We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

The Evidence for Management of Abdominal Aortic Aneurysms: Lessons Learned from Randomised Controlled Trials

Reza Mofidi¹ and Stuart A. Suttie²

¹*James Cook University Hospital, Middlesbrough*

²*University of Dundee, Dundee,
United Kingdom*

1. Introduction

Abdominal aortic aneurysm (AAA) is a common life threatening condition in the western world. In England and Wales alone, over 2500 patients present to hospital with rupture of AAA annually, of whom over two thirds die of their condition¹. The best treatment for AAA is elective repair of pre-symptomatic abdominal aortic aneurysms. Such a therapeutic strategy depends on effective identification of patients with AAA and the subgroup of patients in whom there is a real risk of aneurysm rupture. As the vast majority of patients with AAAs are asymptomatic, timely identification of AAA may be achieved through targeted screening of the at risk populations. Over the last two decades longitudinal studies of patients with smaller AAAs have provided insights into the timing of AAA repair and the need for and frequency of ultrasound surveillance if an expectant management strategy is followed. This chapter discusses the available evidence for screening for AAA as well as all the other measures which have helped to optimise therapeutic strategies in the management of patients with AAA throughout the patients' journey from the initial diagnosis to the eventual repair of AAA.

2. Targeted screening for AAA

In the past 40 years with the advent and generalised use of abdominal ultrasonography there has been an accurate, cheap and non invasive tool for the diagnosis of abdominal aortic aneurysms. Abdominal ultrasonography has been found to be an accurate and reproducible modality in measuring the dimensions of AAA. This has led to the concept of its use for screening of at risk populations. In the last 20 years there have been four population based randomised controlled trials which have assessed the value of targeted screening in reducing mortality from abdominal aortic aneurysms in the unselected elderly male population²⁻⁵. These trials which have been undertaken in Chichester (England)², England (MASS trial)³, Viborg County (Denmark)⁴ and the city of Perth and suburbs (Western Australia)⁵ have together recruited over 120,000 subjects. All of these studies have reported on long term (over 10 years) follow up. Using the predefined criteria set by the US Preventative Screening Task Force USPSTF⁶ the MASS trial has been classified as good with

the other three trials classified as fair i.e. not meeting all the criteria but judged to have no fatal flaws⁷.

The Chichester trial was the first to assess the value of screening for AAA in the at risk population. It was also unique as it included women as well as men. It identified all men and women aged between 65 and 80 years of age from 9 general practices in the catchment area of St Richard's hospital in Chichester between 1988 and 1991^{2,8,9}. The subjects were randomised to undergo a single screening ultrasound (US) or a control group who were followed up. AAA rupture rates, aneurysm related mortality, and overall mortality was compared between the two groups. Upon identification of AAA the therapeutic strategy for AAAs with maximum diameters between 30-44mm was once yearly surveillance US, AAAs between 44 and 59mm underwent 3 monthly ultrasound scans, whilst aneurysms greater than 60mm in diameter were considered for repair^{2,8,9}. Overall 6040 men were randomised, the authors reported a significant reduction in aneurysm related mortality which has been maintained over 15 years. However, to date this study has demonstrated no difference in the all cause mortality between the two groups. The Chichester trial has been criticized for its relative small size, a relatively high aneurysm diameter threshold for repair and including 75-80 year old patients in whom the benefits of screening are marginal. In addition 27-percent of subjects who were invited for screening refused to participate thereby diluting the benefits of screening. Despite these criticisms the Chichester study remains a land mark as it demonstrated the feasibility of US screening for AAA and its potential value and remains a blue print for other aneurysm screening studies. This study identified a low but none the less troubling rate of AAA rupture in patients who had a non aneurysmal aorta on the first screening study². A population based screening study in Gloucester demonstrated that 2.2-percent of men aged 65-73 years have a maximal aortic diameter of 2.5 to 2.9 mm and suggested that this group of patients should undergo repeat US scanning at 5 yearly intervals¹⁰.

The second RCT to study the value of population based screening for AAA was carried out in Viborg County of Denmark. In 1994 all men aged between 65 and 74 were randomised to either undergo a single screening US or the control group. In all 12639 patients were randomised^{4,11,12}. This study reported a 66-percent reduction in the aneurysm related mortality which has been maintained over 14-years. In addition they reported a 2-percent reduction in overall mortality after long term follow-up which did not reach significance⁴.

The Western Australia population based screening was a study of similar design. It randomised 41000 men between the ages of 65 and 85 years to a single US screening and a control groups. They reported no difference in aneurysm outcomes in the full study population but when the analysis was restricted to 65-74 year old men they reported a significant reduction in aneurysm related mortality after 5 years of follow-up⁵. Long term follow-up results of this study have not been published as a separate publication to date, however in a reply to a correspondence by Lederle, Norman and Lindholt did report a surprisingly high, 3-percent reduction in overall mortality in the restricted (65-74 year old) patient population after 10 years of follow-up from the Western Australia trial which was statistically significant¹³.

The MASS trial which was a population based screening RCT for men aged between 65 and 74 years of age included 4 screening centres in the United Kingdom. This study randomised 67770 patients again to single screening ultrasound or a control group and was designed to study cost effectiveness of screening in addition to reductions aneurysm related and overall mortality^{3,14,15}. This study reported a 48-percent relative risk reduction in aneurysm related

mortality as a result of screening. This benefit was present at 4 years ¹⁴ and was maintained at 10 years (Figure-1)³. There was a reduced AAA rupture rate in the patients who were invited for screening. Most of these ruptures occurred in patients who were excluded from the potential benefits of screening, such as patients who refused or did not attend screening, patients who were lost to follow-up and those who either refused or deemed not fit for surgery³. The MASS trial also reported a small rate of AAA rupture in patients who did not have an AAA on the screening scan, this rate was reported as 3 per 10,000 person years after 10 years of follow up³.

Fig. 1. Cumulative deaths related to abdominal aortic aneurysm, by time since randomisation (MASS Trial)³. From: Thompson SG, Ashton HA, Gao L, Scott RA and Multicentre Aneurysm Screening Study Group, Screening men for abdominal aortic aneurysm: 10 year mortality and cost effectiveness results from the randomised Multicentre Aneurysm Screening Study, BMJ 2009; 338: b2307.

