

Current concepts and controversies in endovascular repair of abdominal aortic aneurysms

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Endovascular aneurysm repair (EVAR) has rapidly expanded since its introduction in the early 1990s. Early experiences were understandably associated with relatively high rates of complications including conversion to open repair. Soon, perioperative morbidity and mortality results improved but these concerns were replaced by questions over the long-term effectiveness of the endovascular method highlighted by the increasing numbers of secondary interventions and even reports of aneurysm rupture. A better understanding of the behaviour of stent-grafts in the aorta and the response of aortic aneurysms to endovascular repair has been forged by observations both *in vitro* and *in vivo*. It was only relatively recently that terms such as migration and endoleak were coined. Only now that we are beginning to fully understand the causes and consequences of such problems can further developments be made. The following paper outlines some of the concepts and discusses the controversies and challenges facing clinicians involved in endovascular aneurysm surgery today.

KEY WORDS: Aortic aneurysm, abdominal, surgery - Vascular surgical procedures - Blood vessel prosthesis implantation.

Open aneurysm repair is a major operation, which has reached its zenith. Although significant improvements in outcome have been made since its description by Creech in the 1950s, none have been made within the past 20 years.¹ The mean 30-day mortality rate from open repair in prospective population based studies from 1985 to 1996 was 8.2%.² In that study 10.6% of patients suffered some form of cardiac complication. Further, 1/3 of patients (who were fit and asympto-

matic preoperatively) will not have recovered at a mean follow-up of 34 months and 18% would decide not to undergo the operation again knowing the recovery process involved.³ There is room for improvement.

Attempts at successful endovascular treatment of abdominal aortic aneurysms (AAA) are not a new concept. In 1864 Moore performed wire coiling of a thoracic aneurysm in an attempt to induce aneurysm thrombosis and prevent rupture. Peacock published a similar method in 1968 for the treatment of AAA.⁴ Up to 700 feet of wire was inserted in to 14 patients with AAA considered at prohibitively high risk from conventional open AAA repair owing to medical co-morbidity (Figure 1).

Other ideas have been developed in order to reduce the difficulties associated with open aortic surgery. Cave-Bigley introduced a ringed intraluminal graft to facilitate the proximal aortic anastomosis.⁵ Alternatives include repair *via* a retroperitoneal exposure or laparoscopic routes.⁶

It is over a decade since the pioneering work of Parodi and Volodos culminated in the first EVARs.⁷ Experience and research in tandem with improving technology have ironed out many of the problems associated with the early devices. Conversion to open repair in early reports of EVAR were in the region of 20%.^{9, 10} Conversion itself carries a mortality in excess of 20% and in one series was as high as 43%.¹¹ This complication is now rare. One recent multi-centre study of a 2nd generation

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Figure 1.—Peacock's coiling apparatus for the endovascular treatment of aneurysms.

endovascular stent-graft reported no conversions in a total of 269 cases.¹² Others have also reported that 2nd-generation devices are out-performing their 1st generation counterparts.^{13, 14}

Despite these improvements many aspects of EVAR remain poorly understood. 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.¹⁵ The fate of EVAR will rest, in part, on the mid-term and late results of current devices.

Morphological constraints

The guiding principle of endovascular aneurysm repair has always been to ensure the device bypasses and decompresses the aneurysm and is secured in a normal artery proximally and distally. Early experiences with EVAR demonstrated that outcome was dependent upon the morphology of the aneurysms being treated.¹⁶

The effect of aneurysm morphology on outcome was amply demonstrated in Stanley's review of a single 2nd generation endovascular graft in Australasia.¹⁷ That study showed that breaching the recommended morphological guidelines resulted in a significant number of complications. In particular breaching the proximal neck guidelines resulted in a 4-fold increase in proximal endoleak. Combined deviations multiplied the risk.¹⁷

Unfortunately only a limited number of patients have a normal artery proximal and distal to the aneurysm. Investigation of aneurysm morphology revealed 50-62% of patients would be suitable for repair by the endovascular technique.^{18, 19} Studies suggest that larger aneurysms may have more adverse morphology.²⁰ Adverse proximal neck morphology accounts for the majority of unsuitable aneurysms. Almost 1/3 (29%) of aneurysms have an inadequate neck length (less than 15 mm).¹⁸

Adverse morphological features can be separated under 2 broad headings. First are those features which may prevent delivery of the endovascular stent-graft to its intended site. Second are those which inhibit the formation of a blood-tight seal.

There are a number of generic morphological features which currently make EVAR unsuitable. However, the proportion of aneurysms suitable for EVAR is device specific. Some devices have been designed specifically to circumnavigate adverse morphological features. In addition any surgeon may accept adverse morphological features if he thinks the endovascular approach is in the patients best interests and the chances of successful aneurysm exclusion are high.

Successful access of the endovascular graft to the site of deployment depends largely upon the state of the external and common iliac arteries. The diameter of the external iliac artery necessary will obviously depend upon the diameter of the delivery system, although arteries which are not calcified do tend to stretch and dilate, accommodating larger devices than originally appreciated. Sheath diameter has been reduced but some of the larger grafts still require 24 Fr sheaths for successful insertion. Alternatively, stent-grafts may be inserted directly through the CIA using a conduit. The latter technique tends to be reserved for very large devices, which are intended for placement in the thoracic aorta.

Access difficulties associated with iatrogenic damage of the iliac arteries resulted in a significant proportion of the conversions and considerable morbidity and perioperative mortality in the early series of EVAR. Improved trackability of stent-grafts has reduced access complications. Problematic arteries tend to be excessively calcified, usually with circumferential calcification and are tortuous. Removing stents can increase the flexibility of devices but these grafts are more prone to defor-

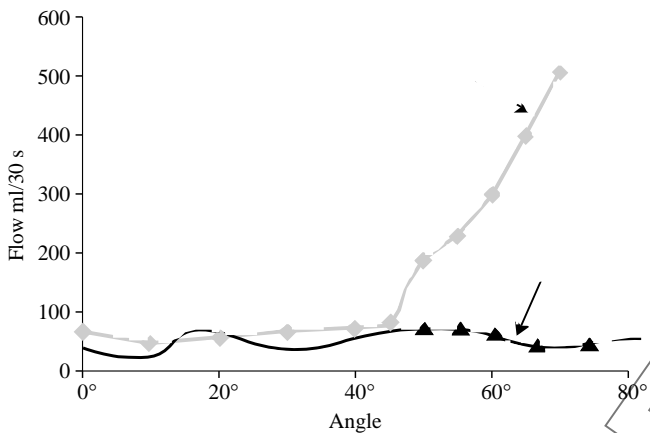


Figure 2.—Graph of proximal peri-endoleak flow against aortic neck angulation in 2 types of endovascular stent-graft (□=Gianturco Z-stent; ▲=embroidered flexible stent-graft with continuous nitinol wire (Anson Medical, Abingdon, UK)).

mation and kinking. In one study almost 46% of unsupported devices required adjunctive stent placement for graft deformation.²¹

Thrombus was thought to represent an obstacle to obtaining a blood-tight seal in the aortic neck. However a study of 184 patients from Nottingham undergoing EVAR with a custom-made uni-iliac endoprosthesis was unable to demonstrate that patients with thrombus-lined necks were at increased risk of developing endoleak.²² Concerns remain regarding the potential for embolization of thrombus from the aortic neck in to the splanchnic, renal or peripheral circulation. This is particularly salient as one study found the degree of embolisation may be more severe following EVAR than with open repair.²³ The results of this early study 1st generation devices may be attributable, in part, to prolonged and difficult catheterisation or perhaps the use of balloon-expandable stents.

