MISSED OPPORTUNITIES FOR THE DETECTION OF ABDOMINAL AORTIC ANEURYSMS: A RETROSPECTIVE STUDY OF EIGHTEEN PATIENTS PRESENTING WITH A RUPTURED OR ACUTE SYMPTOMATIC ABDOMINAL AORTIC ANEURYSM

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DISSERTATION
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" Diseases desperate grown

By desperate appliance are reliev'd

Or not at all "

(Hamlet Act iv Sc iii)

" HOW HISTORY MAY HAVE BEEN CHANGED?"

It is interesting to note that Albert Einstein died from a ruptured abdominal aortic aneurysm. [1] *Had he been alive today and consulted his general practitioner, his aneurysm would have been diagnosed and successfully repaired, ensuring him a normal life expectancy. How would the world not have benefited from this genius ?*

DECLARATION

I, ROY THOMAS MARONEY hereby declare that the work on which this thesis is based is my original work (except where acknowledgements indicate otherwise), and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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Date

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DEDICATION

*I dedicate this thesis to my loving wife **JEANNE** and our 3 sons **LLOYD, UWEN** and **BYRON** for sacrificing precious family time to allow me to complete my task.*

SUMMARY

A ruptured abdominal aortic aneurysm (AAA) has a mortality of 80 percent. The majority of these cases present as medical emergencies, with 50 percent dying before they reach hospital. Twenty percent are not operated on because of an incorrect diagnosis and of the surviving 30 percent, there will be a peri-operative mortality of 40 percent. Thus only 20 percent of patients survive this condition. It is important to state that the long-term survival reported for patients undergoing AAA surgery approximates that of age-matched populations.[2] This is in contrast to patients undergoing a coronary bypass procedure, where the long-term survival is affected by factors such as hypertension, angina and peripheral vascular disease. If the condition is diagnosed electively, the mortality rate is reduced to less than 5 percent.

The researcher obtained the records of 18 patients who had presented to the vascular service at the New Kingsbury Hospital with a diagnosis of a ruptured or acute symptomatic AAA. He interviewed the referring family doctor and also obtained information from the case records to determine whether there were missed opportunities for the detection of such aneurysms. The results of the research showed that 12 general practitioners (GP's) out of a group of 13, were unaware that abdominal ultrasound is a highly specific and sensitive method for detecting AAA's. Only one of the group of 13 GP's regularly screens for AAA. The diagnosis of AAA was missed in 12 of the 18 patients. In this series the mean diameter of the aorta was 7,67 cm which is considered to be easily palpable. Five of the patients were referred to specialists for incidental reasons and they all failed to detect the AAA. The majority of patients with AAA's have at least 2 associated risk factors. The patients consulted their GP at least 5 times over the 24 month period. The GP's are not aware of the different modes of presentation, associated risk factors nor the value of screening for AAA's. Ten of the group of 13 GP's profess to engage in some form of Continuing Medical Education (CME).

I have suggested a few guidelines to encourage family physicians to screen for AAA in all males over the age of 60, especially if they have risk factors, such as hypertension, a current or former cigarette smoker, coronary artery disease, peripheral vascular disease and a family history of AAA. The examination should include a thorough abdominal palpation and referral for an abdominal ultrasound examination to obtain the precise diameter of the AAA as treatment depends on the size of the AAA.

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INTRODUCTION

MOTIVATION FOR THE STUDY

In September 1985, Mr. C.J. first consulted the researcher, complaining of chronic lower lumbar backache. He was duly examined him and found to have a full range of back movements and no neurological signs of a vertebral disc compression. More interest was displayed in controlling his blood pressure, which was incidentally noted to be 160/110, than paying too much attention to his backache, which the researcher thought was due to a musculo-skeletal sprain.

Over an 11-year period, Mr.C.J. was examined by at least 4 doctors in the practice, who all diagnosed his backache as muscular sprain, myalgia, sciatica, arthralgia or chronic low back pain. He was seen on 120 occasions, of which 33 consultations, ie 27 percent, were related to his backache.

Had one considered more than purely mechanical causes for this patient's backache and had one considered his general state of health in relation to his backache, the researcher may have been able to diagnose the cause of his backache earlier.

On the 11th November 1994 the patient again consulted his practitioner with the inevitable complaint of backache. He declined an examination, requested some non-steroidal anti-inflammatory drugs (NSAIDS) and referral to the physiotherapist. Four days later, Mr. C.J. rushed into the surgery, in tears, because of excruciating and continuous back pain. The doctor now only realised that there was something major amiss. He examined the patient and noted that although he was not clinically shocked, his abdomen was very tender. The patient's urinalysis revealed bilirubin 3+ and urobilinigen 1+. As he had an acute abdomen, he was immediately referred to Groote Schuur Hospital, where an abdominal x-ray was done that showed an ectatic aorta. A CT scan revealed a 6 cm infra-renal AAA. Mr.C.J. then had an emergency repair to his leaking abdominal aneurysm. Post-operatively he did well although his blood pressure still proved difficult to control.

In September 1995, the researcher noticed that Mr C.J. had absent peripheral pulses of his left lower limb. As the mechanism in 10% of peripheral emboli is from an AAA,[3] he was referred back to Groote Schuur Hospital. Regrettably he had another AAA and died post-operatively due to a disseminated intravascular coagulopathy.

As the researcher thought about this case, he reflected on the fact that 4 doctors had failed to detect the AAA over a considerable period and wondered how often doctors fail to diagnose this condition in general practice.

DEFINITION OF TERMS

AAA = Abdominal Aortic Aneurysm. The diameter of the aorta exceeds 3 cm

AAA's = plural form. Abdominal Aortic Aneurysms

Acute rupture of an AAA = classically presents with: hypotension, abdominal or back pain and an epigastric mass

A chronic or contained rupture of an AAA = This diagnosis is made on a CT Scan which usually shows bleeding into the psoas muscle, with the leak contained by the psoas sheath. Less commonly one may observe a haematoma that has become walled off or contained

A Medical Audit is defined as the development of techniques to evaluate the quality of clinical care by measuring the performance of diagnostic, therapeutic and other procedures.[4]

Angiography = radiological examination of the blood vessels after the injection of a contrasting agent

Atherosclerosis = degeneration of the arterial wall due to the formation of fatty deposits which impairs blood circulation and predisposes to thrombus formation

COAD = Chronic Obstructive Airway's Disease

CVA = Cerebro-vascular accident ie. any condition that affects the blood vessels of the brain and causes rupture of the affected blood vessels

CME = Continuing Medical Education after graduation in the form of lectures, workshops, etc

CT Scan = Computerized Tomographic Scan

DIC = Disseminated Intravascular Coagulopathy, ie, uncontrolled widespread bleeding which is invariably fatal

Ectatic = a dilated blood vessel or organ but not aneurysmal

Elective = optional or non-urgent as in elective surgery

Embolus = blood clot in an artery which has separated from the original thrombus and traveled via the blood stream to a different site

Genetics = the science of inheritance

Haematemesis = vomiting of blood

Incidence = the number of new occurrences of an event in a defined population during a stated period of observation.