In addition to the above RCTs a number of systematic reviews and meta-analyses have attempted to assess the value of population based screening in the medium and long term. Cosford and Leng in a Cochrane systematic review reported that there was significant evidence of reduction in aneurysm related mortality from AAA in men aged 65 to 80 years who undergo population based ultrasound screening, but no significant reduction in all cause mortality¹⁶. This review was based on the 3-5 year follow up data from the above RCTs. Subsequent to this Norman and Lindholt published a meta-analysis which showed that population based AAA screening after 7-15 years of follow up resulted in a reduction of both AAA and all cause mortality¹⁷. Their findings were contested as the reported 3-percent all cause mortality reduction was larger than what was expected by an approximately 50-percent reduction in aneurysm related mortality, bearing in mind that the mortality from AAA in the patient population is reported to be between 1.1 to 3-percent¹⁸.

Takagi et al. conducted a further meta-analysis of US screening in the male population over the age of 65years using long term 10 to 15 year follow up data from the RCTs. They reported an absolute risk reduction in aneurysm related mortality of 4 per 1000 subjects screened (Figure-2). They also revealed a strong trend towards a significant reduction in all cause mortality⁷. The latter finding was surprising for the reasons mentioned already. The authors hypothesized that screening may coincide with the asymptomatic at risk population for cardiovascular disease coming in contact with health care professionals and becoming aware of smoking risk, their blood pressure etc. The resultant reduction in cardiovascular risk factors may be in part responsible for additional reduction in all cause mortality. Such a hypothesis opens the door to the possibility of risk factor alteration and institution of secondary prevention measures such as commencement of anti-platelet agents and statin therapy during screening programmes thereby increasing the value of the screening⁷.

Fig. 2. Forrest Plot of illustrating the reduction in aneurysm related mortality (A) and the trend towards a reduction in overall mortality (B) as a result of population based screening of men between the ages of 65 and 80 years after 10 years of follow up⁷.

From: Takagi H, Goto SN, Matsui M, Manabe H, Umemoto T. A further meta-analysis of population-based screening for abdominal aortic aneurysm. J Vasc Surg. 2010; 52(4):1103-8.

Cost effectiveness of a population based screening programme is calculated by measuring the costs of ultrasound screening as well as the extra procedures and surveillance that is required for the screen identified AAA and subtracting them from the costs of treating ruptured AAA. It is expressed in cost per life year gained. As the survival advantage in terms of life year gained continues to increase with time, the cost effectiveness of screening continues to improve. A comprehensive analysis of costs of screening was performed by the MASS trial participants. They calculated the cost per life year gained to be £41,000 after 4 years¹⁴, £14,000 after 7 years ¹⁵and £7600 after 10 years³. Using the estimated life span of men aged 65 years the cost per life year gained is estimated to be in the region of £2300, which is well below the guideline figure of £25,000 which is considered acceptable for the adaptation of new medical technologies and interventions in the National Health Service of the United Kingdom¹⁹. Lindholt *et al.* also performed a comprehensive cost analysis of population based AAA screening using data obtained from the Viborg trial. They reported cost per Quality

Adjusted Life Year (QALY) gained as a result of screening to be €179 albeit with relatively wide 95% confidence intervals (€-4083 to €4682)⁴. Both of these values for costs of screening are much lower than the cost analysis carried out by the USPSTF using primarily economic modelling in 2003 and suggest that population based AAA screening in men is more cost effective than the initial assessments suggested²⁰.

The role of screening for AAA in women remains controversial. To date there is no evidence that screening for AAA in an unselected population of women is associated with a reduction even in aneurysm-related mortality. Scott and colleagues conducted the only RCT (Chichester trial) which studied the value of screening in women over the age of 65 in an unselected population (n=9342)²¹. They reported the prevalence of AAA in women to be 1.3 percent, with other authors reporting a similar rate of 0.7–1.3 percent in unselected populations^{22–24}. Scott *et al.* did not demonstrate a difference in rupture rates between the women randomized to screening and control populations of women at 5- and 10-year follow-up²¹. They concluded that screening for women was neither clinically indicated nor economically viable²¹. This study was limited by high rate of non attendance of women for AAA screening which ranged between 27 and 42-percent depending on patients age. They screened an unselected population of women without consideration of risk factors for aneurysm disease and fitness for repair; consequently a large proportion of women who were found to have an AAA did not undergo aneurysm repair²⁵. The UK Small Aneurysm Trial revealed that female sex was an independent risk factor for AAA rupture; the rupture rate in women was three times higher than that in men, despite a smaller initial AP diameter. Furthermore, mean AP diameter preceding rupture was significantly lower in women than men²⁶. A number of other authors have reported a higher growth and rupture rate of AAA in women^{27–33}. A Finnish community-based follow-up study reported that the aortic diameter was less than 5.5 cm in 24 per cent of women with a ruptured AAA, compared with only 5 per cent of men²¹. In light of these findings the 6 cm cut off value for repair of AAA in Chichester trial may have been too large to prevent aneurysm rupture in a proportion of screened women thereby reducing the value of screening in women.

For screening to be effective in reducing aneurysm-related mortality in women, it will need to be limited to high-risk women who are fit to undergo aneurysm repair²². There is increasing evidence that women with atherosclerotic disease are at significantly higher risk of developing AAA. Derubertis and colleagues²² reported that the prevalence of AAA in women with multiple (more than three) atherosclerotic risk factors was 6.4 per cent. When these findings are considered in conjunction with the increased growth rates of AAA²⁶ and higher aneurysm rupture rate in women, screening in women with multiple risk factors for AAA may become clinically and economically viable^{34–36}.

3. Optimum therapeutic strategy for small AAAs

Abdominal aortic aneurysms are treated in order to prevent rupture and the associated mortality. Aneurysm treatment has its own associated morbidity and mortality. Open surgical repair is an invasive procedure which is tolerated poorly by the subgroup of patients with multiple medical co-morbidities. Even endovascular repair cannot be accomplished without an obligatory complication rate as a result of the initial deployment of the stent graft, in addition to which a proportion of patients require secondary procedures necessary to address complications such as endoleaks, device migration and stent thrombosis requiring long term close surveillance³⁷. A small proportion of patients

who have undergone endovascular repair (EVAR) succumb to rupture. Therefore the natural history of the AAA needs to be balanced against the risk associated with treatment. Aneurysm diameter is one variable which has been consistently associated with the risk of rupture and has therefore been used to stratify patients into risk categories which decides whether US based surveillance or intervention is required to repair the aneurysm. In patients who are entered into surveillance programmes the maximum diameter of the aneurysm is used to decide on the frequency of scanning. In case of aneurysms greater than 5.5 cm there is consensus that risk of rupture mandates repair if the patient is fit to undergo the procedure. In the case of aneurysms less than 4.0 cm in diameter, most clinicians agree on a watchful waiting approach. The evidence for the optimum therapeutic strategy in the mid-sized aortic aneurysms (maximum diameter between 4.0 to 5.5 cm in diameter) has been strengthened by a number of randomised controlled trials in the last 20 years which have consolidated the modern management of AAA^{26,38-41}.

