthetic agents Willstatter and Duisberg in 1923 manufactured Avertin which was introduced into practice by
Prof. Eichholz. After searching experiments and clinical use in thousands of cases, it has now become a valued and popular adjunct to the armoury of the surgeon in his battle against pain and shock.

SHOCK

The term shock denotes a depression of all the vital functions of the body.

Clinical and pathological evidence warrant the recognition of two distinct phases in the evolution of shock, an early or primary phase, and a late or secondary phase. Each may arise independently. The physiology of their production differs, but the ultimate pathological state which they produce is identical.

Primary Shock depends on the nervous connections between the vital cerebral centres and the periphery. It appears to result from excessive stimulation of afferent nerves. The degree of shock depends on factors such as the sensitivity of the central controlling nerve cells and the peripheral receiving and conducting nerve endings and fibres, the specialization of the nerve endings and the quantity and quality of the stimuli.

The more sensitive the nerve mechanism, the more marked will be the shock. Sensitisation is produced in many ways. A typical example is that produced by the toxic
secretion of the thyroid gland in Exophthalmic goitre.

The nature of the nerve endings exposed to stimulation is an important factor. Injury of a part supplied by highly specialized nerve endings, such as exist in the palm of the hand, leads to a greater degree of shock than injury of parts supplied by less specialized nerves. Shock-producing stimuli can also reach the brain via the nerves of the special senses of sight and hearing.

The quantity and the quality of the peripheral stimuli bear a relation to the degree of shock. A superficial burn over a wide surface causes much more intensive shock than a more destructive burn involving a small area. Painful stimuli are more shock producing than painless ones. Shock arising in such a manner depends both on the pain and the quality of the affected nerve for its production. By irritating the parietal peritoneum or dragging on the mesentery in the unanaesthetised animal we can produce primary shock. Pain alone is not responsible for this, for a similar experiment will cause shock in an animal under ether anaesthesia. There will be a difference in the degree, but not in the kind of shock. The elimination of pain in the second experiment diminishes the effects of the stimuli, but does not abolish them altogether.
Primary shock may be manifest immediately after the receipt of a severe injury such as a road accident, or it may take a little time to develop when it depends on the summation of repeated smaller injuries such as result during a surgical operation.

Secondary Shock depends on the other channel of communication between the centres and the periphery - the cardiovascular system. Since the work of Bayliss and Cannon, Dale and others there remained little doubt that the causative factor in the production of secondary shock is a toxin absorbed from the seat of trauma. Bayliss and Cannon found that shock did not develop when they tied the main vein before crushing the muscles of a limb, indicating that the products of muscle disintegration absorbed into the circulation were responsible for the development of a shock-like condition. The injection of extracts of crushed muscle will produce clinical shock.

Dale and Laidlaw showed that the injection of histamine will lead to a condition closely resembling shock. Histamine can be extracted from almost every tissue of the body. It is, therefore, not unreasonable to assume that a histamine-like body, perhaps histamine itself, is responsible for the causation of secondary shock.

The manifestations of secondary shock are as a rule not typically developed until 12 or 24 hours after the receipt of trauma, but often they are seen much earlier. This is
especially the case when predisposing causes such as exposure, fear, septic infection etc have been present. After a surgical operation the patient frequently recovers clinically from primary shock before the signs of secondary shock develop. The primary shock may, however, be so severe that its manifestations pass imperceptably into those of secondary shock. This is especially true in cases of much haemorrhage. It is clear that the worse state primary shock leaves the vital centres in, the more readily will they suffer through the onslaughts of the agents causing secondary shock.

PREVENTION OF SHOCK

The surgeon has almost mastered pain and infection, but shock is still his most merciless enemy. Any progress, therefore, in his battle against shock is pregnant with possibilities. With this idea in mind we do not hesitate to put forward our views.

In accidental surgery primary shock usually plays the more important part, but in ordinary routine surgery secondary shock represents the more serious phase and is the one that often leads to the fatal issue. Our struggle against secondary shock must be mostly one of prevention. Assuming that histamine is the causal agent, it will be...
essential to produce as little of it as possible. Gentle handling of tissues, clean cutting etc., are therefore, advisable. No operation, however, can be performed without liberating some histamine and unfortunately we possess no specific antibody which will destroy it or combat its effects. It seems that until such an antibody is forthcoming we must direct our attention towards fortifying the vulnerable centres against the effects of histamine. To do this successfully demands great resourcefulness for so many and varied are the causes that predispose the tissues to the effects of histamine. No purpose will be served by discussing these here.

Everything else being equal, the degree of secondary shock is directly proportional to the degree of primary shock. If primary shock can be eliminated or minimized, secondary shock will correspondingly decrease. If there has been no damage done by primary shock the tissues are left in a more favourable position to withstand the effects of histamine. We believe that primary shock can be almost completely prevented during most operations and that thereby secondary shock can be greatly diminished. This forms the basis of our argument.
DEFINITIONS

Where we use the unqualified term "shock" in the rest of our discussion we intend it to convey the idea of "primary shock".

As primary shock can be caused by psychical as well as traumatic influences we use the term "psychic" and "traumatic" to denote the cause of the primary shock.

In dealing with secondary shock we use the term "histamine" to indicate that the shock was caused by an agent, probably histamine-like, absorbed from the site of trauma.

"Toxic shock" will indicate a shock-like condition caused by the absorption of bacterial products.

THE IDEAL ANAESTHETIC

The aim of anaesthesia is to protect the patient against pain and shock.

The supreme test of an anaesthetic is the highest possibly safety of the patient. If with this we can combine comfort without jeopardising safety, it will be inhuman not to do so. Various anaesthetic agents appear to suit various operative fields. We cannot discuss each separately. We shall chiefly devote our attention to one big surgical field,
the abdomen, and consider what is required of an anaesthetic to stand the supreme test in this sphere.

What does this surgeon require?

I. An agent which prevents all noxious stimuli whether psychic or traumatic from reaching the higher cerebral centres, in other words, COMPLETE ANOCIATION. This demands far more than mere unconsciousness and insensibility to pain.

II. An agent which gives complete MUSCULAR RELAXATION, enabling the surgeon to perform delicate and difficult manoeuvres without undue traction on the mesenteries and the peritoneum and without pressure damage to sensitive viscera.

III. An agent whose beneficial effects (relaxation etc.) will last for the whole period of the operation.

IV. An agent which will, both generally and locally, leave the tissues, as nearly as possible in their normal state, that is, the LEAST TOXIC agent. The local effects concern the parts with which the anaesthetic comes into direct contact. The general effects concern those, with which there is contact after blood absorption or where harm is more indirectly caused. The brain, heart, lungs, liver, kidneys and supra-renal glands are the organs chiefly affected. The more nearly normal the tissues are left, the

Each point is fully considered later on.
less fear of complications there will be and the speedier the recovery.

V. An agent requiring the simplest equipment for its administration and a technique which can be acquired by everybody, called upon to anaesthetise patients.

The chief points to be considered from the patient's point of view are:

VI. An agent which will give a rapid, painless and pleasant induction, thus avoiding the horror of the induction period so prevalent today.

VII. An agent which will lead to a pleasant recovery, without the usual concomitants associated with inhalation narcosis, e.g. headache and pain and discomfort incident to vomiting. In other words, a recovery following anaesthesia produced by the least toxic agent.

A CONSIDERATION OF THE ABOVE QUALITIES - VARIOUS ANAESTHETICS EXAMINED

I. ANOCIATION (Crile)

Crile's work in this field is of such outstanding merit, that one will be excused for referring to him at some length.

(1) Noxious influences originating in the operation area. Crile has produced abundant proof, that certain harmful influences constantly produce identical changes
in certain vital organs of the body. Whether these changes are the real cause or the effects of shock is not certain. They and further changes noticed by Rendel Short are, however, considered to constitute an essential part, if not the whole, of the pathology of shock. The influences whereby these changes can be brought about are now known as the "causes of shock."

Crile (1) describes the following changes:

(a) BRAIN. The brain cells show first stage of hyperacidity characterised by hyperchromatism and later a stage of exhaustion, characterised by chromatolysis, by alteration of the nucleus-plasma relation, by rupture of the nuclear and the cell membranes and finally by disintegration. These changes are most marked in the cerebellum and the cortex and are present also in the medulla and cord. No changes, however, are noticed, when the brain has previously been severed from the part that is injured, whether by local anaesthesia or by section of the cord.

(b) LIVER. Enlargement or swelling of the liver cells, general disappearance of the cytoplasm, the presence of vacuolated spaces, displacement and occasional disappearance of nuclei are the prominent changes seen. They are most marked near the periphery of the lobules.

(1) Surgical interpretation of Shock. pages. 40, 41 & 49
(c) SUPRARENAL GLANDS. The changes in the cortex are most marked. The medullary cells may appear almost normal, while extensive changes exist in the cortex. Histologically there is enlargement of the cells with occasional rupture of their membranes, distortion of their outlines, eccentric displacement of the nuclei, which may be crenated, and general disappearance of cytoplasm.

Such is the destruction caused in three vital organs by trauma, haemorrhage (anaemia), exposure, fatigue, emotion, insomnia, bacterial toxins, certain anaesthetic agents etc. The ideal anaesthetic must prevent these changes.

It has been shown that local anaesthesia or spinal anaesthesia (which amounts to severing of the cord) efficiently bars noxious stimuli from travelling up the afferent paths of peripheral nerves or spinal cord to the brain. But harmful impulses originating elsewhere require to be blocked in some other way. By eliminating consciousness ether acts efficiently in the latter sense, but does it comply with the first? Far from it. It has been shown (2) that Ether affords the brain no protection against the harmful impulses originating in the operation field. What is more, it actually produces changes in the brain etc., which in no way are distinguishable from those produced by trauma, without anaesthesia. In other words, ether is a shock producing agent. So much so, is this the case, that animals subjected to ether anaesthesia for 4 or 6 hours, without trauma, may die the following day (3) The failure of (2) (3) Crile: Surgical interpretation of Shock p. 74
ether to prevent harmful stimuli, originating in the operation field, from reaching the higher centres is thus explained by Crile (4): "The lipoid solvent anaesthetics probably break the arc which maintains consciousness beyond the brain cells, somewhere in the efferent path, perhaps at the synapse. The afferent path from the seat of injury being unbroken, the afferent stimuli reach and modify the brain cells, as readily as if no anaesthetic had been given, and it would seem that the brain cell changes must be due to the response of the brain as if in a futile effort to escape from the injury."

Dale has shown that the anaesthetics (chloroform and Ether, but not nitrous oxide), sensitize the capillaries to the action of histamine, so that in unanaesthetized animals about ten times the dose of the drug is needed to produce an equivalent depression of blood pressure. If histamine is the causal factor in secondary shock, it is clear that ether may contribute considerably towards magnifying this shock.

Apart from certain toxic changes, to which we refer later, we may sum up thus:

(a) Ether does not prevent traumatic shock
(b) Ether produces changes indistinguishable from shock.
(c) Ether enhances histamine shock
(d) Ether prevents psychic shock.

(4) Surgical Interpretation of Shock. p. 43
Is spinal anaesthesia in these respects of any greater advantage? We have seen that, by severing the higher centres from the site of trauma, it prevents traumatic shock. There remains, however, a phenomenon of the gravest concern to be reckoned with. Spinal anaesthesia produces a fall of blood pressure with a consequent anaemia of the brain. Lack of blood, (therefore lack of oxygen) causes damage to the brain cells, essentially similar to the damage caused by ether or unblocked traumatic impulses. Thus, as a secondary effect, spinal anaesthesia produces, to a minor or major degree, depending on the extent and duration of the blood pressure drop, the very condition it is intended to prevent.

We shall have to decide, whether the damage, if any, done by the anaemia, outweighs the advantages derived from prevention of traumatic shock, or vice versa (see under blood pressure). The factor of anaemia assumes the utmost importance when we have to deal with cases of low blood pressure, be that due to haemorrhage, shock or any other cause. We may assume, for the moment, that the drug in itself does not cause shock, as does ether.

\(x\). It must be clearly understood that "traumatic shock" used in this sense does not refer to secondary shock.
As spinal Anaesthesia does not dull the higher centres the brain remains unprotected against the effects of mental influences. Psychic Shock is not prevented.

We conclude that:

(a) **SPINAL ANAESTHESIA PREVENTS TRAUMATIC SHOCK**

(b) **SPINAL ANAESTHESIA MAY PRODUCE CHANGES INDISTINGUISHABLE FROM SHOCK, IF THERE IS SEVERE AND PROLONGED FALL OF BLOOD PRESSURE.**

(c) **SPINAL ANAESTHESIA DOES NOT PREVENT PSYCHIC SHOCK.**

2. **PSYCHIC OR EMOTIONAL SHOCK.**

Noxious stimuli can reach the brain from many sources other than the field of operation. The patient's own mind is a most fruitful source. He fears the pain of the treatment, the anaesthetic and death, he worries about a possible incurable disease, about long unemployment and financial loss. In his strange surroundings harmful stimuli reach him by way of his ears, eyes and nose. All this worry, fear and anxiety produce "lesions that are truly physical lesions as are fractures." Fear may reduce the organism to a state of complete exhaustion even to death. Nobody has yet explained satisfactorily the process by which it takes place. Over-stimulation probably leads to over-reaction on the part of the higher centres, but the reaction does not necessarily present any external manifestations.
Part of the reaction appears to result in efferent impulses to the suprarenal glands for there is always hyper-secretion of adrenalin during emotional states. It has been suggested that sudden death from fear is due to a rapid out-pouring of an overdose of adrenalin into the circulation.

Crile was able to demonstrate changes in the brain etc., after prolonged fear similar to what he found after traumatic shock.

Surgeons have long recognised how important a factor fear is in determining the recovery of their patients. The patient who views matters light-heartedly does well, whereas the one who is convinced that he will die, dies.

Such things may happen to those who are physically normal, apart from the complaint which necessitates the operation. When, however, we deal with patients whose nervous system has become utterly exhausted by prolonged exposure, by the effects of bacterial poisons or by toxic thyroid secretions, psychic shock becomes a matter of far greater importance. The truth of this has been demonstrated over and over again in the "stealing" method of treating exophthalmic goitre.

When operating under spinal anaesthesia emotional influences may profoundly affect the patient. In order to eliminate them morphia and scopolomine are commonly used. More recently several drugs of the barbiturate series and avertin
have become more popular. They possess distinct advantages in producing more prolonged and profound unconsciousness without possessing some of the disadvantages of morphia and scopolomine.

COMPLETE ANOSCIATION. We have drawn attention to the advantages of spinal anaesthesia in preventing traumatic shock and noted the use of Avertin in preventing emotional or psychic shock. In many of our cases a combination of the two has been used. Such might have approached the ideal if it were not for certain drawbacks. Of these the outstanding one is fall of blood pressure. Both spinal anaesthesia and avertin amnesia cause the blood pressure to drop. We have to consider later on, how serious a disadvantage this is, and whether we can do anything to remove or diminish it.

III. RELAXATION

This qualification of an anaesthetic is such an obvious one, that the point need hardly be laboured.

