10

DESCENDING THORACIC AORTIC ANEURYSMS: OPEN REPAIR OR STENT-GRAFTING?

EDOUARD KIEFFER, LAURENT CHICHE

The first endovascular repair of descending thoracic aortic aneurysm (DTAA) by Dake et al. [1] was certainly a major milestone in the management of such lesions. Some investigators, most of whom have no expertise in surgery of the thoracic aorta, have expanded indications to the point of recommending endovascular repair as the treatment of choice for DTAA [2-8]. Other investigators report excellent results with open surgical repair and see apparently no indications at all for endovascular repair [9-11]. In our department we have adopted a middle ground. We began performing endovascular repair of DTAA in 1997. Up to that date, all DTAA except those in patients presenting absolute operative contra-indications related to their general condition were treated via thoracotomy with circulatory assistance, indicating partial cardiopulmonary bypass in most cases. Since 1997, we have been performing endovascular repair in poor surgical risk candidates provided that spinal cord arteriography demonstrated that the origin of the Adamkiewicz was located outside of the zone to be covered by the stent-graft. This cautious attitude is based on the absence of long-term results in most reported series of endovascular repair for DTAA, as well as the relatively disappointing outcome described in the only series with a sufficient follow-up period [12]. The purpose of this study is to assess the results of our strategy by analyzing postoperative mortality and morbidity in function of treatment technique used [13].

Methods

From July 1997 to May 2005, a total of 173 consecutive patients underwent elective treatment of DTAA in the Department of Vascular Surgery of the Pitié-Salpêtrière University Hospital. Patients were divided into two groups according to treatment technique: group I including 52 patients (30.1%) who underwent stent-graft repair and group II including 121 patients (69.9%) who underwent open surgical repair.

Data on patient population, aneurysmal disease, surgical technique and treatment outcome were collected prospectively and analyzed retrospectively. Demographic data involved patient age (mean, range, median and percentage 70 years or over) and comorbidities (hypertension, coronary artery disease, chronic obstructive pulmonary disease, chronic renal insufficiency, diabetes, cerebrovascular insufficiency and concomitant infrarenal abdominal aortic aneurysm).

The etiology of aneurysmal disease was classified into three groups: degenerative aneurysm, aneurysm related to chronic dissection (including intramural hematoma and penetrating ulcer) and aneurysm due to other causes including post-traumatic, dystrophic, inflammatory, infected, congenital and iatrogenic aneurysms.

Aneurysm topography was assessed by panaortography and CT-scanning. Lesions were classified using a personal system [14] into the following four categories (Fig. 1).

- Type I: aneurysms located in the proximal part of the descending thoracic aorta with the lower limit not extending past the midpoint. This type of lesion generally requires clamping of the aortic arch between the left common carotid and left subclavian artery if open repair is performed, and transposition of the left subclavian artery into the left common carotid artery if endovascular repair is performed.

- Type II: aneurysms located in the middle part of the descending throracic aorta. This type of lesion is technically the most favorable indication for both open and endovascular repair.

- Type III: aneurysm located in the distal part of the descending thoracic aorta with the upper limit not extending above the midpoint. Surgical exposure of this type of lesion is often difficult and may require control of the aorta flush with the celiac axis or between the superior mesenteric artery and celiac axis. The origin of the Adamkiewicz artery is often located within the limits of the lesion. In case of endovascular repair, there is a risk of coverage of the Adamkiewicz artery and celiac axis.

