

Terhi Nevala

ENDOVASCULAR
TREATMENT OF
AN ABDOMINAL
AORTIC ANEURYSM

*MID-TERM RESULTS AND MANAGEMENT OF
A TYPE II ENDOLEAK*

FACULTY OF MEDICINE,
INSTITUTE OF DIAGNOSTICS, DEPARTMENT OF DIAGNOSTIC RADIOLOGY,
INSTITUTE OF CLINICAL MEDICINE, DEPARTMENT OF SURGERY,
UNIVERSITY OF OULU

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MEDICA



ACTA UNIVERSITATIS OULUENSIS
D Medica 1046

TERHI NEVALA

**ENDOVASCULAR TREATMENT OF
AN ABDOMINAL AORTIC
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Mid-term results and management of a type II endoleak

Academic dissertation to be presented with the assent of
the Faculty of Medicine of the University of Oulu for
public defence in Auditorium 7 of Oulu University
Hospital, on 19 March 2010, at 12 noon

UNIVERSITY OF OULU, OULU 2010

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Acta Univ. Oul. D 1046, 2010

Supervised by
Docent Jukka Perälä
Docent Fausto Biancari

Reviewed by
Docent Leo Keski-Nisula
Docent Jukka Saarinen

ISBN 978-951-42-6133-6 (Paperback)
ISBN 978-951-42-6134-3 (PDF)
<http://herkules.oulu.fi/isbn9789514261343/>
ISSN 0355-3221 (Printed)
ISSN 1796-2234 (Online)
<http://herkules.oulu.fi/issn03553221/>

Cover design
Raimo Ahonen

JUVENES PRINT
TAMPERE 2010

Nevala, Terhi, Endovascular treatment of an abdominal aortic aneurysm. Mid-term results and management of a type II endoleak

Faculty of Medicine, Institute of Diagnostics, Department of Diagnostic Radiology, Institute of Clinical Medicine, Department of Surgery, University of Oulu, P.O.Box 5000, FI-90014 University of Oulu, Finland

Acta Univ. Oul. D 1046, 2010

Abstract

Endovascular aneurysm repair (EVAR) is a minimally invasive alternative to open surgery to exclude an abdominal aortic aneurysm from the circulation to avert a rupture. The aim of this thesis was to evaluate the early and mid-term results of EVAR using the Zenith[®] stent-graft (Cook Inc, Bloomington, IN, USA) in asymptomatic and symptomatic abdominal aortic aneurysm (AAA) patients in three Finnish university hospitals. Furthermore, the aim was to study whether preoperative embolization of the inferior mesenteric artery (IMA) before EVAR decreases the incidence of a type II endoleak or has an effect on the aneurysm sac shrinkage. Finally, the results after secondary interventions for a type II endoleak were evaluated.

Two hundred six patients underwent elective endovascular repair of an intact AAA. The use of the Zenith[®] stent-graft was associated with good early and mid-term results. The thirty-day mortality rate (2.9%) was in accordance with other EVAR studies. Only one late aneurysm-related death occurred in this series, whilst no patients died of a late aneurysm rupture. No stent-graft migrations or fractures were observed. Endoleak, defined as persistent blood flow outside the graft and within the aneurysm sac, remains a long-term problem with EVAR. The overall endoleak incidence was 34.6%. A type II endoleak (retrograde perfusion via aortic side branches) occurred in 52 patients (25.4%).

EVAR was performed for 14 patients with a symptomatic, unruptured AAA. The median delay from admission to intervention was 4 days. EVAR of a symptomatic, unruptured AAA was associated with a favourable outcome even in patients with a very high operative risk. There were no perioperative deaths.

Altogether forty patients treated at Kuopio University Hospital had a patent IMA on preoperative computed tomography (CT) and were treated successfully with coil embolization before EVAR. Thirty-nine patients who underwent EVAR at Oulu University Hospital without preoperative embolization of a patent IMA served as a control group. Preoperative coil embolization of the IMA significantly reduced the incidence of type II endoleaks after EVAR, but the present study failed to show any influence on late postoperative aneurysm sac shrinkage.

Overall, 14 patients underwent a secondary intervention to repair the type II endoleak. Ten patients had transarterial embolization and four patients had translumbar embolization. The results were unsatisfactory; clinical success after the first secondary intervention was achieved in only two patients in the transarterial embolization group and three patients in the translumbar embolization group. These results seem to favour direct translumbar embolization rather than transarterial embolization.

In conclusion, EVAR with the Zenith[®] stent-graft is effective in excluding AAAs from the circulation and is associated with good mid-term results.

Keywords: abdominal aortic aneurysm, blood vessel prosthesis, inferior mesenteric artery, interventional radiology therapeutic embolization, stents, symptomatic abdominal aortic aneurysm, type II endoleak, Zenith stent-graft

Acknowledgements

This study was carried out during the years 2005–2010 at the Department of Diagnostic Radiology, Oulu University Hospital, in collaboration with the Department of Surgery, Oulu University Hospital.

I wish to express my deepest gratitude to Professor Osmo Tervonen, M.D., Head of the Department of Diagnostic Radiology, for creating an encouraging and inspiring atmosphere in which to carry out this research. He provided the opportunity and facilities for my research.

I owe my greatest gratitude to my supervisors, Docent Jukka Perälä, M.D., Head of the Interventional Radiology Unit, and Docent Fausto Biancari, M.D., Department of Surgery, for their ideas, guidance and support. My deepest and warmest thanks are due to Docent Jukka Perälä, who introduced me to the field of interventional radiology. It has been a privilege to work under his surveillance. His patience with my never-ending and meticulous questions during all phases of this study has been indispensable. I am especially grateful to Docent Fausto Biancari, M.D., for his optimistic attitude towards my work and his expertise in academic writing. This thesis would not have been accomplished without him.

I am sincerely grateful to my closest co-workers, Professor Hannu Manninen, M.D., Department of Radiology, Kuopio University Hospital, Professor Mauri Lepäntalo, M.D., Department of Vascular Surgery, Helsinki University Hospital and Pekka Aho, M.D., Ph.D., Department of Vascular Surgery, Helsinki University Hospital. Their ideas, constructive criticism and encouraging comments were essential to this work. I wish to express my gratitude to my co-authors, Docent Pekka Matsi, M.D., Department of Radiology, Kuopio University Hospital, Docent Kimmo Mäkinen M.D., Department of Surgery, Kuopio University Hospital, Wolf-Dieter Roth M.D. Ph.D., Department of Radiology, Helsinki University Hospital and Kari Ylönen, M.D., Department of Surgery, Oulu University Hospital. They gave numerous hours of their time for this work.

I owe my thanks to Ilpo Hovi, M.D., Ph.D., Pekka Kerimaa, M.D., Anita Mäkelä, Ville Mäki, M.D., Katri Rajala, M.D., and Matti Turtiainen, M.D., for helping me collect the data. I am also grateful to Docent Juha Salenius, M.D., Ilkka Manner M.D., and Jaakko Viljamaa, M.D., for helping me collect additional data during the final stage of writing this thesis.

I am grateful to Docent Leo Keski-Nisula, M.D., and Docent Jukka Saarinen, M.D., for their thorough review of the manuscript, constructive criticism and

advice. I wish to warmly thank authorized translator Keith Kosola for the careful revision of the language of this thesis.

I want to express my warmest thanks to my colleagues and friends in the Interventional Radiology Unit; especially Juho Kariniemi, M.D., for his excellent teaching and guidance in interventional radiology and Kari Palosaari, M.D., Ph.D., for his friendly collaboration. Salla Kokkonen, M.D., Ph.D., Mari Kuisma, M.D., Ph.D., Jaakko Niinimäki, M.D., Ph.D., and Johanna Ronkainen M.D., Ph.D., deserve my warmest thanks for their friendly and essential advice in the final stage of preparing this thesis. I wish to thank Marianne Haapea, M.Sc, for explaining statistical analysis to me in black and white.

I want to thank the staff of the Department of Diagnostic Radiology, especially in the Interventional Radiology unit. It has been a pleasure to work with you. I want to express my thanks to all of my colleagues and friends at the Radiology Departments of Oulu University Hospital. The collaboration and positive attitude towards work and the moments after work have been special and precious. Finally, I want to warmly thank the most treasured people in my life: my family and dear friends.

This study was financially supported by grants from the Radiological Society of Finland, the Finnish Society of Interventional Radiology, the Paavo Ilmari Ahvenainen Foundation and the Finnish Medical Foundation, all of which are gratefully acknowledged.

Oulu, January 2010

Terhi Nevala

Abbreviations

AAA	abdominal aortic aneurysm
CI	confidence interval
CT	computed tomography
EUROSTAR	European collaborators registry on stent-graft techniques for AAA repair
EVAR	endovascular aneurysm repair
EVAR trial 1	Endovascular Aneurysm Repair trial 1
EVAR trial 2	Endovascular Aneurysm Repair trial 2
F	French catheter scale
GAS	Glasgow aneurysm score
HUH	Helsinki University Hospital
IIA	internal iliac artery
IMA	inferior mesenteric artery
KUH	Kuopio University Hospital
MRI	magnetic resonance imaging
OR	odds ratio
OUH	Oulu University Hospital
RAAA	ruptured abdominal aortic aneurysm
SE	standard error
SMA	superior mesenteric artery
US	ultrasonography

List of original publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals:

- I Nevala T, Biancari F, Manninen H, Aho PS, Matsi P, Mäkinen K, Roth WD, Ylönen K, Lepäntalo M & Perälä J (2009) Finnish multicenter study on the midterm results of use of the Zenith stent-graft in the treatment of an abdominal aortic aneurysm. *J Vasc Interv Radiol* 20: 448–454.
- II Nevala T, Perälä J, Aho P, Matsi P, Ylönen K, Roth WD, Manninen H, Mäkinen K, Lepäntalo M & Biancari F (2008) Outcome of symptomatic, unruptured abdominal aortic aneurysms after endovascular repair with Zenith stent-graft system. *Scand Cardiovasc J* 42: 178–181.
- III Nevala T, Biancari F, Manninen H, Matsi P, Mäkinen K, Ylönen K & Perälä J (2010) Inferior mesenteric artery embolization before endovascular repair of an abdominal aortic aneurysm: effect on type II endoleak and aneurysm shrinkage. *J Vasc Interv Radiol* 21: 181–185.
- IV Nevala T, Biancari F, Manninen H, Aho PS, Matsi P, Mäkinen K, Roth WD, Ylönen K, Lepäntalo M & Perälä J (2010) Type II endoleak after endovascular repair of abdominal aortic aneurysm: effectiveness of embolization. *Cardiovasc Intervent Radiol*. In Press.

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1 Introduction

Abdominal aortic aneurysms (AAA) can be insidious, since they can be asymptomatic or mildly symptomatic until a rupture occurs. Ruptured abdominal aortic aneurysms (RAAA) cause approximately 1–2% of all deaths in the Western world (Wilmink *et al.* 1999, Vardulaki *et al.* 1999). The overall mortality rate for ruptured abdominal aortic aneurysm is 78–89%, and almost half of deaths happen outside of the hospital (Bengtsson & Bergqvist 1993, Kantonen *et al.* 1999, Brown & Powell 1999, Wilmink *et al.* 1999).

The goal of the treatment of an AAA is to exclude the aneurysm from the circulation to avert a rupture. Open surgery was the first method for treating an AAA (Dubost *et al.* 1951). This is a major surgical procedure consisting of a laparotomy or lumbotomy and cross-clamping of the abdominal aorta. Since 1991, an alternative minimally invasive method, endovascular aneurysm repair (EVAR), became available (Parodi *et al.* 1991, Volodos *et al.* 1991). EVAR is associated with lower operative mortality and similar mid-term survival compared with surgical repair (Greenhalgh *et al.* 2004, Prinssen *et al.* 2004, Blankensteijn *et al.* 2005, Greenhalgh *et al.* 2005b). Early experiences with first-generation stent-grafts were associated with a high rate of late complications including stent-graft migration, structural failures, limb occlusion and conversions to open repair. Since then, development of stent-grafts has improved the durability of EVAR (Torella 2004).

Signs related to a symptomatic AAA are abdominal pain, back pain and tenderness over the aneurysm. Open repair of a symptomatic, intact AAA is associated with an increased risk of immediate postoperative mortality and morbidity (Leo *et al.* 2005). Treatment of such a condition is challenging, and endovascular treatment is likely to offer an appealing alternative to open repair.

An endoleak, defined as persistent blood flow outside the graft and within the aneurysm sac, remains a long-term problem. Clinical significance and the need for a secondary intervention differ according to the type of endoleak. Type I (leak from the stent-graft attachment site) and type III (structural failure of the stent-graft) endoleaks are considered major complications, potentially leading to an aneurysm rupture, and thus requiring a prompt secondary intervention (van Marrewijk *et al.* 2002). The pathogenesis of a type II endoleak (retrograde perfusion via aortic side branches) and its significance, prevention and treatment are controversial. Type IV (caused by stent-graft porosity) is uncommon and self-

limited. Type V, called also endotension, is defined as a situation in which the sac remains pressurized in the absence of a detectable endoleak.

The aim of this thesis was to evaluate the short- and mid-term results of EVAR using the Zenith[®] stent-graft (Cook Inc, Bloomington, IN, USA) in asymptomatic and symptomatic AAA patients in three Finnish university hospitals. Furthermore, the aim was to study whether preoperative embolization of the inferior mesenteric artery (IMA) before EVAR decreases the incidence of a type II endoleak or has an effect on sac shrinkage. Finally, the results after a secondary intervention for a type II endoleak were evaluated.

2 Review of the literature

2.1 Abdominal aortic aneurysm

2.1.1 Anatomy of the abdominal aorta

The aorta is the main artery which delivers oxygenated blood from the heart to the body. The aorta is divided into ascending, arch, descending thoracic and abdominal parts. The abdominal part begins at the level of the diaphragm, descends to the level of the fourth lumbar vertebra and branches into two common iliac arteries. The common iliac arteries branch into the external and internal iliac arteries at the level at the lumbosacral intervertebral disc. (Gray *et al.* 1989). The normal diameter of the aorta varies with sex, age and bodyweight (Bengtsson *et al.* 1996, Grimshaw & Thompson 1997). It is proposed that an infrarenal abdominal aortic diameter greater than 30 mm is pathologic (McGregor *et al.* 1975). Reporting standards from 1991 suggested that an abdominal aortic aneurysm (AAA) should be defined as a dilatation of the aorta that has at least a 50% increase in diameter compared with the expected normal diameter (Johnston *et al.* 1991). Though an abdominal aortic aneurysm could be located in any part of the infradiaphragmatic aorta, the term AAA commonly refers to an infrarenal aorta (Figure 1).

