Eur J Vasc Endovasc Surg (2009) **37**, 640—645

ELSEVIER

EDUCATIONAL SERIES ON THORACIC AORTA (EDITED BY R. FATTORI)

Thoracoabdominal Aortic Aneurysm Repair: Results of Conventional Open Surgery

M.A. Schepens*, R.H. Heijmen, W. Ranschaert [1], U. Sonker, W.J. Morshuis

Department of Cardio-thoracic Surgery, St. Antonius Hospital, Nieuwegein, The Netherlands

Submitted 18 March 2009; accepted 18 March 2009
Available online 11 April 2009

KEYWORDS
Thoracoabdominal;
Aneurysm;
Aorta;
Dissection;
Complication;
Surgery

Abstract *Objectives:* The aim of this study is to report our experience in the surgical repair of thoracoabdominal aortic aneurysms (TAAAs) over the last 27 years against the background of evolving surgical techniques.

Methods: We reviewed the prospectively collected data of 571 patients who underwent open TAAA repair between 1981 and 2008. Data were analysed using univariate and multivariate analysis (logistic regression). Pre-, intra- and postoperative risk factors were used to develop risk models for in-hospital mortality, spinal cord deficit and renal failure. Recent published series were used to highlight the different treatment modalities and explore results.

Results: Seventy patients (12.3%) died in the hospital, the 30-day mortality was 8.9%, 37 patients (6.5%) required postoperative dialysis and 47 patients (8.3%) developed paraplegia or paraparesis. The incidence of paraplegia in the left heart bypass group was 4.4%. The predictors for hospital mortality were increasing age (odds ratio 1.096 per year, 95% confidence interval (CI): 1.05—1.14) and the need for haemodialysis (odds ratio 10, 95% CI: 4.7—21.1). For postoperative spinal cord deficit, we found three protecting factors: age above 75 years (odds ratio 0.14, 95% CI: 0.19—1.09), the presence of a post-dissection aneurysm (odds ratio 0.4, 95% CI: 0.17—0.94) and the combined use of cerebrospinal fluid drainage and motor-evoked potentials (odds ratio 0.28, 95% CI: 0.14—0.56). The urgency of procedure (odds ratio 4, 95% CI: 1.8—9) and preoperative serum creatinine level (odds ratio 1.007 per micromole per litre, 95% CI: 1.0—1.01) were significant risk factors for renal failure.

Conclusions: Open TAAA repair intrinsically has substantial complications, of which spinal cord ischaemia and renal failure are the most devastating, despite major progress in our understanding of the pathophysiology and operative strategy. An overview of the results of recently published series is given along with an analysis of our data.

* Corresponding author. M.A. Schepens, (Present address), Department of Cardio-thoracic Surgery, St. Jan Hospital, Ruddershove 10, 8000 Brugge, Belgium. Tel.: +32 5045 2690; fax: +32 5045 2689.
E-mail address: marc.schepens@azbrugge.be (M.A. Schepens).
[1] Present address: Department of Cardio-thoracic Surgery, St.Jan Hospital, Brugge, Belgium.

1078-5884/$36 © 2009 European Society for Vascular Surgery. Published by Elsevier Ltd. All rights reserved.
doi:10.1016/j.ejvs.2009.03.011

Thoracoabdominal aortic aneurysm (TAAA) is a highly lethal disorder. Open surgical repair is the only solution that ensures long-term durability and survival. This is evident from the fact that the number needed to treat (the number of patients who need to be treated to prevent one adverse outcome) is only two.[1] However, it is a complex and challenging intervention which tests not only the patient's cardiovascular systems, but also the pulmonary, neurological, haematological and immune systems. It is not surprising that this intervention is associated with a high incidence of serious complications, often leading to respiratory failure, renal failure, neurological deficits or even death. Substantial refinements in the preoperative optimalisation of the patient's condition, anaesthetic and surgical techniques as well as postoperative care over the past decades have reduced mortality and morbidity. Our ageing population with limited reserves confronts us with new challenges. The aim of this study is to review our total experience over the past 27 years and analyse hospital mortality, renal failure and spinal cord deficits.

