Cardiovasc Intervent Radiol (2008) 31:451–459
DOI 10.1007/s00270-008-9295-7

ROESCH LECTURE, CIRSE 2007

Endovascular Aneurysm Repair: Current and Future Status

R. J. Hinchliffe · K. Ivancev

Received: 6 September 2007 / Accepted: 6 December 2007 / Published online: 30 January 2008

Abstract Endovascular aneurysm repair has rapidly expanded since its introduction in the early 1990s. Early experiences were associated with high rates of complications including conversion to open repair. Perioperative morbidity and mortality results have improved but these concerns have been replaced by questions about long-term durability. Gradually, too, these problems have been addressed. Challenges of today include the ability to roll out the endovascular technique to patients with adverse aneurysm morphology. Fenestrated and branch stent-graft technology is in its infancy. Only now are we beginning to fully understand the advantages, limitations, and complications of such technology. This paper outlines some of the concepts and discusses the controversies and challenges facing clinicians involved in endovascular aneurysm surgery today and in the future.

Keywords Aneurysm · Aorta · Endovascular aneurysm repair (EVAR)

Introduction

Open aneurysm repair is a major operation, which reached its zenith some years ago. Population-based studies suggest the morbidity and mortality rates are significant. Mortality may be as high as 8%, and 10% may suffer cardiac complications [1]. One in five patients would not undergo the operation again knowing the recovery process involved [2]. There is room for improvement.

It has been over a decade since the pioneering work of Parodi and Volodos culminated in the first endovascular aneurysm repair (EVAR) [3, 4]. Improving technology and experience have addressed many problems associated with the early devices. Conversion to open repair, as high as 20% in some early reports, is now rare [5, 6]. Long-term results with early-generation devices have been disappointing. There have been numerous secondary interventions and conversions. Ruptures have occurred at a rate of 1% per year, which is the same as for untreated small aneurysms <5.5 cm [7]. However, in randomized controlled trials latest-generation devices have performed equally as well as open repair in short- to midterm follow-up.

Morphological Constraints

Early experiences with EVAR demonstrated that outcome was dependent on the morphology of the aneurysms being treated [8]. These observations were confirmed by later studies with second-generation stent-grafts. In particular, breaching the proximal neck guidelines resulted in a fourfold increase in proximal endoleak. Combined morphological deviations multiplied the risk [9].

Unfortunately only a limited number of patients have a normal artery proximal and distal to the aneurysm. Investigations of aneurysm morphology revealed that about 60% of patients are suitable for EVAR [10, 11]. Larger aneurysms may have more adverse morphology [12]. Adverse proximal neck morphology accounts for the majority of unsuitable aneurysms. Almost a third (29%) of aneurysms have an inadequate neck length (<15 mm) [10]. These

R. J. Hinchliffe (✉) · K. Ivancev
Endovascular Centre, Entrance 59, Malmo University Hospital, Malmo 20502, Sweden
e-mail: robhinchliffe@gmail.com

K. Ivancev
e-mail: krassi.ivancev@med.lu.se

limitations of standard EVAR have been a driving force for fenestrated and branch stent-graft technology.

However, it is possible to successfully exclude aneurysms whose morphology is not "ideal"using standard EVAR. In a series of 13 patients with aortic necks <10 mm long, Greenberg was able to demonstrate that such aneurysms could be excluded using a stent-graft with a suprarenal component [13]. Others have found more complex anatomy possible. Wide necks are a surmountable problem. In a study of 16 patients with wide necks (>30 mm) from Leicester, no endoleaks were observed during a 12-month follow-up period. In addition, the aortic necks appeared to significantly decrease in diameter [14]. More recent controlled studies have also confirmed the ability of standard infrarenal aortic stent-grafts to exclude aneurysms with one or more adverse proximal aortic neck features [15]. All these results are relatively short-term and should be viewed with some caution prior to widespread adoption of adverse morphology.

Improvements in graft technology including a reduction in sheath diameters have increased the applicability of EVAR, allowing more aneurysms to be treated. Delivery systems have increased trackability and conformability, allowing graft insertion through more tortuous iliac arteries. The use of a suprarenal anchor stents has reduced the length of the aortic neck required to provide an adequate seal.

Experimental evidence from Nottingham using an in vitro model (Gianturco-Dacron stent-graft) suggested that an angulation of >30 degrees significantly increases endoleak flow by lifting the graft away from the neck wall [16].

Graft Configuration

Aorto-aortic tube grafts have been largely consigned to history, few patients having the prerequisite distal aortic neck [17]. The uni-iliac configuration was originally developed to facilitate graft manufacture (often by the surgeon) and the use of smaller sheath sizes. It was later demonstrated that more aneurysms were suitable for uni-iliac EVAR than for bifurcated (on account of accommodating unilateral CIA aneurysms) [10]. However, improving technology and the use of coil embolization have allowed more patients to be treated with bifurcated stent-grafts. The bifurcated approach is more favorable because of its "physiological" configuration.

Fenestrated EVAR

Fenestrated stent-graft technology was developed in order to increase the morphological applicability of EVAR and offer an alternative to open repair. The technique was developed in 1996 and has subsequently been refined into a system (based on the Zenith stent-graft; William A. Cook Ltd., Brisbane, Australia) which is now commercially available [18, 19].

Fenestrated stent-grafts are individually customized and require some weeks to manufacture. Consequently they are not suitable for patients presenting emergently with acute symptomatic or ruptured abdominal aortic aneurysm (AAA). Major refinements in the fenestrated EVAR technique have included the use of a modular system to assist alignment of the stent-graft in the aortic neck and bifurcation. Further improvements, including the use of diameter-reducing restraining ties which limit initial stent-graft expansion to permit alignment of the stent-graft prior to catheterization of target vessels and reinforced fenestrations to facilitate catheterization, likely have made an impact on results.