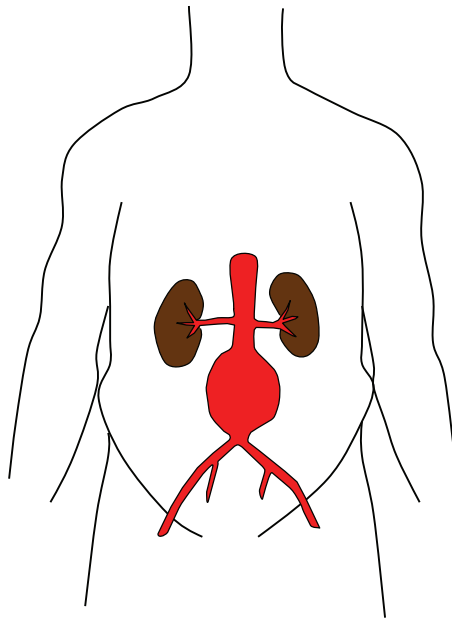


Fig. 1. Infrarenal abdominal aortic aneurysm.

The anterior and anterolateral branches of the abdominal aorta are the celiac, superior mesenteric (SMA), gonadal, phrenic and inferior mesenteric (IMA) arteries. The lateral branches are the renal and middle suprarenal arteries. The posterior branches are four lumbar artery pairs and the middle sacral artery. The fifth lumbar artery pair is small and can occasionally rise from the middle sacral artery, but lumbar arteries from the iliolumbar arteries branching from the internal iliac arteries usually take their place. (Gray *et al.* 1989). The IMA, lumbar arteries and occasionally the accessory renal arteries are the arteries which can arise from infrarenal AAA's. The lumbar arteries have numerous anastomoses with the middle sacral artery, iliolumbar arteries, intercostal arteries, epigastric arteries and deep iliac circumflex artery. The IMA has anastomosis with the SMA through the ascending branch of the IMA and the left colic artery (arcade of Riolan). (Uflacker 2007).

2.1.2 Epidemiology of AAAs

AAAs are more common in men than in women. The prevalence of AAAs varies between 4.1% and 14.2% in men and between 0.35% and 6.2% in women (Cornuz *et al.* 2004). The incidence of AAAs has increased during the last decades (Heikkinen *et al.* 2002a). The reason for this is probably the ageing of the population, an increase in smoking, screening programs and increasing use of ultrasonography.

2.1.3 Aetiology, risk factors and pathophysiology of AAAs

An AAA is quite seldom a direct consequence of a specific cause such as infection, inflammatory disease or connective tissue disorder (Towbin *et al.* 1999, Erentug *et al.* 2003). Most of the time, the aetiology is non-specific. The leading risk factors for development of an AAA include increasing age, smoking, male sex, family history and atherosclerotic diseases (Lederle *et al.* 2000, Singh *et al.* 2001, Frydman *et al.* 2003, Forsdahl *et al.* 2009). Smoking is strongly associated with aneurysm development (Brown & Powell 1999, Vardulaki *et al.* 2000, Brady *et al.* 2004). The risk factors for atherosclerosis are also found to be associated with increasing risk of developing AAA; hypertension, hypercholesterolemia, low HDL (high-density lipoprotein) cholesterol (Forsdahl *et al.* 2009). Numerous other possible aetiologies have been investigated, including connective tissue laxity, protease inhibitors, alcohol consumption, diabetes mellitus and body mass index (Wilmink *et al.* 2000, Defawe *et al.* 2003, Forsdahl *et al.* 2009).

Aneurysm formation is characterized by connective tissue degradation in the aortic wall. Elastic and collagen fibres are essentials for the aorta to have adequate distensibility. There is a growing understanding of the underlying reasons, such as inflammation, a complex remodelling process of matrix proteins and specific genes involved in this process (Knox *et al.* 1997, Rahkonen *et al.* 2004, Sakalihasan *et al.* 2005).

2.1.4 Rupture risk of AAAs

The risk of an aneurysm rupture is directly related to the aneurysm size (Szilagyi *et al.* 1966). The mean growth rate of small aneurysms is 2.0 mm-3.3 mm/year (Powell *et al.* 1998, Brady *et al.* 2000, Biancari *et al.* 2002a). However, expansion of large aneurysms could be as fast as 7.4 mm/year (Limet *et al.* 1991). Two large

trials have shown that surveillance of small aneurysms (4.0–5.4/5.5 cm) is safe and early surgery does not save lives (Powell *et al.* 1998, Lederle *et al.* 2002b). Estimated annual rupture risks are listed in Table 1 (Reed *et al.* 1997, Brown & Powell 1999, Brewster *et al.* 2003). According to a Finnish national database, a ruptured aortic aneurysm was the main cause of death for 276 people (220 men and 56 women) in Finland in 2007 (Statistics Finland 2009).

Table 1. Risk of rupture.

Diameter of the aneurysm (cm)	Annual rupture risk (%)
< 4	0
4–4.9	0.5–5
5–5.9	3–15
6–6.9	10–20
7–7.9	20–40
≥ 8	30–50

2.1.5 Clinical features, diagnosis and imaging of AAAs

Non-ruptured aneurysms are mostly asymptomatic and are diagnosed incidentally. Sometimes an AAA can become symptomatic and the symptoms may be nonspecific, such as abdominal pain and back pain, or more accurate such as tenderness over the aneurysm. According to a Finnish nationwide registry, symptomatic AAAs accounted for 19% of all patients with an unruptured AAA who underwent open repair (Biancari *et al.* 2003).

Abdominal palpation has only moderate sensitivity for detecting an AAA (Fink *et al.* 2000). An abdominal plain image can coincidentally be diagnostic if calcifications are seen, but it is not accurate enough. Ultrasonography (US) is the simplest method for making a diagnosis and measuring an AAA (Brewster *et al.* 1977, Wilmink *et al.* 2002). It is non-invasive, inexpensive and very accurate. If treatment is planned, then contrast enhanced computed tomography (CT) is the next step needed to determine the detailed anatomy of the AAA. Magnetic resonance imaging (MRI) is an alternative method to CT. Nowadays conventional angiography is very seldom necessary. If the detected AAA has a diameter ≤ 40 mm, rescreening with ultrasound is recommended at 24-month intervals. If the AAA has a diameter of 41–45 mm, it is safe to do surveillance at 12-month intervals, and with AAAs ≥ 46mm surveillance should be done every 6 months (Wilmink *et al.* 2000, Brady *et al.* 2004).

Radiologic signs of impending ruptures are difficult to identify or could be missing. The clearest sign of a high likelihood of an aneurysm rupture is increased aneurysm size (Lederle *et al.* 2002a). Imaging findings that predict an impending rupture are discontinuity in aortic wall calcifications and a well-defined peripheral crescent-shaped area of hyperattenuation within the thrombus (crescent sign) seen in CT (Siegel *et al.* 1994). Abdominal aorta aneurysm ruptures are mostly retroperitoneal and occur more often on the left than on the right (Darling *et al.* 1977).

Inflammatory AAAs

Inflammatory AAAs are relatively rare: the incidence range is 2.7–7.1% of the total number of AAAs (Lindblad *et al.* 1991, Puchner *et al.* 2006, Yusuf *et al.* 2007). It is characterized by inflammatory and/or fibrotic changes around the aortic aneurysm. In most cases the aetiology is unclear (Leu 1990, Yonemitsu *et al.* 1996). Inflammatory AAA can be asymptomatic or could show long-lasting non-specific symptoms like back pain and weight loss (Railo *et al.* 2005). Surgery appears to be more difficult due to fibrosis and perioperative mortality is three times higher than with noninflammatory AAAs (Pennell *et al.* 1985).

2.1.6 Treatment of AAAs

Asymptomatic AAAs can be treated electively. There is no strict single threshold diameter for treatment. According to current guidelines, a diameter of 5.5 cm is the best threshold for repair, but individualized consideration in each patient is essential (Brewster *et al.* 2003). In a review of ruptured AAAs from Finland, 24% of women with a rupture had an AAA < 5.5 cm (Heikkinen *et al.* 2002b). This result confirms the conception that with women it is reasonable to treat smaller aneurysms. Annual expansion of 1 cm or more or development of symptoms are also indications for treatment (Brewster *et al.* 2003).

Open surgery

The first open surgery for elective treatment of an AAA was performed almost 60 years ago (Dubost *et al.* 1951). An anterior transperitoneal or left-sided retroperitoneal approach can be used. The retroperitoneal approach is helpful when the AAA involves the pararenal or suprarenal abdominal aorta or when the

anterior transperitoneal approach is rendered because of previous operative scarring or the presence of stomas. Regardless of the approach, the technique is the same; proximal and distal clamping, dissection of the aorta, removal of the clot, suturing the orifices of the lumbar arteries, fixing the aortic graft and closing the aneurysm wall over the graft. (Hobson 2004) Reimplantation or ligation of the IMA at the time of open aortic surgery is controversial (Senekowitsch *et al.* 2006).

Elective open surgery is associated with 30-day mortality of 1.1–7% (Irvine *et al.* 2000, Ashton *et al.* 2002, Elkouri *et al.* 2004, Schermerhorn *et al.* 2008). Open surgery is a major procedure, with a complication rate between 9.4–21% (Hallett *et al.* 1997, Biancari *et al.* 2003, Lovegrove *et al.* 2008). A recent meta-analysis showed that cardiac and respiratory complications are significantly more common in open surgery than in EVAR: the cardiac complication rates were 7.2% in open surgery and 4.2% in EVAR, and the corresponding respiratory complication rates were 12.8% and 3.8%. Open surgery is also associated with a prolonged postoperative stay in the intensive care unit and in hospital. (Lovegrove *et al.* 2008) However, grafts are very durable and patients do not need lifelong surveillance (Hallett *et al.* 1997, Biancari *et al.* 2002b).

EVAR

Almost two decades ago, Volodos and Parodi introduced a less invasive endovascular method, endovascular aneurysm repair (EVAR) (Parodi *et al.* 1991, Volodos *et al.* 1991). During the past two decades this method has spread worldwide, and approximately 30% of AAAs in Finnish university hospitals were treated with EVAR in 2007 (Table 2).

Table 2. AAAs treated in university hospitals in Finland during 2007.

	HUH n (%)	KUH n (%)	OUIH n (%)	TAUH n (%)	TUH n (%)
Open surgery, ruptured	47 (40.2)	9 (16.4)	16 (27.6)	17 (23.0)	16 (28.1)
Open surgery, symptomatic	11 (9.4)	4 (7.3)	8 (13.8)	6 (8.1)	3 (5.3)
Open surgery, elective	48 (41.0)	15 (27.3)	15 (25.9)	16 (21.6)	29 (50.9)
Open surgery	106 (90.6)	28 (50.9)	39 (67.2)	39 (52.7)	48 (84.2)
TOTAL					
EVAR, ruptured	0 (0)	2 (3.6)	0 (0)	1 (1.4)	1 (1.8)
EVAR, symptomatic	2 (1.7)	3 (5.5)	4 (6.9)	3 (4.1)	0 (0)
EVAR, elective	9 (7.7)	22 (40)	15 (25.9)	31 (41.9)	8 (14.0)
EVAR TOTAL	11 (9.4)	27 (49.1)	19 (32.8)	35 (47.3)	9 (15.8)
TOTAL	117	55	58	74	57

HUH = Helsinki University Hospital, KUH = Kuopio University Hospital, OUIH = Oulu University Hospital, TAUH = Tampere University Hospital, TUH = Turku University Hospital.

Symptomatic AAAs

It is widely accepted that in order to prevent haemodynamic instability associated with an aneurysm rupture, repair of a symptomatic AAA should be carried out as soon as possible. However, the prognostic impact of an emergency versus an urgent/semi-elective setting is controversial (Haug *et al.* 2004, Tambyraja *et al.* 2004). Open repair of a symptomatic AAA is associated with a significantly increased risk of immediate postoperative mortality and morbidity. Reported mortality rates after open repair of symptomatic AAA are 5–18% (Biancari *et al.* 2003, Haug *et al.* 2004, Tambyraja *et al.* 2004, Leo *et al.* 2005). Recent reports indicate that emergency repair of symptomatic abdominal aortic aneurysms can be performed by endovascular grafting with encouraging results (Franks *et al.* 2006, Oranen *et al.* 2006).

2.2 Endovascular repair of an abdominal aortic aneurysm

The initial stent-grafts were tube endografts, which necessitated at least 1 cm landing zone in the distal aorta. Quite soon bifurcated stent-grafts with distal attachment to the iliac arteries become preferred. These first-generation devices

could not tolerate the forces exerted on the aorta over time, and structural failures were common. Experiences with these first-generation devices were disappointing considering mid-term results (Aho *et al.* 2002, Väärämäki *et al.* 2007). Since then there has been considerable evolution of stent-grafts and this has improved the results of EVAR (Torella 2004).

During the past decade, data concerning EVAR have been collected by several sources, including two registries, EUROSTAR (European Collaborators Registry on Stent-graft Techniques for AAA repair) (Vallabhaneni & Harris 2001) and RETA (Registry for Endovascular Treatment of Aneurysms) (Thomas *et al.* 2005), as well two large, European, prospective, multicenter randomised controlled trials, EVAR trial 1 (Endovascular Aneurysm Repair trial 1) and DREAM (Dutch Randomised Endovascular Management Trial). These two randomized trials have demonstrated a clear, significant mortality benefit at 30 days in patients with an AAA larger than 5–5.5 cm treated by endovascular repair (Greenhalgh *et al.* 2004, Prinssen *et al.* 2004). The reported 30-day mortality rates were 4.7% and 4.6% for open repair and 1.7% and 1.2% for EVAR, respectively. Recently a third randomized multicenter trial from the United States (Open Versus Endovascular Repair Veterans Affairs Cooperative Study) reported short-term results showing that perioperative mortality was low for both procedures and it was lower for endovascular than for open repair, 0.5% vs. 3% (Lederle *et al.* 2009). There is no longer debate about the other early benefits of EVAR: a shorter hospital stay, less blood loss, shorter operation times and lower early morbidity.

