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Endovascular Repair of Infrarenal Abdominal Aortic Aneurysms in High-Risk-Surgical Patients☆

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Purpose. *Following the publication of a prospective randomized trial (EVAR2) that questioned the benefit of endovascular repair of abdominal aortic aneurysms (AAA) for high-surgical-risk patients, we evaluated our own initial and long-term results with endovascular AAA repair for this patient population.*

Material and methods. *Between January 2000 and December 2005, 115 patients with an AAA managed by an aortic endograft were entered in a registry. Data concerning diagnosis, operative risk, treatment, and follow-up were analyzed on an intention-to-treat basis for all patients considered to be poor candidates for surgery. Patients with a ruptured AAA and those who were good surgical candidates were excluded from analysis. The main goal was evaluation of the operative mortality and the long-term survival of these patients. Secondary goals were determination of the frequency of secondary operations, the outcome of the aneurysm sac, and primary and secondary patency rates after aortic endograft placement.*

Results. *A total of 92 high-surgical-risk patients treated by an endograft were entered in this study. Sixty-seven patients (73%) were classed ASA III and 18 (20%) were ASA IV (20%). Mean aneurysm diameter was 58 mm $\pm$ 9 mm. The technical success rate was 99%. Operative mortality was 4.3% (4 cases). Four patients required re-intervention during the mean follow-up of 18 months. The survival rate at 3 yr was 85%. One type I endoleak (1%) and 9 type II endoleaks (9.7%) occurred during the follow-up period. Primary and secondary patency rates at 3 yr were respectively 96% and 100%.*

Conclusion. *Our initial and long-term results with endograft repair of AAA in high-surgical-risk patients were satisfactory. These results appear to justify endovascular repair for this patient population.*

Keywords: Aneurysm; Abdominal aorta; Endograft; Endovascular; High-surgical-risk patient.

Two randomized prospective studies[1,2] concerning patients who are good surgical risk candidates have reported a significant decrease in immediate morbidity and mortality after endovascular repair of abdominal aortic aneurysms (AAA) compared to conventional open surgery. Nevertheless, the uncertainties concerning the long-term outcome of aortic endografts have prompted several scientific societies to recommend that this approach be reserved for so-called high surgical risk patients. However, recent data on endovascular AAA repair includes both a high frequency of secondary procedures[3–5] and elevated rates of immediate and late morbidity and mortality in high-surgical-risk patients.[5] This study describes our initial and long-term results with endovascular repair of AAA in high-surgical-risk patients.

Material and Methods

Between January 2000 and December 2005, a total of 115 patients with an infrarenal AAA managed at our center by aortic endograft placement were included in a registry. All patients underwent preoperative evaluation consisting of aortography with a graduated pigtail catheter and contrast-enhanced CT with 3–5 mm slices. Data concerning diagnosis, operative risk, treatment, and follow-up were collected prospectively in a registry. Ninety-two of these 115 patients (80%) with a non-ruptured AAA were considered poor candidates for surgery and were included in this study. Patients who underwent emergency repair of a ruptured AAA (10 cases) and good-surgical-risk patients (13 cases) were excluded from analysis. These 92 high-surgical-risk patients were selected for endovascular

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repair after discussion between the surgeons and the anaesthetists of our Department. The decision to include these patients in an endovascular repair protocol was based on the existence of favourable anatomic conditions for aortic endograft placement and the presence of one or more of the following criteria used by the AFSSAPS (Agence Française de Sécurité Sanitaire des Produits de Santé) to define high-surgical-risk candidates:

1. age 80 yr or over
2. coronary artery disease evidenced by a history of myocardial infarction or angina with a positive function test and non-revascularizable coronary artery lesions
3. symptomatic heart failure
4. inoperable tight aortic narrowing
5. left ventricular ejection fraction $< 40\%$ of predicted
6. COPD evidenced by $FEV_1 < 1.2$ l/sec, vital capacity $< 50\%$ of the predicted value as a function of age, sex and weight, arterial $PaCO_2 > 45$ mm Hg or $PaO_2 < 60$ mm Hg, or home oxygen therapy
7. renal failure corresponding to serum creatinine greater than or equal to 200 micromol/l prior to contrast medium injection
8. hostile abdomen owing to the presence of ascites or other signs of portal hypertension.