However, it is possible to successfully exclude aneurysms whose morphology is not "ideal". In a series of 13 patients with aortic necks less than 10 mm long Greenberg was able to demonstrate that such aneurysms could be excluded using a stent-graft with a supra-renal component.²⁴ Others have also found more complex anatomy possible. Wide necks are a surmountable problem. In a study of 16 patients with wide necks (greater than 30 mm) from Leicester, no endoleaks were observed during a 12-month follow-up period. In addition the aortic

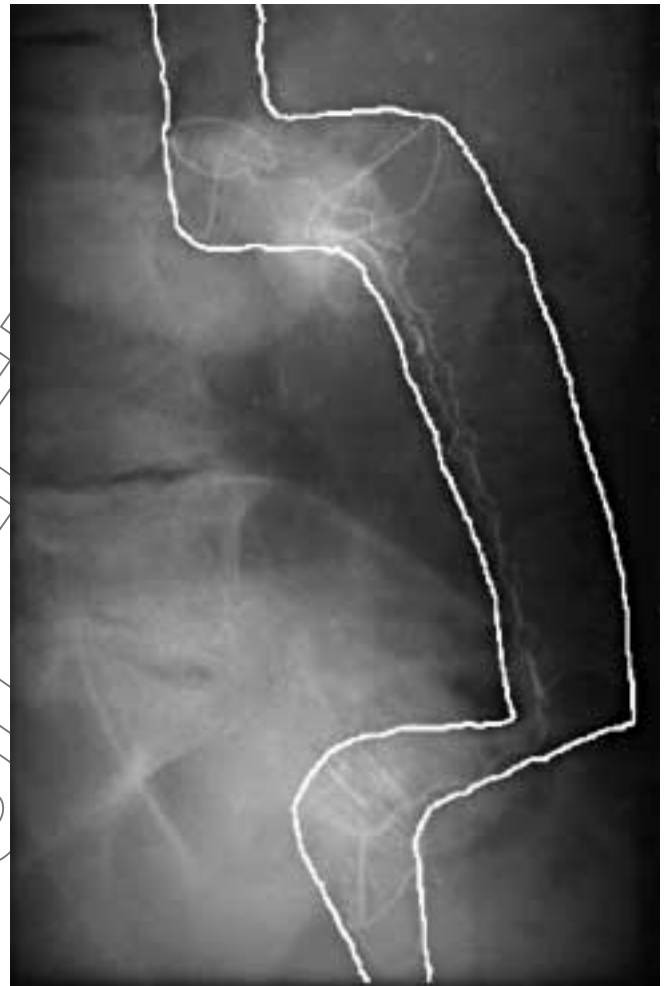


Figure 3.—Plain abdominal X-ray of patient with an angulated and tortuous aneurysm treated by an Anson endovascular stent-graft. Developed with new technology in order to allow successful treatment of angulated necks.

necks appeared to significantly decrease in diameter.²⁵ All these results are relatively short-term and should be viewed with some caution prior to widespread adoption of EVAR in patients with 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 supra-renal anchor stents has reduced the length of

aortic neck required to provide an adequate seal.

Experimental evidence from Nottingham using an *in vitro* model (Gianturco-Dacron stent-graft) suggested that an angulations of greater than 30° significantly increases endoleak flow by lifting the graft away from the neck wall.²⁶ Alternative solutions are required to reduce this problem (Figure 2).

At present the ideal graft has not been manufactured and no one graft possesses all the desirable characteristics. Therefore there are 2 sets of criteria for EVAR. The 1st are a set of procedure specific criteria or a generically accepted morphology for successful EVAR. The 2nd are a set of device specific morphological criteria. The latter will depend upon the type of device and its characteristics. For example, the Anson endovascular stent-graft can accommodate angulation which would not normally be suitable for some designs which use conventional self-expanding stents (Figure 2).

These differences in graft design make risk stratification difficult. However, a number of factors have been identified and graded to help surgeons to predict the outcome for EVAR. Hopefully these systems may be used to allow valid comparisons to be made between new techniques, graft technology and to compare results between centres. The *ad hoc* committee for standardized reporting practices in vascular surgery of the society for vascular surgery/American association for vascular surgery suggested a global scoring system for stratifying the risk of major morbidity and mortality after endovascular repair.²⁷ They calculated a score by the sum of weighted factors in 2 areas. Firstly, medical co-morbidity, with each factor graded from 0 to 3. Secondly were the anatomic severity scores (each graded 0 to 3) related to the presumed risk for failure of device deployment, pelvic ischaemia and embolisation.

Graft configuration

Aorto-aortic tube grafts have been largely consigned to history.²⁸ This configuration is now only really suitable for discreet saccular aortic aneurysms. Only a small number of aneurysms have a suitable distal aortic neck and consequently these grafts were associated with high rates of distal type I endoleak. The distal aorta was recognised as an unsuitable distal landing zone for endovascu-

lar stent-grafts quite early on in the evolution of EVAR. Subsequently the common iliac artery was used as the distal landing zone; bifurcated and uni-iliac grafts were developed. 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).¹⁸ The bifurcated approach appears more favourable because of its "physiological" configuration.

In addition proponents of bifurcated endovascular grafts cite the avoidance of a femoro-femoral extra-anatomic bypass graft as a potential advantage. However, this bypass does not appear to carry the low patency rates associated with occlusive disease and septic complications are uncommon.²⁹ The results of the study suggested the patency rates of femoro-femoral bypass grafts for aneurysmal disease are favourable when compared to occlusive disease.²⁹ Three-year patency rates were 91%, which compares favourably with an accepted 60% for the equivalent bypass grafts inserted for occlusive disease.³⁰

Some institutions, including our own have suggested there may still be a place for the uni-iliac configuration, notably in the emergency situation where rapid aneurysm exclusion is desirable and in the presence of a unilateral common iliac artery aneurysm.^{31, 32}

Intentional or inadvertent occlusion of one or both internal iliac arteries (IIA) is not an uncommon occurrence following EVAR. Occlusion may be a necessary part of exclusion of a common iliac artery aneurysm or occur following maldeployment of the stent-graft.

The consequences of IIA occlusion are variable. Patients may remain asymptomatic, some will suffer buttock claudication whereas others may develop frank bowel and pelvic ischaemia or infarction. In general, unilateral IIA occlusion is rarely associated with severe ischaemic complications.³³ The Montefiore uni-iliac endograft system requires occlusion of the ipsilateral internal iliac artery as an integral part of the procedure. Although rarely associated with severe ischaemic complications, when a similar system was used in Leicester, this approach resulted in buttock claudication in 40% of cases.³⁴ Bilateral IIA occlusion represents a higher

risk of complications but does not necessarily result in clinically significant ischaemia or infarction due to an extensive collateral pelvic blood supply.

Predictors of clinically significant pelvic ischaemia 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 ischaemia and infarction.³⁵ 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 retrospectively identified 154 patients who had undergone elective IIA occlusion either unilaterally or bilaterally during either EVAR or open aneurysm repair.³⁶ They speculated their low incidence (12%) of ischaemic complications was due to the preservation of external iliac artery (EIA) and common femoral artery (CFA) collaterals, the avoidance of shock and distal embolisation.