IHD = Ischaemic Heart Disease

Melaena = the passage of blood in the stool

MI = Myocardial Infarction

MRI Scan = Magnetic Resonance Imaging Scan

Morbidity = the presence of an illness

Mortality = the number of deaths in a given period

NSAIDS = Non-steroidal anti-inflammatory drugs

Peripheral vascular disease = occlusion of peripheral blood vessels due to, eg,. emboli or atheroma

Prevalence = the number of instances of a given disease or other condition in a given population at a designated time

Risk factors = eg, smoking or hypertension which can result in a specific individual's increased probability of developing an illness such as AAA

Sciatica = inflammation or compression of the sciatic nerve

Screening = the process of looking for asymptomatic disease

Tertiary = an institution where specialized and superspecialized care is available

Thrombus = blood changes from its liquid state to a more solid appearance and produces a blood clot

Ultrasound = high frequency sound waves that are used to study internal organs of the body without exposing the patient to irradiation

Vascular surgeon = a surgeon that specializes in reparation of aneurysms, insertion of grafts for vasculo-occlusive disease (excluding coronary bypass surgery) and general vascular trauma

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LITERATURE REVIEW

ABDOMINAL AORTIC ANEURYSMS

DEFINITION OF AN ABDOMINAL AORTIC ANEURYSM

The definition of an AAA is based on the size of the aortic diameter which is a continuous variable. Defining a standard cut-off point is not ideal because of the varying normal diameters of the human aorta. It increases in size throughout life and is much bigger in males compared to females.[5] Thus there is no universally accepted and standard method of defining an AAA. This creates a problem for the epidemiologist because the prevalence of disease may vary in different surveys because of the differences in the criteria used for diagnosis. The definition that is currently most widely accepted and which will be used in this thesis is a permanent localized infrarenal aortic dilatation measuring 2,5 cm or more in the anterioposterior (A.P.) diameter.[6] Other definitions which are not used very often include:

i) when the maximum A.P. diameter of the infrarenal aorta is either greater than 4,0 cm or exceeded the diameter of the supra-renal aorta by at least 0,5 cm.[7]

ii) It is also defined as the largest transverse diameter in either the A.P.or lateral plane with anything above 3 cm considered as aneurysmal.[8]

SYMPTOMS OF ABDOMINAL AORTIC ANEURYSMS

Approximately 78 percent of all patients with AAA may be entirely asymptomatic at the time of initial diagnosis.[9] Thirty eight percent of the rest were discovered during the course of a physical examination, 31 percent on radiological examination and 8 percent during laparotomy. Locally at Groote Schuur Hospital about 15 percent of AAA's are picked up incidentally on routine abdominal ultrasound. (Prof. Immelman-personal communication) On routine examination the AAA may be palpated as an expansile, tender and pulsatile swelling in the epigastrium. The aneurysm may not be palpable in the obese or in the presence of a large retroperitoneal haematoma or shock. Clinical palpation is on the whole unreliable. Even in a thin person, estimation of aneurysm size is imprecise.[9] The birfurcation of the aorta lies at the level of the umbilicus; therefore, the pulsatile mass is located mostly at or above the umbilicus in the

epigastrium. This needs to be differentiated from a transmitted pulsation of the aortic node, or pancreatic tumours.

The most common presenting symptom is abdominal pain. The pain is typically constant or "throbbing" and is located in the epigastrium. Not infrequently the pain is described as penetrating through the epigastric region into the back.[1] Backache usually indicates that the AAA is on the verge of rupture or that there is a contained leak. In 50 percent of these cases the aneurysm has ruptured.[9] Backache due to erosion of the lumbar vertebrae is a very rare occurrence. Rupture of an AAA remains an important cause of sudden death and often less than 40 percent of patients reach hospital alive.[9] In most cases rupture is into the retroperitoneal space and surviving patients present with abdominal pain, tenderness and hypovolaemic shock. AAA can cause low back pain due to compression of surrounding tissues or to extension with stretching of the surrounding peritoneum or rupture of the aneurysm. Patients mainly complain of a dull, steady back pain that is unrelated to activity and that may radiate to the hips or thighs. This is usually due to an expanding, false aneurysm.[3] Occasionally pressure from the contained haematoma results in jaundice from common bile duct compression, as was the case in the index patient Mr.C.J., or in ureteral obstruction, femoral neuropathy, or extension of the haematoma into the femoral sheath, simulating a groin hernia. Unlike patients with acute rupture, most patients with chronic rupture are haemodynamically stable and have no signs or symptoms of acute blood loss.

Aorta-caval fistula or aorta-renal vein fistula usually results from rupture of an AAA into the inferior vena cava or left renal vein.[10] This occurs in less than 4 percent of cases. The typical case presents with a painful aneurysm over which a machinery bruit is audible. The patient may present with high output cardiac failure and evidence of regional venous hypertension.

Rupture into the gastro-intestinal tract usually presents with abdominal or back pain, haematemesis and melaena. Most commonly the aneurysm ruptures into the third part of the duodenum.[3] Symptoms are insidious, and a so-called herald bleed often occurs prior to massive haemorrhage. This premonitory bleeding is typically minor,

occurs intermittently, and results from erosion of the duodenal mucosa. Subsequent rupture into the intestinal lumen produces catastrophic haemorrhage and death. Rupture may also occur into the colon.[11] This is often associated with diverticular disease. The inflamed sigmoid colon adheres to the aneurysm and precipitates rupture. Typically the patient presents with bright red rectal bleeding, fever and a tender mass in the left lower quadrant. This mass is only pulsatile in 50 percent of cases so that a misdiagnosis of acute diverticulitis with perforation and pericolic abscess often results in unnecessary delay before operation. Very rarely a small aorta-enteric fistula presents with iron deficiency anaemia secondary to chronic blood loss.

About 5 percent of aneurysms can develop as a result of primary infections of the aortic wall.[3,12] These inflammatory AAA's are analogous to retro-peritoneal fibrosis. This subgroup occurs mainly in elderly males. They often present with backache and weight loss. The condition is recognised by observing a pale, white, dense fibrous tissue anteriorly over the AAA, and which may later spread to obstruct the ureters. Histologically, the sterile (non-infected) wall contains chronic inflammatory cells, eg, T and B lymphocytes and plasma cells. The cause of these inflammatory AAA's are thought to be due to an auto-immune response to lerutin. (Prof. Immelman- personal communication). Atherosclerotic aneurysms can become secondarily infected from bacterial endocarditis, septicaemia or contiguous spread from an adjacent abscess. Abdominal and back pain are reported in over 70 percent of cases. The aneurysm is usually palpable, though rarely tender, and significant weight loss is observed in 25 percent of cases. The inflammatory process often involves adjacent structures. Some degree of ureteric obstruction is observed in 25 percent of cases and may cause flank pain secondary to hydronephrosis or pyelonephritis. The finding of microscopic haematuria does not exclude rupture of an AAA.[13] Occasionally the patient presents with symptoms of frank duodenal obstruction.[3]

Mycotic aneurysms of the abdominal aorta are rare. The patients present with fever, leukocytosis and a tender pulsatile abdominal mass.[14] In 40 percent of cases, the aneurysm is not palpable and is found during an investigation of systemic sepsis of unknown origin. This aneurysm has mainly been associated with bacterial endocarditis

and patients have often presented with a history of rheumatic fever and clinical features of rheumatic heart disease. Mycotic aneurysms of the abdominal aorta are particularly prone to rupture.

Other rare manifestations of AAA are primary aorto-enteric fistula or atheroembolism (blue toe syndrome)[3] with lower limb ischaemia secondary to embolization of thrombus or atheroma from the aneurysm sac.[15] If the emboli are small, peripheral pulses may be preserved and the patient presents with the characteristic mottling of "trash foot". Mural thrombus may occlude the ostia of visceral or spinal arteries and result in an unusual presentation with colonic ischaemia or paraplegia secondary to cord infarction. Sudden onset of pain and paraesthesia in both lower limbs, accompanied by cyanotic mottling of the skin up to the level of the umbilicus, can very rarely follow acute thrombosis of the aneurysm. This is usually associated with extensive occlusive disease of the distal vessels. Femoral pulses are lost and in two-thirds of cases there is complete paralysis of both lower limbs.[16]

The differential diagnosis of AAA will include acute cholecystitis, appendicitis, acute pancreatitis, renal and diverticular pain. Testicular pain or radiation of pain to the genitals may occur, particularly on the left.[3] Other presentations are nerve root compression, swollen limb, sexual dysfunction and bladder obstruction.

The aims of treatment are to prevent rupture and death and to improve the patient's life expectancy.

PREVALENCE AND AETIOLOGY OF ABDOMINAL AORTIC ANEURYSM

The prevalence of AAA varies in different surveys because of differences in the criteria for diagnosis. It is also difficult to assess as the disease is frequently asymptomatic. Therefore, analysis of data based on clinical presentation, eg hospital admissions or surgical operations, may seriously underestimate the true prevalence of the

disease. However, the advent of diagnostic ultrasound and computed tomography makes the assessment of the true prevalence of disease in the population more feasible.