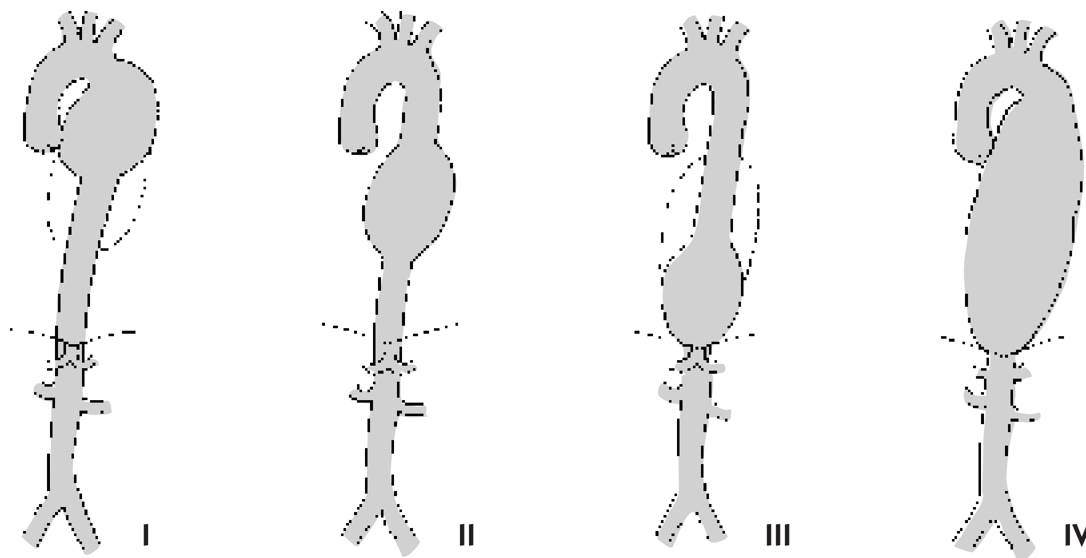

FIG. 1 Classification system for descending thoracic aortic aneurysm [14].

- Type IV: aneurysm involving the full length of the descending thoracic aorta. This type of lesion combines the problems of both type I and type III.

Pre-operative spinal cord arteriography [15] was carried out in all patients except those presenting strictly isthmic aneurysms or, more rarely, those presenting contra-indications for selective catheterization of the intercostal or lumbar arteries (aneurysm presenting with spinal cord ischemia or atheromatous emboli). The results of spinal cord arteriography were classified into four categories [16] depending on the location of the Adamkiewicz artery in relation to the zone between the aortic clamps or zone to be covered by the endograft (Fig. 2):

- group A: Adamkiewicz artery located above the surgical zone;
- group B: Adamkiewicz artery located below the surgical zone;
- group C: Adamkiewicz artery located within the surgical zone;
- group D: failure to visualize the Adamkiewicz artery.

Conventional surgical repair was usually performed by a strictly thoracic approach but the thoracoabdominal route or both techniques was used in some cases. A technique to protect against the effects of aortic clamping was used in all cases. Deep hypothermic circulatory arrest [17] was used in patients either presenting anatomical indications, including large aneurysms not allowing control of the proximal aorta, injury of the aorta or pulmonary artery during control, retrograde dissection of the posterior part of the aortic arch and atheroma or dissection raising risk for aortic clamping, or presenting risk for spinal cord ischemia because of failure to visualize the Adamkiewicz artery. In all other cases partial cardiopulmonary bypass, usually femoro-femoral, was used [18]. Cerebrospinal fluid (CSF) drainage was performed in all patients so as to maintain CSF pressure at 10mmHg or lower for at least 48 hours.

Stent-grafts were usually introduced by the femoral route but the iliac or aortic route was used in some cases. A 24 F introducer was used in most cases. Until 2003 we used custom-made stent-grafts. Since 2003 we have been using *TAG*® thoracic endoprosthesis *(W.L. Gore and Associates, Inc.)*.

Postoperative outcome variables included in-hospital mortality, spinal cord ischemia, central neurological deficits, hemorrhage, cardiac manifestations, pulmonary complications and renal insufficiency. Manifestations related to spinal cord ischemia including not only paraplegia but also partial and/or unilateral deficits and isolated sphincter manifestations were taken into account regardless of whether onset occurred immediately or secondarily after the repair procedure. Central neurological deficits