Materials and Methods

In our hospital, 571 patients (62.5% male) were operated upon for TAAA between February 1981 and November 2008. Aneurysms limited to the descending thoracic aorta were excluded from this analysis. The patients' age ranged from 16 to 89 years (mean: 65.3 years). The distribution of the types of TAAA according to Crawford illustrates that type I, II, III and IV aneurysms were present in 22.8, 47.6, 18.7 and 10.9% of patients, respectively. The status of the cardiac and pulmonary functions was evaluated preoperatively if sufficient time was available. Our standard operative technique has been described in detail previously.[2] In summary, all patients were positioned in the right lateral position with hips flexed at 60° and intubated with a double-lumen endotracheal tube, allowing the collapse of the left lung (facilitating surgical exposure). Electrocardiogram, intra-arterial blood pressure (via the right brachial artery and right femoral or dorsal pedis artery), central venous pressure (via the right internal jugular vein) and pulmonary artery pressure were continuously monitored. Four large-bore intravenous catheters were inserted to allow rapid fluid administration if required. Intraoperative trans-oesophageal echocardiography was not used routinely. Two cell-saver suction tubes were used. Fluids administered to the patient were pre-warmed. Urine output and core (nasopharyngeal and rectal) temperature were recorded continuously. In nearly all the patients, a nasogastric tube was inserted (in some cases, this was not possible due to severe compression of the oesophagus by the aneurysm). Arterial blood gases, electrolytes, glucose, activated clotting time (ACT) and haemoglobin were measured regularly. During the last 15 years, we also monitored the intrathecal pressure using one (sometimes two) cerebrospinal fluid (CSF) catheter(s) inserted into the third or the fourth lumbar space after the induction of general anaesthesia. The pressure was maintained below 10 mm Hg and the amount of fluid drained was unlimited. The drain was kept in place for 72 h postoperatively. We did not use intrathecal naloxone, systemic thiopental before aortic occlusion or epidural cooling. In the case of left heart bypass, no extra heparin was administered (apart from 5000 international units in the fluid reservoir). We allowed a spontaneous lowering of the core temperature to 32 °C, except in cases where deep hypothermia was used. Deep hypothermia was restricted to patients in whom no proximal clamp could be applied due to the extension of the aneurysm into the arch, giant TAAA with a substantial risk of entering into the aneurysm while opening the chest, or inability to adequately ventilate the patient by the right lung alone. The surgical exposure of the aorta was by a left thoracoabdominal incision in the left sixth intercostal space with division of the costal arch. The diaphragm was incised circumferentially keeping about a 1.5 cm muscle at its costal insertion. For the last 10 years, we have approached the aneurysm retroperitoneally by medial rotation of the visceral sac instead of trans-peritoneal dissection. Cannulation of the left side of the heart was performed via the left inferior or superior pulmonary vein (which causes less rhythm disturbances and is easier to control), but rarely via the left atrial appendage. The non-aneurysmal proximal aorta could also be cannulated. The oxygenated blood is returned via the left femoral or iliac artery, resulting in temporary decreased perfusion of the left leg. The aneurysm is replaced mostly in a cranio-caudal fashion by using sequential clamping and after initiating left heart bypass. Since 1987, left heart bypass has been used in 413 patients (72.3%), deep hypothermia in 34 (6%) and simple cross-clamping in 124 patients (21.7%). When left heart bypass was used, distal aortic perfusion pressure was kept above 70 mmHg. Major visceral arteries were re-implanted by direct end-to-side anastomosis to the graft, sometimes after having performed an eversion endarterectomy. The same was performed for major intercostal and lumbar arteries between T8 and L2, guided by motor-evoked potential (MEP) monitoring. Renal protection was achieved by direct cannulation of the renal artery ostia and flushing with cold (4 °C) Ringer's acetate with mannitol and methylprednisolone if the ostia were within the aneurysm (shots of 300 cc per administration, repeated every 20 min, resulting in an approximate total of 1 l). Left heart bypass circuit was not used to provide selective visceral blood perfusion. Over the past years, we have actively re-warmed the patients at the end of the procedure; however, we now prefer to raise the temperature by slow external re-warming at the intensive care unit. Somatosensory-evoked potentials (SEPs) were introduced in 1984 and transcranial MEPs in 1994, both of which were used in every elective patient. All patients were followed up in our own outpatient clinic for at least one postoperative visit (3 months after surgery) and yearly thereafter (sometimes at the referring centre).