Despite the fact that the benefit of overall survival rates at two and four years is lost, the benefit of aneurysm-related deaths persists (Blankensteijn *et al.* 2005, Greenhalgh *et al.* 2005b). A high rate of complications and secondary interventions remains the long-term problem with EVAR.

2.2.1 EVAR in high-risk patients

The evolution of EVAR has allowed higher-risk patients, unfit for open repair, to be offered EVAR as an alternative to conservative treatment. However, there are conflicting data concerning this issue. EVAR trial 2 (Endovascular Aneurysm Repair trial 2) compared patients unfit for open surgery by randomising them into an EVAR group and a best medical therapy group. (Greenhalgh *et al.* 2005a). There were no differences in four-year aneurysm related mortality or all-cause mortality. On the other hand, recent studies have suggested a benefit of EVAR over open surgery in a high-risk population (Sicard *et al.* 2006, Bush *et al.* 2007).

The problem in identifying such high-risk patients is, however, the lack of well-derived and validated risk scoring methods specifically planned for patients with an AAA. Among the currently available risk scoring methods, the Glasgow aneurysm score (GAS) is a simple scoring system specifically derived from patients who have undergone open repair of an AAA (Samy *et al.* 1994) and it has been shown to perform well in predicting postoperative mortality and morbidity in patients undergoing elective open repair of an AAA (Nesi *et al.* 2004). It has also been evaluated as a predictor of survival following EVAR (Biancari *et al.* 2006, Baas *et al.* 2008). The GAS is calculated according to the following formula: risk score = (age in years) + (7 points for myocardial disease) + (10 points for cerebrovascular disease) + (14 points for renal disease).

2.2.2 EVAR technique

EVAR is dependent on and limited by aneurysm morphology. Planning the EVAR involves detailed measurements of the aneurysm anatomy. At the beginning of the EVAR era the EUROSTAR criteria for anatomic suitability were quite strictly followed; the infrarenal aortic neck should be at least 15 mm long, no more than 25 mm wide and less than 60 degrees angulated (Harris *et al.* 1997). The external iliac artery should also be wide enough to permit deployment of the stent-graft to the aorta. There are studies from almost a decade ago reporting that 30–68% of patients are suitable for EVAR (Carpenter *et al.* 2001, Woodburn *et al.* 2001, Wilson *et al.* 2004). As a result of experience, developing techniques and devices, more and more challenging AAAs are nowadays treated with EVAR, and the suitability rates are rising. Even AAAs with a hostile neck anatomy could be nowadays treated with a standard stent-graft (Choke *et al.* 2006).

The attachment of stent-graft devices is based on radial force and in some cases, barbs attached to the arterial wall. Proximal 10–20% oversizing of the stent-graft is necessary to obtain a seal between aortic wall and stent-graft (Mohan *et al.* 2001).

Anaesthesia used with EVAR could be local, regional or general anaesthesia. Especially high-risk patients attain advantages from minimally invasive anaesthesia techniques (Ruppert *et al.* 2007). The stent-graft is installed via femoral arteries using percutaneous or open access. It is proposed that with percutaneous access there are less groin complications (Smith *et al.* 2009).

The most common stent-graft configuration is bifurcated. Aortouni-iliac devices are used for patients with a unilateral iliac artery occlusion or stenosis.

With aortouni-iliac devices, the contralateral common iliac artery is obstructed with an occluder when necessary. The stent-graft is inserted using fluoroscopic guidance.

2.2.3 Stent-grafts

Among the stent-grafts currently in use, the Zenith[®] stent-graft is one of the most employed (Biancari *et al.* 2006). By the autumn of 2009 the Zenith[®] stent-graft was employed in 41500 patients in Europe and in 44000 in the USA (Paananen 2009). In EVAR trial 51% of inserted stent-grafts were Zenith[®] stent-grafts, 33% were Talent[®] (Medtronic, Minneapolis, MN, USA) and 7% were Excluder[®] (W.L. Gore, Flagstaff, AZ, USA) stent-grafts (Greenhalgh *et al.* 2004). These three second/third-generation stent-grafts were the ones most used in the beginning of the current decade in Finland. These stent-grafts are bifurcated, modular, fully supported, self-expandable and have additional components such as proximal cuffs and extension limbs. All the devices have undergone changes and improvements over time. The main differences between stent-grafts concern proximal fixation, deployment mechanism, graft flexibility and the size of the introduction system.

The Talent[®] stent-graft is a modular two-piece system, with a main body with a full length limb and a contralateral limb. It is a woven polyester graft with interspersed self-expanding nitinol stents. It has suprarenal proximal fixation with a non barbed bare metal portion. (Chavan *et al.* 2000)

The Excluder[®] stent-graft is a modular two-piece system made of an expanded polytetrafluoroethylene (PTFE) graft bonded to nitinol stents. It has infrarenal proximal fixation with barbs. The Excluder[®] stent-graft has flexible and thinner wall device limbs and it is suitable for patients with narrow and angulated iliac arteries. It is mounted on a catheter, in contrast to the others, which are preloaded into an introduction system. (Matsumara *et al.* 2001)

The Zenith[®] stent-graft is a modular three-piece stent-graft with multiple stainless steel stents and woven polyester fabric (Figure 2). The characteristic feature of the Zenith[®] stent-graft is an uncovered suprarenal stent portion with fixation barbs. (Kaviani & Greenberg 2004) Suprarenal fixation allows treatment of shorter necks with less risk of migration. The length of both limbs can be chosen after insertion of the main body, thus the graft is very useful when accurate length measurements prove difficult. The main body of the device is longer than that of the other stent-grafts, thus the contralateral limb is closer to the

contralateral iliac artery. It was introduced for clinical use in 1993 (Lawrence-Brown *et al.* 1996), and over time it has undergone several improvements (Verhoeven *et al.* 2006). The main body is available from 22–36 mm and the iliac limbs are 8–24 mm in diameter. The stent-graft is preloaded into an introduction system, which is available in 18 to 22-F (French) sizes, depending on the size of the stent-graft itself. Because of the wide range of sizes, a broad range of aortic and iliac arteries can be treated.

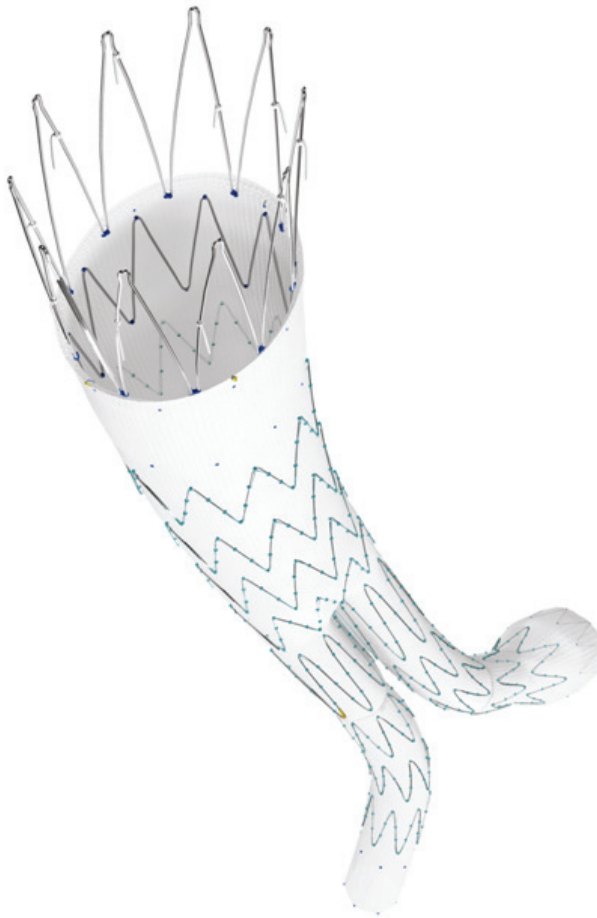


Fig. 2. The Zenith® stent-graft. Reprinted with permission from Cook Inc.

The differences in device design and the introduction system allow for appropriate stent-graft selection according to the patient's vascular anatomy. Because of the obvious clinical selection bias that affects studies, a comparison

between stent-grafts is difficult (Verhoeven *et al.* 2004). There is no evidence of significant differences in the clinical outcome between stent-grafts that are in active use (Brown *et al.* 2007, Abbruzzese *et al.* 2008). However, suprarenal proximal fixation seems to be associated with lower migration rates (Abbruzzese *et al.* 2008). Studies concerning the Zenith[®] stent-graft are summarized in Table 3 (Greenberg *et al.* 2001, Abraham *et al.* 2002, Alric *et al.* 2002, Hinchliffe *et al.* 2004, Greenberg *et al.* 2004b, Lalka *et al.* 2005, Hiramoto *et al.* 2007, Becquemin *et al.* 2008, Bos *et al.* 2008, Greenberg *et al.* 2008, D'Elia *et al.* 2009).

Table 3. Previous studies evaluating the results of the Zenith[®] stent-graft in patients with an AAA.

Study	Study design	Number of patients	Mean follow-up length (years)	30-day mortality (%)
D'elia 2009	Single-center study. Jan 2004-Dec 2008.	501	1.9	1.8
Becquemin 2008	Single-center study. Jan 2000-Nov 2004	212	1.5	0.9
Bos 2008	Single-center study. Mar 1999-Dec 2006.	234	2.2	1.7
Greenberg 2008	Continuation of a pivotal Zenith [®] multicenter study as well continued access. Jan 2000-June 2003.	739	ND	ND
Hiramoto 2007	Single-center study. Oct 1998-Dec 2005.	325	2.3	ND
Lalka 2005	Single-center study. Feb 2000-Jan 2005.	136	3	0
Greenberg 2004	Prospective, multicenter, nonrandomized, Zenith [®] multicenter study. Jan 2000-July 2001.	432	1-2	0.5
Hinchliffe 2004	UK retrospective, multi-center study. July 1996-Nov 2002.	269	1	4.1
Alric 2002	Single-center study. July 1996-Sep 2001.	88	1.7	3.4
Abraham 2002	Single-center prospective study. Oct 1998-July 2001.	116	0.9	0.9
Greenberg 2001	Prospective databases at seven centers. 1995-	528	1.2	ND

ND = no data available

2.2.4 Late complications after EVAR

EVAR is associated with late stent-graft-related complications such as migration, structural failures, occlusion, kinking, infection, endoleak and rupture. Migration is defined as caudal movement of the stent-graft of more than 10 mm (Chaikof *et al.* 2002). Migration and structural failures were significant problems with first-generation devices (Jacobs *et al.* 2003, van Marrewijk *et al.* 2005), but with modern stent-grafts they occur relatively seldom. Reported migration rates among modern stent-grafts are 0–25% (Peterson *et al.* 2007, Pitton *et al.* 2009). Reported migration rates with the Zenith[®] stent-graft are low: 0–2.9% (Bos *et al.* 2008, Greenberg *et al.* 2008). There are only occasional reports of structural failures (Rumball-Smith *et al.* 2006, Hiramoto *et al.* 2007). An occlusion of a stent-graft is also quite a rare complication with modern devices, and it is observed in 0–3.5% of patients (Hiramoto *et al.* 2007, Brown *et al.* 2007). Stent-graft kinking is usually defined as stent-graft bending, which requires a secondary intervention and can appear at the same time as an occlusion. The reported kinking rates are 0–1.6% (Verhoeven *et al.* 2004, Brown *et al.* 2007). The incidence of stent-graft infection is 0.4–0.7 (Fiorani *et al.* 2003, Veraldi *et al.* 2009). In comparison, the reported infection rate with open surgery is 0.7–1.3% (Hallett, Jr. *et al.* 1997, Lovegrove *et al.* 2008). The aneurysm rupture rate is less than one percent (Bos *et al.* 2008, Abbruzzese *et al.* 2008).

Endoleaks

The significance, nature and treatment vary significantly between different types of endoleaks. An endoleak is called primary when it occurs perioperatively (≤ 30 days) and any endoleak occurring thereafter is called secondary. The reappearance of an endoleak is called a recurrent endoleak.

A type I endoleak is defined as a sealing failure at the proximal or distal attachment sites of the stent-graft or an inadequately sealed iliac occluder. A type I endoleak seen immediately after stent-graft placement has been thought to be the result of wrong patient selection with an unsuitable anatomy, a wrong type of stent-graft or maldeployment of the stent-graft. If it develops later, it could be related to artery dilatation. A type I endoleak has been reported in 1.5–10% of patients (Veith *et al.* 2002, van Marrewijk *et al.* 2002, Hinchliffe *et al.* 2004, Bos *et al.* 2008).

A type II endoleak after EVAR is a result of retrograde perfusion of the aneurysm sac via the IMA or lumbar arteries or other arteries, such as an accessory renal artery. A type II endoleak is the most common endoleak, present in 8–23% of patients (Alric *et al.* 2002, Hinchliffe *et al.* 2004, Hiramoto *et al.* 2007, Bos *et al.* 2008). It has been shown that about 50–80% of type II endoleaks resolve spontaneously (Silverberg *et al.* 2006, Jones *et al.* 2007, Higashiura *et al.* 2007). A type II endoleak can appear in any time during surveillance.

A type III endoleak is the result of device failure, usually due to disintegration of the graft fabric or disconnection of the modular limb. A type III endoleak has been reported in approximately in 1% of patients (Alric *et al.* 2002, Bos *et al.* 2008).

A type IV endoleak is a consequence of graft porosity and can only occur within 30 days of stent-graft insertion. It resolves spontaneously. Endotension is a condition where a sac remains pressurized in the absence of a detectable endoleak (Gilling-Smith *et al.* 1999). The exact reason for endotension is unclear. It is thought to be secondary to transmission of aortic pressure to the sac (Dias *et al.* 2004). An alternative mechanism for endotension could also be accumulation of excess fluid around the stent-graft and formation of hygroma (Risberg *et al.* 2001). It could also be that endotension represents a situation where imaging modalities can not detect an existing endoleak.

Secondary interventions

A secondary intervention is defined as any late procedure performed to maintain the functionality of the stent-graft, whether performed percutaneously or surgically. Reported secondary intervention rates vary between 7% and 20% (Alric *et al.* 2002, Greenhalgh *et al.* 2005b, Greenberg *et al.* 2008, Conrad *et al.* 2009).

Type I and III endoleaks are associated with an increased risk of rupture, and they need to be treated without delay (van Marrewijk *et al.* 2002) They are usually treated with a stent-graft extension or additional stenting. Type II endoleaks are often benign but sometimes they can be associated with an expansion of the aneurysm sac and a rupture, and therefore they require a secondary interventions (Jones *et al.* 2007).