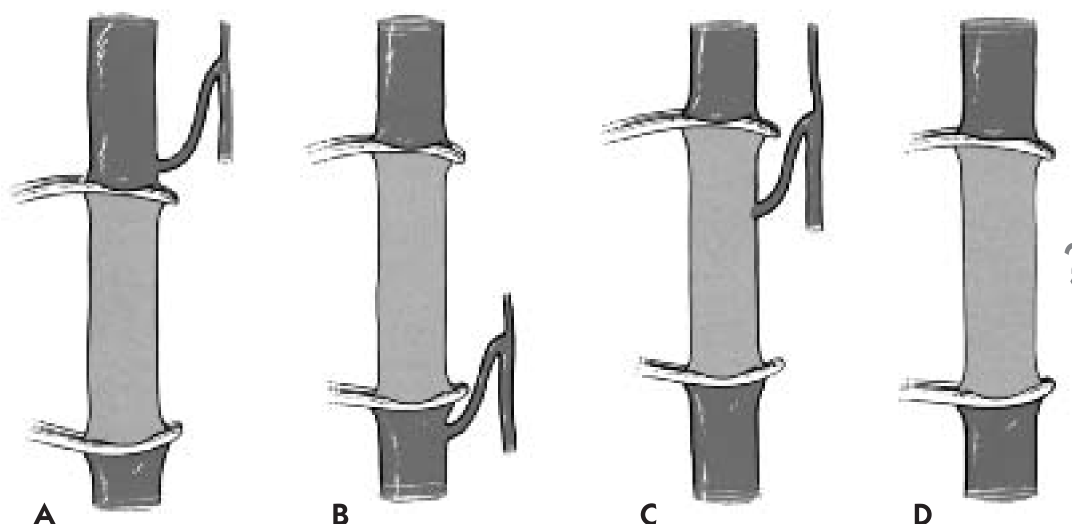

FIG. 2 Classification system for the results of preoperative spinal cord arteriography [16].

included hemispheric ischemic, vertebrobasilar ischemic and hemorrhagic complications. Local hemorrhagic complications were defined as any postoperative bleeding requiring reoperation for hemostasis. Cardiac manifestations included myocardial infarction with or without Q wave, isolated augmentation of Ic troponine level, cardiac insufficiency and supraventricular or ventricular arrhythmia. Pulmonary complications included pneumonia, mechanical ventilation for more than 48 hours or need for reintubation or tracheostomy. Renal insufficiency was defined as an increase in baseline creatinine level greater than 50% or need for hemofiltration or for permanent or temporary hemodialysis.

Statistical analysis was performed by univariate comparison using a chi-square or exact test as appropriate. Probability values less than 0.05 were considered as significant.

Results

Pre-Operative Data

Patient data (sex, age, and co-morbidity), aneurysm characteristics (etiology and anatomy) and findings of pre-operative spinal cord arteriography are presented according to group in Tables I, II and III, respectively. There was a significant difference between groups I and II with regard to age, prevalence of chronic obstructive pulmonary disease, number of type I or type IV aneurysms, number of cases in which spinal cord arteriography was deemed unnecessary and number of patients in whom treatment included a risk of spinal cord ischemia linked to temporary clamping or definitive exclusion of the zone including the origin of the Adamkiewicz artery (group C). Conversely there was no difference between the two groups with

Table I DEMOGRAPHICS AND COMORBIDITIES

	Group 1 (n = 52)		*Group 2 (n = 121)*		*p-value*
	N	*%*	*N*	*%*	
Age (mean ± SD)	69.3 ± 12.5		59.4 ± 13.7		< 0.0001
Age (range)	35 - 89		11 - 82		
Age (median)	72		62		
Age ≥ 71 years	32	61.5	27	22.3	< 0.0001
Male	41	78.8	100	82.6	0.55
Female	11	21.2	21	17.4	
Coronary artery disease	18	34.6	30	24.8	0.18
Myocardial revascularization	6	11.5	20	16.5	0.40
Hypertension	37	71.2	94	77.7	0.36
COPD	30	57.7	42	34.7	0.005
Renal insufficiency*	13	25.0	25	20.7	0.53
Diabetes	3	5.8	5	4.1	0.64
Cerebrovascular insufficiency	15	28.8	20	16.5	0.06
AAA	18	34.6	29	24.0	0.15

* Serum creatinine > 120 µmol/l
AAA: abdominal aortic aneurysm
COPD: chronic obstructive pulmonary disease
SD: standard deviation

regard to the prevalence of hypertension, coronary artery disease, chronic renal insufficiency, diabetes, cerebrovascular insufficiency or concomitant infrarenal abdominal aortic aneurysm. There was also

Table II ANEURYSM CHARACTERISTICS

	Group 1 (n = 52) N	%	*Group 2 (n = 121)* N	%	*p - value*
Etiology					
Degenerative	29	55.8	65	53.7	0.80
Chronic dissection*	12	23.1	32	26.5	0.64
Others	11	21.1	24	19.8	0.84
Anatomy**					
Type I	26	50.0	40	33.1	0.04
Type II	9	17.3	16	13.2	0.48
Type III	10	19.2	24	19.8	0.92
Type IV	7	13.5	41	33.9	0.0006

* Includes intramural hematomas and penetrating ulcers
** Personal classification system [14]

Table III PRE-OPERATIVE SPINAL CORD ARTERIOGRAPHY*

	Group 1 (n = 52) N	%	*Group 2 (n = 121)* N	%	*p - value*
Not performed	25	48.1	18	14.9	< 0.0001
Group A	2	3.9	6	4.9	0.75
Group B	13	25.0	22	18.2	0.30
Group C	6	11.5	70	57.9	0.0001
Group D	6	11.5	5	4.1	0.07

* Personal classification system according to the location of the Adamkiewicz artery [16]

no significant difference regarding etiology, number of patients with type II or type III aneurysm and number of patients in whom the Adamkiewicz artery was located above (group A) or below (group B) the surgical zone or failed to be visualized.