Graft placement was performed in the operating theatre under general or local-regional anaesthesia using a limited femoral surgical approach or, in certain cases, a percutaneous approach (Prostar® system, Abbott). A bifurcated endograft was used except when the diameter of the distal aortic neck was smaller than 24 mm or when there were calcifications, tortuous iliac arteries or iliac occlusion. In the later cases, a uniiliac endograft was preferred and combined with a crossover bypass and occlusion of the contralateral iliac artery. Postoperative follow-up consisted of clinical examination and follow-up CT 1 mo., 6 mo., 12 mo. and 18 mo. after intervention, then annually if the 18 mo. results were satisfactory. Patients with renal failure were followed up on an alternating basis by Doppler ultrasound and CT. All radiological images were reinterpreted conjointly with a vascular radiologist. The data base of clinical and imaging information thus obtained was reviewed, then completed by analysis of the files of all of the patients included in this series. The main goal of this study was to evaluate the operative mortality and the long-term survival of high surgical risk patients with an infrarenal AAA treated by an aortic endograft. The secondary goals were to determine the primary and secondary patency rates, the frequency of secondary procedures, and the outcome of the aneurysm sac after aortic endograft placement. Operative mortality was defined as any death occurring the first 30 days after endograft placement, regardless of the cause. Secondary interventions included any complementary surgical or endovascular procedure performed immediately after or at a distance from endograft placement in order to maintain a satisfactory clinical result or to treat a specific complication linked to the endograft or the aneurysm. The outcome of the aneurysm sac was evaluated by postoperative measurement of aneurysm diameter during follow-up.

Statistical analysis on an intention-to-treat basis was performed using STAT VIEW software (SAS Institute, Inc. 1992–1998; version 5.0). Measured values were expressed as the mean ± standard deviation, or as ranges. The Kaplan-Meier method was used to calculate the survival and patency rates. A t-test was used to compare the mean duration of hospitalization between the subgroup of patients treated with a bifurcated endograft and those treated by a uniiliac endograft combined with a femoro-femoral crossover bypass. A P value < 0.05 was considered statistically significant.

Results

A total of 92 patients (82 men, 10 women) were considered high-surgical-risk candidates and were included in our study. Mean age was 77 yr (range 53–100). The major comorbidities of the patients in our series are listed in Table 1. Of the 66 patients with recognized severe cardiac disease, 29 had coronary artery disease, 9 had a left ventricular ejection fraction less than 40%, and 20 patients had multiple associated cardiac events (heart failure, arrhythmia, coronary artery disease). Seven patients were classed ASA II (8%), 67 were ASA III (73%), and 18 were ASA IV (20%). Multiple severe co-morbidities were present in all ASA IV patients. Those classified ASA III had at least one major life threatening condition (cardiac disease, renal failure or significant chronic respiratory disease) and other minor problems like hypertension, tobacco abuse, or diabetes. Six of the 7 ASA II patients were elderly

Table 1. Risk factor data for the patients in our series ($N = 92$)

Factor	N	%
Diabetes	11	11.9%
Arterial hypertension	58	63.0%
Smoking	47	51.1%
COPD	38	41.3%
Cardiopathy	66	71.7%
Creatinemia > 200 micromol/l	9	9.8%

COPD: chronic obstructive pulmonary disease.

(between 80 and 100 yr); the last ASA II patient had a hostile abdomen owing to obesity, multiple previous laparotomies, and a colostomy. Table 2 displays how many patients were unfit for open repair according to each single AFSSAPS criteria. For comparative purposes we report how many would be at high risk according to each single EVAR2 criteria[6] (Table 2). Mean body mass index in this population was 25 (range 15−33). Mean aneurysm diameter was 58 mm ± 9 mm. The mean length of the proximal aneurysm neck was 18 mm (range 15−30 mm); the mean neck diameter was 23 mm ± 5 mm. Fourteen patients had highly tortuous or severely calcified iliac arteries. The technical success rate was 99% (91/92 cases). A bifurcated endograft was used in 55 cases (14 of them were placed totally by a bilateral percutaneous approach).