A surgeon struggling through a tight abdominal wall, causes marked shock to his patient by irritating the parietal peritoneum, by dragging on the mesentery and by pressing on the sensitive nerve plexuses. Apart from causing "traumatic shock" in such a way, his hand, by bruising muscles and organs, in its struggle, also adds to the degree of "histamine shock", that must necessarily follow absorption from the traumatised areas.
Can we in rigid cases estimate the precise amount of damage the assistant's retractor is doing to such an important and vital organ as the liver, one of the regulators of metabolism? Similarly irreparable damage may be done to the pancreas. Finisterer holds that many deaths after upper abdominal operations are due to a bruised pancreas. Does technique not suffer for lack of relaxation? Relaxation, however, can be obtained, and retained, even in a powerful muscular man, but at what cost? The patient has to be saturated with a highly toxic drug, which damages liver, lungs and kidneys, and yet does not protect him completely against shock, moreover produces shock. Loss of tone in the voluntary muscles depends on sympathetic paralysis. It is the first to go in spinal anaesthesia, the last in inhalation narcosis.

The wonderful and complete relaxation obtained by means of spinal anaesthesia would certainly proclaim it as the ideal anaesthetic, if relaxation were the only point to be considered. As a beginner in abdominal surgery, one considered the relaxation afforded by spinal anaesthesia, one's best assistant.

PROLAPSE OF INTESTINES. It seems quite in place here to remark about the problem of prolapse of the intestines through the operation wound. Apart from being a mere nuisance and prolonging the operation, prolapse has been shown to be one of the most important shock producing factors in
abdominal operations. Basil Hughes (5) has by careful readings shown how the blood pressure fell with each loop of bowel withdrawn from the abdomen. The obvious inference is never to allow the intestine to prolapse and, when it is necessary to withdraw, do this systematically, loop by loop, replacing each loop before the next one is withdrawn. Can spinal anaesthesia assist us here? Everyone is acquainted with the trouble one sometimes has, even under sound general anaesthesia, with protruding gut. When repairing a large ventral hernia, or dealing with an obstructed colon the surgeon may have the greatest difficulty in preventing the bowel from escaping from the abdomen. Excessive handling of distended bowel in obstruction also increases the shock in these cases.

When operating under spinal anaesthesia it is a striking thing to see how the gut appears to recede to the posterior abdominal wall. The small intestine especially is contracted and peristaltic. The anterior abdominal wall is so relaxed that it can literally be lifted off the bowels. It is only in the most advanced cases of obstruction that I have noticed any tendency at all for the intestines to protrude under spinal anaesthesia.

III. DURATION OF ANAESTHESIA

That the effects of the anaesthetic should last for the duration of the operation, appears self evident. Fuller

consideration, however, will show the importance of this statement. Why do we consider it a serious matter for any operation to be prolonged beyond 1½ to 2 hours? The reason is, that many patients will not stand two hours of noxious impulses from the operation field making their onslaughts on an unprotected brain, plus the rapid poisoning by an intensely toxic anaesthetic agent, administered over such a prolonged period. The effects of a general anaesthetic can be made to last the duration of the operation, but not without severely prejudicing the patient’s convalescence.

Does spinal anaesthesia offer any advantage? One can fairly safely assert that, provided the patient is well protected against psychic shock by means of a basal anaesthetic, is not allowed to suffer from the effects of exposure and from anoxaemia, (a potent shock producing factor) the length of the operation will make no difference to him, while adequate anaesthesia lasts, except in respect of histamine shock which will depend on the amount of tissue damage done. One naturally takes it for granted, that all the principles of surgery are adhered to, just as in the case of general anaesthesia. Until recently the great difficulty has been to get spinal anaesthesia to outlast extra long operative procedures. With the introduction of percan, this difficulty has been solved. Not only can complete anociation be maintained
for a three hours' operation, but the patient will be free from pain for several hours longer. My practical experience of percaín is limited to a few cases but through the kindness of Dr Howard Jones I was given excellent opportunities of studying its effects and usefulness. As a result of this and judging by the favourable reports of others, I entirely agree that percaín is a most valuable drug, and should be the one of choice, for any infra-diaphragmatic operation, that will last upwards of 1½ hours. The method to which I have become accustomed in this type of case will become clear from the later description. Briefly the procedure was to supplement spinal with some other form of anaesthesia when it appeared that the operation would be too protracted. The supplementary anaesthetic was either a splanchnic injection, nitrous oxide-oxygeh or Ether. Generally, however, spinal anaesthesia, by the method we employed, could be relied upon to last 1 hour and 20 minutes. During this time most of the ordinary abdominal operations can be completed.

IV. TOXICITY OF ANAESTHETIC AGENT

Next to protecting the patient against pain and shock, the intact preservation of his tissues and organs is the most important principle in anaesthesia. The two principles are really very similar. We have noted how the clinical manifestations of shock are always associated with certain
definite pathological changes in at least three of the vital organs of the body (whether these changes are to be considered as cause or effect we do not know) Experimental and clinical evidence very strongly indicate that these changes are brought about by a wide range of influences or agents or a combination of such influences or agents. Amongst these we have noticed an influence such as a series of charges along unprotected afferent nerve paths and we have discussed the effects of bacterial toxins, of histamine and of certain anaesthetic agents. In our quest for an ideal anaesthetic agent, we must look for one (or a combination) which will not only protect the higher centres but will, per se, do no harm to any of the body tissues. So far this has been unattainable, and, therefore, we have to find those agents that will do least damage. Such an extensive examination being altogether beyond the scope of this thesis. I shall largely limit my remarks to the drugs, concerned in the cases reported on, namely avertin, stovain, spinocain and percaibe.

(1) AVERTIN

(a) Rectal Mucosa. About 50 cases of proctitis were reported during the first few years of the introduction of Avertin. Since then greater carefulness in testing the solution has practically eliminated the condition. It is
caused by the presence of hydrobromic acid in the solution. This substance gives a blue colouration when Congo Red is added to the solution. A negative Congo Red test and a healthy mucous membrane are absolute safe-guards against the condition.

(b) **Brain.** The basal centres are somewhat depressed, but no organic changes are produced.

(c) **Heart.** The heart's action is slightly depressed while amnesia lasts, but no destructive changes are produced. Raginsky and Bourne (6) state that in normal doses Avertin has no deleterious effect on the heart and after repeated doses they were unable to discover any pathological changes. Such also is the evidence of the introducers of the drug.

(d) **Lungs.** Respiratory complications after avertin appear to be much less common than after ether. In 1000 gynaecological operations Young (7) has had 3 cases of mild bronchitis, 1 case of pleurisy and 3 cases of broncho-pneumonia of which one terminated fatally. Probably the most important reason for this was the small quantity of inhalation anaesthetic required. Finisterer (8) and Dandy (9) report to the same effect. Dandy's report is striking. He has not had a

(6) Current Researches in Anaesthesia and Analgesia Jan-Feb 1932
(8†) Personal Communication
(9) J. Am. Med. Assoc. 30th May 1931
single anaesthetic death, no post-operative pneumonia and no other deleterious effect immediate or remote in 250 cranial operations of every type. (We realise, however, that pulomonary complications are seldom seen after cranial operations.)

(e) Liver. Two conflicting views exist concerning the use of avertin in diseases of the liver. The chemical composition of the drug is such, that there are theoretical grounds for the belief, that it may damage the liver (10) (11). Raginsky and Bourne (12) after damaging the liver of dogs with chloroform, and then repeatedly administering avertin, found that they were able to produce an additional 5-30% damage as measured by the tetrabromphenolphthalein dye test. This damage was of the nature of parenchimatous and occasionally fatty degeneration. Widenhorn (13) has found an increased blood sugar following the administration of avertin. Shipway (14) on the other hand, on the strength of his extensive clinical experience disregards the possibility of liver damage. Ashworth (15) too is of opinion that little need be feared in cases of liver damage but bases his remarks on 3 cases only. Two of these were jaundiced and suffered no ill effects from the avertin. Yet Seiffer (16) and others as a result of their clinical experience, have

(10) Wilcox: Lancet 11th July 1931
(11) Parsons. Lancet. 6th June. 1931
(12) Anaesthesia and Analgesia Jan-Feb. 1932
(13) " " March-April 1932
(14) Lancet. 6th June 1931
(16) Zentralblatt fur Chirurgie 1929 Nr 35. 2183. P.
warned against its use in diseases of the liver.

We had only 2 cases of rather serious collapse after avertin. Both occurred after cholecystectomy for gall stones. One patient was jaundiced. In view of the warnings expressed by some this experience made us hesitate to use avertin for cases of hepatic disease, until we had carried out the following investigation.

I performed functional tests for liver efficiency in 12 patients who had Avertin and Stovain combined anaesthesia, avertin being given in the dose of 0.1 grm. per kilogram of body weight. (Details of test will follow) I demonstrated that there was evidence of liver damage in 4 cases. One of the 4 cases will be examined more closely on account of the doubt that exists concerning the use of Avertin in diseases of the liver.

All 4 cases had laparotomy performed. Their livers were inspected. In 3 there was no evidence of disease. In one case (H.J.) with a diagnosis of duodenal ulcer we found no ulcer but an advanced degree of alcoholic cirrhosis of the liver. This case and one other of the four had given a positive laevulose test before the operation, that is, in 2 cases out of 4 we had evidence of deficient liver function before avertin had been administered. The readings of the case H.J. are worth quoting, because here we not only had a positive test before and after operation indicating liver
deficiency, but we actually found a cirrhotic liver during operation. There could exist not the least doubt in this case. The readings we obtained are in entire agreement with the findings of Raginsky and Bourne in the animal experiments we have quoted. On the day before the operation the patient's fasting blood sugar was estimated and a carbohydrate tolerance test was made immediately afterwards. The following readings were obtained:

<table>
<thead>
<tr>
<th>Fasting blood sugar before operation</th>
<th>107</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Sugar ½ hour after giving 50 grm of laevulose orally</td>
<td>98</td>
</tr>
<tr>
<td>&quot; 1 hour &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; 2 hours &quot; &quot; &quot; &quot; &quot; &quot;</td>
<td>153</td>
</tr>
<tr>
<td>131</td>
<td></td>
</tr>
</tbody>
</table>

(The apparent fall at the ½ hour period is within the limits of observational error) We notice here, that the blood sugar level rose from 107 to 153 one hour after laevulose, i.e. a rise of 46 mg. per cent. A rise of over 30 mg. per cent is taken as evidence of deficient function of the liver. The liver in this case was unable to cope with the sugar in the blood as rapidly as it should. We notice too that the 2 hour reading is again on the downward trend. This indicates, that although it was not quite normal, the liver still remained active enough to cope with the sugar if allowed time. A similar test was carried out on the day after the operation and the following readings were obtained:

<table>
<thead>
<tr>
<th>Fasting blood sugar 24 hours after operation</th>
<th>113</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Sugar ½ hour after giving 50 grms of laevulose orally</td>
<td>139</td>
</tr>
<tr>
<td>&quot; 1 hour &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; &quot; 2 hours &quot; &quot; &quot; &quot; &quot; &quot;</td>
<td>195</td>
</tr>
<tr>
<td>243</td>
<td></td>
</tr>
</tbody>
</table>

Here again the rise is far in excess of the normal 30. We also notice that the blood sugar level is still going up 2
hours after the laevulose had been given, Rolleston and McNee (17) consider this an important factor in the interpretation of the test. 

WHEREAS BEFORE THE AVERTIN HAD BEEN GIVEN THE BLOOD SUGAR ROSE 46 MG PER CENT AT THE 1 HOUR PERIOD IT ROSE NO LESS THAN 82 MG PER CENT DURING THE SAME PERIOD AFTER THE AVERTIN HAD BEEN GIVEN. WHEREAS, BEFORE THE AVERTIN HAD BEEN GIVEN THE LIVER WAS ABLE TO DEAL WITH THE EXCESSIVE SUGAR IN 2 HOURS IT BECAME QUITE INCAPABLE OF DOING IT AFTER AVERTIN HAD BEEN GIVEN.

This case confirms the experimental evidence, where it was shown that an already damaged liver will be further damaged by avertin. We are inclined to believe that the damage is of a less transitory nature than Widenhorn (18) thinks. He finds that the blood sugar usually returns to normal in 8 to 24 hours. This we cannot confirm. This clinical test, of course, does not tell us what, if any, the pathological changes in the liver are. It merely indicated that the liver has become incapable of completely discharging its physiological duties. From the experimental evidence obtained by others we may, however, infer that parenchymatous and even fatty degeneration had taken place.

What practical significance do we attach to this?

It seems clear that avertin does damage the liver and it

(17) Diseases of the liver p. 70

would be unwise to disregard this. On the other hand, there appears to be very little cause for pessimism. The case of cirrhosis of the liver, just dealt with, had a convalescence as smooth as could be wished. So did the other three with positive laevulose tests. They certainly showed no other evidence of liver insufficiency. We, therefore, conclude that (a) THE SURGEON NEED NOT FEAR THE EFFECTS OF AVERTIN IF THE LIVER IS NORMAL (b) THE DANGER IN A CASE OF SLIGHT HYPO FUNCTION OF THE LIVER IS SMALL AND AVERTIN IS NOT CONTRAINDICATED. (c) WHERE MARKED LIVER DEFICIENCY EXISTS AVERTIN IS CONTRAINDICATED.

(2) SPINAL ANAESTHESIA

All drugs used in the production of Spinal anaesthesia are tissue poisons. Cocain is the most potent and has been discarded. When used in normal dosage and dilution the others show very little, if any, evidence of direct toxic effect on the tissues. This, however, is not a universally accepted fact. Howard Jones (19) for instance, holds that the fall in blood pressure is largely due to the toxic effect on the vaso-motor centre by the drug, which has been absorbed from the spinal canal. (19) Anaesthesia and Analgesia Sept-Oct. 1932.
We shall consider this subject of toxicity first in relation to the blood pressure and later in relation to the liver, the respiratory system and the cord and meninges.

(1) Fall of Blood Pressure. This is in every respect the most serious problem in spinal anaesthesia and its most dreaded complication. The abundant literature on the subject is both confusing and misleading. Each experimenter proves to his own satisfaction that one particular factor is the source of all the mischief. This is a fallacy. The blood pressure is lowered for several reasons and to a combination of these must we attribute the total fall. We shall endeavour to classify the causes in the order they appear to us most rational, and refer briefly to some controversial points.

CAUSES OF FALL OF BLOOD PRESSURE

(a) Paralysis of the vaso-constrictors. Blood pressure varies as the product of cardiac output and the peripheral resistance. Peripheral resistance consists chiefly of the tonically contracted arterioles, principally those of the splanchnic area and the skin. (2). The state of the arterioles is under continuous control of the vaso-motor centre which is situated in the floor of the Fourth Ventricle. The fibres to the splanchnic viscera arise from the lower 7 dorsal and the upper 2 or 3 lumbar segments. If these fibres are paralysed, as happens in spinal anaesthesia, or if the splanchnic

(20) Samson Wright, Applied Physiology pp. 290-291
nerves are severed a considerable proportion of the body's blood is delayed in the abdominal organs, the so-called "splanchnic pool". That this is the case can be demonstrated experimentally by measuring the size of an organ in a plethysmograph.

This paralysis of the vaso-constrictor nerves is almost certainly the main cause of the fall of blood pressure.

(b) Functional severance of the motor nerves to the voluntary muscles, leads to complete flaccidity allowing a large amount of blood to collect in their veins and venules. It has not been proved that sympathetic paralysis dilates the arterioles in the muscles; Wright believes the reverse to be the case.

