University of Helsinki

Institute of Clinical Medicine

Department of Surgery, Vascular Surgery

And

Department of Vascular Surgery

Helsinki University Central Hospital

Helsinki, Finland

OUTCOMES OF RUPTURED ABDOMINAL AORTIC ANEURYSM

Sani Joanna Laukontaus

Academic dissertation

Helsinki 2006

To be presented, with the assent of the Medical Faculty of the University of Helsinki, for public examination in Auditorium 1 of Meilahti Hospital, Helsinki University Central Hospital, Helsinki, Haartmaninkatu 4, on December 8th, 2006, at 12 noon.

SUPERVISED BY:

Professor Mauri Lepäntalo, MD

Department of Vascular Surgery

Helsinki University Central Hospital, Finland

Docent Ville Pettilä, MD

Department of Anaesthesiology and Intensive Care Medicine

Helsinki University Central Hospital, Finland

REVIEWED BY:

Docent Mikko Hippeläinen, MD

Department of Surgery

University of Kuopio, Finland

Docent Hannu Savolainen, MD

Swiss Cardiovascular Center

University Hospital, Bern, Switzerland

DISCUSSED WITH:

Professor Tatu Juvonen, MD

Department of Surgery

University of Oulu, Finland

To my family

CONTENTS

ABSTRACT

Background: Ruptured abdominal aortic aneurysm (RAAA) is a life-threatening event, and without operative treatment the patient will die. The overall mortality can be as high as 80–90%; thus repair of RAAA should be attempted whenever feasible. The situation should be assessed in the face of a high rate of operative mortality and postoperative quality of life. The operative mortality rate has remained unchanged over the past few decades. In addition to mortality, the quality of life (QoL) has become an increasingly important outcome measure in vascular surgery.

Aim of the study: To evaluate outcomes of RAAA and to find out predictors of mortality.

Patients and Methods: In Helsinki and Uusimaa district 626 patients were identified to have RAAA in 1996–2004. Altogether 352 of them were admitted to Helsinki University Central Hospital (HUCH). Based on data from the nationwide Finnvasc Registry, 836 RAAA patients underwent repair of RAAA in 1991–1999. First, the 30-day operative mortality was assessed in both groups, and hospital and population-based mortality also in Helsinki and Uusimaa district. Regional centralisation and improving in-hospital quality included centralisation of emergency vascular surgery, strengthening of emergency preparedness, and better availability of postoperative care in the intensive care unit. The effect of these changes on the outcome of RAAA was assessed. Secondly, QoL was evaluated by a validated, self-administrated QoL questionnaire (RAND-36) of survivors of RAAA in 1996–2000. The study was retrospective with cross-sectional QoL assessment. Thirdly, quality-adjusted life years (QALYs), which measure length and QoL, were calculated for each RAAA patient, using the EQ-5D index (EQ-5D QoL questionnaire) and estimation of life expectancy according to age- and sex-adjusted population (1996–2002). Fourth, the predictors of outcome after RAAA were assessed at admission and 48 hours after repair of RAAA (1999-2003).

Main results: The 30-day operative mortality rate was 38% in HUCH and 44% nationwide, whereas the hospital mortality was 45% in HUCH. Hospital mortality for RAAA patients over 80 years of age was 66%. Population-based mortality was 69% in 1996–2004 and 56% in 2003–2004. After organisational changes were undertaken, the mortality decreased significantly at all levels, even at the population level. Among the survivors, the QoL after RAAA was almost equal

when compared with norms of age- and sex-matched controls; only physical functioning (one out of eight domains) was slightly impaired (RAND-36). Successful repair of RAAA gave a mean of 4.1 (0–30.9) QALYs for all RAAA patients, although also non-survivors were included. The preoperative Glasgow Aneurysm Score was an independent predictor of 30-day operative mortality after RAAA with reasonable discriminatory ability (AUC 0.75 (95% CI 0.72–0.78)). It also predicted the outcome at 48- hours for initial survivors of repair of RAAA (AUC 0.67 (95% CI 0.56–0.78)). A high Glasgow Aneurysm Score and high age were associated with low numbers of QALYs to be achieved. Organ dysfunction measured by the Sequential Organ Failure Assessment (SOFA) score at 48 hours after repair of RAAA was the strongest predictor of death (AUC 0.79 (95% CI 0.70–0.89)).

Conclusion: Surgery of RAAA is a life-saving and cost-effective procedure. The centralisation of vascular emergencies improved the outcome of RAAA patients; mortality due to RAAA decreased at all measured levels. If the level of vascular or emergency services are analysed, it is mandatory to analyse the fate of all patients admitted alive to hospital, not only the patients selected for operative treatment. The survivors had a good QoL after RAAA, and successful repair of RAAA resulted in a mean 4.1 QALYs. Predictive models can be used on individual level only to provide supplementary information for clinical decision-making due to their moderate discriminatory value. These results support an active operation policy, as there is no reliable measure to predict the outcome after RAAA.

ORIGINAL ARTICLES

This thesis is based on the following original articles. The studies will be referred to in the text by their Roman numerals.

I

Korhonen SJ, Kantonen I, Pettilä V, Keränen J, Salo JA, Lepäntalo M. Long-term survival and health-related quality of life of patients with ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2003;25:350-353.

II

Laukontaus SJ, Pettilä V, Kantonen I, Salo JA, Ohinmaa A, Lepäntalo M. Utility of surgery after ruptured abdominal aortic aneurysm. Ann Vasc Surg 2006;20:42-48.

III

Korhonen SJ, Ylönen K, Biancari F, Heikkinen M, Salenius J.-P., Lepäntalo M for the Finnvasc Study Group. Glasgow Aneurysm Score as a predictor of the immediate outcome after surgery for ruptured abdominal aortic aneurysm: a Finnvasc study. Br J Surg 2004;91:1449-1452.

IV

Laukontaus SJ, Lepäntalo M, Hynninen M, Kantonen I, Pettilä V. Prediction of survival after 48-h maximal intensive care after repair for ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2005;30:509-519.

V

Laukontaus SJ, Aho P-S, Pettilä V, Albäck A, Kantonen I, Railo M, Hynninen M, Lepäntalo M. Decrease of mortality of ruptured abdominal aortic aneurysm after centralisation and in-hospital quality improvement of vascular service. Submitted.

ABBREVIATIONS AND DEFINITIONS

AAA	Abdominal aortic aneurysm
APACHE II	Acute Physiologic and Chronic Health Evaluation
AUC	Area Under the (ROC) Curve
ECG	Electrocardiograph
EQ-5D	EuroQol 5 Dimensions
EVAR	Endovascular aneurysm repair
GAS	Glasgow Aneurysm Score
ICU	Intensive care unit
POSSUM	The Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity
P-POSSUM	Portsmouth POSSUM
V-POSSUM	Vascular POSSUM
QoL	Quality of life
QALY	Quality-adjusted life year
RAAA	Ruptured abdominal aortic aneurysm
ROC curve	Receiver operating characteristic curve
SF-36	Medical Outcomes Study Short-form 36-item survey
SOFA	Sequential Organ Failure Assessment
VBHOM	Vascular Biochemistry and Haematology Outcome Models

30-day operative mortality	30-day mortality of operated RAAA patients
Operative hospital mortality	Mortality of operated RAAA patients during the same hospitalisation irrespective of its length
Hospital mortality	Mortality of all RAAA patients admitted alive to hospital irrespective of the length of hospitalisation
Overall mortality	Mortality of all RAAA patients including RAAA deaths outside hospital

INTRODUCTION

A rupture of the abdominal aortic aneurysm (AAA) is always a surgical emergency situation. AAA is often asymptomatic unless and until it ruptures. Symptoms of rupture are abdominal pain, back pain, nausea, fainting, or loss of consciousness (Bengtsson and Bergqvist 1993). Patients may have several symptoms, and various other symptoms are described as well. The purpose of the present study was to evaluate the outcomes of ruptured abdominal aortic aneurysm (RAAA), especially mortality, its predictors, and quality of life.

The most commonly used definition of AAA is aortic diameter of 3.0 cm or more (McGregor et al. 1975). Asymptomatic aneurysms are in many cases detected by abdominal ultrasonography done for some other medical reason. The prevalence of AAA was 4.9% among 65–74-year old men according to a population-based screening study, The Multicentre Aneurysm Screening Study (MASS) in the UK (Ashton et al. 2002). Based on the same study, the prevalence of large aneurysms, diameter more than 5.0 cm, was 0.6%. In men, AAA was uncommon before the age of 55 years, but its prevalence increased rapidly, reaching a peak (5.9%) at the age of 80 years (Bengtsson et al. 1992). AAA was 2–4 times more common in men than in women, in whom AAA was detected about 15 years later than in men, reaching a peak (4.5%) at age over 90 years (Bengtsson et al. 1992).

Johnston et al. (1991) classified arterial aneurysms as degenerative, inflammatory, mycotic, mechanical, traumatic, and anastomotic aneurysms, as well as aneurysms associated with congenital connective tissue disorders (Ehler-Danlos, Marfan syndrome). Degenerative AAA is the most common type with unknown etiology, whereas there are well-known risk factors for AAA. Age, smoking, male gender, and family history of AAA are reported to be independent risk factors. Hypertension, atherosclerosis, and hypercholesterolemia are controversial risk factors (Singh et al. 2001, Wanhainen et al. 2005). Singh et al. reported that smoking increased the risk of AAA 6–7 times when current smokers were compared to non-smokers (Singh et al. 2001).

Elective surgery is indicated when the risk for aneurysm rupture is clearly higher than operative mortality. The maximum diameter of the AAA is the most important indicator for the risk of rupture (Darling et al. 1977). Current accepted thresholds for elective treatment of AAA are:

AAA diameter 5.5 cm or more, growth rate over 1.0 cm in a year, or symptoms caused by AAA. Among women the threshold may be lower, and 5.0 cm diameter has been suggested, althoug clear evidence for this does not exist (Brown et al. 2003). The mortality of elective open repair of AAA is 4-5% (Heller et al. 2000, Dueck et al. 2004b, Rigberg et al. 2006).

Endovascular repair of AAA is a less invasive procedure which is used in both elective and emergency repair. The Dutch Randomized Endovascular Aneurysm Management (DREAM) and the Endovascular Aneurysm Repair (EVAR) Trial 1 compared endovascular repair with open repair of AAA (Blankensteijn et al. 2005, EVAR trial participants 2005a). Both trials reported lower 30-day mortality in the endovascular group, but after two years the survival rates for endovascular and open repair were equalized (Blankensteijn et al. 2005). Four years after randomisation, the EVAR 1 Trial demonstrated a 3% reduction in aneurysm-related mortality after endovascular repair. However, all-cause mortality was equal in both groups (EVAR trial participants 2005a). The EVAR Trial 2 compared endovascular repair with best-medical treatment in patients unfit for open repair of AAA (EVAR trial participants 2005b). After four years of follow-up the overall mortality rates were high in both groups, 66% after endovascular repair and 62% in the non-intervention group, with no statistical difference between the groups. This might be due to the fact that only patients unfit for surgery were included in the EVAR 2 Trial.

RAAA causes 1 to 2% of all deaths in industrialized countries (St Leger et al. 1996). In Finland there were 270 RAAA deaths in 2004 according to the cause of death registry of Statistics Finland. In the future the number of RAAAs will increase as the number of aged persons is growing.

REVIEW OF THE LITERATURE

TYPES OF RUPTURE

Rupture of AAA can occur in a number of ways: most frequently as a closed rupture with the formation of a retroperitoneal haematoma, a free rupture in to the intraperitoneal cavity, with the site of perforation usually in the anterior wall, or a rupture in to an adjacent organ; vena cava or duodenum (Szilagyi 1982).

The site of the aneurysm rupture varies and affects the outcome (Golledge et al. 1999). The aneurysm diameter was greatest in the middle third of the infrarenal aorta where most aneurysm ruptures occurred. In transverse dimensions, the rupture site was distributed all around the aneurysm. The site of aneurysm rupture in the transverse dimension was closely related with retroperitoneal haematoma, which occurred in 98% of the cases. The autopsy study, based on 118 non-operated RAAAs, revealed intraperitoneal rupture in 15% of the cases (Darling et al. 1977). Golledge et al. (1999) reported free intraperitoneal rupture in 22% of the cases. Gloviczki et al. (1992) reported that 69% (146/213) of the operated RAAA patients had retroperitoneal staining or haematoma, 31% had free intraperitoneal rupture, and 2% aortocaval fistula. Aortoduodenal fistula is unusual especially before aortic reconstruction (Miani et al. 1984).

Contained RAAA is uncommon (2.8–3.3%). The rupture site has most commonly been reported to be posterior, in 63% in the study of Sterpetti et al. (1990) and 85% in the study of Bonamigo et al. (2006) of the cases. Haematoma is chronically contained by the surrounding structures such as psoas muscle, and anterior and posterior renal fascia.

RISK OF RUPTURE

Incidence

The incidence of RAAA is increasing or has not changed over the past few decades (Mealy and Salman 1988, Thomas and Stewart 1988, Drott et al. 1992, Bengtsson and Bergqvist 1993, Reitsma et al. 1996, Katz et al. 1997, Choksy et al. 1999, Heller et al. 2000, Heikkinen et al.

2002). In Finland the incidence of RAAA was 6.1/100 000 (Kantonen et al. 1999b) and 6.3/100 000 in the well-defined geographic region of Pirkanmaa (Heikkinen et al. 2002). Similar incidence rates of 5.6-6.9/100 000 have been reported in the Scandinavian countries (Johansson and Swedenborg 1986, Drott et al. 1992, Bengtsson and Bergqvist 1993). Bengtsson et al. (1993) estimated that incidence would increase from 5.6/100 000 to 6.3/100 000 assuming the number of not autopsied included. The incidence rate in the UK was 8/100 000 (Basnyat et al. 1999). In the USA Heller et al. (2000) reported unchanged age- and population adjusted incidences of 6.6/100 000 to 16.3/100 000 over the past two decades, including the roughly estimated number of RAAA patients dying outside hospital. Recently Dillavou et al. (2006) reported decreased incidence of RAAA during 1994–2003 in the USA. In their series, the incidence of RAAA surgery for patients older than 65 years declined from 18.7/100 000 (1994) to 13.6/ 100 000 (2003). Endovascular repair for elderly patients who are at a high risk of rupture may be one reason for the decreased number of ruptures in the recent years. The reported RAAA incidences vary due to a multitude of data retrieval methods, sample selection, age, sex, and autopsy rate.

Men have a 2–4-fold RAAA incidence compared to women (Mealy and Salman 1988, Bengtsson and Bergqvist 1993, Reitsma et al. 1996, Katz et al. 1997, Choksy et al. 1999). In Malmö, Sweden, Swedish men had a mean incidence of 8.4/100 000 and women 3.0/100 000 (Bengtsson and Bergqvist 1993), and in Pirkanmaa, Finland, Finnish men 10.5/100 000 and women 2.7/100 000 (Heikkinen et al. 2002). Katz et al. (1997) reported for hospitalised patients over 50 years of age in the USA an incidence of 22.6/100 000 for men and 4.5/100 000 for women. Men under 50 years of age had an incidence of 0.1/100 000 (Bengtsson and Bergqvist 1993) and those under 65 years 2.1/100 000 (Heikkinen et al. 2002). Heikkinen et al. (2002) reported an incidence of 35.5/100 000 for the population aged 65 years and over and the age-specific incidence was highest in men over 80 years (138.9/100 000). Bengtsson et al. (1993) found the highest incidence in male patients older than 80 years (118/100 000). However, the largest number of RAAAs in relation to the number of deaths was observed among 70 to 80-year old men.

Growth rate and risk of rupture

The current standard for estimating the risk of rupture is the maximum AAA diameter (Darling et al. 1977). 25% of men and 15% of women may be prone to aortic dilatation (Wilmink et al. 1998). Of all AAAs, 13% will eventually rupture (Bengtsson et al. 1992). The risk of rupture increases with the increasing diameter of aneurysm. AAA grows exponentially, and the growth rate is faster in large aneurysms. Based on the results of The UK Small Aneurysm Trial, the annual rupture rate of AAA of diameter 4.0 to 5.5 cm is 1% (The UK Small Aneurysm Trial participants 1998). After the AAA diameter reaches 5.0–6.0 cm, the risk of rupture increases (Nevitt et al. 1989, Vardulaki et al. 1998, Lederle et al. 2002).

Vardulaki et al. (1998) used data collected from two prospective population-based screening studies in the UK. Within one year after AAA detection, the risk of rupture for AAA with initial diameter of 3.0 ranged from 0.2–0.4% depending on the diameter at first follow-up, for a diameter of 4.0 cm 0.8–1.1%, for 4.5 cm 1.2–2.1%, and for 5.0 cm 2.2–3.6%, respectively (Table 1). For AAA with an initial diameter <5 cm, the probability that the diameter will reach 6 cm within three years or will rupture, is very small. Based on the same study, the risk that an aneurysm of 5.5 cm would rupture within 5 years was estimated to be 31.3% (Vardulaki et al. 1998).

Lederle et al. (2002) reported results of a prospective cohort of 198 patients with large AAA (>5.5 cm) for whom elective repair was not planned due to poor medical condition or patient refusal. Within one year, if the initial diameter was 5.5–5.9 cm, the risk of rupture was 9.4%, if the diameter was 6.0-6.9 cm the risk was 10.2%, and if the diameter was >7.0 cm the risk was 32.5% (Table 1). They also reported that after secondary measurement when the AAA reached a diameter of at least 8 cm, the risk of rupture was 36.4% during the first year. For large aneurysms (>5.5 cm) the AAA growth rate was median 0.43 cm per year (interquartile range, 0.08–0.79 cm per year) (Lederle et al. 2002). The growth rate of small aneurysms was slower: with AAA diameter 3.0–3.9 cm the mean growth was 0.18 cm per year; 4.0–4.4 cm 0.23 cm; 4.5–4.9 cm 0.34 cm, respectively (Solberg et al. 2005). Although a large AAA will eventually rupture, 62% of the deaths were due to causes other than RAAA, such as cardiovascular diseases (Lederle et al. 2002). Law et al. (1994) estimated the AAA rupture risk using data from 13 different reports (Table 1). Studies concerning the risk of rupture of large aneurysms are based on autopsy reports or patients unfit for surgery studies, so the number of patients is small.

Table 1. Studies focussing on the annual risk of rupture of AAA.

| AAA diameter (cm) | Risk of rupture (%) | | |
	Law et al.*	Vardulaki et al.	Lederle et al.
<3.0	0	NA	NA
3.0–3.9	0.4	0.2	NA
4.0–4.4	1.1	0.8	NA
4.5–4.9	1.1	1.4	NA
5.0–5.4	3.3	2.5	NA
5.5–5.9	3.3	NA	9.4
6.0–6.4	9.4	NA	10.2
6.5–6.9	9.4	NA	19.1
>7.0	24.5	NA	32.5

NA = not available; * = used 1 cm discrimination

Women have a smaller aortic diameter than men (Sonesson et al. 1993). Solberg et al. (2005) recently reported an increased growth rate of AAA in women; the mean growth rate for women was 0.243 cm per year compared with 0.165 cm for men. The current threshold for elective operative treatment is nevertheless similar for both genders. Thus women are at a 3–4-fold higher risk for RAAA rupture than men (Brown and Powell 1999, Brown et al. 2003) and a lower threshold should therefore be considered (Table 2).

Table 2. The annual risk of rupture of AAA according to gender and diameter, data based on Brown et al. 2003.

AAA diameter, cm	Risk of rupture (%)	Relative risk (95% CI)
Men, 5.0-5.9	1.0	1.0
Women, 5.0–5.9	3.9	4.0 (1.2,13.0)
Men, ≥6.0	14.1	14.3 (5.9,34.5)
Women, ≥6.0	22.3	22.6 (8.4,61.1)

Risk factors

Risk factors associated with rupture in the study cohort based on The UK Small Aneurysm Trial were female sex (3.0 Adjusted Hazard Ratio (95% Confidence Interval 1.99–4.53)), larger initial aneurysm diameter (2.94 (2.49–3.48) per cm), reduced lung function (FEV_1 0.62 (0.45–0.86) per L), smoking (never smoked 0.65 (0.27–1.53)), and hypertension (1.02 (1.00–1.03) per mmHg) (Brown and Powell 1999). In addition to initial diameter risk factors affecting the expansion rate of AAA included age, smoking, and hypertension (Cronenwett et al. 1990, Chang et al. 1997, Tornwall et al. 2001). Vardulaki et al. (1998), however, did not find hypertension to increase the growth rate of AAA. A recent population-based study showed that risk factors associated with the development of large AAA were male gender, smoking, physical inactivity, and high serum total cholesterol (Lindblad et al. 2005).

Other parameters than maximum diameter have been proposed as potential predictors of AAA rupture. The AAA ruptures when the strength of the aortic wall is insufficient to sustain the stress on it. Taking into consideration both the stress and strength of the wall the identification of patients with AAA who are at highest risk for rupture could be improved (Vorp and Vande Geest 2005). Fillinger et al. (2003) demonstrated that elevated peak wall stress was superior to AAA diameter for predicting rupture risk. Peak wall stress was analysed with three-dimensional (3D) computed tomography reconstructions. Also Hall et al. (2000) demonstrated that wall stress predicted AAA rupture. They calculated the wall tension from computer tomography images using the Law of Laplace, which assumes that AAA is a symmetric cylinder. However, the actual anatomic characteristics of AAA reflected rupture risk; RAAAs were less tortuous with greater cross-sectional diameter asymmetry than electively evaluated AAAs (Fillinger et al. 2004).

A weakening of the aortic wall was associated with rupture (Di Martino et al. 2006) but little is known about the mechanism involved (Vorp and Vande Geest 2005). Thickening of the intraluminar thrombus caused local hypoxia in the aneurysm wall, and could lead to regional wall weakening (Vorp et al. 2001). An inflammatory reaction in the aortic wall causes degeneration and remodelling, and an imbalance between them favouring degeneration, which may lead to rupture (Petersen 2003). A reduction of elastin-derived peptides could possible indicate impending rupture, as could higher levels of matrix metalloproteinase MMP-9 (Petersen

et al. 2001, Petersen et al. 2002). Although all these factors have been shown to predict AAA rupture, measuring the wall stress and strength is still used only in laboratory settings.

The reason for AAA ruptures at any particular time is unknown. Studies on seasonal variation have revealed a peak in wintertime (Ballaro et al. 1998, Bown et al. 2003), and low atmospheric pressure has also been associated with rupture (Bown et al. 2003, Harkin et al. 2005).

OUTCOME OF RUPTURED ABDOMINAL AORTIC ANEURYSM

Natural outcome

The natural outcome of RAAA is death. Surgery is the only treatment that can save the life of RAAA patient. Without operative treatment, survival time is a few hours; only 2 out of 69 patients survived 6 days (Lloyd et al. 2004, Boyle et al. 2005). In very rare cases it has been possible to survive after RAAA without operative treatment (Christenson et al. 1984, Sundhagen et al. 2006). A small number of studies or case reports have demonstrated the outcomes of contained rupture (Table 3) (Christenson et al. 1984, Sterpetti et al. 1990, Defraigne et al. 2001, Dorrucci et al. 2001, Al-Koteesh et al. 2005, Bonamigo et al. 2006, Sundhagen et al. 2006). The symptoms of these patients were less acute or even missing, and operative treatment was performed weeks after the probable time of the initial symptoms in patients with contained RAAA (Sterpetti et al. 1990, Defraigne et al. 2001).

Table 3. Studies on contained RAAAs.

Authors	Year	N	Surgery (%)	Time from symptoms to surgery	Mortality (%)
Bonamigo et al.	2006	13	13 (100)	NA	1 (8)
Sundhagen et al.	2006	1	0 (0)		0
Al-Koteesh et al.	2005	1	1 (100)	Months	0
Dorrucci et al.	2001	1	1 (100)	6 weeks	0
Defraigne et al.	2001	5	5 (100)	Mean 6 weeks	1 (20)
Sterpetti et al.	1990	16	13 (100)	Mean 17 weeks	1 (6)
Christenson et al.	1984	1	0 (0)		0

NA=not available

Mortality

Overall mortality

Roughly half of the RAAA patients died before reaching hospital, and the other half underwent surgery. Half of the operated patients survived, whereas overall mortality was as high as 70-90% (Thomas and Stewart 1988, Drott et al. 1992, Bengtsson and Bergqvist 1993, Kantonen et al. 1999b, Heikkinen et al. 2002).