Reports on fenestrated stent-grafts were published between 2005 and 2006. Sun and co-workers performed a systematic review of the literature from 1999 to 2006 [20]. Short-term results are encouraging. Perioperative target vessel patency rate was 97% (95% CI, 92%–100%) and 90% (85%–95%) during follow-up. No conversions to open surgery were required. Perioperative mortality was 1.1% (0.4% – 2.7%) and the endoleak rate after 30 days was 9.4% (2.6%–16.3%). Long-term data are limited, with only four studies reporting more than 12 months' mean follow-up.

Iliac Branch Grafts

The consequences of internal iliac artery (IIA) occlusion are variable. Patients may remain asymptomatic, some may suffer buttock claudication, and others may develop frank bowel and pelvic ischemia or infarction. In general, unilateral IIA occlusion is rarely associated with severe ischemic complications [21]. Although rarely associated with severe ischemic complications, when a similar system was used in Leicester, this approach resulted in buttock claudication in 40% of cases [22]. Bilateral IIA occlusion represents a higher risk of complications but does not necessarily result in clinically significant ischemia or infarction due to an extensive collateral pelvic blood supply. In a recent study from St. George's Vascular Institute bilateral IIA embolization prior to EVAR resulted in claudication in 31% of patients. There were no limb/life-threatening complications [23]. Unsurprisingly, embolization of the main IIA trunk resulted in fewer complications than distal embolization. There was no benefit found from undertaking a sequential compared with a simultaneous approach to embolization.

Predictors of clinically significant pelvic ischemia would be desirable if either unilateral or bilateral IIA is being considered to facilitate EVAR. Endovascular repair of ruptured AAA appears to confer a higher risk of bowel ischemia and infarction [24]. The authors of that study consequently modified their practice, suggesting that no patient having EVAR of ruptured AAA should undergo bilateral IIA occlusion. Presumably the hypoperfusion resulted in the occlusion of collateral vessels.

Mehta et al. retrospectively identified 154 patients who had undergone elective IIA occlusion either unilaterally or bilaterally during either EVAR or open aneurysm repair [25]. They speculated that their low incidence (12%) of ischemic complications was due to the preservation of external iliac artery and common femoral artery collaterals, the avoidance of shock, and distal embolization.

The method of IIA occlusion also appears to play a role in the development of ischemic complications [26]. Endovascular coil embolization increases the possibility of distal ischemia because of the technical difficulties involved in occluding the IIA. In one retrospective study, coil embolization resulted in buttock claudication in 45% of patients, compared to 27% of those who received occlusion of the IIA at its origin through placement of a stent-graft across it [27].

In order to prevent the complications associated with IIA occlusion and increase the number of patients treatable by the endovascular method, iliac branch stent-grafts have been developed. Both currently available systems are based on the Zenith stent-graft and incorporate a small side arm which is cannulated with a wire into the IIA. A covered stent is then placed from the branch graft into the IIA, thus maintaining patency of the IIA. Experience with this system is limited to a small number of experienced centers but initial results are encouraging, with high rates of technical success.

Assessment of Aneurysm Morphology

Preoperative assessment of aneurysm morphology is vital to the successful outcome of EVAR. Spiral CT with intravascular contrast (spiral CTA) remains the most popular method of assessment and provides all the necessary information for successful EVAR [28]. Invasive calibration angiography, once used routinely, is no longer required. Angiography was used to aid the determination of aorto-iliac length and the detection of accessory renal arteries. However, spiral CTA with three-dimensional (3D) multiplanar reconstruction provides adequate prediction of length, although some length measurement discrepancy between the two modalities may occur within large aneurysm sacs [29]. Measurements of aorto-iliac length are much less critical with modular systems. Consequently computer software has been developed to facilitate endograft sizing. They enable the user to accurately and reproducibly predict the required size of endograft from 3D spiral CT reconstructions (e.g., TeraRecon) [30].

Magnetic resonance angiography (MRA) is a valid alternative method of preoperative assessment of aneurysm morphology, which is particularly useful in patients with renal impairment [31]. It remains more expensive than spiral CTA and requires detailed postimaging processing. Additional problems arise with the change of imaging modality which will be required if a stainless-steel endograft is deployed. Stainless-steel stents cause considerable image distortion on MRA.

The value of intraoperative intravascular ultrasound (IVUS) continues to be debated. It is currently expensive and requires adequate training before it can be used effectively. Proponents of the technique suggest that it may almost completely replace periprocedural angiography, with its attendant nephrotoxicity [32]. Others argue that IVUS is an unnecessary luxury [33]. In the latter study, selectively used IVUS was only able to detect one lesion that required treatment and had not been identified on angiography. Clearly it is not vital to the satisfactory outcome in the majority of patients. A more realistic view may be that it is an additional instrument of quality control.

Contrast nephrotoxicity is not an uncommon problem in patients undergoing EVAR, where large volumes of contrast can be used in patients who frequently have pre-existing renal impairment. Strategies to reduce the incidence of contrast nephrotoxicity include preoperative intravenous hydration in addition to the use of carbon dioxide [34]. The role of antioxidants (such as *N*-acetyl cysteine) in preventing renal impairment continues to be debated. Any beneficial effect is likely to be small.

Anesthesia and Percutaneous Stent-Graft Delivery

The feasibility of EVAR under local anesthesia has been demonstrated at a number of centers [35]. Percutaneous delivery of stent-grafts also has the potential to further reduce the adverse physiological consequences of EVAR. However, these techniques may not be suitable for all patients and the impact of these techniques on patient outcome has not been confirmed. In one study the physiological effects of local anesthesia were only slightly reduced compared with those of general anesthesia [36]. In fact in another study, de Virgilio et al. were unable to demonstrate any difference in the number of postoperative cardiac or pulmonary complications in a total of 229 patients undergoing EVAR (general anesthesia, n=158; local anesthesia, n=71) [37].