(c) Paralysis of the intercostal and abdominal wall muscles (21) Gray and Parsons as quoted by Severs and Waters, (22) attribute the "main fall" of blood pressure to paralysis of the intercostal muscles and the consequent diminution of the aspiration action of the thorax, whereby blood is sucked from the abdomen to the heart and paralysis of the abdominal muscles which normally pump the blood out of the abdomen. The diaphragm tries to compensate by larger excursions but does not succeed on account of the flaccidity of the muscles of the

(21) Evans: Spinal Anaesthesia p. 84.
(22) Anaesthesia & Analgesia. March - April 1932.
abdominal wall. After paralysing the intercostal muscles of dogs by spinal anaesthesia, they were able to restore the blood pressure to normal by means of artificial respiration. To exclude the effect of better oxygenation produced by artificial respiration, they controlled their experiments by others, where the dog received a liberal supply of oxygen but no chest movements. In such cases the pressure rose to some extent, but yet remained well below normal.

(d) Anoxaemia. When the majority of the intercostal muscles have been paralysed by a high spinal anaesthesia respiration is chiefly carried on by the diaphragm. With the diaphragm alone operating, lung expansion takes place chiefly in the lower lobes and very little in the upper. This results in inadequate oxygenation of the blood. The pulmonary circulation is already diminished as a result of splanchnic and muscular stagnation of the blood. Thus inadequate oxygenation may become more important still.

This lack of oxygen is termed anoxaemia. The manner by which it brings about a fall of blood pressure is a complicated one. A moderate deficiency of oxygen usually causes a rise of pressure by stimulating the vaso-motor centre. It seems, therefore, contradictory to state that a greater deficiency could cause a fall. The explanation is obtained in the effect of anoxaemia on the respiratory centre. This too is a complicated
chemico-physiological process, but can be simply explained thus: Anoxaemia is a cause of increased fatigue of the centre and shallow breathing, which again accentuates the tendency to anoxaemia. A vicious circle is thus established which, unless broken, inevitably leads to respiratory arrest. (Haldane)

Meanwhile a state of alkalaemia has developed. This results in a less sensitive vaso-motor centre and the blood pressure falls.

(e) **Paralysis of the Cardiac augmentor nerves.** The Augmentor nerve fibres belong to the sympathetic system and arise from the first 4 dorsal segments. Normally they stimulate the heart to greater activity as soon as the blood pressure falls. The vagus (parasympathetic) has the reverse action. It slows the heart. When the accelerating influence of the Augmentor nerves is eliminated or diminished the vagus has free play and inhibits the heart’s action considerably. In high spinal anaesthesia some of, if not all, the augmentor fibres are partially or totally paralysed. The action of the heart is inhibited, the pulse rate falls and does not respond to the falling blood pressure.

(f) **Direct toxic effect on the vital centres.** That the toxicity of the drug is an important, if not the chief cause of the fall of blood pressure, is a view that is gaining popularity just now. Hewer (23) states that percan

(23) Recent advances in Anaesthesia p. 121.
causes a smaller drop in blood pressure than novocain or stovain, because in the concentrations used it is less toxic when absorbed into the blood. Howard Jones (24) claims that drugs used in spinal anaesthesia affect the vasomotor centre, after blood absorption, to such an extent as to lead to "severe collapse to a pulseless state."

It is quite clear that this is a secondary effect altogether, but it is of first rate importance. The sympathetic fibres in the motor roots are amongst the earliest to be affected and the result is soon reflected in a fall of blood pressure after a spinal injection has been made. This is the primary fall. Then the drug is gradually absorbed into the veins and to some extent it accumulates in the blood before being destroyed by the liver. During this period it exerts a depressing effect on the vasomotor centre, and causes a fall in the blood pressure. This is the secondary fall. It is most marked at the time of maximum concentration of the drug in the blood. In the case of stovaine this takes place 15-20 minutes after injection, whereas with percain it takes place about 35 minutes after. The reason is that stovaine is used in concentrated form (5-10%) whereas percain is used in


I am using the terms primary and secondary merely for the sake of lucidity. In practice the fall is a continuous one and no stages can be recognised.
extremely dilute solution (1: 1500). Moreover the fall is less with percairn than with stovaine. Granted that an equal number of spinal roots were paralysed, the primary fall must be equal in the two cases. What difference there is must be due to a difference in the secondary fall.

Occasionally one meets with an individual who is hypersensitive to the drug. In such a case the poisoning symptoms will be acute and resemble anaphylactic shock (25). There is also the possibility of injecting the drug intravenously with serious collapse and even death.

(g) Adrenalin Deficiency. Adrenalin is a product of the medulla of the suprarenal gland. It is carried into circulation by way of the adrenal veins. Its action is to reinforce the activity of the sympathetic nervous system during conditions of stress (26). Its secretion is controlled by the splanchnic nerves. During times of stress, such as a marked fall in arterial blood pressure, the suprarenal takes on "emergency functions", constricts the arterioles chiefly in the splanchnic area and the skin, increases the peripheral resistance and raises the blood pressure. It also redistributes the blood directing the main flow to such important organs as the brain, heart and lungs. The stimulus for such emergency function is the fall of pressure itself. It is possible that this starts in the aorta and in the carotid sinus. The impulse to secrete is conveyed along the splanchnic nerve. (As far as we know there

(26) Samson Wright: Applied Physiology p. 130
is no hormonal influence.

In high spinal anaesthesia this secretory nerve is paralysed. The demand for adrenalin is present, but the gland remains ignorant of the fact; consequently it does not respond.

Although we have no experimental proof that this is the case in spinal anaesthesia, we feel justified on theoretical grounds to regard adrenalaemia as a factor in the fall of blood pressure. Perhaps it is more correct to say that adrenalaemia is an important factor in preventing the blood pressure from rising after the initial fall. Clinical evidence strongly supports this conclusion.

IS LOW BLOOD PRESSURE IN SPINAL ANAESTHESIA IDENTICAL TO THE LOW BLOOD PRESSURE OF SHOCK?

Crile (27) states that "surgical shock is an exhaustion of the vaso-motor centre. Neither the heart muscle, nor the cardiac centres, nor the respiratory centre, are other than secondarily involved. Collapse is due to a suspension of the function of the cardiac or of the vaso-motor mechanism or to haemorrhage. In shock cardiac stimulants have but a limited range of possible usefulness, and may be injurious. In collapse stimulants may be useful because the centres are not exhausted."

The low blood pressure of spinal anaesthesia is in kind similar to that of collapse and may also, in cases of disaster, be similar in degree. On the other hand, there are distinct pathological and clinical differences between low blood pressure of spinal anaesthesia and low blood pressure of shock. The following are some of the more important:

**SHOCK**

(a) In severe cases of shock, vasomotor centre is exhausted. Brain cells are damaged. There is little or no response to artificial raising of the blood pressure or to the administration of oxygen.

(b) The capillaries are dilated and more permeable. The blood is more concentrated.

(c) Recovery is slow. The patient is restless and apathetic. This may be due to brain cell changes and severe metabolic disturbances.

**SPINAL ANAESTHESIA**

(a) In ordinary cases of spinal anaesthesia the vasomotor centre is altogether unaffected. In "dangerous" cases the activity of the centre is diminished. In most cases this is due to anoxaemia (rarely toxicity of the drug). The centre will respond at once on increasing the supply of oxygen (28) or artificially raising the blood pressure.

(b) The arterioles are dilated. Blood is not concentrated. This is important because the arterioles will respond to adrenalin.

(c) Recovery is rapid as the effect of the anaesthetic on the sympathetic nerves wears off. There is no apathy. The patient will often declare himself to feel almost normal after a considerable fall of blood pressure.

The following two cases illustrate these differences.

The first, a middle-aged male was operated on by Mr. H. Stewart at Bradford, March 1932, for duodenal ulcer. Gastro-enterostomy was performed under general anaesthesia. This was followed by "vicious circle" vomiting. Some days later it was decided to do another laparatomy, this time under spinal anaesthesia, for fear of chest complications. This being one of the "opposition firms" in the hospital I was very anxious to procure an entirely successful anaesthesia. The usual dose of stovain was given, but the head of the table was tilted down slightly more than usual. The patient also had avertin. Perfect anaesthesia and relaxation were obtained, and retained, during the whole of a long and difficult duodeno-jejuno-stomy. It, however, soon became evident that we had obtained far more anaesthesia than was necessary. Practically all the intercostal muscles were paralysed and very shallow breathing resulted.

In addition the blood pressure fell so low that the pulse became imperceptible at the wrist. The patient's colour remained fair, the skin remained warm and there was very little sweating. We were alarmed, but not unduly so, knowing that the condition was not one of shock, and that the patient's condition would improve as soon as the effect of the stovain on the spinal nerve roots began to wear off. Towards the end of the operation a strong pulse had returned at the wrist.
After the effects of the avertin had worn off the patient not only declared that he felt very well, but his condition was just the reverse of what it would have been, had shock been the cause of his low blood pressure.

It is quite evident that we were dealing with a case of what might be described as uncomplicated low blood pressure. There was no suspicion of vaso-motor exhaustion as in shock. The vaso-motor centre was undamaged and entirely alive, it merely had to await a larger supply of blood and better oxygenation to assume full control of its duties once more. We disagree with Pitkin (29) when he describes such a state as one of "profound shock."

Frequently as Normal Lake (30) says the "fall in blood pressure is more alarming than serious." We wish however to emphasize that our attitude towards this state of low blood pressure is not one of complacent tolerance. It is a state that should be avoided by all possible means, and when encountered, should receive the most prompt and skilful treatment. Although we deny it to be shock, in the first instance, we are only too well aware that if allowed to persist, it will, by deficient blood supply, bring about a pathological change in the brain cells in no way distinguishable from that produced by shock.

(30) Centenary Meeting B.M.A. 1932.
The second case was a man of 56 years we treated at Tilbury. He had been quite well until 10 hours before admission, when he was suddenly overpowered by acute abdominal pain, while feeding his pigs early in the morning. A doctor was called in the afternoon and diagnosed appendicitis. When we saw him, he had an ominous cyanosed appearance about the mucous membranes suggesting an overpowering toxaemia, his eyes were sunken, he was apathetic and did not complain of much pain. Pulse was fast, but the pressure still fair. On examining the abdomen there was board-like rigidity of both recti below and above the umbilicus, with maximum intensity in the right iliac region, where also tenderness appeared to be most marked. We diagnosed perforated peptic ulcer. In spite of the patient’s shock state, we decided that spinal anaesthesia would probably be less dangerous than general. Laparotomy revealed a perforation of a diverticulum of the pelvic colon. The abdomen was full of faeces. There was acute general peritonitis.

During the operation the blood pressure did not drop very much, neither did the pulse rate rise appreciably. Yet the recovery of pressure, that one is accustomed to look for towards the termination of and after the operation never set in. Pressure gradually dropped and pulse rate gradually increased until the patient died the same night. He died of shock. This case illustrates the low blood pressure
of shock.

The vaso-motor mechanism was exhausted by the shock produced by perforation of the bowel and toxaemia. The additional lowering of the blood pressure by the spinal anaesthesia probably increased the shock.

No doubt this was an extreme case. He would probably have died, even had only local anaesthesia been used. But he serves as a warning against taxing an already over-powered vaso-motor centre by an additional lowering of the blood pressure. At the same time every detail of the case is in line with our argument that the low blood pressure of spinal anaesthesia is essentially different from the low blood pressure of shock.

11. Effects on the Liver.

We became interested in the liver, not because we believed that drugs used in spinal anaesthesia would directly damage its tissues to any appreciable extent, but because we considered the liver to be a convenient organ, whereby we could measure the effects of shock on account of the destructive changes seen in its cells. We argued that, if spinal anaesthesia damaged the liver either directly, or through a fall of blood pressure or through failure in protecting the patient from shock in any way, we should be able to obtain evidence of this by means of a functional test. We were
hoping to repeat the test in cases of ether anaesthesia, but were unable to do so on account of the almost inevitable vomiting that follows the administration of 50 grms of laevulose in such cases.

The method we adopted was as follows: On the day before the operation a normal fasting blood sugar reading was made. This was followed by 50 grms of laevulose and half a pint of water by mouth. Blood samples were again obtained ½ hour, 1 hour and 2 hours later, and sugar estimations made by a modified Calvert's method. The whole procedure was repeated on the day after the operation. We thus obtained (a) Fasting blood sugar reading before and after the operation.

(b) a liver function test before and after the operation.

On account of vomiting and technical errors some of the readings had to be discarded and one has not had another opportunity of adding to this small number of cases.

Of the 19 'spinal' cases whose blood sugar we estimated we found that there was a post-operative rise in 17 and a fall in 2 cases. The average was a rise of 24.2 mg. per cent. From this we concluded that, to a certain degree, there was impairment of sugar metabolism after spinal anaesthesia. This impairment appeared less marked in cases where pure spinal anaesthesia was performed, (18.6 mg. per cent) than in cases where spinal anaesthesia was combined with avertin, (33 mg. per cent) We know that even after local anaesthesia
there is a rise in blood sugar (31)

The question arises whether this was a purely functional phenomenon or whether we could produce any clinical evidence to show that spinal anaesthesia damaged the liver (directly or indirectly). The clinical evidence that we have taken as our standard, is the same, as we adopted in our discussion on the effect of avertin on the liver, namely, a rise of over 30 mg per cent in the blood sugar after administration of 50 gm. of laevulose. By using laevulose (and not glucose) changes in the pancreas and muscles which may influence blood sugar are eliminated. As far as is known laevulose is metabolised by the liver only. (32).

Our figures showed that the highest level of blood sugar was almost invariably reached, 1 hour after giving the carbohydrate. To simplify matters we have, therefore, disregarded the ½ hour and the 2 hours' readings and merely took into account the 1 hour figure.

The result of this investigation was to show that out of 18 cases there was evidence of post-operative liver damage in 7. As this figure seems extraordinarily high, we thought it necessary to examine all 7 cases more closely. On going over our records we found that in every case there was present

(32) Rolleston and McNee. Diseases of the Liver p. 71
some factor which might account for the positive test, i.e. the liver damage. For convenience we group the cases into:

(1) Those that had shown liver hypofunction, as evidenced by an exactly similar test, before the operation, and in addition received avertin.

(2) Those that showed no pre-operative evidence of liver damage, but received avertin

(3) Those that received no avertin, but showed evidence of liver damage before the operation.

In the accompanying table we call positive (or pos.) those that gave evidence of liver damage, that is, had a rise in blood sugar of 30 - mg. per cent after 50 gm of laevulose.

<table>
<thead>
<tr>
<th>Case</th>
<th>Pos. before operation</th>
<th>neg. before op.</th>
<th>pos. before op.</th>
</tr>
</thead>
<tbody>
<tr>
<td>E.E.</td>
<td>(pre. op. rise 44)</td>
<td>(pre. op. 55)</td>
<td>(pre. op. rise 55)</td>
</tr>
<tr>
<td>H.M.</td>
<td>(pre. op. rise 47)</td>
<td>(pre. op. 56)</td>
<td>(pre. op. rise 56)</td>
</tr>
<tr>
<td>G.W.</td>
<td>(pre. op. rise 42)</td>
<td>(pre. op. 56)</td>
<td>(pre. op. rise 56)</td>
</tr>
<tr>
<td>E.T.</td>
<td>(pre. op. rise 44)</td>
<td>(pre. op. 55)</td>
<td>(pre. op. rise 55)</td>
</tr>
</tbody>
</table>

It is clear from this table that in each case, where our test gave evidence of liver damage, following spinal
anaesthesia, an additional factor had to be reckoned with. Five of the cases (groups I and III) gave positive tests before the operation. We were, therefore, not surprised to find them positive again after the operation. This leaves us with 2 cases (group II) out of 18. These two apparently had normal livers before the operation and pathological livers after. They both received avertin. We have previously fully discussed the possibility of avertin causing demonstrable liver damage. We have no clear proof that the avertin was the responsible factor in these two cases, but it certainly appears to be a reasonable supposition. At any rate, we have no proof either that the spinal anaesthesia was responsible for the damage.