The study by Collin et al.[17] conducted in the community on a relatively large population restricted by age and sex probably provides one of the more accurate estimates, namely the prevalence of AAA (of 4 cm) in men aged 65-74 years is about 2 percent.

The incidence of rupture of AAA's is also of considerable interest, particularly for the planning of surgical services and the assessment of screening programmes. The overall incidence in the population can best be estimated by attempting to trace all ruptures in a community during a fixed period of time. Ingoldby[18] et al. did this in Swansea, Wales and found a population incidence of ruptured AAA's of 17 per 100,000 in 1983. A similar study in Stockholm, Sweden, found an incidence of 6 per 100,000.[19]

AAA's occur more commonly in males than in females. In Western Australia, a study examining all deaths and admissions due to AAA in a defined area found a male to female ration of about 4:1.[20]

Several studies have shown a marked change in prevalence with age; in Western Australia, eg, the annual incidence of deaths and admissions due to AAA increased from 60 per 100,000 men aged 55-59 years to 500 per 100,000 in those over 80 years of age.[20]

Racial differences in the occurrence of aneurysms have been studied in the United States. A comprehensive survey of AAA's detected either at autopsy or by CT in a major teaching hospital in North Carolina, found the prevalence to be twice as high in Whites as in blacks.[21] However, the prevalence in White females was similar to that in Black males and females, and so it was concluded that the White male was the group with the particularly high prevalence.

It is difficult to describe geographical variation in the prevalence of AAA, not only because of problems with the definition and measurement of disease, but because international routine statistics are not available. However, comparison of mortality statistics in England and Wales and in the United States in 1984 suggest an almost six-fold higher occurrence of disease in England and Wales compared to the United States.[22,23] But these statistics are prone to all the vagaries of mortality data. Also the age-standardized rates were not based on the same standard population, and this may have contributed to some of the differences.

Patients with coronary atherosclerosis have a 5% prevalence, and this rises to more than 10% in those with peripheral or cerebrovascular disease.[24] Accepted risk factors are advancing age,[25] pre-existing cardiac disease,[26] documented myocardial infarction and angina,[27] renal disease and hypertension, often untreated and unsupervised.[28]

Recent studies have also documented a strong genetic component to this disease.[29] First order relatives (parents, siblings, and offspring) of patients with AAA are considerably more likely to have the disease than the general population. At least 18% of patients with AAAs have a first-degree relative also affected. The mode of inheritance is multifactorial.

There has been a very apparent increase in the number of patients presenting to vascular surgeons with AAA's.[20] This may be due to an increase in the number of doctors per capita, or be related to the increasing age of the population as a whole, because the diagnosis of AAA increases sharply with age. The effect of provision of ready access to abdominal ultrasound and CT scanners may also have had some effect. Another possibility is that AAA is in itself becoming more common in affluent communities. The true incidence is increasing but the trends are not wholly compatible with advances in diagnosis and surgery; and increases have occurred in the number of complicated as well as uncomplicated cases. The admission rates have increased across the full range of severity of the disease, implying a true change in incidence and not just improved case ascertainment.

There appears to be a correlation between both hypertension and smoking and the development of aneurysms, and there is a substantial predominance of white men among the patients.[29,30] An 8:1 preponderance of AAA existed in smokers versus non-smokers. Aortic size as determined by ultrasound is greater in men who smoke and have hypertension, as compared with men without these risk factors.[31]

The infra-renal AAA is the most common aneurysm.[1] The etiology is probably not atherosclerotic but degenerative and age related. Atherosclerosis is usually secondary to circulatory disease elsewhere and is usually associated with thorasic, femoral and popliteal aneurysms. There is a reduction in elastin from 35% in the normal aorta, to 8% in the aneurysmal aorta.[29] Elastin cannot be regenerated and has a half-life = 70 years. Several biochemical abnormalities have been noted in those with AAA, including increased proteolysis. Aneurysm formation depends on several factors that interact to increase the expansile forces on the aortic wall or to decrease its ability to withstand them. Once vessel dilatation begins, regardless of the initiating factors, mural tension in the aneurysm increases dramatically, promoting further enlargement. The relationship between wall tension (T), vessel radius r, and transmural pressure (p) generally conforms to the law of La Place, in which T= pr.

THE NATURAL HISTORY OF ABDOMINAL AORTIC ANEURYSMS

Enlargement of the aorta normally occurs with ageing and also varies with gender.[32] CT measurement of the infra-renal aorta in adult men averages 2,3 cm, whereas the corresponding diameter in women is 1,9 cm.[33] Information on the natural history of AAA comes from three types of study. In patients with aneurysms discovered for the first times at necropsy, 23,4 percent of those aneurysms measuring 4,1-5,0 cm in diameter had caused death.[34] For aneurysms known to be present before death but measured at necropsy, 35,5 percent of those less than 6 cm caused death.[35] A follow-up study on a group of patients with an aneurysm less than 6 cm in diameter and rejected for surgery because the aneurysm was too small or because their general health was too poor, showed that the annual rate of rupture was the same as the mortality rate from all

other causes.[36] Patients with AAA which remain inoperable have a limited life expectancy with an estimated rupture rate of 25 percent p.a. They have a 3- year survival of 30-50 percent, and a 5-year survival of 20 percent. Szilagyi[35] demonstrated that the risk of rupture and the long-term survival of patients without operation were directly related to the size of the aneurysm. For patients with unrepaired small aneurysms, the 5-year survival was 47,8 percent, compared with only 6 percent for those with large aneurysms. Two-thirds of these patients die because of rupture. The rest die from coronary heart disease and other manifestations of atherosclerosis. The mean growth rate of aneurysms less than 6,0-cm in diameter is 0,5 cm per year.[36] The presence of an untreated small aneurysm raises the expected annual death rate by 230% for men aged 60-64 years and 38% for men aged 80-84 years.[37] Craig[38] says that the incidence of asymptomatic AAA in men aged 55-75 years is 5,4 percent, and once dilatation has occurred, the aorta can be expected to continue to expand. Rupture can, however, occur while the aneurysm is still small. After rupture, more than half the patients will die before reaching hospital. The overall survival rate has been estimated to be as low as 14 percent.

There is a significant difference in operative mortality rate between elective (4,3 percent) and emergency (36 percent) aneurysm surgery, leading to calls for increased rate of referral of patients with asymptomatic lesions. Urgent operations are usually associated with increased mortality, primarily due to the lack of time available for careful evaluation and compensation for concurrent disease states.[39] In some instances quite impressive mortality rates are attributable to pre-selection of patients on grounds of stability and fitness, rejecting the seriously ill and poor risk cases.[40] These figures cannot be compared fairly to mortality from centers where all cases come to surgery on the basis that any salvage in a condition which essentially represents 100% mortality will be an improvement.

Thirty to 50% of patients with AAA's will die because of rupture if not treated.[41] There is an 80% mortality of ruptured aneurysms compared with a 5% mortality of elective repairs.[42] Thus, it can be said that every 3 elective aneurysm repairs prevents a premature death from aneurysmal rupture. Thus on the basis of the three aforementioned

criteria, it seems that an excellent case can be made for large-scale population screening of asymptomatic AAA's.

Screening for small AAA's has also been advocated. The mean time from the diagnosis of an aneurysm to its rupture has been estimated at 18-24 months.[43] The overall incidence of rupture of an untreated asymptomatic aneurysm is approximately 25 percent per annum, rising to 50 percent per annum if the lesion is symptomatic. Referral of patients with small aneurysms may also be considered inappropriate, yet 10 percent of all ruptures occur when the dilatation is less than 5 cm.

THE DIAGNOSIS OF ABDOMINAL AORTIC ANEURYSMS

The diagnosis of an AAA can be made clinically with the reservations previously discussed. Fifty six percent of AAA have calcified walls on the lateral view of a penetrated abdominal x-ray. [30,44] As the size of an AAA is the most important predictor of the risk of rupture, newer non-invasive diagnostic imaging techniques capable of precise measurement now play an increasingly important role in operative decision making. The mainstay of diagnosis remains the use of abdominal ultrasound, which is sensitive and specific, cheap and readily available. It provides adequate information in about 85 percent of cases but presents a problem in the obese and those with distension of gas in their bowels. It is a rapid and safe procedure that does not inconvenience the patient.