The method of IIA occlusion also appears to play a role in the development of ischaemic complications.³⁷ Endovascular coil embolisation increases the possibility of distal ischaemia because of the technical difficulties involved in occluding the IIA. In one retrospective study, coil embolisation 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.³⁸

The importance of IIA preservation has been recognised during open repair. The incidence of ischaemic complications following unilateral IIA occlusion appears remarkably similar to those reports following EVAR. In a Canadian aneurysm study when flow into at least one of the internal iliac arteries was not maintained, the incidence of diarrhoea and ischemic colitis increased from the 12% where both were preserved.³⁹

Because of these ischaemic complications there has been great interest in developing methods in order to preserve IIA patency in patients undergoing EVAR. IIAs are most frequently occluded because the CIA presents an unfavourable landing site. The main endovascular technique involves using a flared or wide iliac limb ("bell-bottom" technique) to allow a seal to be developed in a wide CIA.⁴⁰ An alternative and evolving approach in its infancy is the fenestrated or branched graft.

Open approaches to maintain IIA patency require surgical exposure of the iliac bifurcation and consequently can be technically difficult and not without significant morbidity. These include opening the CIA and suturing the endograft to the CIA bifurcation in a "hybrid" approach or alternatively relocating the IIA to the EIA.^{41, 42}

The primary patency of aorto-bifemoral bypass grafts placed at open repair may be as high as 83% at 10 years even when performed for occlusive disease.⁴³ In comparison the patency rates of iliac limbs of endovascular grafts have been disappointing. In our own series from Nottingham with a 1st generation device with unsupported iliac limbs, 87% required intraoperative placement of Wallstents.⁴⁴ Many of the early devices comprised unsupported iliac limbs, however, there is now clear evidence that supported limbs offer improved patency rates over their unsupported counterparts.⁴⁵

Iliac limb occlusion is more common when the limb is deployed in the external iliac artery. Smaller limb diameters are also at greater risk.⁴⁶

There does not appear to be any significant difference in the failure rate of iliac limbs according to graft configuration. In a report of the failure of iliac limbs, the patency rates of uniiliac (97%) and bifurcated (90%) grafts were similar 18 months following the procedure.⁴⁷

The advantages of the unitary design are the potential for reducing endoleak at the junctions between components and ease of deployment. The modular system allows intraoperative customisation of length. This facility is particularly useful because aortoiliac length is difficult to measure. The required endograft length will depend upon the position which the device adopts within the aorta. Preoperative prediction of this position is difficult.

Some of the 1st generation modular stent-grafts were plagued by high rates of modular limb disconnection. Modular devices rely upon the frictional forces between their component parts to prevent dislocation. In some early designs these forces were inadequate. Case reports have underscored the requirement for correct deployment of the iliac limb to ensure stability and prevent distraction.⁴⁸

Thin-walled grafts were introduced in order to reduce sheath diameter and facilitate graft delivery. Some of these devices have ruptured in the medi-

um-term. A combination of the orientation of the stents and the thin-walled nature of the grafts resulted in abrasion of the stents through the graft wall. Some grafts remain porous in the early post-operative period. This results in a 'blush' seen on completion angiography, a type IV endoleak. Seam defects in the graft fabric have also resulted in failure.

A variety of other modes of stent-graft failure have been noted including wire and suture breaks and stent fracture. Some of these failures have been due to flaws in design, usually due to the use of materials with inadequate strength. Others have been attributed to manufacturing problems, notably with nitinol. Undeniably a number of failures have been consequent to device migration and kinking producing abnormal loads on the endograft.

Lessons have been learned from these graft failures. In a "survival of the fittest" many 1st generation endovascular stent-grafts have been withdrawn due to high numbers of graft related complications. Thin walled grafts with stent row separation and weak modular component fixation have been discarded.⁴⁹ The more robust designs with standard gauge graft and stents appear to be out-performing their frail counterparts.

Lawrence-Brown used computational fluid dynamics to investigate the potential forces placed upon a stent-graft under physiological conditions. A mathematical model suggested the maximum downward force placed on a stent-graft would be of the order of 9N. Larger grafts were subject to greater forces than smaller ones.⁵⁰ A number of methods have been developed in an attempt to oppose this distal force. These include the radial force of a stent applied to the aortic neck; hooks and/or barbs to engage the aortic wall either in the aortic neck or supra-renal aorta and finally stent-grafts possessing high column strength.

Hooks and barbs are designed to engage the full thickness aortic wall. One of the problems of this approach is in the patient with a diseased aorta. It is possible that one or more of the hooks or barbs will not engage an atherosclerotic aorta properly thereby reducing the efficacy of the fixation. If the hooks or barbs are positioned in the infra-renal aortic neck seal may be compromised. Damage to extra-aortic tissues such as the IVC or duodenum by these hooks/barbs remains a theoretical possibility but extremely unlikely.

In an attempt to surmount the problems associated with achieving stable fixation in the aortic neck an alternative method or site of fixation has been employed. Supra-renal fixation involves a system crossing the level of the renal artery ostia. The rationale for this technique is several fold. Firstly, the supra-renal aorta tends not to suffer the same degree of atherosclerotic disease as the infra-renal aorta. Secondly, separating the zones of fixation and seal is likely to promote a better blood-tight seal. Finally, the supra-renal aorta is less subject to the aneurysmal process which may befall the infra-renal aortic neck.

Clearly, concerns were raised as soon as it was suggested that bare metallic stents may cross the renal arteries. It was suggested that the stents may act as a source of emboli or even precipitate renal artery occlusion. Early experimental work in pigs suggested some stents were more thrombogenic than others.⁵¹ When deployed, stents with large interstices (such as the Palmaz or Memotherm) may not cover the ostium. If they do cross the ostium then up to 40% may be covered. In contrast, the Wallstent with smaller interstices covers in the region of 36% of the ostium in the majority of cases. Unpolished nitinol stents resulted in partial occlusion of porcine arteries at 6 months follow-up, whereas only 1 Palmaz stent and no Wallstents (haemodynamically similar to the Gianturco Z stent) caused this complication.

Early results *in vivo* were encouraging with no significant difference in renal injury (measured by radionuclide scintigraphy and serum creatinine) when comparing 2 groups of patients, one receiving a conventional infra-renal stent and another using supra-renal.⁵² Likewise during intermediate follow-up there appears to be little evidence of clinically significant renal injury in patients with supra-renal stents.⁵³ A recent study compared serum creatinine and renal artery patency in 32 patients undergoing EVAR with supra- and 57 with infra-renal stents over a 12 months period.⁵⁴ There was no significant deterioration in renal function in those with supra-renal stents and no difference in renal function between the groups.

One publication contradicts these findings. In this large single-centre series of 748 cases the renal infarction rate was 23.2% following supra-renal fixation compared to 6.6% infra-renal.⁵⁵ It is not clear

why this group reported such a high incidence of renal infarction, although aneurysm morphology (case selection) or operative technique may be responsible. In contrast, Kramer reported a much lower rate of renal infarction (8.7%) during the follow-up of supra-renal stents.⁵⁶ The rate of renal infarction did not significantly differ from infra-renal stents (5.6%) in patients at 12-month follow-up. The effects of trans-renal stenting in patients with renal artery stenosis are less well established. Questions remain over the possibility for accelerated disease progression and occlusion (although the placement of a supra-renal stent does not prevent subsequent endovascular therapy of the renal arteries).