(I wish here to draw attention to a point which has no bearing on the thesis, but which confirms the conclusion of Mekie (33) and other observers. They state that there is no physiological foundation for the common practice of giving glucose to a patient suffering from surgical shock, because there was already more sugar in the blood than the liver could metabolise. Their observations were made after general anaesthesia, where there was always a high sugar content. From my observations in spinal anaesthesia it appears that there was a rise in 17 out of 19 cases. It seems, therefore, that

also in spinal anaesthesia there is no call for post-operative glucose medication.)

The clinical significance of liver damage during anaesthesia is obvious and need not hold our attention for long. It probably matters little in the end, even though it adds to morbidity and discomfort, whether the anaesthetic causes a certain amount of parenchymatous degeneration in the liver of the strong and more or less normal patient, undergoing an operation for some chronic non-infective complaint. But we have to consider whether impaired function of this organ does not become a matter demanding our serious attention in cases whose livers are being over-powered by toxins. Is the additional burden we put on such a liver, by administering a destructive anaesthetic, not more than once the very means of turning the scale to the wrong side. The liver plays a most important part in the metabolism of protein, carbohydrate and fat and in the destruction of bacterial and other toxins circulating in the blood. If it has already been injured by infection or disease the addition of a toxic anaesthetic will add to the damage and result in diminution of the alkali reserve and an increase of acidosis, owing to an insufficiency of the liver tissue. Without abundant experimental confirmation, one cannot be dogmatic about the question as to whether the anaesthetic is to be blamed as the cause of death in these cases. But one
suggests that many cases of acute abdominal infections die, not on account of the infection, but on account of a failure of metabolism. This is the result of destroying too many cells of an organ already severely damaged. Every surgeon meets with the case of acute appendicitis with peritonitis which dies on the 3rd, 4th or 5th day, where on autopsy it is difficult to explain exactly why the patient should have died. In fact, there appears to be no reason why the patient should not have overcome the infection. Death may be ascribed to shock or to peritonitis. Is a failing liver not responsible for some of these deaths? Normal metabolic function and a rapid destruction of the products of infection may have averted the disaster.

III. Effects on the meninges, cord, nerve roots and cranial nerves due to irritation and trauma.

We are at the moment considering the toxic effects of drugs used in Spinal Anaesthesia. It is convenient, however, to deal with traumatic lesions at the same time as their effects may be very similar.

**Causes of Nerve and Meningeal Lesions.**

(a) Injury to the cord or nerve roots by the needle or the injection.
(b) Pressure of a haematoma on a nerve root.
(c) Irritation of the drug
(d) Infection.
Types of Lesion.

1. **Headache** has in the past been an annoying complication of spinal anaesthesia. With better understanding of its causes it has become less frequent and its treatment has become more efficient. Headache after spinal anaesthesia is not necessarily due to the anaesthesia. Mild forms may occur after any type of anaesthesia. Such headaches as a rule disappear quickly after an analgesic tablet. This type of headache occurred in over 20 per cent of our patients. There remain, however, two types which are definitely due to the spinal anaesthesia. They may be termed Low Pressure Headache and High Pressure Headache, respectively.

**Low Pressure Headache** is due to leaking of cerebrospinal fluid into the tissues. It may occur after any lumbar puncture whether fluid is injected or not. It is worse on raising the head and improves on lowering the head. It is not accompanied by irritative signs such as photophobia and a stiff neck. The introduction of the small bore needle has very largely eliminated this type of headache. By keeping the patient's head low for 24 hours after the operation a certain number will be avoided. If encountered the best treatment is to raise the foot of the bed and to give analgesics. Hypotonic saline intravenously is said to cure persistent cases.

**High Pressure Headache** is due to meningeal irritation usually aseptic, rarely bacterial. The spinal meninges are
injected and the cerebro-spinal fluid is increased in amount and cellular content.

This type of headache usually starts on the day after operation, sometimes as late as the third day, and may last over a week. It is severe, is not improved by lowering the head and is accompanied by signs of irritation such as photophobia, stiff neck and even a slight Kernig's sign.

The cause of the non-infective irritation is undoubtedly the drugs used in spinal anaesthesia. Experimenting on dogs Davis and his associates (34) found that all drugs used for this purpose caused a varying degree of inflammatory reaction in the leptomeninges.

Treatment consists in keeping the patient quiet and administering analgesics. If this does not bring relief hypertonic saline or glucose intravenously must be given. In one case we had to resort to hypertonic saline. It brought immediate relief. Lumbar puncture has the same effect. We have seen this type of headache on three occasions. In one case the pain was localised in the neck.

Meningitis may be

(a) non-infective or irritative and
(b) purulent

Little more need be added after what has been discussed under headache.

Purulent meningitis is seldom due to anything but careless technique and should not be encountered. A blood born infection could take place, but must surely be exceedingly rare.

The treatment consists in frequent lumbar puncture.

3. Abducens Nerve Palsy.

From the practical point of view far too much stress has been laid on this rare complication. It causes some inconvenience but always clears up spontaneously. Babcock has not seen it in 15000 consecutive cases of spinal anaesthesia. The cause is by no means clear. The sixth nerve has a long intracranial course and is the commonest cranial nerve to be affected in any form of meningitis. It is possible that the lesion is due to a basal meningitis. Irritative signs sometimes accompany it. The condition may be bi-lateral. Whether the meningitis is infective or not is difficult to determine.

Babcock (35) states definitely that the condition is due to an infection which has an incubation period of one week. It usually clears up by the fourth week, but may last six or eight weeks. We have not seen the condition.

Urinary Incontinence, parathesias, weakness of muscle groups, Trophic changes.

These are sequelae we occasionally meet with. They are of extreme importance, especially urinary incontinence. Probably

most of these changes have no connection with the spinal anaesthesia. Unfortunately we seldom examine our patients preliminarily from this point of view and, therefore, may involve ourselves in court proceedings. Amongst our patients there are two who developed incontinence. In neither case was this due to the anaesthesia. We deal with them in the next section. It is not always very easy to decide whether a change such as this is functional or organic. If organic any of the four causes which we have mentioned earlier may be responsible.

Injury of a nerve root by the needle is probably responsible for some cases of parathesias. The condition always clears up, but may last several months. At the time of puncture the patient will feel a sharp pain down his limb. In the two cases we have seen, no evidence of organic nerve lesion could be obtained. They are reported on in the next section.

A group of muscles may be similarly affected.

We have come across patients complaining of weakness of the legs. No organic lesion can be discovered. It is possible that in such people the cauda equina was temporarily compressed by an extra-dural haematoma. It seems reasonable that such damage may also be the cause of urinary incontinence. Carefulness and practice will minimise the risk; some cases will be unavoidable. Such patients, however, always recover. There remains nevertheless a type of patient who will not recover
completely. This is due to damage of the cord. Under the next heading we point out how the careful surgeon need not fear ever to come across such a case.

Can irritation of the drug cause degenerative changes in the cord or nerves? Davis and his collaborators, to whose work we have already referred, noticed the following changes in dogs:

(a) Passive changes in the ganglion cells of the cord similar to those seen in retrograde or so-called Wallerian degeneration.

(b) Swelling and fragmentation of the axis cylinders.

(c) Signs of degenerative changes in the fibre tracts of the cord.

But on killing their animals 90 days later these changes had entirely or almost entirely disappeared. This work is of extreme importance and should dispel any fears of a permanent toxic degeneration we may have had. There is also the authoritative clinical evidence of Babcock (36) after an experience of more than 20,000 operations under spinal anaesthesia. His words are: "May spinal degeneration and paralysis follow the intradural injection? I doubt the existence of medullary paralysis from the proper use of spinal anaesthesia. One of our patients had eleven inductions

of spinal anaesthesia, without evidence of injury to the cord."

(5) **Paraplegia.** Most surgeons, if not all, would rather see their patient die on the table than face him paralytic afterwards. Such cases are not unknown. If due to spinal anaesthesia at all, only extreme carelessness could have been the cause. Damage to the cord is the responsible lesion. Mere puncture of the cord is said to do no harm, but even for this there is no need, one must keep below the level of the cord. The lesion is caused by forcibly injecting the anaesthetic into the cord and disrupting it. We must be careful not to miss cases of meningeal lesions, tabes, transverse myelitis, secondary carcinoma of the vertebrae and other rarer lesions. Should we be unfortunate enough to meet a case of paraplegia following spinal anaesthesia.

IV. **Effect on the respiratory System**

1. **Respiratory embarrasment** may be produced in the following ways:

   (a) Reflex inhibition of respiration as a result of the lumbar puncture.

   (b) By paralysis of the abdominal and intercostal muscles

   (c) By paralysis of the phrenic nerves. This can only occur if anaesthesia extends beyond the 5th cervical segment.
(d) By interference with the respiratory centre. It used to be taught that this effect was produced directly by the drug rising to and anaesthetising the medulla. This view has been criticised by Johnson and Henderson (37) who were unable to paralyse the respiratory centre by direct application of the drug. They injected the drug into the 4th ventricle, in the floor of which the centre is located, without influencing it in the least. By using coloured fluids and noticing the staining afterwards, they were able to prove that the drug was actually injected into the ventricle. Cotui and Standard (38) on the other hand, as a result of similar experiments, were of opinion that there was direct action of the drug on the centre, after it had reached the 4th ventricle through the foramina of Luschka and Magendi.

Howard Jones claims that the centre becomes poisoned by the drug present in the circulation. His arguments are similar to those used in case of the vaso-motor centre, with which we have dealt. We have previously pointed out how poor oxygenation which normally stimulates the centre, may under certain conditions depress it and lead to total failure of respiration.

2. Pulmonary Complications. Whereas the above refers to the immediate and more or less direct effects of spinal

anaesthesia on the respiratory system, our consideration has also to take into account the more remote effects. It was hoped by the introduction of spinal anaesthesia to eliminate pulmonary complications altogether. This did not materialise. Pulmonary complications are seen after spinal anaesthesia and even after local anaesthesia. Some claim a great advantage for spinal anaesthesia in this respect. Others state it makes no difference to the incidence of pulmonary complications. In order to be of value, it is necessary to consider cases operated on under exactly similar conditions. The most reliable recent report is that of Sise (39). His figures are 1008 spinal and 973 ether. He reports from a clinic where for a certain time ether used to be the routine anaesthetic, where as later spinal became the routine. These cases were operated on by the same surgeons and postoperative treatment remained similar. Sise found that in the ether cases pulmonary complications amounted to 6.5 per cent and in the spinal cases 3.35 per cent. On analysis it appeared that pulmonary complications had actually increased, if slightly, in lower abdominal operations under spinal anaesthesia, namely from 2.3 per cent to 2.5 per cent. In upper abdominal operations, however, there was a definite improvement in the incidence of pulmonary complications. With

ether anaesthesia the figure was 10.7 per cent and with spinal anaesthesia 4.2 per cent.

Pulmonary complications were found in 7, i.e. 3.8% of our 182 cases of spinal anaesthesia, with or without avertin. All occurred in upper abdominal operations.

V. SIMPLICITY OF TECHNIQUE

Neither spinal anaesthesia nor avertin amnesia require any elaborate outfit. The technique is simple and easily acquired. If the surgeon administers the spinal himself a few minutes are lost, but these are made good by the smooth, uninterrupted anaesthesia and absolute relaxation.

Simplicity of administration should not infer inefficient supervision. If spinal patients were half as carefully watched as ether ones, many fatalities would be avoided. The patient must be attended to by a competent anaesthetist, who knows the signs of vaso-motor and respiratory failure and of shock, one who is capable to deal with such contingencies promptly and efficiently.

VI & VII. PLEASANT INDUCTION AND PLEASANT RECOVERY

In this respect it will be difficult to improve on the action of avertin. We found it specially useful in children, nervous women and alcoholics. The patient falls into a
a pleasant sleep, even before the rectal injection has been completely given. Unconsciousness lasts several hours after operation. During this time the patient appears somewhat restless and may require more attention than after ether. Heroin may be given to the ordinary patient without much fear of the chest.

Vomiting is seen much less frequently than after ether. One seldom sees severe and repeated vomiting.

In cases where avertin or some other basal anaesthetic is contraindicated, and we wish to give a spinal anaesthetic, the mental factor becomes a serious consideration. The patient may be so nervous as to make a spinal anaesthetic almost impossible. Our practice in such cases is to induce with ether and then give the spinal anaesthetic. We generally find that it is not necessary to carry on with the ether at all once the spinal has been given, or it may be necessary merely to pour a few drops on the mask now and then. In all not more than 1½ - 2 ozs will be used, whereas, without spinal anaesthesia 12 - 16 ozs may have been required.
SECTION II.

CASE REPORTS
Explanatory.

In this section we report on the cases we dealt with. For the purpose of the investigation we drew up a special spinal anaesthetic scheme. Our report will closely follow the lines of this scheme. We shall briefly describe the methods of anaesthesia employed and the drugs used. Our observations on liver functions have been so completely reported on in the previous section, that we propose not to deal with the liver in this section again.

As our number of cases is so small, we have considered it helpful to make use of some additional material under two headings, namely, mortality and blood pressure. The cases are all from the same clinic. For mortality consideration they consist of 536 cases operated on by Mr Basil Hughes under spinal anaesthesia. For blood pressure consideration Dr. R. Hanson, the honorary anaesthetist to Mr Hughes' unit at the Bradford Royal Infirmary, has kindly supplied me with blood pressure records of an additional number of cases.

Our report, therefore, deals with the following cases: 128 operated on mostly by Mr. Basil Hughes at Bradford Royal
Infirmary. I assisted at all the operations and gave the anaesthetic for some.

54 at the Seaman's Hospital, Tilbury, most of them being both anaesthetised and operated on by myself.

This makes a total of 182 for full report.

74 at Bradford for blood pressure considerations.

536 at Bradford for mortality considerations.

Through the contingencies of the operating theatre and the ward, every detail was not recorded in all cases. We stipulate, therefore, where necessary, under the different headings in how many cases any particular observation had been made.

APPARATUS

Barker's and Pitkin's needles have been used. The Pitkin needle has a smaller bore (20-22 gauge) and a shorter bevel (45°) than Barker's needle. Three advantages accrue from these features.

(a) The small puncture made in the meninges prevents the escape of the anaesthetic drug on withdrawal of the needle. In the days when large bore needles were in use such escape was a frequent cause of unsatisfactory anaesthesia. It also prevents seepage of cerebro-spinal fluid which is recognised as one of the causes of headache after spinal anaesthesia.
(b) The shorter bevel prevents any of the anaesthetic fluid being injected into the tissues, an accident which sometimes happens, when only part of a long bevel has penetrated the arachnoid mater.

