

# Endovascular stent grafting of abdominal aortic aneurysms: a radiologist's view on a new potential issue in renovascular disease

A Nicholson

*Vascular Radiology Department, Hull & East Yorkshire Hospitals Trust, Anlaby Road,  
Kingston upon Hull, HU2 3JZ, UK*

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## Abstract

Endovascular abdominal aortic aneurysm repair was first described in 1991. Initial enthusiasm for the procedure has now diminished to a point where many consider the procedure to be a 'failed experiment'. Is this true or is it simply a good idea still under development?

## Keywords

EVAR; endoleak; rupture; randomised controlled trial.

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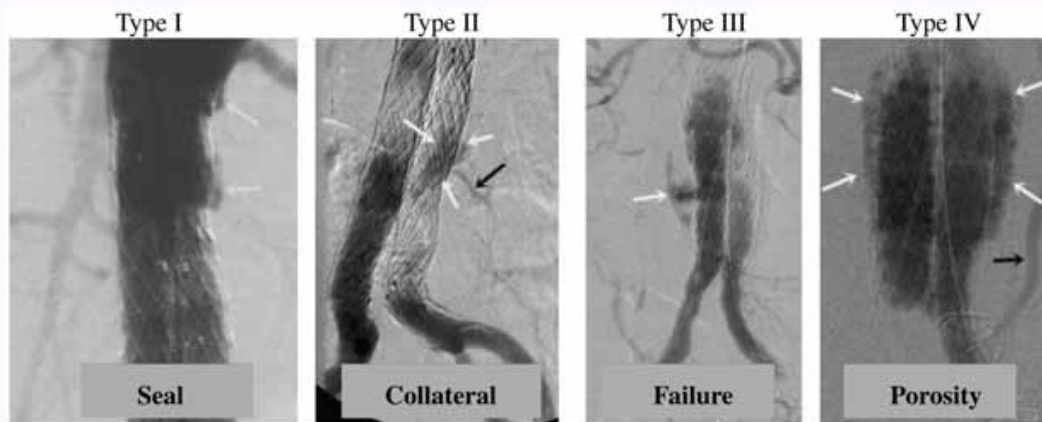


Fig. 1. Endoleak classification.

Endovascular stent grafting of abdominal aortic aneurysms (EVAR), first proposed by Dotter in 1969<sup>[1]</sup> and eventually first performed by Parodi in 1991<sup>[2]</sup>, has enjoyed a meteoric rise in enthusiasm but more recently a meteoric fall in ‘popularity’. The publication of a leading article in the *British Journal of Surgery* suggesting that the technique and technology should be abandoned<sup>[3]</sup> is a nadir, which suggests that the term popularity rather than scientific merit is well used. To understand what has been happening it is important to look at the evidence and history of abdominal aortic aneurysm (AAA) treatment over the last 10 years.

The publication of the UK Small Aneurysm Study not only demonstrated that aneurysms of less than 5.5 cm had an annual rupture rate of 1%, but it also showed the variability in mortality figures following open repair<sup>[4]</sup>. This had been noted in previous publications<sup>[5]</sup>. Though many surgeons claim mortality figures for AAA surgery of less than 3%, the truth is that many have rates as high as 12%. Parodi and Palmatz’s work<sup>[2]</sup> induced a sense