Hospital mortality

In assessing the outcome of surgery, the most common monitoring interval is 30-day mortality (Russell et al. 2003). The mortality figures for RAAA, however, vary considerably, depending on the patients included in a study (Callam et al. 1991). A varying number of the RAAA patients who are alive on arrival to the emergency room are selected for surgery. The mortality figures are affected by the inclusion or exclusion of the patients selected to undergo surgery, of those intended to undergo surgery but who die before its start or during it as well as of deaths outside the primary surgical unit. The 30-day mortality alone is not adequate to reflect the true surgical mortality. In-hospital mortality should include deaths also after 30 days, but before hospital discharge, and this should be viewed as hospital mortality. Stenbaek et al. (2004) used 60-day operative mortality because deaths between day 30 and 60 were related to the surgical procedure. Thus 60-day operative mortality reflected surgically related operative hospital mortality better than did crude 30-day operative mortality. An unanswered question is how to include in the analyses all these deaths during the whole hospitalisation period, including further treatment at a lower care level.

If 30-day mortality or hospital mortality include only operated patients, it should be called operative mortality. The operative mortality rate overlooks the actual hospital mortality and does not reflect the vascular service of the area. The proportion of RAAA patients who arrive alive at the hospital, but who are considered unfit for surgery, varied from 7–43% (weighted mean 24%) in numerious series (Table 4) (Hardman et al. 1996, Bradbury et al. 1997, Barry et al. 1998, Semmens et al. 1998, Adam et al. 1999, Basnyat et al. 1999, Evans et al. 2000, Kantonen et al. 1999b, Noel et al. 2001, Heikkinen et al. 2002, Neary et al. 2003, Bown et al. 2004, Dueck et al.

2004a, Tambyraja et al. 2005d). In the Finnvasc study Kantonen et al. found huge variations in the selection criteria between hospitals in Finland, resulting in the exclusion of 12–63% RAAA patients from emergency repair (Kantonen et al. 1999b). In the recent Medicare data from the USA, 98% of the patients being 65 years or older, 28 859 RAAAs were recorded, of whom 8 669 (30%) were not operated, 19 420 underwent open repair, and 770 endovascular repair (McKinsey et al. 2006). Contraindications for operative treatment were age, co-morbidities, malignancy with short life expectancy, poor general status, and refusal of surgery (Bengtsson and Bergqvist 1993, Hardman et al. 1996, Heikkinen et al. 2002). Hewin et al. (1998) reported the factors influencing the decision of the vascular surgeon not to operate some of the RAAA patients. Almost every vascular surgeon (97%) (313/323) decided not to operate on selected patients: age >80 years influenced 77% of the decisions seldom or never to operate a RAAA patient. A severe neurological disease and cardiac arrest were also influencing factors.

The weighted mean operative mortality rate for RAAA was 42% (range 29–64%) in the recent studies (Table 4) (Chen et al. 1996, Hardman et al. 1996, Rutledge et al. 1996, Bradbury et al. 1997, Koskas and Kieffer 1997, Barry et al. 1998, Dardik et al. 1998, Semmens et al. 1998, van Dongen et al. 1998, Adam et al. 1999, Basnyat et al. 1999, Kantonen et al. 1999b, Sasaki et al. 1999, Evans et al. 2000, Noel et al. 2001, Heikkinen et al. 2002, Neary et al. 2003, Roddy et al. 2003, Bown et al. 2004, Dueck et al. 2004a, Stenbaek et al. 2004, Davidovic et al. 2005, Hadjianastassiou et al. 2005, Tambyraja et al. 2005d, Visser et al. 2005, Rigberg et al. 2006). Table 4 presents the 30-day operative mortality, operative hospital, and hospital mortality rates after RAAA during the past 10 years in the series of more than 150 RAAA patients. Interestingly, the total weighted mean mortality rates of 30-day operative and operative hospital mortality did not differ, but this was due to the fact that the mortality rates were collected from different publications. Indisputable RAAA deaths have been reported between 30-day and discharge; the weighted mean from four series was 1.7%. Omitting deaths that occurs after 30 days, and the high percentage of non-operated patients with a lower mortality rate, may lower the publication threshold. There are a number of reasons for unintentional or intentional data enhancement that bias the results of AAA surgery (Campbell 1991).

Table 4. Literature review of mortality after ruptured abdominal aortic aneurysm.

Authors	Year	N	Non-operated %	30-day operative mortality %	Operative hospital mortality %	Hospital mortality %
Rigberg et al.	2006	2628	Excl.	45	46	NA
Davidovic et al.	2005	406	Excl.	48		NA
Hadjianastassiou et al.	2005	605	Excl.		47	NA
Tambyraja et al.	2005	378	21		44	56
Visser et al.	2005	5593	Excl.		41	NA
Bown et al.	2004	222	18	46	48	57
Dueck et al.	2004	3570	27	41		53
Stenbaek et al.	2004	249	Excl.	51	57*	NA
Neary et al.	2003	232	18	54		63
Roddy et al.	2003	323	Excl.	29		NA
Heikkinen et al.	2002	221	13	51		63
Noel et al.	2001	413	7	37	41	45
Evans et al.	2000	692	15		34	44
Adam et al.	1999	381	17	41		51
Basnyat et al.	1999	233	43	64		79
Kantonen et al.	1999	454	31		54	68
Sasaki et al.	1999	183	Excl.		54	NA
Barry et al.	1998	258	7		43	47
Dardik et al.	1998	527	Excl.	47		NA
Semmens et al.	1998	494	43	36		63
Van Dongen et al.	1998	309	Excl.		25	NA
Bradbury et al.	1997	632	19		35	47
Koskas et al.	1997	158	Excl.	53		NA
Chen et al.	1996	157	Excl.	46		NA
Hardman et al.	1996	175	12		39	46
Rutledge et al.	1996	1480	Excl.			55
Weighted mean (95% CI)		20973	24 (17–31)	42 (38–46)	42 (37–47)	54 (49–59)

Excl. = excluded from the study; NA = not available; * = 60-day operative mortality

Registry data are vulnerable to omissions (Lepäntalo et al. 1994, Kantonen et al. 1997). In Finland, the operative hospital mortality rate was 49% and 54%, based on the data of the vascular registry and the data of Statistics Finland, respectively (Kantonen et al. 1999b). Different mortality rates indicate that data escapes from the registries. According to the same

report, hospital mortality was 68% in 1991–1994. Reviews and reports which used data from random, stratified samples representing discharges from hospitals are not included in Table 4 (Heller et al. 2000, Hallin et al. 2001, Bown et al. 2002, Dillavou et al. 2006). Bown et al. (2002) reported an overall operative mortality rate of 48% in a meta-analysis of RAAA in 1955–1998. A gradual reduction over time, approximately 3.5% per decade, was observed. In a literature review covering the years 1985–1997 Hallin et al. (2001) reported a 30-day operative mortality rate of 47% with no improvement over the years. Similarly, Heller et al. (2000) reported an operative mortality rate of 46% on a national level in the USA, with no improvement over the years 1979–1997. A recent study from the USA over the years 1994–2003 reported a decrease in the incidence of RAAA surgery, but 30-day operative mortality remained unchanged (Dillavou et al. 2006). The extensive Medicare data also from the USA revealed an operative mortality of 54% for open repair of RAAA, of 61% for endovascular repair, and a hospital mortality of 68% including RAAA patients unfit for surgery (McKinsey et al. 2006).

Mortality in the postoperative year is an important measure of outcome (Rigberg et al. 2006). In a large group of RAAA patients (n=2628) the 30-day operative mortality was 45.1%, hospital mortality 45.7%, and 1-year mortality 53.5%. The mortality was 8.4% between days 31 to 365 after repair of RAAA. Dueck et al. (2004b) reported a 30-day operative mortality of 40% and a 1-year mortality of 48% after repair of RAAA. Mortality remained unchanged over time (1993–1999). These studies on mortality during the first year after RAAA did not describe the discharge status of the patients, although the proportion of patients discharged home, to nursing homes and further institutional care would be of great interest.

Volume outcome associations

The type of hospital influenced mortality. High-volume hospitals had lower mortality rates than low-volume hospitals (Birkmeyer et al. 2003). Comparison of the reports is difficult because the definition of hospital volume differs. Rigberg et al. (2006) demonstrated an increased operative mortality associated with low-volume hospitals for patients with RAAA. Low-volume was defined as <16 cases/year (high-volume >32.5 cases/year, medium-volume between these two). Dimick et al. (2002) used the term high-volume hospital when the number of elective AAA procedures was more than 30 per year, and found lower operative mortality among these hospitals. Heller et al. (2000) reported hospital bed number <500 as a predictor for operative

mortality after RAAA based on a large database. Rutledge et al. (1996) found better survival after surgery in hospitals with a bed number >100. On the contrary, Visser et al. (2005) reported better survival in hospitals with <400 beds or in university hospitals, in the Netherlands, but the number of patients unfit for surgery was not available. Based on an other study in the Netherlands, 34% of admitted RAAA patients did not undergo surgery in 1992 (Reitsma et al. 1996).

Some studies have shown that hospital volume has no effect on mortality, but that high-volume surgeons or vascular surgeons had better outcome rates than general surgeons (Ouriel et al. 1990, Dardik et al. 1998, Dueck et al. 2004c). Basnyat et al. (1999) found operative mortality to be similar between vascular surgeons and non-vascular surgeons, but vascular surgeons turned down significantly fewer patients.

In Finland high-volume hospitals (>15 elective AAA procedures per year) had lower overall hospital mortality than low-volume hospitals, indicating that more RAAA patients were defined as unfit for surgery in low-volume hospitals (Kantonen et al. 1999b). Consequently, operative mortality did not differ between high-volume and low-volume hospitals. This clearly demonstrated that there is bias related to operative mortality, due to the different rates of omission.

The risk related to transportation has been debated due to the trend towards specialisation and centralisation in vascular surgery. The question has been whether to transport the patient to the nearest surgical unit or to a vascular centre. Dueck et al. (2004c) reported that distance was not associated with mortality after RAAA. Similar results were reported in Finland (Kantonen et al. 1999a). Adam et al. (1999) examined RAAA patient transferral between hospitals. Transferring patients from other units to a vascular centre did not affect mortality either. They nevertheless reported a very low operative rate of 6% for those not transferred.

Long-term survival

Long-term survival is as important measure of outcome as hospital mortality, but it is infrequently reported. The population screening for AAA is increasing, and endovascular repair for RAAA is deemed an acceptable treatment (Greco et al. 2006, Peppelenbosch et al. 2006).

Long-term survival after any given type of repair of RAAA is especially important to allow comparison between different treatment methods.

Data on long-term survival of survivors after RAAA have caused controversy. The long-term survival of patients after RAAA has been compared with that of a group treated with elective repair, and with an age- and sex-adjusted population. Dueck et al. (2004b) showed no difference on long-term survival for 30-day survivors after RAAA compared to a group of elective repair of AAA in a large study (n=2280). Similar results have been reported previously (Rohrer et al. 1988, Stonebridge et al. 1993, Soisalon-Soininen et al. 1995). Contradictory results with worse long-term survival have been reported by others (Johnston 1994b, Cho et al. 1998). Cho et al. (1998) reported cumulative survival rates of 86%, 64%, and 33% after successful repair of RAAA at 1, 5, and 10 years, respectively. After elective repair, the survival rates at the same intervals were 97%, 74%, and 43%, respectively, and the survival rates of the general population were 95%, 75%, and 52%, respectively. Based on the previous reports, the range of crude five-year survival after RAAA is 40–60% (Rohrer et al. 1988, Soisalon-Soininen et al. 1995, Cho et al. 1998, Evans et al. 1999, Dueck et al. 2004b).

Several studies have reported long-term survival after repair of RAAA equal to that of the age- and sex-adjusted population (Rohrer et al. 1988, Stonebridge et al. 1993, Hinterseher et al. 2004). Others have reported poorer outcomes in similar comparisons (Cho et al. 1998, van Dongen et al. 1998).

The life expectancy of elderly patients after RAAA was similar to that of an age- and sex-adjusted control population in previous studies (Evans et al. 1999, Aune et al. 2004, Stenbaek et al. 2004). Norman et al. reported even better survival for over 80-year-olds than expected life expectancy, although this group included elective AAA patients (Norman et al. 1998). Van Dongen et al. (1998) found worse long-term survival for RAAA patients older than 70 years compared to the general population. For patients age over 80 years, 1-year mortality was >70%; this was worse compared to the general population in the age group of 81–90 years (n=514) but better in the age group of 91–100 (n=59) (Rigberg et al. 2006).

The influence of gender is also ambiguous. Women displayed worse long-term survival than either the elective surgery group or the age- and sex-adjusted population (Norman et al. 1998, Stenbaek et al. 2004). After five years 30% of women were alive compared to 50% of men after

repair of RAAA (Norman et al. 1998). The reason for the worse outcome is not clear, but the main cause of death was related to atherosclerotic diseases after discharge (Norman et al. 1998). On the contrary, gender did not influence long-term survival (Johnston 1994a, Evans et al. 2000).

It has been suggested that the relative survival ratio (Norman et al. 1998) or standardised mortality ratio (Stenbaek et al. 2004) should be used to allow a proper comparison. In general, women have a longer life expectancy than men, but after RAAA this advantage disappears. The 5-year relative mortality after repair of RAAA was 1.04 for men and 1.92 for women (Norman et al. 1998). Stenbaek et al. (2004) reported a 1.96 standardised mortality ratio for RAAA patients compared with the normal population, and Van Dongen et al. (1998) reported a standardised mortality ratio of 2.1 (95% CI 1.7–2.5) after RAAA.

Quality of life

The outcome of surgery is often measured by mortality and morbidity, but these measures fail to take the patient's perspective of outcome into account. QoL assessment has become increasingly important in many fields of medicine, also in vascular surgery. Health-related QoL measures are divided into two groups: generic and disease-specific measures. Generic measures are designed for any disease or health problem in any population sample, while disease-specific measures focus on aspects of health that are important to specific health problems (Patrick and Deyo 1989).

A few studies have focused on QoL assessment after RAAA (Rohrer et al. 1988, Currie et al. 1992, Magee et al. 1992, Hennessy et al. 1998, Bohmer et al. 1999, Joseph et al. 2002, Piper et al. 2003, Hinterseher et al. 2004, Tambyraja et al. 2005b). The measures used were generic, and not necessarily validated, and the number of patients was in general small. These studies provided results that were mainly equal with those of the age- and sex-adjusted general population or patients undergoing elective repair of AAA (Table 5).

Self-designed questionnaires were used to assess QoL (Currie et al. 1992, Piper et al. 2003). Currie et al. (1992) designed a QoL questionnaire comprising three parts. The questionnaire was completed in the patient's home by the interviewer, and the results of rupture and elective AAA

patients were compared with age- and sex- matched control subjects chosen by the survivors' general practitioner. The QoL of the patients over 80-years old was comparable to that of the control subjects, but the sample consisted of only seven patients. Piper et al. (2003) asked survivors after RAAA to rate their recovery compared to their preoperative status, and assigned a quality score of one to four describing coping with daily routines. After a median of 45 months follow-up time, the RAAA patients reported their QoL to be equal to the preoperative status (n=39).

One of the earliest QoL studies on RAAA by Rohrer et al. (1998) used a modified version of Self-evaluation of Life Function Scale and compared the results with patients undergoing elective repair (n=29). The validation of this modified version was uncertain. Magee et al. (1992) and Hennessy et al. (1998) used a validated Rosser index classification. Hennessy et al. (1998) reported a QoL equal to that of elective controls (n=14), and Magee et al. (1992) reported impaired QoL after RAAA (n=45). The QoL -index was lower postoperatively than preoperatively. Patients undergoing elective repair, on the other hand, had an improved QoL index after repair (Magee et al. 1992). The rupture group and elective group were not age-matched; the RAAA patients were older than those undergoing elective surgery.

The latest QoL studies have used the validated SF-36 measure (Ware and Sherbourne 1992). Comparison of RAAA survivors with the norms of age- and sex-adjusted population revealed no significant differences between these groups (n=28) (Bohmer et al. 1999). Joseph et al. (2002) reported the same or even better QoL compared to norms (n=26). In their recent study, Tambyraja et al. (2005b) reported equal QoL after emergency and elective repair of RAAA (n=30). When comparison was made with the norms of an age- and sex-adjusted population, RAAA patients had worse outcomes in two out of eight health domains, i.e. role limitations due to physical and emotional problems. The value of this study was the fixed follow-up time, as the QoL questionnaire was sent to the survivors six months after surgery of RAAA. Although, six months was usually the time for patients with an elective AAA operation to regain their preoperative health-related levels, it may nevertheless be too short to recover after major surgery of RAAA (Perkins et al. 1998). Critically ill ICU patients (including 40 emergency surgery cases of any nature) had a worse QoL one year after intensive care than the age- and sex-adjusted population (Pettilä et al. 2000).

Hinterseher et al. (2004) used the WHO-QOL-BREF test because of its international comparability and its brevity. They considered the SF-36 questionnaire to be lengthy, although it is more widely used. The QoL was equal in all five health domains compared to the age- and sex-adjusted control population.

All previous QoL studies are retrospective with different follow-up times, except the one by Tambyraja et al. (2005b). Furthermore, preoperative QoL assessment is not feasible nor even possible after RAAA. In addition, a small sample size is sensitive to bias and may reflect an absence of differences.

Table 5. Studies regarding quality of life (QoL) among RAAA patients.

Authors	Year	N	Measure	Findings in QoL
Tambyraja et al.	2005	30	SF-36	Good QoL, equal to elective group but worse than norms
Hinterseher et al.	2004	24	WHO-QOL-BREF-test	QoL equal to the general population
Piper et al.	2003	39	Authors' own questionnaire	QoL equal to preoperative status
Joseph et al.	2002	26	SF-36	Equal or better QoL compared to norms
Bohmer et al.	1999	28	SF-36	QoL equal to norms
Hennessy et al.	1998	14	Rosser index	Good QoL, equal to elective group
Currie et al.	1992	7 RAAA 13 AAA	Authors' own questionnaire	QoL equal to normal subjects
Magee et al.	1992	45	Rosser index	Impaired QoL
Rohrer et al.	1988	29	Modified Self-evaluation of Life Function Scale	Good QoL

SF-36

The Medical Outcomes Study Short-form 36-item survey (SF-36) is the most widely used and validated generic health-related QoL measure (Ware and Sherbourne 1992, Garratt et al. 2002). The SF-36 was found to be valid and reliable for use in a vascular setting and was recommended for QoL assessment in vascular patients (Beattie et al. 1997). The RAND-36 is based on the SF-36 measure (Hays et al. 1993) and consists of the same 36 questions that assess eight health domains: 1) physical functioning, 2) limitations in daily routines activities because of physical health problems, 3) social functioning, 4) limitations in daily routine activities because of emotional health problems, 5) general mental health, 6) vitality (energy and fatigue), 7) pain, and 8) perception of general health. For each domain a score can be calculated from 0 to 100, the higher scores indicating better QoL (0 lowest, 100 perfect health). A validated Finnish version with norms of the age- and sex-matched population is available (Aalto et al. 1999).

The results of the SF-36 QoL assessment after RAAA are presented in Table 5 (Bohmer et al. 1999, Joseph et al. 2002, Tambyraja et al. 2005b).

EQ-5D

The EuroQol, EQ 5 Dimensions (EQ-5D) is a generic measure (The EuroQol Group 1990). Its advantage is the use of the results as a single index which allows the calculation of quality-adjusted life years (QALY) and thus economic evaluation. The higher index indicates better QoL, ranging from 0 (death) to 1 (perfect health). The EQ-5D consists of five questions, defining five health dimensions such as mobility, self-care, usual activities, pain/discomfort, and anxiety/depression. The patients also assess their current health status on the Visual Analogue Scale (VAS), which ranges from 0 (worst imaginable health state) to 100 (best imaginable health state). Ohinmaa et al. have done the validation in the Finnish population (Ohinmaa and Sintonen 1999). There were no studies available in which the EQ-5D measure was used for RAAA patients.

Cost-utility

A quality-adjusted life year (QALY) is a measure combining the quantity and the quality of life (Taylor and Gerrard 2002). It is calculated by multiplying the length of life, including life expectancy by quality adjustment. Quality adjustment ranges from 0 (death) to 1 (alive in perfect health). Cost-effectiveness assesses the costs of treatment and measures the health effects of that treatment. Three methods have been used for economic evaluation: 1) a cost-benefit analysis, where the benefit is assessed only as monetary values; 2) a cost-effectiveness analysis where the outcome is assessed in clinically relevant scales, such as life years gained; 3) a cost-utility analysis, where the outcome is assessed as QALYs (Taylor and Gerrard 2002). If the net costs of the treatment are divided by the net effects of the treatment, it is called the cost-effectiveness ratio, which is usually expressed as a cost per QALY saved. QALYs allow the comparison of cost-effectiveness ratios between diseases and treatments. To enhance the comparability across health interventions, cost-effectiveness analyses are ideally based on lifetime simulations of related costs and quality-adjusted survival (Shepard 1999).

Patel et al. (2000) used a hypothetical cohort of RAAA patients, calculating 3.4 QALYs to be achieved after RAAA surgery. They used estimates and calculated the costs for initial hospitalisation to be 22 700 € and for lifetime 28 800 € (US Dollars converted to Euros). Thus the cost-effectiveness ratio was 8 500 €/QALY. A mortality of 50% was used as a base-case of mortality. If mortality rose to a level of 80%, the cost-effectiveness ratio rose to 24 800 €/QALY. The impact of age was also calculated; if age and operative mortality were adjusted, the cost-effectiveness ratio for a 95-years old RAAA patient was 47 600 €/QALY.

Tang et al. (2003) calculated costs for 47 RAAA patients. They found huge variation in the costs. For complicated aneurysms, which by definition developed one or more dysfunction in the organ system needing system support, the median costs were 30 150 € and for uncomplicated aneurysms 9 600 € (UK Sterling converted to Euros). The median overall cost was 12 100 € and nearly twice that of an elective repair. Costs were calculated for RAAA patients selected for surgery; 8 non-operated patients were excluded from the analysis.

Seiwert et al. (1995) calculated the cost of additional, adjusted life years for 119 patients undergoing surgery for RAAA; 6 patients were excluded from the analysis as unfit for surgery. In the age group of 65-69 years, 31 patients had the lowest overall mortality (22%) and they

consumed the greatest total costs. The mean cost for each RAAA patient was 17 900 €, but with successful outcome the mean cost per survivor was 32 800 € (US Dollars converted to Euros). The cost per additional adjusted life year was 3 200 €.

Aune et al. (2004) reported the life years gained in over 80-year-olds after RAAA. The operative mortality was 47% (25/53). Mean survival was 2.7 years for all the patients and 5.2 years for survivors. The over 80-year-olds gained 145 life years in total and the estimated cost per gained life year was 6 800 €.

Predictors of outcome

Attempts have been made to identify predictors of mortality on different levels of the treatment chain of RAAA patients in the hospital. Most of these studies have evaluated risk factors predicting 30-day or hospital mortality. The initial decision on whether to operate or not is usually made in the emergency room. Factors predicting mortality are presented in Table 6.

Age

Advanced age is associated with increased operative mortality after RAAA (Katz et el. 1994, Samy et al. 1994, Chen et al. 1996, Hardman et al. 1996, Rutledge et al. 1996, Dardik et al. 1998, Heller et al. 2000, Hsiang et al. 2001, Noel et al. 2001, Alric et al. 2003, Roddy et al. 2003, Visser et al. 2005, Dillavou et al. 2006). The cut-off age for worsening the outcome ranged from 70 to 80 years (Katz et al. 1994, Hardman et al. 1996, Heller et al. 2000, Roddy et al. 2003). Gloviczki et al. (1992) found operative mortality of 56% for patients 80 years or older, compared with 47% for those under 80. Barry et al. (1998) found mortalities of 51% and 48%, respectively. Hallin et al. (2001) demonstrated relative risk estimates of 1.9–3.1/10 years in their literature review of 3 138 RAAA patients (in 1985–1997). The mean operative mortality among patients over 80 years was 53% (147/272) with a wide range (37–90%) after repair of RAAA, based on 14 different reports (Rosset et al. 2006). Opposite results have been reported as well (Harris et al. 1991, Aune et al. 1995, Halpern et al. 1997, Janczyk et al. 2004). Although high age is a risk factor for surgery, survival over 40% is acceptable (Gloviczki et al. 1992). Age is considered to be a minor clinical predictor of peri-operative cardiovascular risk by the American College of

Cardiology and the American Hearth Association (ACC/AHA) guidelines for peri-operative cardiovascular evaluation for non-cardiac major surgery (Eagle et al. 2002).