The other 36 patients were managed with an aortic uniiliac endograft combined with a crossover bypass. The sole technical failure concerned one of the 14 patients with severely tortuous iliac arteries and calcification. This patient had already had a crossover bypass for an occlusive left iliac lesion and it was impossible to insert the endograft. Immediate conversion to surgery was successful despite this patient's COPD.

In four other patients, a complementary surgical or endovascular procedure was required to enable endograft placement. Two of these patients underwent percutaneous transluminal iliac angioplasty to facilitate advancement of the endograft delivery system. Another patient required a hepato-renal bypass following accidental injury of the right renal artery during endograft deployment. In the last patient, a bifurcated endograft was converted to a uniiliac endograft combined with a crossover bypass following per-operative endograft limb occlusion. Eight patients underwent prior unilateral hypogastric embolization because the aneurysm extended to the internal iliac arteries. The endografts used included 6 Excluder® (WL Gore), 5 Aneurix® (Medtronic), 14 Talent® (Medtronic), and 66 Zenith® (Cook).

The mean duration of hospitalization was 9 days (range 1−32). However, the hospital stay was longer in the patients managed with an aortic uniiliac endograft plus a femoro-femoral crossover bypass than in those who received a bifurcated endograft: 11 days vs 7 days of hospitalization respectively ($P = 0.0007$). The mean hospital stay of patients treated by a percutaneous approach (14 patients) was 5 days. Mortality at Day 30 was 4.3% (4 patients), corresponding to 1 myocardial infarction, 1 multi-organ failure, 1 colonic ischemia associated with postoperative paraplegia in a patient who already had a thoracic endograft, and 1 iliac rupture (small, tortuous and calcified iliac artery). The four patients who died before the 30th postoperative day were all males over 80 yr of age classed ASA IV who had multiple cardiac problems and COPD. The procedure for these 4 patients were performed under general anaesthesia.

Mean follow-up was 18 mo. Five late deaths occurred during this period: 1 heart failure, 1 rupture of a type I endoleak, 1 deterioration of general status, 1 mesenteric infarction, and 1 case of aspiration pneumonia. Actuarial survival at 3 yr was 85% (Fig. 1). Primary and secondary patency rates at 3 yr were 96% and 100% respectively (Fig. 2). Three patients developed buttock claudication that completely resolved in all cases between 6 and 18 mo. The contralateral internal iliac artery remained patent in these 3 patients who had all undergone hypogastric artery embolization prior to endograft placement. Four patients

Table 2. Repartition of our patients who would be unfit for open repair according to each AFSSAPS and each EVAR2 criteria ($N = 92$)

Function	AFSSAPS Criteria	N_1*	EVAR2 Criteria[6]	N_2†
Age	>80 yrs	38	No	0
Local factors	Hostile abdomen	1	No	0
Cardiac	CAD‡	29	?? (local decision§)	?
	Heart failure**	24	Heart failure††	24
	Inoperable tight aortic stenosis	2	Severe valve disease	2
Renal	Serum creatinine > 200mcmoles/l	9	Serum creatinine > 200mcmoles/l	9
Respiratory	FEV1 < 1.2 l/sec		FEV1 < 1.0 l/s	
	Vital capacity < 50%		−	
	$PaCO_2 > 45$ mm Hg	38	$PaCO_2 > 6.5$ KPa	38
	Pa $O_2 < 60$ mm Hg		Pa $O_2 < 8.0$ KPa	
	Home oxygen therapy		Short of breath after a flight of stairs	

* N_1: Number of our patients deemed unfit for open conventional repair according to each AFSSAPS Criteria.
† N_2: Number of our patients who could be deemed unfit for open conventional repair according to each EVAR2 criteria.[6]
‡ CAD (coronary artery disease) as evidenced by: prior myocardial infarction, or angor with positive function test and non-revascularizable coronaropathy.
§ decision about patient fitness and enrolment into EVAR1 or EVAR2 were dedicated to local surgeon, anaesthetist and radiologist.
** Heart failure: patent manifestations or ejection fraction <40%.
†† Uncontrolled congestive heart failure.