(c) The short bevelled needle, being relatively blunt, gives a very obvious sensation to the finger on piercing the dura mater. Not only can its passage through the dura be felt, it can be heard.

This last we consider as the most important advantage. One knows exactly where the needle point is and need have no fear of damaging the nerve roots. In order to save the point of the needle and to simplify penetration we found it useful first to pierce the skin over the site of puncture with an ordinary cutting needle.

With a Barker's needle a record syringe is used, (3 cc. for stovaine and spinocain and 20 cc. for percaine) with Pitkin's a Luer-Lock syringe. The latter is fitted with a screw catch which grasps the needle and prevents it from becoming detached from the syringe.

**HYDROSTATICS AND SPINAL ANAESTHESIA**

Before discussing the individual drug it is useful to remind ourselves of how the physical quality of a drug may influence us in adopting a particular method of administration.

The specific gravity of the cerebro-spinal fluid is 1.005 - 1.008.
Solutions used in spinal anaesthesia are either hyperbaric, iso-baric or hypo-baric as their sp. gr. is more than equal to or less than that of the cerebro-spinal fluid. The specific gravity of the solution will, to a certain extent, determine its mode of administration.

The following methods are used or have been used.

1. Direct. An iso-baric solution is injected opposite the nerve roots that are to be anaesthetised. This method is dangerous and has been discarded.

2. Gravitational diffusion. An hypo-baric solution will rise; an hyper-baric one will sink in cerebro-spinal fluid. The level of anaesthesia is thus determined to a certain extent by tilting the anaesthetic table up - or downwards as the case may be. Other factors, however, come into play. One has to reckon with the curvatures of the spine and with the tendency of solutions of different specific gravity to equalise their specific gravities when mixed. There is a fair difference between the specific gravity of cerebro-spinal fluid and that of the hyper-baric solutions in use. Such solutions will, therefore, diffuse towards the lowest point of the canal to a considerable extent. The hypo-baric solutions in common use are relatively only slightly "lighter" than cerebro-spinal fluid. They, therefore, do not diffuse so readily towards the highest point of the spinal canal. The difference in quantity between the cerebro-spinal
fluid and the anaesthetic solution will also determine the rate of diffusion.

3. Barbotage or mixing the dose in the syringe with spinal fluid and the repeated withdrawal and re-injection of fluid. This is usually completed by gravitational diffusion.

4. Filling the lower part of the subarachnoid space with a large quantity of the anaesthetic solution and displacing the cerebrospinal fluid to a point above the required level of anaesthesia (Howard Jones)

5. Displacing the cerebrospinal fluid from the caudal end of the canal by means of oxygen, then injecting an hypobaric solution which will float on the remaining column of cerebrospinal fluid (Kirschner's Spinal Zone Anaesthesia). To achieve this result the patient must not be moved from the Trendelenburg position. Anaesthesia is obtained in the zone supplied by those nerve roots which are placed between the oxygen and the cerebrospinal fluid and bathed in the anaesthetic solution.

We have used methods (2) and (3) only.

Drugs, preliminary medication, treatment, technique: Stovaine (Benzol-ethyl-dimethyl-aminopropinol hydrochloride) Ampoules contain 5% stovain, i.e. 1 grm. and 5% glucose in 2 ccs. This was used at Bradford. At Tilbury 10% Stovaine in saline was used. The specific gravity of either is more than that of the cerebrospinal fluid.
The patient is examined on the day previous to the operation. If the blood pressure is less than 110 m.m. of mercury Ephedrin gr. ½ is given subcutaneously 20 minutes before operation. If the blood pressure is more than 110 ephedrin gr ½ is given at the same interval.

**Morphia gr ½** is given to men hypodermically and gr 1/6 to women half an hour before the spinal is due to be administered. This is usually avoided when avertin is used.

Before the patient leaves the ward his legs are bandaged with elastic bandages, the legs being elevated before the application is begun at the feet. This is an attempt to minimise the fall of blood pressure due to stagnation in the muscles and veins of the lower limbs.

**Position.** The patient lies on the anaesthetic table, which must be flat, with an adjustable head piece, in the left (or right) lateral position. The legs are drawn up towards the chest and the head bent forward, thus opening up the intervertebral spaces and making the puncture easy. The sitting posture makes the puncture easier still ensuring both wide interspaces and a straight spine. We deprecate this, because we believe that it leads to additional cerebral anaemia, especially in those patients who suffer from "lumbar puncture shock". With due attention to the position of the shoulders and hips there will be no rotation of the spine in the lateral position. The skin of the back is prepared in the ward at the same time as the site for operation.
Anaesthetising the skin is done with ½ or 1½ solution of novocain with adrenalin. An intradermal wheal is first raised, using the finest of hypodermic needles. Following this the subcutaneous tissues and the interspinous ligament are anaesthetised. We pay special attention to this detail, because lumbar puncture without proper anaesthesia, is a very unpleasant experience for the patient, when not under avertin. Our experience is that a great deal of lay hostility to spinal anaesthesia has its origin in painful lumbar punctures. Half a dozen insertions of a spinal needle, through an unanaesthetised back, to be followed by failure and general anaesthesia, are sufficient to make anybody lose faith in this cruel method of allaying pain. By adopting the above procedure we find that our lumbar punctures are absolutely painless. We attribute the complete absence of lumbar puncture shock in our cases to proper local anaesthesia and to the fact that we invariably warn our patient before inserting the hypodermic needle. Moreover, when there is good anaesthesia the patient relaxes his sacrospinalis muscles. This allows of proper dorsal arching of the spine and ensures an easy puncture.

We give a local anaesthetic even in cases under the influence of avertin, because the sense of pain is not abolished with the basal anaesthetic dose of avertin.

It may seem superfluous to expand on such a detail, but in spinal anaesthesia, as in so many other things,
the strictest attention to detail comes second only to the proper knowledge and application of important principles in deciding between success and failure. Also our depressing experiences in different English and continental clinics prompt us to emphasize this point.

Blood is often seen to well from the spinal needle. Its significance is seldom appreciated, judging by the very inadequate reference to it in textbooks on anaesthesia. In order to get a clear conception of the origin of this blood, we have to remind ourselves of the venous drainage of the parts involved.

(a) The external vertebral venous plexuses (anterior and posterior (40) drain the external parts of the vertebrae and the surrounding soft tissues. They communicate freely with the internal plexuses. Two parallel branches of the posterior plexus proceed dorsally, one along each side of every vertebral spine. As the needle passes through the centre of the fibrous interspinous ligament, there is very little chance of puncturing these.

(b) The internal vertebral venous plexuses (anterior and posterior) lie external to the dura mater. Each consists of two parallel longitudinal veins joined by communicating branches. There is also free communication between the two plexuses. The veins of the anterior plexus are the larger and

(4) Gray’s Anatomy p. 684
Quain’s Anatomy vol III. pt. I. pp. 63 and 335
they receive large branches - basi-vertebral veins - from the posterior surface of the bodies of the vertebrae. The basi-vertebral veins remain permanently open like the diploic veins.

(c) Some of the veins of the piamater are continued down between the layers of the pia which covers the filum terminale.

When the needle is inserted the first likely vein from which blood may be drawn is a communicating branch of the posterior internal plexus. This blood is blue and it appears before any cerebro-spinal fluid is seen. All that is required is to push the needle boldly forward. It will pierce the dura and bleeding will cease. A few drops of blood remaining in the needle will tinge the first flow of cere-spinal fluid. As soon as the fluid runs clear the injection is made.

It is extremely unlikely that the veins of the piamater will suffer damage. They are very small opposite the site of puncture and will slip away before the needle point.

If the needle is pushed right through the canal its point may enter one of the veins of the anterior internal plexus. Using a long bevelled needle and not feeling it slip through the dura, this may possibly happen. There follows a free flow of dark venous blood. If the anaesthetic drug is now injected it will be an intravenous injection and immediate collapse and probably death will follow. On withdrawing the
Vertebral body and venous plexuses (modified after Gray) with the spinal needle in a "dangerous" position. The point and part of the bevel have entered a communicating branch of the Anterior Internal Plexus of veins, while part of the bevel has remained in the subarachnoid space. A mixture of blood and cerebro-spinal fluid escapes under such circumstances.
needle slightly blood tinged cerebrospinal fluid will again be obtained.

There is another possibility. The books entirely disregard this. When giving an intravenous injection into the forearm everyone must at times have noticed that the drug would freely flow into the vein, and at the same time, form a small collection under the skin. The explanation is obvious. A long bevelled needle was used, the point and part of the bevel were pushed into the vein while part of the bevel remained subcutaneous. Some of the drug will therefore, escape under the skin. Similarly the opening of the spinal needle may be partly in the vein and partly in the subarachnoid cavity. The pressure of the cerebrospinal fluid is more or less equal to that of the venous blood. A mixture of blood and fluid will be obtained. Can we recognize this? The cerebro-spinal fluid will not be tinged as we have previously seen and the colour of the flow will not be dark like that of venous blood. There will be a flow of a red mixture, resembling very watery arterial blood. We could not have punctured any arteries. The only explanation is that we have a mixture of venous blood and cerebro-spinal fluid. The intensity of the colour will depend on the proportion of blood in the mixture. Should an injection be made under these circumstances, part of the drug will enter the vein and again the consequences may be disastrous. That this is not merely a theoretical possibility, will be confirmed
by any who has had experience of lumbar punctures.

The practical point is never to make an injection until there is a clear flow of cerebro-spinal fluid.

The site of puncture varies to some extent with the nature of the operation. For upper abdominal operations, the space between L2 and L3 is generally selected. Immediately the fluid is injected the patient is turned on his back. The head and knees are raised, so that diffusion of the agent may be limited to the required section of the cord. In the case of an upper abdominal operation, e.g. gastro-enterostomy, one pillow is placed under the buttocks to aid the upward diffusion of the heavy stovain. When an adjustable table is used slight downward sloping towards the dorsal spine ensures a similar effect. The head and shoulders are always tilted upwards. The patient is kept in this position for five minutes, when anaesthesia is complete and, as the drug is then fixed, (some people prefer to wait 10 minutes) he may be placed in any position desired.

On return to the ward it is best to keep the patient flat for 24 hours, or even raise the foot of the bed. This counteracts cerebral anaemia and the low pressure type of headache. Where Fowler's position is desirable, it is better to wait 1½ to 2 hours after the operation. By this time blood pressure is well restored, and syncope need not be feared. Fluids by mouth are immediately allowed, if not contra-indicated by the type of operation. A desire for solid food
and the newspaper is not infrequently expressed by patients on return to the ward.

SPINOCaina. Solution A to be used for local infiltration of the skin contains:

- Ephedrin hydrochloride gr \( \frac{1}{4} \)
- Novocain 1% ad cc. i.

Solution B for spinal injection contains:

- Novocain 200 mgs
- Strych. Sulph. 2.2 mgs
- Alcohol 14.5%
- Gliadin
- Distilled water to 2 cc

The specific gravity is less than that of cerebro-spinal fluid.

The preliminary medication, treatment and technique are the same as for stovaine, with the following exceptions:

(a) Bandages are not required as the fall of blood pressure is not so great

(b) No preliminary ephedrin is given on the ward. This is given in solution A.

(c) As soon as the injection has been made, the patient is turned over on to his face, with the head anteflexed at a lower level than the body. This position is maintained for 2 or 3 minutes during which time the posterior roots are soaked. If the operation is to be a unilateral one, e.g. for inguinal hernia, we keep the patient in the lateral position. Anaesthesia is found to be more intense on the uppermost side. He is then turned on to his
back for a further 5 to 8 minutes, with head still lower than the body, when anaesthesia is complete. The lowered position of the head limits upwards diffusion. Turning the patient on to his face first ensures a higher and more intense anaesthesia.

We have not used a tiltometer as recommended by Pitkin. Only in 3 cases have 3 cc of spinocain for higher abdominal incisions been given. The results were less satisfactory than with Stovaine.

Percaine (sold in South Africa as Nupercaine) is the hydrochloride of a butyloxycinchoninic acid diethylethylenediamide. It is a derivative of quinoline and does not belong to the cocaine group. It possesses two outstanding advantages over other analgesic drugs:

1. It is extremely potent being 20 times more effective than novocaine. On this account it can be used in very dilute solution whereby toxicity is enormously diminished.

2. Its effect is more lasting than that of any of the known analgesic drugs. A 1:1500 solution will give good surgical anaesthesia for 3 hours and freedom from post-operative pain for 3 to 7 hours more.

By the introduction of percaine several of the objections to spinal anaesthesia have been overcome. Its relative non-toxicity ensures that the vaso-motor and respiratory centres will not be unduly depressed.
The duration of anaesthesia which has hitherto been a serious drawback of spinal anaesthesia, can now be arranged to suit the operation. More dilute solutions and smaller quantities can be used for short operations.

Dissolved in 5% saline the solution is hypo-baric. Specific gravity of 1:1500 solution is 1.003. The patient can, therefore, be placed in the Trendelenberg position right from the start. This ensures a very much better blood supply to the brain guarding against serious complications.

Its action is regular and reliable.

It is almost non-irritating to the tissues.

Preliminary medication, treatment and technique

Ephedrin: gr. ½ – 1½ is given intramuscularly immediately before the spinal injection.

Dosage. Maximum dose for tissue infiltration is 200 milligrams. In spinal anaesthesia one very seldom, if ever, need use more than 12 m.g. Toxicity therefore, is extremely low.

Solutions in most common use are 1:200 (1-2 cc), 1:1500 and 1:2000. The latter two are given in amounts of 6 to 20 cc. As a rule 9% saline is used to make up the 1:200 solution and 5% for the other two.

Fairlie (41) finds that 14 cc gives satisfactory anaesthesia for upper abdominal operations. Howard Jones calculates the dose by the length of vertebral column between the 7th cervical spine and the intercristal line. The measurement is made

in inches and from this figure 4 is subtracted in the case of men and 6 in the case of women. Thus he obtains a figure which gives the dose in ccs of the 1;1500 solution. It generally works out at 16 to 18 ccs. I agree with Fairlie that this dose is unnecessarily large. I have not found it necessary to use more than 15 ccs. Measuring the spine certainly is useful, for it gives one a rough idea of what the length is of the column of cerebro-spinal fluid which has to be displaced.

The Injection. The subdural space is treated exactly as ordinary tissues. The patient lies in the lateral position. The needle is inserted in the second or third lumbar space and not more than a few drops of cerebro-spinal fluid are allowed to escape. The percin solution, contained in a 20 cc. record syringe, is injected slowly. One should take 1 - 2 minutes to complete the injection, so that the brain can accommodate itself to the increased pressure. It is also as well to delay a few seconds before withdrawing the spinal needle. This will minimize the escape of fluid through the dural puncture, which may be expected, after such an increase of pressure into the spinal canal.

Position. The patient is now turned into the ventral decubitus for 5 minutes and the foot of the table is raised about 6 inches. This allows the posterior roots to be thoroughly soaked. If it is not done anaesthesia will be incomplete on
account of the "lightness" of the drug compared with the cerebro-spinal fluid. The patient's face is turned sideways so that there results a definite downward slope from the dorsal curvature of the spine to the cervical region. By adopting this position the hypo-baric solution reaches to a point above the 5th dorsal segment which is necessary for good splanchnic anaesthesia, but remains well below the origin of the phrenic roots.