Co-morbidities

Co-morbidities increase along with ageing, especially vascular diseases. Among 72 over 80-year-olds (including 63 AAA and 9 RAAA patients) 56% had hypertension, 30% ischemic heart disease, 21% peripheral vascular disease, 17% renal failure, and 14% respiratory insufficiency (Rosset et al. 2006). Coronary heart disease (Samy et al. 1994, Kniemeyer et al. 2000), cerebrovascular disease (Samy et al. 1994, Koskas and Kieffer 1997), chronic obstructive pulmonary disease (Samy et al. 1994, Dardik et al. 1998), and chronic renal failure (Samy et al. 1994, Sasaki et al. 1999, Heller et al. 2000) affected mortality after RAAA. Some studies have demonstrated no influence of preoperative health status (Seiwert et al. 1995, Halpern et al. 1997, Harris et al. 2005). A literature review of preoperative risk factors based on multivariate analyses demonstrated that renal failure and COPD were risk factors, but coronary heart disease was not (Hallin et al. 2001). Dardik et al. (1998) found that hypertension and pulmonary disease correlated with operative mortality. They proposed that diseases might have been overdiagnosed by more aggressive coding strategies in many hospitals, as described by Katz et al. (1994). Patients who died before reaching the hospital may have had more severe co-morbidities, thus introducing a bias by selection or underreporting on assessing the influence of co-morbidities on outcome.

Gender

There are large databases on the influence of gender after repair of RAAA. Women were less likely to undergo surgery (Katz et al. 1997, Evans et al. 2000, Noel et al. 2001, Dueck et al. 2004a) and had a poorer outcome after repair of RAAA compared with men (Katz et al. 1994, Rutledge et al. 1996, Heller et al. 2000, Semmens et al. 2000, Dueck et al. 2004a, Dillavou et al. 2006). Men were 1.4 times more likely than women to undergo surgery after RAAA, and women had a 1.4-fold greater risk for operative death (Katz et al. 1997). However, these results were flawed by low operative rates: men 61% and women 49%. The proportion of women was larger in the rupture population than in a group of elective repair of AAA patients in a recent study

(Dillavou et al. 2006). The reason for these discrepancies is not clear. The advanced age of women at the time of rupture has been shown in many studies. Advanced age is associated with an increased incidence of co-morbidities such as coronary heart disease and cerebrovascular disease. In contrast, gender was not found to be a risk factor for postoperative mortality based on some previous reports (Samy et al. 1994, Dardik et al. 1998, Evans et al. 2000, Visser et al. 2005).

Other pre- and intra-operative factors

Free intraperitoneal rupture was associated with higher mortality as compared with retroperitoneal rupture (Rantakokko et al. 1983, Turton et al. 2000). In the series of 135 RAAA patients, Rantakokko et al. (1983) reported that all 14 out of 65 operated patients with free intraperitoneal rupture died. The operative mortality among patients with free intraperitoneal rupture was 58%, whereas among patients with massive retroperitoneal haematoma it was 43%, and with retroperitoneal staining 24%, respectively (Gloviczki et al. 1992).

Chen et al. (1996) reported three major preoperative predictors for operative mortality, namely, unconsciousness OR 3.1 (95% CI 2.2–4.4), advanced age 1.9 (1.5–2.3), and cardiac arrest 1.8 (1.4–2.4). Co-morbidities were not predictors of operative mortality after RAAA. However, after elective repair of AAA, co-morbidities such as previous heart failure OR 2.6 (95% CI 1.7–3.9) and peripheral vascular disease 2.9 (2.0–4.3) affected mortality (Chen et al. 1996).

Patients who died in the emergency room (7%) were included in the study of Gloviczki et al. (1992) who reported systolic blood pressure <90 mmHg to predict hospital mortality by OR 3.0 (95% CI 1.5–6.1) and haematocrit 1.3 (1.1–1.7). Preoperative haematocrit <28 predicted 72% mortality among RAAA patients (Gloviczki et al. 1992). Haemoglobin below 90 g/l predicted operative mortality (Hardman et al. 1996, Kniemeyer et al. 2000) as did acute renal failure measured by serum creatine level >190 μmol/l (Hardman et al. 1996).

Samy et al. (1994) demonstrated that shock was a pre-eminent prognostic factor in assessing outcome after repair of RAAA. The diagnosis of shock was based on retrospective clinical information of hypotension, tachycardia, pallor, and sweating. It could be assumed that the latter

two were not so easily retrievable from the medical files. Without a strict definition of shock the results may be biased.

A predictor of mortality after repair of RAAA was ischemia confirmed by electrocardiograph (ECG) by OR of 6.6 (95% CI 1.8–24.3) (Hardman et al. 1996). Ischemia was defined as greater than a 1 mm ST segment depression or an associated T-wave change present on the admission ECG. In a recent study by Tambyraja et al. (2005a), an increase in cardiac troponin I (cTnI) predicted operative mortality after RAAA. 23 of the 50 patients had an elevated cTnI level, and 11 of these had ischemia confirmed by ECG. Also the serum creatine level was higher in the patients with an elevated cTnI level. Elevated cTnI levels, however, were associated with other diseases, such as renal failure (Ammann et al. 2004).

Intraoperative temperatures of 36, 34, and 32 $^{\circ}$C predicted operative mortality: 15%, 49%, and 84%, respectively (Janczyk et al. 2004). Base deficit was also an intraoperative predictor, survivors having a mean of –5.1 ($\pm$5.4) base deficit and non-survivors –12.9 ($\pm$7.2) (Janczyk et al. 2004)

Table 6. Preoperative factors predicting mortality after RAAA, data from multivariate analyses.

Table 6.

Authors	Year	N	Mortality (%)	Co-morbidities	Age	Gender	Unconsciousness	Shock/Hypotension	Cardiac arrest	ECG changes	Haemoglobin	Creatinine	Hypothermia	Base deficit
Calderwood et al.	2004	137	56		>76		0			1	<90 g/l	>180 µmol/l		
Janczyk et al.	2004	100	47		0	0	0	0			0		1	1
Piper et al.	2003	147	35	0				0					1	0
Noel et al.	2001	413	37		1				1		Low hematocrit			
Hsiang et al.	2001	134	53		1		1		1		0			
Kniemeyer et al.	2000	57	30	CHD	0	0		SBP <80 mmHg		1	<90 g/l	0		
Turton et al.	2000	102	53		0	0		SBP <91 mmHg	1					
Heller et al.	2000	67751	46	RF	>70	1								
Sasaki et al.	1999	183	35	RF	0			Hypotension						
Dardik et al.	1998	527	47	COPD, HT	1	0								
Halpern et al.	1997	96	56	0	0		1	SBP <90 mmHg			<100 g/l	>150 µmol/l		
Koskas et al.	1997	158	53	CVD	1	0		0				0		
Chen et al.	1996	157	46		1		1		1					
Hardman et al.	1996	154	39		>76	0	1			1	1	>190 µmol/l		
Samy et al.	1994	203	40	CHD, CVD, RF	1	0		Shock	1					
Gloviczki et al.	1992	231	42	COPD	0	0		SBP <90 mmHg	1		Low hematocrit	0		
Harris et al.	1991	113	64	0	0	0	1	0	1					0

CHD=Coronary heart disease;RF=renal failure;COPD=Chronic obstructive pulmonary disease; HT=hypertension;CVD=cerebrovascular disease;SBP=Systolic blood pressure

Postoperative factors

After postoperative treatment of at least 24–48 hours in the ICU, the predictors of mortality differed from the predictors for preoperative or overall mortality (Maziak et al. 1998, Kniemeyer et al. 2000, Bown et al. 2004). In a series of 222 RAAA patients, 18% were considered unfit for surgery (Bown et al. 2004). Of the 181 operated patients, 141 survived at least 24 h after initial surgery. Mortality after 24 h was 32%, and multiple organ dysfunction was responsible for 60% of the deaths. Kniemeyer et al. (2000) reported that multiple organ dysfunction was the cause of death in 93% of the postoperatively deceased patients. The cause of death was haemorrhagic shock for the patients who died intraoperatively (Kniemeyer et al. 2000, Bown et al. 2004).

Chen et al. (1996) reported predictors of postoperative mortality for patients who survived after the initial surgery. These were: coagulopathy, (defined as a prothrombin time >4 seconds versus control or thrombocytopenia <60 x 10^9/l) OR 7.9 (95% CI 4.1–15.5), ischemic colitis 6.4 (3.4–12.2), adrenergic agents used >48 hours 4.8 (3.0–7.6), admission to operating room >1 hour 4.6 (2.6–8.3), perioperative myocardial infarction 4.0 (2.1–7.7), and renal failure (serum creatinine level >230 μmol/l) 3.7 (1.9–7.5). Advanced age was also a predictor by OR 4.0 (2.1–8.0) per 10 years.

Acute renal failure after RAAA is a severe complication. Bown et al. (2004) described renal complications in 32% of the patients who survived at least 24 h after initial surgery; half of them required renal replacement therapy. Renal complications were associated with mortality as high as 70%, OR 9.5 (3.1–28.6). Barratt et al. (2000) analysed the outcome of acute renal failure after repair of RAAA in a series of 65 patients. Acute renal failure was defined by serum creatinine level >600 μmol/l, or necessary renal replacement therapy. Acute renal failure occurred at a median of 4 days (range 1–14 days) after surgery. The presence of a vascular disease, additional surgery required, and multiple organ dysfunction were predictors of mortality. Of the 65 patients 75% died before discharge, and after five years over half of the survivors had died.

Measures predicting mortality

Few scoring methods have been tested in a retrospective fashion in patients with RAAA. In order for scoring methods to be useful, they should be readily available already in the emergency room to facilitate initial patient selection. In an emergency situation, only few data are available. In the case of an unconscious patient, the patient's history may remain totally unknown and his/her co-morbidities unidentified, as laboratory test results might not be available at the time of deciding to operate or not. Variables of different scoring methods are presented in Table 7.

Glasgow Aneurysm Score

The preoperative Glasgow Aneurysm Score (GAS) was established to provide a scoring system for predicting the outcome after elective or emergency repair of AAA (Samy et al. 1994). The same authors validated the scoring system prospectively using a different study population (Samy et al. 1996). This simple preoperative score is calculated using the following formula: risk score = (age in years) + (17 points for shock) + (7 points for myocardial disease) + (10 points for cerebrovascular disease) + (14 points for renal disease). A diagnosis of shock is based on clinical information on hypotension, tachycardia, pallor, and sweating. Myocardial disease refers to a previously documented myocardial infarction and ongoing angina pectoris, or both. Cerebrovascular disease refers to all grades of stroke and included transient ischemic attack. Renal disease refers to a history of chronic and acute renal failure, and/or serum urea level >20 μmol/l and/or serum creatinine level >150 μmol/l at the presentation. GAS >95 predicted death in 80% of the cases in the series of 92 RAAA patients (Samy et al. 1996).

The preoperative GAS predicted mortality after elective AAA surgery (Biancari et al. 2003a, Leo et al. 2005, Nesi et al. 2004). The best cut-off level to predict poor outcome was GAS >78 in the study by Biancari et al. (2003a) and >76 in the Finnvasc study (Biancari et al. 2003b). The Area under the Receiver operator characteristic (ROC) curve was 0.80 (95% CI 0.71–0.90), operative mortality was 1.4% with a score of ≤78, and 8.7% with a score of >78 (Biancari et al. 2003b). As regards RAAA, Tambyraja et al. (2005c) showed in a series of 82 RAAA patients that GAS was a poor predictor of outcome, AUC 0.61 (95% CI 0.48–0.73). The median GAS for survivors was 93 and for non-survivors 96. The median GAS of the 18 patients unfit for surgery and excluded from the analysis was 102.

Hardman Index

Hardman et al. (1996) identified five risk factors that were easily determined on presentation to the emergency room. The risk factors had cumulative predictive value when three or more of the five factors were associated with 100% mortality. These risk factors were: age >76 years, loss of consciousness after arrival, ischemia confirmed by ECG, haemoglobin <90 g/l, and serum creatinine level >190 μmol/l. In the first studies, a score of three or more risk factors predicted fatal outcome, although the number of patients at risk was small (n=20) (Boyle et al. 2003, Prance et al. 1999). In later studies there were 7/19 hospital survivors with three or more risk factors after RAAA (Neary et al. 2003, Calderwood et al. 2004, Tambyraja et al. 2005c). Furthermore, one of these succumbed at a nursing home six weeks after hospital discharge (Neary et al. 2003) and two were in the communal rehabilitation hospital (Tambyraja et al. 2005c). Follow-up time of the study was not available (Tambyraja et al. 2005c).

POSSUM

Copeland et al. (1991) derived a scoring method called The Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity (POSSUM) for surgical patients. It was later developed and modified with regression equations for vascular patients (V-POSSUM) (Prytherch et al. 1998) and for RAAA patients (RAAA-POSSUM) (Prytherch et al. 2001). The POSSUM consists of two components: 12 physiology and 6 operative variables. The RAAA-POSSUM has two variants: one uses both a physiological and operative score; the other a preoperative physiological score only. Of the POSSUM scores, only the RAAA-POSSUM physiology score can be calculated before the operation. The RAAA-POSSUM as well as Portsmouth-POSSUM (P-POSSUM), and V-POSSUM predicted mortality after repair of RAAA, as did the Hardman Index in a recent study, but the Hardman Index was easier to calculate (Neary et al. 2003). Lazarides et al. (1997) used the conventional POSSUM score and failed in predicting the outcome after RAAA, as did Bown et al. (2004), in all POSSUM models except the V-POSSUM and P-POSSUM.

Models based on the POSSUM data set have been observed to be vulnerable to incomplete data, as a large amount of information is required on every patient. Furthermore, some components of the data set are subjective rather than objective (Prytherch et al. 2005).

VBHOM

The Vascular Biochemistry and Haematology Outcome Models (VBHOM) were designed to minimize data gathering and quality problems (Prytherch et al. 2003). The VBHOM uses seven items which can be constructed from hospital pathology and patient administration computer systems. It failed to model the outcome as a combined group for RAAA and elective repair of AAA, and necessitated the formation of two separate models for elective and emergency surgery (Prytherch et al. 2005).

APACHE II

The Acute Physiologic and Chronic Health Evaluation (APACHE II) scoring system is validated and used widely in critical care to assess the severity of diseases (Knaus et al. 1985). It is based on 12 physiologic and laboratory factors, as well as on age and chronic health status. Many studies have indicated that the APACHE II score is a good predictor of outcome after RAAA (Gloviczki et al. 1992, Maziak et al. 1998, Ho et al. 1999). Gloviczki et al. (1992) found the mean APACHE score at admission to be 11.3 for survivors and 19.1 for non-survivors, OR 1.50 (95% CI 1.2-1.9). Patients unfit for surgery were included. Also Lazarides et al. (1997) found a high score to be associated with death, but its power to predict outcome was limited in individual patients. They reported a mean APACHE II score of 11.3 for survivors and 14.5 for non-survivors (Lazarides et al. 1997), although APACHE II was never intended to predict death at the individual level (Chang 1989).

The APACHE II has been modified and internally validated for AAA patients (APACHE II-AAA) to predict mortality after elective and emergency surgery (Hadjianastassiou et al. 2005). The following components from APACHE II were chosen: Acute Physiology Score, chronic health status classified as binary, operative urgency, and chronological age. The APACHE-AAA model had better calibration than the original APACHE II model in predicting outcome after repair of AAA. Of the 1896 cases, 605 were emergency ones. Also in different age groups the APACHE-AAA predicted operative mortality better than the APACHE II. The hierarchical methodology used to develop the APACHE-AAA model makes it flexible to adjust for the hospital-related factors, which also affect outcome in addition to the patient-casemix.

Hadjianastassiou et al. (2006) compared different prediction models in the ICU after repair of RAAA. Statistical modelling by multiple regression analysis was the best model of assessing hospital mortality when compared to the clinician's estimate or artificial neural network, which is a computational, mathematic tool for information processing. The covariates included in the multiple regression analysis were age, Acute Physiology Score, chronic health status, and operative urgency.

SOFA

Various organ dysfunction scores have been described for general use in the ICU. The Sequential Organ Failure Assessment (SOFA) is a scoring instrument used to quantify the severity of organ dysfunction in critically ill patients (Vincent et al. 1998). The SOFA score (score 0–4) is defined as follows: Pa02/Fi02 for respiratory failure, creatinine level, urine output, or renal hemofiltration or hemodialysis for renal, bilirubin for liver, mean arterial pressure, dopamine/doputamine or epinephrine/ norepinephrine administered for the cardiovascular system, platelet count for coagulation, and Glasgow Coma Scale for central nervous system (Vincent et al. 1998). There were no studies available in which SOFA score was used for RAAA patients.

Table 7. Variables of different scores predicting outcome.

Glasgow Aneurysm Score (Samy et al. 1994)	Hardman Index (Hardman et al. 1996)	SOFA (Vincent et al. 1998)	VBHOM (Prytherch et al. 2003)	POSSUM Physiology score (Prytherch et al. 1998)	POSSUM Operative score (Prytherch et al. 1998)	APACHE II (Knaus et al. 1985)
Preoper.	Preoper.	Preoper.	Postoper.	Preoper.	Postoper.	Preoper.
Age	Age	Pa02/Fi02	Age	Age	Grade of operation	Age
Shock	Consciousness	Platelets	Alive/ dead	Systolic blood pressure	Number of procedures	Chronic health status
Myocardial disease	ECG	Serum bilirubin	Hospital stay	ECG	Total blood loss	Haematocrit
Cerebro-vascular disease	Haemoglobin	Mean arterial pressure	Haemoglobin	Haemoglobin	Peritoneal soiling	Haemoglobin
Renal disease or acute failure	Serum creatinine	Glasgow Coma Scale	Serum urea	Serum creatinine	Presence of malignancy	Serum creatinine
		Serum creatinine/ Urine output	Serum sodium and potassium	Serum urea	Timing of operation	Serum sodium and potassium
			White blood cell count	Serum potassium		White blood cell count
				White blood cell count		Temperature
				Cardiac signs		Mean arterial pressure
				Respiratory signs		Pulse rate
				Heart rate		Respiratory rate
				Glasgow Coma Scale		Arterial pH
						Oxidation (p02)

SOFA = Sequential Organ Failure Assessment; VBHOM = Vascular Biochemistry and Haematology Outcome Models; POSSUM = The Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity; APACHE II = Acute Physiologic and Chronic Health Evaluation; Preoper. = preoperative score; Postoper. = postoperative score; ECG = Electrocardiograph

AIMS OF THE PRESENT STUDY

The main purpose of this study was to evaluate the outcome and factors predicting mortality in ruptured abdominal aortic aneurysm (RAAA) in Helsinki and Uusimaa district.

The specific aims were to assess:
1. The 30-day operative, hospital, and population-based mortality, and effects of organisational changes on outcome after RAAA. (I–V)
2. Quality of life of patients undergoing repair of RAAA. (I, II)
3. The number of quality-adjusted life years (QALY) following RAAA and factors related to QALYs. (II)
4. Predictors of outcome after RAAA. (II–IV)

PATIENTS AND METHODS

Data source

Data from Helsinki University Central Hospital (HUCH) was retrieved from the patient records, Hospital Discharge Registry, and the Cause of Death Registry of Statistics Finland. The outcome status of all patients was cross-referenced by a manual case record review, and computerised patient administration system of HUCH was used, if needed. All Helsinki and Uusimaa data were retrieved from the Cause of Death Registry of Statistics Finland. The death certificates of all patients were recorded. National Finnvasc registry data were collected from the beginning of 1991 to 1999. Details of the Finnvasc registry have been described previously (Salenius 1992).

The protocols of the quality of life (QoL) studies (I, II) were approved by the Ethics Committee of the HUCH. All retrospective studies had a licence for document research approved by the Surgical Board of HUCH.

PATIENTS AND STUDY DESIGNS

Altogether 1381 RAAA patients were included in five studies (I–V) in 1991–2004 (Figure 1). Of the 1381 RAAA patients, 1087 underwent repair for RAAA. 13 patients were excluded from the analysis of operative treatment due to their operation outside HUCH, and they were not included in the Finnvasc data either. The mean age of the 1074 operated RAAA patients was 71 years (median 71, range 43–94 years) and 86% were men (Table 8). 13% (139/1074) were over 80 years of age and 65% (90/139) of them were men (Table 8).

Study III
836

*81

Study V
626

Study IV
197

Study I
220

Study II
269
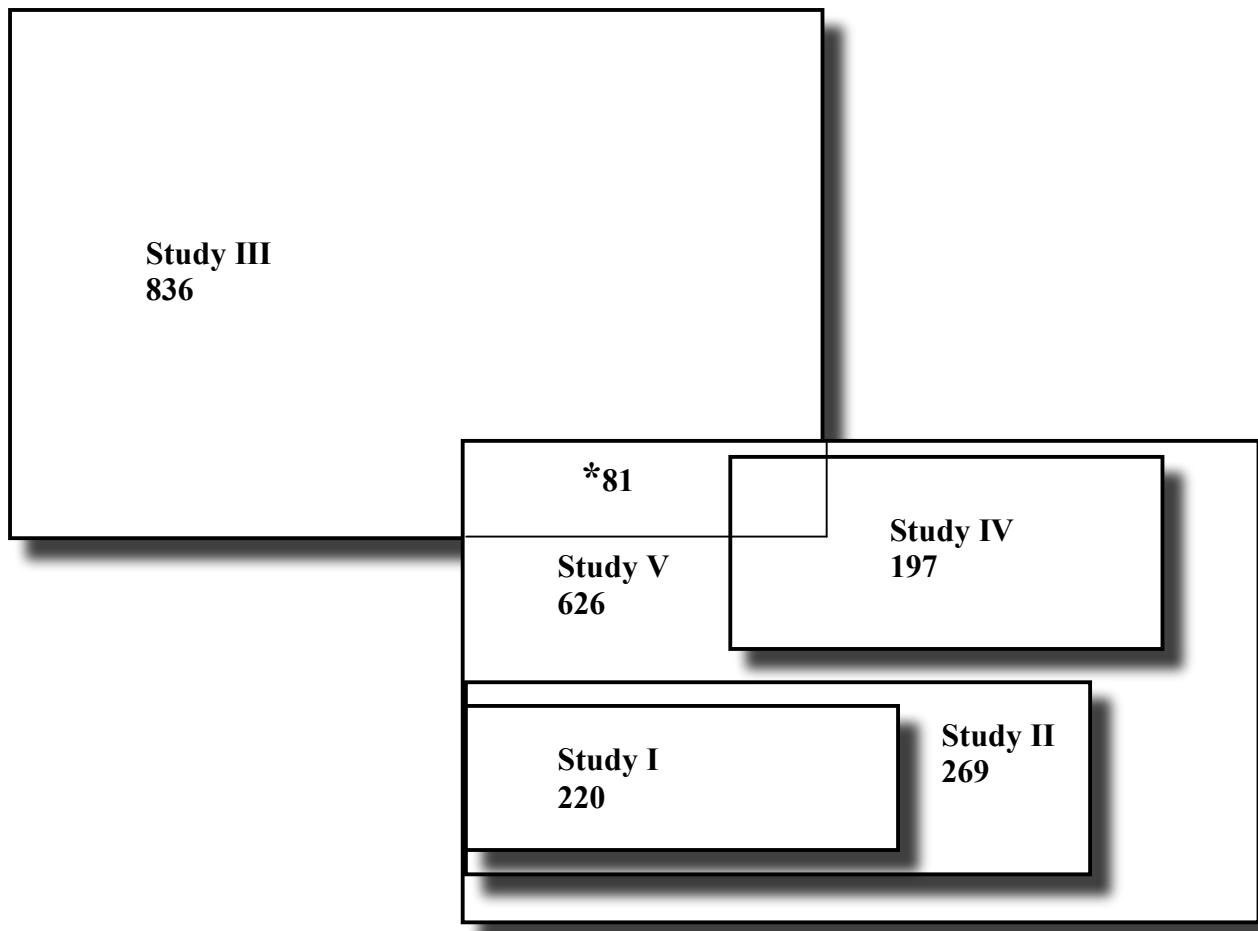
| 1991 | 1992 | 1993 | 1994 | 1995 | 1996 | 1997 | 1998 | 1999 | 2000 | 2001 | 2002 | 2003 | 2004 |

*81 RAAA patients were included in both the Helsinki and Uusimaa (V) and the Finnvasc (III) study population

Figure 1. A total of 1381 RAAA patients in the different study years (1996–2004) and the number of patients (I–V).