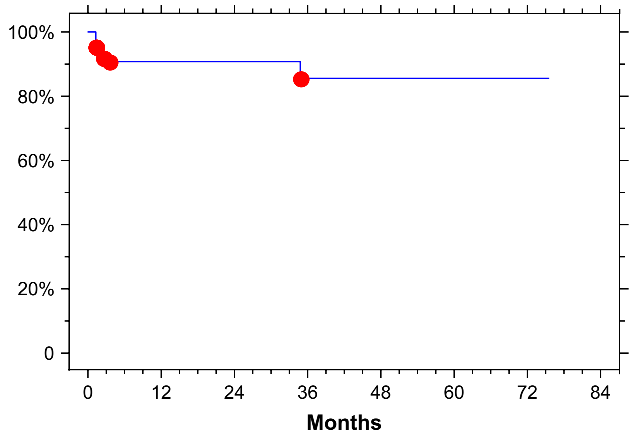

Fig. 1. Kaplan-Meier survival curve.

(4.3%) required repeat surgery during follow-up: 3 repeat endovascular repairs and one surgical repair. The reasons for these repeat procedures are listed in Table 3. During follow-up, one Type I endoleak (1%) resulted in a fatal rupture. There were 9 type II endoleaks (9.7%): 3 healed spontaneously, 5 persisted with a stable or regressive aneurysm diameter, and 1 endoleak associated with an increase in the size of the aneurysm sac was successfully embolized (Table 3). The aneurysm sac decreased in size by 2 to 30 mm in 33 patients in this series (36%). In the remaining patients, the aneurysm sac remained stable during the entire follow-up period (Fig. 3).

Discussion

Operative mortality in our series of high-surgical-risk patients who underwent endovascular AAA repair was 4.3%. The frequency of secondary procedures during the mean follow-up period of 18 mo. was 4.3%. Survival at 3 yr was 85%, with a significant reduction in the size of the aneurysm sac in over one third of cases. Only one of the 5 deaths that occurred during long term follow-up was related to the aneurysm and was due to a specific complication of endografts (type I endoleak). Long-term morbidity was dominated by the development of type II endoleaks (9.7% of cases) and thrombosis or stenosis of the endograft limb (2.1% of cases). Type II endoleaks all had a benign course and usually required only surveillance.

The limitations of our study are those of a retrospective analysis of data collected in a registry without prior definition of a control population. Nevertheless, no patient in this series was lost to follow-up and their clinical characteristics are comparable to those of most other studies published on the subject.

Previous studies suggest that endovascular AAA repair may be a valuable alternative to open repair for patients with severe comorbidities.[7–12] Published operative mortality rates vary between 0 and 5.3% for high surgical risk patients. The EVAR2[5] randomized prospective study comparing surveillance and endovascular repair of AAA in patients for whom open repair was contraindicated nevertheless reported a mortality rate on Day 30 of 9% in the endovascular group. This is more than twice the mortality in our series. Likewise, long-term survival in EVAR2[5] (34% at 4 yr) was markedly lower than the long-term survival of 85% at 3 yr in our study. One might argue that better results were achieved in our study because of more lenient inclusion criteria, especially 8% of our patients were ASA class II patients. However, most of our patients would have probably qualified as EVAR2 patients (Table 2). Since the design of both EVAR1 and EVAR2 trials, it has become clear that there are difficulties in using strict ASA classification for defining risk among vascular surgery patients.[6] For both trials, decisions about patient fitness were made by local surgeons, anaesthetists and radiologists.[6] ASA 2 patients are frequently not high risk for open AAA repair, but in our study the ASA 2 patients (7 cases) were octogenarians (6 cases) or had a hostile abdomen (1 case). Nevertheless, advanced age as a risk factor for aortic cross-clamping surgery has remained a controversial issue.[13] Haug *et al.*, in a recently published study, reported an operative mortality rate of 11% for octogenarians who underwent conventional AAA repair.[14] In a post-hoc analysis of the low risk DREAM trial population, Baas *et al.* have found a 2.2 fold increased risk of death for patients older than 70 years who underwent open aortic repair as compared to patients younger than 70 years (6.3% vs. 3.2% respectively).[15] A possible explanation is that elderly patients, because of limited daily activity, could have unnoticed cardiac