Intra-Operative Data

The 52 endovascular stent-grafts (group I) were introduced by the femoral route in 37 patients (71%), iliac route in 8 patients (15%) or aortic route using an accessory prosthetic graft in 7 patients (14%) who underwent simultaneous treatment of an abdominal aortic aneurysm. To obtain an adequate landing zone in 24 patients (46%), it was necessary to perform either left subclavian/carotid artery transposition or revascularization of the left common carotid and left subclavian arteries from the right common carotid artery by cervical exposure.

Conventional surgical management in 121 patients (group II) was carried out with partial cardiopulmonary bypass in 78 cases (64.5%) and deep hypothermic circulatory arrest in 43 cases (35.5%). The most frequent surgical procedure was resection and grafting, applied in 108 patients (89.3%). Resection with end-to-end anastomosis was feasible in 6 patients (4.9%) with post-traumatic aneurysm of the aortic isthmus. Prosthetic patch angioplasty was performed in 7 patients (5.8%) presenting saccular aneurysms. Intercostal arteries were reimplanted or preserved in 63 patients (58.3%) that underwent resection and grafting.

Postoperative Mortality

In-hospital mortality in the overall series was 8.1% (14/173). A statistically significant difference ($p = 0.02$) was found between mortality after endovascular stent-graft repair (group I) and open surgical repair (group II): 15.4% (8/52) versus 5.0% (6/121). Three deaths after endovascular repair (group I) were due to factors related to the patient's general condition, being heart disease in two patients (day 2 in both) and multi-organ failure in one patient (day 5). The other 5 deaths after endovascular repair were attributed to technical causes, including perforation of the left ventricle (intra-operative), perforation of the aorta (day 4), embolic stroke (day 8), kinking of the stent-graft (day 17) and retrograde dissection of the ascending aorta (day 21). All deaths after conventional surgical management (group II) were due to factors related to the patient's general condition, being multi-organ failure in four patients (days 2, 4, 8 and 72), heart failure in one patient (day 2) and respiratory insufficiency in one patient (day 21).

Postoperative Spinal Cord Ischemia

The spinal cord ischemia complication rate in the overall series was 5.2% (9/173). All complications (9/121, 7.4%) occurred after open surgical repair (group II). In 3 cases (2.8%) the complication consisted of immediate irreversible flaccid paraplegia that led to death in one patient. The remaining 6 complications (4.6%) involved partial deficits or isolated sphincter disturbances. The onset of these complications was delayed in 3 cases. In all 6 cases symptoms disappeared or regressed within weeks or months after the procedure. No spinal cord ischemia complications (0/52) occurred after endovascular stent-graft repair (group I). The difference between the two groups was statistically significant ($p = 0.04$).

Other Postoperative Complications

Table IV lists all fatal and non-fatal neurological, cardiac, respiratory, renal, digestive and hemorrhagic complications observed as well as the conversion rate for patients treated endovascularly. Patients in group I had significantly less respiratory ($p = 0.002$) and renal ($p = 0.05$) complications but significantly more strokes ($p = 0.01$).

Discussion

Introduction of endovascular technology into the therapeutic armamentarium has revolutionized treatment of DTAA. However, although many studies reporting endovascular repair have been published, these series have not addressed the respective roles of open and endovascular repair. To our knowledge this is the first study to analyze the breadth of that revolution in a large single-center series in which indications for endovascular repair and open surgical repair were decided based on consistent selection criteria. A previous report by Glade et al. [19] described a multicenter series with a smaller patient population selected based on criteria that changed during the course of study.

In our series postoperative in-hospital mortality following open surgical repair and endovascular repair was significantly different: 5% versus 15.4% respectively ($p = 0.02$). However, it should be emphasized that endovascular treatment was used exclusively in poor surgical risk patients. This find-

ing is consistent with previous studies showing lower mortality ranging from 1.5% to 9% [2-8] after elective endovascular repair in good surgical risk patients. However, it should be stressed that 5 of the 8 deaths after endovascular repair were due to technical causes that could probably have been avoided, at least in part, with better deployment skill and improvement in endovascular technology.