Trendelenburg Position. Five minutes later (occasionally longer if it is feared that anaesthesia will not extend high enough) the patient is turned on to his back and the table is tilted into a slight Trendelenburg position. This is maintained throughout the operation. It ensures the brain of a good blood supply in addition to checking higher extension of anaesthesia. The tilt should not be an exaggerated one, for then the pressure of the abdominal viscera will interfere with the excursions of the diaphragm and further complicate the difficulties of respiration. In certain operations (e.g. cholecystectomy) this position is awkward, but good relaxation more than compensates for it.

Care must be exercised in moving the patient from this position. Sudden elevation of the head may cause syncope. On return to the ward the foot of the bed is raised and only one pillow given.
Avertin. (tri-brom-ethyl alcohol)

**Dosage:** 0.08 to 0.1 grm. per kilogram of body weight dissolved at 35-40°C in distilled water to make a 3% solution. This dose is intended to give basal anaesthesia. We have no experience of larger doses (0.12 or even 0.15 grm.) whereby general narcosis is obtained.

**Effects of overheating the solution.** Hydrobromic acid is split off and dibromacetaldéhyde is formed. This causes severe irritation of the rectal mucous membrane.

The adoption of fluid for solid avertin has brought into the technique a drug, amylene hydrate, which has a stimulating effect on the respiratory centre and is therefore valuable.

**Testing the solution.** It is essential that this should be done before every case. Two drops of cango red are added to 5 cc of the solution. In the presence of hydrobromic acid, a blue colour appears.

**Preliminary medication, technique etc.**

No morphia or morphine is given when used in conjunction with spinal anaesthesia owing to the depressant action on the respiratory centre. A soap and water enema is given on the previous night and a simple water lavage on the morning of the operation. The avertin is given 15 - 20 minutes before the patient leaves the ward for the theatre. In administering the solution which should be freshly prepared, the patient is placed in the Sim's position and the solution run into the
rectum through funnel, tube and catheter. The whole process is completed in 5 minutes, when the catheter is clamped and left in the rectum. Ashworth (42) considers it better to take at least 10 minutes for the injection. He claims more constant results. We see no special advantage in this. Our results have invariably been constant.

In 3 to 10 minutes, from starting the injection the patient has forgotten all about his surroundings and remembers nothing further until he regains consciousness 5 to 7 hours afterwards. On return to the ward a rectal washout is given and the catheter removed.

(1) Amount of Anaesthetic solution used. Level of Introduction. Level of Anaesthesia.

(Amongst these cases none received percam) Spinocain. In most cases 2 cc were given. Three patients had 3 cc. Others had 1½ and 1 cc.

Stovain. 1 - 2 cc of 5% and 0.5 - 1 cc of 10% solution. The smaller dose was given for a lower abdominal operation e.g. appendicectomy, whereas the larger was given for an upper abdominal one. The amount is determined by the site of the operation, and to a degree, by the size of the patient, a tall man, requiring more than a small female.

The usual site of introduction was between L2 and L3 for the upper abdominal operations and between L3 and L4 for the
lower ones. In a few cases the space between L1 and L2 was used. The site of introduction has a definite influence on the height of anaesthesia, but, at the same time, a lower puncture can be partly compensated for by increased tilting.

(2) Additional Agents. Following the above procedure the level of anaesthesia as a rule was satisfactory. Amongst our spinocain cases 4 received ether in addition. Two of these were extremely nervous and the other two were complete failures. Of the stovain cases two required ether for the stitching up, the abdominal muscles becoming rigid, 48 and 60 minutes respectively after induction. In the first case it is possible that some of the drug may have been injected extrathecally. The average duration of full anaesthesia is well over an hour.

When however, a long operation was anticipated it was necessary to supplement the spinal anaesthesia. (We have pointed out that with percan this is unnecessary). For this purpose a splanchnic injection was made. It was given immediately after the abdomen had been opened and a thorough investigation made. With the forefinger and thumb of the left hand the pulsations of the coeliac axis are felt and the splanchnic needle inserted immediately above, while the left forefinger guides its point between the aorta and the inferior vena cava. Before the injection and once or twice during the procedure the piston of the syringe is withdrawn to make sure that the needle has not entered a blood vessel. 50-70 cc of ½% novocain (without adrenalin) are injected.
(3) Previous Medication. Most of these details are to be found under the headings of the different drugs. We, therefore, merely give a general outline of the scheme we followed. When avertin and spinal are combined no morphia is given. There is no real need for it; it adds to the respiratory depression. When spinal alone is given or when avertin and nitrous oxide are combined morphia is given. In very apprehensive people morphia and scopolomine are both given. Much, however, depends on the type of patient, so that strict rules cannot always be followed. A powerful young man can be given morphia with a combination of avertin and spinal; whereas morphia ought never to be given in a feeble person, even if avertin and gas are combined. This is especially so if there is any respiratory trouble. In such cases morphia is best avoided also when spinal anaesthesia is given alone without avertin. Nembutal is to be preferred. It does not interfere with the cough reflex when given in moderate doses (1o grs.).

Ephedrin. Vaso- tonic treatment is of as great importance as sedative pre-medication. We have used both Caffein and Ephedrin. The former has probably no effect at all on the blood pressure. It was soon discarded. Ephedrin, on the other hand, is of real value. In 20 stovaine cases, watched from this point of view, Ephedrin was given in 10 and withheld in 10. The dose never exceeded 1/6 gr. In those that had no ephedrin the blood pressure fell on an average 40 m.m. of mercury, whereas in those that had
ephradin the average fall was 33 mm of mercury. This is in accordance with many views expressed. The action of ephedrin is analogous to that of adrenalin. Its effect, however, is less powerful, although more prolonged. It causes a rise of blood pressure lasting 1 1/2 - 2 hours. It is best to anticipate a fall and give the drug before the fall occurs. Our practice is to give 1/4 or 1/3 gr intramuscularly immediately before the patient is taken to the operating theatre. (Many anaesthetists give it just after the spinal injection). Following the advice of Pitkin (43) we have also given it on the day before the operation. If a patient's blood pressure is found to be under 120 mm of mercury we give him 1/4 gr. ephedrin on the day before and 1/3 gr. immediately before the operation. Our numbers are too small to draw any conclusions as to the value of this practice.

Adrenalin is used for the same purpose. Evans highly commends it. We have used it only during the operation, when there was excessive fall of blood pressure.

(4) Alteration in Pulse Rate. There was an average increase of 12.9 beats per minute in spinocain cases and 9 in those who had Stovaine. The comparison was made between the average rate on the day before the operation, when the nervous

element was still in abeyance, and the rate registered on return of the patient to the ward, after the operation. The maximum increase of pulse rate was 44 beats per minute.

(5) Temperature Changes. These were registered like the pulse changes. There was an average fall of 0.28°F.

(6) Blood Pressure changes were recorded in 58 cases of Spinocain and 58 cases of Stovaine. The latter mostly had avertin as well. The normal is taken as the pressure recorded on the day before operation and comparisons made with that figure.

During Operation:

Spinocain. In one case the blood pressure rose 2 m.m. of mercury. In all other cases there was a fall. The maximum was 50 m.m. of mercury. The average was 15 m.m. of mercury.

Stovaine and Avertin, except for one case, where the pressure remained constant, there was a fall in pressure averaging 41 m.m. of mercury. There appears to be no greater drop when avertin and Stovaine are combined, than when Stovaine is given alone. Our figures, however, are not enough to be conclusive. The biggest drop, 116 m.m. of mercury, occurred in a case whose blood pressure before operation was 196.

In a case, whose pre-operative pressure was 216, the drop was 60 m.m. of mercury. The lowest pre-operative pressure recorded was 108 m.m. of mercury.
After Operation. Pressure readings taken at various times after operation indicated a fairly rapid recovery. But the normal was not reached, for several hours. In the case of the 210 pressure the fall of 60 was followed by a rise of 90 in a few hours.

Additional Comments. We have previously discussed the effects of spinal anaesthesia on blood pressure. In considering the above case reports some additional remarks are called for.

We note that the fall of blood pressure which occurs after spinocain is not nearly so marked as that after Stovain. This may be accounted for by several factors:

(a) In our case spinocain was used for operations below the umbilicus, whereas Stovain was chiefly used for operations above the umbilicus. This meant a greater sympathetic nerve paralysis.

(b) Spinocain is combined with Gliadin. This makes absorption more gradual. Stovaine is a more toxic drug. It is difficult to know how much these factors influence the blood pressure.

The maximum fall of pressure occurs as a rule 15-20 minutes after the injection of the drug (later with percaine). This fall may become more marked as the operation progresses, in which case treatment should not be delayed. We have generally found ephedrin gr. $\frac{1}{4}$ sufficient, although many hold that ephedrin is only of use before the operation and
that a second dose is useless. When the fall occurs very rapidly and indicates collapse, other methods must be adopted. The only alarming fall of which we had experience, was where a drop of 116 occurred in a patient whose normal pressure was 196 m.m. of mercury. Adrenalin was given intravenously. The patient's condition recovered at once. She left the hospital alive and well.

Treatment of severe fall of blood pressure.

(i) Trendelenburg position. If a hypo-baric solution is used, the patient will already be in this position. It may be slightly increased. If a hyper-baric solution is used no fear need be felt lest the anaesthetic runs up to the medulla, for by the time a severe fall of blood pressure occurs the drug will be "fixed". This position must, therefore, be adopted as soon as possible.

(ii) Oxygen and Carbon dioxide. Either of these gases may be used, but a combination is better. A severe fall is often due to anoxaemia. Oxygen is, therefore, indicated. Carbon dioxide, on the other hand, is a strong respiratory stimulant. Should no carbon dioxide be available the patient can be made to rebreathe his own carbon dioxide, while oxygen is freely administered through a nasal tube or by some other convenient method. If a nitrous oxide apparatus is used this is a simple matter.
Adrenaline is our standby in extreme cases. Given intravenously, as it should be in such bad cases, it works magic. A pulseless patient can be restored to “good condition” within seconds. We have used the ordinary 1:1000 solution giving M 3 + 5 but the method of transfusing with a saline-adrenaline solution (adrenaline M 1. to 100 c.c. normal saline) is better. Just enough of the solution is run in to restore the pressure. This naturally presupposes that the apparatus and solution are by the side of the anaesthetist for immediate use. Subcutaneous injection is useless. It takes too long for the drug to reach the circulation in such a depressed condition.

The action of adrenaline is analogous to that of the sympathetic nervous system. It acts on the same structures. Its site of action appears to be an hypothetical myo-neural substance, or junction material, between the nerve endings and the organ of supply. It does not act on the nerve endings themselves, for it will still act even after the sympathetic nerves have been divided and allowed to degenerate. Although it is by no means certain that the capillaries are innervated by the sympathetic nervous system there is some evidence that adrenaline constricts the capillaries as well as the arterioles. According to Wright (44) the capillaries can resist greater force tending to distend them if they have first been constricted.

(44) Applied Physiology, p. 312.
by adrenaline. (Perhaps it is partly on this account that ephedrin
is more valuable when given before the blood pressure falls).
From the point of view of spinal anaesthesia the important
effects of adrenaline are (a) Acceleration of the heart beat and
increased force of the beat by stimulation of the junctions of the
cardio-augmentor nerve fibres.
(b) Constriction of the arterioles of the skin.
(c) Constriction of the arterioles of the splanchnic area.

By constricting the arterioles the peripheral resistance
is raised, pressure rises and the basal centres are better
supplied by blood.

A comforting factor in spinal anaesthesia is the
knowledge that the worst trouble will be over after 15-20
minutes. In long operations we may proceed with confidence once
this mark is passed, knowing that the blood pressure will
gradually rise as the effect of the drug diminishes. In ether
the blood pressure is affected in the reverse way. First there
is a slight rise to be followed by a gradual fall of pressure.
In long operations this fall may become pronounced towards the
end, when the patient can ill afford it. What is worse, the
tendency to fall may continue for some time after the conclusion
of the operation. In the first case one works away from the
danger, in the second towards it.
(7) Effects noted during the Operation.

Pallor occurred in 39% of spinocain and in 57% of Stovaine cases.

Nausea occurred in 20% of spinocain and in 17% of Stovaine cases.

Vomiting occurred in 3.6% in spinocain and in 7% of Stovaine cases.

Sweating occurred in 25% of spinocain and in 37% of Stovaine cases.

Causes and treatment of Nausea and Vomiting.

(a) Cerebral anaemia. Frequent deep breaths and keeping the head low brings speedy relief as a rule.

(b) "Psychic nausea" is due to insufficient pre-operative sedative treatment and usually occurs in patients who had no morphia.

(c) Reflex vomiting is produced by packing the upper abdomen and manipulations of the stomach. To prevent this Hewer (45) and Norman Lake (46) recommend the injection of a few ccs of local anaesthetic into the lower end of the oesophagus. This blocks the afferent impulses from the stomach.

(8) Early after effects.

Headache, was noted in 22% of our cases. All but 2

were mild and responded to aspirin or veramon. In one patient
the pain started on the third day and lasted for a week. In
another case there was rather severe pain in the neck. Both
these cases fall under the class, non-infective or irritative
meningitis, to which we have referred.

Backache occurred in 28% of cases, but the report was
usually made in response to a direct question (as in the
case of headache). One would probably find an equally high
percentage after other types of anaesthesia. In two cases,
however, there was severe lumbar backache.

Causes of Backache.

(a) Trauma by the needle. We have often noticed that
when it was difficult to insert the needle in one inter-space
it would slip in easily at another. We have made it our
practice not to traumatise the tissues by persisting in one
space, but rather to anaesthetise over a second and insert
the needle there.

(b) Extrathecal injection and seepage of cerebro-spinal
fluid are said to be causes.

Vomiting occurred in about 50% of our cases. It was
very seldom of severe character. In 20% only was it repeated
more than once. This is an improvement on general anaesthesia,
but not such a striking one as others report.
Retention of urine occurred in 8 cases out of 82 observed. As this has been mentioned a drawback of Spinal anaesthesia we specify the cases in whom it occurred:

- Cystoscopy
- Cholecystectomy
- Dilatation and curettage
- Cholecystectomy
- Herniectomy
- Ovariectomy and Herniectomy
- Colpo-perineorrhaphy
- Partial gastrectomy

Judging by these cases and the fact that all cleared up in a few days, most requiring only one catheterisation, we can hardly call retention of urine a special drawback of spinal anaesthesia.

**Ileus.** In no case could we blame the anaesthetic for producing intestinal paralysis. Where such cases were encountered there always existed some other obvious cause.

There was no evidence of increased frequency of post-operative distension.

**Paralyses** were never seen.

**Trophic Ulcers.** One of our cases, a well nourished healthy young man developed a bed sore over the sacrum on the day after an herniectomy was performed. The spinal anaesthesia may have been a factor in its production. It is difficult to explain how it could have occurred otherwise.

The above are always mentioned as special complications of Spinal Anaesthesia.
RESPIRATORY COMPLICATIONS

(a) Embarrassment of breathing 2 cases
(b) Collapse of Lung 1 case
(c) Broncho-pneumonia 4 cases
(d) Bronchitis 2 cases
(e) Cough without physical signs occurred frequently especially after acute abdominal conditions.

None of these cases terminated fatally. The case of collapse of the lung was treated early with carbon dioxide and oxygen inhalations and made an exceptionally rapid recovery, without the least sign of superadded sepsis.