Computerised Tomography (CT) is highly sensitive and specific. It is not affected by bowel gas or boney structures, as is ultrasound. It is good when there are complications, but is expensive and not always available. It visualises the renal arteries and anomalies very well. It is of value in the inflammatory aneurysm because it indicates the possibility of ureteric obstruction and a need for a detailed investigation of the urinary tract; and forewarns the surgeon of possible technical difficulties.[45] Spiral CT is a specialised procedure that facilitates the planning of reconstruction of the aorta. Magnetic resonance imaging (MRI) is equal in sensitivity to the CT scan. Not only can CT diagnose the presence of an aneurysm, but it can also demonstrate the leakage of

blood into para-aortic tissues. CT can also diagnose chronic contained rupture. Angiography is now only used in specific cases.

SCREENING IN GENERAL PRACTICE

RATIONALE FOR SCREENING

Population screening for a variety of diseases is becoming widespread. Certain basic criteria must be satisfied before embarking on a major screening programme.[4]

- The condition must be relatively common and cause sufficient mortality or morbidity for a screening programme to be justified.

AAA is predominantly a disease of men past middle age and is uncommon in women except for the very elderly.[41] It is found between 2 and 3 percent of males over the age of 65.[18] Results from other studies in the United Kingdom have been broadly similar.[17,46,47] Their collective evidence suggests that a programme to screen all apparently healthy men once, on or about their 65th birthday, will detect the majority of AAA's. Other high risk groups are male siblings of patients with AAA[48], and patients who present with other manifestations of chronic arterial disease, in whom the incidence is 10 percent.[49] They require special provision for screening irrespective of any plans to screen apparently healthy people.

An important factor which can seriously undermine the value of any screening programme is low patient compliance. A policy of involving local general practitioners and arranging examinations close to patients' homes appears to contribute significantly to a high rate of compliance,[50] and there is therefore a strong case for any future national programme to be implemented with the close involvement of local general practitioners.

- The condition must be detectable by a suitable test which is cheap, easy to apply to large numbers of the population and, above all, accurate, with minimum false negatives or false positive results.

Portable ultrasound scanners have been shown to meet these needs. The technique of examination is non-invasive, safe, free of discomfort for the patient, and readily repeatable. The equipment can be operated by a radiographer or vascular nurse in a general practitioner's surgery, or even in the patient's home, and the cost per examination is extremely low.[46]

- Suitable treatment must be available to cure or modify the outcome of the disease once it has been detected at an early or asymptomatic stage.

It has been estimated that between 30 percent and 50 percent of patients with AAA will die as a result of aneurysm rupture if the condition is not treated.[41] The 80 percent mortality of ruptured aneurysm contrast sharply with the mortality of elective repair which is reported as less than 5 percent in most centres.[42] Major surgery could be regarded as a relative drawback but for the future there is optimism that an effective endovascular alternative to conventional surgery may not be too far away. The successful evolution of endovascular procedures is pertinent to screening programmes since two thirds of aneurysms identified by this means are under 4 cm diameter.[51]

Given the uncertain aetiology of the disease and its essentially occult nature, neither attempts at prevention nor the introduction of innovative methods of treatment can be expected to have much impact on the number of deaths from this cause. The most important requirement is to detect a higher proportion of lesions before rupture when most are asymptomatic, and from the evidence presented, it is apparent that this objective is both feasible and affordable. If it is accepted that the current high mortality from AAA must be reduced, then it follows that there is a need for a national screening programme.

SCREENING METHODS

AAA has been aptly nicknamed " U-boats of the abdomen."[52] The researcher has witnessed the near catastrophic consequence of failing to detect an aneurysm and feel that there is a need to alert the public at large to these dangers and educate our fellow physicians continuously of the potential for fatal rupture. The concept of screening the

population at risk for occult AAA is slowly gaining support. If the mortality rate attributable to AAA is to be reduced, the disease must be recognised and treated before rupture occurs. Unfortunately, the majority of aneurysms remain asymptomatic until they rupture. Most aneurysms are discovered incidentally on routine physical examination or by chance from calcifications visible on abdominal x-rays. Thus, a large proportion of potentially lethal aneurysms remains undetected. This fact has fostered interest in screening high-risk populations for aneurysmal disease.[30]

A large AAA can easily be palpated abdominally, although a small AAA is often missed, especially in the obese patient. A tortuous or ectatic aorta or even an abdominal mass can feel exactly like an AAA on examination. Conventional radiography reveals the presence of an AAA in only about 56% of cases, when the dilated aortic wall is identified by a fine rim of calcium and occasionally is falsely positive.[30] In addition, measurement of size is relatively imprecise even for heavily calcified aneurysms.[44]

It is imperative to find a method that accurately identifies the presence or absence of the disease, but it should also define the extent and diameter of the aneurysm. The latter requirement is particularly important, because it is generally accepted that the risk of rupture is directly related to the degree of dilatation. [30] Arteriography defines only that portion of the lumen not filled with clot and not the extent of the aneurysmal dilatation. Thus it is unsatisfactory as a diagnostic method and as it is invasive, its use as a routine diagnostic study is precluded, especially when the index of suspicion is low.

B-mode ultrasonography and computed tomography (CT) are two methods that meet the criteria of an appropriate tool: safety, reproducibility and a high degree of accuracy. They are non-invasive and do not cause any discomfort. Although CT scanning is somewhat more accurate, ultrasonography has the advantages of not requiring contrast media, of using no ionizing radiation, and of being less expensive.[30] Many institutions are now offering a limited CT study for aneurysm diagnosis and size estimation, which decreases the radiation exposure, time and expense of the procedure.[8] Spiral computed tomographic angiography (CTA) has been advocated as the best available technique for the preoperative assessment of aortic aneurysms.[53] Its main

advantage is that it can manipulate data acquired by the scanner to provide images in multiple planes, allowing visualization of the aorta from any angle. This is essential for accurate measurement of blood vessels, which rarely run in convenient planes for conventional CT or angiography. Mural thrombus is readily distinguished from the arterial lumen when contrast is used.[44]

Magnetic resonance angiography (MRA), like spiral CTA, also provides the facility to reconstruct aortic anatomy in any plane and is thus an acceptable alternative. In a comparative study it was found to be significantly better at visualizing AAA morphology than conventional CT and colour duplex ultrasonography, but no better than arteriography.[53] No comparison has been made with spiral CTA. Magnetic resonance imaging (MRI) does tend to overestimate iliac artery stenoses owing to loss of signal from downstream turbulence and can also be hampered by artifacts caused by respiratory and bowel motion.[53] The three-dimensional imaging of MRI is superior to that of CT reconstructions.[44] MRI does not need contrast agents for excellent intravascular enhancement. MRI can quantitate and characterize blood flow patterns in AAAs. Conventional CT overestimates proximal aortic neck diameter and angiography overestimates neck length.[53]

ULTRASONOGRAPHY

Ultrasonic images of the abdominal aorta can be obtained with either a static B-mode gray-scale instrument or a real-time device. Static scans have the advantage of displaying the full course of the aorta in a single longitudinal field of view. Real-time scanning is more rapid and is well adapted to studying the abdomen in oblique planes, which may be advantageous when there is excessive bowel gas or when the aorta is tortuous.

The accuracy of ultrasound in identifying the presence or absence of an aneurysm is close to 100 percent.[30] Examinations performed at intervals of 3 months rarely varied by more than 2 mm, thereby permitting accurate determination of growth rates.

Ultrasound examinations are rendered more difficult by excess intestinal gas. Having the patient fast prior to the study will reduce this problem. Rarely, a patient is too obese to be studied. Nonetheless, in the majority of patients, a diagnosis can be made without preparation. Equipment problems, poor application techniques, inexperienced technicians and lack of skill in interpreting the scans probably account for most of the errors associated with abdominal ultrasonography.