Study variables and definitions

The preoperative, intraoperative and postoperative data were collected from a prospectively maintained database which allowed us to use a set of 28 potential predictors: age (years), gender, year of operation, arterial hypertension, left ventricular ejection fraction, stroke or transient ischaemic attack (TIA), chronic obstructive pulmonary disease (COPD), serum creatinine level (micromole per litre), previous surgery on ascending aorta, aortic arch, descending or abdominal aorta, dissection, acute presentation (acute symptoms such as back pain, dyspnoea based on haematothorax or bronchial compression, hoarseness or dysphagia), urgency of the procedure (elective, meaning a stable situation with the possibility to delay surgery without a serious risk, versus urgent, meaning operation performed before the beginning of the next working day because of deterioration in clinical status), type of TAAA (according to the Crawford classification), surgeon, the adjunct used, Marfan syndrome, aetiology, CSF drain used or not, MEPs used or not, rupture, cold renal perfusion used or not, the appearance time of the blue dye in the urine after reperfusion of the kidneys, the need for postoperative dialysis and the incidence of spinal and central neurological damage. The term rupture was only used when it was confirmed during surgery. Renal insufficiency was defined as the need for haemodialysis. Operative mortality included all deaths occurring within 30-days from surgery and those occurring during the initial hospitalisation. Deaths occurring after hospital-to-hospital or hospital-to-nursing home discharge were counted as operative deaths. Spinal cord deficits, immediate (upon awakening) or delayed (with a symptom-free interval), were categorised as paraplegia or paraparesis in the absence of unilateral lower extremity deficit with an associated deficit of the ipsilateral arm indicating a central nervous problem. This study was approved by the Institutional Review Board with a waiver of informed consent.

Statistical analysis

All statistical analyses were performed using the SPSS (release 17, SPSS Inc., Chicago, IL, USA) statistical software package. Pre-, intra- and postoperative variables were evaluated for their association with operative mortality, spinal cord deficit and renal failure. Standard univariate methods (Yates-corrected chi-square or Student's *t*-test) were used. The null hypothesis was rejected and the associations with outcomes were considered statistically significant when $p < 0.05$. Factors found to be significant after univariate analysis were entered into stepwise logistic regression analysis.

Results

Operative mortality

Seventy patients (12.3%) died during their initial hospitalisation. The 30-day mortality was 8.9% ($n = 51$). The causes of death are illustrated in Table 1. Significant univariate predictors of hospital mortality are higher age, severely compromised left ventricular ejection fraction, previous aortic arch surgery, acute presentation, preoperative serum creatinine $\geq$150 μmol l^{-1} and postoperative need for dialysis. Post-dissection aneurysm was associated with a decreased risk. After logistic regression analysis, only age and the need for postoperative dialysis remained significant (Table 2). This indicates that a 50 year-old patient without the need for haemodialysis has a 2% risk of dying in the hospital, while a 78 year-old patient with renal failure necessitating dialysis has only a 29% chance of survival. Fig. 1 shows how operative mortality varies with age and with the necessity of dialysis. The annual hospital mortality is depicted in Fig. 2.