Table 8. Demographic data on 1074 RAAA patients who underwent repair of RAAA in 1991-2004.

	All RAAA patients	Age ≥80 years	All survivors	Survivors age ≥80	All non-survivors
N (%)	1074	139 (13%)	600 (56)%	50 (36%)	474 (44%)
Age, years median (range)	71 (43–94)	83 (80–94)	69 (46–94)	83 (80–94)	73 (43–93)
Male (%)	925 (86%)	90 (65%)	522 (87%)	32 (64%)	403 (85%)
GAS median (range)	85 (49–126)	99 (81–126)	80 (49–120)	93 (81–119)	92 (54–126)

GAS = Glasgow Aneurysm Score

Study I

The study included 220 RAAA patients in HUCH in 1996–2000 (Table 9). The long-term survival and QoL of 220 RAAA patients were studied. The validated Finnish version of the RAND 36-item Health Survey 1.0 (RAND-36) questionnaire, along with a cover letter explaining the study, were mailed to 93 long-term surviving RAAA patients (Aalto et al. 1999). The results were compared with the norms of the age- and sex-adjusted Finnish population.

Study II

The study comprised 269 RAAA patients admitted in HUCH for RAAA in 1996–2002 (Table 9). QALYs were calculated for all RAAA patients and for survivors. The date of death of each patient based on the social security number was checked from Statistics Finland, and the life expectancies of the surviving patients were then calculated using the life expectancy of the age- and sex-adjusted population obtained from Statistics Finland. The EQ-5D questionnaire was used for QoL evaluation. Of the surviving 129 patients (129/269), 111 patients were available for QoL assessment. Factors affecting the number of QALYs and hospital survival were evaluated. Preoperative scores of APACHE II, SOFA, Glasgow Aneurysm Score (GAS), and Hardman index were calculated retrospectively from the medical files. The Hardman index was calculated and handled as a dichotomous variable: if 3 or more preoperative factors were positive, the score was considered positive.

Table 9. Number of RAAA patients (I, II).

	Study I	Study II
RAAA patients	220	269
Operated	199	242
Not operated	21	27
Moribund	15	18
Diagnosed at autopsy	6	9
Survivors	117	129
QoL questionnaire sent		
RAND-36	93	
EQ-5D		111

QoL = Quality of life

Study III

From January 1991 to December 1999, 836 patients underwent open repair of RAAA in Finland according to the Finnvasc registry. Pre-, intra-, and postoperative data were collected from the national Finnvasc registry on 836 RAAA patients. Details of the Finnvasc registry have been described previously (Salenius 1992). The influence of the GAS and that of analysed risk factors on 30-day operative mortality were evaluated. We also included a number of other risk factors and used the Finnvasc definition criteria for coronary artery disease, cerebrovascular disease, diabetes, hyperlipidemia, hypertension, pulmonary disease, renal failure, and previous arterial revascularisation, or amputation.

Study IV

This retrospective study comprised 197 RAAA patients in 1999–2003 in HUCH. The aim was to identify predictive factors for 30-day operative mortality after 48 h of maximal treatment in the ICU after repair of RAAA. Of these 197 RAAA patients, 185 underwent surgery and 138 survived at least 48 h. The survivors were included in a study, and 122 factors were reviewed from the medical files. The effect of multiple organ dysfunction, defined according to the SOFA classification, was evaluated. Preoperative GAS was recorded.

Study V

The study included all RAAA patients identified in Helsinki and Uusimaa district in 1996–2004. A total of 626 patients had RAAA, 352 (56%) were admitted to HUCH. Regional centralisation and quality improvements in the treatment chain of RAAA were performed in the years 1999 to 2002 in HUCH. During the regional reorganisation, the vascular service was centralised at the Department of Vascular Surgery of HUCH in the Meilahti Hospital from 1999 onwards in the hospital district of Helsinki and Uusimaa. In-hospital reorganisation included a new surgical intensive care unit (ICU), established in 2001. The on-call system has also been reformed over the years from a single vascular surgical consultant at home to a vascular trainee in the hospital and a senior vascular surgeon on call at home, as of 2003. The study period was divided into three phases: I control period (1996–1998), II change period (1999–2002), and III present

organisational period (2003–2004). The impact of organisational changes on mortality was assessed by comparing the time periods. Data were collected retrospectively from the medical files and death certificates. Patients having undergone open surgery and treated with endovascular repair were included (Table 10). The profile of patients according to their age, gender, and GAS was recorded.

Table 10. Demographic data on 352 RAAA patients in the Helsinki University Central Hospital in 1996–2004.

	Non-operated	Endovascular	Open surgery	Survivors	Non-survivors
N (%)	33 (9)	4 (1)	315 (90)	195 (55)	157 (45)
Age, years median (range)	77 (54–95)	68 (64–73)	72 (43–93)	71 (46–92)	74 (42–95)
Male (%)	24 (73)	4 (100)	260 (83)	165 (85)	123 (78)
GAS median (range)	102 (77–130)	71 (64–86)	86 (50–126)	80 (50–119)	95 (56–130)

GAS = Glasgow Aneurysm Score

OUTCOME MEASURES

Quality of life (I, II)

RAND 36 (I)

The validated Finnish version (Aalto et al. 1999) of the RAND 36-item Health Survey 1.0 (Hays et al. 1993) questionnaire was mailed for self-administration to the patients known to be alive after repair of RAAA. Any help required in the administration was recorded. In cases of no initial response, the questionnaire was resent. If there was no response after two mailings, the patients were contacted by phone. The details of the RAND-36 measure are given on the page 27.

EQ-5D (II)

The validated EQ-5D questionnaire (The EuroQol Group 1990) was chosen because the results could be used as a single index for calculating QALYs. The questionnaire was sent for self-administration to the patients known to be alive after repair of RAAA. Any help required in administration was recorded. In cases of no initial response, the questionnaire was resent.

The details of the EQ-5D measure are given on the page 27. The results were expressed as the EQ-5D index and EQ-VAS. The EQ-5D index and the EQ-VAS values of the study population were compared with the age- and sex-adjusted general Finnish population values; that for the EQ-5D index was 0.795 (median 0.785, range 0.600 to 1.000) and that for the EQ-VAS was 68 (median 68, range 56–77) (Ohinmaa and Sintonen 1999).

Quality-adjusted life years (II)

The assessment of quality-adjusted life years (QALY) aims at combining the length and quality of life. The quality adjustment ranges from 0 (death) to 1 (perfect health). The EQ-5D index was used to assess the QoL of the RAAA patients. A patients with hospital mortality had a value of 0 QALY. If the endpoint was not reached during the follow-up period, then estimates of life expectancies derived from the age- and sex-adjusted population were used. For the non-respondents also pessimistic QALYs were calculated as 75% of the QoL indices and 75% life expectancy according to the age- and sex-adjusted population for survivors.

Glasgow Aneurysm Score (II–V)

The preoperative Glasgow Aneurysm Score (GAS) for each RAAA patient was calculated according to the score formula (II–V). Details of the score are given on the page 35. The value of the GAS as a preoperative predictor of hospital mortality was evaluated (II–IV) as well as GAS affecting the number of QALYs (II). The GAS was used to assess the preoperative profile of the hospitalised RAAA patient (V).

SOFA (II, IV)

Organ dysfunction was defined as a SOFA score; the SOFA is described on page 38. The SOFA was used for preoperative assessment (II). In Study IV the SOFA scores were calculated postoperatively on day 1, 2 (48-h), 3, 7, 14, and 21 until the patients were discharged. If a laboratory value was missing, the previous day's value was used, and if the previous day's value was missing, the next day's value was used. If both of these values were missing, the values from two earlier days or the following two days were used. If all these values were missing, the value was considered as unavailable and was calculated as a normal value. The Glasgow Coma Scale was considered to be normal in sedated patients if there was no known cause for abnormality.

Mortality (I–V)

The death certificates of all RAAA patients deceased during 1996–2004 in the Helsinki and Uusimaa district were reviewed from the Cause of Death Registry, Statistics Finland. Mortality was presented as 30-day operative mortality (only operated RAAA patients) and hospital mortality, which included all RAAA deaths during the same hospitalisation. Hospital mortality included also moribund RAAA patients who were unfit for surgery, and patients having RAAA as the cause of death at autopsy. Population-based mortality was calculated including all identified RAAA deaths in the whole district (V).

STATISTICAL ANALYSES

Statistical analysis was performed with the SPSS statistical program, SPSS version 10.5, Chicago, IL, USA (I–III); SPSS version 11.0, Chicago, IL, USA (IV); SPSS version 12.0, Chicago, IL, USA (V).

The non-parametric Mann-Whitney U-test (for continuous variables) and the Chi-square test were used for comparisons between survivors and non-survivors (II–IV), and the study population versus norms for an age- and sex-adjusted normal population (I). The results were expressed as Z scores, i.e. the difference between the mean and the adjusted population mean

divided by the adjusted population standard deviation (I). The nonparametric Spearman's correlation test was performed to detect significant correlations between QALYs, and pre-, peri- and postoperative factors (II). Chi-square test was used to compare the number of deaths between the three study phases (V). Differences in continuous variables between the three phases were tested by the nonparametric Kruskal-Wallis test (V).

The receiver operating characteristic (ROC) curve (Harrell et al. 1996) was used to evaluate the value of the GAS in predicting postoperative death (III) and independent predictive factors for mortality (IV). AUC values (Area under the curve) between 0.7 and 0.8 represented reasonable discrimination, whereas values >0.8 could be considered good.

Logistic regression with backward stepwise selection was used for the multivariate analysis of the variables affecting the outcome (III). The significant effects of the factors on QALYs were assessed using forward stepwise linear regression analysis (II). Forward stepwise logistic regression analysis was performed to evaluate factors affecting 30-day operative (IV) and hospital mortality (II). Only those variables with a $p<0.05$ at univariate analysis were included in the regression models (III, IV). The correct classification rate (CCR) was determined for the best model (IV). An accessory Bayes model (B-Course 2.0, Helsinki Institute of Information Technology, Helsinki, Finland) was created to evaluate the factors affecting QALYs (II).

A $p<0.05$ was considered statistically significant. In Study II, however, $p<0.01$ was considered statistically significant due to multiple comparisons.

RESULTS

MORTALITY

A total of 626 patients with RAAA were identified in the Helsinki and Uusimaa district in 1996–2004 (Figure 2). 352 (56%) of the 626 patients, were admitted to HUCH. Hospital was the place of death for 61% of all RAAA mortalities (Table 11).
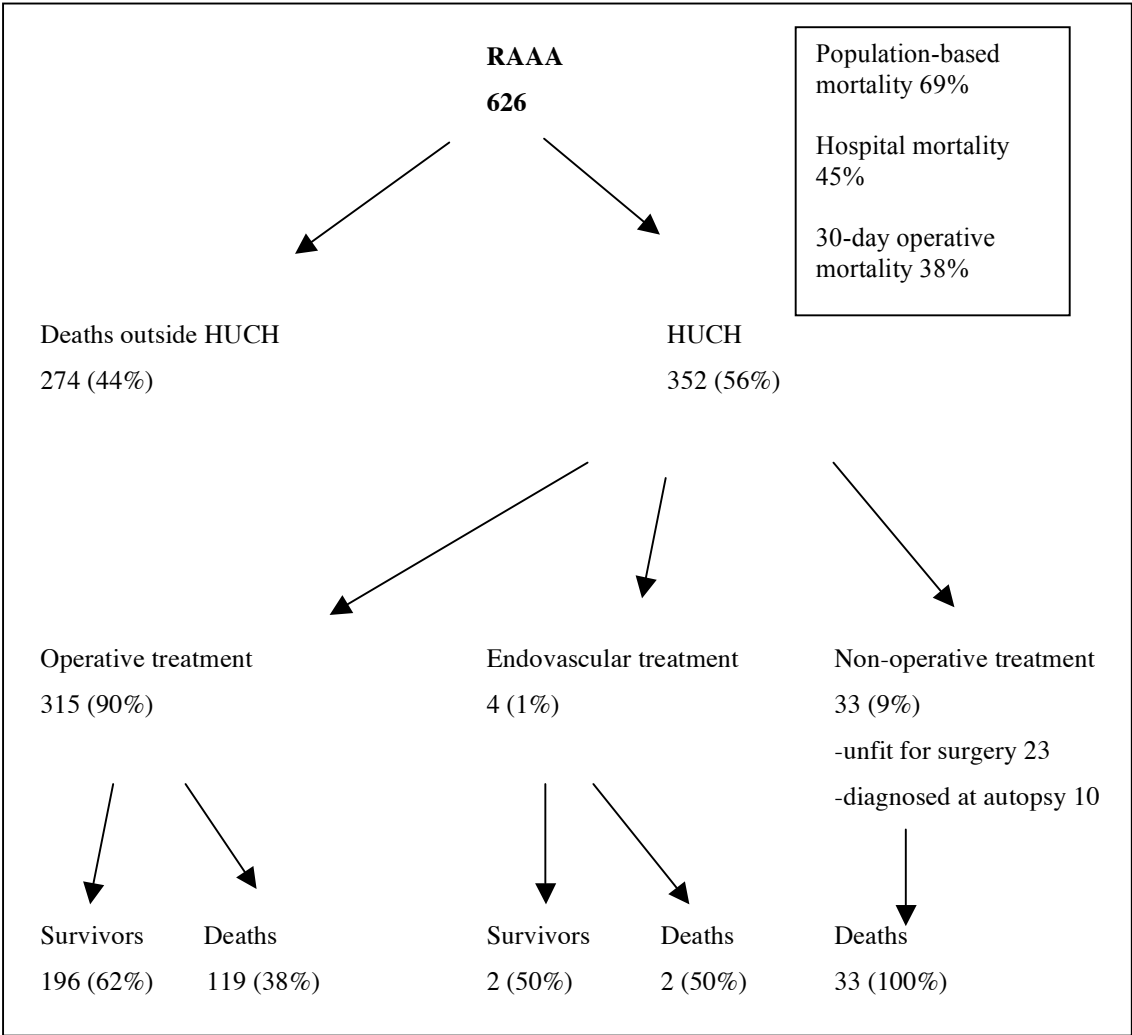
Figure 2. RAAA patients in the Helsinki and Uusimaa district in 1996–2004.

Table 11. Place of death of patients with RAAA in the Helsinki and Uusimaa district in 1996–2004.

Place of death	N	%
HUCH	157	36
Other Hospitals	106	25
Home	135	31
Elsewhere	33	8
Total	431	100

In HUCH 33 patients did not undergo surgery; 21 of them were assessed as moribund or unfit for surgery, and 12 were missing primary identification and diagnosed RAAA at autopsy without any vascular consultation. The reasons for non-operative management of diagnosed RAAA were cardiac arrest or loss of consciousness (33%), age-related co-morbidities (18%), malignancy (6%), and patient refusal (6%) (Table 12).

Table 12. Reasons for non-operative management in 33 RAAA patients in Helsinki University Central Hospital.

Reason	N	%
Cardiac arrest/ Loss of consciousness	11	33
Age-related co-morbidity	6	18
Malignancy	2	6
Patient refusal	2	6
RAAA diagnosed at autopsy	12	36
Total	33	100

The profile of patients according to age, gender, and GAS did not change over the years 1996–2004, nor did time from admission to beginning of the operation. The 30-day operative mortality was 38%, hospital mortality 45%, and population-based mortality 69% (V) (Table 13). Mortality decreased significantly during the third time period (2003–2004); 30-day mortality was 19%, hospital mortality 28%, and population-based mortality 56% (Figure 3).

Table 13. Proportions of 30-day operative, hospital, and population-based mortality of RAAA patients (I-V).

Study	30-day operative mortality (%)	Hospital mortality (%)	Population-based mortality (%)
I	82/199 (41)	NA	NA
II	106/242 (44)	140/269 (52)	NA
III	395/836 (47)	NA	NA
IV	78/185 (42)	93/197 (47)	NA
V	115/315 (38)	157/352 (45)	431/626 (69)

NA = not available
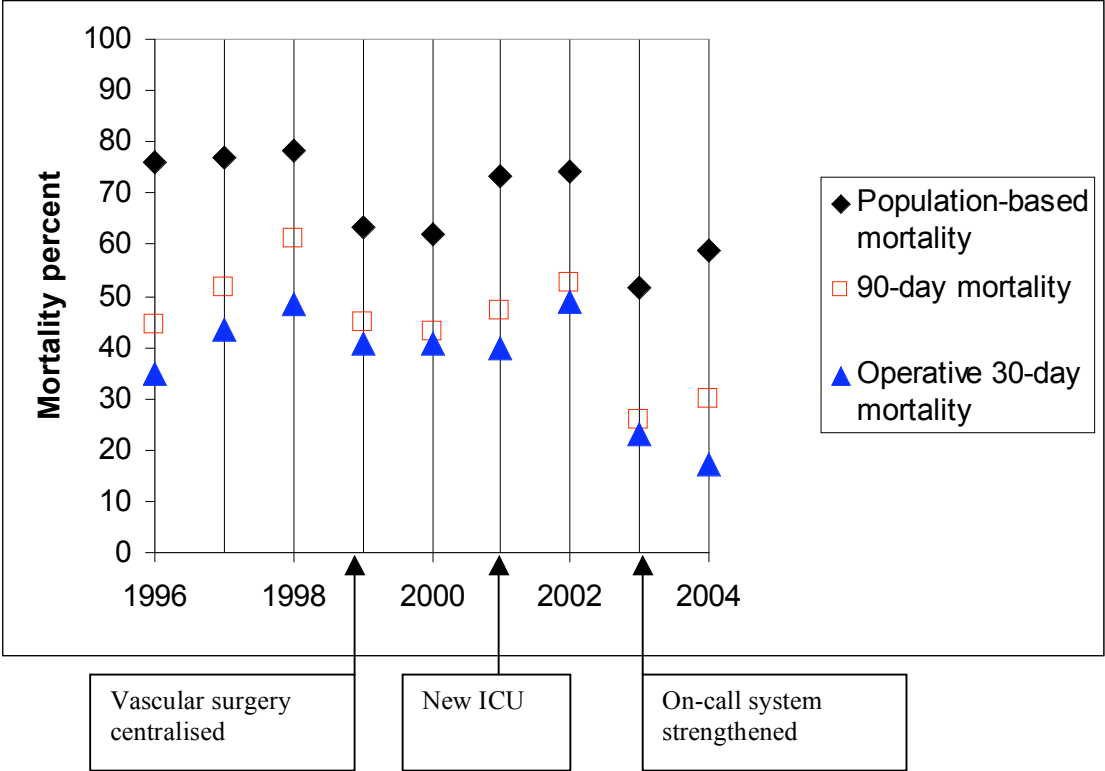
Figure 3. Mortality of RAAA patients in the Helsinki and Uusimaa district in 1996–2004.

Of 352 RAAA patients in HUCH, 67 (19%) were over 80 years of age and 15 of them were not operated. Of the 52 aged patients who underwent surgery, 56% (29/52) died. Hospital mortality was thus 66% (44/67) for patients older than 80 years.

The mean frequency of RAAA during the study was 5.4 RAAAs/100 000 population per year (range 4.5-6.5/100 000) (Table 14).

Table 14. The frequency of RAAA in the Helsinki and Uusimaa hospital district in 1996–2004 retrieved from the Hospital Discharge Registry and the Cause of Death Registry, Statistics Finland.

Year	Population[1] N	All RAAA N	RAAAs/ 100 000 population	RAAA deaths/ 100 000 population
1996	1 240 301	63	5.1	3.9
1997	1 257 702	70	5.6	4.3
1998	1 274 475	83	6.5	5.2
1999	1 290 618	60	4.7	2.9
2000	1 304 595	76	5.8	3.6
2001	1 318 324	68	5.2	3.8
2002	1 329 004	78	5.9	4.4
2003	1 338 180	60	4.5	2.3
2004	1 346 958	68	5.1	2.9

[1] Population according to Statistics Finland.

QUALITY OF LIFE

The response rate to the RAND-36 questionnaire was 88% (82/93). Non-responders were contacted by phone: 2 out of 11 were still working, 6 lived at home without outside help, and 3 could not be contacted. The questionnaire was self-administered by 71% and with assistance by proxy in the case of 29%. Eight domains of QoL were compared with norms for the age- and sex-adjusted general population, and only physical functioning was significantly impaired (p=0.01) (Figure 4). When expressed as a Z score, the difference was –1.21. (I)

The EQ-5D questionnaire response rate was 85% (94/111). The questionnaires were self-administered by 71% and with assistance by 29%. The mean EQ-5D index for respondents was 0.685 (median 0.676, range 0.072–1.000) and significantly lower as compared to the age- and sex-adjusted Finnish general population 0.795 (median 0.785, range 0.600–1.000) (p=0.001). The mean EQ-VAS was 61 (median 63, range 10–100) and did not differ from that of the age- and sex-adjusted Finnish general population, 68 (median 68, range 56–77) (p=0.76). (II)

QUALITY-ADJUSTED LIFE YEARS

The mean (range) number of QALYs after RAAA was 4.1 (0–30.9) for all RAAA patients and 8.5 (0.2–30.9) for hospital survivors. The mean numbers of QALYs according to a pessimistic assessment were 2.2 for all RAAA patients and 4.5 for hospital survivors. Young age and low GAS were associated with a high number of QALYs. There were 2 survivors among the 21 patients with a Hardman score of three or more; they gained 7.3 and 6.5 QALYs, respectively.

Of the 129 survivors, 120 gained at least one QALY, of whom 120 patients 16 were ≥80 years old (34 non-survivors aged ≥80 years old). Of the patients ≥80 years old and with GAS ≥100, 5 patients gained at least one QALY (23 non-survivors aged ≥80 and GAS ≥100).