AIMS AND OBJECTIVES

AIMS

The aim of this study is to review the records of 18 patients attending a tertiary centre for repair of their AAA, with a view to determine:

a) whether there were missed opportunities for an earlier diagnosis of AAA's.

b) whether there would be a reduction in the mortality rate if the diagnosis was made earlier.

OBJECTIVES

The objectives of this study are:

1. To determine the risk factors for AAA identified and extracted from patient records
2. To establish the reasons for consultation in 12 patients with asymptomatic AAA examined in the 24-month period before presenting with acute AAA
3. To determine the outcome of abdominal aortic surgery in this study series
4. To determine the General Practitioners' diagnosis in patients who presented with an acute ruptured AAA
5. To determine the diagnosis by specialists of patients with an asymptomatic AAA
6. To determine some characteristics of the 12 GP's in this series

LIMITATIONS

This study was limited by the following factors:

1.COMPLETENESS OF RECORDS

The records at Groote Schuur Hospital did not always have the name and or address of the referring General Practitioner and thus we were unable to use them. However, the records at the New Kingsbury Hospital was very comprehensive with full details of the referring doctor as well as the surgeon's assessment of the case and his recommendations to the GP.

The records of the majority of General Practitioners were unsatisfactory and incomplete. Only 6 of the 13 GP's recorded blood pressure and abdominal palpation readings.

2. AVAILABILITY

The majority of GP's ran well past their appointment schedules. This created a problem for the researcher as other appointments then had to be rescheduled. The records of the hospital was freely available but in 25 percent of the interviews the GP's notes were incomplete.

3. ACCESSIBILITY

At the New Kingsbury Hospital the records were accessible after consultations with the surgeons, at times that were appropriate for the researcher. The clerical staff were very helpful in accessing all the required data. The minority of GP's gave free access to their data. These were the ones where the data was adequate for purposes of the study.

METHODS

STUDY DESIGN

The Study design was a retrospective case review study. Data were hand extracted from The New Kingsbury Hospital's records. It consisted of patients who presented to the hospital from 1st January 1995 to the 31st December 1996 with a diagnosis of acute or

Eighteen patients and their referring doctors, who numbered thirteen, were identified and contacted. The response rate was 100 percent. Individual patient records from the 13 General Practitioners were assessed. Then followed recorded interviews with these General Practitioners. The interviews were then transcribed, analyzed, hand tallied and tabulated.

STUDY POPULATION AND SAMPLING

The population examined was the 13 family doctors. They referred 18 patients referred to the regional vascular clinic at the New Kingsbury Hospital with a diagnosis of acute symptomatic or ruptured AAA.

MEASUREMENTS

Information was gathered from the family doctor and hospital record regarding an arbitary two-year period before the date of acute presentation.

Initially the researcher was given permission to access the data of the vascular clinic at Groote Schuur Hospital by kind permission of Dr J. Tunnicliffe. Regrettably, the name of the referring GP was not always recorded and the majority of those whose details were available resided too far away to conduct a personal interview. Dr Peter Jefferey and partners at the New Kingsbury Hospital in Claremont then kindly consented to provide data on eighteen of their most recent cases of AAA's.

Methods to be used:

1. **Questionnaire**:

   The author devised the following questionnaire that assisted him in compiling information during the interview with the referring doctor and while extracting data from the General Practitioner's records.

PLEASE ENTER THE FOLLOWING DETAILS FROM YOUR RECORDS:

Patient's name: Mr./Mrs./Ms _____

Patient' Address: _____

Sex:_____

Date of birth:_____

Number of consultations in the last 24 months? _____

Please list the consultations

_____

_____

_____

_____

Tick yes or no if your patient has had one or more of the following

MEDICAL HISTORY	**YES**	**NO**
SMOKER		
PERIPHERAL VASCULAR DISEASE		
MYOCARDIAL INFARCTION		
HYPERTENSION		
CEREBRO VASCULAR ACCIDENT		
FAMILY HISTORY OF ABDOMINAL ANEURYSM		

SPECIAL INVESTIGATIONS		**DATE**	**RESULT**
ULTRASOUND	RENAL		
ULTRASOUND	ABDOMINAL		
X-RAY	LUMBAR- SPINE		
CT SCAN	LUMBAR SPINE		
CT SCAN	ABDOMEN		

2. Interview with the referring doctor

The author explained the nature of the study and obtained informed consent from the doctor.The interview was recorded and transcribed within 24 hours.

It is important to emphasize that this was not a qualitative study. The information that the researcher extracted from the interviews were quantifiable, based on the interviews with the General Practitioners and the patient and hospital case records. From the patient records the researcher was able to ascertain whether they had any risk factors associated with the development of AAA and whether the GP took due cognizance of these. It also indicated how many consultations the patient had with their GP over a period of 24 months during which the AAA may have been diagnosed. The researcher could also establish whether the patient had the benefit of other medical specialists opinions.

The **selection of these methods** was based on practical consideration, eg:

1. The acceptability of the procedure to the subjects
2. The absence of inconvenience, unpleasantness, or untoward consequences
3. The probability that the method would provide a good coverage, ie, would supply the required information about all or almost all members of the sample

DATA MANAGEMENT AND ANALYSIS

The interviews were transcribed within 24 hours of their being recorded. Each transcription took between 2-4 hours. Sometimes the doctor forgot to speak into the microphone which resulted in loss of data. The data was analyzed and transcribed. From these interviews the researcher could determine whether the general practitioner was aware of the various risk factors associated with AAA, whether he/she had screened patients for AAA and whether he/she correctly diagnosed the acute or ruptured AAA.

ETHICAL AND LEGAL CONSIDERATIONS

Informed consent was obtained from all the interviewees. They were assured of confidentiality and anonymity. All informants will receive acknowledgment and feedback of results. The protocol was submitted to and approved by the University of Cape Town ethics committee.

RESULTS

Eighteen patients were referred to the vascular surgeons at the New Kingsbury Hospital with a diagnosis of acute symptomatic or ruptured AAA. They were entered into the study and recruited over a 2-month period that commenced in April 1997. There were 14 males and 4 females, an expected male predominance.[20,38] The mean age was 69,44 (range 55-86) years. The mean maximum transverse diameter of the aneurysm at presentation was 7,15 cm; only three were less than 5 cm. At the time of acute presentation the size was measured directly at operation, or by ultrasonography or computed tomography.

Sixteen patients in this series had risk factors well recognized with the development of AAA.[24,29,30,54] The results are tabulated in Table I.

Table I. *Demographic and clinical characteristics of this series of 18 Patients*

No. of patients	18
Male: Female Ratio	14:4
Average age (years)	69
Risk factors: No. of patients	16
Smokers	11(61%)
Hypertension	10(55%)
Chronic Obstructive Airways Disease	5 (27%)
Peripheral vascular disease	4 (22%)
Myocardial Infarction	4 (22%)
Family history of AAA	3 (16%)
Cerebro- vascular accident	2 (11%)

The family doctor had an opportunity to see 12 of the 18 patients over a period averaging 21,58 months but still failed to diagnose AAA. (See Table II.) Thus only 34 percent of this sample of patients with acute or ruptured AAA had no contact with their general practitioner in the 24-month period before the emergency operation.

Table II. *Reasons given for the consultation in the 24-month period before acute presentation without an AAA being diagnosed*

No. of patients	12
Average diameter of AAA	7,67 cm
Time to presentation (months)	21,58
Age (mean) years	70
Male:Female ratio	10:2
Reasons for coming as a %	Routine examination 33,3 %
	Backache 25%
	Peripheral Vascular Disease 16.6 %
	Collapse 16,6 %
	Right Iliac Fossa Pain 8,3 %

The mean age of these patients was 70 (range of 58-86 years). Ten (83%) of these patients were males and 2 (17%) were females. The mean time to presentation was 21,6 months. The average transverse diameter of the aorta was 7,67 cm (range 4,5-12). It is generally accepted that aneurysms of more than 4 cm are easily palpable.

Table III. *Post-operative Outcome of Abdominal Aortic Surgery in this series of 18 Patients*

No. of patients	18
Average age (Years)	70
Acute AAA	11
Ruptured AAA	7
Outcome: Alive:Died	15:3

Seven of the 18 patients (40%) presented as an acute ruptured aortic aneurysm, of which two patients died post-operatively. (See Table III.) Of the remaining 11 patients who had an AAA, one died.