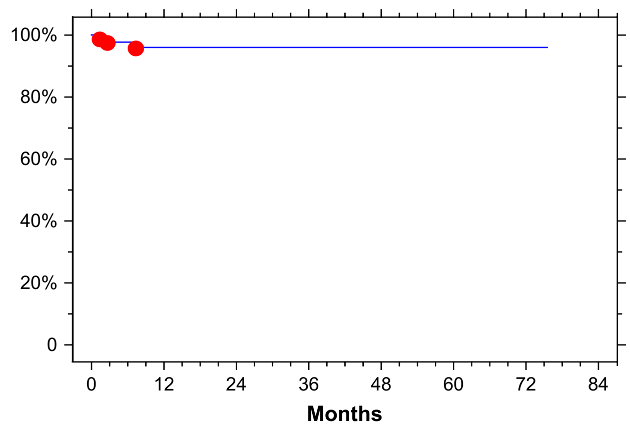

Fig. 2. Kaplan-Meier actuarial curve of primary patency.

Table 3. Secondary procedures

Event	Moment of diagnosis	Treatment	Moment of treatment	Result
Type II endoleak	6 mo.	Ilio-lumbar embolization	36 mo.	Good Follow-up 3 mo.
Type I endoleak (rupture)	2 mo.	Surgical conversion	2 mo.	Death
Endograft limb thrombosis	Day 6	Thrombectomy	Day 6	Blue toe syndrome
Endograft stenosis	2 mo.	Percutaneous transluminal angioplasty	2 mo.	Good Follow-up 9 mo.

or respiratory morbidities prone to decompensate by a surgical insult. Conversely, octogenarians with AAA should be considered as high risk patients and be offered the less invasive surgical approach even without previous cardiac and respiratory problems.

Moreover, other studies[8–11] including greater numbers of patients (Table 4) corroborate our favourable results obtained with EVAR in patients unfit for conventional repair. In the EUROSTAR[11] register, the survival rate at 3 yr was 68% for the group of high-risk patients who were refused surgery. Sicard *et al.*[8] reported a mean survival rate of 56% at 4 yr. Differences in outcome noted in these registries (Table 4), though minor, might reflect the lack of universal, homogenous, and easy-applicable guidelines for defining high risk populations. With the exception of those patients with a limited life expectancy for whom the risk of death at 1 yr due to co-morbidities greatly exceeds the risk of aneurysm rupture, we and most authors[8–12] advocate endovascular repair of AAA for high-surgical-risk patients, in contrast to the EVAR2.[5] Factors including the therapeutic approach, anatomic features, and statistical bias may explain some of the discrepancies observed as compared to EVAR2. For example, medical treatment, a factor that has been proven to influence patient survival,[16,17] was not optimal in nearly half of the patients in the EVAR2[5] study. Aside from aneurysm

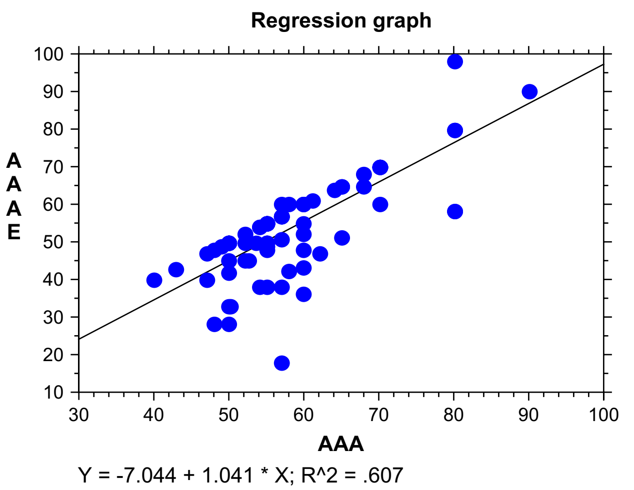

Fig. 3. Evolution of aneurysm diameter after aortic endograft placement.

diameter, EVAR2[5] did not report any anatomic characteristics. Mean diameter in EVAR2 was 6.7 cm versus 5.8 cm in our study. However, several literature studies[18–23] suggest that anatomic conditions worsen as the aneurysm enlarges. Large aneurysm diameter, like a short, calcified and/or highly angulated neck, are predictive factors of aortic endograft complications, both immediately and at long term.[22,24–28] Strict respect of anatomic feasibility conditions for aortic endografts and a smaller mean aneurysm diameter in our series compared to EVAR2 may explain our good results. Clinical and statistical factors might also explain the results in EVAR2.[5] Nine of the 20 deaths recorded in the EVAR2 endovascular group (i.e. 45% of the mortality in the group) were due to aneurysm rupture in patients who were waiting for an endograft. The mean interval between randomization and treatment (mean 57 days, maximum 767 days) was indeed very long for patients whose AAA measured a mean diameter of 6.7 cm. Furthermore, 47 patients of the EVAR2[5] surveillance group (27%) were operated on in violation of the study protocol, although curiously the mortality rate was only 2.1%, which is similar to our results for this type of patient.