The BAYES model, using all data independently and quartiles of QALYs as the dependent variable, revealed that GAS was the only factor independently associated with the number of QALYs.
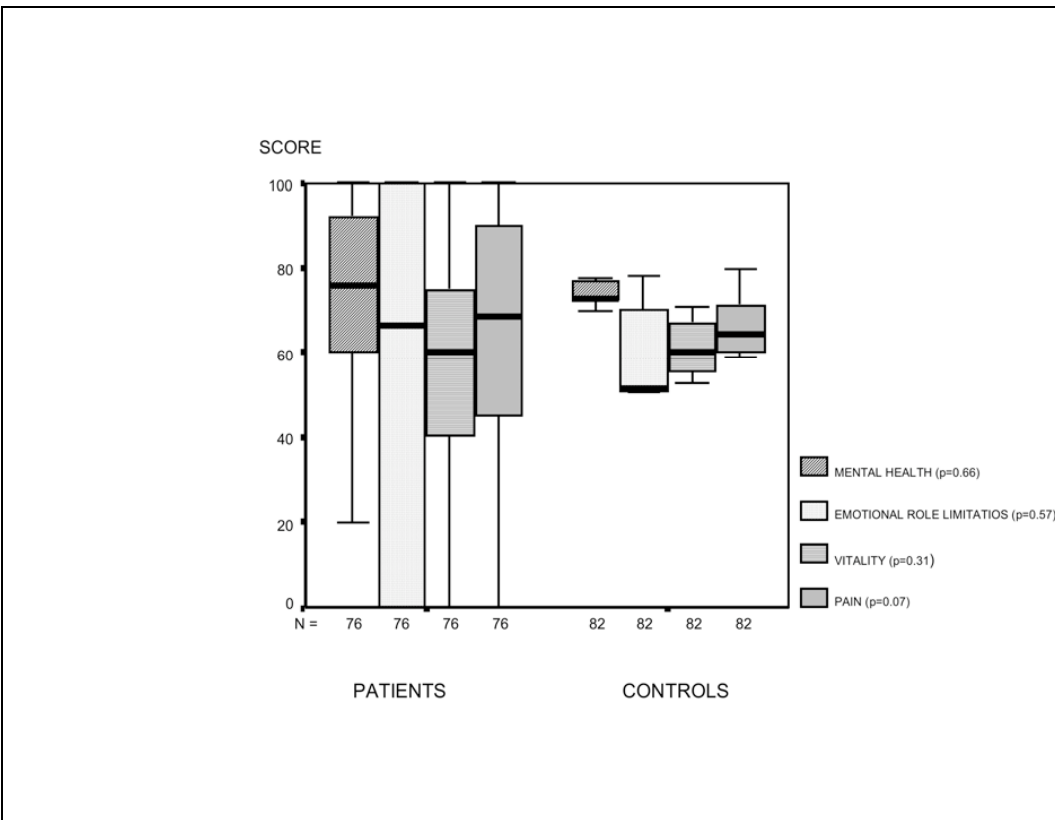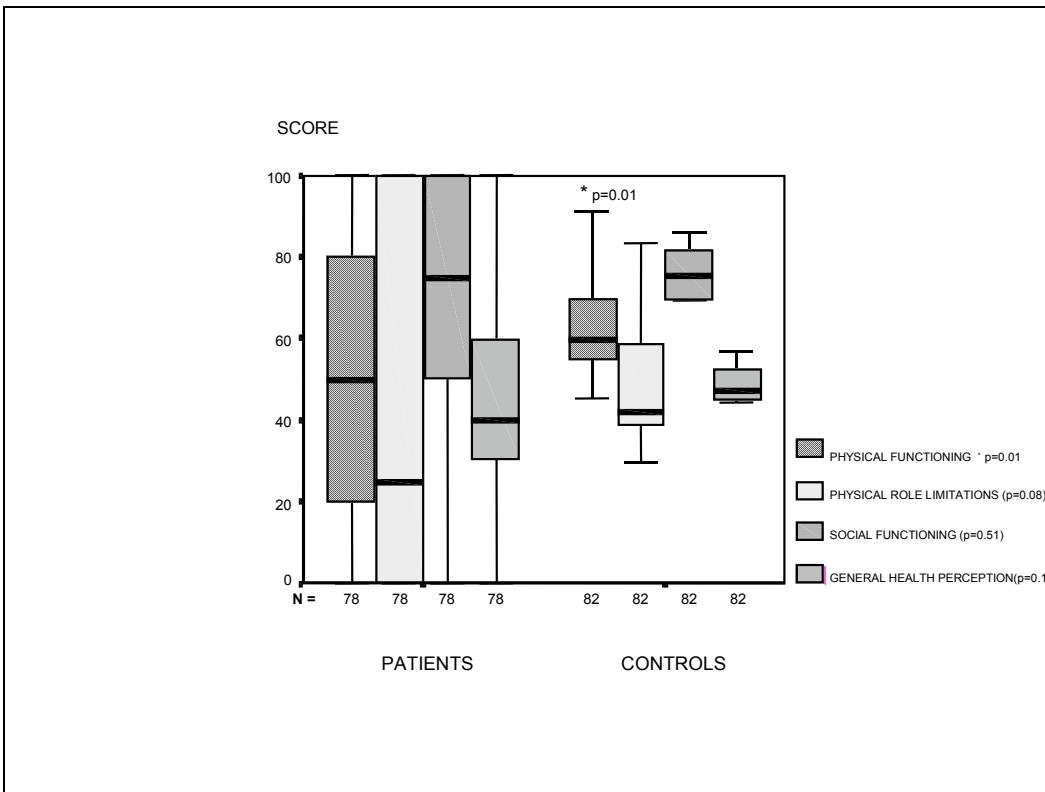
Figure 4. The patients' quality of life after RAAA according to the eight health domains of the RAND-36, compared with the norms of the Finnish age- and sex-adjusted population. The results as median, inter-quartile range and range. Reproduced from Korhonen et al. 2003, with permission.

PREDICTORS OF OUTCOME

Glasgow Aneurysm Score

The mean GAS among RAAA patients who died (395/836) was 91.1 (standard deviation 11.5), whereas it was 79.6 (12.8) among the survivors (441/836) (p<0.001). Quintiles of the GAS predicted 30-day operative mortality (p<0.001) (Figure 5). Predictors of mortality in multivariate analysis were preoperative shock OR 2.13 (95% CI 1.45–3.11) and an increase of 10 units in the GAS OR 1.81 (95% CI 1.54–2.12). The ROC curve showed that the best cut-off point of the GAS in predicting 30-day operative death was 84 (AUC 0.75, 95% CI 0.72–0.78, Standard Error 0.17) (Figure 6). Patients with a GAS ≤ 84 had a 30-day mortality rate of 28.2% (114/404), whereas it was 65.0% (281/432) among those with a score >84 (III). During the years 2003–2004 with an optimised treatment chain of RAAA, if cut-off points of GAS ≥105 and age ≥80 were used, 4 survivors out of 12 RAAA patients would have been missed (V). When all RAAA patients in the period of 1996 to 2004 were included and tested with different GAS cut-off points (95–115) and age ≥80, the mortality rate ranged from 75–82% (Figure 7). With a GAS ≥120 and age ≥80 all five patients died.

In order to test the potential confounding effect of factors related to different hospital practices, the nationwide data were divided according to the hospital volume. Six hospitals with more than 50 cases of repair of RAAA were analysed separately, whereas other hospitals with less than 50 repair cases of RAAA were clustered together as one. In 6 of the 7 hospitals tested, GAS predicted outcome (P<0.05): the lower the GAS, the better the outcome.

Preoperative GAS was an independent predictor of death after 48-h survival after RAAA repair (AUC 0.67 (95% CI 0.56–0.78)) together with organ dysfunction by SOFA score at 48-h, and suprarenal clamping during the operation. (IV)

APACHE II

The preoperatively measured APACHE II scores differed significantly between survivors and non-survivors after RAAA. All RAAA patients had an APACHE II score median of 14 (range 3–37), the survivors 12 (3–30), and non-survivors 16.5 (5–37), respectively (p<0.001). (II)
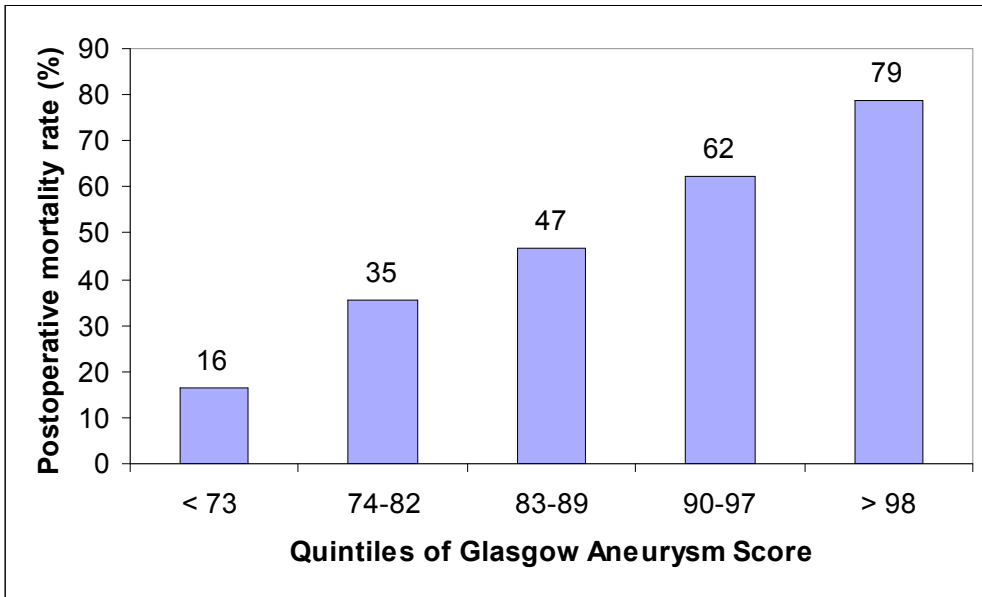
Figure 5. The 30-day mortality rates in different quintiles of the Glasgow Aneurysm Score in patients who underwent open repair for RAAA. Reproduced from Korhonen et al. 2004, with permission.
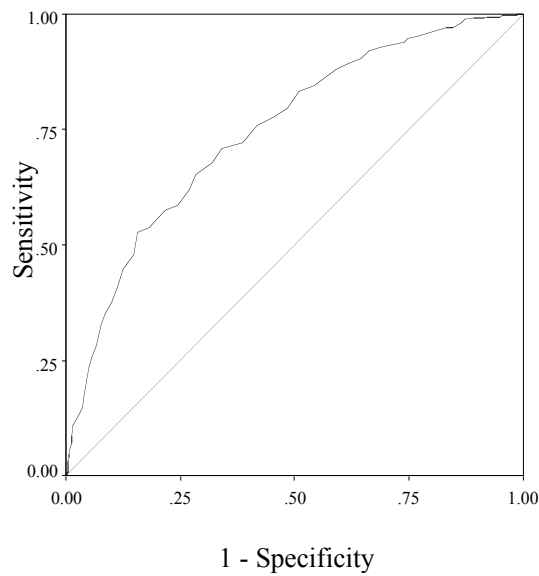
Figure 6. Receiver operating characteristics (ROC) curve for the Glasgow Aneurysm Score in predicting postoperative mortality (Area Under the Curve 0.75, 95% CI 0.72–0.78, SE 0.017, p<0.001).
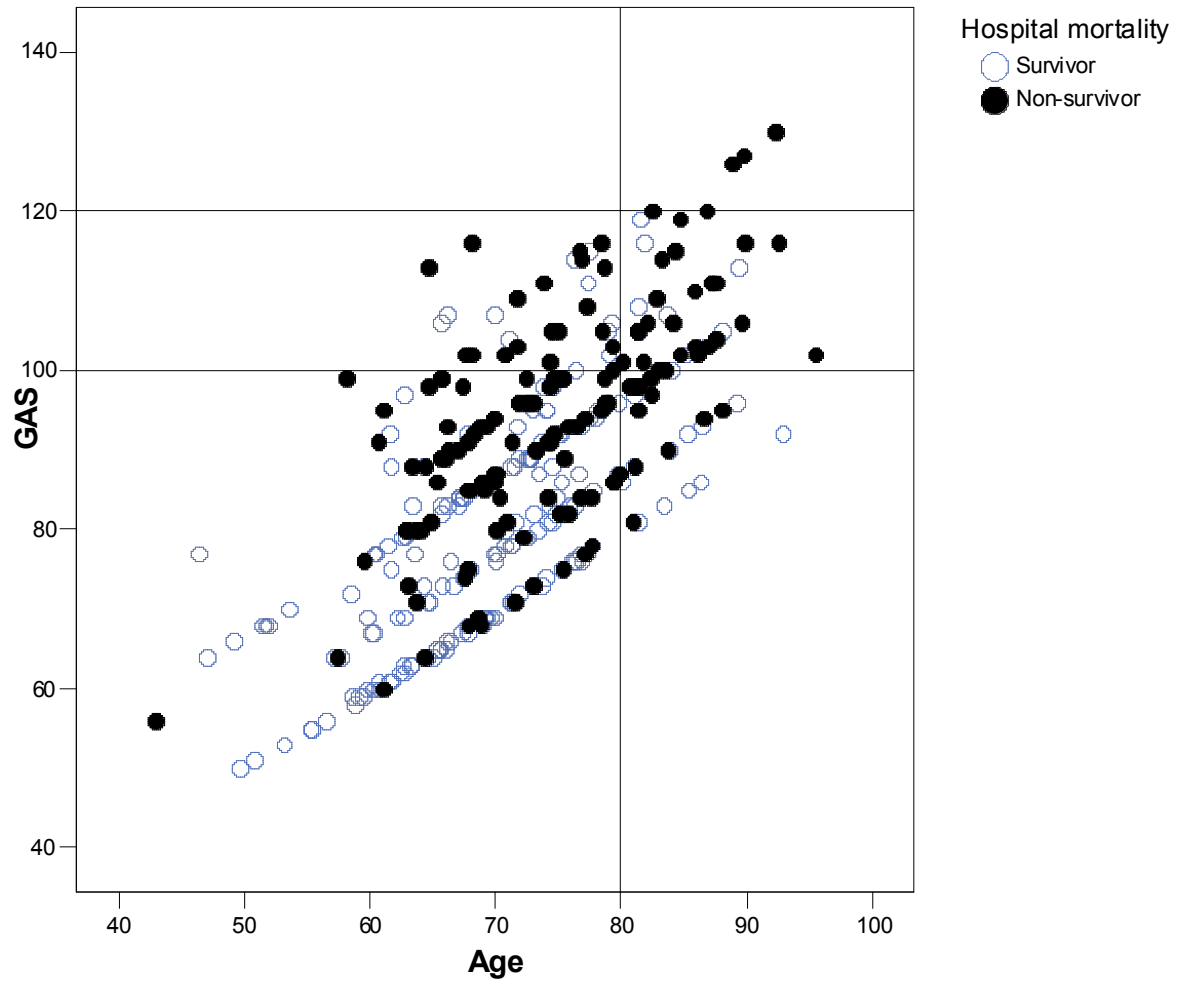
Age, years	Glasgow Aneurysm Score	Survivors (n)	Non-survivors (n)	Mortality (%)
≥80	≥95	13	39	75
≥80	≥100	10	31	76
≥80	≥105	7	21	75
≥80	≥110	3	14	82
≥80	≥115	2	9	82
≥80	≥120	0	5	100

Figure 7. Glasgow Aneurysm Score (GAS) and age related to hospital mortality after RAAA with different cut-off points of the GAS and age.

Predictors at 48 hours

The 30-day operative mortality of 197 RAAA patients was 42% (78/185) and hospital mortality 47% (93/197) (Figure 8). Of the 12 patients not operated, five were considered moribund, and seven RAAA deaths were diagnosed at autopsy. 10% (19/185) died intraoperatively. All survivors at 48 hours (n=138) were included in the analysis.
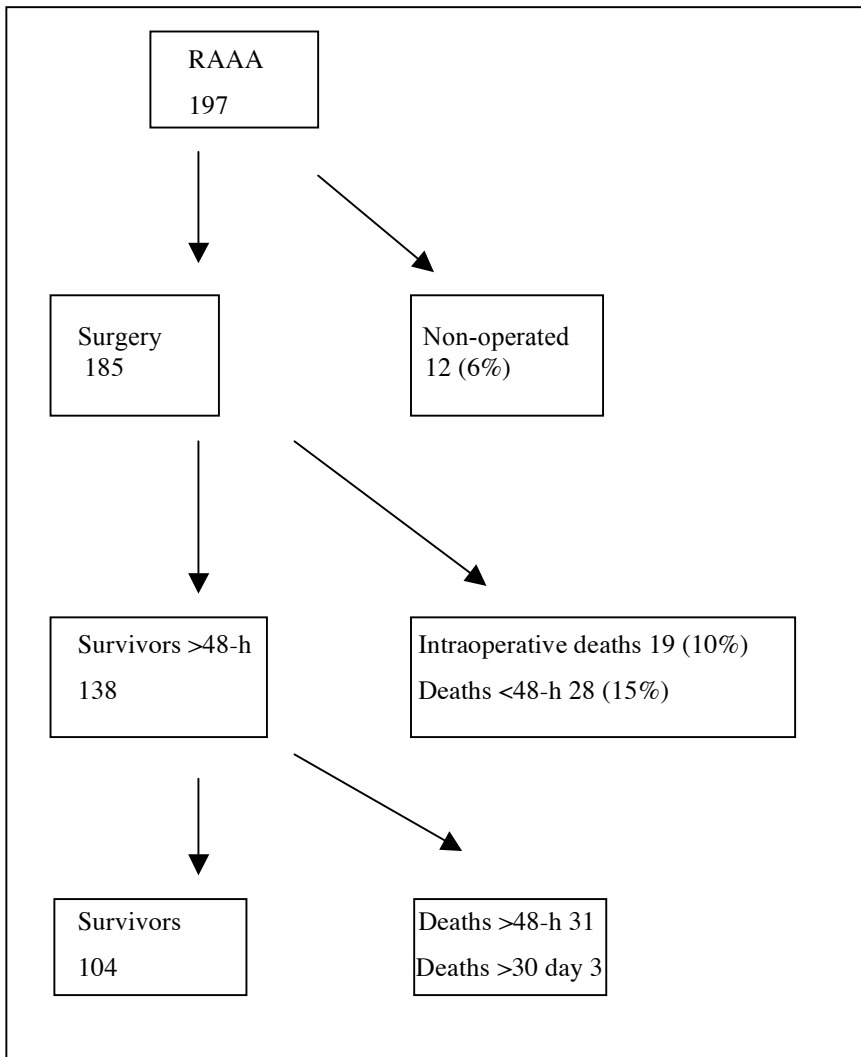
Figure 8. A total of 197 RAAA patients in 1999–2003.

When the characteristics and preoperative values of the RAAA patients were compared, age, preoperative GAS, and shock differed significantly between the survivors and non-survivors. Suprarenal clamping was used more often for non-survivors (42% versus 19%, p=0.009).

In the multiple logistic regression analysis, the SOFA score at day 2 (48-h) (p<0.001), preoperative Glasgow Aneurysm Score (p=0.03), and suprarenal clamping (p=0.04) were independent predictors of 30-day operative mortality 48 h after repair of RAAA. The Areas under the ROC curves (AUC) of significant measures of multivariate analysis were 0.79 (95% CI 0.70–0.89) for day 2 SOFA score, and 0.67 (95% CI 0.56–0.78) for preoperative Glasgow Aneurysm Score, respectively. At day 2 (48-h), a cut-off point for the SOFA score >11 predicted mortality with a sensitivity of 39% and a specificity of 93%, and at day 7 63% and 98%, respectively.

The mean daily measured SOFA scores for survivors and non-survivors differed from day 1 onwards, displaying the greatest difference at day 7 (Figure 9). Out of 138 SOFA scores, 118 overlapped at day 2 (48-h) after repair of RAAA (Figure 10). Development of cardiovascular and/or renal failure differed significantly between the survivors and non-survivors as measured by daily SOFA scores (Figure 11).
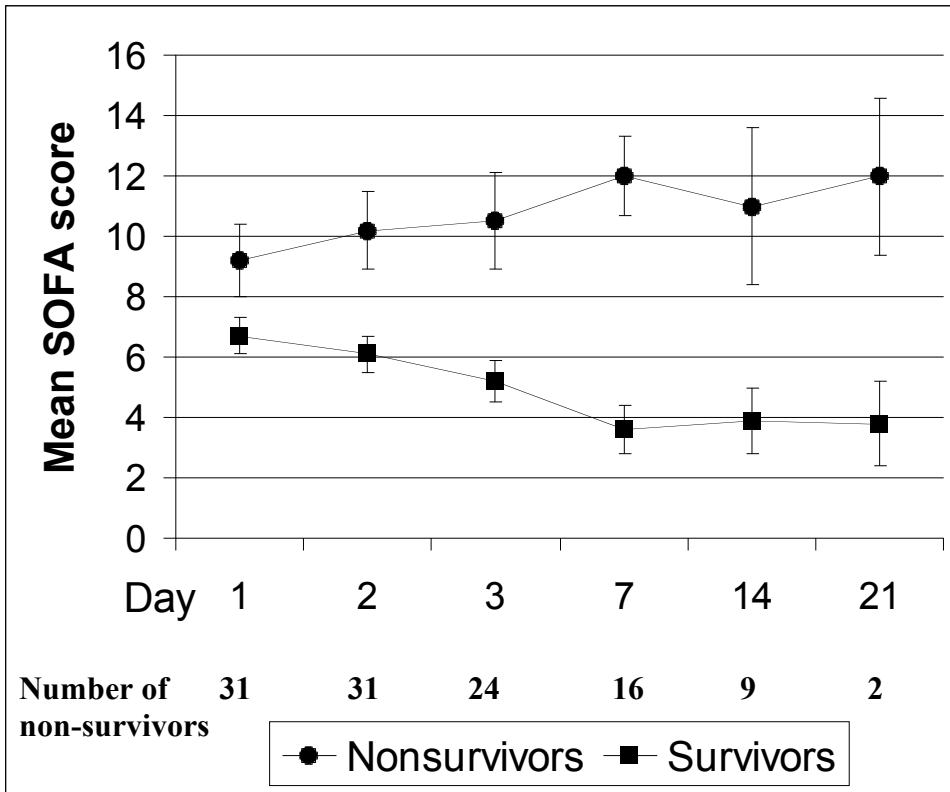
Figure 9. Development of mean (CI 95%) daily SOFA scores in 138 patients after repair of RAAA. Reproduced from Laukontaus et al. 2005, with permission.
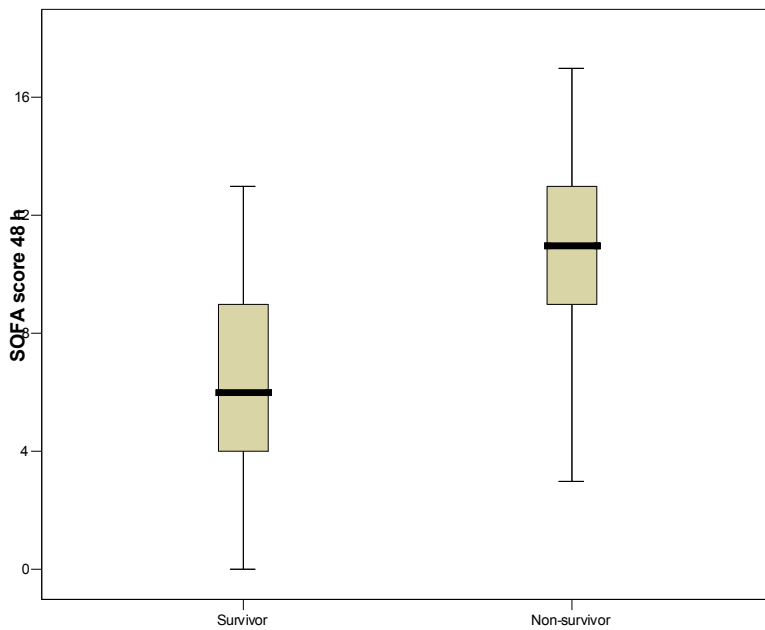
Figure 10. SOFA Score at 48 h (day 2) after repair of RAAA. The results are shown as median, interquartile range, and range.
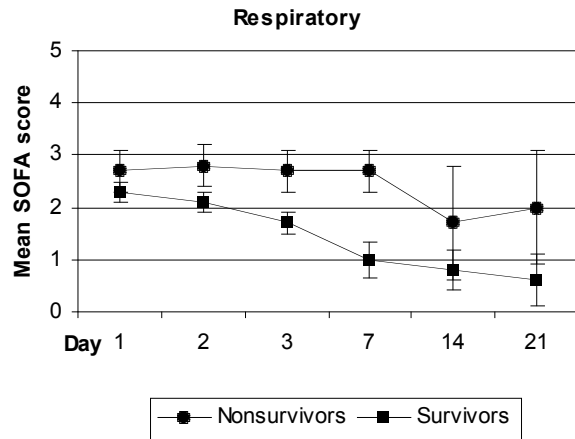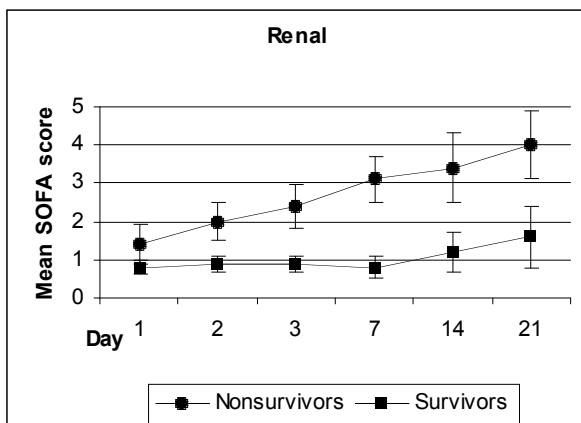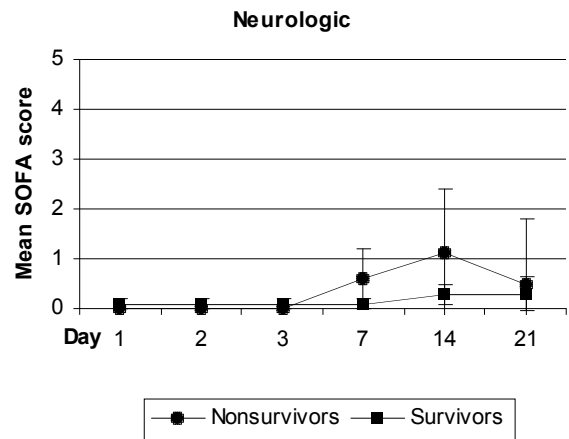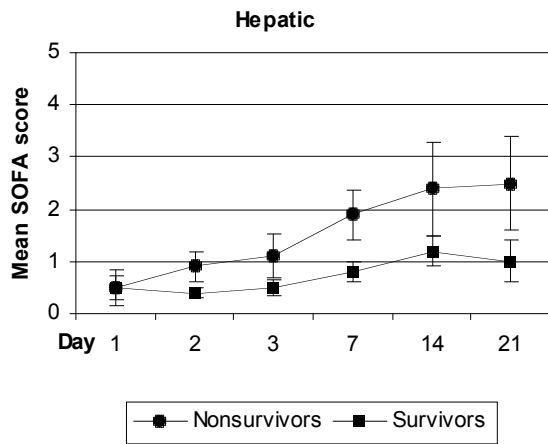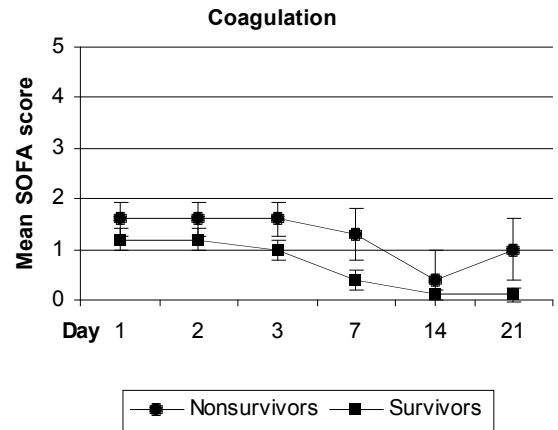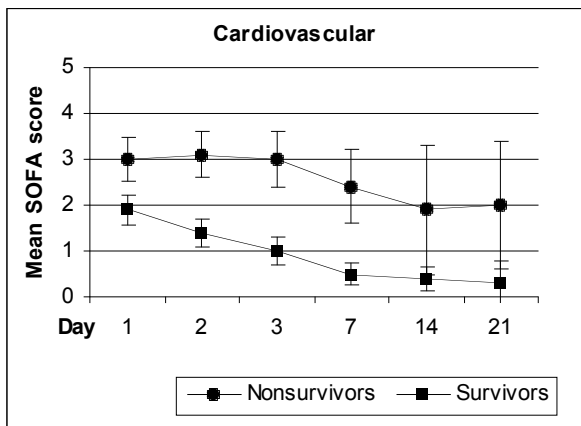
Figure 11. Development of single organ dysfunction as measured by mean (95% CI) SOFA scores in survivors and non-survivors after surgery for RAAA. The significant differences are framed. Reproduced from Laukontaus et al. 2005, with permission.