Table 1 Causes of in-hospital mortality

Cause of death	Number (%)
Cardiac	14 (2.5)
Intraoperative mortality	5 (0.9)
Oesophageal perforation	1 (0.2)
Intestinal ischemia	5 (0.9)
Pulmonary	7 (1.2)
Multi-organ failure	26 (4.6)
Aorta related	3 (0.5)
Cerebrovascular accidents	8 (1.4)

Spinal cord deficits

Immediate or delayed-onset paraplegia occurred in 30 patients (5.3%) and immediate or delayed paraparesis in 17 patients (3%). Significant univariate predictors related to an increased risk of spinal cord deficits include operations performed before the year 2000, acute presentation and the operating surgeon. The type of adjunct (in favour of the left heart bypass), the use of an adjunct, the use of CSF drainage, the use of MEPs, age above 75 years and post-dissection aneurysm were significant univariate predictors related to a decreased risk of spinal cord deficits. The annual incidence of spinal cord deficits is illustrated in Fig. 3. Paraplegia occurred in 6.6% of the patients operated upon using simple cross-clamping, in 12.5% of whom extra-corporeal circulation was used and in 4.4% left heart bypass operation was performed ($p = 0.09$). The incidence of paraplegia was significantly higher in the patient group that had undergone a prior abdominal aortic aneurysm (AAA) repair (10.7 versus 3.8%, $p = 0.003$). After multivariate analysis, age above 75 years, the fact that the TAAA was a consequence of a dissection and the combination of CSF drainage and the performance of MEPs remained significant (Table 3). All three factors were protective for spinal cord damage.

Renal failure

Renal failure, defined as the need for haemodialysis, occurred in 6.5% of patients. After univariate analysis, we

Table 2 Multivariate analysis of hospital mortality

Risk factor	Log regression coefficient	*p*-value	Estimated odds ratio	95% CI
Age (per year)	0.091	0.001	1.096	1.05–1.14
Need for haemodialysis	2.303	0.001	10.0	4.7–21.1
Constant	−8.57	0.001	–	–

CI = confidence interval global χ^2 of final model = 7.1 ($p = 0.52$).

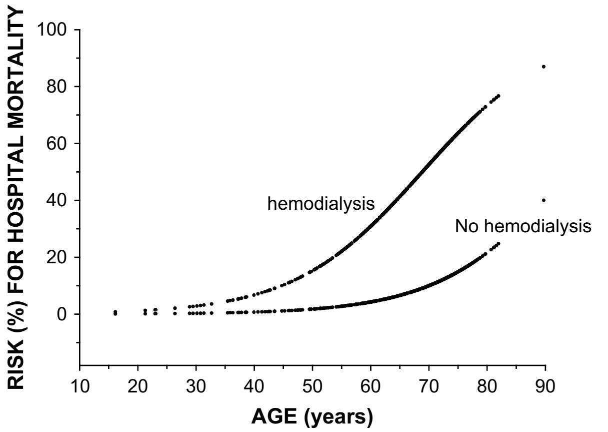

Figure 1 Influence of age and postoperative haemodialysis on in-hospital mortality.

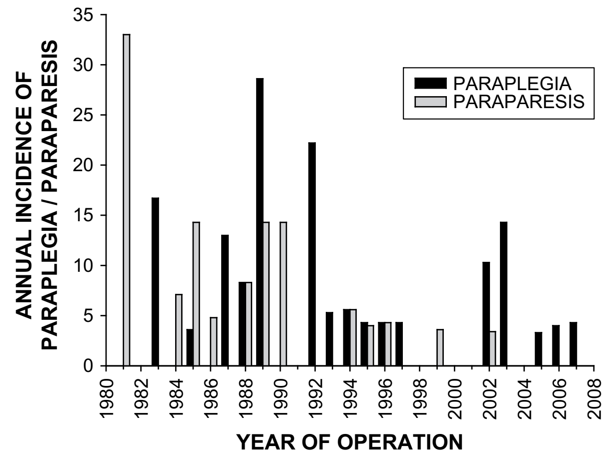

Figure 3 Annual incidence of spinal cord deficits.