In addition to crossing the renal artery ostia, "supra-renal" stents frequently cross or reach the level of the coeliac or superior mesenteric arteries. In a study of 192 patients with supra-renal stents during a mean follow-up of 25 months there was no evidence of distal embolisation on CT scanning and aortography was unable to demonstrate any stenoses or occlusions of the splanchnic circulation.⁵⁷ However, there have been cases of colonic infarction and massive micro-embolisation in patients undergoing EVAR with both infra- and supra-renal stents. It is difficult to attribute any of these complications directly to supra-renal stenting and reported cases underscore the importance of multiple aetiological factors.⁵⁸

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.⁵⁹ Invasive calibration angiography, once used routinely is no longer required. Angiography was used to aid the determination of aortoiliac length and the detection of accessory renal arteries. However, spiral CTA with 3D multiplanar reconstruction provides adequate prediction of length, although some length measurement discrepancy between the 2 modalities may occur within large aneurysm sacs.⁶⁰ Measurements of aorto-iliac length are much less critical with modular systems.



Figure 4.—Computer assisted prediction of stent-graft requirements (StentView).

Exclusion of accessory renal arteries may be considered desirable in order to create an adequate proximal neck. Occlusion appears well tolerated in the majority of patients. In a study of 24 patients, 26 accessory renal arteries were intentionally occluded.⁶¹ There were only 2 significant sequelae during a 12-month follow-up period. One patient developed a renal infarct and hypertension and another progressive renal failure despite a normal perfusion scan. Consequently, the detection of accessory renal arteries by DSA is not necessary for successful outcome in the majority of patients.

Angiography consistently undersizes the proximal neck diameter because it only measures luminal diameter, which is frequently diseased.⁶² In addition the inter- and intraobserver variability with any imaging technique is a potential source of error when planning EVAR. Even with the gold-standard spiral CTA, Van Bockel suggested that interobserver variability regularly exceeded 2 mm.

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 3-D spiral CT reconstructions.⁶³ (Figure 4).

MRA is a valid alternative method of preopera-

tive assessment of aneurysm morphology, which is particularly useful in patients with renal impairment.⁶⁴ 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 it may almost completely replace perprocedural angiography with its attendant nephrotoxicity.⁶⁵ Others argue IVUS is an unnecessary luxury.⁶⁶ In the latter study, selectively used IVUS was only able to detect one lesion, which 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 a not 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 or gadolinium, both of which have been used successfully (alternatively used reduced doses of contrast).⁶⁷ Potential future strategies include the use of antioxidants (such as N-acetyl cysteine) which have shown encouraging results when used with hydration in preventing renal damage associated with radiographic contrast (in those with preoperative chronic renal insufficiency).⁶⁸

Anaesthesia

The feasibility of EVAR under local anaesthesia (LA) has been demonstrated in a number of centres.⁶⁹ Allied to LA, percutaneous delivery of stent-grafts has the potential to further reduce the adverse physiological consequences of EVAR. The technique certainly appears attractive, especially for medically high-risk patients or those with ruptured AAA. Whether LA is suitable for all patients and

what impact it can make upon patient outcome remains to be fully demonstrated. Not all patients will be able to tolerate the technique (patients must lie still for the duration of the procedure) and conversions to general anaesthesia (GA) may be required in some. LA EVAR maintains significant although somewhat reduced systemic physiological disturbance compared with GA.⁷⁰ In fact de Virgilio was unable to demonstrate any difference in the number of postoperative cardiac or pulmonary complications in a total of 229 patients undergoing EVAR (GA, n=158; LA, n=71).⁷¹ It is hoped further refinements of the LA technique may increase the applicability and improve the outcome.

Percutaneous EVAR is now possible due to low profile delivery systems and percutaneous arterial closure devices. Percutaneous arterial closure devices offer an alternative to surgical repair of arteriotomies (and with smaller sheaths, manual compression). A variety of devices are currently used which achieve haemostasis using several different approaches. Some use anchoring sutures with or without collagen plugs and others use collagen haemostatic plugs without any anchoring mechanism.

Percutaneous delivery of stent-grafts has allowed some centres to perform EVAR on a day-case basis.⁷² Others have found that less than 30% of patients who have their EVAR performed under LA are even suitable for discharge the following day.⁶⁹ 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 a recent study of patients undergoing EVAR, 15% of patients required conversion to a conventional groin incision and 1 patient died from retroperitoneal haemorrhage.⁷³ Similar results have been published by others.⁷⁴ 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. It may be argued therefore that larger aneurysms with more difficult morphology and patients with greater co-morbidity — just those who are likely to benefit from percutaneous procedures — are most likely to be those who are unsuitable.

Endoleak

Endoleak is a condition associated with endoluminal vascular grafts defined by the persistence of blood flow outside the lumen of the endoluminal graft but within an aneurysm sac or adjacent vascular segment being treated by the graft.^{75, 76}

Endoleak is a condition peculiar to EVAR. There are some similarities with pseudoaneurysm formation following a conventional (aortic) anastomosis. They are classified according to their site of origin. Type I endoleaks occur from endograft attachment zones (or additionally in the case of a uni-iliac graft around an occluding device). Type II endoleak or "retrobleak" follows flow from aortic side branch vessels.⁷⁷ Type III endoleak is associated with graft failure, either dislocation of a modular limb or through a damaged graft fabric.⁷⁸ Type IV endoleak is associated with some thin-walled porous endografts. It is self-limiting and resolves within 1 month. Type V endoleak or endotension is evidence of a pressurised aneurysm sac in the absence of a demonstrable endoleak.

Further, endoleak may be classified on the basis of its 1st time of detection. Perioperative endoleaks occur within 24 hours of the operation; early within 1-90 days of EVAR; late after 90 days.

Experimental work using a bench-model has successfully demonstrated that all endoleaks irrespective of diameter and length are able to transmit systemic pressure.⁷⁹ In contrast, *in vitro* work suggests that thrombosed endoleaks may not behave in a similar fashion.⁸⁰ 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 found that the presence of an outflow channel in an aneurysm sac with endoleak might bring about pressure reduction.⁸¹ The results suggested that increasing the outflow reduced both mean and systolic intrasac pressures

Type I endoleak

Type I endoleak is associated with subsequent aneurysm rupture and occurs in 0-10% of cases.⁸²

When occurring early, type I endoleak may be due to incorrect case selection with unsuitable aneurysm morphology; incorrect choice of stent-graft or maldesired deployment of the stent-graft. In effect this is a complication of "bad preoperative planning or bad deployment". The majority of late cases of type I endoleak may be attributed to migration and/or neck dilation.

The benefits of graft oversizing in the prevention of attachment site endoleak have been recognised for sometime. This was only clearly demonstrated, however, with an analysis of large numbers of patients in the EUROSTAR database.⁸³ In theory oversizing allows for any measurement discrepancy between observed and expected values and accommodates any non-uniformity in the landing zones.