Percutaneous delivery of stent-grafts has allowed some centers to perform EVAR on a day-case basis [38]. Others have found that <30% of patients who have their EVAR performed under local anesthesia are even suitable for discharge the following day [39]. The benefits of a percutaneous procedure appear small. Despite perceived benefits, many of the encouraging results of these percutaneous closure devices have been obtained following interventional cardiological procedures, which require smaller sheaths, possibly in less diseased vessels. In one study of patients undergoing EVAR, 15% of patients required conversion to a conventional groin incision and one patient died from retroperitoneal hemorrhage [40]. Similar results have been published by others [41]. Both studies underscored the importance of careful patient selection to reduce complications. Obesity, scarred groins, and calcification in the former study, and large sheath size in the latter, predicted procedural failure.

Endoleak

Experimental work using a bench-model has successfully demonstrated that all endoleaks, irrespective of diameter and length, are able to transmit systemic pressure [42]. In contrast, in vitro work suggests that thrombosed endoleaks may not behave in a similar fashion [43]. Short and wide thrombosed endoleaks are capable of transmitting greater pressures than long narrow ones. This may offer the reason why thrombosed type I endoleaks do not appear to be safe, whereas long, narrow thrombosed type II endoleaks are usually benign.

In an experimental model, Parodi and Ferreira found that the presence of an outflow channel in an aneurysm sac with endoleak might bring about pressure reduction [44]. The results suggested that increasing the outflow reduced both mean and systolic intrasac pressures.

Type I endoleak is usually the result of incorrect case selection with unsuitable aneurysm morphology, incorrect choice of stent-graft, or maldeployment of the stent-graft. Late cases of type I endoleak may be attributed to migration and/or neck dilation. It is generally accepted that type I endoleaks are associated with rupture, and consequently, treatment is mandatory no matter when they are discovered.

Type II endoleak does not appear to be a graft-related complication of EVAR. The etiology is retrograde perfusion of the aneurysm sac via the inferior mesenteric artery (IMA) or lumbar arteries (especially the fourth pair) and, rarely, other branches such as an accessory renal artery. It has been estimated that one-quarter of preoperatively patent IMAs will subsequently persist to perfuse the aneurysm [45]. Thrombosed IMAs do not appear to reperfuse the aneurysm sac (although this may not be the case following attempted treatment of the endoleak). Flow of blood in patent side branches is variable. In addition to simple inflow to the aneurysm sac, a to-and-fro movement of blood within type II endoleaks can often be detected on duplex ultrasound. The clinical significance of this motion is unknown. Patent side branches can also act as outflow vessels for other types of endoleak, including inflow from either the IMA or lumbar arteries or even other types of endoleak [46].

A number of centers have been unable to identify any preoperative factors which will reliably predict the development of type II endoleak [47]. In contrast, others have found that patients with a large, patent IMA or more than two lumbar arteries on preoperative spiral CTA are at higher risk for the development of persistent type II endoleaks [48].

The natural history of type II (side-branch) endoleak is generally benign but remains incompletely understood. Roughly two-thirds of these endoleaks will spontaneously thrombose in the perioperative period. There have been a minority of cases in which type II endoleaks appear to behave in an aggressive fashion. These include isolated aneurysm ruptures, but it is not yet clear why these type II endoleaks should behave so differently than the majority [49, 50].

Intraoperative studies during EVAR and open AAA repair have suggested that the pressure within side branches would likely be insufficient to result in persistent pressurization of the aneurysm sac [51]. These findings led the authors to hypothesize that high intrasac pressure was more likely to be transmitted directly through the graft rather than patent side branches. This report is in contrast with the intrasac pressure measurements performed by Baum and colleagues [52]. They recorded systemic or near-systemic pressures with pulsatile waveforms in all patients with endoleak, irrespective of type. Pulsatile pressures have also been found in type II endoleaks at laparotomy [46]. One suggestion for these discrepancies and the general benign nature of type II endoleaks is that type II endoleaks only cause a localized rise in sac pressure. Consequently the decision about which type II endoleaks require intervention has been controversial. The prevailing opinion among experts in the field is to take a noninterventional approach unless the aneurysm is getting bigger [53].

Type III endoleak is the result of graft failure. It is associated with subsequent aneurysm rupture. There are two main modes of failure. First is disintegration of the graft fabric and second is modular limb disconnection. These failures were more common with some of the less robust first-generation endovascular stent-grafts. Graft fabric disintegration was associated with some of the thin-

walled graft fabrics [54]. Modular limb disconnection with earlier devices occurred because there was insufficient frictional force to prevent distraction at the junctional zone.

Type IV endoleaks are a consequence of thin-walled grafts which remain porous in the perioperative period. Many of these endoleaks will resolve spontaneously within 1 month of EVAR [55].

Endotension is a condition associated with endoluminal vascular grafts, defined by persistent or recurrent pressurization of an aneurysm sac after endovascular repair, without evidence of endoleak [56]. There are a number of reasons why pressure may be transmitted to the aneurysm sac in the absence of detectable blood flow (endoleak). The first and most widely accepted is the transmission of pressure by thrombus. This phenomenon has been recognized for some time in vascular surgery and is the reason why some aneurysms rupture despite being thrombosed [57]. Other plausible explanations include the presence of an intermittent or low-flow endoleak not amenable to detection by current imaging modalities or, alternatively, hygroma and seroma formation. Ultrafiltration of blood through PTFE grafts has been noted in open surgery [58, 59]. There have even been suggestions that an underlying infective process may be responsible in some patients with endotension.

In vitro analysis of pressure transmission through thrombus suggested that thrombosed endoleak channels do not behave in the same fashion as channels that are patent [43]. In thrombosed endoleaks in vitro, pressure reduction is directly proportional to the length and inversely proportional to the diameter of the channel. As previously suggested, this may account for the benign nature of thrombosed type II endoleaks, in contrast to the aggressive behavior of their type I counterparts.