The UK small aneurysm trial (UKSAT) was a multicentre RCT which randomised 1090 patients, who were diagnosed as having an AAA with maximum AP diameter of 4.0 to 5.5cm and were deemed fit to undergo an open repair of AAA to either immediate open repair or 3 monthly ultrasound surveillance. They reported the rupture rate of these AAA in the surveillance group to be in the 1-percent per year. They did not find any significant difference in aneurysm related or all cause mortality between the two groups after a follow up period of 7 years (Figure-3)²⁶. During the follow up period over two thirds of patients who were randomised to surveillance had undergone repair of their aneurysms based on clinical grounds.²⁶ Long term follow up data from the small aneurysm trial has confirmed the initial findings of the UKSAT³⁸.

Fig. 3. Kaplan-Meier survival curves comparing survival of patients with small abdominal aortic aneurysms randomised to ultrasound surveillance and early surgery from UK small aneurysm trial²⁶. From: United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. Lancet 1998;352: 1649-55.

A number of years after the publication of the UKSAT, the Veterans Affairs Cooperative Study group published the Aneurysm Detection and Management ADAM study³⁹. This study involved screening of 126,196 veterans aged between 50 and 79 years of age for AAA with a single abdominal US. Those with AAA measuring 4.0 to 5.4 cm in diameter were offered entry to the trial. In all, 1136 subjects were randomly assigned to undergo early elective repair or ultrasound surveillance. Annualized rupture rate in the surveillance arm of the study was 0.6-percent, with no difference in aneurysm related and overall mortality between the two arms of the study ³⁹. In this study as in UKSAT the majority of patients in the surveillance arm of the study had undergone elective repair after 8 years of follow up based on clinical grounds (symptomatic aneurysm, growth to greater than 5.5cm in diameter or rapid expansion by greater than 1 cm per year) ³⁹. Completion of these two landmark trials which utilised open elective repair coincided with the advent and generalised use of endovascular repair as a primary modality treatment of AAA. This resulted in some authors questioning the validity of these landmark trials in the era of endovascular repair and suggested that as endovascular repair can be performed with significantly lower peri-procedural morbidity and mortality a policy of surveillance for smaller AAAs should be examined against endovascular repair.

To date two randomised controlled trials (PIVOTAL⁴⁰ and CAESAR⁴¹) have been conducted to compare early endovascular repair of small AAAs with ultrasound surveillance. The prerequisite for both studies was that the patients which were randomised had AAAs which were anatomically suitable for endovascular repair.

The PIVOTAL trial which was published in 2010, randomised 728 patients with AAAs measuring 40 to 50 mm in diameter to ultrasound based surveillance or early endovascular repair⁴⁰. The mean duration of follow up was 20 months (+/-12 months) they found no difference in all cause or aneurysm related mortality between the two groups ⁴⁰. At the end of the relatively short follow up duration almost one third of patients who were in the surveillance group had undergone an aneurysm repair based on clinical grounds⁴⁰. The other study of a similar design was the CAESAR trial which randomised 360 patients with AAAs measuring between 40 and 54 mm to early endovascular repair or a watchful waiting strategy. ⁴¹ After 54 months of follow up there was no significant difference in rupture rates, aneurysm related and overall mortality between the two groups (Figure-4). This study revealed that the probability of the patients in the surveillance arm of the study requiring delayed repair based on clinical grounds during the duration of follow up was 60-percent⁴¹. In addition they reported that 16.4-percent of aneurysms which upon entry into the trial were suitable for endovascular repair will be no longer suitable for EVAR after 36 months⁴¹.

A constant finding in these trials has been that a significant proportion of AAAs under ultrasonographic surveillance come to require repair within the duration of the study^{26,39}. This, taken together with the low but present annual risk of rupture has lead to differing interpretations of the results of these trials with some authors still advocating in favour of early repair of small AAA using the justification that a policy of early EVAR is as safe as a policy of US Surveillance⁴². To date there is no objective data to recommend either open or endovascular repair of smaller AAAs over a policy of watchful waiting and US surveillance. A policy of early EVAR is associated with a risk of early and delayed complications and a need for secondary procedures, thus mandating the need for close surveillance in patients who undergo early EVAR. It is therefore unlikely that there will be an economic justification for early endovascular repair.

Fig. 4. Kaplan–Meier estimates of survival at 54 months from time of randomisation in EVAR versus Surveillance groups. $P = 0.6$. Numbers at risk are shown. CAESAR trial⁴¹. From: Cao P; DeRango P, Verzini F, Parlani G et al. Comparison of surveillance vs Aortic Endografting for Small Aneurysm Repair (CAESAR) trial: results of a randomised controlled trial. *Eur J Vasc Endovasc Surg.* 2011; 41(1): 13-25.

4. Open versus endovascular repair of AAA

Ever since its inception, EVAR has offered the promise of reducing the perioperative morbidity and mortality which has been associated with open elective repair. By the end of last century, data from EVAR registries such as RETA⁴³ and EUROSTAR⁴⁴ suggested that endovascular repair, although safe was associated with an immediate complication rate in addition to events such as endoleak and device migration which mandate lifelong surveillance and in a group of patients re-intervention. As with any new or emerging technology or intervention the case for primacy of EVAR over open repair in terms of perioperative mortality rate, post operative complications and cost effectiveness needs to be made using good quality evidence. A number of trials with a similar design have been commissioned in order to compare the outcomes following EVAR and open repair of AAA in patients who are anatomically suitable to undergo endovascular repair and fit to undergo open repair. These include the Dutch Randomised Endovascular Aneurysm Management (DREAM)^{45,46} trial, EVAR-1 Trial (United Kingdom)⁴⁷, ACE trial (France)⁴⁸ and Open Versus Endovascular Repair (OVER) of abdominal aortic aneurysms trial (United States)⁴⁹. The DREAM trial which was the first to report its results enrolled 351 patients between November 2000 and December 2003 from 24 centres in the Netherlands and 4 centres in Belgium. This study focused on short term combined mortality and morbidity outcomes⁴⁵. It

reported a significantly lower operative mortality and severe complication rates in the EVAR group compared to the patients who had been randomised to open repair. At 2 years follow up aneurysm related mortality following EVAR was still significantly lower than open repair (2.1% versus 5.7%) however after 2 years of follow up there was no significant difference in the overall survival rates or freedom from moderate to severe complications between the two groups. The conclusions drawn from this trial was that there was a significant reduction in early morbidity and mortality following EVAR compared to open aneurysm repair but this difference is not sustained past 2 years^{45,46}.