Three quarters of secondary interventions are performed using an endovascular approach (Conrad *et al.* 2009). Open conversion is needed when a complication can not be treated with the endovascular approach. These situations

include stent-graft infection, endotension, a proximal type I endoleak when insertion of a proximal cuff is not feasible and a stent-graft main body occlusion. Elective late conversions have a mortality rate similar to that of primary open repair, but the mortality rate in emergency late conversions has been reported to be up to 19% (Kelso *et al.* 2009).

2.2.5 Follow-up imaging

Lifelong surveillance is thought to be necessary, particularly because late complications could appear after several years (Vallabhaneni & Harris 2001). CT has been the most commonly used imaging method and it has been performed at 1, 3, 6, 12 and 18 months after operation and annually thereafter. We do not yet have long-time data on the durability of second generation stent-grafts, but because of the encouraging results of mid-term data, many centers have loosen the surveillance intervals (Kranokpiraksa & Kaufman 2008).

With follow-up imaging it is essential to measure aneurysm size, to detect and classify a possible endoleak and to detect any morphologic changes in the stent-graft. Maximum aneurysm sac diameter has generally been used to follow the sac size, although time-consuming volume measurements have been proposed as being more accurate (Bargellini *et al.* 2005). According to the reporting standards guidelines, an aneurysm diameter increase ≥ 5 mm or a volume increase $\geq 5\%$ is considered significant (Chaikof *et al.* 2002).

Follow-up modalities include CT, plain radiograph, MRI and color duplex US with or without contrast enhancement. Plain radiographs are still used, and some authors considered them superior to CT in detecting device integrity (Fearn *et al.* 2003).

Contrast-enhanced CT is the most widely used surveillance modality. Along with multidetector scanners, volumetric data with isotropic voxel size are nowadays routinely available. Improvements in CT with advanced workstation tools have caused re-evaluation of the clinical value of radiographs in detecting morphologic changes in stent-grafts. Triphasic CT angiography (precontrast, arterial and delayed phases) has been recommended, but because of concerns regarding radiation, it is suggested that a precontrast scan may be necessary only in the first post-EVAR imaging (Iezzi *et al.* 2006). Contrast-enhanced CT is more sensitive for detecting an endoleak than is conventional catheter angiography (Armerding *et al.* 2000).

The advantage of a catheter angiography is its capability to detect flow direction and to classify endoleaks. Catheter angiography should be reserved for complex and ambiguous cases.

Gadolinium-enhanced MR is at least as sensitive as CT (Pitton *et al.* 2005), but its usefulness depends on the composition of the stent-graft. Certain materials cause artefacts, and with some materials there is also a lack of data concerning the safety issues.

US is widely used in surveillance with small AAAs before open surgery or EVAR, and in some cases it is also used in post-EVAR surveillance. Aneurysm size measurements in US correlate well with those obtained with CT (Raman *et al.* 2003, Aburahma *et al.* 2005), but colour duplex US has sensitivity of only up to 69% for detecting endoleaks (Aburahma *et al.* 2005, Ashoke *et al.* 2005). However, contrast-enhanced US (CEUS) has markedly improved the diagnostic value of US in detecting endoleaks (Iezzi *et al.* 2009).

2.2.6 Costs of EVAR

EVAR trial 1 showed that the cost of EVAR was higher than that of open repair in similar patient groups (Greenhalgh *et al.* 2005b). Smaller, retrospective studies have come to same conclusion (Clair *et al.* 2000, Angle *et al.* 2004). On the other hand, a recent study from Sweden showed that in a population-based setting the total cost was similar for EVAR and open repair (€29786 versus €26382). There were important differences in patient characteristics and the cost structure. The EVAR patients were older and they had more comorbidity. Preoperative costs were lower for open repair while peri- and postoperative costs were similar. The cost of intensive care was higher for open repair patients while EVAR had a higher implant cost. (Mani *et al.* 2008) An activity-based cost analysis of a Finnish interventional radiologic unit also showed that the cost of the stent-graft formed 87% of the total cost in EVAR (Rautio *et al.* 2003).

2.3 Management of type II endoleaks

2.3.1 Prevention of type II endoleaks

IMA embolization

The clinical significance and treatment of patent IMA-related type II endoleaks is controversial. It has been shown that most preoperatively patent IMAs occlude after EVAR (Dorffner *et al.* 2001). There are studies that did not find any correlation between the patency status of IMAs and type II endoleaks (Sheehan *et al.* 2006) or were unable to demonstrate an association between patent aortic side branches and postoperative endoleaks (Petrik & Moore 2001, Back *et al.* 2003). On the other hand, preoperative IMA embolization has been reported to reduce the incidence of type II endoleaks (Axelrod *et al.* 2004). Some authors reported that type II endoleaks correlate with a higher prevalence of IMA patency (Velazquez *et al.* 2000, Fan *et al.* 2001, van Marrevjik *et al.* 2002, Sampaio *et al.* 2005). It has also been observed that the diameter of the patent IMA correlates with the development of type II endoleaks (Arko *et al.* 2001).

Internal iliac artery embolization

When the common iliac artery is aneurysmatic, the stent-graft limb has to extend to the external iliac artery. To prevent a type II endoleak from the internal iliac artery (IIA), it should be embolized before stent-graft insertion. Bilateral IIA embolization is generally avoided, but when necessary, it could be done with limited morbidity (Mehta *et al.* 2004). A unilateral IIA occlusion only rarely causes severe ischaemic complications, such as colon ischaemia, disabling claudication and impotence (Karch *et al.* 2000).

Lumbar artery embolization

The effectiveness of preoperative lumbar artery embolization is doubtful, and this procedure is not generally used. There are a few studies that reported feasibility and a positive effect of lumbar artery embolization on type II endoleak rates (Parry *et al.* 2002, Bonvini *et al.* 2003). On the other hand, there are also studies that suggest a lack of influence on the incidence of type II endoleak (Gould *et al.*

2001). Studies on this issue include both lumbar and IMA embolization, which prevent any conclusion on this matter. Further, it is extremely challenging to embolize all the lumbar arteries.

Other techniques

It is difficult and almost impossible to preoperatively embolize all the aortic branches, and therefore injection of liquid embolization material into the aneurysm sac during stent-graft insertion appears to be an attractive technique. Injection of fibrin glue (a mixture of fibrinogen and thrombin) into the aneurysm sac immediately after the initial stent-graft insertion has been shown to be a safe and effective technique; there were only 2.4% delayed type II endoleaks (Zanchetta *et al.* 2007). It has also been shown that intraoperative embolization of the IMA and thrombin injection into the aneurysm sac has a trend toward lower type II endoleak rates (Muthu *et al.* 2007). Blood pressure control may also be an important factor in the management of type II endoleak (Hiramoto *et al.* 2008).

2.3.2 Treatment of type II endoleaks

Treatment of type II endoleaks is challenging because there could be many potential inflow and outflow arteries, and an endoleak behaves similarly to an arterial malformation (Baum *et al.* 2002).

Indications

The understanding of and treatment approach to type II endoleaks have changed significantly during the EVAR era. In the early years embolization was liberally done if the patient had a permanent type II endoleak (seen in 6-month surveillance imaging). In the light of current knowledge, a type II endoleak should be treated only in case of an increase in aneurysm size (van Marrewijk *et al.* 2002, Steinmetz *et al.* 2004, Tolia *et al.* 2005). According to reporting standards an AAA diameter change ≥ 5 mm or a volume change $\geq 5\%$ is considered significant (Chaikof *et al.* 2002). Two of the above-mentioned studies used a 5 mm threshold and one (van Marrewijk *et al.* 2002) used a threshold of 8 mm.

Techniques

The treatment options for type II endoleaks include transarterial embolization (Sheehan *et al.* 2004, Stavropoulos *et al.* 2009), translumbar embolization (Baum *et al.* 2002, Ellis *et al.* 2003), transcaval embolization (Mansueto *et al.* 2005), endoscopic ligation of the lumbar and mesenteric arteries (Wisselink *et al.* 2000), and open surgical repair. The embolization materials used are coils, glue, ethylene–vinyl alcohol copolymer and thrombin.

The results of previous studies reporting on the treatment of type II endoleaks are summarized in Table 4 (Gorich *et al.* 2000, Martin *et al.* 2001, Haulon *et al.* 2001, Liewald *et al.* 2001, Chuter *et al.* 2001a, Baum *et al.* 2002, Solis *et al.* 2002, Kasirajan *et al.* 2003, Faries *et al.* 2003, Sheehan *et al.* 2004, Stavropoulos *et al.* 2009). The number of patients treated is quite small, and the success rates of transarterial embolization vary greatly, from 11% to 100%. The success rates in the three studies on translumbar embolization varied between 72% and 92%.

Table 4. Clinical reports on the outcomes of secondary interventions for type II endoleaks.

Study	Technique	Number of patients	Success n (%)
Stavropoulos 2009	transarterial	23	18 (78)
	translumbar	62	45 (72)
Sheehan 2004	transarterial	19	15 (78.9)
Kasirajan 2003	transarterial	8	6 (75)
Faries 2003	transarterial	16	14 (87.5)
Solis 2002	transarterial	10	4 (40)
Baum 2002	transarterial	20	4 (20)
	translumbar	13	12 (92)
Haulon 2001	transarterial	18	17 (94.4)
Chuter 2001	transarterial	9	1 (11)
Martin 2001	translumbar	4	3 (75)
Liewald 2001	transarterial	14	8/13 (62)*
Gorich 2000	transarterial	11	11 (100)

*One endoleak resolved spontaneously

3 Purpose of the study

The purpose of the present study was:

1. To assess the short- and mid-term results of an endovascular repair of an AAA with the Zenith[®] stent-graft.
2. To evaluate the outcome of patients who underwent EVAR for a symptomatic, unruptured AAA.
3. To evaluate the effect of preoperative IMA embolization on type II endoleaks and sac shrinkage.
4. To evaluate the results of treating a type II endoleak after EVAR.

4 Material and methods

4.1 Study population

The present study was a retrospective, multicenter study which was conducted in Oulu University Hospital (OUH), Kuopio University Hospital (KUH) and Helsinki University Hospital (HUH) in Finland. From January 2000 to October 2006, a total of 256 patients underwent endovascular repair of a nonruptured infrarenal AAA with the Zenith® stent-graft. Patients with an isolated iliac artery aneurysm were excluded from this study. The institutional review board did not require their approval for this retrospective study. Permission to use nationwide data was obtained from the Ministry of Social Affairs and Health. Causes of death were obtained from the hospitals' patient documents and from Statistics Finland. The number of patients in the original papers is presented in Figure 3.

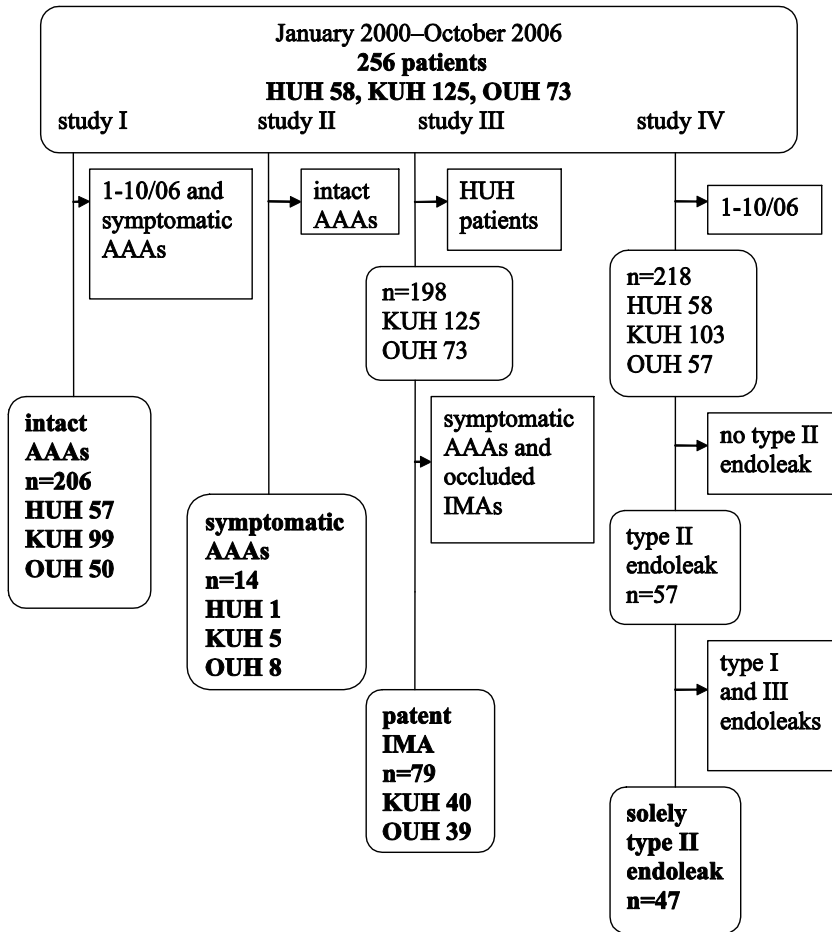


Fig. 3. Number (n) of patients in the original papers. Excluded patients are marked with arrows.

Study I

Study I included 206 patients who underwent elective EVAR of an intact AAA during January 2000–December 2005. Patients who underwent urgent repair of a symptomatic AAA were excluded from this study. The operative risk of the patients was assessed according to the Glasgow aneurysm score (GAS) (Samy *et al.* 1994). As shown by the high GAS (mean 80.0 ± 9.7 , range 53–115), most of the patients had a significantly increased operative risk and thus were not fit for major open surgery.

Study II

Study II included 14 patients who underwent urgent EVAR for a symptomatic, unruptured AAA during January 2000-October 2006. The median delay from admission to intervention was 4 days. All the patients were treated within 15 days.

Study III

Study III included 198 patients who underwent elective or urgent endovascular repair of an AAA in KUH and OUH. In KUH the clinical rationale included routine preoperative embolization if a patent IMA was detected on CT, while in OUH preoperative IMA embolization was not done. Altogether forty patients treated at KUH had a patent IMA on CT and were treated successfully with coil embolization before EVAR. Thirty-nine patients who underwent EVAR at OUH without preoperative embolization of a patent IMA served as a control group.