Endotension is usually identified indirectly by increasing sac diameter, volume, or, possibly, aneurysm pulsatility. Unfortunately, however, endotension may be present in the absence of these signs and can, if left unrecognized or untreated, present with aneurysm rupture [60].

The measurement of pressure within the aneurysm sac may help in the management of patients following EVAR. At present it is only possible to measure pressure for short periods of time by invasive means. Catheters must be placed either by endovascular (with the potential for erroneous readings created by an endoleak channel around the catheter) or translumbar routes and connected to a pressure transducer. An indirect method of detecting a pressurized aneurysm is by echo-tracking ultrasound, which measures sac compliance by detecting wall motion [61]. In vivo measurements have confirmed a pressure reduction in successfully excluded aneurysm sacs [62]. Likewise, the pressure remains high in the presence of endoleak [63].

New technology currently undergoing in vitro and animal testing holds the promise of implantable, chronic pressure telemetry. Preliminary results from animal experiments of 3 months' duration have been encouraging, with close agreement of measurements taken from wireless versus wired pressure sensors [64]. Although these devices would appear to be extremely useful, much work must be done to define their role. They must be shown to detect endoleak as reliably as current imaging techniques. In addition, their position in the aneurysm sac will be vital (as the pressure within a nonhomogenous sac is likely to be variable), as will their long-term accuracy and repeatability. Most critically, we have yet to define what level of pressure renders any particular aneurysm safe.

Endoleak Treatment

Endovascular treatment for endoleak is nearly always possible, and open conversion should be performed only as a last resort. Large balloon-expandable stents (e.g., the giant Palmaz stent) are useful in the treatment of type I endoleak. They improve the apposition of the stent-graft to the aortic neck, which is not attainable by simple angioplasty alone [65].

Type II endoleak is usually managed by coil embolization. Coils are deployed via the endovascular or translumbar routes. Special care must be taken to ensure that the channel is completely occluded, as pressure may be transmitted through an incompletely embolized vessel [66]. Preoperative occlusion (coil embolization) of side branches has been advocated in some publications in order to reduce the incidence of type II endoleak [67]. However, others have suggested that this policy may not be beneficial. In one study, type II endoleak developed in 20% of patients who had undergone preoperative embolization, compared with 23% of patients who had not undergone preoperative treatment [68].

Migration

The "healing," which occurs between a polyester graft and the aorta, is insufficient and consequently requires an alternative method of fixation [69]. Incorporated in endovascular stent-grafts are one or more design features which take the place of nonabsorbable sutures during open surgery. These include radial force of the stents in the aortic neck and hooks (±barbs) to engage either the aortic neck or the suprarenal aorta. Some stent-grafts have also incorporated high columnar strength into their design.

Many first-generation stent-grafts suffered from high rates of migration due to inadequate fixation. The Chuter

device had vestigial hooks, which did not penetrate the full thickness of the aortic wall. In Nottingham, 57% migrated during a 7-year follow-up period [70]. Migrations tend not to appear until a number of months following device insertion. In one study the mean delay was 18 months, and in another, 24 months [70, 71]. It is not clear why migration appears to peak at this time but it may be intimately related with neck dilation. Some authors have suggested that as the neck appears to dilate at 1 mm per year, migration might not occur for 2 to 3 years because of graft oversizing [72]. It is still unclear which precedes the other, neck dilation or migration (suffice to say some grafts migrate without evidence of neck dilation).

A study by Resch et al. revealed that there was a great difference in the forces required to pull out stent-grafts from cadaveric aorta. Balloon-expandable stents and those with robust barbs placed in the suprarenal aorta were associated with the most secure fixation in that study [73]. A number of the other designs provided insufficient strength to prevent the forces placed on the grafts in vivo, which equate to $\sim$10 N. However, caution must be used when considering the results of this paper, as there are many more factors that contribute to endograft fixation and migration in vivo, including aneurysm morphology and, possibly, columnar strength which some endografts possess. Because there are many variables, it is difficult to identify which patients are likely to be at greater risk of developing migration. Hence, the precise risk factors for migration remain uncertain [74].

The effect of stent-graft design on the incidence of migration has been demonstrated in a number of studies. Stent-grafts with robust barbs placed in the suprarenal aorta, engaging the full thickness of the aortic wall, appear to be associated with less migration than their first-generation counterparts with or without vestigial hooks and barbs [75]. The evidence for the efficacy of columnar strength is not yet known because some of the stent-grafts with this feature have additional forms of fixation including either hooks and barbs or suprarenal stents. In addition, longer-term follow-up data, which may reveal the true migration prevalence, are not yet available for these grafts. New techniques are being developed to prevent and treat migration. These include endovascular and laparoscopic stapling [76].

Results of EVAR

In a recent systematic review of EVAR, the 30-day mortality rate was 1.6% in randomized controlled trials and 2% in nonrandomized trials and case series [77]. The results of the EVAR 1 trial suggested that patients considered to be "normal" risk for aortic surgery had a 3% better aneurysm-related survival compared with patients undergoing open repair.

Some patients are not offered open repair because they are "high risk." Using one definition of high risk, Jordan et al. experienced a perioperative mortality of 8.3% for open AAA repair and 2.3% after EVAR [78].

These encouraging results in high-risk patients undergoing EVAR mean that current scoring systems in vascular surgery may not be valid. In a retrospective study, Ananda and colleagues found that all variants of POSSUM overpredicted mortality and suggested that aneurysm morphology needed to be incorporated in future risk-stratification tools for vascular surgery [79].

The long-term outcome following EVAR is improving. In the EVAR 1 trial, the early reduction in aneurysm-related mortality benefit persists to at least 4 years (although all-cause mortality was not different).

Emergency EVAR

Critically ill patients with ruptured AAA are just the cohort of patients who may benefit the most from the reduced physiological insult associated with EVAR.