No postoperative spinal cord ischemia complications were observed after endovascular repair in this series. The most likely explanation is that we tried to limit the use of stent-grafts to patients at low risk for spinal cord ischemia (type I aneurysms) and avoided patients at high risk (group C aneurysms) insofar as possible. Neurological complication rates reported in major series describing stent-grafting of the descending thoracic aorta in the literature [2-8,20-22] have varied from 3% to 6% but have never been nil in sufficiently important series.

The underlying mechanisms, clinical manifestations and outcome of spinal cord ischemia are different after endovascular repair and open surgical repair. Since it is not required, cross-clamping and subsequent spinal cord reperfusion are not causative factors for ischemia after endovascular repair. The main causes of spinal cord ischemia after endovascular repair are thrombo-embolism [20] and, most commonly, permanent obstruction of the arteries supplying the spinal cord (especially the Adamkiewicz artery). However, electromagnetic

Table IV POSTOPERATIVE COMPLICATIONS

	Group 1 (n = 52)		*Group 2 (n = 121)*		*p-value*
	N	*%*	*N*	*%*	
In-hospital mortality	8	15.4	6	5.0	0.02
Paraplegia/Paraparesis	0	-	9	7.4	0.04
Stroke	8	15.4	5	4.1	0.01
Cardiac complications					
*Myocardial infarction**	3	5.8	8	6.6	0.83
Rhythm disturbances	3	5.8	17	14.0	0.11
All cardiac complications	6	11.6	25	20.6	0.15
Respiratory complications	15	28.8	69	57.0	0.002
Renal complications					
Hemodialysis	0	-	9	7.4	0.04
No hemodialysis	4	7.7	15	12.4	0.36
All renal complications	4	7.7	24	19.8	0.05
Digestive complications	1	1.9	10	8.3	0.11
Hemorrhagic complications**	0	-	3	2.5	0.25
Conversions	2	3.8	0	-	0.03

* Based on abnormal troponin I values
** Reoperations for hemostasis

sensor measurements showing persistent pressure after endovascular repair of DTAA [23] proves that circulation continues inside the aneurysmal sac for several weeks. Ongoing circulation is probably due to small type II endoleaks that occlude only gradually thus allowing time for collateral circulation to develop. Open surgical repair rules out such development since the intercostal or lumbar artery giving rise to the origin of the Adamkiewicz is sutured. Endoleak followed by development of collateral circulation along with the fact that a significant number of patients generally undergo exclusion of a limited segment of the descending thoracic aorta likely accounts for the lower incidence of spinal cord ischemia after endovascular repair than open surgical repair of the DTAA. These mechanisms could also explain the higher frequency of delayed, regressive spinal cord ischemia after endovascular repair [2,6,20-22]. Complete regression has been reported after CSF drainage and/or augmentation of systemic blood pressure [2,20-22]. Dependence of spinal cord blood supply on collateral circulation after endovascular repair is consistent with the frequent observation that neurological deficits were more common in patients who underwent previous or simultaneous open repair of an infrarenal abdominal aortic aneurysm [2,6,7,20-22,24].

Conclusion

In summary, this study presents our recent experience with open surgical repair and stent-grafting for elective treatment of DTAA. Endovascular repair was used exclusively in poor surgical risk patients in whom pre-operative selective arteriography usually demonstrated that the arteries supplying blood to the spinal cord were located outside the zone to be covered by the stent-graft. Using this approach we were able to achieve a postoperative mortality rate of 5.0% after open surgical repair. All neurological deficits due to spinal cord ischemia (7.4% including 2.8% paraplegia) were observed after conventional surgical management. No neurological deficits were observed after endovascular repair but the mortality rate was high (15.4%). More than half of deaths after endovascular repair were due to technical causes directly related to the method. Improvement of deployment skill and endovascular technology should lead to better results.

Due to yet unresolved concerns about the long-term outcome of stent-graft treatment of thoracic aneurysms [12], we will for the time being continue not to use endovascular repair in good surgical risk patients.

REFERENCES

1 Dake MD, Miller DC, Semba CP et al. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. *N Engl J Med* 1994; 331: 1729-1734.

2 Bell RE, Taylor PR, Aukett M et al. Mid-term results for second-generation thoracic stent-grafts. *Br J Surg* 2003; 90: 811-817.

3 Criado FJ, Clark NS, Barnatan MF. Stent-graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. *J Vasc Surg* 2002; 36: 1121-1128.

4 Ellozy SH, Carroccio A, Minor M et al. Challenges of endovascular tube graft repair of thoracic aortic aneurysm: midterm follow-up and lessons learned. *J Vasc Surg* 2003; 38: 676-683.