found that low preoperative creatinine level, post-dissection aneurysm, elective intervention, use of adjunct, use of left heart bypass, renal perfusion, the operating surgeon and the appearance of the blue dye into urine less than 30 min after reperfusion were related to a lower incidence of haemodialysis. After multivariate analysis in a model that predicts 94% of the cases correctly, we found that the urgency of the procedure and the preoperative creatinine level were independent predictors for haemodialysis (Table 4). In practice, this means that a patient with an elevated preoperative creatinine of 200 μmol l^{-1} and an urgent intervention has a risk of 25% for haemodialysis. On the other hand, if the creatinine level is 60 μmol l^{-1} and the intervention is planned, the risk is limited to 2.7%. The relationship of the risk of postoperative dialysis to the preoperative serum creatinine level and urgency is illustrated in Fig. 4.

Discussion

The most debilitating complication of TAAA repair is spinal cord injury. Spinal cord ischaemia remains an ever-present threat despite the use of simultaneous protective measures such as CSF drainage, distal aortic perfusion using left heart bypass, liberal re-implantation of intercostals and lumbar arteries between T8 and L2, permissive mild hypothermia and MEP monitoring. We have previously reported our experience using simple cross-clamping[3] resulting in a paraplegia rate of 5.7% and a paraparesis rate of 8%. The use of left heart bypass alone decreased paraplegia and paraparesis rate to 4.4 and 1.7%, respectively. Our results confirm that the use of CSF drainage and left heart bypass is protective as shown by Safi et al. in 1998.[4] Aneurysms resulting from a previous dissection apparently are protective not only for spinal cord damage, but also for renal failure and in-hospital mortality; this fact is intriguing, but not new. In this relatively young patient category, more intercostal arteries are patent and therefore re-implanted in higher numbers as compared to atherosclerotic aneurysms. This has also been reported by Coselli et al.[5] In the initial Crawford series,[6] however, dissection was noted to be a risk factor for neurological disturbances. The finding that patients older than 75 years have a lower risk of spinal cord damage still remains unexplained.

Griepp et al.[7] commented that no prospective studies have demonstrated the efficacy of SEP and MEP in reducing the incidence of spinal cord injury. We support the findings of Keyhani et al.[8] that normal SEP and MEP have a strong negative predictive value indicating that patients who have

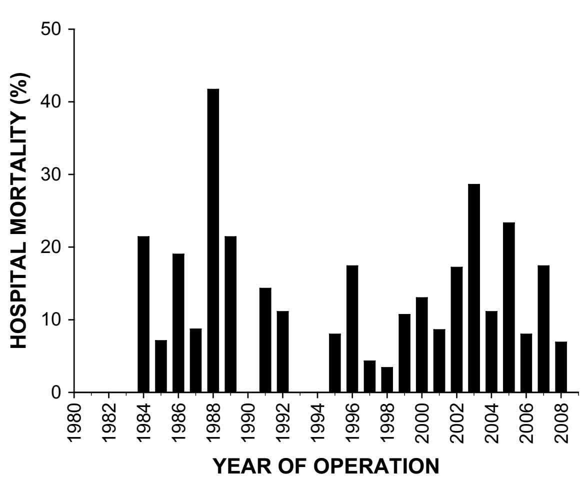

Figure 2 Annual in-hospital mortality.

Table 3 Multivariate analysis of spinal cord deficit

Risk factor	Log regression coefficient	*p*-value	Estimated odds ratio	95% CI
Age above 75	−1.91	0.06	0.14	0.19–1.09
Presence of aortic dissection	−0.90	0.03	0.40	0.17–0.94
CSF-d × MEP	−1.27	0.001	0.28	0.14–0.56
Constant	− 1.47	0.001	–	–

CI = confidence interval global χ^2 of final model = 1.6 (p = 0.80); CSF-d = cerebrospinal fluid drainage; MEP = motor-evoked potentials.