Early reports demonstrated the feasibility of the technique and identified and surmounted a number of the challenging areas [80, 81]. Subsequent reports have continued the early work, with improved outcome in both selected patients and "all-comers" [82]. Additional refinements have also been made, including the use of local anesthesia [83]. One of the greatest obstacles to its success is the organization and delivery of an emergency EVAR service.

Future Prospects

The large multicenter randomized trials have demonstrated that EVAR is a viable alternative to open repair for the majority of patients presenting with AAA. It is associated with lower perioperative morbidity and mortality and outcomes which are at least as good at midterm follow-up. EVAR continues to be developed to become more applicable and durable. The prospects of new graft technology, fenestrated and branched grafts, in addition to endovascular stapling, hold promise to fulfill these requirements [84, 85]. A greater understanding of endoleak, particularly type II endoleak, is required. Intrasac pressure telemetry remains unproven but may offer rationalization of follow-up from the current intensive schedules.

EVAR has come a long way in a short time. Many challenges lie ahead but the future for EVAR appears bright.

References

1. Blankensteijn JD, Lindenburg FP, Van der Graaf Y, Eikelboom BC (1998) Influence of study design on reported mortality and morbidity rates after abdominal aortic aneurysm repair. Br J Surg 85:1624–1630
2. Williamson WK, Nicoloff AD, Taylor LM Jr, Moneta GL, Landry GJ, Porter JM (2001) Functional outcome after open repair of abdominal aortic aneurysm. J Vasc Surg 33:913–920
3. Parodi JC, Palmaz JC, Barone HD (1991) Transfemoral intraluminal graft implantation for abdominal aortic aneurysms. Ann Vasc Surg 5(6):491–499
4. Volodos NL, Karpovich RP, Troyan VI et al (1991) Clinical experience of the use of self-fixing synthetic prostheses for remote endoprosthetics of the thoracic and the abdominal aorta and iliac arteries through the femoral artery and as intraoperative endoprosthesis for aorta reconstruction. Vasa Suppl 33:93–95
5. Yusuf SW, Whitaker SC, Chuter TA, Ivancev K, Baker DM, Gregson RH, Tennant WG, Wenham PW, Hopkinson BR (1997) Early results of endovascular aortic aneurysm surgery with aortouniiliac graft, contralateral iliac occlusion, and femorofemoral bypass. J Vasc Surg 25:165–172
6. Thompson MM, Sayers RD, Nasim A, Boyle JR, Fishwick G, Bell PR (1997) Aortomonoiliac endovascular grafting: difficult solutions to difficult aneurysms. J Endovasc Surg 4:174–81
7. Harris PL, Vallabhaneni SR, Desgranges P et al (2000) Incidence and risk factors of late ruptures, conversion and death after endovascular repair of infrarenal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 32:739–749
8. May J, White GH, Yu W, Waugh RC, Stephen MS, Harris JP (1996) Results of endoluminal grafting of abdominal aortic aneurysms are dependent on aneurysm morphology. Ann Vasc Surg 10:254–261
9. Stanley BM, Semmens JB, Mai Q, Goodman MA, Hartley DE, Wilkinson C, Lawrence-Brown MD (2001) Evaluation of patient selection guidelines for endoluminal AAA repair with the Zenith stent-graft: the Australasian experience. J Endovasc Ther 8:457–464
10. Armon MP, Yusuf SW, Latief K, Whitaker SC, Gregson RH, Wenham PW, Hopkinson BR (1997) Anatomical suitability of abdominal aortic aneurysms for endovascular repair. Br J Surg 84:178–180
11. Bayle O, Branchereau A, Rosset E, Guillemot E, Beaurain P, Ferdani M, Jausseran JM (1997) Morphologic assessment of abdominal aortic aneurysms by spiral computed tomographic scanning. J Vasc Surg 26:238
12. Hinchliffe RJ, Alric P, Owen V, Rose D, Davidson IR, Armon MP, Hopkinson BR (2003) A comparison of the morphology of intact and ruptured aneurysms of the infra-renal abdominal aorta. J Vasc Surg 38(1):88–92
13. Greenberg R, Fairman R, Srivastava S, Criado F, Green R (2000) Endovascular grafting in patients with short proximal necks: an analysis of short-term results. Cardiovasc Surg 8:350–354
14. Ingle H, Fishwick G, Thompson MM, Bell PR (2002) Endovascular repair of wide neck AAA—preliminary report on feasibility and complications. Eur J Vasc Endovasc Surg 24:123–127