COST-EFFECTIVENESS ANALYSIS

The costs were analysed further, as measuring the QALYs enables the calculation of cost-effectiveness ratios. The costs were assessed using the following pricelist of HUCH, 2006: RAAA operation 4000 €, one day at the ICU 2 500 €, one day at the ward 350 €, and deaths in the emergency room 400 €. The costs of RAAA were 15 650 € per patient, 19 400 € per survivor, and 11 000 € per non-survivor (Table 15). With the hospital mortality rate of 45% (157/352) the mean cost for a survivor of RAAA was 28 300 €. The cost-effectiveness ratio was thus 6 900 €/QALY (28 300 €/4.1 QALY). To assess the reliability of these cost estimates, further calculation of the actual costs was done for 70 consecutive RAAA patients, resulting in a mean cost of 17 700 € (range 140–76 500 €).

In the subgroup of age ≥80 years and GAS ≥100, the cost of RAAA was 9 500 € per patient, 16 000 € per survivor, and 7 300 € per non-survivor (Table 15). With the hospital mortality rate of 76% (31/41) the mean cost for a survivor of RAAA was 38 800 €. The cost-effectiveness ratio was thus 35 300 €/QALY (38 800 €/1.1 QALY).

One RAAA patient used a mean of 3.9 ICU days and 6.5 ward days (Table 15). A survivor used a mean of 4.8 ICU days and a mean of 10.4 ward days, and a non-survivor 3.1 and 2.2 days, respectively. When the ICU and ward days of all patients were divided among the survivors, the mean figures were 7.4 and 13.5 days, respectively. When the ICU and ward days of all patients in the subgroup of age ≥80 years and GAS ≥100 were divided among the survivors, the mean figures were 7.4 and 14.5 days, respectively.

For 28 patients aged ≥80 years and Glasgow Aneurysm Score ≥100, the mean QALY was 1.1 (0–7.9). Five of the RAAA patients survived and gained together 29.5 QALYs, which comes to about 8 500 €/QALY in the cost-effectiveness analysis (Table 16). (II)

Table 15. Costs and length of stay in the intensive care unit (ICU) and in the ward for all survivors and non-survivors, for the group age ≥80 years and for the subgroup of age ≥80 years, and the Glasgow Aneurysm Score ≥100 after RAAA, in 1996–2004.

	Survivor			Non- survivor		
	All	Age ≥80	Age≥80, GAS ≥100	All	Age ≥80	Age≥80, GAS ≥100
N	195	23	10	157	44	31
ICU days, mean	4.8	3.5	3.6	3.1	1.5	1.2
median	2.0	2.0	2.0	0	0	0
(range)	(0–36)	(0–14)	(0–14)	(0–30)	(0–17)	(0–15)
Ward days, mean	10.4	9.0	8.6	2.2	2.0	1.9
median	8.0	6.0	6.5	1.0	1.0	1.0
(range)	(0–41)	(3–30)	(3–30)	(0–60)	(0–15)	(0–15)
No need for ICU (n)	30	4	3	85	33	24
Cost €/RAAA	19 400	16 000	16 000	11 000	7 500	7 300
QALY, mean	4.1	1.4	1.1	0	0	0
Cost €/QALY	4 700	11 400	14 500			

ICU = Intensive care unit; GAS = Glasgow Aneurysm Score; QALY = Quality-adjusted life year

Table 16. The total number of QALYs and the cost-effectiveness ratio for RAAA patients aged ≥80 years and with different Glasgow Aneurysm Scores.

Age, years	GAS	Survivors n	Non-survivors n	Total QALYs	Cost-effectiveness ratio (€/QALY)
≥80	≥95	8	29	34.75	8 900
≥80	≥100	5	23	29.51	8 500
≥80	≥105	4	14	22.46	4 800
≥80	≥110	2	10	9.25	11 400
≥80	≥115	2	6	9.25	8 200
≥80	≥120	0	2	0	

GAS = Glasgow Aneurysm Score; QALY = Quality-adjusted life year

DISCUSSION

From rupture to emergency room

In the Helsinki and Uusimaa district, some 70 persons had RAAA annually during the study years 1996–2004. Almost one third of the deaths resulting from RAAA occurred at home and were diagnosed at autopsy. One fifth of the RAAA deaths occurred in other hospitals, outside HUCH. Only a small minority of the patients admitted to other hospitals were operated on. These results were parallel to those reported recently by Heikkinen et al. (2002) in the Pirkanmaa district.

More than half of all RAAA patients were admitted to the emergency unit of HUCH, which has had 'all comers policy', and principally all were candidates for surgery. In the present study 6% of the patients diagnosed to have RAAA were not operated. The operative rate was one of the highest in the published literature, and in line only with the series of Barry et al. (1998) and Noel et al. (2001). Attempts have been made to identify predictors of mortality, but no single preoperative parameter to indicate definite outcome is available (Halpern et al. 1997, Sasaki et al. 1999 Heller et al. 2000, Calderwood et al. 2004, Janczyk et al. 2004). Knowledge of a single risk factor adds little to the decision-making process in the emergency room, whereas knowledge of the global operative risk may ideally help the surgeon to decide whether to operate or not if the patient has a high operative risk.

A selection criterion is to identify patients who are not cases for salvage attempts. To discover individual operative risk by using the risk stratification scoring system is practical in the emergency setting, but only if it can be done reliably. The preoperative Glasgow Aneurysm Score (GAS) is a simple prediction tool with only five easily available preoperative variables. In fact, the patient's age and evidence of shock were variables associated with the highest weight (Samy et al. 1994, Samy et al. 1996) and were also easily retrievable. Preoperative risk factors such as co-morbidities could not be reliably evaluated in the emergency room, as observed by Kantonen et al. (1997). The co-morbidities nevertheless had relatively less predictive weight, as seen in other studies as well (Samy et al. 1994, Samy et al. 1996).

The scoring system will hardly lead to the identification of a cut-off point beyond which the mortality is 100%. To the best of my knowledge, Study III is the most extensive study investigating the GAS after RAAA so far. If the GAS was 84 or more, 65% of patients died. If the GAS was $\geq$100 and age $\geq$80 years, the mortality rate was 76%. Only if the GAS was $\geq$120, irrespective of the patient's age, mortality was 100%. However, there were only five patients and they were all over 80 years old. Tambyraja et al. (2005c) reported that in 14 patients with GAS $\geq$110 the operative mortality was below 50%, but they excluded the patients not selected for operative treatment. The subgroups of patients with advanced age and high GAS were relatively small for drawing definitive conclusions on how to identify patients facing immediate fatal outcome.

Although one third of all RAAA patients aged over 80 years survived after RAAA, it was possible even at the oldest age quintile (80–95 years) to gain 7.9 QALYs. These results are in a line with other studies on over 80-year-olds: Dueck et al. (2004c) reported a 30-day operative mortality of 48%, Roddy et al. (2003) 41%, and Barry et al. (1998) 51%. However, these figures excluded elderly patients unfit for surgery, and are therefore skewed. Operative mortality rates for over 80-year-old patients were only slightly worse than for younger ones, which supports the active surgical approach. From the ethical point of view, age as such should not be used as a means for prioritisation (Mikkola and Bergström 1994).

Initial survivors

Some 10% of the operative RAAA patients died intra-operatively, and a further 32% died during the next month, which is in accordance with previous studies (Bown et al. 2004). Postoperative treatment after repair of RAAA should be carried out in the intensive care unit (ICU) or high dependency unit. In Study IV only 2% (3/138) of the patients were not treated in the ICU, and they all survived as the fittest patients were transferred directly to the ward. Optimally the decision on whether to withdraw or continue treatment should be made only after at least 24 to 48 hours of maximal ICU treatment. In the ICU, the cause of death was usually organ dysfunction (Bown et al. 2004, Meesters et al. 1994). Study IV revealed that organ dysfunction by day 2 SOFA score was an independent predictor for 30-day mortality. Daily measured SOFA scores, however, showed that the ability to distinguish between patients who die and those who survive was the best on day 7. Thus, day 2 seems too early for making decisions on continuing or

withdrawing the treatment. Despite its good predictive value, the SOFA score was unable to discriminate survivors from non-survivors at individual level. The SOFA score has proven its potential to describe organ dysfunction in critically ill patients (Vincent et al. 1998, Moreno et al. 1999). In fact, this was the first study used SOFA to predict mortality after RAAA.

The SOFA score was assigned daily, which was valuable in the standardised assessment of severity and duration of organ failure (Bernard 1998). The cut-off point for SOFA score is difficult to select, i.e. whether to simply exceed the score value or maintain it for a few days. At day 2 a SOFA score >11 identified 39% of non-survivors, and at day 7 63% with a high specificity. Junger et al. (2002) selected a cut-off point >11.5 with a sensitivity of 37% and a specificity of 99% in the surgical ICU with unselected patients. Cabré et al. (2005) demonstrated in a prospective study that 34 unselected ICU patients aged over 60 years and with a SOFA score above 10 for 5 days had a mortality of 100%. Recently Kaarlola et al. (2006) reported that all patients aged ≥65 years with a SOFA score greater than 15 died during their stay in the ICU. The patient material consisted of medical and surgical patients, 132 out of 882 were in the ICU for postoperative care after repair of AAA, but the share of RAAA patients was not available.

Preoperative GAS maintained its value in predicting postoperative mortality for initial survivors at day 2, although the discrimination was only moderate. GAS was designed to model mortality of RAAA in a preoperative setting. The timing of the construction of a risk-stratification model dictates the potential uses of such models. The construction of such a model at the earliest time-point in the treatment chain of a RAAA patient is unlikely to be accurate, due to the large number of events that the RAAA patient will have.

The limitations of assessing different discrimination models need to be addressed. First, scoring measures such as GAS and SOFA were assigned retrospectively. As in most retrospective analyses, complete data were not available. In the present study, a missing variable was either considered normal (Glasgow Aneurysm Score II–IV) or a previous day's value was used (SOFA IV). This strategy would rather underestimate than overestimate the score and its predictability. The decision to operate, however, was clinical, and thus the scores did not affect outcome. Second, as SOFA scores were not collected daily, some valuable data might be missed. In general, as the number of patients in the study was large, the operative rate rather high, and the amount of missing data small, the results seem reliable.

Quality of life

As more than half of the hospitalised RAAA patients survived, the main question is whether these patients were able to live unrestricted lives postoperatively. The survivors lived at home at the time of the questionnaire in the present study. Dillavou et al. (2006) reported that one third of all RAAA patients (including non-survivors) were discharged home, and every tenth transferred to long-term care. Among the survivors of RAAA, QoL was nearly the same when compared to the norms of the age- and sex-adjusted general population; only impairment in physical functioning was observed (RAND-36) (I). Older patients had lower QoL in their physical functioning, which was also seen in the control population (Aalto et al. 1999). Co-morbidities also influenced adversely the functioning of the general population older than $\geq$70 years (Dunlop et al. 2002). When a different QoL method (EQ-5D) was used, a lower QoL index was obtained than that for the age- and sex-adjusted Finnish general population, although EQ-VAS did not differ (II). These results with slight impairment were in line with previous studies (Magee et al. 1992, Tambyraja et al. 2005b). Tambyraja et al. (2005b) reported functional outcome using the SF-36 measure. They found that role limitations due to physical and emotional problems after RAAA were worse than in the general population but equal with the group of elective repair of AAA. They also noted lower scores more often in other health domains after elective repair of AAA than after RAAA when compared to the general population. The good functional outcome after RAAA may be interpreted so that surviving from near-death has a positive effect on the perception of QoL. The other studies demonstrated QoL equal to that of the general population (Currie et al. 1992, Bohmer et al. 1999, Joseph et al. 2002, Hinterseher et al. 2004) or to the group of elective repair of RAAA (Hennessy et al. 1998, Tambyraja et al. 2005b).

QoL is a subjective concept, based on the different perceptions of each patient's QoL. Discordance between the QoL studies (I, II) could be explained by the difference in the set up of the health questionnaires. A wide range of scores may have reflected different outcomes after major surgery, and as the RAND-36 and the EQ-5D are generic measures, this may have influenced the sensitiveness of the measure. The RAND-36 and the EQ-5D were compared in critically ill patients (Kaarlola et al. 2004). The measures correlated well, but the RAND-36 discriminated better, especially in regard to physical functioning, self-care, or low quality of life levels. A disease-specific measure might indicate disease severity better as observed in patients with peripheral arterial disease (de Vries et al. 2005). QoL measures are crude, but the present

data strongly contradict the belief that life after the repair of RAAA would be so poor that the attempt would not be worthwhile.

Age or preoperative heath status based on GAS did not differ during the study years. The response rate was high, even though the questionnaires were filled out with the aid of a proxy in more than one-fourth of the cases. Bohmer et al. (1999), however, reported that the next of kin were prone to assess global QoL worse than the patients themselves. The importance of the different aspects questioned may also differ between the patient and the proxy. The study was retrospective, with cross-sectional QoL assessment, which may introduce bias. In this kind of study, it is impossible to evaluate QoL in emergency settings before the repair of RAAA. Patients who survived after RAAA and were able to answer the questions, represented a naturally selected population. Thus comparison with the appropriately adjusted general population as controls was fundamental. Those patients who died after hospitalisation, but before QoL evaluation, might have presented with lower scores due to the presence of co-morbidities. However, this is by far the largest study investigating QoL after repair of RAAA.

The RAND-36 (based on the SF-36) was chosen because the availability of the validated Finnish version with age- and sex-adjusted population norms made comparison possible. The SF-36 has confirmed reliability, construct validity, responsiveness, and sensitivity (Black et al. 2001). The SF-36 is the most widely used QoL instrument in the medical literature, and it has been recommended in the QoL assessment of vascular diseases (Beattie et al. 1997). Results of the RAND-36 cannot be used as a single index for measuring QALYs and comparisons with other studies are difficult. Therefore we used the EQ-5D to assess the feasibility of surgery for RAAA using QALYs.

Cost-utility

RAAA patients will die without operative treatment, thus gaining 0 QALYs. Surviving patients gained a mean of 8.5 QALYs, but measuring QALYs only among survivors exaggerates the ability of the vascular service to produce QoL. Thus all RAAA patients were included, and with non-survivors a mean of 4.1 QALYs was obtained. Clear association was seen between QALYs and preoperative GAS, high GAS indicating low QALYs to be achieved.

Measuring QALYs is an underused approach for determining outcome after RAAA. To the best of my knowledge, this was the first study measuring QALYs after repair of RAAA. Life expectancy after the follow-up was an estimation calculated by using the age- and sex-adjusted life expectancy from Statistics Finland. Many studies have shown, as did Study I, that patients surviving more than 30 days have a life expectancy similar to that of the age- and sex-adjusted population (Rohrer et al. 1988, Stonebridge et al. 1993, Hinterseher et al. 2004). If the life expectancy had been worse among the survivors of RAAA, it would have affected the number of QALYs gained.

The repair of RAAA was a cost-effective procedure. The cost-effectiveness ratio per survivor was considerably lower than the 40 000–80 000 €/QALY (50 000–100 000 USD/QALY) limit usually considered acceptable (Lee et al. 1996, Hamel et al. 1997, Azimi and Welch 1998, Finlayson and Birkmeyer 1998, Angus et al. 2003). It is comparable to the hypothetical model of Patel et al. (2000) (8 500 €/QALY). There were unfortunately limitations in the calculation of the costs, as the treatment after hospitalisation at the lower health care level was not accurately available. However, in their hypothetical model, Patel et al. (2000) calculated hospitalisation costs of 22 700 € and lifetime cost of 28 800 €. Accordingly, the cost-effectiveness ratio involving the lifetime costs would be 8 300 €/QALY in the present material.

Cost-effectiveness was also demonstrated in patients 80 years or older. The cost-effectiveness ratio with a mortality rate of 76% was further lower than 40 000 €/QALY limit. The mean length of stay per aged survivor in the ICU was comparable with all RAAA patients, and the net costs were even lower.

In general, the treatment of RAAA corresponds well with other surgical and medical treatments (Table 17) (Kuntz and Kent 1996, Patel et al. 1999, Patel et al. 2000, Schermerhorn et al. 2000, Eefting et al. 2003, Åhlstrom et al. 2005).

Table 17. Cost-effectiveness ratios for different medical procedures.

Procedure	Authors, year	Cost-effectiveness ratio (€/QALY)
CEA for symptomatic patients (ICA stenosis 70–99%)	Kuntz et al., 1996	3 300*
Open repair of RAAA	The present study	6 900
Open repair of RAAA	Patel et al., 2000	8 500*
Open repair of AAA	Schermerhorn et al., 2000	8 500*
Coronary bypass surgery	Eefting et al., 2003	11 400*
Endovascular repair of AAA	Patel et al., 1999	18 200*
Renal replacement therapy	Åhlström et al., 2005	25 500
CEA for asymptomatic patients (ICA stenosis 60–99%)	Kuntz et al., 1996	42 200*

RAAA = ruptured abdominal aortic aneurysm; AAA = abdominal aortic aneurysm; CEA = carotid endarterectomy; ICA = internal carotid artery; * = USD converted to Euros

Vascular service

Our vascular unit provides the only 24-hour, 365-day emergency vascular service in the Helsinki and Uusimaa district. When suspected RAAA patients' arrive to the emergency room, bedside ultrasonography examination is undertaken for all RAAA patients, and computer tomography in selected cases. Computer tomography has been located in the emergency room since 2003. After the decision to operate, an RAAA patient is transferred to the operating theatre, and prepared for surgery before general anesthesia with a minimum of preoperative fluid resuscitation.

After the organisational changes in the hospital chain were carried out, including regional centralisation and quality improvements, there was a decrease in the population-based mortality, the 90-day hospital mortality, and 30-day operative mortality. It is nevertheless not clear what the true causes of the improvement were. Hypotensive haemostasis, as the initial approach, has been advocated during the past few years. There was no preadmittance data available to clarify how well this treatment regimen was really used. In other series its benefit has been demonstrated (Roberts et al. 2006). The incidence of RAAA and demographic variables related to outcome did not change over the study period. The mortality rates during the years 2003–2004 were lower than those published earlier in Finland (Kantonen et al. 1999b, Heikkinen et al. 2002). Bown et al. (2002) reported a gradual reduction in RAAA mortality between the years 1955–1998. However, several studies have shown no reduction in RAAA mortality over the years (Heller et al. 2000, Hallin et al. 2001, Visser et al. 2005).

Most studies reporting only operation-related mortality of RAAA surgery are skewed by the patient selection. 30-day mortality alone is a notoriously biased figure and apt to data enhancement (Campbell 1991). It is therefore mandatory to analyse the fate of all patients admitted alive to any given hospital, if the level of vascular or emergency services is analysed. Hospital mortality is a far better measure of vascular service than operative mortality. The inclusion of non-operated patients with known AAA, and even those diagnosed at autopsy, worsens the mortality rates (Callam et al. 1991). In the present study there was a small number of non-operated patients with diagnosed RAAA. Great variation was seen in operative activity, with exclusion rates ranging 7–43% (Hardman et al. 1996, Bradbury et al. 1997, Barry et al. 1998, Semmens et al. 1998, Adam et al. 1999, Basnyat et al. 1999, Kantonen et al. 1999b, Evans et al. 2000, Noel et al. 2001, Heikkinen et al. 2002, Neary et al. 2003, Bown et al. 2004, Dueck et al. 2004a, Tambyraja et al. 2005d). None of these specially included patients with undiagnosed RAAA. As vascular service is analysed as an entity, those patients missed by this service, irrespective of the reason, reflect the ability of the treatment chain to function as whole, and therefore even these patients need to be included.

The total RAAA incidence rate of 5.4/100 000 inhabitants/year in the Helsinki and Uusimaa district was similar to that reported by Bengtsson et al. (1993) (5.6/100 000) and slightly lower than 6.1/100 000 in Finland in 1991–1994 by Kantonen et al. (1999b) or 6.3/100 000 in the Pirkanmaa district in 1991–1997 by Heikkinen et al. (2002). These incidences were based on all identified RAAA patients, also RAAA deaths outside hospital. The exact autopsy rate of the patients who died suddenly remains unknown during the study period. However, the autopsy rate in Finland was 31% of all deaths, and it was the highest in the Nordic countries (Saukko 1995). Registration of the cause of sudden death of elderly people (>65 years) may also be based on prior knowledge of a chronic/fatal disease without autopsy. Some RAAA deaths must therefore have escaped the data, but there was no indication that the number of missed RAAA deaths would have changed over the study years.

Emergency surgery of AAA should be carried out by vascular specialists (Campbell and Chester 2002). Our results of improved survival support the data of better outcomes in high-volume hospitals and operated by high-volume surgeons (Ouriel et al. 1990, Rutledge et al. 1996, Heller et al. 2000, Birkmeyer et al. 2003). The transfer times and distances from the outskirts of the Helsinki and Uusimaa hospital district are probably longer than before the regional centralisation, but this is not a time problem, as the longest distance is less than 140 km.

The results of prehospital care in Helsinki are good, and have been shown to be parallel in Europe in the chain-of-survival after cardiac arrest (Herlitz et al. 1999). Emergency medical service including prehospital care has improved over the years (Kuisma and Martikainen 1998) and may have had an impact on survival. However, most of the studies on the transfer of RAAA patients have shown no adverse effect of transfer time or distance on survival (Adam et al. 1999, Kantonen et al. 1999a, Cassar et al. 2001, Vogel et al. 2005).

Future prospects

The selective approach to operative treatment seems reasonable in some subgroups of RAAA patients, but the problem of selection still remains. Discriminator models for prognostication did not identify the patients with immediate fatal outcome at individual level. Prospective studies evaluating different preoperative scoring methods might be valuable. Measures predicting mortality might be used for auditing or for comparisons between hospitals.