Another major problem linked to endovascular AAA repair is the frequency of secondary procedures required to maintain the immediate results and the resultant increase in the cost of patient management. Endoleaks are the main cause of repeat intervention after placement of an aortic endograft.[29] In certain series, 27–35% of the patients treated with an aortic endograft required a secondary procedure at some point during the first seven years of follow-up.[3,4] In the EVAR2 study, 43% of the endovascular group patients had at least one postoperative complication and 26% of the patients required a secondary procedure after

Table 4. Literature review

Study	*N*	Mean age (yr)	Mean aneurysm diameter (mm)	Deaths at 30 days	Survival
EUROSTAR[11]	550	72.6	58.3	4.8–5.3%	68% (at 3 yr)
SVS[8]	565	76.6	64	2.9%	56% (at 4 yr)
EVAR2[5]	166	76.8	64	9%	34% (at 4 yr)
This series	92	77	58	4.3%	85% (at 3 yr)

4 yr of follow-up.[5] These repeat procedures account for a good percentage of the financial costs linked to this group.[5] However, several other studies have reported surgical or endovascular reintervention rates varying from 2 to 13%.[18,30,31] These figures agree with the 4.3% reintervention rate in our series. Improvement of the endografts on the market may explain the more favourable results in the most recent series.[28] However, the disparities in reintervention rates might be due to differences in the diagnostic attitude as well as to differences in therapeutic choices. Indeed, up to 19% of all type I endoleaks and 49% of all type II endoleaks have been reported following systematic arteriography after 1 year of survival.[32] The rate of CT-detected endoleaks varies from 4% to 20%, depending on the series.[9,30,31] Henao and coll.[33] emphasized the role of contrast-enhanced Doppler ultrasound for the diagnosis of endoleaks. In EVAR2, the authors reported 5.6% type I endoleaks and 9.5% type II endoleaks. While type I endoleaks justify an aggressive approach because they can result in rupture in up to 21% of cases in less than 30 days,[26] type II endoleaks with an aneurysm sac that remains stable or regresses in size can be treated conservatively.[34,35]

Endograft limb thrombosis and stenosis are less common complications of endovascular AAA repair. In our series, as in EVAR2 and the series of Allaqaband and coll.,[10] 2−4% of patients developed this type complication that always prompted a repeat surgical or endovascular procedure. According to a recent study, placement of an additional bare metal stent in the endograft limb during the initial procedure might improve long-term patency by reducing the risk of thrombosis.[36]

Conclusion

Our initial and long-term results with endografts for the treatment of AAA in high-surgical-risk patients were satisfactory as the operative mortality was 4.3%, survival at 3 yr was 85%, and the secondary procedure rate was only 4.3%. In our opinion, these results justify endovascular repair for this group of patients. The most important factor for improvement of the short and long-term results of aortic endografts is respect of anatomic feasibility conditions.

References

1 EVAR1 trial participants. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm: randomised controlled trial. *Lancet* 2005;**365**:2179−2186.

2 DREAM trial participants. A randomized trial comparing conventional and endovascular repair of abdominal aortic aneurysms. *N Engl J Med* 2004;**351**:1607−1618.

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

4 Becquemin JP, Kelley L, Zubilewicz T, Desgranges P, Lapeyre M, Kobeiter H. Outcomes of secondary interventions after abdominal aortic aneurysm endovascular repair. *J Vasc Surg* 2004; **39**:298−305.

5 EVAR trial participants. Endovascular aneurysm repair and outcome in patients unfit for open repair of abdominal aortic aneurysm (EVAR trial 2): a randomised controlled trial. *Lancet* 2005; **365**:2187−2192.

6 Brown LC, Epstein D, Manca A, Beard JD, Powell JT, Greenhalgh MR. The UK endovascular aneurysm repair (EVAR) trials: design, methodology and progress. *Eur J Vasc Endovasc* 2004;**27**:372−381.