Of the 4 cases (patient numbers 1,3,9,18) that presented for the first time, only one was correctly diagnosed. One patient was a visitor from Germany who volunteered the

fact that he was diagnosed 3 years earlier, but had declined the operation without informing his next of kin.

The general practitioner failed to diagnose the acute ruptured AAA in 5 of the 7 cases (70 percent). (These results are shown in Table IV)

Table IV. *General Practitioners' Diagnosis in patients who presented with an acute Ruptured AAA*

No. of patients	7
Age (years)	65
Male: Female Ratio	6:1
Time to presentation (months)	0-24
Outcome: Alive/Dead	5:2
GP's Diagnosis	
Renal Colic	2
Ruptured AAA	2
Convulsion	1
Deep Vein Thrombosis	1
Myocardial Infarction	1

The following 4 patients waited on average approximately 2 years and 5 months for their AAA to be repaired, after the initial diagnosis. Patient number 2's condition was considered to be too risky for an operation. However, as his AAA had increased by nearly 2 cm in the space of 8 months, the risk of rupture outweighed the operative risk and he had his aneurysm repaired. Similarly, patient number 7's AAA doubled in size after 2 years and thus he was then operated on. The ninth patient in this series declined the operation until the AAA ruptured 3 years later. Patient number 14's AAA was followed up for 2 years until it had increased to 5,7cm. At this stage the risk of rupture was greater than 25 percent and thus he had his AAA repaired.

In our series of 18 patients, 5 were referred to specialists for reasons other than the diagnosis of an AAA. They too failed to detect the aneurysm. (See Table V.)

Table V. *Diagnosis by specialists of patients with an asymptomatic AAA*

No. of patients	5
Average Age(Years)	74
Sex	Males
Time to presentation (months)	0-24
Size at Diagnosis (cm)	8,6
Diagnosis	Cerebro-vascular Accident
	Orthopnoea
	Chronic Obstructive Airways Disease
	Hypertension
	Peripheral Vascular Disease

Although we did not study CME habits of the participating general practitioners and thus are unable to draw any conclusions, it is nevertheless interesting to note that 76 percent of the doctors sampled say that they attend continuing medical education lectures, albeit not regularly, due to time constraints and the demands of their families. Only 2 are aware of risk factors associated with AAA's; and 1 screens for AAA's in their practice. Six patients were correctly diagnosed as having an AAA. (These results are shown in Table VI.)

Table VI. *Characteristics of the 12 GP's in this series*

No. of GP's	13
No. of Patients	18
GP's who do Continuing Medical Education	10 (76%)
GP's aware of risk factors	2 (16%)
GP's who screen for AAA	1 (8%)
GP's who diagnosed AAA	6 (33%)

Table VII. *Analysis of 26 Patients who had emergency surgery at Groote Schuur Hospital for AAA from 3rd January 1995 – 2nd December 1996*

No. of patients	26
Male: Female Ratio	20:6
Average age (years)	62
Outcome: Alive/Dead	13:13
Race: White Males	7
Coloured Males	13
White Females	1
Coloured Females	4
Black Male	1

DISCUSSION

This small study series has confirmed the literature reports of a male preponderance in AAA's.[20.29] It is also in agreement with the literature by identifying the following associated risk factors: smoking, hypertension, chronic obstructive airways disease, peripheral vascular disease and a previous myocardial infarction. [24,26,28,29,31] Each patient in this study had at least 2 risk factors associated with AAA.

The family doctor had the opportunity to see 12 of the 18 patients, ie, 66% over a period averaging 21,58 months, but still failed to detect the AAA. All these patients were considered to have aneurysms that were easily palpable. A common problem that was identified was the inability to perform adequate abdominal examinations.

The mortality rate of 11 percent in this series compares favourably to the average of nearly 50 percent mortality in acute ruptured AAA's.[38] Of the remaining 11 patients who had an acute AAA, one died, ie, a mortality of 9 percent which is also in keeping with international standards.[42] This study was not designed to discuss these mortality figures, which probably reflect a privileged and highly selected population group receiving first class medical care. All the patients who had the benefit of an elective diagnosis of AAA survived the procedure.

In 12 of the 18 patients, the diagnosis of a ruptured AAA was undetected. Patient number 7,10 and 17 had calcification of their abdominal aorta on x-ray of the lumber spine. Althoug this sign only occurs in 56 percent of cases of AAA,[30] it does increase one's suspicion. Had the GP's at that stage investigated with an abdominal ultrasound examination, the diagnosis would have been made earlier. As this carries such a high mortality, attempts should be made to make GP's more aware of the condition.

Only 1 GP screens for AAA and 2 are aware of risk factors associated with the development of AAA. The majority of GP's in this study have no awareness of AAA's, its associated risk factors, especially family history and the necessity for screening. The

fact that only 33 percent of the GP's correctly diagnosed the AAA means that the majority are unaware of the different modes of presentation of AAA.

GUIDELINES

The researcher would like to recommend the following guidelines:

1. All adult males over the age of sixty should have an annual abdominal palpation.
2. If they have any risk factors such as hypertension, smoking, peripheral vascular disease, a previous myocardial infarction, angina or a cerebro-vascular accident they need an abdominal ultrasound examination.
3. If one's patient has had a AAA their brothers and sons need to be warned that they have an increased chance of developing an AAA and needs 5-yearly ultrasound examinations
4. Any palpable mass in the epigastrium should be ultrasonically examined.
5. Suspect a ruptured AAA in a 60 year old male who presents with abdominal and or back pain, hypotension and a pulsatile epigastric mass.

CONCLUSION

There were missed opportunities for the detection of asymptomatic AAA . They were 1. The failure of GP's to be aware and recognise well known risk factors associated with the development of AAA.

2. The lack of screening for AAA
3. A presymptomatic period of nearly 20 months was identified in the this study during which there was an opportunity to make the correct diagnosis.
4. There was a lack of diagnostic skill in recognising an acute ruptured AAA.

Replacement of the ruptured aneurysm offers the only hope of survival, a situation which has also lead others to quote from Shakespeare:

----------diseases desperate grown
By desperate appliance are reliev'd
Or not at all *(Hamlet Act iv Sc iii)*

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APPENDIX 1.

INTERVIEWS WITH DOCTORS

PATIENT NO.1: 81-year-old White female. **DOCTOR NO. 1:** Male GP aged 50. An initial consultation. New GP. She felt that previous GP had disappointed them with medical management of her late husband. Routine examination revealed a palpable 6cm AAA. I am impressed at the doctor's insistence of a clinical examination even though the patient only requested him to fill a prescription. This is very important especially as this was her first visit to the doctor. Very often doctors are just too grateful for these short consultations as they have such a heavy workload. This can lead to serious complications as can be seen in another patient. The previous GP failed to detect the patient's AAA despite her history of chronic low back pain. The current GP does not screen for AAAs and is not convinced of its benefits in the elderly. He says that he often watches the emergency medical series on television and concluded that these screening measures are of no value in the elderley. He is unaware of the genetic link in AAA. The patient's brother had surgery for AAA. The referring surgeon suggested that both the patient's sons should be referred for abdominal ultrasound examinations but this was not done. This GP attends CME lectures on a regular basis.

PATIENT NO.2: 71 year old Coloured male. **DOCTOR NO. 2:**Male GP aged 38. This colleague declined to be taped as he felt that he was inadequately prepared. Has been seeing patient on a monthly basis for over 2 years before discovering the 5cm AAA during a routine visit. During that time the patient had a CVA and was consulting a physician who also failed to detect the AAA. Risk factors were smoking, CVA and hypertension. This GP is unaware of the need to screen for AAAs and its associated risk factors. As the AAA increased in size over 8 months to 6,5cm the patient had an operation despite being considered at high risk due to his dense CVA. The doctor rarely attends CME meetings and reads the occasional journal. He also felt that he might have missed the AAA previously as he had

always examined the patient sitting in a wheelchair, whereas when he examined the patient lying in bed on another home visit he could easily palpate the AAA.