There is some suggestion that proximal type I endoleak may behave differently in unsupported grafts, although this is not a widely held view. A review of a single-centre experience discovered that 7% of patients who underwent EVAR with such a device (Ancure, Guidant/EVT, Menlo Park CA) had an endoleak at the end of the procedure.⁸⁴ The authors speculated that the low radial force at the proximal stent in combination with an unsupported graft was responsible for the early endoleaks. At 1-month follow-up, however, 82% of these endoleaks had resolved and consequently the authors feel safe observing type I endoleaks until 6 months unless associated with sac enlargement. This experience is in marked contrast to the general consensus for the natural history and management of type I endoleak. These results contradict other authors observations using unsupported grafts.⁴⁴ It is generally accepted that type I endoleaks are associated with rupture and consequently treatment is mandatory no matter when discovered.

Type II endoleak

The accepted type II endoleak rate is of the order of 10-25% and does not appear to be a graft related complication of EVAR. The aetiology is retrograde perfusion of the aneurysm sac *via* the inferior mesenteric (IMA) or lumbar arteries (especially the 4th pair) and rarely other branches such as an accessory renal artery.

It has been estimated that 1/4 of preoperatively

patent IMAs will subsequently persist to perfuse the aneurysm.⁸⁵ 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. Finally, patent side branches can also act as outflow vessels for other types of endoleak, including inflow from either IMA or lumbar arteries or even other types of endoleak.⁸⁶

In Nottingham we have been unable to identify any preoperative factors, which will reliably predict the development of type II endoleak, although patients with no side branches on injection of contrast in to the aneurysm sac at the end of the operation ("sacogram") rarely develop type II endoleak.⁸⁷ In contrast others have found patients with a large, patent IMA, or >2 lumbar arteries on preoperative spiral CTA are at higher risk for the development of persistent type-II endoleaks.⁸⁸

The natural history of type II (side-branch) endoleak is generally benign but remains incompletely understood. In the region of 2/3 of these endoleaks will spontaneously thrombose in the perioperative period. Some type II endoleaks will appear where they have not been present on previous imaging studies.⁸⁹ Of those, which persist, the overwhelming majority will not result in aneurysm expansion. Reports on behaviour of aneurysm sacs in response to type II endoleak varies widely, however, the majority will either stay stable with no tendency to either shrinkage or expansion.^{90, 91} Resnikoff's report of 844 cases of aneurysm bypass and exclusion from a single centre provides an insight in to the behaviour of side-branch endoleak.⁹² Seventeen (2%) of aneurysm sacs remained perfused despite the use of intraoperative ultrasound to detect (and ligate) patent side-branches. Fourteen patients subsequently required operative treatment of the "endoleak" because of marked sac expansion, rupture or because they had become symptomatic.

In a similar fashion, the Montefiore group observed a ruptured IIA aneurysm due to back-bleeding from branches ("type II endoleaks"), which had originally been thrombosed (the proxi-

mal IIA had been occluded with a stent-graft and the distal IIA had been coil embolised).⁹³

In accordance with these reports there have been a minority of cases in which type II endoleaks appear to behave in an aggressive fashion. There have been isolated aneurysm ruptures, but it is not yet clear why these type II endoleaks should behave in such a different manner to the majority.^{94, 95}

Intraoperative studies during EVAR and open AAA repair have suggested that the pressure within side-branches would likely be insufficient to result in persistent pressurisation of the aneurysm sac.⁹⁶ These findings led the authors to hypothesise 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 *et al.*⁹⁷ 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.⁸⁶ One suggestion for these discrepancies and the general benign nature of type II endoleaks may be that type II endoleaks only cause a localised rise in sac pressure.

Warfarin may have the potential to affect endoleak rate, especially related to side-branch patency. One group investigated the effects of warfarin on endoleak and there did not appear to be any difference in the incidence of early or late type II endoleak.⁹⁸ There were insufficient numbers to determine whether warfarin precluded spontaneous thrombosis of these endoleaks. However, at 1-year follow-up the aneurysm sacs in those on warfarin had not reduced in size as quickly as those in the control group.

At present there are no known factors, which are able to predict the behaviour of type II endoleak. Consequently the decision on which type II endoleaks to intervene has been controversial. The prevailing opinion among experts in the field is for a non-interventional approach unless the aneurysm is getting bigger.⁹⁹

Type III endoleak

This type of endoleak is the result of graft failure (Figure 5). It is associated with subsequent aneu-

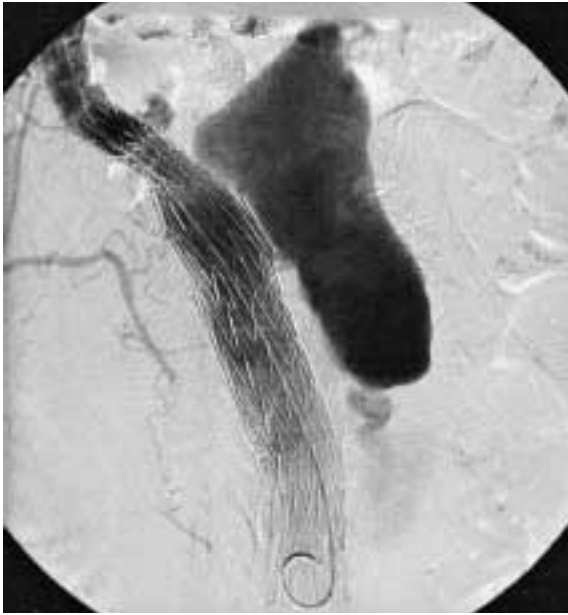


Figure 5.—Type III endoleak. Angiogram demonstrating type III endoleak due to modular limb disconnection.

rysm rupture. There are 2 main modes of failure. First is disintegration of the graft fabric and 2nd is modular limb disconnection.

These failures were more common with some of the less robust 1st generation endovascular stent-grafts. Graft fabric disintegration was associated with some of the thin walled graft fabrics.¹⁰⁰ Modular limb disconnection with earlier devices occurred because there was insufficient frictional force to prevent distraction at the junctional zone. A proportion of these failures were the result of insufficient overlap between the main device and iliac limb or were precipitated by graft deformity, usually the result of caudal migration. The more distal placement of the modular limb join may lessen the risk of disruption by the “splinting” effect of the intact other limb.

Type IV endoleak

Type IV endoleaks are a consequence of thin-walled grafts which remain porous in the perioperative period. The result may be seen as an arterial blush on completion angiography following deployment. As many as 1/3 of these devices will show evidence of contrast within the aneurysm sac

in the first few days following EVAR. However, many of these endoleaks will resolve spontaneously within 1 month of EVAR.¹⁰¹ Bench-model testing of porous endografts has revealed that they transmit systemic pressures to the aneurysm sac.¹⁰² Consequently, the long-term durability of these thin-walled grafts remains to be determined and is probably suspect.

One of the problems associated with type IV endoleak is the diagnostic dilemma it frequently presents. It can be difficult to distinguish from other types of endoleak on completion angiography and spiral CTA and therefore becomes a diagnosis of exclusion.

Endotension

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.¹⁰³

It is an uncommon condition following EVAR. In a series from Sidney, there were only 7 cases out of a total of 400 (1.8%) EVARs performed.