7 Iannelli G, Monaco M, Di Tomasso L, Piscione F, Stassano P, Mainenti PP *et al*. Endovascular vs open surgery of abdominal aortic aneurysm in high risk patients: a single center experience. *Thorac Cardiovasc Surg* 2005;**53**:291−294.

8 Sicard GA, Zwolak RM, Sidawy AN, White RA, Siami FS. Society for Vascular Surgery Outcomes Committee. Endovascular abdominal aortic aneurysm repair: long-term outcome measures in patients at high-risk for open surgery. *J Vasc Surg* 2006;**44**: 229−236.

9 Sukhija R, Aronow WS, Mathew J, Yalamanchili K, Kakar P, Frishman WH *et al*. Treatment of abdominal aortic aneurysms with an endovascular stent-graft prosthesis in 96 high-risk patients. *Cardiol Rev* 2005;**13**:165−166.

10 Allaqaband S, Mortada ME, Tumuluri R, Bajwa TK. Endovascular stent-graft repair of abdominal aortic aneurysms in high-risk patients; a single center experience. *J Interv Cardiol* 2004;**17**: 71−79.

11 Buth J, Van Marrewijk CJ, Harris PL, Hop WC, Riambau V, Laheij RJ. EUROSTAR collaborators. Outcome of endovascular abdominal aortic aneurysm repair in patients with conditions considered unfit for an open procedure: a report on the EUROSTAR experience. *J Vasc Surg* 2002;**35**:211−221.

12 Sbarigia E, Speziale F, Ducasse E, Giannoni MF, Ruggiero M, Palmieri A *et al*. What is the best management for abdominal aortic aneurysm in patients at high surgical risk? A single-center review. *Int Angiol* 2005;**24**:70−74.

13 Rosset E, Rovani G, Aublet-Cuvellier B, Lietin B, Tran X, Halden MA *et al*. Surgical treatment of infrarenal aortic aneurysms in octogenarians. In: Branchereau A, Jacobs M, eds. *Vascular Procedures in the Elderly Patient*. Oxford: Paris consultants Ltd; 2006:83−93.

14 Haug ES, Romundstad P, Aune S, Hayes TBJ, Myhre HO. Elective open operation for abdominal aortic aneurysm in octogenarians. Survival analysis of 105 patients. *Eur J Vasc Endovasc Surg* 2005; **29**:489−495.

15 Baas A, Jong SD, Prinssen M, Buskens E, Grobbee D, Blankensteijn J. The benefit of endovascular aneurysm repair: the role of age. In: Branchereau A, Jacobs M, eds. *Vascular Procedures in the Elderly Patient*. Oxford: Paris consultants Ltd; 2006:95−104.

16 Kertai MD, Boersma E, Westerhout CM, Klein J, Van Urk H, Bax JJ *et al*. A combination of statins and beta-blockers is independently associated with reduction in the incidence of perioperative mortality and non-fatal myocardial infarction in patients undergoing abdominal aortic aneurysm surgery. *Eur J Vasc Endovasc Surg* 2004;**28**:343−352.

17 Kertai MD, Boersma E, Westerhout CM, van Domburg R, Klein J, Bax JJ *et al*. Association between long-term statin use and mortality after successful abdominal aortic aneurysm surgery. *Am J Med* 2004;**116**:96−103.

18 Carpenter JP, Baum RA, Barker CF, Golden MA, Mitchell ME, Velazquez OC *et al*. Impact of exclusion criteria on patient