EVAR-1 trial was a multicentre RCT which was conducted in 37 hospitals in the UK. It randomised 1252 patients with large AAA to either open or endovascular repair. Unlike the DREAM trial, EVAR-1 was designed to perform a comparison of long term survival, graft durability, quality of life and hospital costs associated with open repair and EVAR in addition to comparing short term mortality and morbidity between the two groups⁴⁷. They reported a significantly lower in perioperative morbidity and mortality following EVAR. Four years after randomisation, all cause mortality was similar between the two groups, although there was a persistent reduction in aneurysm related mortality in the EVAR group,(Figure-5)⁴⁷. After 12 months there was no difference in quality of life scores between the two groups with a greater number of complications and re-interventions at 4 years in the EVAR arm of the study. The hospital costs of EVAR were 25-percent higher than open repair⁴⁷.

Fig. 5. EVAR-1 Kaplan-Meier survival curves comparing aneurysm related and overall mortality between patients who have been randomised to open elective and endovascular (EVAR) repair of AAA (EVAR-1 trial)⁴⁷. From: EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. *Lancet* 2005; 365(9478): 2179-86.

The OVER trial is a RCT which included 42 Veterans Affairs medical centres in the United States. It randomised 881 patients who had AAA with a greater than 50 mm in maximal diameter, an iliac aneurysm greater than 30mm in diameter or rapid sac expansion, to elective open repair or EVAR. The preliminary results from this study indicated that the

EVAR group had significantly lower 30-day mortality as well as all cause mortality⁴⁹. After a mean follow up of 1.8 years the complication rate was not significantly different between the two groups nor was the secondary reintervention rate. As in the DREAM trial, the reintervention following EVAR was mainly due to a device related complications whereas the commonest reason for reintervention following open repair was for incisional hernia^{46,49}. Early results from the ACE trial suggest similar early mortality benefit following endovascular repair which is lost after medium term follow up⁴⁸.

Some subgroups of patients such as those who have significant co-morbidities such as cardiovascular or respiratory disease, octogenarians and women with AAA, require an individualised approach and revised criteria for the management of AAA. From its inception EVAR has provided the promise of repairing AAA in patients in whom open repair poses a high risk. Therefore armed with the knowledge that smaller AAAs are best managed by a policy of watchful waiting, EVAR appeared to be an ideal modality for the management of patients with larger AAAs which are anatomically suitable for endovascular repair, have a reasonable predicted longevity but are unfit to undergo open repair. The EVAR-2 trial was designed to answer this question. EVAR-2 trial was a randomised controlled trial of 338 patients who had an AAA with a maximum diameter of greater than 5.5cm and their aneurysm morphology was anatomically suitable for EVAR, but were medically unsuitable to undergo open repair. Primary endpoint was all-cause mortality, with secondary endpoints of aneurysm-related mortality, health-related quality of life, postoperative complications, and hospital costs⁵⁰.

The 30-day operative mortality in the EVAR group was 9.0-percent and the no intervention group had an annual rupture rate of 9.0-percent per year. Aneurysm related mortality in the patient population was 13-percent and all cause mortality after 4 years of follow up was 64-percent ⁵⁰.

Fig. 6. Kaplan-Meier curves comparing aneurysm related and overall mortality between patients who have been randomised to EVAR and no intervention group (EVAR-2 trial)⁵⁰. From: EVAR trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): randomised controlled trial. Lancet 2005; 365(9478): 2187–92.

There was no significant difference in all-cause mortality between the EVAR group and the no intervention group (hazard ratio 1.21, 95% CI 0.87–1.69). There was no difference in aneurysm-related mortality (Figure-6)⁵⁰. A policy of early endovascular repair was significantly more expensive than expectant management and was associated with a higher complication and reintervention rate. There was no difference in quality of life scores between the two arms of the study⁵⁰. Therefore the conclusion drawn by the authors was that this population of patients are best served by conservative treatment. Clearly the design of such a study provides one difficulty and that is the definition of not fit for open AAA repair is subject to clinical opinion and may be related to factors that do not affect patient's longevity. The other group of patients are those with one organ morbidity such as respiratory disease or border line medical fitness, who have a large AAA and favourable anatomy for endovascular repair. Therefore clinical judgement is exercised in the application of results of EVAR-2 trial.

5. Medical treatment of patients with AAA

In addition to risk of growth and rupture, patients with AAA are at risk from other cardiovascular events by the virtue of their age, medical co-morbidities and male preponderance of AAA. Medical management of patients with known AAA follows two parallel but different aims, reducing cardiovascular event rates perioperatively and during follow up in addition to aneurysm specific therapy which is aimed at slowing aneurysm growth and reducing the risk of rupture⁵¹⁻⁵³.

Hyperlipidaemia, a known modifiable risk factor in the development of cardio-vascular disease, can be treated with the use of drugs such as the statins (3-hydroxyl-3-methylglutaryl coenzyme A reductase inhibitors). Patients with AAA are known to be at high risk of cardio-vascular disease as well as increased risk of cardio-vascular complications following AAA repair⁵⁴. Statin therapy has been associated with improved survival due to decreased risk of cardio-vascular complications, in both open and endovascular repair⁵⁴⁻⁵⁸. Although the primary mechanism of statins is in reducing low density lipoproteins and total cholesterol levels along with increasing levels of high density lipoproteins, other protective non lipid mechanism may be at work. These so called pleiotropic effects describe a diversity of cellular events which have an effect on several components of the arterial wall, including: endothelial cells; smooth muscle cells; platelet function, monocytes and macrophages, which together help to modify the inflammatory process in the vessel wall. Statins have been shown to be beneficial in the secondary prevention of coronary heart disease even in those patients with normal lipid profiles⁵⁹⁻⁶⁰.