Endovascular repair of RAAA has begun to emerge as a treatment option for RAAA: 4-6% of RAAA patients were treated by endovascular repair in the USA in 2003 (Greco et al. 2006, McKinsey et al. 2006). Long-term overall mortality was lower in the endovascular group compared to open repair, but selection bias is obvious (Greco et al. 2006). The HUCH participated in a prospective multicentre study in Europe investigating endovascular repair for RAAA (Peppelenbosch et al. 2004, Peppelenbosch et al. 2006). The 30-day mortality was 35% in the endovascular group and 39% in the open repair group. It was noteworthy that a large number of RAAA patients was excluded from the study. However, successful endovascular repair needs a skilful radiology team which is available 24 hours a day. The suitability of RAAA for endovascular repair is also a matter of concern (Wilson et al. 2004).

Cost-utility analysis, i.e. calculating QALYs and the cost-effectiveness ratio, allows future comparisons with other types of interventions, such as endovascular aneurysm repair.

In Finland the number of people over 65 years of age will increase in the next decades by 70%. In the beginning of the study (1996) 14.5% of the Finnish population were over 65 years and at the end of the study (2004) 15.9% (Statistics Finland). In 2020 it is estimated that 23% of the population will be over 65 years old (Statistics Finland). If the RAAA incidence remains

72

unchanged, the number of RAAAs will increase dramatically. Even though the repair of RAAA is a cost-effective procedure, it may be questioned how all RAAA patients will be treated in the future while the resources of the society decrease. An active treatment policy of RAAA is advisable as the preoperative prediction of RAAA is unreliable.

CONCLUSIONS

In a nationwide study population, the 30-day operative mortality was 44% for ruptured abdominal aortic aneurysm (RAAA) patients (III). The 30-day operative mortality and hospital mortality were 38% and 45%, respectively in the Helsinki University Central Hospital between the years 1996 to 2004 (V), whereas the population-based mortality was 69% in the Helsinki and Uusimaa district (V). The hospital mortality of RAAA patients over 80 years old was 66%. After organisational changes were undertaken, including regional centralisation, and in-hospital quality improvements, mortality decreased at all levels, even at the population level (V). If the level of vascular or emergency services is to be analysed, it is mandatory to analyse the fate of all patients admitted alive to hospital, not only those selected for operative treatment.

Among the survivors of RAAA repair, the quality of life was nearly the same as the norms of an age- and sex-adjusted general population (I, II), despite impairment in physical functioning (I).

RAAA patients will die without operative treatment, thus gaining 0 quality-adjusted life years (QALY). Successful repair of RAAA gives a mean of 4.1 (0–30.9) QALYs for all RAAA patients, including non-survivors (II). The repair of RAAA is a cost-effective procedure, also in patients aged ≥80 years.

The preoperative Glasgow Aneurysm Score was an independent predictor of 30-day operative mortality at admission (III) and at 48 hours (IV) after repair of RAAA. Organ dysfunction measured by the Sequential Organ Failure Assessment (SOFA) score was the best predictor of 30-day operative mortality in RAAA patients alive at 48 hours after repair of RAAA (IV). A high Glasgow Aneurysm Score and increased age were associated with a low number of QALYs after repair of RAAA (II). These results strongly support an active operation policy, as there is no reliable measure to predict the outcome after RAAA.

ACKNOWLEDGEMENTS

My very warm thanks go to my dear friends Anna-Maija Korhonen, Sanna Lepola, Outi Lehtiniemi, Mari Rissanen, and Maarit Tuorila for being true friends during all these years.

My heart-felt thanks go to my mother Heleena Luusua, my father Eero Korhonen, Tiina Kitkiöjoki, my sister Pinja, and brothers Pietu, Joonas, and Samuel Korhonen for their continuous support and love. I also want to thank Kaija Laukontaus, Raija Laukontaus, Pietu and others for their kind help in taking care of Sea while I was finishing this book.

Finally, my most deep-felt thanks belong to my family, my beloved husband Petri and our lovely daughter Sea. You are the sunshine of my life.

Helsinki, November 2006
Sani Laukontaus

REFERENCES

Aalto A, Aro S, Aro AR, Mähönen M. RAND 36-Item Survey 1.0. Suomenkielinen versio (Finnish version). STAKES (National Research and Development Centre for Welfare and Health). Helsinki, 1999.

Adam DJ, Mohan IV, Stuart WP, Bain M, Bradbury AW. Community and hospital outcome from ruptured abdominal aortic aneurysm within the catchment area of a regional vascular surgical service. J Vasc Surg 1999; 30:922-928.

Al-Koteesh J, Masannat Y, James NV, Sharaf U. Chronic contained rupture of abdominal aortic aneurysm presenting with longstanding back pain. Scott Med J 2005; 50:122-123.

Alric P, Ryckwaert F, Picot MC, Branchereau P, Colson P, Mary H, Marty-Ane C. Ruptured aneurysm of the infrarenal abdominal aorta: impact of age and postoperative complications on mortality. Ann Vasc Surg 2003; 17:277-283.

Ammann P, Pfisterer M, Fehr T, Rickli H. Raised cardiac troponins. BMJ 204; 328:1028-1029.

Angus DC, Linde-Zwirble WT, Clermont G, Ball DE, Basson BR, Ely EW, Laterre PF, Vincent JL, Bernard G, van Hout B, PROWESS Investigators. Cost-effectiveness of drotrecogin alfa (activated) in the treatment of severe sepsis. Crit Care Med 2003; 31:1-11.

Ashton HA, Buxton MJ, Day NE, Kim LG, Marteau TM, Scott RA, Thompson SG, Walker NM, 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; 360:1531-1539.

Aune S, Amundsen SR, Evjensvold J, Trippestad A. The influence of age on operative mortality and long-term relative survival following emergency abdominal aortic aneurysm operations. Eur J Vasc Endovasc Surg 1995; 10:338-341.

Aune S, Laxdal E, Pedersen G, Dregelid E. Lifetime gain related to cost of repair of ruptured abdominal aortic aneurysm in octogenarians. Eur J Vasc Endovasc Surg 2004; 27:299-304.

Azimi NA, Welch HG. The effectiveness of cost-effectiveness analysis in containing costs. J Gen Intern Med 1998; 13:664-669.

Ballaro A, Cortina-Borja M, Collin J. A seasonal variation in the incidence of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 1998; 15:429-431.

Barratt J, Parajasingam R, Sayers RD, Feehally J. Outcome of acute renal failure following surgical repair of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2000; 20:163-168.

Barry MC, Burke PE, Sheehan S, Leahy A, Broe PJ, Bouchier-Hayes DJ. An "all comers" policy for ruptured abdominal aortic aneurysms: how can results be improved? Eur J Surg 1998; 164:263-270.

Basnyat PS, Biffin AH, Moseley LG, Hedges AR, Lewis MH. Mortality from ruptured abdominal aortic aneurysm in Wales. Br J Surg 1999; 86:765-770.

Beattie DK, Golledge J, Greenhalgh RM, Davies AH. Quality of life assessment in vascular disease: towards a consensus. Eur J Vasc Endovasc Surg 1997; 13:9-13.

Bengtsson H, Bergqvist D, Sternby NH. Increasing prevalence of abdominal aortic aneurysms. A necropsy study. Eur J Surg 1992; 158:19-23.

Bengtsson H, Bergqvist D. Ruptured abdominal aortic aneurysm: a population-based study. J Vasc Surg 1993; 18:74-80.

Bernard GR. Quantification of organ dysfunction: seeking standardization. Crit Care Med 1998; 26:1767-1768.

Biancari F, Heikkinen M, Lepäntalo M, Salenius JP, Finnvasc Study Group. Glasgow Aneurysm Score in patients undergoing elective open repair of abdominal aortic aneurysm: a Finnvasc study. Eur J Vasc Endovasc Surg 2003a; 26:612-617.

Biancari F, Leo E, Ylönen K, Vaarala MH, Rainio P, Juvonen T. Value of the Glasgow Aneurysm Score in predicting the immediate and long-term outcome after elective open repair of infrarenal abdominal aortic aneurysm. Br J Surg 2003b; 90:838-844.

Birkmeyer JD, Stukel TA, Siewers AE, Goodney PP, Wennberg DE, Lucas FL. Surgeon volume and operative mortality in the United States. N Engl J Med 2003; 349:2117-2127.

Black NA, Jenkinson C, Hayes JA, Young D, Vella K, Rowan KM, Daly K, Ridley S. Review of outcome measures used in adult critical care. Crit Care Med 2001; 29:2119-2124.

Blankensteijn JD, de Jong SE, Prinssen M, van der Ham AC, Buth J, van Sterkenburg SM, Verhagen HJ, Buskens E, Grobbee DE, Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med 2005; 352:2398-2405.

Bohmer RD, Fleischl J, Knight D. Quality of life after emergency abdominal aortic aneurysm repair. Aust N Z J Surg 1999; 69:447-449.

Bonamigo TP, Becker M, Weber EL, Bianco C, Miranda F,Jr, Poli de Figueiredo LF. Outcome after surgical repair of sealed rupture abdominal aortic aneurysms: a case-control study. Clinics 2006; 61:29-34.

Bown MJ, Sutton AJ, Bell PR, Sayers RD. A meta-analysis of 50 years of ruptured abdominal aortic aneurysm repair. Br J Surg 2002; 89:714-730.

Bown MJ, McCarthy MJ, Bell PR, Sayers RD. Low atmospheric pressure is associated with rupture of abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2003; 25:68-71.

Bown MJ, Cooper NJ, Sutton AJ, Prytherch D, Nicholson ML, Bell PR, Sayers RD. The post-operative mortality of ruptured abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2004; 27:65-74.

Boyle JR, Gibbs PJ, King D, Shearman CP, Raptis S, Phillips MJ. Predicting outcome in ruptured abdominal aortic aneurysm: a prospective study of 100 consecutive cases. Eur J Vasc Endovasc Surg 2003; 26:607-611.

Boyle JR, Gibbs PJ, Kruger A, Shearman CP, Raptis S, Phillips MJ. Existing delays following the presentation of ruptured abdominal aortic aneurysm allow sufficient time to assess patients for endovascular repair. Eur J Vasc Endovasc Surg 2005; 29:505-509.

Bradbury AW, Makhdoomi KR, Adam DJ, Murie JA, Jenkins AM, Ruckley CV. Twelve-year experience of the management of ruptured abdominal aortic aneurysm. Br J Surg 1997; 84:1705-1707.

Brown LC, Powell JT, UK Small Aneurysm Trial Participants. Risk factors for aneurysm rupture in patients kept under ultrasound surveillance. Ann Surg 1999; 230:289-296.

Brown PM, Zelt DT, Sobolev B. The risk of rupture in untreated aneurysms: the impact of size, gender, and expansion rate. J Vasc Surg 2003; 37:280-284.

Cabre L, Mancebo J, Solsona JF, Saura P, Gich I, Blanch L, Carrasco G, Martin MC, and the Bioethics Working Group of the SEMICYUC. Multicenter study of the multiple organ dysfunction syndrome in intensive care units: the usefulness of Sequential Organ Failure Assessment scores in decision making. Intensive Care Med 2005; 31:927-933.

Calderwood R, Halka T, Haji-Michael P, Welch M. Ruptured abdominal aortic aneurysm. Is it possible to predict outcome? Int Angiol 2004; 23:47-53.

Callam MJ, Haiart D, Murie JA, Ruckley CV, Jenkins AM. Ruptured aortic aneurysm: a proposed classification. Br J Surg 1991; 78:1126-1129.

Campbell B, Chester J. Emergency vascular surgery. BMJ 2002; 324:1167-1168.

Campbell WB. Mortality statistics for elective aortic aneurysms. Eur J Vasc Surg 1991; 5:111-113.

Cassar K, Godden DJ, Duncan JL. Community mortality after ruptured abdominal aortic aneurysm is unrelated to the distance from the surgical centre. Br J Surg 2001; 88:1341-1343.

Chang JB, Stein TA, Liu JP, Dunn ME. Risk factors associated with rapid growth of small abdominal aortic aneurysms. Surgery 1997; 121:117-122.

Chang RW. Individual outcome prediction models for intensive care units. Lancet 1989; 2:143-146.

Chen JC, Hildebrand HD, Salvian AJ, Taylor DC, Strandberg S, Myckatyn TM, Hsiang YN. Predictors of death in nonruptured and ruptured abdominal aortic aneurysms. J Vasc Surg 1996; 24:614-620.

Cho JS, Gloviczki P, Martelli E, Harmsen WS, Landis ME, Cherry KJ,Jr, Bower TC, Hallett JW,Jr. Long-term survival and late complications after repair of ruptured abdominal aortic aneurysms. J Vasc Surg 1998; 27:813-819.

Choksy SA, Wilmink AB, Quick CR. Ruptured abdominal aortic aneurysm in the Huntingdon district: a 10-year experience. Ann R Coll Surg Engl 1999; 81:27-31.

Christenson JT, Norgren L, Ribbe E, Steen S, Thorne J. A ruptured aortic aneurysm that "spontaneously healed". J Cardiovasc Surg 1984; 25:571-573.

Copeland GP, Jones D, Walters M. POSSUM: a scoring system for surgical audit. Br J Surg 1991; 78:355-360.

Cronenwett JL, Sargent SK, Wall MH, Hawkes ML, Freeman DH, Dain BJ, Cure JK, Walsh DB, Zwolak RM, McDaniel MD. Variables that affect the expansion rate and outcome of small abdominal aortic aneurysms. J Vasc Surg 1990; 11:260-268.

Currie IC, Scott DJ, Robson AK, Horrocks M. Quality of life of octogenarians after aneurysm surgery. Ann R Coll Surg Engl 1992; 74:269-273.

Dardik A, Burleyson GP, Bowman H, Gordon TA, Williams GM, Webb TH, Perler BA. Surgical repair of ruptured abdominal aortic aneurysms in the state of Maryland: factors influencing outcome among 527 recent cases. J Vasc Surg 1998; 28:413-420.

Darling RC, Messina CR, Brewster DC, Ottinger LW. Autopsy study of unoperated abdominal aortic aneurysms. The case for early resection. Circulation 1977; 56:II 161-164.

Davidovic L, Markovic M, Kostic D, Cinara I, Markovic D, Maksimovic Z, Cvetkovic S, Sindjelic R, Ille T. Ruptured abdominal aortic aneurysms: factors influencing early survival. Ann Vasc Surg 2005; 19:29-34.

de Vries M, Ouwendijk R, Kessels AG, de Haan MW, Flobbe K, Hunink MG, van Engelshoven JM, Nelemans PJ. Comparison of generic and disease-specific questionnaires for the assessment of quality of life in patients with peripheral arterial disease. J Vasc Surg 2005; 41:261-268.

Defraigne JO, Sakalihasan N, Lavigne JP, Van Damme H, Limet R. Chronic rupture of abdominal aortic aneurysm manifesting as crural neuropathy. Ann Vasc Surg 2001; 15:405-411.

Di Martino ES, Bohra A, Vande Geest JP, Gupta N, Makaroun MS, Vorp DA. Biomechanical properties of ruptured versus electively repaired abdominal aortic aneurysm wall tissue. J Vasc Surg 2006; 43:570-576.

Dillavou ED, Muluk SC, Makaroun MS. A decade of change in abdominal aortic aneurysm repair in the United States: Have we improved outcomes equally between men and women? J Vasc Surg 2006; 43:230-238.

Dimick JB, Stanley JC, Axelrod DA, Kazmers A, Henke PK, Jacobs LA, Wakefield TW, Greenfield LJ, Upchurch GR,Jr. Variation in death rate after abdominal aortic aneurysmectomy in the United States: impact of hospital volume, gender, and age. Ann Surg 2002; 235:579-585.

Dorrucci V, Dusi R, Rombola G, Cordiano C. Contained rupture of an abdominal aortic aneurysm presenting as obstructive jaundice: report of a case. Surg Today 2001; 31:331-332.

Drott C, Arfvidsson B, Ortenwall P, Lundholm K. Age-standardized incidence of ruptured aortic aneurysm in a defined Swedish population between 1952 and 1988: mortality rate and operative results. Br J Surg 1992; 79:175-179.

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 2004a; 39:784-787.

Dueck AD, Kucey DS, Johnston KW, Alter D, Laupacis A. Long-term survival and temporal trends in patient and surgeon factors after elective and ruptured abdominal aortic aneurysm surgery. J Vasc Surg 2004b; 39:1261-1267.

Dueck AD, Kucey DS, Johnston KW, Alter D, Laupacis A. Survival after ruptured abdominal aortic aneurysm: effect of patient, surgeon, and hospital factors. J Vasc Surg 2004c; 39:1253-1260.

Dunlop DD, Manheim LM, Sohn MW, Liu X, Chang RW. Incidence of functional limitation in older adults: the impact of gender, race, and chronic conditions. Arch Phys Med Rehabil 2002; 83:964-971.

Eagle KA, Berger PB, Calkins H, Chaitman BR, Ewy GA, Fleischmann KE, Fleisher LA, Froehlich JB, Gusberg RJ, Leppo JA, Ryan T, Schlant RC, Winters WL,Jr, Gibbons RJ, Antman EM, Alpert JS, Faxon DP, Fuster V, Gregoratos G, Jacobs AK, Hiratzka LF, Russell RO, Smith SC,Jr. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery - executive summary a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). Circulation 2002; 105:1257-1267.

Eefting F, Nathoe H, van Dijk D, Jansen E, Lahpor J, Stella P, Suyker W, Diephuis J, Suryapranata H, Ernst S, Borst C, Buskens E, Grobbee D, de Jaegere P. Randomized comparison between stenting and off-pump bypass surgery in patients referred for angioplasty. Circulation 2003; 108:2870-2876.

The EuroQol Group. EuroQol - a new facility for the measurement of health-related quality of life. The EuroQol Group. Health Policy 1990; 16:199-208.

Evans SM, Adam DJ, Brittenden J, Bain M, Bradbury AW. Vascular surgical society of Great Britain and Ireland: long-term survival following repair of ruptured abdominal aortic aneurysm in patients over 75 years of age. Br J Surg 1999; 86:696.

Evans SM, Adam DJ, Bradbury AW. The influence of gender on outcome after ruptured abdominal aortic aneurysm. J Vasc Surg 2000; 32:258-262.

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 2005b; 365:2187-2192.

EVAR trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet 2005a; 365:2179-2186.

Fillinger MF, Marra SP, Raghavan ML, Kennedy FE. Prediction of rupture risk in abdominal aortic aneurysm during observation: wall stress versus diameter. J Vasc Surg 2003; 37:724-732.

Fillinger MF, Racusin J, Baker RK, Cronenwett JL, Teutelink A, Schermerhorn ML, Zwolak RM, Powell RJ, Walsh DB, Rzucidlo EM. Anatomic characteristics of ruptured abdominal aortic aneurysm on conventional CT scans: Implications for rupture risk. J Vasc Surg 2004; 39:1243-1252.

Finlayson SR, Birkmeyer JD. Cost-effectiveness analysis in surgery. Surgery 1998; 123:151-156.

Garratt A, Schmidt L, Mackintosh A, Fitzpatrick R. Quality of life measurement: bibliographic study of patient assessed health outcome measures. BMJ 2002; 324:1417.

Gloviczki P, Pairolero PC, Mucha P,Jr, Farnell MB, Hallett JW,Jr, Ilstrup DM, Toomey BJ, Weaver AL, Bower TC, Bourchier RG. Ruptured abdominal aortic aneurysms: repair should not be denied. J Vasc Surg 1992; 15:851-857.

Golledge J, Abrokwah J, Shenoy KN, Armour RH. Morphology of ruptured abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 1999; 18:96-104.

Greco G, Egorova N, Anderson PL, Gelijns A, Moskowitz A, Nowygrod R, Arons R, McKinsey J, Morrissey NJ, Kent KC. Outcomes of endovascular treatment of ruptured abdominal aortic aneurysms. J Vasc Surg 2006; 43:453-459.

Hadjianastassiou VG, Tekkis PP, Goldhill DR, Hands LJ. Quantification of mortality risk after abdominal aortic aneurysm repair. Br J Surg 2005; 92:1092-1098.

Hadjianastassiou VG, Franco L, Jerez JM, Evangelou IE, Goldhill DR, Tekkis PP, Hands LJ. Optimal prediction of mortality after abdominal aortic aneurysm repair with statistical models. J Vasc Surg 2006; 43:467-473.

Hall AJ, Busse EF, McCarville DJ, Burgess JJ. Aortic wall tension as a predictive factor for abdominal aortic aneurysm rupture: improving the selection of patients for abdominal aortic aneurysm repair. Ann Vasc Surg 2000; 14:152-157.

Hallin A, Bergqvist D, Holmberg L. Literature review of surgical management of abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2001; 22:197-204.

Halpern VJ, Kline RG, D'Angelo AJ, Cohen JR. Factors that affect the survival rate of patients with ruptured abdominal aortic aneurysms. J Vasc Surg 1997; 26:939-945.

Hamel MB, Phillips RS, Davis RB, Desbiens N, Connors AF,Jr, Teno JM, Wenger N, Lynn J, Wu AW, Fulkerson W, Tsevat J. Outcomes and cost-effectiveness of initiating dialysis and continuing aggressive care in seriously ill hospitalized adults. SUPPORT Investigators. Study to Understand Prognoses and Preferences for Outcomes and Risks of Treatments. Ann Intern Med 1997; 127:195-202.

Hardman DT, Fisher CM, Patel MI, Neale M, Chambers J, Lane R, Appleberg M. Ruptured abdominal aortic aneurysms: who should be offered surgery? J Vasc Surg 1996; 23:123-129.

Harkin DW, O'Donnell M, Butler J, Blair PH, Hood JM, Barros D'Sa AA. Periods of low atmospheric pressure are associated with high abdominal aortic aneurysm rupture rates in Northern Ireland. Ulster Med J 2005; 74:113-121.

Harrell FE,Jr, Lee KL, Mark DB. Multivariable prognostic models: issues in developing models, evaluating assumptions and adequacy, and measuring and reducing errors. Stat Med 1996; 15:361-387.

Harris JR, Forbes TL, Steiner SH, Lawlor DK, Derose G, Harris KA. Risk-adjusted analysis of early mortality after ruptured abdominal aortic aneurysm repair. J Vasc Surg 2005; 42:387-391.

Harris LM, Faggioli GL, Fiedler R, Curl GR, Ricotta JJ. Ruptured abdominal aortic aneurysms: factors affecting mortality rates. J Vasc Surg 1991; 14:812-818.

Hays RD, Sherbourne CD, Mazel RM. The RAND 36-Item Health Survey 1.0. Health Econ 1993; 2:217-227.

Heikkinen M, Salenius JP, Auvinen O. Ruptured abdominal aortic aneurysm in a well-defined geographic area. J Vasc Surg 2002; 36:291-296.

Heller JA, Weinberg A, Arons R, Krishnasastry KV, Lyon RT, Deitch JS, Schulick AH, Bush HL,Jr, Kent KC. Two decades of abdominal aortic aneurysm repair: have we made any progress? J Vasc Surg 2000; 32:1091-1100.

Hennessy A, Barry MC, McGee H, O'Boyle C, Hayes DB, Grace PA. Quality of life following repair of ruptured and elective abdominal aortic aneurysms. Eur J Surg 1998; 164:673-677.

Herlitz J, Bahr J, Fischer M, Kuisma M, Lexow K, Thorgeirsson G. Resuscitation in Europe: a tale of five European regions. Resuscitation 1999; 41:121-131.

Hewin DF, Campbell WB. Ruptured aortic aneurysm: the decision not to operate. Ann R Coll Surg Engl 1998; 80:221-225.

Hinterseher I, Saeger HD, Koch R, Bloomenthal A, Ockert D, Bergert H. Quality of life and long-term results after ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2004; 28:262-269.

Ho K, Burgess KR, Braude S. Ruptured abdominal aortic aneurysm - outcome in a community teaching hospital intensive care unit. Anaesth Intensive Care 1999; 27:497-502.

Hsiang YN, Turnbull RG, Nicholls SC, McCullough K, Chen JC, Lokanathan R, Taylor DC. Predicting death from ruptured abdominal aortic aneurysms. Am J Surg 2001; 181:30-35.

Janczyk RJ, Howells GA, Bair HA, Huang R, Bendick PJ, Zelenock GB. Hypothermia is an independent predictor of mortality in ruptured abdominal aortic aneurysms. Vasc Endovascular Surg 2004; 38:37-42.

Johansson G, Swedenborg J. Ruptured abdominal aortic aneurysms: a study of incidence and mortality. Br J Surg 1986; 73:101-103.