5 Leurs LJ, Bell R, Degrieck Y et al. Endovascular treatment of thoracic aortic diseases: combined experience from the EUROSTAR and United Kingdom Thoracic Endograft registries. *J Vasc Surg* 2004; 40: 670-680.

6 Makaroun MS, Dillavou ED, Kee ST et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. *J Vasc Surg* 2005; 41: 1-9.

7 Mitchell RS, Miller DC, Dake MD et al. Thoracic aortic aneurysm repair with an endovascular stent-graft: the "first generation". *Ann Thorac Surg* 1999; 67: 1971-1980.

8 Orend KH, Scharrer-Pamler R, Kapfer X et al. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. *J Vasc Surg* 2003; 37: 91-99.

9 Biglioli P, Spirito R, Porqueddu M et al. Quick, simple clamping technique in descending thoracic aortic aneurysm repair. *Ann Thorac Surg* 1999; 67: 1038-1044.

10 Coselli JS, LeMaire SA, Conklin LD, Adams GJ. Left heart bypass during descending thoracic aortic aneurysm repair does not reduce the incidence of paraplegia. *Ann Thorac Surg* 2004; 77: 1298-1303.

11 Estrera AL, Miller CC 3rd, Chen EP et al. Descending thoracic aortic aneurysm repair: 12-year experience using distal aortic perfusion and cerebrospinal fluid drainage. *Ann Thorac Surg* 2005; 80: 1290-1296.

12 Demers P, Miller DC, Mitchell RS et al. Mid-term results of endovascular repair of descending thoracic aortic aneurysms with first-generation stent-grafts. *J Thorac Cardiovasc Surg* 2004; 127: 664-673.

13 Kieffer E, Chiche L, Cluzel P et al. La chirurgie conventionnelle des anévrysmes de l'aorte thoracique descendante à l'heure du traitement endovasculaire: une expérience monocentrique de 9 ans. *Ann Chir Vasc* (in press).

14 Kieffer E. Chirurgie de l'aorte thoracique descendante. Encyclopédie médico-chirurgicale, techniques chirurgicales-chirurgie vasculaire. Paris: Elsevier. 43-148A.

15 Kieffer E, Fukui S, Chiras J et al. Spinal cord arteriography: a safe adjunct before descending thoracic or thoracoabdominal aortic aneurysmectomy. *J Vasc Surg* 2002; 35: 262-268.

16 Kieffer E, Richard T, Chiras J et al. L'artériographie médullaire préopératoire dans la chirurgie des anévrysmes de l'aorte thoracique et thoracoabdominale: résultats préliminaires chez 45 malades. *Ann Chir Vasc* 1989; 3: 34-46.
17 Kieffer E, Koskas F, Walden R et al. Hypothermic circulatory arrest for thoracic aneurysmectomy through left-sided thoracotomy. *J Vasc Surg* 1994; 19: 457-464.
18 Kieffer E, Chiche L, Touchot B. Surgical treatment of thoracoabdominal aortic aneurysms with cardiopulmonary bypass. In: Chiesa R, Melissano G, eds. *Chirurgia aortica: how to do it.* Milano, Universita Vita-Salute San Raffaele 2004: pp 107-119.
19 Glade GJ, Vahl AC, Wisselink W et al. Mid-term survival and costs of treatment of patients with descending thoracic aortic aneurysms; endovascular vs. open repair: a case-control study. *Eur J Vasc Endovasc Surg* 2005; 29: 28-34.
20 Cheung AT, Pochettino A, McGarvey ML et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. *Ann Thorac Surg* 2005; 80: 1280-1289.
21 Chiesa R, Melissano G, Marrocco-Trischitta MM et al. Spinal cord ischemia after elective stent-graft repair of the thoracic aorta. *J Vasc Surg* 2005; 42: 11-17.
22 Gravereaux EC, Faries PL, Burks JA et al. Risk of spinal cord ischemia after endograft repair of thoracic aortic aneurysms. *J Vasc Surg* 2001; 34: 997-1003.
23 Ohki T, Ouriel K, Silveira PG et al. Initial results of wireless pressure sensing for endovascular aneurysm repair: the APEX-Trial. Acute pressure measurement to confirm aneurysm sac excusion. *J Vasc Surg* 2007; 45: 236-242.
24 Moon MR, Mitchell RS, Dake MD et al. Simultaneous abdominal aortic replacement and thoracic stent-graft placement for multilevel aortic disease. *J Vasc Surg* 1997; 25: 332-340.