PATIENT NO. 3: White male aged 61. **DOCTOR NO.3:** Male GP aged 40. Risk factors include smoking and COAD. First consultation. Diagnosed as renal colick. Two hours later he was recalled and found patient in a shocked state. Referred to hospital. Surgeon noted that the weakly pulsatile aneurysm was palpable. GP unaware of screening methods for and risk factors associated with AAA and failed to detect the AAA that had ruptured or initially the absent peripheral pulse. Doctor does attend CME lectures.

PATIENT NO. 4. White male aged 86. **DOCTOR NO. 3:** Male GP aged 40. Patient of the practice for 3 years and saw GP 7 times. At no stage was his abdomen examined. He was referred to a physician because of orthopnoea. Six months later, the latter palpated the abdomen and found a 9cm AAA. Risk factors for this patient was hypertension, COAD and previous smoker. The GP would value guidelines for screening of AAAs.

PATIENT NO.5. White female aged 73. **DOCTOR NO.4:** Male GP aged 50. Attended the surgery for many years. No notes on any abdominal examination. Was incidentally seen on one occasion. According to the surgeon she had complained of a pulsation in the epigastrium for many months and had a palpable AAA of 6cm. Risk factors: heavy smoker; Husband had surgery for AAA 15 years ago. Thus son and daughter at risk for developing AAA. GP not aware of this and does not screen for AAA. He does not attend CME meetings. The practice has poor record keeping skills.

PATIENT NO.6: White female aged 70. **DOCTOR NO.4:** Male GP aged 50. A patient of the practice for more than 6 years during which she saw the GP on 11 occasions. Complained of lower abdominal pain and low back pain for 3 years. Abdomen was only examined once i.e. 1994 and no abnormalities were detected. While on holiday in Johannesburg her previous GP diagnosed an AAA of 4,6 cm.

Risk factors noted by surgeon (i.e. not in GP's notes): COAD and smoking. The GP has no idea of screening for AAAs and has no time for CME.

PATIENT NO.7. White Male aged 55. **DOCTOR NO.5:** Female GP. Aged 38. Foreign graduate. Recently taken over the practice. Patient had complained of lower abdominal pain for a no. of years. Treated as Peptic Ulcer with H2 antagonist and PPI's. A note in the records of alcoholic duodenitis after gastroscopy but no details of the endoscopist or path reports. Eventually an abdominal ultrasound was done which revealed an AAA of 3cm. Repeat scan 2 years later showed a doubling in size and the patient was referred for operation. Patient was never screened although he had several risk factors viz. COAD, AMI (myocardial infarct), hypertension, smoker and peripheral vascular disease. (PVD). Doctor says that in her country of origin patient records remain the property of the patient and moves with him. She regularly attends CME meetings but has no policy for screening of AAAs. She has requested a set of guidelines.

PATIENT NO.8. White male aged 60. **DOCTOR NO. 6:** Male GP aged 43 who is also a qualified pharmacist. Saw doctor a month previously for the first time, requesting an ear syringe. GP obliged, as this was "a nice quick consultation". He did not inquire into the medical background nor examined the patient. Then GP was asked to see patient at home, who had been experiencing severe back pain since the previous night which became so severe that he had collapsed. He explained that he also suffered from back pain for several months. The doctor's differential diagnoses were convulsion, spinal shock or hypoglycaemia. He was referred to hospital. The surgeon's notes were that the patient previously suffered from hypertension and now presents with hypotension, back pain and an easily palpable epigastric mass. Although this triad is diagnostic of a ruptured AAA the GP was completely unaware of the possibility and does not routinely screen for AAA in his practice. He does not attend CME meetings and was unaware of any recommendations for screening of AAAs. He would value a set of guidelines.

PATIENT NO. 9. White male aged 70. **DOCTOR NO. 7:** Male GP aged 45. Patient on vacation. Daughter phoned that dad had collapsed. Admitted to hospital. He complained of severe abdominal pain and backache and volunteered the fact that he had an AAA that was diagnosed 3 years ago but that he had refused an operation. On examination he was shocked and hypotensive, in severe pain with a huge pulsatile abdominal mass. The GP could hardly miss the diagnosis. Although more than 50% of his patients are geriatric, he does not screen for AAA as he does not see any benefit in it. He expressed the opinion that should he be diagnosed with an AAA at the age of 70, he would decline an operation. He does attend the local CME group and diagnoses about one AAA per annum.

PATIENT NO. 10. White male aged 74. **DOCTOR NO.7:** GP aged 45 (male). Patient of the practice for 5 years. He had a squamous carcinoma of the skin, right hemicolectomy for caecal carcinoma, acute myocardial infarct, COAD and a smoker. The GP felt that it was difficult to palpate his abdomen, as he was very obese. The patient consulted with a pulmonologist on a few occasions that also did not detect the AAA. The patient had complained of backache for several months, which the GP did not regard as significant. Eventually he tried a chiropractor that immediately ordered an X-ray of his lumbar sacral spine. Why did the para-medical professional regard the symptom of backache with far more suspicion? The X-ray revealed a calcified AAA and the patient was referred via the GP to the vascular surgeon who found an 8cm AAA on abdominal palpation which, on CT scan had already ruptured. This GP as well as the specialist would benefit from guidelines for assessing backache and screening for AAA especially in the light of well-recognised risk factors. This patient died during the operation.

PATIENT NO.11. Patient White male aged 65. **DOCTOR NO. 7:** .Male GP aged 45. Past medical history of hypertension, smoking, seborrhoeic warts and solar keratosis. Has attended the practice since 1984 and since then has had chronic backache. He was also once referred to a physician. He presented with sudden left abdominal pain radiating down the left flank and groin while playing golf. GP diagnosed renal colick and fortunately requested an ultrasound. This revealed an

8,5-9cm AAA. The vascular surgeon noted that the patient had a large palpable AAA that had ruptured. This repetitive picture shows that GP's are not examining the abdomen properly and that they fail to entertain a differential diagnosis. There was no record in the notes that the patient's abdomen was ever examined over the last 12 years. The GP does not know the cost of an abdominal ultrasound and does not refer any AAA under 4,5cm. He is unaware that even small AAAs can rupture.

PATIENT NO.12. White male aged 58. **DOCTOR NO.8:** Male GP aged 30. This patient has been attending the practice for 5 years. He suffers from hypertension and gout. No. of consultations per annum are 4 and abdominal examination was always recorded as normal. He presented with sudden right renal angle pain and a swollen right leg. The GP diagnosed a DVT and referred him to the vascular surgeon who diagnosed a ruptured AAA. The GP does not routinely screen for AAAs and is unaware of risk factors. He does attend CME sessions occasionally.

PATIENT NO.13. White male aged 71. **DOCTOR NO. 9:** Male GP aged 43. Attends the practice for over 5 years. Stopped smoking after his heart attack 7 years ago. Saw his cardiologist last 5 years ago but no record of any abdominal palpation. He presented with chronic backache for over 2 years. GP ordered an X-ray of his L/S spine eventually which revealed the aneurysm. Surgeon noted that the AAA was easily palpable. Patient's mother died from a ruptured AAA (notes from surgeon-GP unaware of this). Despite the surgeon's recommendations no follow up of siblings done as GP was unaware of the increased genetic risk for developing AAAs. This GP arranges the monthly CME meetings but despite the high incidence of AAAs in their area (they serve a predominantly retired community) their has never been a talk on AAAs. This is an ideal opportunity to do needs analysis for evaluating appropriate CME. The GP does not have guidelines for assessing backache.

PATIENT NO.14. White male aged 69. **DOCTOR NO. 10:** Male GP aged 57. Patient tends to wander from doctor to doctor but has been at the practice for over 14 years. He also consults his own physician annually. He has Patget's disease of

the pelvis and had a TUR for benign prostatic hypertrophy. He presented with acute cholecystitis in 1990 and had an ultrasound that revealed an AAA. He had it repaired in 1996 when it had grown to 5,6cm and a cholecystectomy. This GP has commendable CME habits. He keeps notes on various diseases, in alphabetical fashion and manually updates them after reviewing journals etc. He does one subject at a time during which he consults a textbook. Thereafter he arranges an interview with a specialist to clear up any difficulties and this practice is repeated until he feels competent in a certain field. When the patient's prostate problem was identified he probably should have had an abdominal ultrasound although there is no record of this. This investigation would have revealed the AAA earlier.