15. Choke E, Munneke G, Morgan R, Belli AM, Loftus I, McFarland R, Loosemore T, Thompson MM (2006) Outcomes of endovascular abdominal aortic aneurysm repair in patients with hostile neck anatomy. CardioVasc Interv Radiol 29:975–980
16. Albertini JN, Macierewicz JA, Yusuf SW, Wenham PW, Hopkinson BR (2001) Pathophysiology of proximal perigraft endoleak following endovascular repair of abdominal aortic aneurysms: a study using a flow model. Eur J Vasc Endovasc Surg 22:53–56
17. Faries PL, Briggs VL, Rhee JY, Burks JA Jr, Gravereaux EC, Carroccio A, Morrissey NJ, Teodorescu V, Hollier LH, Marin ML (2002) Failure of endovascular aortoaortic tube grafts: a plea for preferential use of bifurcated grafts. J Vasc Surg 35:868–873
18. Park JH, Chung JW, Choo IW, Kim SJ, Lee JY, Han MC (1996) Fenestrated stent-grafts for preserving visceral arterial branches in the treatment of abdominal aortic aneurysms: preliminary experience. J Vasc Interv Radiol 7:819–823
19. Faruqi RM, Chuter TA, Reilly LM, Sawhney R, Wall S, Canto C, Messina LM (1999) Endovascular repair of abdominal aortic aneurysm using a pararenal fenestrated stent-graft. J Endovasc Surg 6:354–358
20. Sun Z, Mwipatayi BP, Semmens JB, Lawrence-Brown MM (2006) Short to midterm outcomes of fenestrated endovascular grafts in the treatment of abdominal aortic aneurysms: a systematic review. J Endovasc Ther 13:747–753
21. Karch LA, Hodgson KJ, Mattos MA, Bohannon WT, Ramsey DE, McLafferty RB (2000) Adverse consequences of internal iliac artery occlusion during endovascular repair of abdominal aortic aneurysms. J Vasc Surg 32:676–683
22. Ingle H, Fishwick G, Garnham A, Thompson MM, Bell PR (2002) Long-term results of endovascular AAA repair using a homemade aortomonoiliac PTFE device. J Endovasc Ther 9:481–487
23. Bratby MJ, Munneke GM, Belli AM, Loosemore TM, Loftus I, Thompson MM, Morgan RA (2007) How safe is bilateral internal iliac artery embolization prior to EVAR? CardioVasc Interv Radiol [Epub ahead of print]
24. Yilmaz N, Peppelenbosch N, Cuypers PW, Tielbeek AV, Duijm LE, Buth J (2002) Emergency treatment of symptomatic or ruptured abdominal aortic aneurysms: the role of endovascular repair. J Endovasc Ther 9:449–457
25. Mehta M, Veith FJ, Ohki T, Cynamon J, Goldstein K, Suggs WD, Wain RA, Chang DW, Friedman SG, Scher LA, Lipsitz EC (2001) Unilateral and bilateral hypogastric artery interruption during aortoiliac aneurysm repair in 154 patients: a relatively innocuous procedure. J Vasc Surg 33(Suppl 2):S27–S32
26. Kritpracha B, Pigott JP, Price CI, Russell TE, Corbey MJ, Beebe HG (2003) Distal internal iliac artery embolization: a procedure to avoid. J Vasc Surg 37:943–948
27. Wyers MC, Schermerhorn ML, Fillinger MF, Powell RJ, Rzucidlo EM, Walsh DB, Zwolak RM, Cronenwett JL (2002) Internal iliac occlusion without coil embolization during endovascular abdominal aortic aneurysm repair. J Vasc Surg 36:1138–1145
28. Broeders IA, Blankensteijn JD, Olree M, Mali W, Eikelboom BC (1997) Preoperative sizing of grafts for transfemoral endovascular aneurysm management: a prospective comparative study of spiral CT angiography, arteriography, and conventional CT imaging. J Endovasc Surg 4:252–261
29. Armon MP, Whitaker SC, Gregson RH, Wenham PW, Hopkinson BR (1998) Spiral CT angiography versus aortography in the assessment of aortoiliac length in patients undergoing endovascular abdominal aortic aneurysm repair. J Endovasc Surg 5:222–227
30. Whitaker SC (2001) Planning endovascular aneurysm repair (EVAR) wiyh three-dimensional modelling. In: Greenhalgh RM (ed) Vascular and endovascular surgery techniques. W. B. Saunders, London, pp 209–214
31. Ludman CN, Yusuf SW, Whitaker SC, Gregson RH, Walker S, Hopkinson BR (2000) Feasibility of using dynamic contrast-enhanced magnetic resonance angiography as the sole imaging modality prior to endovascular repair of abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 19:524–530
32. von Segesser LK, Marty B, Ruchat P, Bogen M, Gallino A (2002) Routine use of intravascular ultrasound for endovascular aneurysm repair: angiography is not necessary. Eur J Vasc Endovasc Surg 23:537–542