LATE AFTER EFFECTS

We examined 84 of our cases at periods varying from 4 to 10 months after the anaesthetic with the following result:

(1) Indefinite complaints such as occasional numbness weakness or cramp in the legs were obtained in about half a dozen cases.

In two cases there was a definite complaint of paraesthesia. In both cases this was limited to the lateral side of the left thigh. Both were gradually improving. We could find no sign of organic nerve lesion. We believe the cause of the pain was injury to a nerve root.
(ii) There were 3 cases with bladder symptoms. We give their details:

(a) A young man had a stone removed from his ureter in Jan 1932. Since then he suffered from urgency and frequency of micturition.

(b) A young woman whose abdomen had previously been explored with negative result, was given a spinal anaesthetic in Feb 1932 for cystoscopy, pyelography and ordinary physical examination on account of frequency of micturition and dysuria. Nothing abnormal was found. Since this examination she suffered from incontinence of urine. In view of all the circumstances it would be unfair to blame spinal anaesthesia for the incontinence in this woman. She is a type of patient who may develop incontinence after any form of anaesthesia.

(c) A middle aged woman had partial gastrectomy performed for carcinoma of the stomach in Feb. 1932. In October 1932 she developed incontinence of urine. The spinal anaesthetic almost certainly has no relation to this. No cause could be found.

The patient's attitude towards the Anaesthetic. To a number of these patients we put the question, "If you had to have another operation what kind of anaesthetic would you prefer?" Their answers can be divided into 3 groups:
(a) Men practically without exception preferred to have spinal again.

(b) The majority of women "would rather sleep".

(c) Both men and women who had previous experience of general anaesthesia most definitely preferred spinal anaesthesia.
SECTION III

(1) The Usefulness of Avertin.

Since Blomfield and Shipway (47) published a report on 198 cases of Avertin Amnesia the scope and safety of this agent has undergone a vast change. In the beginning, when the recommended dose was 0.15 grm per kilogram of body weight, some fatalities were recorded and numerous alarming phenomena, chief of which were depression of respiration, anoxaemia and the concomitant collapse were noted. With the introduction of a smaller dose the administration became absolutely safe. To those who consider Avertin an amnesic, its use will be attended by great success, but not to those who try to use it as an anaesthetic. It is a great mistake to give it in such doses, as would induce surgical anaesthesia, without additional agents. Avertin was never claimed to give surgical anaesthesia in safe doses. It was styled from the first as a basal narcotic.

As with other anaesthetics there is some variation in the reaction of different patients to Avertin. A dose, which will keep one patient on the "light side" will procure complete surgical anaesthesia in another. Why this should happen is not always clear. We have noticed that fat women seldom require

(47) Proc. Roy. Soc. of Med. (Sec of Anaes.) Nov. 1929
more than 0.09 grm per kilogram of body weight, whereas such a small dose would be insufficient for an ordinary male. Possibly one should take into account the quantity of metabolically inert fat. Children on the other hand will stand more; and one may err on the bigger side. A dose of 0.11 grm per kilogram of body weight will not be too much.

The type of operation has a certain bearing on the dose, a long operation requiring a slightly bigger one.

One of the chief advantages avertin narcosis has over inhalation narcosis with chloroform or ether is that there is a quiet and uneventful period of induction, none of the dread attending the surroundings of the operating theatre. The patient wakes up 6 or 7 hours afterwards to find that the operation is over - a pleasant return to consciousness. In many instances loss of memory is retrograde and the patient will not remember the events of the 5 minutes preceding unconsciousness.

Contraindications are few.

Absolute ones are:

1. Old people suffering from extreme degrees of thyroid deficiency, toxaemia, or uraemic symptoms.

2. Shock or haemorrhage with excessive fall of blood pressure.
Relative contra-indications.

1. Very poor lung expansion. Bronchitic subjects are ideal cases for this type of anaesthesia, but one must be careful in conditions where a large amount of lung tissue has been destroyed or put out of action by some means or other. Such patients will not stand full doses on account of the respiratory depression that follows.

2. Hepatic deficiency. Only very severe degrees of hepatic deficiency will contraindicate the use of avertin, but it is advisable to give smaller doses when liver disease is known to exist. Smaller doses will probably have good effect. Raginsky and Bourne have pointed out that in dogs smaller doses produced deep anaesthesia after their livers had been damaged by chloroform.

3. Cardiac diseases. Avertin is useful in people suffering from cardiac complaints because it reduces struggling to a minimum. But it must be used with care. It depresses the heart's action and only small doses should be given.

4. Renal deficiency. About 99% of the drug is excreted by the kidney. Severe hypofunction of the kidney is therefore a contra-indication. In actual practice and experimentally however, it has been found that no poisoning symptoms result in cases of very obvious renal disease. There is merely a moderate extension of the excretion time. Maddox (48) has

(48) Avertin Rectal Anaesthesia p. 45.
given an overdose of avertin to a dog with a blood urea of 243 mg per cent, without producing any harmful effects. It must nevertheless be remembered that operation alone frequently suffices to precipitate an attack of uraemia in such cases.

It has been our practice never to give avertin in any case of acute or subacute infection. The reason was our fear of the parenchymatous changes in the liver and the kidneys so frequently associated with infective conditions. I have since learnt that avertin is regularly given to such patients without any harm ever resulting. Shipway (49) invariably gives it and has never regretted doing so. Maddox does the same. This only shows that moderate liver and kidney changes do not affect detoxication and excretion of the drug to an extent that will cause harmful symptoms.

5. Diseases of rectum and anus. An ulcerated mucous membrane bars the use of avertin on account of the irritation that may follow. If there is no ulceration it can be given. In such cases we administer it 30 minutes before the operation and wash the rectum out with plain water immediately before the operation. Anaesthesia is less lasting but still very beneficial.

Anaesthetists (5) warn against the use in cases where a rapid fall of blood pressure is to be expected. Hewer (51)

(49) Personal Communication, p. 49
(50) Raginsky and Bourne: Anaes. & Anal. Jan-Feb 1932
(51) Recent advances in Anaesthesia. p. 34
especially mentions spinal anaesthesia as such a condition. Surgeons, on the other hand, have come to regard this fall of blood pressure to be of little practical significance. Young (52) with an experience of over a thousand cases almost disregards it now. Theoretically one would expect a very considerable fall when Avertin and spinal anaesthesia were combined. In actual practice this does not take place. We are unable to show from our cases that the combination of avertin and stovaine, caused any greater fall in blood pressure, than would have been caused by the stovaine alone. We do not know what the explanation of this phenomenon is. There is one possible factor which may have a bearing. Avertin does not protect the central nervous system against the noxious stimuli originating in the field of operation nearly so well as other anaesthetics. If used alone or with ether it is quite likely that a certain degree of primary shock would result from the operation. This would further decrease the blood pressure. When used in conjunction with spinal anaesthesia such a factor can certainly be eliminated.

Indications: Keeping in mind its few contra-indications, avertin amnesia offers an extremely wide range of usefulness. We do not desire to enumerate them, but rather to point out a few which in our experience have been outstanding.

(a) Chronic appendicitis. In combination with nitrous oxide oxygen, as originally used in Germany, we have found it very satisfactory.

(52) Br. Med. Jour. 5th Dec. 1931
(b) In major abdominal operations, such as gastro­enterostomy, partial gastrectomy, resection of bowel etc., where the operation is protracted, avertin, combined with spinal anaesthesia, is a wonderful help to the patient, relieving the mind of all anxiety, while the spinal anaesthetic gives perfect relaxation. It has been our practice to give Avertin in all such cases.

(c) In hysterical women and chronic alcoholics Avertin is of the greatest usefulness. One does not see the struggling on the anaesthetic table so often associated with the first stage of ether anaesthesia.

(d) In children generally we have found it of the utmost advantage.

(e) In major operations above the diaphragm, such as mastectomy, we have found it very beneficial. The small amount of ether that is necessary to produce surgical anaesthesia is surprising. We have referred to Dandy’s extremely satisfactory and encouraging report on 250 brain operations.

(f) Of greater use, however, than in any of the above, do we consider it in cases of Exophthalmic goitre, where the psychic element plays such an important role. In combination with local infiltration anaesthesia extremely gratifying results are obtained. It does away with the necessity of the great speed with which it is customary to tackle these cases. It enables the surgeon to do a more deliberate dissection with greater care to
technique and avoidance of all unnecessary trauma. Since the introduction of pre-operative iodine therapy, this method of anaesthesia has probably been the greatest advance in the surgery of the thyroid gland. It has reduced the death rate in the "poor risk" cases of Grave's disease.

There is another important factor to be reckoned with in this connection. Pribram (53) observed that Avertin and thyroxine appeared to have antagonistic actions. He gave 2-3 cc thyroxine intravenously to patients suffering from respiratory depression after avertin. Response was prompt. Parsons (54) states that thyroxine detoxicates the drug and minimizes the risk of toxic symptoms. Patients with a high metabolic rate benefit most from the drug. Conversely, avertin may prove harmful in patients with a low basal metabolic rate.

Dangers. The likelihood of serious complications is extremely small. One naturally has to select one's cases and not forget that Avertin is a potent drug. Measuring of the dose and heating of the distilled water must not be left to a nurse. The freshly prepared solution must be tested with Congo Red in every case. Apart from these technical considerations there is really only one danger, that of respiratory embarrassment which if not promptly treated may lead to collapse. In such case the patient's head must be lowered at

(53) Zentralblatt für Chirurgie, 14th Dec. 1929
once and oxygen administered, or better still a mixture of oxygen and carbon dioxide. In extreme cases 1 cc. of coramine has been highly recommended. There is a tendency for the tongue to fall back and this must be watched for both in the theatre and in the ward. The head should be kept low on return to the ward. An injection of Ephedrin gr \(\frac{1}{2}\) hastens recovery. A disadvantage in the use of avertin is that the patient is sometimes restless in the theatre and on his return to the ward. In the theatre a few drops of ether on the open mask are quite sufficient to control restlessness, and heroin gr 1/12 may be given safely in the ward during the period of recovery. Morphia should be withheld on account of its depressive effect on the respiratory centre.

(2) THE USEFULNESS OF SPINAL ANAESTHESIA

That spinal anaesthesia has an extremely wide field of usefulness in operative surgery below the diaphragm, nay, that it is the anaesthetic of choice when combined with a basal narcotic, has been our experience in the clinics whence this report eminates. There it has been the routine procedure. The results have justified its use. We are, however, aware of the fact that similar results are claimed for other forms of anaesthesia. We, therefore, propose not to discuss the general application of spinal anaesthesia but to confine ourselves to its special indications, where we consider spinal to be superior to any form of anaesthesia, on condition
that some of its absolute and well-defined contra-indications are not present simultaneously. In defining these indications, we shall almost entirely confine ourselves to the cases reported above, but there are a few others, outside our experience, of which we have to take note.

**INDICATIONS.**

(1) In certain organic visceral diseases where general anaesthesia is contra-indicated and local anaesthesia not possible or advisable.

(a) **Lungs.** (1) Acute conditions, such as bronchitis and pneumonia, associated with a surgical condition that requires immediate operation.

(2) Chronic diseases such as bronchitis, emphysema, bronchiectasis and tuberculosis.

(If, however, there is marked deficiency of useful pulmonary tissue, such as one finds in advanced tuberculosis, or not empyema, a high spinal must be given. Such a patient will not stand the respiratory embarrassment incident on paralysis of the intercostal muscles. A low spinal need not be feared.)

(b) **Cardio-Vascular.** Arterio-sclerosis is considered by some to be a contra-indication, on account of the fall of blood pressure and the danger of sudden heart failure. Such cases are bad for any type of anaesthesia. Most observers agree that the great fall of pressure
in these cases does not as a rule lead to serious consequences. The fall can be greatly controlled if anticipated. Amongst our cases there were several with high blood pressures. The results have been good. Where there is evidence of heart failure in the arterio-sclerotic, e.g. cyanosis, swelling of ankles, and a low pulse pressure spinal anaesthesia should not be given.

(c) Liver. Marked liver insufficiency is a strong contra-indication for general anaesthesia. Where cholaemia has advanced spinal anaesthesia is also contra-indicated. Only local should be used.

(d) Pancreas. Diabetes cases give very excellent results with spinal anaesthesia.

(Acute pancreatitis is a contra-indication on account of the shock present.)

(e) Kidneys. Cases with renal dysfunction, as evidenced by high blood urea, albumen or casts, do far better under spinal than under general anaesthesia. A temporary diminution, or even suspension, of kidney function is possible on account of the low blood pressure, but this appears to have no serious consequences. The results obtained in prostatic surgery with spinal anaesthesia are very good. In many of these cases the problem of renal failure is the main consideration.
Caesarean section for eclampsia is a special indication that should be mentioned in connection with renal failure.

(ii) Where extensive operative procedures are carried out below the umbilicus.

Some typical examples are excision of rectum, Wertheim's operation and plating of the femur. Spinal anaesthesia is responsible for the prevention of a great deal of shock in these cases. It need occasion very little fall in blood pressure, therefore, causing no harm. The patient is saved a prolonged saturation of his tissues by a toxic volatile anaesthetic.

(iii) Certain Abdominal emergencies.

(a) Intestinal Obstruction. The sympathetic nervous system exerts an inhibitory influence on the intestine. With spinal anaesthesia this inhibition is abolished and active peristalsis follows. This brings about the emptying of the distended coils through an artificial opening or the anus. Nobody will dispute the advantage of thus ridding the patient of a collection of toxic intestinal contents. Following relief of obstruction in the small bowel intestinal contents are frequently passed per rectum while the patient is still on the table. In large bowel obstruction we have invariably seen large quantities of faecal matter discharge through a Paul's tube immediately after insertion, without any aid of the hand.

Russel and Sworn (55) have published a series of cases.

where the induction of spinal anaesthesia without operation brought relief. Amongst these cases were 3 of spontaneous reduction of a strangulated hernia. With due regard to the value of the method in other forms of obstruction, e.g. impacted faeces, we cannot convince ourselves of the safety of this procedure in cases where incipient gangrene of the bowel cannot be excluded. The writers state that they have found no cases recorded where the bowel had perforated following similar treatment. Their argument is not very convincing.

Norman Lake (56) warns that in very debilitated people the rapid evacuation of the bowels and consequent loss of fluid may prove fatal. He cites two cases. It appears that both were almost moribund before the operation. One feels that this disadvantage is amply compensated for by the advantages that spinal anaesthesia gives. Moreover, as long as we possess water and salt we have a powerful weapon with which to fight such dehydration.

A practical point in connection with operations for intestinal obstruction under spinal anaesthesia is to remember that the patient is not free from the risk of aspirating his vomitus. He should, therefore, not receive much sedative premedication. It is also safest to leave a small tube, such as Jutt's tube, in the stomach after washing it out, so that the anaesthetist can aspirate any fluid that collects in the stomach.

(56) B.M.A. Centenary meeting 1932.
The usefulness of Spinal Anaesthesia in Ileus

We are using the term "Ileus" in a wider sense than would be indicated by "paralytic ileus". Ileus occurring in general peritonitis is generally called paralytic ileus. It is not uncommonly due to mechanical interference such as fibrinous adhesions. Other cases are said to be produced by overaction of the sympathetic nervous system due to toxic absorption and irritation. There does not seem to be sufficient foundation for this theory. If it were true then spinal anaesthesia should relieve such cases. There is no evidence that it does.