Table 4 Multivariate analysis of postoperative renal failure necessitating haemodialysis

Risk factor	Log regression coefficient	*p*-value	Estimated odds ratio	95% CI
Urgency	1.42	0.001	4.13	1.8–9.1
Preoperative serum creatinine	0.0077	0.001	1.0	1.00–1.01
Constant	−4.04	0.001	–	–

CI = confidence interval global χ^2 of final model = 12.7 ($p = 0.12$).

no loss of signals are unlikely to awake with neurological deficit. Previous studies have shown that evoked potentials are useful monitoring tools, allowing relevant adjustments in operative strategy.[9–11]

We have investigated the role of the release of biochemical markers such as protein S100 into the CSF[12] to detect ischaemia of the spinal cord. The practical value of this is limited for intraoperative monitoring since elevation of these markers occurs late, except for glial fibrillary acidic protein.[13] We could not find any association between spinal cord deficits and the clamping of the left sub-clavian artery.

Over the past decade, a downward trend has been observed in mortality and complication rate after TAAA repair. While Etz et al.[14] reported a 30-day mortality of 6.4% in 140 patients, Coselli et al.[15] reported 5% in 2286 patients and Safi et al.[16] 14% in 1004 patients. At first glance, our 30-day mortality of 8.9% seems high compared to some of these studies; it is important to stress, however, that our results are a summary of our complete experience since 1981. We have noted that in-hospital mortality and 30-day mortality are sometimes not clearly defined in all studies. The perspective of our operative mortality changes when we apply the predictive model of LeMaire et al.[17] (based on the number of patients with: (1) Crawford extent II TAAA; (2) ruptured aneurysms; (3) renal insufficiency; and (4) symptomatic aneurysms, in addition to the total number of TAAAs). If we apply this risk model to our data, we obtain an expected 30-day mortality of 33%, while our 30-day mortality is only 8.9%. Our results also compare favourably to 19% mortality at 30-days in elective cases and 48.4% in ruptured cases documented in California.[18] Most of our patients were operated upon using left heart bypass, which was introduced in 1986, before which we solely relied on simple cross-clamping.[3] Deep hypothermic circulatory arrest was used only in selected patients as described in the method session. We agree with Coselli et al.[19] that the use of this technique should be limited to specific situations. It achieves optimal protection of various organs, which is reflected in a lower paraplegia rate; however, the downside is the increased incidence of serious complications.

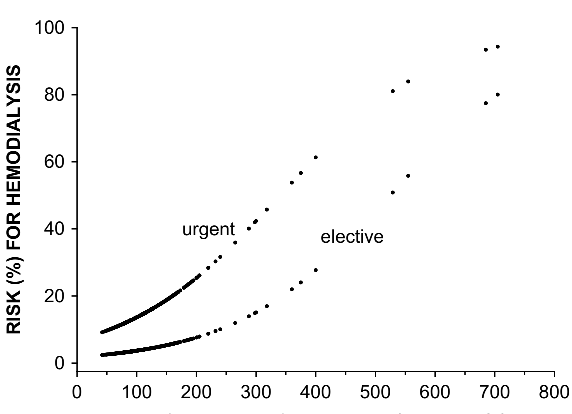

Figure 4 Relation between preoperative serum creatinine, urgency and the risk of haemodialysis.