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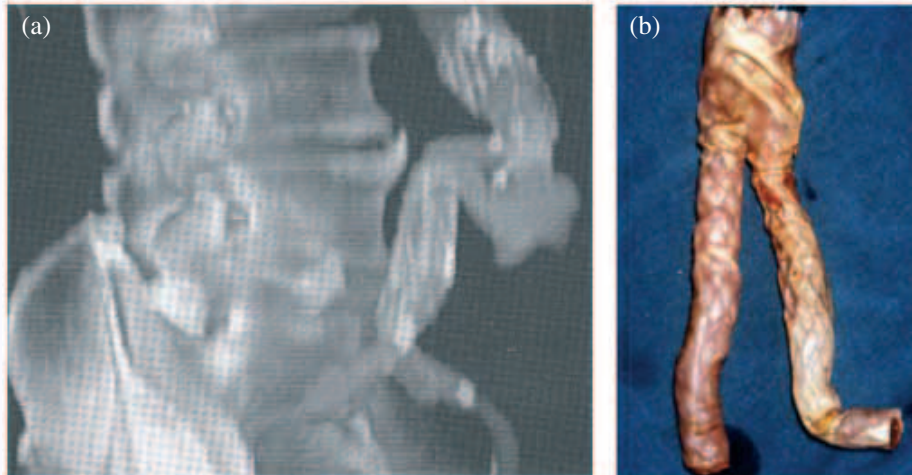
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**Fig. 2.** (a) First-generation graft: migration and kinking. Longitudinal reduction in sac size has resulted in severe superior to inferior kinking. (b) Second-generation stent graft removed at open surgery because of severe limb kinking.

of excitement that suddenly put vascular radiology at the forefront of vascular surgical thinking. Surgeons started making home-made devices and inserting them at a prodigious rate. When the first generation of commercial devices was introduced, the way forward seemed clear. Studies at that time demonstrated that whatever physiological parameter was measured from kidney function to reperfusion bowel injuries, the data demonstrated the clear advantage of EVAR over open surgery<sup>[6]</sup>. In addition, quality of life was improved at 3 months (by SF36), ITU stay was minimal, blood loss improved by up to 200% and in-patient stay reduced to a few days<sup>[7]</sup>. Between 1995 and 1998 there was a proliferation of positive personal cohort studies, all signalling the death knell for open aneurysm repair.

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However, by 1998 the home-made and first-generation commercial devices started to have problems. The term 'endoleak' was invented as the devices began increasingly to demonstrate their limitations (Fig. 1). There were so many of these endoleaks that a classification had to be invented (Types 1-4). Vascular radiologists were spending hours trying to fix them and those that could not be fixed had to be removed at open surgery. In addition we were learning that as aneurysm sacs shrank, the stent grafts dislocated and kinked (Fig. 2). Late rupture following EVAR was becoming recognised. However the second-generation EVAR devices were now being introduced. These were modular, and seemed to have advantages over the first-generation devices. There was a second wave of enthusiasm which, by 2000, was also tempered by the same problems as the first-generation devices. In addition, aneurysm sacs were not shrinking despite the absence of demonstrable leak (a Type 5 endoleak).

However, it was becoming clear that the majority of endoleaks were self-limiting and that 60% spontaneously resolved<sup>[8]</sup>. In addition, third-generation devices were with us, many of which were better designed and constructed and some of which could be fixed above the renal arteries. Data from my own centre and from others suggest that where endoleak survival at 3 years had been less than 70% it is now 94%. Where intervention-free survival had been 64% it is now 88%. Where average percentage change in aneurysm sac size had been less than 4% it is now greater than 20% (Fig. 3)<sup>[9]</sup>. The new devices also allow more aneurysm types to be treated. Registry of endovascular treatment of abdominal aortic aneurysm (RETA) data from 1000 patients, the majority of whom had first- and second-generation devices, suggest that the acute rupture and conversion rate is 3.3%<sup>[10]</sup>.

The EVAR 1 & 2 trials began recruiting in 1999 in the UK. The DREAM (Dutch Randomised Endovascular Aneurysm Management Trial) study is similar in Holland<sup>[11]</sup>. These are nationally funded randomised studies that are recruiting ahead of schedule. EVAR 1 is randomising patients who are fit for surgery to EVAR or open surgery. EVAR 2 is randomising patients who are considered unfit for surgery. Until these studies report it is far too early to consider that EVAR should be abandoned. The practical and physiological advantages of EVAR still stand<sup>[6]</sup>. Fourth-generation devices are now being introduced that result from all the lessons we have learnt over the last 11 years. It should be remembered

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