Study IV

Study IV included 218 patients who underwent elective or urgent endovascular repair of an AAA. A type II endoleak was observed in 57 patients. After exclusion of ten patients who also had a type I or type III endoleak, 47 patients who had solely a type II endoleak at some point during the follow-up constituted the material for this study. Overall, 14 patients underwent a secondary intervention to repair the type II endoleak.

The patients' characteristics are reported in Table 5. The anatomic criteria of all the patients before EVAR were assessed with preoperative contrast-enhanced CT. At the beginning of the study, most of the patients had also catheter angiography. Vascular surgeons and radiologists together determined their suitability for EVAR. The follow-up was planned as a contrast-enhanced CT before discharge, at 3 and 12 months, and annually thereafter. Anteroposterior and lateral abdominal radiographs were taken at the same time points. The clinical surveillance was done by a vascular surgeon.

Table 5. Preoperative patients' characteristics.

Risk factors	Study I	Study II	Study III	Study IV
	n (%)	n (%)	n (%)	n (%)
Mean age (years)	73.2 ± 7.3	78.4 ± 5.9	71.2 ± 7.1	73.8 ± 8.2
Males	181 (87.9)	10 (71)	34 (85)	40 (85.1)
Diabetes	27 (13.1)	1 (7)	7 (17.5)	6 (12.8)
Smoking habit	116 (56.3)	5 (36)	13 (32.5)	25 (53.2)
Transient ischaemic attack	11 (5.3)	0	2 (5)	2 (4.3)
Stroke	28 (13.6)	3 (21)	4 (10)	6 (12.8)
Coronary artery disease	111 (53.9)	9 (64)	24 (60)	23 (48.9)
Myocardial infarction	76 (36.9)	5 (36)	12 (30)	18 (38.3)
Previous coronary artery revascularization	61 (29.6)	5 (36)	13 (32.5)	12 (27.7)
Previous peripheral vascular interventions	19 (9.2)	0	3 (7.5)	1 (2.1)
Lower limb ischaemia	21 (10.2)	0	9 (22.5)	3 (6.4)
Dyslipidemia	88 (42.7)	5 (36)	21 (52.5)	22 (46.8)
Haemo-/peritoneal dialysis	4 (1.9)	0	0	2 (4.3)
Atrial fibrillation	39 (18.9)	3 (21)	5 (12.5)	10 (21.3)
Chronic heart failure	31 (15.0)	2 (14.3)	2 (5)	6 (12.8)
Hypertension	115 (55.8)	8 (57)	30 (75)	26 (55.3)
Chronic obstructive pulmonary disease/asthma	51 (24.8)	6 (43)	10 (25)	8 (17)
Serum creatinine > 150 µmol/l	11 (5.5)	0	0	4 (8.5)
Mean Glasgow aneurysm score	80.0 ± 9.7	85.1 ± 7.2	76.8 ± 8.1	81.0 ± 11.0

4.2 Methods

The data were collected from the hospitals' patient data, from either electronic or paper versions. The data collection form is shown in appendix 1. If the surveillance was carried out in a central hospital, local colleagues were contacted to obtain follow-up data.

4.2.1 Definitions

The results were reported according to the reporting standards for EVAR specified by the Society for Vascular Surgery / American Association for Vascular Surgery (Chaikof *et al.* 2002).

The technical success of EVAR on an intention-to-treat basis was defined as successful introduction and deployment of the device in the absence of surgical conversion or mortality, type I or III endoleak, or graft limb obstruction. The term assisted primary technical success was used when unplanned endovascular or

surgical procedures were necessitated. These procedures were percutaneous transluminal angioplasty, stenting, coil embolization, embolectomy, endarterectomy, insertion of a proximal cuff, insertion of an occluder and a femoral-femoral bypass. Based on reporting standards criteria, for technical success we used information from the first 24 hours: thus, we used the data from the initial procedure.

Stent-graft patency was given as primary, assisted primary or secondary patency. Primary patency was defined as a patent stent graft without a significant stenosis ($> 30\%$) or occlusion. Assisted primary patency was defined as a failing patent stent graft that underwent a further intervention, such as stenting, to improve patency. Secondary patency was defined as a patent stent graft after additional endovascular or surgical procedures.

Primary clinical success was reported on an intention-to-treat basis and was considered a success if there was no need for additional procedures. In study I, data from 198 patients were used for the success analysis. Access failures and 30-day operative deaths were excluded. If there was a type I endoleak that disappeared without intervention, we considered it a clinical success. Clinical failure was considered when a type I or III endoleak, graft thrombosis or infection, aneurysm expansion, aneurysm rupture, aneurysm-related death, migration or failure of device integrity occurred.

Secondary procedures were defined as aneurysm-related procedures, whether percutaneous or surgical, including conversions. Kinking was defined as noteworthy when it required a secondary intervention. Aneurysm size was measured by the maximum diameter of axial images on CT. A change in aneurysm size was considered significant if the change was ≥ 5 mm. Primary conversion was defined as converting to open repair during the same operation. Secondary conversion was defined as conversion to open repair at any time during the follow-up. Secondary conversion was also classified as elective or urgent.

Technical success for embolization of a type II endoleak was defined as successful deployment of embolization material to the endoleak cavity (an intention-to-treat basis). Embolization was considered clinically successful when resolution of the endoleak was achieved without enlargement of the aneurysm sac on a follow-up CT scan.

4.2.2 Operative technique

Stent-graft placement

The stent grafts were installed through surgically exposed common femoral arteries utilizing fluoroscopic guidance with large image field angiographic equipment. The procedure was performed using either local, epidural or general anaesthesia. The procedure was carried out in all cases as a collaborative effort by vascular surgeons and interventional radiologists. The hospitals in this study are of the same size and the treatment teams in the three centers have equal experience, and they employ the same type of technology for such interventions. Most of the Zenith® stent-grafts were Trifab version devices. Aortouni-iliac grafts were used if another iliac artery was occluded, severely stenosed or the contralateral limb was not successfully catheterized. A tubular stent graft was used in patient who had an abdominal aorta aneurysm with a long distal neck.

Preoperative aortic side branch embolization

If the aneurysm involved the distal common iliac artery or external iliac artery, the stent-graft limb was extended to the external iliac artery. These patients underwent IIA embolization before stent grafting. Besides this, some patients underwent preoperative IMA and/or lumbar artery embolization. Indications for these preoperative coiling procedures varied among the three institutions. In KUH, preoperative embolization of the IMA was routinely done one day before EVAR. The lumbar artery was embolized if it was remarkable large and considered to be especially prone causing an endoleak. The IMA, IIA or lumbar artery was selectively catheterized with a diagnostic 5F catheter, and generally a 0.018-inch microcatheter was introduced into the artery. Embolization was performed using coils. The coils were delivered just proximal to the first side branch.

Embolization of a type II endoleak

Transarterial embolization or a direct aneurysm sac puncture in association with embolization was used to treat type II endoleaks. The embolization agents were coils, thrombin, ethylene–vinyl alcohol copolymer and glue. Interventional radiologists chose the embolization material according to their preferences.

Transarterial embolization was carried out through percutaneous femoral artery access. Endoleaks emanating from the IMA were accessed through the Riolan's arcade from the superior mesenteric artery (SMA). Lumbar artery endoleaks were catheterized through collateral vessels from the iliolumbar arteries. The aim was to put the catheter into the endoleak cavity and embolize the entire endoleak cavity. If this was not possible, the inflow vessels were embolized as close as possible to the aneurysm sac. The SMA or the internal iliac artery was selectively catheterized with a 4F diagnostic catheter. A 0.018-inch microcatheter was then navigated coaxially to the embolization site. Heparin was not systematically administered.

The direct aneurysm sac punctures were carried out under fluoroscopy and ultrasound guidance or CT and fluoroscopy guidance. If the puncture was done under CT guidance, embolization was carried out at the CT table using fluoroscopy with mobile C-arm equipment. Translumbar puncture patients were placed in a prone position and the puncture was carried out through the back at the level of the endoleak. In one slim patient, the puncture was accomplished transabdominally because the stent graft was located dorsally in the aneurysm sac and there was no access to the endoleak cavity through the translumbar route. After puncture of the aneurysm sac with an 18-gauge needle, a 4F catheter was routinely introduced into the cavity over a 0.035 wire. If the puncture was not in the endoleak cavity, an attempt to reach the cavity was made with the catheter. The exact position was determined by the free and pulsatile return of blood through the catheter. Endoleak angiography and in many cases pressure measurements were done. The endoleak cavity was filled with embolization material. With liquid embolization materials, the tip of the catheter was placed as close as possible to the feeding vessels and embolization was started from that position.

4.3 Statistical methods

Statistical analysis was performed with SPSS statistical software (SPSS v. 12.0.1 and 13.0.1, SPSS Inc., Chicago, Ill., USA).

Continuous variables are reported as the mean \pm standard deviation, the median and the range. Receiver operating characteristic curve analysis was used to assess the association of the GAS and the immediate outcome end-points. The linearized rate of reintervention in study I was calculated as the number of reinterventions per individual follow-up period. The linearized rate of aneurysm

sac shrinkage was calculated as the overall aneurysm size change per individual follow-up period (study III). Univariate analysis was performed using Pearson's test, Fisher's exact test, and the Mann-Whitney test (study III). The Kaplan-Meier method (study I, II and III) and the Cox model (study I) were used to assess survival outcome and the outcome end-points. Logistic regression and Cox regression, both with backward selection, were used for multivariate analysis in study I. Unless otherwise specified, continuous variables were not dichotomized. Only variables with $p < 0.05$ were included in the regression models. P-values less than 0.05 were considered statistically significant.

5 Results

5.1 Mid-term results of using the Zenith[®] stent-graft (I)

5.1.1 Operative details and initial technical success

The mean preoperative aneurysm size was 62 ± 10 mm (median 60 mm, range 38–97 mm). The anaesthetic technique was epidural in 159 (77.2%) patients, general in 46 (22.3%) and local in one (0.5%). The mean duration of the procedure was 3.8 ± 1.1 hours (median 3.5 hours, range 1.8–8.0 hours).

The primary technical success rate at 24 hours was 79.1% and the assisted technical success rate was 86.4%. In ten patients a type I endoleak was suspected during the primary intervention, but was not seen at the first follow-up CT, resulting in technical success rates of 82.0% and 90.3%, respectively, one week after EVAR. Other reasons for technical failures were 12 type I endoleaks, two type III endoleaks, one external iliac artery rupture, one 50% stenosis in the left stent graft limb junction and two access failures.

5.1.2 30-day outcome

The 30-day mortality rate was 2.9%. Two patients died of a myocardial infarction. One patient succumbed to a pulmonary artery perforation and intrathoracic haemorrhage after insertion of a Swan-Ganz catheter. One died of complications after intraoperative bleeding from the iliac artery. One patient developed renal and liver failure as well as bleeding from a gastric ulcer. One patient died because of colon necrosis and peritonitis. Nineteen patients (9.2%) required a postoperative follow-up in the intensive care unit for a mean of 4.1 ± 3.3 days (median 2.0, range 1–13). Thirty-day postoperative morbidity is summarized in Table 6.

Table 6. Thirty-day postoperative morbidity.

Complication	Number of patients (%)
Myocardial infarction	10 (4.9)
Transient ischaemic attack	1 (0.5)
Haemorrhage requiring only blood transfusion	9 (4.4)
Haemorrhage requiring operation	11 (5.3)
Acute renal failure	1 (0.5)
Respiratory failure necessitating intubation	4 (1.9)
Intestinal ischaemia	1 (0.5)
Other minor complications	27 (13.1)

Univariate analysis showed that ongoing angina pectoris ($p = 0.031$), a history of lower limb ischaemia ($p = 0.015$), patient age ($p = 0.017$), low patient weight ($p = 0.011$), preoperative serum creatinine $> 150 \mu\text{mol/l}$ ($p = 0.036$), and the GAS ($p = 0.004$) were predictors of 30-day postoperative mortality. The GAS had an area under the curve of 0.843 ($p = 0.004$), its best cut-off value in predicting 30-day postoperative death was 90. Patients with a GAS $<$ and ≥ 90 had a 30-day postoperative mortality rate of 0.6% (1 out of 177) and 17.9% (5 out of 28) ($p < 0.0001$, O.R. 38.3, 95% C.I. 4.3 - 342.1), respectively.

Logistic multiple regression analysis showed that patient age ($p = 0.013$; O.R. 1.4, 95% C.I. 1.1–1.9), low patient weight ($p = 0.014$; O.R. 0.9, 95% C.I. 0.7–1.0) and lower limb ischaemia ($p = 0.007$; O.R. 196.6, 95% C.I. 4.2–9168.7) were independent predictors of 30-day postoperative mortality. When the GAS was included in the regression analysis as a dichotomous variable (GAS $<$ or ≥ 90) ($p = 0.009$; O.R. 70.4, 95% C.I. 2.8–1751.5), it was also an independent predictor of 30-day postoperative death along with lower limb ischaemia ($p = 0.031$; O.R. 44.0, 95% C.I. 1.4–1380.1) and low patient weight ($p = 0.017$; O.R. 0.8, 95% C.I. 0.71–1.0).

5.1.3 Late survival

The mean follow-up time was 2.4 ± 1.7 years (median 2.4, range 0–6.2 years). The overall survival rates at the 1-, 3- and 5-year follow-ups were 93.3%, 78.7% and 64.5% (S.E. < 0.059 , Fig. 4). The most common causes of death were heart diseases (37.8%) and cancer (27%). No patients died of an abdominal aneurysm rupture. Aneurysm-related death occurred in an 81-year-old man. No growth of his aneurysm was observed at the 6-month control, but at the 10-month control the aneurysm had grown 8 mm. A CT scan was repeated because of abdominal

pain after eight months. Surprisingly, it showed a further aneurysm growth of 10 mm. However, no endoleak was detected either on a CT scan or with angiography. Despite the fact that we did not detect an endoleak or a rupture, the abdominal pain was suspected to be aneurysm-related. Thus, he underwent urgent open repair of the aneurysm. No sign of aneurysm rupture was detected at the operation. He died of multiorgan failure two days after the operation.