Matrix Metallo Proteinase-9 (MMP-9) expression is closely linked to aneurysm formation in animal models. In vitro experiments have shown that addition of Cerivstatins to human organ cultures from AAA reduces tissue levels of both total and active MMP-9 in a concentration dependent manner. Evans et al reported significantly reduced MMP-9 levels in excised tissue obtained from the aneurysm sac at the time of the aneurysm repair in patients who had been started on statins 3-weeks preoperatively compared with controls⁵⁹. Schouten et al monitored 150 patients with small AAAs for 12 months and reported a reduction in the aneurysm expansion rate in patients receiving statin therapy⁶⁰. In an observational study of 130 patients under surveillance, Sukhija reported no aneurysm expansion in 75 patients who were on statin therapy over a 2 year follow up period⁶¹. Schlosser et al in an analysis of the results of a large observational cohort study which

involved 5057 patients with vascular disease (Second Manifestation of ARterial disease (SMART) study) and included 230 patients with small AAA revealed an independent association between statin therapy and reduced aneurysm growth rate. This reduced growth and rupture rates were independent of serum lipid values^{62,63}.

Over the years there has been some interest in β -blockers, both to slow the growth rate of AAA and to reduce perioperative morbidity from cardiovascular events. The benefit was postulated partly due to their haemodynamic properties and partly due to the effect of β -blockers on matrix proteins. In a trial reported by Lindholt and colleagues the use of Propranolol did not reduce the rate of expansion of AAA, admittedly in the treatment arm of the study the compliance was poor with only 22-percent continuing on Propranolol by 2-years⁶⁴. Another trial which was carried out in Canada came to a similar conclusion owing to poor patient compliance in the treatment arm of the study⁶⁵.

In the last 15 years there has been significant interest in using peri-operative β -blockade as a means of increasing myocardial oxygen delivery thereby reducing the risk of perioperative myocardial infarction and death. Mangano et al randomised 200 patients who were undergoing major elective non-cardiac surgery to either receive Atenolol or placebo. This was started before the induction of anaesthesia. Patients with evidence of congestive cardiac failure, systolic blood pressure of less than 100mmHg or pulse rate of less than 55 beats /minute, 3rd degree heart block or broncho-spasm were excluded. This treatment was continued for 6 months postoperatively. They reported a significant reduction in cardiovascular event rate and death from cardiac causes⁶⁶.

Poldermans and colleagues performed a similar study in patients undergoing elective aneurysm or infrainguinal arterial reconstruction. They screened 1351 patients for cardiac disease using Dobutamine stress testing, 173 patients had a positive test of whom 59 were randomised to receive Bisoprolol and 53 placebo⁶⁷. They also reported a significant reduction in non fatal cardiac events as well as cardiac death. In these patients β -blockade was started at least a week in advance of the operation and they were screened for bradycardia and hypotension preoperatively⁶⁷.

POISE was a large international randomised controlled trial of the use of extended release Metoprolol in patients undergoing non-cardiac surgery, the study randomised 8351 patients to either receive Metoprolol or placebo which was started 2-4 hrs before surgery and continued for 30 days. They reported a significantly reduced risk of myocardial infarction in the Metoprolol group but at the expense of higher mortality and stroke rate in the treatment arm of the study⁶⁸. Similarly, Yang et al randomised such patients undergoing major vascular surgery, not already β -blocked, to dose adjusted Metoprolol or placebo 2 hours prior to surgery and until discharge or maximum of 5 post-operative days, and found no protective effects of β -blockade in terms of 30 day myocardial infarction and death rates⁶⁹. β -blockade did result in significantly more episodes of bradycardia and hypotension. In light of these findings the American Heart Association guidelines regarding perioperative β -blocker therapy in patients undergoing non cardiac surgery have been altered to be more cautious and circumspective (Table-)⁷⁰.

In a large observational study, Hackham et al have shown that the use of Angiotensin Converting Enzyme Inhibitor (ACE_I) therapy taken 3-12 months prior to data analysis significantly reduced the risk of rupture from AAA, independently of blood pressure⁷¹. This data was obtained from a large administrative database of 3379 patients with ruptured and 11947 with non ruptured AAA. Other anti-hypertensive medications had no such effect⁷¹. Interestingly, patients who had stopped ACE_I therapy prior to admission were more likely

to present with ruptured AAA⁷¹. The effect of ACE_i on expansion of AAA is still equivocal, with some studies demonstrating no protective effect of ACE_i therapy⁷²⁻⁷³. Thompson et al in a recent observational study of 1269 patients with small AAA who were followed up for a mean of 3.4 years, reported a significant reduction in aneurysm growth rate as a result of ACE inhibitor therapy⁷². The follow up data from UK small aneurysm trial does not support the above finding⁷⁴.

Infection with *Chlamydiae pneumoniae* has been postulated as a risk factor for AAA expansion, as the organism has been isolated from atherosclerotic plaque and the walls of AAA^{75,76}. Three small trials have aimed to elucidate the effect of the antibiotics Doxycycline and Roxithromycin in AAA growth, two of which have shown reduced aortic expansion associated with treatment^{77,78}, whilst another one by Baxter and colleagues showed no effect of doxycycline on aortic diameter⁷⁹. These three trials were limited by their small numbers. In addition administration of Doxycycline has been shown to suppress MMP-9 in both human and animal studies⁷⁹⁻⁸¹, suggesting that the reduction in aneurysm expansion rate with administration Doxycycline may be mediated through a mechanism which is independent from treatment of *Chlamydiae pneumoniae* infection.

To date there is no conclusive evidence that any medical therapy is associated with a reduction in aneurysm growth or risk of rupture. However diagnosis of AAA provides a forum for instituting appropriate secondary prevention therapies, which will reduce morbidity and mortality in the peri-operative period as well reduce long term cardiovascular risk. There is some evidence that instituting some of these treatments such as statin therapy, ACE inhibitors may well have an effect on aneurysm growth and rupture rates.