There are a number of reasons why pressure may be transmitted to the aneurysm sac in the absence of detectable blood flow (endoleak). The 1st and most widely accepted is the transmission of pressure by thrombus.¹⁰⁴ This phenomenon has been recognised for sometime in vascular surgery and is the reason why some aneurysms rupture despite being thrombosed. 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.¹⁰⁵ It may be speculated that this phenomenon could be responsible for endotension in a number of patients with PTFE grafts. Risberg reported hygroma formation in a series of 4 cases in which they found an accumulation of clear fluid around the graft.¹⁰⁶ There have even been suggestions 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 ones which are patent.⁸⁰ 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 behaviour 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 unrecognised or untreated present with aneurysm rupture.¹⁰⁷

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 pressurised aneurysm is by echo-tracking ultrasound, which measures sac compliance by detecting wall motion.¹⁰⁸

In vivo measurements have confirmed a pressure reduction in successfully excluded aneurysm sacs.¹⁰⁹ Likewise, the pressure remains high in the presence of endoleak.¹¹⁰

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 between measurements taken from wireless and wired pressure sensors.¹¹¹ 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 non-homogenous 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.

Diagnosis of endoleak

The gold standard for the detection of endoleak remains spiral CTA and selective angiography both

with delayed views (although the latter is invasive). Alternative non-invasive modalities include MRA and duplex ultrasound. Spiral CTA and MRA are reliable in establishing a diagnosis of endoleak but it can be difficult to decide the origin and type. Of course this is of particular importance as it may affect subsequent management.

Ultrasound is a useful tool in the diagnosis of endoleak and may be improved by the use of contrast agents. The ability to identify the direction of flow in endoleaks can modify subsequent management. The technique is, however, operator dependent and views may be obscured by bowel gas although bowel preparation does improve image quality.¹¹²

Type II endoleaks can be difficult to identify on imaging because there may be a delay before flow is seen from the side-branch vessel into the aneurysm sac. Consequently, delaying the image acquisition may increase the sensitivity of spiral CTA for type II endoleak. Ultrasound scanning with or without contrast enhancement is not as reliable as spiral CT in diagnosing type II endoleak.¹¹³

The measurement of pulsatile wall motion using ultrasound technology has been suggested as an indirect method of detecting endoleak and pressurisation of the aneurysm sac following EVAR. In an *in vitro* and animal experimental model using 2 types of ultrasound, the investigators were able to attain a sensitivity of 64% and specificity of 100% for the detection of endoleak. For the detection of pulse pressure the combined sensitivity was 90% and specificity of 100%. Pulsatile wall motion was not reliable for the detection of mean aneurysm pressure.¹¹⁴

Although most surgeons think they are able to assess the pulse pressure within an aneurysm clinically, a more critical appraisal of pulse pressure within the aneurysm sac following EVAR with and without endoleak suggested this was not the case. Clinical examination is therefore not a reliable technique with which to assess the exclusion of the aneurysm sac.^{115, 116}

Other novel approaches suggested for the detection of an incompletely excluded aneurysm sac involve the measurement of fibrin degradation products (FDPs) and matrix metalloproteinases (MMPs), which act as surrogate markers of aneurysm activity.

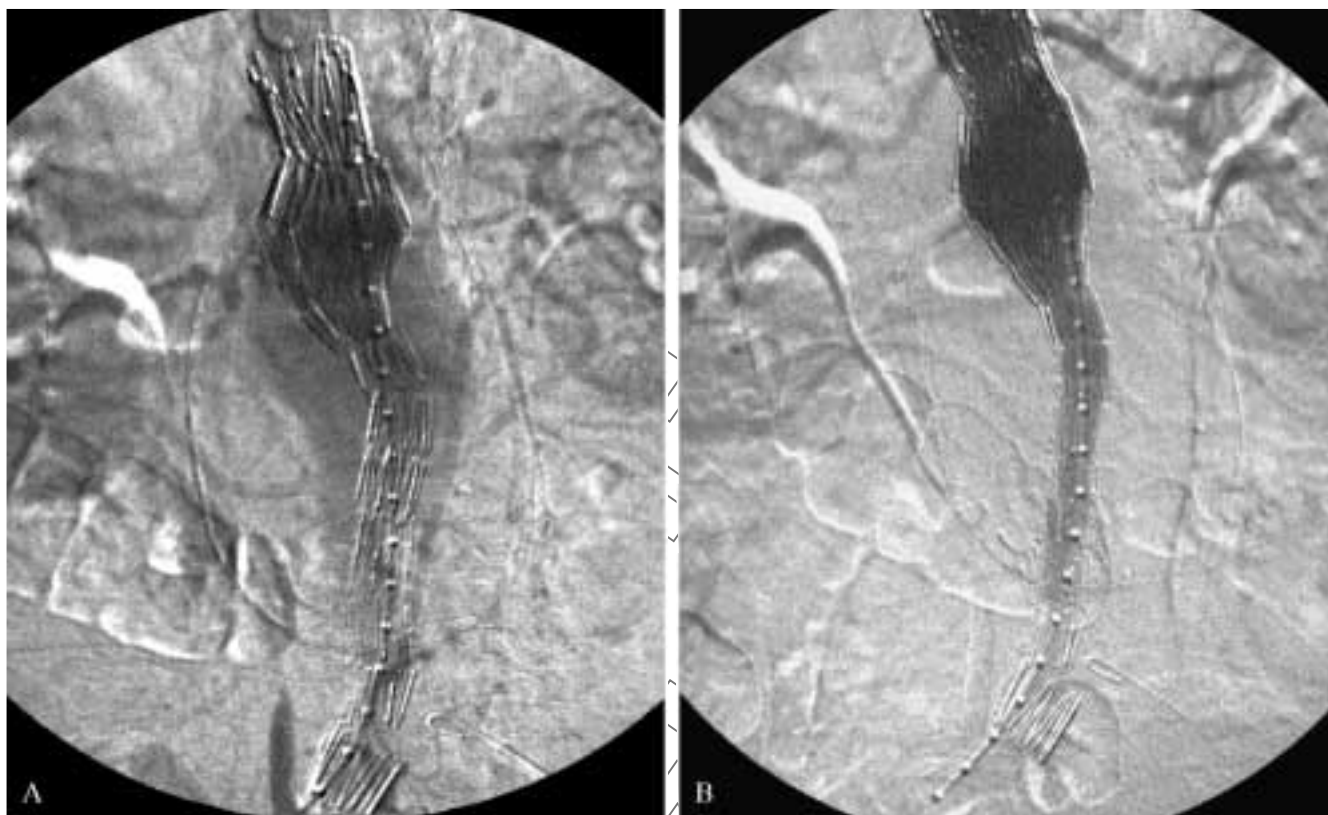


Figure 6.—A) Proximal endoleak at completion angiography following stent-graft deployment in a short neck. B) Successful treatment of the endoleak in Figure 6A with a Palmaz stent.

Treatment of endoleaks

The treatment of type I endoleak when detected at “completion angiography” should always be performed before the patient gets off the table if at all possible. Prevention may be affected by recognition of the morphological risk factors and accurate deployment of the endograft. Endovascular management should always be attempted prior to more invasive manoeuvres, which carry a high morbidity and mortality. Treatment will depend on the perceived cause of the endoleak. If the graft has been deployed too low in the aortic neck an extension may be placed up to the level of the renal arteries. Other endovascular manoeuvres include the use of angioplasty balloons in an attempt to improve the apposition of the graft to the aortic neck and balloon expandable stent such as the giant Palmaz stent (Figure 6). Palmaz stents have been found to

be useful in a variety of situations, where an improved seal is required.¹¹⁷

More invasive manoeuvres include the use of periaortic ligatures, which may be placed at laparotomy.¹¹⁸ Whilst this technique avoids aortic cross-clamping it is associated with significant morbidity and mortality as it often comes at the end of a complicated procedure in a patient who would not otherwise be fit for an open repair.¹¹⁹ Tying the periaortic ligatures with a Palmaz stent or balloon inside the aortic neck may prevent overtightening and stenosis and at the same time probably improving seal. The final alternative is conversion to a traditional open repair.