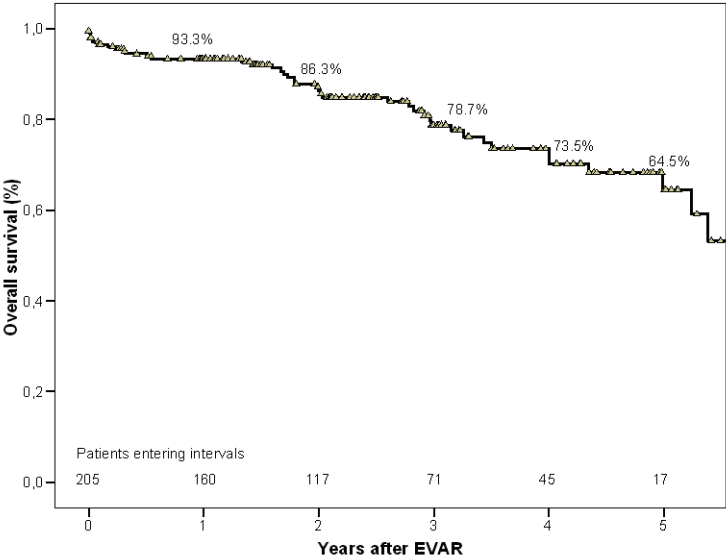


Fig. 4. Kaplan-Meier estimate of all-cause mortality (at 5 years: SE < 0.059).

In the univariate analysis, a history of transient ischaemic attack or stroke ($p = 0.021$), preoperative serum creatinine $> 150 \mu\text{mol/l}$ ($p = 0.045$), lower limb ischaemia ($p = 0.001$), low body mass index ($p = 0.028$) and GAS ($p < 0.0001$) were predictors of long-term all-cause mortality. The Cox regression analysis showed that the GAS ($p = 0.001$, O.R. 1.1, 95% C.I. 1.0–1.1) and the presence of lower limb ischaemia ($p = 0.002$, O.R. 3.9% C.I. 1.7–9.3) were independent predictors of long-term all-cause mortality.

5.1.4 Long-term results

The primary patency rates at the 1-, 3- and 5-year follow-ups were 97.9%, 95.6% and 95.6% (S.E. < 0.017), respectively. At the same intervals, the primary assisted patency rates were 98.4%, 97.1% and 97.1% (S.E. < 0.013) and the secondary patency rates were 100%, 99.3%, and 99.3% (S.E. < 0.007), respectively. The primary clinical success rates at the 1-, 3- and 5-year follow-ups were 90.6%, 85.6%, and 83.5% (S.E. < 0.035, Fig. 5).

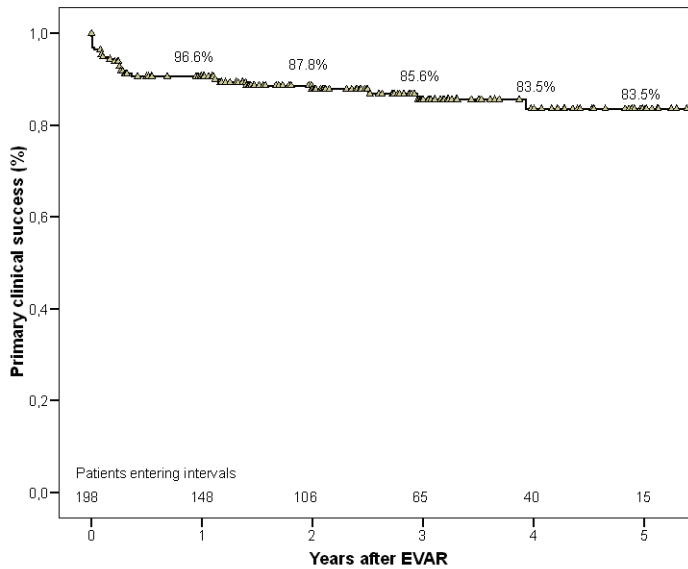


Fig. 5. Kaplan-Meier estimate of primary clinical success (at 5 years: SE < 0.035).

5.1.5 Endoleaks

Overall endoleak incidence was 34.6%. Type I endoleaks were observed in 26 patients (12.6%), 22 of them during the primary procedure. Ten endoleaks disappeared before the first surveillance imaging (within 7 days). Two endoleaks that were not detected at the initial procedure were seen at the first imaging within 7 days. Thus, at one week there were 14 type I endoleaks (7.8%). Two patients had type I endoleaks that were secondary, detected 7 months and 28 months after

EVAR. Six patients were treated with either an endovascular intervention or conversion. Altogether nineteen patients' endoleak disappeared spontaneously. One patient with a type I endoleak was treated conservatively and he died of a myocardial infarction on the third postoperative day. A type III endoleak was observed in two patients (1.0%). Survival with freedom from type I and III endoleaks was 95.3% at 1, 3 and 5 years (S.E. < 0.015).

A type II endoleak occurred in 52 patients (25.4%). There were 27 primary (13.2 %) and 25 secondary (12.2%) type II endoleaks. Of the type II endoleaks, 51.9% were detected during the first week. The rate of appearance of a secondary type II endoleak at six months, one, two and beyond two years was 15.4%, 13.5%, 11.5% and 7.7%, respectively. Altogether 51.9% of the type II endoleaks resolved spontaneously. Thirteen type II endoleaks were treated with either catheter embolization or direct puncture embolization. In six cases (3.0%) a type II endoleak recurred after a period of resolution. The aneurysm sac grew in ten patients (10/52, 19.2%).

We did not observe any type IV endoleaks in this series. There were 4 patients (2.0%) with an aneurysm enlargement without any evident endoleak. One of these patients had Alzheimer's disease and surveillance was ended. He died of a myocardial infarction 4 years and 4 months after EVAR. Three other patients were treated conservatively.

During the follow-up period, the aneurysm diameter decreased ≥ 5 mm in 53.2% of the patients, remained unchanged in 31.2%, and increased ≥ 5 mm in 7.8%. This information was missing for 16 patients (7.8%) because of early death or a missing control.

5.1.6 Secondary interventions

Neither stent-graft fractures nor migration were detected in this series. Five patients (2.5%) had graft limb thrombosis. There were altogether six secondary conversions (2.9%). Five of them were elective. The reasons for these elective conversions were access failure, stent-graft infection, stent-graft main body folding and two type I endoleaks. One patient had an emergency conversion because of abdominal pain.

Overall, twenty-seven patients (13.1%) had a secondary intervention during the study period. Twenty-three patients had one reintervention, three patients had two and one had three reinterventions. The secondary interventions are summarized in Table 7. The second and third interventions were all transarterial

embolization of aortic side branches. Survival from any vascular reintervention at 1, 3 and 5 years was 89.7%, 83.2% and 83.2% (S.E. < 0.031), respectively.

Table 7. Secondary interventions in 27 patients.

Secondary Interventions	n (%)
Conversion	6 (22.2)
Transarterial embolization of an aortic side branch (IMA or lumbar artery)	6 (22.2)
Iliac internal artery embolization with or without stenting	4 (14.8)
Thrombolysis with or without percutaneous transluminal angioplasty/stenting.	3 (11.1)
Stenting	3 (11.1)
Translumbar embolization	3 (11.1)
Left common iliac artery ligation	1 (3.7)
Femoral-femoral crossover bypass surgery.	1 (3.7)

5.2 Symptomatic, unruptured AAAs (II)

5.2.1 Initial success

The primary technical success rate was 86% (12 out of 14 patients). The assisted technical success rate was 93% (13 out of 14 patients). Stent-grafting failed in one patient because of excessive kinking and atherosclerotic lesions of both external iliac arteries. The mean operative time in these 13 patients was 234±74 min (median 220 min, range 123–390 min). Blood loss was recorded in ten patients, median blood loss was 475 ml, range 100–1800 ml.

5.2.2 Complications and long-term results

None of these patients died during the immediate postoperative period. There was no need for blood transfusion or reoperation because of bleeding. One patient had chest pain after the procedure, and because of that he was admitted to the intensive care unit for one day. He did not develop a myocardial infarction. Another patient had symptoms related to intestinal ischaemia after the procedure. A third patient developed a dissection of the right common femoral artery. No other major complication occurred in this series.

The mean follow-up time was 1.9 ± 1.4 years. The 2-year survival rate was 69%. One patient died of rupture of a dissecting thoracic aortic aneurysm, one of cancer, one of cardiac heart failure and yet another after trauma. There were no abdominal aneurysm related deaths. The survival freedom from a secondary procedure was 71%. One patient underwent stent-grafting for a distal type I endoleak five months after the procedure. Another patient underwent femoro-femoral cross-over bypass surgery because of a right limb graft thrombosis which occurred nine months after the procedure. At the same time he had percutaneous transluminal angioplasty and stenting for a stenosis of the left iliac artery.

A type II endoleak was detected in three patients at the first CT control within 4 days. In two of them the endoleak disappeared 2 and 17 months after the procedure, and in the third patient the aneurysm did not grow, so there was no indication for further treatment. Additionally, one patient developed a type II endoleak 3 months after the procedure and, because the AAA is slowly growing, he has been scheduled for a secondary procedure.

5.3 IMA embolization before EVAR (III)

There were significantly more type II endoleaks in the non-IMA embolization group (23/39 vs. 10/40, 59% vs. 25%; $p = 0.002$). There were 63.5% primary, 21.2% secondary, and 15.2% recurrent type II endoleaks. Preoperative IMA embolization did not correlate with the classification of type II endoleaks.

There were significantly more secondary interventions in the non-IMA embolization group (11/38 vs. 1/40, 28% vs. 2.5%; $p = 0.001$). A majority of the secondary interventions were transarterial embolizations of the aortic side branches.

At the last follow-up, 35 (47.3%) of the AAAs showed no change in diameter, 7 (9.5%) grew ≥ 5 mm, and shrinkage ≥ 5 mm was detected in 32 (43.2%). Preoperative IMA embolization did not correlate with aneurysm size changes. The overall linearized aneurysm shrinkage rate/year was 1.4 ± 3.8 mm/year among preoperatively embolized IMA patients versus 1.7 ± 2.4 mm/year among non-IMA embolized patients ($p = 0.72$).

No aneurysm ruptures, stent-graft migrations, or aneurysm-related deaths occurred during the study period. There were no complications related to preoperative IMA embolization.

5.4 Treatment of a type II endoleak (IV)

5.4.1 Conservatively treated endoleaks

Thirty-three type II endoleaks (70.2%) were treated conservatively. During the follow-up, the aneurysm diameter decreased by at least 5 mm in 13 out of 32 patients (41%) and remained unchanged in 15 patients (47%). The aneurysm diameter increased in four patients (13%). During the follow-up, one of them had an enlarging aneurysm and a late endoleak that vanished spontaneously. The aneurysm eventually became painful and necessitated an emergency open repair. Yet, neither the endoleak nor the rupture was identified. This patient died postoperatively of multiorgan failure. This was considered as an aneurysm-related death. Three patients were not considered for intervention because one patient had no further growth of the aneurysm after an initial increase of 5 mm and two had fatal comorbidities. None of the patients died of an abdominal aneurysm rupture.

5.4.2 Embolized endoleaks

Fourteen patients underwent secondary intervention for a type II endoleak. Details of these interventions are shown in Table 8. No procedure-related complications were observed.

Table 8. Results of secondary interventions for type-II endoleaks in 14 patients.

Patient	1 st secondary intervention	TS	CS	2 nd secondary intervention	TS	CS	3 rd secondary intervention	TS	CS
1	Transarterial (ethylene–vinyl alcohol copolymer)	Yes	NA						NA
2	Transarterial (thrombin, coils)	Yes	No						Yes
3	Transarterial (coils)	Yes	Yes						Yes
4	Transarterial (coils)	No	No						No
5	Transarterial (coils)	No	No						Yes
6	Transarterial (coils)	No	Yes						Yes
7	Transarterial	No	No						No

Patient	1 st secondary intervention	TS	CS	2 nd secondary intervention	TS	CS	3 rd secondary intervention	TS	CS
8	Transarterial (stent-graft extension, coils)	No	No	Transarterial (coils) 81 mo after 1 st secondary intervention	No	Yes			Yes
9	Transarterial (coils)	No	No	Transarterial (coils) 18 mo after 1 st secondary intervention	No	No	Transarterial (coils) 5 mo after 2 nd secondary intervention	No	Yes
10	Transarterial (glue)	Yes	No	Transarterial (glue) 13 mo after 1 st secondary intervention	Yes	No	Transarterial (attempt) 32 mo after 2 nd secondary intervention	No	No
11	Translumbar (coils, gelatin)	Yes	Yes						Yes
12	Translumbar (ethylene–vinyl alcohol copolymer)	Yes	Yes						Yes
13*	Translumbar (coils, gelatin)	Yes	Yes	Transarterial (coils) 40 mo after 1 st secondary intervention	Yes	NA			NA
14	Transabdominal puncture (ethylene–vinyl alcohol copolymer)	Yes	No						No

TS = technical success, CS = clinical success, NA = not assessed/no surveillance imaging after intervention, mo = months, * In patient 13 a type II endoleak reappeared 4 years after initial disappearance, and the patient had a second secondary intervention

One patient without surveillance imaging after the secondary intervention was excluded from further analyses. Clinical success was achieved in only five patients (5/13) after the first intervention. Out of the eight patients with a type II endoleak after the first intervention, the endoleaks of two patients disappeared spontaneously two years later. After additional interventions in four patients,

overall clinical success was achieved in eight patients (8/12) at the end of the study period. One patient did not have surveillance imaging after the second secondary intervention; consequently, the data of this patient are missing. Four patients still had a type II endoleak. The aneurysm sac did not grow in these four patients with a permanent type II endoleak.

Ten out of the fourteen patients with a secondary intervention underwent transarterial embolization. After the first intervention, technical success was registered in four of them (4/10) and clinical success in two patients (2/9). Surveillance imaging was missing for one patient. After an additional transarterial intervention in three patients, clinical success was registered in six patients (6/9). It is worth noting that two patients still had a type II endoleak after the first secondary intervention, which disappeared spontaneously by the 2-year follow-up.

Four out of the fourteen patients had a direct puncture of the aneurysm sac and embolization through either the translumbar (3/4) or transabdominal (1/4) route. All four interventions were technically successful, and clinical success was achieved in three patients. In one of these patients with clinical success, a type II endoleak reappeared after four years of resolution, and he underwent a second secondary intervention by transarterial embolization because the diameter of the aneurysm increased. He did not have surveillance imaging after the last intervention.