6. References

- [1] Filipovic M, Seagroatt V, Goldacre MJ. Differences between women and men in surgical treatment and case fatality rates for ruptured aortic abdominal aneurysm in England. *Br J Surg*. 2007; 94: 1096-1099.
- [2] Scott RA, Wilson NM, HA Ashton, Kay DN. Influence of screening on the incidence of ruptured abdominal aortic aneurysm: 5-year results of a randomized controlled study, *Br J Surg* 1995; 82: 1066-1070.
- [3] Thompson SG, Ashton HA, Gao L, Scott RA and Multicentre Aneurysm Screening Study Group, Screening men for abdominal aortic aneurysm: 10 year mortality and cost effectiveness results from the randomised Multicentre Aneurysm Screening Study, *BMJ* 2009; 338: b2307.
- [4] Lindholt JS, Sørensen J, Sogaard R and Henneberg EW. Long-term benefit and cost-effectiveness analysis of screening for abdominal aortic aneurysms from a randomized controlled trial, *Br J Surg* 2010; 97; 826-834.
- [5] Norman PE, Jamrozik K, Lawrence-Brown MM, Le MT *et al*. Population based randomised controlled trial on impact of screening on mortality from abdominal aortic aneurysm, *BMJ* 2004;329; 1259.
- [6] Harris RP, Helfand M, Woolf SH, Lohr KN, et al. Current methods of the US Preventive Services Task Force: a review of the process. *Am J Prev Med*. 2001;20:21-35.
- [7] Takagi H, Goto SN, Matsui M, Manabe H, Umemoto T. A further meta-analysis of population-based screening for abdominal aortic aneurysm. *J Vasc Surg*. 2010; 52:1103-8.

- [8] Scott RAP, Wilson NM, Ashton HA, Kay DN. Influence of screening on the incidence of ruptured abdominal aortic aneurysm: 5-year results of a randomised controlled study. *Br J Surg* 1995; 82: 1066-70.
- [9] Ashton HA, Gao L, Kim LG, Druce PS, et al. Fifteen-year follow-up of a randomized clinical trial of ultrasonographic screening for abdominal aortic aneurysms. *Br J Surg*. 2007;94:696-701.
- [10] Heather BP, Poskitt KR, Earnshaw JJ, Whyman M, Shaw E. Population screening reduces mortality rate from aortic aneurysm in men. *Br J Surg*. 2000;87:750-3.
- [11] Lindholt JS, Vammen S, Juul S, Henneberg EW, Fasting H. The validity of ultrasonographic scanning as screening method for abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg* 1999; 17:472-475.
- [12] Lindholt JS, Juul S, Fasting H, Henneberg EW. Screening for abdominal aortic aneurysms: single centre randomised controlled trial. *BMJ* 2005; 330:750.
- [13] Lindholt JS, Norman P. Response to comment on "Screening for abdominal aortic aneurysm and overall mortality in men". *Eur J Vasc Endovasc Surg* 2008;36:621-622.
- [14] The Multicentre Aneurysm Screening Study Group. The Multicentre Aneurysm Screening Study (MASS) into the effect of abdominal aortic aneurysm screening on mortality in men: a randomised controlled trial *Lancet* 2002; 360: 1531-39
- [15] Kim LG, P Scott RA, Ashton HA, Thompson SG. et al. A sustained mortality benefit from screening for abdominal aortic aneurysm. *Ann Intern Med*. 2007;146:699-706.
- [16] Cosford PA, Leng GC. Screening for abdominal aortic aneurysm. *Cochrane Database Syst Rev*. 2007; (2):CD002945.
- [17] Lindholt JS, Norman P. Screening for abdominal aortic aneurysm reduces overall mortality in men. *Eur J Vasc Endovasc Surg*. 2008;36:167-171.
- [18] Lederle FA. Comment on "Screening for abdominal aortic aneurysm reduces overall mortality in men.". *Eur J Vasc Endovasc Surg*. 2008;36:620-621.
- [19] Rawlins MD, Culyer AJ. National Institute for Clinical Excellence and its value judgments. *BMJ* 2004; 329:224-7.
- [20] Fleming C, Whitlock EP, Beil TL, Lederle FA. Screening for Abdominal Aortic Aneurysm: A Best Evidence Systematic Review for the U.S. *Ann Intern Med*. 2005;142:203-211.
- [21] Scott RA, Bridgewater SG, Ashton HA. Randomized clinical trial of screening for abdominal aortic aneurysm in women. *Br J Surg*. 2002; 89:283-5.
- [22] Derubertis BG, Trocciola SM, Ryer EJ, Pieracci FM, et al. Abdominal aortic aneurysm in women: prevalence, risk factors, and implications for screening. *J Vasc Surg*. 2007; 46: 630-635.
- [23] Solberg S, Singh K, Wilsgaard T, Jacobsen BK. Increased growth rate of abdominal aortic aneurysms in women. The Tromso study. *Eur J Vasc Endovasc Surg*. 2005; 29: 145-149.
- [24] Norman PE, Castleden WM, Hockey RL. Prevalence of abdominal aortic aneurysm in Western Australia. *Br J Surg*. 1991; 78: 1118-1121.
- [25] Mofidi R, Suttie SA, Howd A, Dawson AR, Griffiths GD, Stonebridge PA. Outcome from abdominal aortic aneurysms in Scotland, 1991-2006. *Br J Surg*. 2008; 95:1475-9.
- [26] United Kingdom Small Aneurysm Trial Participants. Long-term outcomes of immediate repair compared with surveillance of small abdominal aortic aneurysms. *Lancet* 1998;352: 1649-55.