Johnston KW, Rutherford RB, Tilson MD, Shah DM, Hollier L, Stanley JC. Suggested standards for reporting on arterial aneurysms. Subcommittee on Reporting Standards for Arterial Aneurysms, Ad Hoc Committee on Reporting Standards, Society for Vascular Surgery and North American Chapter, International Society for Cardiovascular Surgery. J Vasc Surg 1991; 13:452-458.

Johnston KW. Influence of sex on the results of abdominal aortic aneurysm repair. Canadian Society for Vascular Surgery Aneurysm Study Group. J Vasc Surg 1994a; 20:914-923.

Johnston KW. Ruptured abdominal aortic aneurysm: six-year follow-up results of a multicenter prospective study. Canadian Society for Vascular Surgery Aneurysm Study Group. J Vasc Surg 1994b; 19:888-900.

Joseph AY, Fisher JB, Toedter LJ, Balshi JD, Granson MA, Meir-Levi D. Ruptured abdominal aortic aneurysm and quality of life. Vasc Endovascular Surg 2002; 36:65-70.

Junger A, Engel J, Benson M, Bottger S, Grabow C, Hartmann B, Michel A, Rohrig R, Marquardt K, Hempelmann G. Discriminative power on mortality of a modified Sequential Organ Failure Assessment score for complete automatic computation in an operative intensive care unit. Crit Care Med 2002; 30:338-342.

Kaarlola A, Pettilä V, Kekki P. Performance of two measures of general health-related quality of life, the EQ-5D and the RAND-36 among critically ill patients. Intensive Care Med 2004; 30:2245-2252.

Kaarlola A, Tallgren M, Pettilä V. Long-term survival, quality of life, and quality-adjusted life-years among critically ill elderly patients. Crit Care Med 2006; 34:2120-2126.

Kantonen I, Lepäntalo M, Salenius JP, Forsström E, Hakkarainen T, Huusari H, Jaakkola A, Kaarne M, Kaartinen P, Kivivuori R, Kostiainen S, Lehtonen J, Loponen P, Luther M, Mäenpää I, Nikula P, Riekkinen H, Rissanen K, Vilkko P, Ylönen K. Auditing a nationwide vascular registry - the 4-year Finnvasc experience. Finnvasc Study Group. Eur J Vasc Endovasc Surg 1997; 14:468-474.

Kantonen I, Luther M, Salenius J, Huusari H, Jaakkola A, Lehtonen J, Nikula P, Rissanen K, Savolainen H, Ylönen K, Lepäntalo M. Influence of transportation distance on mortality in ruptured abdominal aortic aneurysms. Int J Ang 1999a; 8:134-137.

Kantonen I, Lepäntalo M, Brommels M, Luther M, Salenius JP, Ylönen K. Mortality in ruptured abdominal aortic aneurysms. The Finnvasc Study Group. Eur J Vasc Endovasc Surg 1999b; 17:208-212.

Katz DJ, Stanley JC, Zelenock GB. Operative mortality rates for intact and ruptured abdominal aortic aneurysms in Michigan: an eleven-year statewide experience. J Vasc Surg 1994; 19:804-815.

Katz DJ, Stanley JC, Zelenock GB. Gender differences in abdominal aortic aneurysm prevalence, treatment, and outcome. J Vasc Surg 1997; 25:561-568.

Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med 1985; 13:818-829.

Kniemeyer HW, Kessler T, Reber PU, Ris HB, Hakki H, Widmer MK. Treatment of ruptured abdominal aortic aneurysm, a permanent challenge or a waste of resources? Prediction of outcome using a multi-organ-dysfunction score. Eur J Vasc Endovasc Surg 2000; 19:190-196.

Korhonen SJ, Kantonen I, Pettilä V, Keränen J, Salo JA, Lepäntalo M. Long-term survival and health-related quality of life of patients with ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2003; 25:350-353.

Korhonen SJ, Ylönen K, Biancari F, Heikkinen M, Salenius J.-P., Lepäntalo M for the Finnvasc Study Group. Glasgow Aneurysm Score as a predictor of the immediate outcome after surgery for ruptured abdominal aortic aneurysm: a Finnvasc study. Br J Surg 2004; 91:1449-1452.

Koskas F, Kieffer E. Surgery for ruptured abdominal aortic aneurysm: early and late results of a prospective study by the AURC in 1989. Ann Vasc Surg 1997; 11:90-99.

Kuisma M, Martikainen M. 112 is functioning. Duodecim 1998; 114:1631-1638.

Kuntz KM, Kent KC. Is carotid endarterectomy cost-effective? An analysis of symptomatic and asymptomatic patients. Circulation 1996; 94:II 194-198.

Laukontaus SJ, Lepäntalo M, Hynninen M, Kantonen I, Pettilä V. Prediction of survival after 48-h maximal intensive care after repair for ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2005; 30:509-519.

Law MR, Morris J, Wald NJ. Screening for abdominal aortic aneurysms. J Med Screen 1994; 1:110-115.

Lazarides MK, Arvanitis DP, Drista H, Staramos DN, Dayantas JN. POSSUM and APACHE II scores do not predict the outcome of ruptured infrarenal aortic aneurysms. Ann Vasc Surg 1997; 11:155-158.

Lederle FA, Johnson GR, Wilson SE, Ballard DJ, Jordan WD,Jr, Blebea J, Littooy FN, Freischlag JA, Bandyk D, Rapp JH, Salam AA, Veterans Affairs Cooperative Study Investigators. Rupture rate of large abdominal aortic aneurysms in patients refusing or unfit for elective repair. JAMA 2002; 287:2968-2972.

Lee KH, Angus DC, Abramson NS. Cardiopulmonary resuscitation: what cost to cheat death? Crit Care Med 1996; 24:2046-2052.

Leo E, Biancari F, Hanhela R, Karlqvist K, Romsi P, Ylönen K, Rainio P, Satta J, Juvonen T. Baseline oxygen delivery is associated with an increased risk of severe postoperative complications after elective open repair of abdominal aortic aneurysm. J Cardiovasc Surg 2005; 46:279-284.

Lepäntalo M, Salenius JP, Luther M, Ylönen K. Introduction of a population-based vascular registry: validity of data and limitations of registration. The Finnvasc Study Group. Br J Surg 1994; 81:979-981.

Lindblad B, Borner G, Gottsater A. Factors associated with development of large abdominal aortic aneurysm in middle-aged men. Eur J Vasc Endovasc Surg 2005; 30:346-352.

Lloyd GM, Bown MJ, Norwood MG, Deb R, Fishwick G, Bell PR, Sayers RD. Feasibility of preoperative computer tomography in patients with ruptured abdominal aortic aneurysm: a time-to-death study in patients without operation. J Vasc Surg 2004; 39:788-791.

Magee TR, Scott DJ, Dunkley A, St Johnston J, Campbell WB, Baird RN, Horrocks M. Quality of life following surgery for abdominal aortic aneurysm. Br J Surg 1992; 79:1014-1016.

Maziak DE, Lindsay TF, Marshall JC, Walker PM. The impact of multiple organ dysfunction on mortality following ruptured abdominal aortic aneurysm repair. Ann Vasc Surg 1998; 12:93-100.

McGregor JC, Pollock JG, Anton HC. The value of ultrasonography in the diagnosis of abdominal aortic aneurysm. Scott Med J 1975; 20:133-137.

McKinsey JF, Rosinberg A, Egorova NN, Chanter-Ouw K, Gelijns A, Mskowitz A, Nowygrod R, Morrissey N, Kent KC. National outcomes for the treatment of ruptured abdominal aortic aneurysm: comparison of open vs endovascular repairs. Abstract. Society for Vascular Surgery, Philadelphia, Annual meeting, June 1-4, 2006:76.

Mealy K, Salman A. The true incidence of ruptured abdominal aortic aneurysms. Eur J Vasc Surg 1988; 2:405-408.

Meesters RC, van der Graaf Y, Vos A, Eikelboom BC. Ruptured aortic aneurysm: early postoperative prediction of mortality using an organ system failure score. Br J Surg 1994; 81:512-516.

Miani S, Mingazzini P, Piglionica R, Biasi GM, Ruberti U. Influence of the rupture site of abdominal aortic aneurysms with regard to postoperative survival rate. J Cardiovasc Surg 1984; 25:414-419.

Mikkola T, Bergström M, toim. Terveydenhuollon valinnat–lääketieteelliset perusteet. Jyväskylä: Stakes, 1994 Raportteja 133.

Moreno R, Vincent JL, Matos R, Mendonca A, Cantraine F, Thijs L, Takala J, Sprung C, Antonelli M, Bruining H, Willatts S. The use of maximum SOFA score to quantify organ dysfunction/failure in intensive care. Results of a prospective, multicentre study. Working Group on Sepsis related Problems of the ESICM. Intensive Care Med 1999; 25:686-696.

Neary WD, Crow P, Foy C, Prytherch D, Heather BP, Earnshaw JJ. Comparison of POSSUM scoring and the Hardman Index in selection of patients for repair of ruptured abdominal aortic aneurysm. Br J Surg 2003; 90:421-425.

Nesi F, Leo E, Biancari F, Bartolucci R, Rainio P, Satta J, Rabitti G, Juvonen T. Preoperative risk stratification in patients undergoing elective infrarenal aortic aneurysm surgery: evaluation of five risk scoring methods. Eur J Vasc Endovasc Surg 2004; 28:52-58.

Nevitt MP, Ballard DJ, Hallett JW,Jr. Prognosis of abdominal aortic aneurysms. A population-based study. N Engl J Med 1989; 321:1009-1014.

Noel AA, Gloviczki P, Cherry KJ,Jr, Bower TC, Panneton JM, Mozes GI, Harmsen WS, Jenkins GD, Hallett JW,Jr. Ruptured abdominal aortic aneurysms: the excessive mortality rate of conventional repair. J Vasc Surg 2001; 34:41-46.

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; 317:852-856.

Ohinmaa A, Sintonen H. Inconsistencies and modelling of the Finnish EuroQol (EQ-D) preference values. In: Gainer W, von dem Schulenberg G, Piercy J (eds), EuroQol, Plenary Meeting, Hannover 1998, 1-2 October; Discussion Papers. University of Hannover Centre of Health Economics and Health System Research 1999; 57-74.

Ouriel K, Geary K, Green RM, Fiore W, Geary JE, DeWeese JA. Factors determining survival after ruptured aortic aneurysm: the hospital, the surgeon, and the patient. J Vasc Surg 1990; 11:493-496.

Patel ST, Haser PB, Bush HL,Jr, Kent KC. The cost-effectiveness of endovascular repair versus open surgical repair of abdominal aortic aneurysms: A decision analysis model. J Vasc Surg 1999; 29:958-972.

Patel ST, Korn P, Haser PB, Bush HL,Jr, Kent KC. The cost-effectiveness of repairing ruptured abdominal aortic aneurysms. J Vasc Surg 2000; 32:247-257.

Patrick DL, Deyo RA. Generic and disease-specific measures in assessing health status and quality of life. Med Care 1989; 27:S217-232.

Peppelenbosch N, Zannetti S, Barbieri B, Buth J, ERA study collaborators. Endograft treatment in ruptured abdominal aortic aneurysms using the Talent AUI stentgraft system. Design of a feasibility study. Eur J Vasc Endovasc Surg 2004; 27:366-371.

Peppelenbosch N, Geelkerken RH, Soong C, Cao P, Steinmetz OK, Teijink JA, Lepantalo M, De Letter J, Vermassen FE, Derose G, Buskens E, Buth J. Endograft treatment of ruptured abdominal aortic aneurysms using the Talent aortouniiliac system: An international multicenter study. J Vasc Surg 2006; 43:1111-1123.

Perkins JM, Magee TR, Hands LJ, Collin J, Galland RB, Morris PJ. Prospective evaluation of quality of life after conventional abdominal aortic aneurysm surgery. Eur J Vasc Endovasc Surg 1998; 16:203-207.

Petersen E, Gineitis A, Wagberg F, Angquist KA. Serum levels of elastin-derived peptides in patients with ruptured and asymptomatic abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2001; 22:48-52.

Petersen E, Wagberg F, Angquist KA. Proteolysis of the abdominal aortic aneurysm wall and the association with rupture. Eur J Vasc Endovasc Surg 2002; 23:153-157.

Petersen E. Biochemical markers for increased risk of rupture. Abstarct. Abdominal Aortic Aneurysms Bringing Basic Science into Clinical Practice. Stockholm, Sweden, January 30- February 1, 2003:15-16.

Pettilä V, Kaarlola A, Mäkeläinen A. Health-related quality of life of multiple organ dysfunction patients one year after intensive care. Intensive Care Med 2000; 26:1473-1479.

Piper G, Patel NA, Chandela S, Benckart DH, Young JC, Collela JJ, Healy DA. Short-term predictors and long-term outcome after ruptured abdominal aortic aneurysm repair. Am Surg 2003; 69:703-709.

Prance SE, Wilson YG, Cosgrove CM, Walker AJ, Wilkins DC, Ashley S. Ruptured abdominal aortic aneurysms: selecting patients for surgery. Eur J Vasc Endovasc Surg 1999; 17:129-132.

Prytherch DR, Whiteley MS, Higgins B, Weaver PC, Prout WG, Powell SJ. POSSUM and Portsmouth POSSUM for predicting mortality. Physiological and Operative Severity Score for the enUmeration of Mortality and morbidity. Br J Surg 1998; 85:1217-1220.

Prytherch DR, Sutton GL, Boyle JR. Portsmouth POSSUM models for abdominal aortic aneurysm surgery. Br J Surg 2001; 88:958-963.

Prytherch DR, Sirl JS, Weaver PC, Schmidt P, Higgins B, Sutton GL. Towards a national clinical minimum data set for general surgery. Br J Surg 2003; 90:1300-1305.

Prytherch DR, Ridler BM, Ashley S, Audit Research Committee of the Vascular Society of Great Britain and Ireland. Risk-adjusted predictive models of mortality after index arterial operations using a minimal data set. Br J Surg 2005; 92:714-718.

Rantakokko V, Havia T, Inberg MV, Vanttinen E. Abdominal aortic aneurysms: a clinical and autopsy study of 408 patients. Acta Chir Scand 1983; 149:151-155.

Reitsma JB, Pleumeekers HJ, Hoes AW, Kleijnen J, de Groot RM, Jacobs MJ, Grobbee DE, Tijssen JG. Increasing incidence of aneurysms of the abdominal aorta in The Netherlands. Eur J Vasc Endovasc Surg 1996; 12:446-451.

Rigberg DA, Zingmond DS, McGory ML, Maggard MA, Agustin M, Lawrence PF, Ko CY. Age stratified, perioperative, and one-year mortality after abdominal aortic aneurysm repair: a statewide experience. J Vasc Surg 2006; 43:224-229.

Roberts K, Revell M, Youssef H, Bradbury AW, Adam DJ. Hypotensive resuscitation in patients with ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2006; 31:339-344.

Roddy SP, Darling RC,3rd, Maharaj D, Ozsvath KJ, Mehta M, Paty PS, Kreienberg PB, Choi D, Chang BB, Shah DM. Should ruptured abdominal aortic aneurysms be repaired in the octogenarian? Cardiovasc Surg 2003; 11:337-340.

Rohrer MJ, Cutler BS, Wheeler HB. Long-term survival and quality of life following ruptured abdominal aortic aneurysm. Arch Surg 1988; 123:1213-1217.

Rosset E, Rovani G, Aublet-Cuvelier B, Lietin B, Tran X, Halden MA, Hermier M, Poirier M, Ribal J. Surgical treatment of infrarenal aortic aneurysms in octogenarians. In Branchereau A, Jacobs M (eds). Vascular procedured in the elderly patient. Paris Consultans Ltd, Oxford, 2006, 85-88.

Russell EM, Bruce J, Krukowski ZH. Systematic review of the quality of surgical mortality monitoring. Br J Surg 2003; 90:527-532.

Rutledge R, Oller DW, Meyer AA, Johnson GJ,Jr. A statewide, population-based time-series analysis of the outcome of ruptured abdominal aortic aneurysm. Ann Surg 1996; 223:492-502.

Salenius JP. National vascular registry in Finland. FINNVASC Study Group. Ann Chir Gynaecol 1992; 81:257-260.

Samy AK, Murray G, MacBain G. Glasgow aneurysm score. Cardiovasc Surg 1994; 2:41-44.

Samy AK, Murray G, MacBain G. Prospective evaluation of the Glasgow Aneurysm Score. J R Coll Surg Edinb 1996; 41:105-107.

Sasaki S, Sakuma M, Samejima M, Kunihara T, Shiiya N, Murashita T, Matsui Y, Yasuda K. Ruptured abdominal aortic aneurysms: analysis of factors influencing surgical results in 184 patients. J Cardiovasc Surg 1999; 40:401-405.

Saukko P. Medicolegal investigative system and sudden death in Scandinavia. Nippon Hoigaku Zasshi 1995; 49:458-465.

Schermerhorn ML, Birkmeyer JD, Gould DA, Cronenwett JL. Cost-effectiveness of surgery for small abdominal aortic aneurysms on the basis of data from the United Kingdom small aneurysm trial. J Vasc Surg 2000; 31:217-226.

Seiwert AJ, Elmore JR, Youkey JR, Franklin DP. Peter B. Samuels Award. Ruptured abdominal aortic aneurysm repair: the financial analysis. Am J Surg 1995; 170:91-96.

Semmens JB, Norman PE, Lawrence-Brown MM, Bass AJ, Holman CD. Population-based record linkage study of the incidence of abdominal aortic aneurysm in Western Australia in 1985-1994. Br J Surg 1998; 85:648-652.

Semmens JB, Norman PE, Lawrence-Brown MM, Holman CD. Influence of gender on outcome from ruptured abdominal aortic aneurysm. Br J Surg 2000; 87:191-194.

Shepard DS. Cost-effectiveness in Health and Medicine. In Gold MR, Siegel JE, Russell LB, and Weinstein MC (eds). New York: Oxford University Press, 1996. J Ment Health Policy Econ 1999; 2:91-92.

Singh K, Bonaa KH, Jacobsen BK, Bjork L, Solberg S. Prevalence of and risk factors for abdominal aortic aneurysms in a population-based study : The Tromso Study. Am J Epidemiol 2001; 154:236-244.

Soisalon-Soininen S, Salo JA, Takkunen O, Mattila S. Comparison of long-term survival after repair of ruptured and non-ruptured abdominal aortic aneurysm. Vasa 1995; 24:42-48.

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.

Sonesson B, Hansen F, Stale H, Lanne T. Compliance and diameter in the human abdominal aorta - the influence of age and sex. Eur J Vasc Surg 1993; 7:690-697.

St Leger AS, Spencely M, McCollum CN, Mossa M. Screening for abdominal aortic aneurysm: a computer assisted cost-utility analysis. Eur J Vasc Endovasc Surg 1996; 11:183-190.

Stenbaek J, Granath F, Swedenborg J. Outcome after abdominal aortic aneurysm repair. Difference between men and women. Eur J Vasc Endovasc Surg 2004; 28:47-51.

Sterpetti AV, Blair EA, Schultz RD, Feldhaus RJ, Cisternino S, Chasan P. Sealed rupture of abdominal aortic aneurysms. J Vasc Surg 1990; 11:430-435.

Stonebridge PA, Callam MJ, Bradbury AW, Murie JA, Jenkins AM, Ruckley CV. Comparison of long-term survival after successful repair of ruptured and non-ruptured abdominal aortic aneurysm. Br J Surg 1993; 80:585-586.

Sundhagen JO, Florenes T, Staxrud LE. Patienten som ikke vill dö. Abstract. Norwegian Society for Vascular Surgery 2006; 16:20.

Szilagyi DE. Clinical diagnosis of intact and ruptured abdominal aortic aneurysms. In Bergan JJ Yao JST (eds). Aneurysms: diagnosis and treatment. New York: Grune & Stratton, Inc, 1982; 205-216.

Tambyraja AL, Dawson AR, Murie JA, Chalmers RT. Cardiac troponin I predicts outcome after ruptured abdominal aortic aneurysm repair. Br J Surg 2005a; 92:824-827.

Tambyraja AL, Fraser SC, Murie JA, Chalmers RT. Functional outcome after open repair of ruptured abdominal aortic aneurysm. J Vasc Surg 2005b; 41:758-761.

Tambyraja AL, Fraser SC, Murie JA, Chalmers RT. Validity of the Glasgow Aneurysm Score and the Hardman Index in predicting outcome after ruptured abdominal aortic aneurysm repair. Br J Surg 2005c; 92:570-573.

Tambyraja AL, Murie JA, Chalmers RT. Outcome and survival of patients aged 65 years and younger after abdominal aortic aneurysm rupture. World J Surg 2005d; 29:1245-1247.

Tang T, Lindop M, Munday I, Quick CR, Gaunt ME, Varty K. A cost analysis of surgery for ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2003; 26:299-302.

Taylor PR, Gerrard DJ. The economic evaluation of arterial intervention. Cardiovasc Surg 2002; 10:98-101.

Thomas PR, Stewart RD. Abdominal aortic aneurysm. Br J Surg 1988; 75:733-736.

Tornwall ME, Virtamo J, Haukka JK, Albanes D, Huttunen JK. Life-style factors and risk for abdominal aortic aneurysm in a cohort of Finnish male smokers. Epidemiology 2001; 12:94-100.

Turton EP, Scott DJ, Delbridge M, Snowden S, Kester RC. Ruptured abdominal aortic aneurysm: a novel method of outcome prediction using neural network technology. Eur J Vasc Endovasc Surg 2000; 19:184-189.

The UK small Aneurysm Trial Participants. Mortality results for randomised controlled trial of early elective surgery or ultrasonographic surveillance for small abdominal aortic aneurysms. Lancet 1998; 352:1649-1655.

van Dongen HP, Leusink JA, Moll FL, Brons FM, de Boer A. Ruptured abdominal aortic aneurysms: factors influencing postoperative mortality and long-term survival. Eur J Vasc Endovasc Surg 1998; 15:62-66.

Vardulaki KA, Prevost TC, Walker NM, Day NE, Wilmink AB, Quick CR, Ashton HA, Scott RA. Growth rates and risk of rupture of abdominal aortic aneurysms. Br J Surg 1998; 85:1674-1680.

Vincent JL, de Mendonca A, Cantraine F, Moreno R, Takala J, Suter PM, Sprung CL, Colardyn F, Blecher S. Use of the SOFA score to assess the incidence of organ dysfunction/failure in intensive care units: results of a multicenter, prospective study. Working group on "sepsis-related problems" of the European Society of Intensive Care Medicine. Crit Care Med 1998; 26:1793-1800.

Visser P, Akkersdijk GJ, Blankensteijn JD. In-hospital operative mortality of ruptured abdominal aortic aneurysm: a population-based analysis of 5593 patients in The Netherlands over a 10-year period. Eur J Vasc Endovasc Surg 2005; 30:359-364.

Vogel TR, Nackman GB, Brevetti LS, Crowley JG, Bueno MM, Banavage A, Odroniec K, Ciocca RG, Graham AM. Resource utilization and outcomes: effect of transfer on patients with ruptured abdominal aortic aneurysms. Ann Vasc Surg 2005; 19:149-153.

Vorp DA, Lee PC, Wang DH, Makaroun MS, Nemoto EM, Ogawa S, Webster MW. Association of intraluminal thrombus in abdominal aortic aneurysm with local hypoxia and wall weakening. J Vasc Surg 2001; 34:291-299.

Vorp DA, Vande Geest JP. Biomechanical determinants of abdominal aortic aneurysm rupture. Arterioscler Thromb Vasc Biol 2005; 25:1558-1566.

Wanhainen A, Bergqvist D, Boman K, Nilsson TK, Rutegard J, Bjorck M. Risk factors associated with abdominal aortic aneurysm: a population-based study with historical and current data. J Vasc Surg 2005; 41:390-396.

Ware JE,Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care 1992; 30:473-483.

Wilmink AB, Pleumeekers HJ, Hoes AW, Hubbard CS, Grobbee DE, Quick CR. The infrarenal aortic diameter in relation to age: only part of the population in older age groups shows an increase. Eur J Vasc Endovasc Surg 1998; 16:431-437.

Wilson WR, Fishwick G, Sir Peter RFB, Thompson MM. Suitability of ruptured AAA for endovascular repair. J Endovasc Ther 2004; 11:635-640.

Åhlstrom A, Tallgren M, Peltonen S, Räsänen P, Pettilä V. Survival and quality of life of patients requiring acute renal replacement therapy. Intensive Care Med 2005; 31:1222-1228.