6 Discussion

EVAR is increasingly used to treat AAAs with encouraging results. It has been shown to be associated with an immediate outcome that is superior to open repair. The purpose of the current study was to assess the short- and mid-term results of EVAR with the Zenith[®] stent-graft in asymptomatic and symptomatic AAA patients and to investigate whether preoperative IMA embolization has an effect on type II endoleak rates and sac shrinkage, and also to evaluate the results of treating type II endoleaks

In the present study, the Zenith[®] stent-graft was shown to be effective in excluding AAAs from the circulation and to be associated with good short- and mid-term results in both asymptomatic and symptomatic patients. Preoperative coil embolization of the IMA reduced the incidence of type II endoleaks after EVAR, but it did not show any effect on late postoperative aneurysm shrinkage. Finally, the overall results of secondary interventions of type II endoleaks were quite poor, but these preliminary results favour direct translumbar embolization rather than transarterial embolization.

6.1 Short- and mid-term results of EVAR with the Zenith[®] stent-graft

In the present study, the 30-day postoperative mortality rate was 2.9% and the late aneurysm-related death rate was 0.5%. These rates along with the migration (0%), thrombosis (2.5%) and secondary intervention rates (13.1%) are in concordance with previous studies dealing with the Zenith[®] stent-graft (Table 9) (Greenberg *et al.* 2001, Abraham *et al.* 2002, Alric *et al.* 2002, Hinchliffe *et al.* 2004, Greenberg *et al.* 2004b, Lalka *et al.* 2005, Hiramoto *et al.* 2007, Bos *et al.* 2008, Greenberg *et al.* 2008, Becquemin *et al.* 2008, D'Elia *et al.* 2009). A characteristic feature of the Zenith[®] stent-graft is that its suprarenal bare metal fixation with barbs is reflected by a very low migration rate. In previous studies two different thresholds for migration have been used; ≥ 5 mm and ≥ 10 mm. Reporting standards for EVAR recommend a 10-mm threshold (Chaikof *et al.* 2002). In the present study we used a 5-mm threshold and still we did not observe any migration. This was the only case where we did not follow the reporting standards.

The 5-year freedom rate from a secondary intervention was 83.2%. Open repair of AAA is associated with freedom rates from graft-related reintervention between 94% and 98% at 5 years and between 88% and 94% at 10 years

(Biancari *et al.* 2002b, Conrad *et al.* 2007). Despite being lower than the freedom rates observed after open repair, the need for a secondary intervention in this high-risk population is acceptable. However, analysis of the need for a secondary intervention is complicated by the aggressive approach toward type II endoleaks at the beginning of the study period. Nowadays, in light of current knowledge, we treat them only in case of an increase in aneurysm size (van Marrewijk *et al.* 2002, Steinmetz *et al.* 2004, Tolia *et al.* 2005). Thus, it is likely that nowadays these secondary intervention rates would be smaller. Indeed, if we count only imperative secondary interventions done because of type I and III endoleaks, migration, graft kinking and graft occlusion, the secondary intervention rate would be closer to open repair rates. Hiramoto reported such a secondary intervention rate of 2.8 % (Hiramoto *et al.* 2007), and in our study it would be 7.3%.

Table 9. Our results compared with other Zenith® stent-graft studies.

	30-day mortality (%)	Stent-graft migration (%)	Stent-graft thrombosis (%)	AAA-related late death (%)	Late aneurysm rupture (%)	Secondary intervention (%)
Present study	2.9	0	2.5	0.5	0	13.1
D'Elia 2009	1.8	0	2.8	ND	ND	11.2
Becquemin 2008	0.9	0	3.3	0.9	0.9	14.6
Bos 2008	1.7	ND	1.7	0 *	0.4	9.2
Greenberg 2008	ND	0.27	2.6	ND	0.1	20
Hiramoto 2007	ND	ND	0.6	0.9	0.3	8.6
Lalka 2005	0	ND	ND	ND	ND	12.5
Greenberg 2004	0.5	2	ND	0.5	0.3	11
Hinchliffe 2003	4.1	0.4	ND	ND	0.7	7.5
Alric 2002	3.4	ND	3.4	3.4	2.3	6.8
Abraham 2002	0.9	0	0.9	0.9	0.9	6.9
Greenberg 2001**	ND	ND	1.7	0.2	0.2	ND

ND = no data available, * 1.7 % unknown cause of death, ** 297/528 (57%) had follow-up information

The mean GAS of 80 is in accordance with the EUROSTAR series (mean GAS 79) (Biancari *et al.* 2006), but markedly higher than that of patients (mean GAS 73) included in the Finnvasc registry who underwent open repair mostly in the pre-EVAR era (Biancari *et al.* 2003). Thus, most of the patients in the present study had a significantly increased operative risk and therefore were not ideal candidates for major open surgery. Against this background, the 30-day mortality rate of 2.9% is a very good result. This clear advantage over open repair in terms of immediate outcome does not make any comparison with the results of the latter feasible, as nowadays patients at high risk are often offered EVAR treatment as the only alternative to conservative treatment.

This occurs despite the results of EVAR trial 2. EVAR trial 2 compared patients unfit for open surgery by randomised them into an EVAR group and a best medical therapy group. There were no differences in four-year aneurysm related mortality or all-cause mortality. (Greenhalgh *et al.* 2005a) This trial had many problems; it was planned on an intent-to-treat basis and 20% of the patients crossed over between groups. Also, 19% of deaths in the EVAR group occurred in patients who never underwent EVAR. The other problem was that although a recommended guideline was given, the “unfit” definition was based on a subjective determination by the clinician who initially evaluated the patient (Brown *et al.* 2004). The controversy over this matter is far from being settled. Anyhow, there is a lower level evidence suggesting the benefit of EVAR over open surgery in a high-risk population (Sicard *et al.* 2006, Bush *et al.* 2007).

A significant finding of the present study was that there was only one aneurysm-related death, while no patients died of a late aneurysm rupture. However, we observed a rather large number of type I endoleaks (12.6%, 26/206), whereas other authors reported an incidence of this event in 0 to 3.4% of patients with the Zenith[®] stent-graft (Abraham *et al.* 2002, Alric *et al.* 2002, Hincliffe *et al.* 2004, Hiramoto *et al.* 2007). Our result of 12.6% is the figure from the initial procedure (within 24 hours). Ten of these endoleaks disappeared by the first imaging control (within 7 days), thus our type I endoleak rate at one week was 7.8%. When 27 world-known physicians with extensive experience with EVAR, who had studied and published articles on endoleaks, were inquired about their experience with the total percentage of type I endoleaks at their institution (after 24 hours), the answers ranged from 0% to 30%, with a mean of 7.5% (Veith *et al.* 2002). This is in accordance with our results. Additionally 9 type I endoleaks disappeared spontaneously later on. The overall rate of spontaneously disappearing type I endoleaks was 73% and there were 7 (2.9%) permanent type I

endoleaks. Six of them were treated with a secondary intervention. These findings suggest that a type I endoleak might be a rather “benign” condition after EVAR using the Zenith[®] stent graft, as very often it disappears spontaneously shortly after the procedure or within one year. Some patients had also a type II endoleak, thus it is possible that some of the type II endoleaks were incorrectly diagnosed as type I endoleaks. It is also worth noting that all the interventions in this study were guided by modern angiographic equipment with large image intensifiers, providing excellent quality of angiography which revealed even minor leaks. One may also argue that minor leakages due to graft porosity immediately after the delivery were erroneously interpreted as type I endoleaks during the intervention. We did not have data about the infrarenal aortic neck anatomy, and thus we were unable to assess the impact of neck anatomy in a rather large number of type I endoleaks. Common guidelines for establishing patient suitability for EVAR were followed and we believe that in this series there were no outstandingly challenging neck anatomies.

The assisted technical success rate (90%) assessed at the time of the first CT follow-up, within 1 week after the intervention, is slightly lower than the figures (94%-100%) reported in previous studies dealing with this stent graft (Greenberg *et al.* 2001, Abraham *et al.* 2002, Hinchliffe *et al.* 2004, Greenberg *et al.* 2004b). Still, our results are acceptable and may reflect differences in the study populations in various studies.

The 5-year survival rate of previously reported patients who underwent EVAR with a Zenith[®] stent graft ranged from 50% to 70% (Hinchliffe *et al.* 2004, Hiramoto *et al.* 2007, Bos *et al.* 2008), and in our series it was as high as 64.5%. Renal dysfunction is the important predictor of poor survival after both open and endovascular repair of an AAA. Preoperative renal insufficiency has been reported to be a risk factor for postoperative renal failure (Miller & Myers 1987). With EVAR, renal dysfunction can be worsened by contrast medium, wire manipulation and atheroembolism. The uncovered suprarenal stents of the Zenith[®] stent-graft traverse the renal ostia and there is concern whether this can predispose to renal dysfunction. However, there is no difference in renal dysfunction rates between EVAR (suprarenal fixation) and open repair patients (Greenberg *et al.* 2004a), and the stent position does not have a significant effect on renal function (O'Donnell *et al.* 2007). In the present study only one patient (0.5%) had postoperative acute renal failure.

6.1.1 Symptomatic patients

The reported mortality rates after open repair of a symptomatic AAA range from 9% to 18% (Haug *et al.* 2004, Tambyraja *et al.* 2004, Leo *et al.* 2005). In the present study there were no deaths during the 30-day period. This study confirmed the preliminary good results of other centers (30-day mortality 0–5%) (Franks *et al.* 2006, Oranen *et al.* 2006) and importantly showed that this treatment method is feasible in very-high-risk patients as herein shown by the markedly increased GAS (85). In fact, in two previous studies dealing with patients who underwent open repair of a symptomatic, unruptured AAA, the median GAS was about 77 (Franks *et al.* 2006, Antonello *et al.* 2007), whereas patients undergoing elective open repair have a median score of about 73 (Biancari *et al.* 2003). Despite such an increased operative risk, the immediate and late results of endovascular treatment with the Zenith[®] stent-grafting system are very encouraging.

6.2 Type II endoleak

We observed 25% type II endoleaks in 206 patients with an asymptomatic AAA. This is slightly more than in previous studies (8–23%) (Alric *et al.* 2002, Hinchliffe *et al.* 2004, Greenberg *et al.* 2004b, Hiramoto *et al.* 2007) and may reflect various imaging protocols in individual studies. The spontaneously resolving type II endoleak rate was 51.9% which is in concordance with previous reported rates between 50–80% (Silverberg *et al.* 2006, Jones *et al.* 2007, Higashiura *et al.* 2007).

6.2.1 Prevention of type II endoleaks

The clinical significance and treatment of patent IMA-related type II endoleaks is controversial. In this controversial scenario, our results clearly showed that preoperative coil embolization of the IMA significantly reduced the incidence of type II endoleaks. However, its clinical benefits are still unknown. In fact, the effect of preoperative IMA embolization on aneurysm sac size change is unclear. A few reports have shown that IMA embolization is beneficial in terms of AAA shrinkage (Axelrod *et al.* 2004, Sheehan *et al.* 2006), but the present study failed to confirm these previous observations. We did not observe aneurysm growth in any of the non-IMA embolization patients who underwent a secondary

intervention. This is related to a previous aggressive policy of treating type II endoleaks at OUH; embolization was performed in any patient who had a persistent type II endoleak at the 3-month follow-up. Despite the fact that secondary interventions were one of the three main outcome endpoints of this study (III), our retrospective analysis does not warrant any conclusions about the efficacy of pre-EVAR IMA embolization in decreasing the need for interventions due to type II endoleaks as the policy of treating them has changed significantly during the study period. Our result that there was no difference in aneurysm growth between the study groups implies that routine IMA embolization is not worthwhile. However, we cannot discount the possibility that the significantly greater number of secondary interventions performed in the non-IMA embolization group had an impact on the aneurysm sac growth rate which masked the possible actual effect of preoperative IMA embolization on aneurysm shrinkage.

The problem is that to achieve complete success, every side branch should be embolized. Peroperatively delivered liquid material has been shown to be a feasible method for reducing the rate of type II endoleaks (Walker *et al.* 1999, Muthu *et al.* 2007, Zanchetta *et al.* 2007). It is theoretically possible that liquid material can cause paralysis by occluding the lumbosacral plexus or anterior spinal artery (Gorich *et al.* 2000, Haulon *et al.* 2001) Although these pre- and perioperative embolization techniques appear attractive, most patients with patent side branches will not develop a type II endoleak. Thus, routine embolization before EVAR exposes many patients to unnecessary procedure-related risks.

There were no complications related to preoperative IMA embolization in the present study. Theoretically, there is a risk of creating colonic ischaemia if embolization material is not placed proximal to the first side branch (Bush *et al.* 2001). It is suggested that chronic anticoagulation may interfere with spontaneous sealing of a type II endoleak (Chuter *et al.* 2001a, Fairman *et al.* 2002). Because of the retrospective design of this study, exact data about anticoagulation treatment during and after the procedures were not available.

6.2.2 Treatment of type II endoleaks

The understanding and management of type II endoleaks have evolved over time. More and more conservative approaches have been adopted, and it is even suggested that type II endoleaks should be treated only if there is an increase in sac size of more than 5 mm over a 6-month period or an overall increase of 10mm

(Rayt *et al.* 2009). The rupture rate from a type II endoleak appears to be low. Data from EUROSTAR suggest an incidence of rupture after a type II endoleak of 0.5% (van Marrewijk *et al.* 2002). However, because a persistent type II endoleak has been shown to be associated with aneurysm sac growth and rupture (Jones *et al.* 2007), it is widely accepted to treat type II endoleaks with significant sac growth. There is ongoing debate about this matter.

Previously reported results of secondary interventions, especially transarterial embolization, are variable. Some studies have reported very poor results (Chuter *et al.* 2001a, Baum *et al.* 2002, Solis *et al.* 2002) while other studies have reported satisfying results of transarterial embolization (Gorich *et al.* 2000, Haulon *et al.* 2001, Kasirajan *et al.* 2003, Faries *et al.* 2003, Sheehan *et al.* 2004, Stavropoulos *et al.* 2009). In this controversial scenario our clinical success rate of 20% after the first transarterial embolization suggests that transarterial embolization is not an adequate treatment method. A type II endoleak behaves similarly to an arterial malformation, which is the reason for single-vessel arterial embolization often being ineffective. It is essential to eliminate the entire endoleak cavity, the nidus. Embolization of a single feeding artery will only divert flow to the neighbouring vessels and enlarge them. With transarterial embolization it could be very challenging to occlude the whole nidus. Stavropoulos showed in a recent study that good results are achievable with modified transarterial embolization (embolization of the whole nidus in addition to the feeding artery) (Stavropoulos *et al.* 2009). We were able to embolize the nidus in only four patients, and two of them had clinical failure nevertheless. This could be one of the reasons for the poor outcome after transarterial embolization observed herein.