A second type of ileus, so-called adynamic ileus, is not infrequently encountered. It is due to inhibitory nervous influences. No mechanical cause for obstruction exists. It is usually observed after comparatively simple operations on the abdominal viscera where there has been little or no interference with the intestines. The belief that it is caused by overaction of the sympathetic nervous system is borne out by the fact that relief follows administration of a spinal anaesthetic.

There is another form of ileus, the paralysis that develops in the distended coils of bowel above a mechanical obstruction which has been allowed to persist too long. The condition is probably due to interference with Auerbach's plexus by toxic absorption from the bowel. In such cases spinal anaesthesia cannot induce peristalsis. Peristalsis is dependent on an intact intrinsic nervous system. The following two cases
illustrate its importance. One was an old man with a strangulated inguinal hernia, involving the small intestine, the other a middle aged woman with gall stone obstruction of the small bowel. Both had been obstructed for several days with regurgitant vomiting etc. Both were operated on under spinal anaesthesia. In neither case did we see visible peristalsis in the distended bowel above the obstruction. Resection of intestine was necessary. In the female case the distended bowel was emptied through the hole by which the stone was removed. Both died. At autopsy I found the bowel, above the seat of the old obstruction, enormously dilated. The sites of stone and strangulation respectively were in excellent condition. But, apart from what had passed by mere hydrostatic action to the coil immediately below, there were no intestinal contents in the collapsed bowel below the original site of obstruction. The only explanation I can offer, is that the bowel had become so completely paralysed, that no more peristalsis was possible even though sympathetic action had been temporarily controlled. It is clear that no other form of anaesthesia would have been of any greater value.

As a result of this experience I believe that its reaction to spinal anaesthesia definitely indicates what the condition of the bowel is. If no peristalsis is seen in the bowel above the obstruction one must regard it as intrinsically
paralysed. In such cases mere relief of the mechanical cause of the obstruction is insufficient. One must leave a tube in the bowel above, preferably more than one tube. Even then the chances of recovery are few.

Intussusception is a form of obstruction where spinal anaesthesia has proved so successful, as to have become an absolute indication. Hamilton Bailey (57) advises 0.2 cc of stovain in saline to be given for infants under 2 years. He specially comments on the relaxed condition of the abdomen.

(b) Acute Appendicitis, with or without localised or generalised peritonitis. We can find nothing in the literature to support this view. Our results, however, have been so satisfactory that we feel justified to draw this conclusion. We are aware of Labat’s (58) warning that the increased peristalsis may spread the infection. On the other hand, there is the immense advantage of spinal anaesthesia causing a minimum disturbance of metabolism. The relaxation, too, is of the utmost value in helping the surgeon do to the minimum amount of trauma. We hope at some future date to be able to substantiate this view.

What has been said of acute appendicitis is not altogether true of a perforated peptic ulcer. Our results have been good. The relaxation obtained is again a strong argument in favour of

(57) Emergency Surgery, p. 205
(58) Regional Anaesthesia, p. 509
spinal anaesthesia. But there are two other considerations:

(1) In early cases there is marked shock, the blood pressure is low.

(2) A high spinal anaesthesia is required. This means a great fall of blood pressure.

We believe that once shock has been overcome, spinal anaesthetic is to be preferred.

IV. FRACTURES OF THE LOWER LIMBS. In simple fractures of the bones of the leg local anaesthesia is to be preferred. When dealing with a femur, or a compound fracture of the leg bones, or a case where a septic skin contra-indicates the use of local anaesthesia, spinal is the anaesthetic of choice.

Relaxation is complete that the fragments literally fall together. Spinal anaesthesia not only simplifies reduction, it is an aid to perfect reduction.

V. VASO-SPASTIC DISEASES. The value of spinal anaesthesia in these cases is that it aids in the prognosis. To Telford and Stopford (59) we owe the elucidation of this important principle, although other means to the same end had previously been employed. In the normal individual the surface temperature of the lower limbs rises on the average by 8°F after the administration of a spinal anaesthetic. This temperature is measured by a delicate electrical skin thermometer. In thrombo-angitis obliterans - a condition where spasm precedes thrombosis

of the vessels - they found that after spinal anaesthesia the skin temperature of the lower limbs, as measured on the dorsum of the foot, rose to a lesser or greater degree. The rise usually was far less than that in the normal individual, but they were able to conclude, that the higher the rise, whether in relation to the two limbs together or to either limb separately the better the prognosis after lumbar sympathectomy.

Spinal anaesthesia is first felt by the sympathetic nerves, the immediate effect being a "feeling of warmth in the feet and legs" due to peripheral vaso-dilatation. This always precedes loss of sensation and motor power. Hughes (60) lays great stress on this as indicating the possibility of a good peripheral circulation and confirming the fact that ramisectomy or ganglionectomy is going to bear fruit.

In cases of gangrene due to arterio-sclerosis, this feeling of warmth is often experienced at a certain level, and gives the surgeon a good idea as to where amputation can be successfully and economically performed.

CONTRA-INDICATIONS

(1) Hypo-tension of any kind contra-indicates spinal anaesthesia. The lowest limit quoted for spinal anaesthesia to be safe is 100, although most people prefer 110 m.m. of mercury. Amongst our material the lowest pre-operative pressure was 108 m.m. of mercury. A great deal depends on

(60) Personal communication.
whether the anaesthesia is to be a high or a low one. The following are the types we may expect to find a low pressure:

(a) General debility
(b) Failing heart
(c) Haemorrhage
(d) Shock.

Should spinal anaesthesia be very specially indicated, for some reason or other, in a case of severe haemorrhage, the objection to its use may be overcome by blood transfusion.

One of our cases demonstrates the importance of haemorrhage as a contra-indication during or after the operation. Spinal anaesthesia was induced to clear the uterus of the remains of an aborted ovum. Severe bleeding took place at the operation. The woman later developed double internal iliac vein thrombosis. The low pressure caused by the haemorrhage plus the low pressure of the spinal anaesthesia cannot be disregarded as the aetiological factor in this case.

In shock spinal anaesthesia is decidedly dangerous. The patient will not stand the additional lowering of the blood pressure. Evans states that, where shock exists, without haemorrhage, spinal anaesthesia might be safer than general anaesthesia. We are of opinion that a short emergency operation, under such circumstances, is much more safely performed under ether anaesthesia.
(ii) Local Conditions.

(a) Sepsis in the skin, in the deeper tissues or in the spinal canal itself precludes spinal anaesthesia. Neurosyphilis is a contra-indication in that the vitality of the tissues is so low that damage may result. For medico-legal reasons spinal anaesthesia is better avoided in any nerve disease, where it may be blamed for a later paralysis.

(b) Deformities of the Spine may make a lumbar puncture impossible.

(iii) Cerebral and cerebellar tumours. In cases of increased intracranial pressure such a delicate balance exists between the blood pressure and the pressure in the cranium that the lowering of blood pressure produced by a high spinal anaesthesia may so disturb the compensation as to lead to death.

MORTALITY

Death may be due to the anaesthetic directly, or it may be caused indirectly by the harmful effects of the anaesthetic on the already devitalised tissues of the patient. In such cases the anaesthetic is a contributing factor. As a rule, when the anaesthetic is directly responsible, the patient will die on the operating table or very soon afterwards. Where the influence is indirect, the patient may live hours or days afterwards, e.g. death from pneumonia caused by an irritating volatile anaesthetic.
When death takes place on the table the anaesthetic is usually blamed, and very often wrongly. It appears that the immediate or direct mortality after spinal anaesthesia is greater than that after ether anaesthesia. Figures vary so widely as from 1 in 200 to 1 in 10,000. They obviously are unreliable. Where spinal anaesthesia is used as a routine the death rate is very low. But where it is used as an expediency the death rate is high. This fact is accounted for by inexperience, which one can expect under such circumstances, and wrong selection of cases. The usual attitude is "Oh, his condition is too bad for a general anaesthetic, we'll give him a spinal." Naturally, if spinal anaesthesia is given to such poor risk cases only, the death rate will be high. We have not seen a spinal anaesthetic death, and in the series of cases from Bradford namely 536, there have been no deaths in the theatre and none in the ward within 12 hours of operation.

With regard to the deaths that may be indirectly or partly attributed to the anaesthetic, we are again on insecure ground. No matter how carefully statistics are kept it remains beyond our knowledge to tell exactly how many patients die as a result of the added destructive effects of the anaesthetic. It is only when we come to consider the figures of individual surgeons, who for a certain period have operated under spinal, and for a similar period, on similar material under general
anaesthesia, that we are able to approach a little nearer to
the truth, even though it is not the whole truth. We are also
justified in making deductions, when we compare results in any
special disease or group of diseases, e.g. intestinal
obstruction, genito-urinary diseases.

In the series of Basil Hughes 22 cases died out of a
total of 536, i.e. 4.1 per cent. As we have no figures to
compare these with we shall quote some from the literature.
These are not selected figures, neither did we specially
search for them in order to establish our point. They were
observed while in search of other material.

For operations for rectal carcinoma Miles (61) had a
mortality of 37% when using chloroform and ether and 12.8%
when using spinal anaesthesia. Since the war he had done only
2 cases under ether and both died.

W.F. Campbell (62) states the death rate is 4.4% less
after spinal than after general anaesthesia, and there is
an especially striking reduction in upper urinary tract surgery
and prostatectomy.

Norman Lake's (63) mortality for partial gastrectomy
under general is 10% whereas under spinal anaesthesia it
is 4%.

The mortality figures for acute intestinal obstruction
collected by Vick (64) from 21 English hospitals is 38.8%

(61) Modern operative Surgery Vol II. p. 81 (Carson 1927)
(63) B.M.A. centenary meeting Sec Anaes 1932.
(64) ditto.
excluding external hernias, and 26.2% for hernias. Basil Hughes (65) who routinely uses spinal anaesthesia, has a mortality of 15% in intestinal obstruction.

Foss (66) states that of 400 acute appendix cases, in 70 of whom there was peritonitis, 200 were operated under ether and 200 under spinal anaesthesia. The first group gave a mortality of 7.4% and the latter 4.2%. His findings in perforations and in gall bladder surgery were similar. He puts the improvement down to the anaesthetic.

From what we have just seen, it appears that we may possibly expect a higher table mortality from spinal anaesthesia but the later, or indirect anaesthetic mortality will be considerably reduced. By using spinal anaesthesia we may have to attend the inquest court more often, but we shall have fewer funerals to go to.

SUMMARY

Section I.

1. The pathology of shock is briefly discussed.
2. The qualities of an ideal anaesthetic are enumerated. Ether, Avertin and Spinal Anaesthetics are examined in this light.
3. Ether is noted to fail in the prevention of traumatic shock. It causes shock.

(66) Annals of Surgery. p. 738
Spinal anaesthesia protects against traumatic shock, but not against psychic shock. Its great disadvantage is the fall of blood pressure.

(4) The causes of fall of blood pressure are fully discussed.
Distinction is made between the low pressure of Spinal anaesthesia and the low blood pressure of shock. Two cases are cited.

(5) An attempt is made by liver function tests to determine whether clinical proof of shock, severe enough to cause liver damage, could be obtained in cases of spinal anaesthesia. After taking into consideration the effects of Avertin, it appeared that spinal anaesthesia did not damage the liver.

(6) The effects of avertin on the liver are studied. Avertin appeared to produce additional damage in the diseased liver. Practical suggestions are made for the use of avertin in the presence of disease of the liver.

SECTION II.

(7) A report is presented on 182 cases of spinal anaesthesia with or without avertin.

(8) Details of technique are given and some minor points - local anaesthesia and haemorrhage in connection with lumbar puncture, are considered in detail.

(9) Effects of the anaesthetic during and after the operation are reported and discussed. Late after effects are reported.
SECTION III.

(10) The usefulness, indications and contra-indications of Avertin and of Spinal Anaesthesia are discussed with special reference to the case reports.

(11) Mortality is discussed. Spinal anaesthesia when not used routinely appears to have a higher immediate mortality than general anaesthesia but a definitely lower total mortality.

CONCLUSION

Before deciding to use spinal anaesthesia we must satisfy ourselves about two, possibly three, disadvantages. In comparison with these all its other disadvantages dwindle into insignificance. Yet if we cannot satisfy ourselves that these two or three disadvantages can be overcome or that the advantages of spinal anaesthesia do not weigh up against them, we dare not use spinal anaesthesia.

What is our position with regard to (1) Fall of blood pressure (ii) Failure of Respiration (iii) Paraplegia?

With regard to paraplegia I would not use spinal anaesthesia, except as a last resort, if I had to fear such a grave disaster. There is no doubt, however, that it cannot occur in the hands of the careful and conscientious surgeon who makes his injections below the level of the cord. Such cases as have
occurred are exceedingly rare and can only be accounted for by
an error in technique or the co-existence of a neural lesion.

Failure of respiration is so intimately bound up with
excessive fall of blood pressure that we would not have
considered it separately as one of the important disadvantages
except to state that paralysis of the phrenic nerves is an
extremely rare occurrence. Moreover, it should not lead to a
fatal result. The effect of the anaesthetic on the phrenic nerves
will wear off very soon and meanwhile the patient can be kept
alive by artificial respiration.

Fall of blood pressure remains the one outstanding
disadvantage. To a certain degree it is unavoidable and will
remain so, unless a drug can be discovered which, by selective
affinity will paralyse the motor and sensory fibres without
affecting the sympathetic fibres of the spinal roots.
With a thorough understanding of the causes of the fall of
blood pressure we can, however, select our cases in such
a way that they will not suffer as a result of the low
pressure. Even though the arterial pressure falls to a low
level in the forearm it does not necessarily imply that the
pressure in the brain is low. With the head down the brain will
rarely have an inadequate supply of blood. A low peripheral
pressure may be a result of the redistributary functions of the
vaso-motor centre which deviates the blood flow from relatively
unimportant areas to the vital organs of the body. Moreover,
modern methods have been so successful as to have almost overcome
this disadvantage. By the use of relatively non-toxic drugs, the adoption of the Trendelenburg position (with hypobaric drugs immediately and with hyperbaric ones a short while after injection), bandaging of the lower limbs, the prophylactic injection of ephedrin and the emergency treatment with adrenalin intravenously and inhalations of oxygen and carbon-dioxide, the fall of blood pressure has been so successfully combated as to have become a minor disadvantage in the hands of the experienced.

Confident of the elimination of these drawbacks we find ourselves in a position to utilise the outstanding advantages of spinal anaesthesia. Of these the most important is prevention of traumatic (primary) shock. We can combine with it the use of an agent which will allay all mental pain and prevent psychic shock. In other words, we can produce complete anociation by employing such a combination. "Anociation" says Crile, "is the goal of operative surgery."

We have, in this light examined a series of cases and taken into account the views of others. We have noted that no one anaesthetic is perfect or ideal, in the sense that it protects the brain completely against the effects of local operative trauma and destructive psychic strain. Spinal anaesthesia protects against the effects of local operative trauma, but not against psychic strain. Inhalation anaesthesia protects against the latter, but not against the first.
Each covers only a part of the field. We have observed the
great usefulness of Avertin as a basal anaesthetic. A
combination of spinal anaesthesia and avertin has been
used in a series of cases. The results have been extremely
satisfactory, both immediate and remote.