PATIENT NO.15. White male aged 74. **DOCTOR NO. 11:** Male GP aged 40. Been attending the practice for 2 years. Risk factors include hypertension, peripheral vascular disease and hypercholesterolaemia. Although his abdomen was regularly examined, no masses were palpable, as he was very obese. He presented with peripheral vascular disease and his GP referred him to the vascular surgeon who also missed the AAA! An arteriogram showed the AAA that was shown to be 12cm in size on ultrasound. The GP is not aware of risk factors and screening for AAAs. The patient's notes does not reveal any smoking cessation strategy.

PATIENT NO.16. White male aged 60. **DOCTOR NO. 11:** Male GP aged 40. 7 previous consultations. Risk factors include Peripheral Vascular Disease, Hypertension and smoking. He presented with a mass in the right iliac fossa with pain. The GP suspected an appendix mass. The surgeon diagnosed a large palpable AAA. This patient would have benefited from routine screening as he had several risk factors. CME is not high on the list of priorities of this GP.

PATIENT NO.17. White Male aged 69. **DOCTOR NO. 12:** Male GP aged 50. Patient has been attending the practice for 18 years .The present GP took over the practice 10 years ago.Patient's risk factors were hypertension and smoking. 15 years ago an X-ray of his lumbar spine revealed calcification of the abdominal aorta. Although this is not pathonomonic of an AAA it does heightens one's suspicion.

According to the GP this patient obtains his medicines from the pharmacy and very rarely consulted the GP. Three years ago absent peripheral pulses were noted and a year later the patient complained of claudicating pain. The patient was then referred to a vascular surgeon who noted that the AAA was easily palpable. This GP organises CME meetings regularly. However the intervention could have been much earlier had screening occurred. This patient died post-operatively.

PATIENT NO.18. White female aged 73. **DOCTOR NO. 13:** Male GP aged 50.This was the only telephonic interview as the GP felt the details were so sketchy that a personal interview would not be worth my while. This was a new patient who presented with chest pain and in shock. He referred her to Constantiaberg Mediclinic. The surgeon's notes was that she presented with abdominal and back pain and a low BP, i.e. classical diagnosis of a ruptured AAA. As she had no medical aid cover she was transferred to Groote Schuur Hospital. The GP was in such a hurry to save her life that he failed to make the proper diagnosis. He thinks that the delay in operating and the time it took to transfer her to another hospital may have contributed to her demise.

APPENDIX 2

DATASETS

Table I. *Demographic and clinical characteristics of this series of 18 Patients*

No.	Race & Sex	Age	HT	PVD	S	CVA	MI	COAD	AAA F.H.	R.F.
1	WF	81	+				+		+	3
2	CM	71	+		+	+				3
3	WM	61			+			+		2
4	WM	86	+					+		2
5	WF	73			+				+	2
6	WF	70			+			+		2
7	WM	55	+	+	+	+	+	+		6
8	WM	60	+							1
9	WM	70								*
10	WM	74			+		+	+		3
11	WM	65	+		+					2
12	WM	58	+							1
13	WM	72			+		+		+	3
14	WM	69								0
15	WM	74	+	+	+					3
16	WM	60	+	+	+					3
17	WM	69	+	+	+					3
18	WF	73	*							
Totals			10	4	11	2	4	5	3	

* = No data available

WF = White Female = 4

CM = Coloured Male = 1

WM = White Male = 13

HT = Hypertension

S = Smoking

AAA F.H. = Family History of AAA

R.F. = Risk Factors

PVD = Peripheral Vascular Disease

CVA = Cerebro vascular accident

MI = Myocardial Infarction

COAD = Chronic Obstructive Airways Disease

Table II. *Reasons given for the consultation in the 24-month period before acute presentation without an asymptomatic AAA being diagnosed*

No.	Age(years)	Sex	Reason for consultation	Time to presentation (months)	AAA size at Presentation (cms.)
2	71	M	Routine	24	5
4	86	M	Routine	24	9
5	73	F	Routine	24	6
6	70	F	Routine	24	4,6
8	60	M	Collapsed	1	Ruptured
10	74	M	Backache	24	8
11	65	M	Backache	24	9
12	58	M	Collapsed	24	Ruptured
13	72	M	Backache	24	4 ,5
15	74	M	P.V.D.*	24	12
16	69	M	R.I.F# Pain	18	11
17	69	M	P.V.D.*	24	-

*P.V.D. = peripheral vascular disease. # R.I.F. = right iliac fossa

Table III. *Post-operative Outcome of Abdominal Aortic Surgery in this series of 18 Patients*

No.	Age (years)	Acute AAA	Ruptured AAA	Alive	Died
1	81	+		+	
2	71	+		+	
3	61		+	+	
4	86	+		+	
5	73	+		+	
6	70	+		+	
7	55	+		+	
8	60		+	+	
9	70		+	+	
10	74		+		+
11	65		+	+	
12	58		+	+	
13	72	+		+	
14	69	+		+	
15	74	+		+	
16	69	+		+	
17	69	+			+
18	73		+		+
TOT 18	MEAN=70	11	7	15	3

Table IV. *General Practitioners Diagnosis in patients who presented with an acute Ruptured AAA*

No.	Age(years)	Sex	Time to presentation(months)	GP's diagnosis	Outcome
3	61	M	Initial consultation	Renal Colic	Alive
8	60	M	1	convulsion	Alive
9	70	M	Initial consultation	Ruptured AAA	Alive
10	74	M	24 months	Ruptured AAA	Died
11	65	M	24 months	Renal Colic	Alive
12	58	M	24 months	Deep vein Thrombosis	Alive
18	73	F	Initial consultation	Myocardial Infarction	Died

Table V. *Diagnosis by specialists of patients with an asymptomatic AAA*

No.	Age(years)	Sex	Reason for examination	Time to presentation (months)	Size in cms.at presentation
2	71	M	CVA	24	5
4	86	M	Orthopnoea	5	9
10	74	M	COAD	24	8
11	65	M	Hypertension	24	9
15	74	M	P.V.D.	1	12

Table VI. *Characteristics of the 12 General Practitioners in this series*

GP.Number.	Patient number.	CME	Aware of risk factors	Screens for AAA	Correct diagnosis
1	1	+			+
2	2	+	–	–	+
3	3	+	–	–	–
3	4	+	–	–	–
4	5	–	–	–	–
4	6	–	–	–	–
5	7	+	+	–	+
6	8	–	–	–	–
7	9	+	–	–	+
7	10	+	–	–	–
7	11	+	–	–	–
8	12	+	–	–	–
9	13	+	–	–	+
10	14	+	+	+	+
11	15	+	–	–	–
11	16	+	–	–	–
12	17	+	–	–	–
13	18	*	*	*	–
Totals: 13	18	10	2	1	6

* = Data not available

Table VII. *Analysis of 26 Patients who had emergency surgery at Groote Schuur Hospital for AAA from 3rd January 1995 – 2nd December 1996*

Patient No.	Age	Race	Outcome
1	64	1	D
2	72	4	A
3	74	3	D
4	58	3	A
5	70	3	A
6	65	3	D
7	70	1	D
8	73	1	A
9	83	4	A
10	71	3	D
11	71	3	D
12	58	3	A
13	51	3	A
14	60	1	A
15	86	3	A
16	76	1	H
17	66	1	H
18	70	3	H
19	58	3	A
20	71	4	A
21	31	3	A
22	74	7	A
23	84	2	D
24	71	4	D
25	58	3	D
26	80	1	D
TOTALS	MEAN=62	20 MALES	13 DEATHS 50 % MORTALITY

D= Died in Intensive Care Unit
A=Discharged from Hospital Alive
H=Died in hospital after having Intensive Care

1= White Male
2=White Female
3=Coloured Male
4=Coloured Female
5=Indian Male
6=Indian Female
7=African Male
8=African Female