Although the numbers of patients considered herein are too small to allow reliable comparisons between groups, the present results seem to favour direct translumbar embolization rather than transarterial embolization. It is possible that through translumbar embolization, access to the nidus could be achieved more easily and could facilitate the occlusion of communication between side branches. However, it could be that modified transarterial embolization is a good alternative when the feeding artery and the whole nidus can be embolized. Thus, it seems that careful preoperative imaging is essential in order to determine whether the endoleak is accessible with the translumbar approach or with the transarterial route.

6.3 Follow-up after EVAR

Because EVAR is associated with late complications that could appear after several years (Vallabhaneni & Harris 2001), lifelong surveillance of these patients is indicated. In the present study (I), the rate of appearance of a secondary type II endoleak at six months, one, two and beyond two years was 15.4%, 13.5%, 11.5% and 7.7%, respectively. During recent years the follow-up policy has changed toward longer imaging intervals, and non-contrast CT and US with or without contrast enhancement are also used alongside contrast-enhanced CT (Hiramoto *et al.* 2007, Chaer *et al.* 2009, Iezzi *et al.* 2009). Recent recommendations for Zenith[®] stent-grafts suggest that every EVAR patient should have contrast-enhanced CT and plain x-rays at 30 days. If there is no endoleak and good component and iliac overlap is seen, the next contrast-enhanced CT is indicated at 1 year, and after that yearly abdominal US should be performed. If there is an endoleak or under one stent component or iliac overlap, the patient should have contrast-enhanced CT every six to twelve months. (Sternbergh III *et al.* 2008) Overall, imaging modalities and specific protocols vary widely among different institutions, depending on their experience and expertise with different imaging modalities.

After secondary interventions with coils and ethylene–vinyl alcohol copolymer there is a problem with artefacts on CT imaging. When an endoleak cavity is successfully embolized, it can be difficult to detect small endoleaks in surveillance imaging. In these cases, it is essential to evaluate the size of the aneurysm sac.

None of the currently used imaging modalities mentioned above can measure aneurysm sac pressure. A study employing direct sac pressure measurements after EVAR indicated that high sac pressure was associated with AAA expansion while low sac pressure was associated with shrinkage (Dias *et al.* 2004). These measurements were done by percutaneous translumbar puncture, thus they were invasive procedures. During recent years there has been a growing interest toward wireless intrasac pressure measurements. A wireless sensor is implanted in the aneurysm sac at the time of stent-graft insertion. Pressure data can be obtained repeatedly and noninvasively during follow-ups. This technique is reported to be feasible, safe and efficient (Ellozy *et al.* 2004, Ohki *et al.* 2007, Hoppe *et al.* 2008).

6.4 Limitations of the present study

The retrospective nature is a major limitation of the present study. Some patients were lost during the follow-up. However, the deaths and causes of death were obtained from Statistics Finland, so we have complete data on late deaths. Another limitation is that there were no control groups (apart from study III). In study III the significantly greater number of secondary interventions performed in the non-IMA embolization group could have had an impact on the aneurysm sac growth rate and could have masked the possible actual effect of preoperative IMA embolization on aneurysm shrinkage. Another limitation is that data on the financial burden of preoperative embolization of the IMA were missing, so we could not make any conclusions about cost effectiveness.

The number of patients in studies II and IV was small. In study II there were only 14 symptomatic AAA patients and furthermore one can not be sure that there was no selection bias. Patients treated with EVAR could have been more stable than average patients going to open surgery. In study IV the number of patients was too small to allow reliable comparisons between different treatment methods in the case of type II endoleaks.

6.5 Ruptured AAAs

Operative mortality after open surgery for RAAAs is very high, 41–54% (Kantonen *et al.* 1999, Bown *et al.* 2002, Giles *et al.* 2009). The first published experience of endovascular repair of a RAAA is from 1994 (Yusuf *et al.* 1994). Since then several centers have employed EVAR for RAAA. Recently published data have shown that the 30-day mortality rate is approximately 21–33% (McPhee *et al.* 2009, Veith *et al.* 2009, Giles *et al.* 2009), thus remarkable lower than in open repair. Anyhow, the results of the studies may have been biased by patient selection, as EVAR is often used for “stable” RAAA patients and an unstable patient tends to go straight to the operating room for open surgery. Furthermore, more anatomically difficult patients are subjected to open surgery. A nonrandomized New ERA study concluded that approximately half of RAAA patients are suitable for EVAR, and an unsuitable infrarenal neck was the most frequent reason for selecting open surgery (Peppelenbosch *et al.* 2006). There are no concluded randomized prospective controlled comparisons of EVAR and open repair in RAAA patients. One randomized controlled trial was halted after a pilot study, and the preliminary results showed no difference in mortality rates at 30-

days (Hinchliffe *et al.* 2006). Three randomized controlled trials are underway (Hoomweg & Balm 2006, Powell 2009, Desgranges *et al.* 2010).

The ability to perform EVAR under local anaesthesia with maintenance of abdominal wall smooth muscle tone may promote retroperitoneal tamponade of bleeding and improve patient outcomes. However, abdominal compartment syndrome is a potential complication after EVAR and it has been seen in 20% of patients (Mehta *et al.* 2005, Mayer *et al.* 2009). Intra-abdominal hypertension leads to respiratory, cardiac and renal dysfunction and successful treatment depends on early diagnosis and surgical decompression.

The natural history of type II endoleaks after EVAR of RAAA is unknown. A ruptured aneurysm sac behaves differently than an intact sac and there are few reported cases of ongoing bleeding through the lumbar arteries or IMA (Veith *et al.* 2009). However, it is speculated that periaortal haematoma could compress the lumbar arteries and cause thrombosis and thus prevent a type II endoleak from developing, at least in the early postoperative period (Orend *et al.* 2002, Scharrer-Pamler *et al.* 2003).

Logistic and practical barriers in this matter have also to be perceived; whether the patient is stable enough to undergo to the anatomic imaging before EVAR and whether the hospital has the appropriate services available 24 hours a day .

6.6 Future prospects

Decreased periprocedural morbidity and mortality and encouraging mid-term results of EVAR have opened the door to its use in patients with a more complex anatomy, such as juxtarenal or thoracoabdominal aortic aneurysms. A fenestrated stent-graft was first described in 1996 (Park *et al.* 1996). Fenestrated stent-grafts have holes in the fabric that can be positioned adjacent to the aortic side branch orifices. These fenestrations can be fixed in position with covered stents through fenestration to the side branches. With these fenestrated stent-grafts it is now possible to circumvent the limitations of short or missing aortic necks. Promising intermediate-term results of fenestrated stent-grafts have been published (Muhs *et al.* 2006, Semmens *et al.* 2006, O'Neill *et al.* 2006, Ziegler *et al.* 2007), although mid- and long-term results of device performance, target vessel patency and adverse renal events are still forthcoming. Branched stent-grafts differ from fenestrated stent-grafts by having true pre-attached branches that can be extended into the visceral and renal arteries using covered stents. The first multibranched stent-graft

was implanted in 2000 (Chuter *et al.* 2001b), and the first short-term results are encouraging (Chuter *et al.* 2008).

During the past decade, the role of screening for AAAs has been investigated and debated. Screening programs with US have been advocated, because AAAs are usually asymptomatic. Screening has been shown to be associated with a significant reduction in mortality from an AAA in men aged 65 or more (Ashton *et al.* 2002, Cosford & Leng 2007, Thompson *et al.* 2009). The largest randomized trial of screening for abdominal aortic aneurysms, the Multicentre Aneurysm Screening Study (MASS) concluded that their result concerning cost-effectiveness was at the margin of acceptability (Thompson *et al.* 2009). Anyhow, a recent study from Denmark concluded that screening is not cost-effective and that further research on this issue is needed (Ehlers *et al.* 2009).

7 Summary and conclusions

1. The Zenith[®] stent-graft is associated with good short- and mid-term results. The thirty-day mortality rate is 2.9%. Noteworthy is that there was only one late aneurysm-related death, while no patients died of a late aneurysm rupture. There were no migrations or stent-graft fractures.
2. EVAR of a symptomatic, unruptured AAA is associated with a favourable outcome even in patients with a very high operative risk. There were no deaths during the 30-day.
3. Preoperative coil embolization of the IMA significantly reduces the incidence of type II endoleaks after EVAR, but the present study failed to show any influence on late postoperative aneurysm shrinkage.
4. The results of transarterial embolization of type II endoleaks are poor. The results appear to favour direct translumbar embolization rather than transarterial embolization.

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Appendix 1 Data collection form

Outcome after endovascular treatment of an AAA with the Zenith stent-graft A Finnish multicenter study

Patient number _____

Center: Helsinki Kuopio Oulu

Date of operation __/__/__

Known preoperative variables (N=no or no data)

Age _____		Gender M F	
Weight _____ kg		Serum creatinine _____	
Length _____ cm		Haemo-/peritoneal dialysis	Y N
Symptomatic, non-ruptured AAA	Y N	C-reactive protein _____	
Non-insulin dependent diabetes	Y N	Lower limb ischaemia	Y N
Insulin dependent diabetes	Y N	Hyperlipidemia	Y N
Current smoking	Y N	Atrial fibrillation	Y N
Ex-smoker	Y N	Congestive heart failure	Y N
< 1 year _____			
> 1 year _____			
TIA	Y N	Hypertension	Y N
Stroke	Y N	COPD/asthma	Y N
Ongoing angina pectoris	Y N	Aortic valve disease	Y N
History of myocardial infarction	Y N	Mitral valve disease	Y N
Prior percutaneous coronary intervention	Y N	Prior aortic valve surgery	Y N
Prior coronary artery bypass surgery	Y N	Prior mitral valve surgery	Y N
Prior peripheral vascular interventions	Y N		

Preoperative diagnostic variables

CT angiography	Y N	Catheter angiography	Y N
MR angiography	Y N		
Maximum aneurysm diameter _____ mm			

Operative variables (primary operation)

Preoperative embolization of the IMA	Y N	Preoperative embolization of the internal iliac artery	Bil. R L
General/regional/local anaesthesia	G/R/L	Blood loss ____ mL	
Graft bifurcated __		Size of the graft	
uni-iliac__		body_____	
tubular__		right limb_____	
		left limb_____	
Operation time ____ min		Fluoroscopy time ____ min	
Concentration of contrast medium_____		Amount of contrast medium ____ mL	
Primary technical success	Y N	Assisted primary technical success	Y N

Primary conversion to open repair	Y N	Access failure	Y N
	__/__/__	Insertion failure	Y N

Cause of conversion _____

30-day postoperative mortality and morbidity

Postoperative death	Y N	Max. postop. serum creatinine ____	
Date of death	__/__/__	Need of haemo-/peritoneal dialysis	Y N
Cause of death _____		Max. C-reactive protein ____	
Stroke	Y N	Atrial fibrillation	Y N
TIA	Y N	Respiratory failure	Y N
Myocardial infarction	Y N	Pneumonia	Y N
Bleeding	Y N	Bleeding requiring reoperation	Y N
Aortic rupture	Y N	Intestinal ischaemia	Y N
ICU/highdependency unit stay	Y N days_____		
Other complications _____			

Follow-up

	modality	aneurysm diameter (mm)	endoleak type I	II	III	IV	V	undetermined	x-ray	other
preoper.. / /	----		----	--	---	--	--	-----	----	
perioper.. / /	----	----							----	
postoper.. / /		----							----	
3-month / /										
6-month / /										
1-year / /										
2-year / /										
3-year / /										
4-year / /										
5-year / /										
6-year / /										
/ /										
/ /										

1st reintervention: _____ / / /

_____ / / /

2nd reintervention: _____ / / /

_____ / / /

3rd reintervention: _____ / / /

_____ / / /

4th reintervention: _____ / / /

_____ / / /

5th reintervention: _____ / / /

_____ / / /

Secondary conversion

Elective: _____ / /

Urgent: _____ / /

Rupture: _____ / /

- hyperlipidemia = on medication
- prior peripheral vascular interventions = surgery or/and endovascular procedures
- aneurysm size = maximum diameter from axial CT/MR image
- respiratory failure = extubation >1 day
- intestinal ischaemia – proven by endoscopy or imaging
- bleeding – needing blood transfusion
- graft kinking – needing secondary intervention

Original Publications

This thesis is based on the following articles, which are referred to in the text by their Roman numerals:

- I Nevala T, Biancari F, Manninen H, Aho PS, Matsi P, Mäkinen K, Roth WD, Ylönen K, Lepäntalo M & Perälä J (2009) Finnish multicenter study on the midterm results of use of the Zenith stent-graft in the treatment of an abdominal aortic aneurysm. *J Vasc Interv Radiol* 20: 448–454.
- II Nevala T, Perälä J, Aho P, Matsi P, Ylönen K, Roth WD, Manninen H, Mäkinen K, Lepäntalo M & Biancari F (2008) Outcome of symptomatic, unruptured abdominal aortic aneurysms after endovascular repair with Zenith stent-graft system. *Scand Cardiovasc J* 42: 178–181.
- III Nevala T, Biancari F, Manninen H, Matsi P, Mäkinen K, Ylönen K & Perälä J (2010) Inferior mesenteric artery embolization before endovascular repair of an abdominal aortic aneurysm: effect on type II endoleak and aneurysm shrinkage. *J Vasc Interv Radiol* 21: 181–185.
- IV Nevala T, Biancari F, Manninen H, Aho PS, Matsi P, Mäkinen K, Roth WD, Ylönen K, Lepäntalo M & Perälä J (2010) Type II endoleak after endovascular repair of abdominal aortic aneurysm: effectiveness of embolization. *Cardiovasc Intervent Radiol*. In Press.

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1030. Kellokoski, Eija (2009) Ghrelin and atherosclerosis. Human, experimental animal and cell culture studies
1031. Kortesuoma, Riitta-Liisa (2009) Hospitalized children as social actors in the assessment and management of their pain
1032. Niinimäki, Maarit (2009) Medical compared with surgical management in induced abortions and miscarriages
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PUBLICATIONS EDITOR

Publications Editor Kirsti Nurkkala

ISBN 978-951-42-6133-6 (Paperback)

ISBN 978-951-42-6134-3 (PDF)

ISSN 0355-3221 (Print)

ISSN 1796-2234 (Online)