- [27] Brown LC, Powell JT. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. UK Small Aneurysm Trial Participants. *Ann Surg.* 1999; 230:289-96
- [28] Heller JA, Weinberg A, Arons R, Krishnasastri KV, et al. Two decades of abdominal aortic aneurysm repair: have we made any progress? *J Vasc Surg.* 2000; 32: 1091-1100.
- [29] Dueck AD, Johnston KW, Alter D, Laupacis A, Kucey DS. Predictors of repair and effect of gender on treatment of ruptured abdominal aortic aneurysm. *J Vasc Surg.* 2004; 39:784-7.
- [30] Dimick JB, Stanley JC, Axelrod DA, Kazmers A, et al. Variation in death rate after abdominal aortic aneurysmectomy in the United States: impact of hospital volume, gender, and age. *Ann Surg.* 2002;235:579-85.
- [31] Semmens JB, Norman PE, Lawrence-Brown MM, Holman CD. Influence of gender on outcome from ruptured abdominal aortic aneurysm. *Br J Surg.* 2000;87:191-4.
- [32] McPhee JT, Hill JS, Eslami MH. The impact of gender on presentation, therapy, and mortality of abdominal aortic aneurysm in the United States, 2001-2004. *J Vasc Surg.* 2007; 45:891-9.
- [33] Mofidi R, Goldie VJ, Dawson ARW, Murie JA, Chalmers RTA. The influence of gender on the growth rate of abdominal aortic aneurysms. *Br J Surg.* 2007; 94: 310-314.
- [34] Norman PE, Semmens JB, Lawrence-Brown MM, Holman CD. Long term relative survival after surgery for abdominal aortic aneurysm in western Australia: population based study. *BMJ.* 1998; 317: 852-6.
- [35] Akkersdijk GJ, Prinssen M, Blankensteijn JD. The impact of endovascular treatment on in-hospital mortality following non-ruptured AAA repair over a decade: a population based study of 16,446 patients. *Eur J Vasc Endovasc Surg.* 2004; 28:41-6.
- [36] Wanhainen A, Lundkvist J, Bergqvist D, Bjorck M. Cost-effectiveness of screening women for abdominal aortic aneurysm. *J Vasc Surg.* 2006; 43:908-914.
- [37] Ouriel K. Treating Smaller Aneurysms: is there a rationale? Advanced Endovascular Therapy of Aortic Disease. Blackwell Publishing, London 2007: 171-180.
- [38] Powell JT, Brown LC, Forbes JF, Fowkes FG, et al. Final 12-year follow-up of surgery versus surveillance in the UK Small Aneurysm Trial. *Br J Surg.* 2007; 94:702-8.
- [39] Lederle FA, Wilson SE, Johnson GR, Reinke DB, et al. Immediate repair compared with surveillance of small abdominal aortic aneurysms. *New England Journal of Medicine* 2002;346:1437-44.
- [40] Ouriel K, Clair DG, Kent KC, Zarins CK; (PIVOTAL) Investigators. Endovascular repair compared with surveillance for patients with small abdominal aortic aneurysms. *J Vasc Surg.* 2010; 51:1081-7.
- [41] Cao P; DeRango P, Verzini F, Parlani G et al. Comparison of surveillance vs Aortic Endografting for Small Aneurysm Repair. *Eur J Vasc Endovasc Surg.* 2011; 41: 13-25.
- [42] Ouriel K, Srivastava SD, Sarac TP, O'hara PJ, et al. Disparate outcome after endovascular treatment of small versus large abdominal aortic aneurysm. *J Vasc Surg.* 2003;37:1206-12.
- [43] Thomas SM, Beard JD, Ireland M, Ayers S, et al. Results from the Prospective Registry of Endovascular Treatment of Abdominal Aortic Aneurysms (RETA): mid term results to five years. *Eur J Vasc Endovasc Surg* 2005; 29: 563-70.

- [44] Vallabhaneni SR, Harris PL. Lessons learnt from the EUROSTAR registry on endovascular repair of abdominal aortic aneurysm. *Eur J Radiol* 2001; 39: 34–41.
- [45] Blankensteijn JD, de Jong SECA, Prinssen M, van der Ham AC, et al. Two-Year Outcomes after Conventional or Endovascular Repair of Abdominal Aortic Aneurysms. *N Engl J Med* 2005;352:2398-405.
- [46] De Bruin JL, Baas AF, Buth J, Prinssen M, et al. Long-term outcome of open or endovascular repair of abdominal aortic aneurysm. *N Engl J Med*. 2010; 362:1881-9.
- [47] EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1). *Lancet* 2005; 365: 2179-86.
- [48] Becquemin JP, Pillet JC, Lescalie F, Sapoval M, et al; ACE trialists. A randomized controlled trial of endovascular aneurysm repair versus open surgery for abdominal aortic aneurysms in low- to moderate-risk patients. *J Vasc Surg*. 2011;53(5):1167-1173.
- [49] Lederle FA, Freischlag JA, Kyriakides TC, et. al. Outcome following endovascular vs open repair of abdominal aortic aneurysm: a randomised trial. *JAMA* 2009; 302: 1535-42.
- [50] EVAR trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2). *Lancet* 2005; 365: 2187–92.
- [51] Golledge J, Powell JT. Medical management of abdominal aortic aneurysm. *Eur J Vasc Endovasc Surg*. 2007; 34: 267-73.
- [52] Miyake T, Morishita R. Pharmacological treatment of abdominal aortic aneurysm. *Cardiovasc Res*. 2009; 83(3):436-43.
- [53] Baxter BT, Terrin MC, Dalman RL. Medical Management of Abdominal Aortic Aneurysm. *Circulation* 2008; 117: 1883-9.
- [54] Heart Protection Study Collaborative Group. *Lancet* 2002; 360: 7-22.
- [55] McNally MM, Agle SC, Parker FM, Bogey WM, et al. Preoperative statin therapy is associated with improved outcomes and resource utilization in patients undergoing aortic aneurysm repair. *J Vasc Surg* 2010;51:1390-6.
- [56] Kertai MD, Boersma E, Westerhout CM, van Domburg R, et al. Association between long-term statin use and mortality after successful abdominal aortic aneurysm surgery. *Am J Med* 2004; 116:96-103.
- [57] Leurs LJ, Visser P, Laheij RJ, Buth J, et al. Statin use is associated with reduced all-cause mortality after endovascular abdominal aortic aneurysm repair. *Vascular* 2006; 14; 1-8.
- [58] Kapoor AS, Kanji H, Buckingham J, Devereaux PJ, et al. Strength of evidence for perioperative use of statins to reduce cardiovascular risk: systematic review of controlled studies. *BMJ* 2006; 333:1149-1152.
- [59] Evans J, Powell JT, Schwalbe E, Loftus IM, Thompson MM. Simvastatin Attenuates the activity of matrix metalloprotease-9 in aneurysmal aortic tissue. *Eur J Vasc Endovasc Surg*. 2007; 34: 302–303.
- [60] Schouten O, van Laanen JH, Boersma E, Vidakovic R, et al. Statins are associated with a reduced infrarenal abdominal aortic aneurysm growth. *Eur J Vasc Endovasc Surg*. 2006; 32: 21–26.