The treatment modality and timing of type II endoleaks is controversial. A variety of methods exist including endovascular, percutaneous, laparoscopic and open procedures. In Nottingham we have advocated the use of intrasac thrombogenic (either gelatin or more recently polyvinylalcohol)

sponge inserted intraoperatively.¹²⁰ Patients receive sponge if patent side-branches are demonstrated on contrast injection in to the aneurysm sac ("sacogram"). The sponge is inserted either through the contralateral iliac limb during aortouni-iliac endovascular EVAR or alongside the limb of a bifurcated endograft. A negative sacogram is able to predict those patients who are unlikely to subsequently develop a type II endoleak. Since we started using sponge our type II endoleak rate has reduced from 9% to 1.3%.¹²¹ If permanent sac ("void") fillers are used they may have the potential to prevent late type II endoleak and help stabilise the endograft. These void fillers could be incorporated in to the endovascular graft itself.

Other centres prefer to treat type II endoleaks later. Coil embolisation is an attractive technique because of its minimally invasive nature. Coils are deployed *via* the endovascular or translumbar routes. Some questions remain regarding the efficacy of this technique. Special care must be taken to ensure the channel is completely occluded as pressure may be transmitted through an incompletely embolised vessel.¹²²

The translumbar treatment appears more effective in the elimination of type II endoleaks than transarterial because the endoleak cavity can be treated more easily. With transarterial embolisations only one component of the endoleak is treated (*i.e.* the inflow) which leaves a dynamic situation where other endoleak channels may open up or convert from outflow to inflow channels. In this way, type II endoleaks appear to behave in a similar fashion to arterio-venous malformations.¹²³

A variety of embolic agents (including glues) exist which may be used in order to thrombose type II endoleaks. They have the advantage of not distorting postprocedural spiral CTA although there remains a small but definite risk of embolisation in to and damage to the spinal circulation.

Preoperative occlusion (coil embolisation) of side-branches has been advocated in some publications in order to reduce the incidence of type II endoleak.¹²⁴ 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 embolisation compared with 23% of patients who had not undergone preoperative treatment.¹²⁵

The management of type III endoleak is usually

permissible via the endovascular route using a covered stent to reline the failing endograft. Occasionally conversion to conventional open repair is required where the endograft has become distorted. Type IV endoleaks should be managed expectantly and may not require intervention.

Endotension, usually observed as an increasing aneurysm sac requires some form of intervention. It is important to try and identify an endoleak using a variety of imaging modalities including selective angiography. Migration may also cause endotension. Being able to identify and correct an endoleak or migration may save the patient from conversion to open repair, which is associated with considerable morbidity and mortality in this situation.

Aneurysm sacs, which appear stable, present another potential difficulty during follow-up. Some surgeons would feel uncomfortable observing stable aneurysm sacs for fear that they remain pressurised and at risk of rupture. Others would prefer to continue observation in the absence of endoleak.⁹⁹ The use of aneurysm volume studies and measurement of intra-sac pressures may determine whether the patient remains at risk.

Migration

The "healing", which occurs between a polyester graft and the aorta, is insufficient and consequently requires an alternative method of fixation.¹²⁶ Incorporated in endovascular stent-grafts are one or more design features, which take the place of non-absorbable sutures during open surgery. These include, radial force of the stents in the aortic neck and hooks (\pm barbs) to engage either the aortic neck or supra-renal aorta. Some stent-grafts have also incorporated high columnar strength into their design.

Many 1st 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.⁴⁴ Worryingly, migration was clearly identified as a risk factor for subsequent aneurysm rupture.¹⁵

Migrations do not tend to appear until a number of months following device insertion. In one study

the mean delay was 18 months and in another 24 months.^{44, 127} It is not clear why migration appears to peak at this time but 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.¹²⁸ 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 Resch¹²⁹ revealed there was a great difference in the forces required to pullout stent-grafts from cadaveric aorta. Balloon-expandable stents and those with robust barbs placed in the supra-renal aorta were associated with the most secure fixation in that study.¹²⁹ A number of the other designs provided insufficient strength to prevent the forces placed on the grafts *in vivo*, which equate approximately to 10N. However, caution must be placed on the results of this paper, as there are many more factors, which 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.¹³⁰

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 supra-renal aorta, engaging the full thickness of the aortic wall appear to be associated with less migration than their 1st generation counterparts with or without vestigial hooks and barbs.¹³¹ 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 supra-renal stents. Secondly, the longer-term follow-up data, which may reveal true migration prevalence, is not yet available for these grafts.

The diagnosis of migration may be made on antero-posterior or lateral abdominal X-ray or on spiral CTA either using reconstructions or plain axial slices using aortic side branches and the renal vein as landmarks. Spiral CTA may be better because of the problems associated with parallax during X-ray.¹³² Regular surveillance is mandatory to detect migration and prevent any complications, including endoleak, graft occlusion and aneurysm rupture.

Conventional endovascular management of migration involves the placement of a covered stent-graft ("cuff" extension) to reline the aorta to the level of the renal arteries. It is now possible to prevent or treat minor degrees of migration with laparoscopic techniques. A report from Dusseldorf described using sutures to attach the endograft to the aortic neck in a small series of patients.¹³³ This technique may prevent conversion to conventional open repair but it is not clear whether it offers any benefit over an endovascular procedure. Other alternatives are being explored and developed including endovascular stapling.

Graft infection

Graft infections are unusual following EVAR and appear less common than open repair. Local wound complications such as seroma, haematoma and superficial wound infection occur in the order of 8% of patients.¹³⁴ Direct spread of infection to the EVAR is unusual although aortouni-iliac endografts have a concomitant femoro-femoral bypass, which is at greater risk of becoming infected. In our series 4.3% of femoro-femoral bypasses became infected but there was no spread to or involvement of the stent-graft.²⁹ The graft is not usually exposed to the air prior to delivery and it is difficult justify EVAR being confined to the operating theatre on the basis of infection rates if superior imaging facilities are available in the endovascular suite. Incidentally, postoperative spiral CTA may demonstrate gas within the aneurysm sac (especially following insertion of thrombogenic material).¹²⁰ The gas is not pathognomonic of infection and is usually absorbed within a week.

Antibiotic prophylaxis is mandatory. Graft infections appear more likely during secondary procedures but there is no evidence to suggest whether patients should receive antibiotic prophylaxis for subsequent non-EVAR related procedures. Intuitively there is a wisdom in giving antibiotic prophylaxis to post-EVAR patients being treated for focal sepsis such as dental abscess.