33. Lipsitz EC, Ohki T, Veith FJ, Berdejo G, Suggs WD, Wain RA, Mehta M, Valladares J, McKay J (2001) Limited role for IVUS in the endovascular repair of aortoiliac aneurysms. J Cardiovasc Surg (Torino) 42:787–792
34. Yusuf SW, Whitaker SC, Hinwood D, Henderson MJ, Gregson RH, Wenham PW, Hopkinson BR, Makin GS (1995) Carbon dioxide: an alternative to iodinated contrast media. Eur J Vasc Endovasc Surg 10:156–161
35. Henretta JP, Hodgson KJ, Mattos MA, Karch LA, Hurlbert SN, Sternbach Y, Ramsey DE, Sumner DS (1999) Feasibility of endovascular repair of abdominal aortic aneurysms with local anesthesia with intravenous sedation. J Vasc Surg 29:793–798
36. Bettex DA, Lachat M, Pfammatter T, Schmidlin D, Turina MI, Schmid ER (2001) To compare general, epidural and local anaesthesia for endovascular aneurysm repair (EVAR). Eur J Vasc Endovasc Surg 21:179–184
37. de Virgilio C, Romero L, Donayre C, Meek K, Lewis RJ, Lippmann M, Rodriguez C, White R (2002) Endovascular abdominal aortic aneurysm repair with general versus local anesthesia: a comparison of cardiopulmonary morbidity and mortality rates. J Vasc Surg 36:988–991
38. Bleyn J, Schol F, Vanhandenhove I, Vercaeren P (2003) Endovascular abdominal aortic aneurysm repair should be carried out as a day-case procedure. In: Greenhalgh RM (ed) Vascular and endovascular controversies. BIBA, London, pp 37–41
39. Henretta JP, Hodgson KJ, Mattos MA, Karch LA, Hurlbert SN, Sternbach Y, Ramsey DE, Sumner DS (1999) Feasibility of endovascular repair of abdominal aortic aneurysms with local anaesthesia with intravenous sedation. J Vasc Surg 29:793–798
40. Teh LG, Sieunarine K, van Schie G, Goodman MA, Lawrence-Brown M, Prendergast FJ, Hartley D (2001) Use of the percutaneous vascular surgery device for closure of femoral access sites during endovascular aneurysm repair: lessons from our experience. Eur J Vasc Endovasc Surg 22:418–423
41. Rachel ES, Bergamini TM, Kinney EV, Jung MT, Kaebnick HW, Mitchell RA (2002) Percutaneous endovascular abdominal aortic aneurysm repair. Ann Vasc Surg 16:43–49
42. Schurink GW, Aarts NJ, Van Baalen JM, Kool LJ, Van Bockel JH (2000) Experimental study of the influence of endoleak size on pressure in the aneurysm sac and the consequences of thrombosis. Br J Surg 87:71–78
43. Mehta M, Ohki T, Veith FJ, Lipsitz EC (2001) All sealed endoleaks are not the same: a treatment strategy based on an ex-vivo analysis. Eur J Vasc Endovasc Surg 21:541–544
44. Parodi JC, Ferreira LM (2003) Endoleak and endotension: the Buenos Aires perspective. In: Veith FJ, Baum RA (eds) Endoleaks and endotension; current consensus on their nature and significance. Marcel Dekker, NewYork, pp 91–100
45. Velazquez OC, Baum RA, Carpenter JP, Golden MA, Cohn M, Pyeron A, Barker CF, Criado FJ, Fairman RM (2000) Relationship between preoperative patency of the inferior mesenteric artery and subsequent occurrence of type II endoleak in patients undergoing endovascular repair of abdominal aortic aneurysms. J Vasc Surg 32:777–788
46. Hinchliffe RJ, Singh-Ranger R, Whitaker SC, Hopkinson BR (2002) Type II endoleak: transperitoneal sacotomy and ligation of side branch endoleaks responsible for aneurysm sac expansion. Endovasc Ther 9:539–542
47. Walker SR, Halliday K, Yusuf SW, Davidson I, Whitaker SC, Gregson RH, Hopkinson BR (1998) A study on the patency of the inferior mesenteric and lumbar arteries in the incidence of endoleak following endovascular repair of infrarenal aortic aneurysms. Clin Radiol 53:593–595
48. Arko FR, Rubin GD, Johnson BL, Hill BB, Fogarty TJ, Zarins CK (2001) Type-II endoleaks following endovascular AAA repair: preoperative predictors and long-term effects. J Endovasc Ther 8:503–510
49. White RA, Donayre C, Walot I, Stewart M (2000) Abdominal aortic aneurysm rupture following endoluminal graft deployment: report of a predictable event. J Endovasc Ther 7:257–262
50. Hinchliffe RJ, Singh-Ranger R, Davidson IR, Hopkinson BR (2001) Rupture of an abdominal aortic aneurysm secondary to type ii endoleak (lesson of the month). Eur J Vasc Endovasc Surg 22:563–565
51. Vallabhaneni SR, Gilling-Smith GL, How TV, Brennan JA, Gould DA, McWilliams RG, Harris PL (2003) Aortic side branch perfusion alone does not account for high intra-sac pressure after endovascular repair (EVAR) in the absence of graft-related endoleak. Eur J Vasc Endovasc Surg 25:354–359
52. Baum RA, Carpenter JP, Cope C, Golden MA, Velazquez OC, Neschis DG, Mitchell ME, Barker CF, Fairman RM (2001) Aneurysm sac pressure measurements after endovascular repair of abdominal aortic aneurysms. J Vasc Surg 33:32–41
53. Veith FJ, Baum RA, Ohki T, Amor M, Adiseshiah M, Blankensteijn JD, Buth J, Chuter TA, Fairman RM, Gilling-Smith G, Harris PL, Hodgson KJ, Hopkinson BR, Ivancev K, Katzen BT, Lawrence-Brown M, Meier GH, Malina M, Makaroun MS, Parodi JC, Richter GM, Rubin GD, Stelter WJ, White GH, White RA, Wisselink W, Zarins CK (2002) Nature and significance of endoleaks and endotension: summary of opinions expressed at an international conference. J Vasc Surg 35:1029–1035
54. Matsumura JS, Ryu RK, Ouriel K (2001) Identification and implications of transgraft microleaks after endovascular repair of aortic aneurysms. J Vasc Surg 34:190–197
55. Zarins CK, White RA, Schwarten D, Kinney E, Diethrich EB, Hodgson KJ, Fogarty TJ (1999) AneuRx stent graft versus open surgical repair of abdominal aortic aneurysms: multicenter prospective clinical trial. J Vasc Surg 29:292–305
56. White GH, May J (2000) How should endotension be defined? History of a concept and evolution of a new term. J Endovasc Ther 7:435–438
57. Dalal S, Donlon M, Beard JD (2001) Thrombosed abdominal aortic aneurysms. Do they need surveillance to prevent late rupture? Eur J Vasc Endovasc Surg 22:570–572
58. Williams GM (1998) The management of massive ultrafiltration distending the aneurysm sac after abdominal aortic aneurysm repair with a polytetrafluoroethylene aortobiiliac graft. J Vasc Surg 28:551–555
59. Risberg B, Delle M, Eriksson E, Klingenstierna H, Lonn L (2001) Aneurysm sac hygroma: a cause of endotension. J Endovasc Ther 8:447–453
60. Politz JK, Newman VS, Stewart MT (2000) Late abdominal aortic aneurysm rupture after AneuRx repair: a report of three cases. J Vasc Surg 31:599–606
61. Schurink GW, Aarts NJ, Malina M, van Bockel JH (2000) Pulsatile wall motion and blood pressure in aneurysms with open and thrombosed endoleaks—comparison of a wall track system and M-mode ultrasound scanning: an in vitro and animal study. J Vasc Surg 32:795–803
62. Chuter T, Ivancev K, Malina M, Resch T, Brunkwall J, Lindblad B, Risberg B (1997) Aneurysm pressure following endovascular exclusion. Eur J Vasc Endovasc Surg 13:85–87
63. Stelter W, Umscheid T, Ziegler P (1997) Three-year experience with modular stent-graft devices for endovascular AAA treatment. J Endovasc Surg 4:362–369
64. Ohki T, Yadav J, Gargiulo N, Kurvers H, Rhee S, Veith FJ, Stern D (2003) Preliminary results of an implanatable wireless aneurysm pressure sensor in a canine model. J Endovasc Ther 10(Suppl):I-32
65. Dias NV, Resch T, Malina M, Lindblad B, Ivancev K (2001) Intraoperative proximal endoleaks during AAA stent-graft repair:

evaluation of risk factors and treatment with Palmaz stents. J Endovasc Ther 8:268–273
66. Marty B, Sanchez LA, Ohki T, Wain RA, Faries PL, Cynamon J, Marin ML, Veith FJ (1998) Endoleak after endovascular graft repair of experimental aortic aneurysms: Does coil embolization with angiographic "seal" lower intraaneurysmal pressure? J Vasc Surg 27:454–461
67. Bonvini R, Alerci M, Antonucci F, Tutta P, Wyttenbach R, Bogen M, Pelloni A, von Segesser L, Gallino A (2003) Preoperative embolization of collateral side branches: a valid means to reduce type II endoleaks after endovascular AAA repair. J Endovasc Ther 10
68. Gould DA, McWilliams R, Edwards RD, Martin J, White D, Joekes E, Rowlands PC, Brennan J, Gilling-Smith G, Harris PL (2001) Aortic side branch embolization before endovascular aneurysm repair: incidence of type II endoleak. J Vasc Interv Radiol 12:337–341
69. Malina M, Brunkwall J, Ivancev K, Jonsson J, Malina J, Lindblad B (2000) Endovascular healing is inadequate for fixation of Dacron stent-grafts in human aortoiliac vessels. Eur J Vasc Endovasc Surg 19:5–11
70. Alric P, Hinchliffe RJ, Wenham PW, Whitaker SC, Chuter TA, Hopkinson BR (2003) Lessons learned from the long-term follow-up of a first-generation aortic stent graft. J Vasc Surg 37:367–373
71. Mohan IV, Harris PL, Van Marrewijk CJ, Laheij RJ, How TV (2002) Factors and forces influencing stent-graft migration after endovascular aortic aneurysm repair. J Endovasc Ther 9:748–755
72. Prinssen M, Wever JJ, Mali WP, Eikelboom BC, Blankensteijn JD (2001) Concerns for the durability of the proximal abdominal aortic aneurysm endograft fixation from a 2-year and 3-year longitudinal computed tomography angiography study. J Vasc Surg 33(Suppl 2):S64–S69
73. Resch T, Malina M, Lindblad B, Malina J, Brunkwall J, Ivancev K (2000) The impact of stent design on proximal stent-graft fixation in the abdominal aorta: an experimental study. Eur J Vasc Endovasc Surg 20:190–195
74. Lee JT, Lee J, Aziz I, Donayre CE, Walot I, Kopchok GE, Heilbron M Jr, Lippmann M, White RA (2002) Stent-graft migration following endovascular repair of aneurysms with large proximal necks: anatomical risk factors and long-term sequelae. J Endovasc Ther 9:652–664
75. Marin ML, Parsons RE, Hollier LH (1998) Impact of transrenal aortic endograft placement on endovascular graft repair of abdominal aortic aneurysms. J Vasc Surg 28:638–646
76. Trout HH, Tanner HM (2001) A new vascular Endostaple: a technical description. J Vasc Surg 34:565–568
77. Drury D, Michaels JA, Jones L, Ayiku L (2005) Systematic review of recent evidence for the safety and efficacy of elective endovascular repair in the management of infrarenal abdominal aortic aneurysm. Br J Surg 92:937–946
78. Jordan WD, Alcocer F, Wirthlin DJ, Westfall AO, Whitley D (2003) Abdominal aortic aneurysms in "high-risk" surgical patients: comparison of open and endovascular repair. Ann Surg 37:623–630
79. Ananda HA, Travis SJ, Hancock J, Davies JN, Woodburn KR (2002) Abdominal aortic aneurysm morphology, POSSUM and the outcome following elective aortic aneurysm surgery. Presented to the Vascular Surgical Society of Great Britain and Ireland, Belfast, November
80. Ohki T, Veith FJ, Sanchez LA, Cynamon J, Lipsitz EC, Wain RA, Morgan JA, Zhen L, Suggs WD, Lyon RT (1999) Endovascular graft repair of ruptured aortoiliac aneurysms. J Am Coll Surg 189:102–112
81. Hinchliffe RJ, Yusuf SW, Macierewicz JA, MacSweeney ST, Wenham PW, Hopkinson BR (2001) Endovascular repair of ruptured abdominal aortic aneurysm—a challenge to open repair? Results of a single centre experience in 20 patients. Eur J Vasc Endovasc Surg 22:528–534
82. Yilmaz N, Peppelenbosch N, Cuypers PW, Tielbeek AV, Duijm LE, Buth J (2002) Emergency treatment of symptomatic or ruptured abdominal aortic aneurysms: the role of endovascular repair. J Endovasc Ther 4:449–457
83. Lachat ML, Pfammatter T, Witzke HJ, Bettex D, Künzli A, Wolfensberger U, Turina MI (2002) Endovascular repair with bifurcated stent-grafts under local anaesthesia to improve outcome of ruptured aortoiliac aneurysms. Eur J Vasc Endovasc Surg 23:528–536
84. Stanley BM, Semmens JB, Lawrence-Brown MM, Goodman MA, Hartley DE (2001) Fenestration in endovascular grafts for aortic aneurysm repair: new horizons for preserving blood flow in branch vessels. J Endovasc Ther 8:16–24
85. Chuter TA, Gordon RL, Reilly LM, Goodman JD, Messina LM (2001) An endovascular system for thoracoabdominal aortic aneurysm repair. J Endovasc Ther 8:25–33