- [61] Sukhija R, Aronow WS, Sandhu R, Kakar P, Babu S. Mortality and size of abdominal aortic aneurysm at long-term follow-up of patients not treated surgically and treated with and without statins. *Am J Cardiol.* 2006; 97: 279–280.
- [62] Schlösser FJ, Tangelder MJ, Verhagen HJ, van der Heijden GJ, et al. Growth predictors and prognosis of small abdominal aortic aneurysms. *J Vasc Surg.* 2008; 47:1127-1133.
- [63] EVAR-2 trial participants. Does aneurysm rupture risk decrease in patients who are anatomically suitable for endovascular repair. *Br J Surg* 2008; 95 (S1): 10
- [64] Lindholt JS, Henneberg EW, Juul S, Fasting H. Impaired results of a randomised double blinded clinical trial of propranolol versus placebo on the expansion rate of small abdominal aortic aneurysms. *Int Angiol.* 1999; 18: 52–57.
- [65] Propranolol for small abdominal aortic aneurysms: results of a randomized trial. *J Vasc Surg.* 2002; 35: 72–79.
- [66] Mangano DT, Layug EL, Wallace A, Tateo I. Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery. Multicenter Study of Perioperative Ischemia Research Group. *N Engl J Med.* 1996; 335(23):1713-20.
- [67] Poldermans D, Boersma E, Bax JJ, Thomson IR, et al. The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography Study Group. *N Engl J Med.* 1999; 341(24):1789-94.
- [68] POISE Study Group. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. *Lancet* 2008; 371(9627):1839-47.
- [69] Yang H, Raymer K, Butler R, Parlow J, et al. The effects of perioperative beta-blockade: results of the Metoprolol after Vascular Surgery study, a randomized controlled trial. *Am Heart J.* 2006; 152:983-90.
- [70] Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof E et al. ACC/AHA 2006 guideline update on perioperative cardiovascular evaluation for noncardiac surgery: focused update on perioperative beta-blocker therapy. *Circulation* 2009; 120: e169-e276.
- [71] Hackam DG, Thiruchelvam D, Redelmeier DA. Angiotensin-converting enzyme inhibitors and aortic rupture: a population-based case-control study. *Lancet.* 2006; 368: 659–665.
- [72] Thompson A, Cooper JA, Fabricius M, Humphries SE, et al. An analysis of drug modulation of abdominal aortic aneurysm growth through 25 years of surveillance. *J Vasc Surg.* 2010; 52: 55-61.
- [73] Sweeting MJ, Thompson SG, Brown LC, Greenhalgh RM, et al. Use of angiotensin converting enzyme inhibitors is associated with increased growth rate of abdominal aortic aneurysms. *J Vasc Surg.* 2010; 52:1-4.
- [74] Brady AR, Thompson SG, Fowkes FG, Greenhalgh RM, Powell JT; et al Abdominal aortic aneurysm expansion: risk factors and time intervals for surveillance. *Circulation* 2004; 110:16-21.
- [75] Lindholt JS, Ashton HA, Scott RA. Indicators of infection with *Chlamydia pneumoniae* are associated with expansion of abdominal aortic aneurysms. *J Vasc Surg.* 2001; 34: 212–215.
- [76] Nieto FJ. Infective agents and cardiovascular disease. *Semin Vasc Med.* 2002; 2: 401–415.

- [77] Mosorin M, Juvonen J, Biancari F, Satta J, et al. Use of doxycycline to decrease the growth rate of abdominal aortic aneurysms: a randomized, double-blind, placebo-controlled pilot study. *J Vasc Surg.* 2001; 34: 606–610.
- [78] Vammen S, Lindholt JS, Ostergaard L, Fasting H, Henneberg EW. Randomized double-blind controlled trial of roxithromycin for prevention of abdominal aortic aneurysm expansion. *Br J Surg.* 2001; 88: 1066–1072.
- [79] Baxter BT, Pearce WH, Waltke EA, Littooy FN, et al. Prolonged administration of doxycycline in patients with small asymptomatic abdominal aortic aneurysms: report of a prospective multicenter study. *J Vasc Surg.* 2002; 36: 1–12.
- [80] Petrinc D, Liao S, Holmes DR, Reilly JM, et al. Doxycycline inhibition of aneurysmal degeneration in an elastase-induced rat model of abdominal aortic aneurysm: preservation of aortic elastin associated with suppressed production of 92 kD gelatinase. *J Vasc Surg.* 1996; 23: 336–346.
- [81] Curci JA, Mao D, Bohnert DG, Allen BT, et al. Preoperative treatment with doxycycline reduces aortic wall expression and activation of matrix metalloproteinases in patients with abdominal aortic aneurysms. *J Vasc Surg.* 2000; 31: 325–342.

IntechOpen

Aneurysmal Disease of the Thoracic and Abdominal Aorta

Edited by Dr. Ruth Bush

ISBN 978-953-307-578-5

Hard cover, 226 pages

Publisher InTech

Published online 16, December, 2011

Published in print edition December, 2011

The first successful open surgical repair of an abdominal aortic aneurysm was in 1951 by Dubost and represented a tremendous milestone in the care of this challenging disease. The introduction of endovascular repair in 1991 by Parodi furthered the care of these patients by allowing for lower morbidity and mortality rates and also, enabling surgeons to extend surgical treatment to patients traditionally deemed too high of a surgical risk. This new book on Aortic Disease covers many interesting and vital topics necessary for both the practicing surgeon as well as a student of vascular disease. The book starts with background information on the evolution of aortic management from traditional open surgical repair to modern endovascular therapies. There is also a chapter covering the data supporting current treatment modalities and how these data have supported modern management. Also, the use of endovascular means for care of the challenging situation of ruptured aneurysms is discussed. In addition to management of abdominal aneurysm, there is a chapter on treatment of aneurysms of the ascending aorta. Along with surgical treatment, one must also understand the molecular basis for how blood vessels remodel and thus, the role of cathepsins in aortic disease is elucidated. Lastly, chapters discussing the perioperative management of radiation exposure and ultrasound-guided nerve blocks as well as the need for high-quality postoperative nutrition will lend well to a full understanding of how to management patients from presentation to hospital discharge. We hope you enjoy this book, its variety of topics, and gain a fuller knowledge of Aneurysmal Disease of the Thoracic and Abdominal Aorta.

How to reference

In order to correctly reference this scholarly work, feel free to copy and paste the following:

Reza Mofidi and Stuart A. Suttie (2011). The Evidence for Management of Abdominal Aortic Aneurysms: Lessons Learned from Randomised Controlled Trials, Aneurysmal Disease of the Thoracic and Abdominal Aorta, Dr. Ruth Bush (Ed.), ISBN: 978-953-307-578-5, InTech, Available from:
<http://www.intechopen.com/books/aneurysmal-disease-of-the-thoracic-and-abdominal-aorta/the-evidence-for-management-of-abdominal-aortic-aneurysms-lessons-learned-from-randomised-controlled>

INTECH
open science | open minds

InTech Europe

University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia

InTech China

Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元

www.intechopen.com

Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

Phone: +86-21-62489820
Fax: +86-21-62489821

IntechOpen

IntechOpen

© 2011 The Author(s). Licensee IntechOpen. This is an open access article distributed under the terms of the [Creative Commons Attribution 3.0 License](https://creativecommons.org/licenses/by/3.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

IntechOpen

IntechOpen