Postoperative renal insufficiency is another major complication after TAAA repair, often leading to death. The results of our analysis support this. Multivariate analysis shows that the need for haemodialysis increases the risk of early death by a factor of 10. Our method to limit postoperative renal failure has not changed over the years except for the introduction of left heart bypass and sequential clamping, which reduces renal ischaemic time substantially as compared to simple cross-clamping. The analysis of our initial experience with simple cross-clamping showed a 14% incidence of haemodialysis. Higher age and elevated preoperative serum creatinine were important risk factors for postoperative haemodialysis.[20] Our analysis highlights that besides urgency, the preoperative serum creatinine level remains an important determinant of postoperative renal failure. When we compared the results of using simple cross-clamping with those of left heart bypass group, the incidence of renal failure dropped to 4.6%. It has been shown that intermittent cold (4 °C) crystalloid perfusion of the kidneys lowers the incidence of renal dysfunction when compared to continuous perfusion with isothermic blood. The results of LeMaire et al.[21] confirm that cold blood perfusion is no better than cold crystalloid perfusion. Perfusion with cold Ringer's acetate is certainly less cumbersome than perfusion with cold blood. Miller et al.[22] have shown that ischaemia of the left leg is related to postoperative renal dysfunction; however, we could not confirm this.

An important weakness of our study is that it covers a long time frame during which operative techniques have changed from simple cross-clamping to left heart bypass. However, it provided an opportunity to evaluate these operative techniques using univariate and multivariate analysis. In most large series, nearly all interventions were performed by a single surgeon with the potential for an improved learning curve and, thus, the results. In our experience, surgeons involved in this type of surgery have changed over the last 27 years: for example, one surgeon performed 45% of all the interventions and three surgeons 11% each, thus illustrating the variability. The operating surgeon was a univariate predictor for spinal cord deficits and renal failure. In most cases, these four surgeons assisted each other during the TAAA repair. Anaesthetic support was limited to only two anaesthesiologists, except in urgent operations. We agree to the fact that, given the complexity of this intervention, familiarity within the surgical team is absolutely necessary to improve team performance.[23] However, increasing individual experience in a certain surgical procedure may raise the acceptance threshold.

More patients with higher risk profiles may be accepted and consequently operated upon, which may in turn have a negative impact on the results. Since there are no mutually agreed upon risk models, the comparison of results between two series remains ambiguous.

Our current analysis clearly proves that the surgical repair of TAAA remains a challenge even in the 21st century. Open TAAA repair intrinsically has substantial complications, of which spinal cord ischaemia and renal failure are the most devastating, despite major progress in our understanding of the pathophysiology and operative strategy. Important risk factors for in-hospital mortality, renal failure and spinal cord deficits have been demonstrated in the present study.

References

1 Miller III CC, Porat EE, Estrera AL, Vinnerkvist AN, Huynh TT, Safi HJ. Number needed to treat: analyzing of the effectiveness of thoracoabdominal repair. *Eur J Vasc Endovasc Surg* 2004;**28**:154–7.

2 Schepens MA, Dossche KM, Morshuis WJ, Heijmen RH, van Dongen EP, ter Beek HT, et al. Introduction of adjuncts and their influence on changing results in 402 consecutive thoracoabdominal aortic aneurysm repairs. *Eur J Cardiothorac Surg* 2004;**25**:701–7.

3 Schepens MA, Defauw JJ, Hamerlijnck RP, De Geest R, Vermeulen FE. Surgical treatment of thoracoabdominal aortic aneurysms by simple crossclamping. *J Thorac Cardiovasc Surg* 1994;**107**:134–42.

4 Safi HJ, Vinnerkvist AN, Miller III CC, Iliopoulos DC, Reardon MJ, Espada R, et al. Effect of extended cross-clamp time during thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg* 1998;**66**:1204–9.

5 Coselli JS, LeMaire SA, de Figueiredo LP. Paraplegia after thoracoabdominal aortic aneurysm repair: is dissection a risk factor? *Ann Thorac Surg* 1997;**63**:28–36.

6 Crawford ES, Crawford JL, Safi HJ, Coselli JS, Hess KR, Brooks B, et al. Thoracoabdominal aortic aneurysms: preoperative and intraoperative factors determining immediate and long-term results of operations in 605 patients. *J Vasc Surg* 1986;**3**:389–404.

7 Griepp RB, Griepp EB. Spinal cord perfusion and protection during descending thoracic and thoracoabdominal aortic surgery: the collateral network concept. *Ann Thor Surg* 2007;**83**:S865–9.