The effect of EVAR on patient physiology

One of the potential advantages of EVAR is its ability to offer more aneurysm patients a successful

repair. A number of patients are currently turned down for open repair because of medical co-morbidity. Open aneurysm repair can be performed in high-risk patients with low mortality but the operation carries significant major (14%) and total (29%) morbidity.¹³⁵

There is no doubt that EVAR carries less physiological stress on patients than open repair. The degree of bowel ischaemia, endotoxaemia and cytokine generation is less with EVAR than open repair.¹³⁶ Cytokine release is a major factor in the development of multiple organ failure (MOF) following aneurysm repair, a common mode of perioperative death in these patients.¹³⁷ Cardiovascular, respiratory and renal insults are all attenuated during and following EVAR.¹³⁸⁻¹⁴⁰

As a result of the reduced physiological upset, the recovery times are more rapid and hospital stays shorter with EVAR.¹⁴¹ In contrast, the same study was unable to demonstrate any difference in the late (>6 months) functional outcome between the 2 techniques. Arko's study revealed 95% of patients undergoing EVAR had made a full-recovery 6 months postoperatively compared to 75% in the open group ($p < 0.01$).¹⁴¹

The results of EVAR

Many papers have been published giving the results of EVAR. Conversion rates, morbidity, mortality, endoleak, secondary intervention and ruptures are all quoted but vary considerably. The reasons for some of these difference have already been discussed and include type of graft, morphology and date of publication. In a meta-analysis of the published literature between 1995 and 1999, Walschot *et al.* were able to identify 39 articles.¹⁴² The perioperative death rate was 3.7%. Endoleak and conversion were 13.1% and 5% respectively in the perioperative period and 5.4% and 1.4% *per annum* thereafter.

Some patients are not offered open repair because they are "high risk". Using a definition of high risk as those patients with at least one of the following: age more than 80 years, chronic renal failure (creatinine > 2.0), compromised cardiac function (diminished ventricular function or severe coronary artery disease), poor pulmonary function, preoperative aortic procedure, a "hostile" abdo-

men, or an emergency operation, Jordan *et al.* experienced a perioperative mortality of 8.3% for open AAA repair and 2.3% after EVAR.¹⁴³

In a series of 116 high-risk patients from Chuter *et al.* were able to achieve successful aneurysm exclusion with low perioperative morbidity and mortality rates.¹⁴⁴ The definition of "high risk" was made on the basis of a number of unspecified assortment of clinical and laboratory criteria. The 30-day rates of mortality, major morbidity, and minor morbidity were 3.4%, 20.7%, and 12%, respectively, in the first 58 patients. With increased experience and improved technique the results were improved to 0%, 3.4%, and 3.4%, respectively, in the last 58. Clearly some patients will survive EVAR who would not have survived open repair.

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 *et al.* 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.¹⁴⁵

One concern regarding the repair of aneurysms in unfit patients is the natural history of their co-morbidity. During a 9-year period, 57 patients who were deemed too unfit for elective AAA repair were followed up. Fifty patients died during follow-up. Median survival was 18 months and only 20 (35%) died from rupture.¹⁴⁶ In another study of medically high risk patients undergoing open repair, 5-year survival was just 46%.¹³⁵

Early during EVAR results were reported from single centres. It was clear that more meaningful data could be acquired from the aggregation of experiences from multiple centres. Consequently, important data regarding the outcomes of EVAR have been gleaned from the analysis of the UK RETA registry and the EUROSTAR database.

More recently national multicentre randomised trials have commenced in order to try and define the role of both EVAR and open repair in the management of AAA. The UK EVAR studies were commenced in 1998 and consist 2 arms. EVAR I randomises fit patients suitable for EVAR to open surgery or EVAR and EVAR II randomises unfit patients to EVAR and best medical treatment (BMT) or BMT and aneurysm surveillance. The trials are

scheduled to stop recruiting in 2004 unless stopped. The Dutch DREAM trial has been organised on similar grounds but compares only those suitable for both techniques.¹⁴⁷

Postrepair aneurysm morphology

Following successful endovascular aneurysm exclusion it was generally believed that the sac would progressively shrink and disappear. This certainly happens in some patients but not all excluded sacs behave in the same fashion. Confusingly, some excluded sacs will not start to shrink immediately whereas others will stop shrinking after 18 months.¹⁴⁸ The significance of a stable non-shrinking, non-growing aneurysm sac continues to be debated.¹⁴⁹ It is difficult to draw any conclusions from these data. Aneurysm size, aneurysm wall and sac composition in addition to patient and stent-graft factors may all account for the differences in sac behaviour between patients.

In addition EVAR appears to cause a peri-aneurysmal inflammatory response in some patients. Inflammation has been noted both on follow-up spiral CTA and during laparoscopy.¹⁵⁰ The significance of the inflammation is as yet undetermined but similar phenomena have been observed following stent-placement in the iliac arteries for occlusive disease.¹⁵¹ It is also observed following open repair of aortoiliac lesions by prosthetic grafts.

The fate of the aneurysm neck following EVAR is variably reported. If progressive neck dilation is expected then EVAR with current devices would be fundamentally flawed. The behaviour of the aneurysm neck following open repair suggests 5% of patients will develop true juxta-anastomotic supra-graft aneurysms following open aneurysm repair.¹⁵² Sonesson¹⁵² reported a growth of the aortic neck of 0.5 mm per year following open repair.¹⁵³ There have been contrasting reports following EVAR with some suggesting progressive dilation whilst others conclude that EVAR prevents aortic neck enlargement.^{154, 155} The early response of the aortic neck appears to differ whether a balloon- or self-expanding stent is placed within side it.¹⁵⁶ Overall there appears to be a subset of patients whose aneurysm neck continues to dilate following either open or endovascular repair. The difficulty

lies in identifying this subset of patients prior to treatment. A report from Nottingham suggested that those patients with hypertension and who continued to smoke appeared to be those most at risk of developing juxta-renal aneurysms in the residual infra-renal aortic neck following open repair.¹⁵² Our anecdotal experience would suggest that relining the aorta to the level of the renal arteries may confer some protection to aortic neck dilation following EVAR.

In contrast the supra-renal aorta does not appear to be subject to the same aneurysmal process as the infra-renal aorta.¹⁵⁷ This observation may add weight to the argument for the use of supra-renal stent-graft fixation techniques.

Changes in aortic morphology following apparent successful aneurysm exclusion has been blamed for mid-term device failure. In one study of a 1st generation device, 69% became buckled or kinked at 1 year. These failures were attributed to longitudinal shrinkage of the aneurysm sac.¹⁵⁸ Others, however, have rejected this hypothesis. They have been unable to identify foreshortening of an excluded arterial segment following EVAR, which may suggest a problem with the device.¹⁵⁹ The kinking is worsened and may be caused by migration of both ends of the stent-graft.

Emergency EVAR

The mortality from open repair of ruptured AAA remains excessive and the prospect of reducing the mortality from this condition would appear long overdue. 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 have demonstrated the feasibility of the technique and identified and surmounted a number of the challenging areas.^{160, 161} Importantly, stent-grafts have been developed for use in the emergency setting. Subsequent reports have continued the early work with improved outcome in both selected patients and "all-comers".³² Additional refinements have also been made including the use of local anaesthesia.¹⁶¹ One of the greatest obstacles is likely to be the organisation and delivery of an emergency EVAR service.

Future prospects

The large multi-centre randomised trials will hopefully tell us which patients will benefit most from current EVAR technology. Paradoxically, those patients who may benefit most are those who are not included in these current trials, namely those patients with ruptured aneurysms.

EVAR must be made more applicable and durable. The prospects of new graft technology, fenestrated and branched grafts in addition to endovascular stapling all hold promise to fulfil these requirements.^{162, 163}

A greater understanding of endoleak, particularly type II endoleak is required. Intrasac pressure telemetry may provide the answers and allow rationalisation of follow-up from the current intensive schedules.

There are still many challenges but the future for EVAR appears bright.

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