selection for endovascular aortic aneurysm repair. *J Vasc Surg* 2001;**34**:1050−1054.
19 Greenberg R, Fairman R, Srivastava S, Criado F, Green R. Endovascular grafting in patients with short proximal necks: an analysis of short term results. *Cardiovasc Surg* 2000;**8**:350−354.
20 Resch T, Ivancev K, Lindh M, Nirhov V, Nyman U, Lindblad B. Abdominal aortic aneurysm morphology in candidates for endovascular repair evaluated with spiral computed tomography and digital subtraction angiography. *J Endovasc Surg* 1999; **6**:227−232.
21 Amon MP, Yusuf SW, Whitaken SC, Gregson RH, Whenam PW, Hopkinson BR. Influence of abdominal aortic aneurysm size on the feasibility of endovascular repair. *J Endovasc Surg* 1997;**4**: 279−283.
22 Peppelenbosch N, Buth J, Harris PL, van Marrewijk C, Fransur G. For the EUROSTAR collaborators. Diameter of abdominal aortic aneurysm and outcome of endovascular aneurysm repair: does size matter? A report from EUROSTAR. *J Vasc Surg* 2004;**39**:288−297.
23 Sampram ESK, Karafa MT, Mascha EJ, Clair DG, Greenberg RK, Lyden SP *et al*. Nature, frequency and predictors of secondary procedures after endovascular repair of abdominal aortic aneurysm. *J Vasc Surg* 2003;**37**:930−937.
24 Rockman C. Reducing complications by better case selection: anatomic considerations. *Semin Vasc Surg* 2004;**17**:298−306.
25 Waasdorp EJ, de Vries JP, Hobo R, Leurs LJ, Buth J, Moll FL. EUROSTAR Collaborators. Aneurysm diameter and proximal aortic neck diameter influence clinical outcome of endovascular abdominal aortic repair: a 4-year EUROSTAR experience. *Ann Vasc Surg* 2005;**19**:755−761.
26 Thomas SM, Beard JD, Ireland M, Ayers S. Vascular Society of Great Britain and Ireland; British Society of Interventional Radiology. Results from the prospective registry of endovascular treatment of abdominal aortic aneurysms (RETA): mid term results to five years. *Eur J Vasc Endovasc Surg* 2005;**29**:563−570.
27 Sampaio SM, Panneton JM, Mozes GI, Andrews JC, Bower TC, Karla M *et al*. Proximal type I endoleak after endovascular abdominal aortic aneurysm repair: predictive factors. *Ann Vasc Surg* 2004;**18**:621−628.
28 Hobo R, Buth J. EUROSTAR collaborators. Secondary interventions following endovascular abdominal aortic aneurysm repair using current endografts. A EUROSTAR report. *J Vasc Surg* 2006; **43**:896−911.
29 Carpenter JP, Baum RA, Barker CF, Golden MA, Velasquez OC, Mitchell MF *et al*. Durability of benefits of endovascular versus conventional abdominal aortic aneurysm repair. *J Vasc Surg* 2002; **35**:222−228.
30 Saratzis N, Melas N, Larzaridis J, Ginis G, Antonitsis P, Lykopoulos D *et al*. Endovascular AAA repair with the aortomonoiliac Endofit stent graft: two years' experience. *J Endovasc Ther* 2005;**12**:280−287.
31 Biebl M, Hakaim AG, Oldenburg WA, Lau LL, Klocker J, Neuhauser B *et al*. Midterm results of a single-center experience with commercially available devices for endovascular aneurysm repair. *Mt Sinai J Med* 2005;**72**:127−135.
32 Haulon S, Devos P, Willoteaux S, Mounier-Vehier C, Sokoloff A, Halna P *et al*. Risk factors of early and late complications in patients undergoing endovascular aneurysm repair. *Eur J Vasc Endovasc Surg* 2003;**25**:118−124.
33 Henao EA, Hodge MD, Felkai DD, McCollum CH, Noon GP, Lin PH *et al*. Contrast-enhanced duplex surveillance after endovascular abdominal aortic aneurysm repair: improved efficacy using a continuous infusion technique. *J Vasc Surg* 2006;**43**:259−264.
34 Sheehan MK, Ouriel K, Greenberg R, McCann R, Murphy M, Fillinger M *et al*. Are type II endoleaks after endovascular aneurysm repair endograft dependent? *J Vasc Surg* 2006;**43**: 657−661.
35 Tolia AJ, Landis R, Lamparello P, Rosen R, Macari M. Type II endoleaks after endovascular repair of abdominal aortic aneurysms: natural history. *Radiology* 2005;**235**:683−686.
36 Sivamurthy N, Schneider DB, Reilly LM, Rapp JH, Skovobogatyy H, Chuter TA. Adjunctive primary stenting of Zenith endograft limbs during endovascular abdominal aortic aneurysm repair: implications for limb patency. *J Vasc Surg* 2006;**43**:662−670.

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