8 Keyhani K, Miller III CC, Estrera AL, Wegryn T, Sheinbaum R, Safi HJ. Analysis of motor and somatosensory evoked potentials during thoracic and thoracoabdominal aortic aneurysm repair. *J Vasc Surg* 2009;**49**:36–41.

9 van Dongen EP, Schepens MA, Morshuis WJ, ter Beek HT, Aarts LP, De Boer A, et al. Thoracic and thoracoabdominal aortic aneurysm repair: use of evoked potential monitoring in 118 patients. *J Vasc Surg* 2001;**34**:1035–40.

10 Galla JD, Ergin MA, Lansman SL, McCullough JN, Nguyen KH, Spielvogel D, et al. Use of somatosensory evoked potentials for thoracic and thoracoabdominal aortic resections. *Ann Thorac Surg* 1999;**67**:1947–52. discussion 1953–1958.

11 Jacobs MJ, Mess W, Mochtar B, Nijenhuis RJ, Statius van Eps RG, Schurink GW. The value of motor evoked potentials in reducing paraplegia during thoracoabdominal aneurysm repair. *J Vasc Surg* 2006;**43**:239–46.

12 van Dongen EP, ter Beek HT, Boezeman EH, Schepens MA, Langemeijer HJ, Aarts LP. Normal serum concentrations of S-100 protein and changes in cerebrospinal fluid concentrations of S-100 protein during and after thoracoabdominal aortic aneurysm surgery: is S-100 protein a biochemical marker of clinical value in detecting spinal cord ischemia? *J Vasc Surg* 1998;**27**:344–6.

13 Winnerkvist A, Anderson RE, Hansson LO, Rosengren L, Estrera AE, Huynh TT, et al. Multilevel somatosensory evoked potentials and cerebrospinal proteins: indicators of spinal cord injury in thoracoabdominal aortic aneurysm surgery. *Eur J Cardiothorac Surg* 2007;**31**:637–42.

14 Etz CD, Di Luozzo G, Bello R, Luehr M, Khan MZ, Bodian CA, et al. Pulmonary complications after descending thoracic and thoracoabdominal aortic aneurysm repair: predictors, prevention, and treatment. *Ann Thorac Surg* 2007;**83**:S870–6.

15 Coselli JS, Bozinovski J, LeMaire SA. Open surgical repair of 2286 thoracoabdominal aortic aneurysms. *Ann Thorac Surg* 2007;**83**:S862–4.

16 Safi HJ, Miller III CC, Huynh TT, Estrera AL, Porat EE, Vinnerkvist A, et al. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair. Ten years of organ protection. *Ann Surg* 2003;**238**:372–81.

17 LeMaire SA, Miller III CC, Conklin LD, Schmittling ZC, Coselli JS. Estimating group mortality and paraplegia rates after thoracoabdominal aortic aneurysm repair. *Ann Thorac Surg* 2003;**75**:508–13.

18 Rigberg DA, McGory ML, Zingmond DS, Maggard MA, Agustin M, Lawrence PF, et al. Thirty-day mortality statistics underestimate the risk of repair of thoracoabdominal aortic aneurysms: a statewide experience. *J Vasc Surg* 2006;**43**:217–22.

19 Coselli JS, Bozinovski J, Cheung C. Hypothermic circulatory arrest: safety and efficacy in the operative treatment of descending and thoracoabdominal aortic aneurysms. *Ann Thorac Surg* 2008;**85**:956–64.

20 Schepens MA, Defauw JJ, Hamerlijnck RP, Vermeulen FE. Risk assessment of acute renal failure after thoracoabdominal aortic aneurysm surgery. *Ann Surg* 1994;**219**:400–7.

21 LeMaire SA, Jones MM, Conklin LD, Carter SA, Criddell MD, Wang XL, et al. Randomized comparison of cold blood and cold crystalloid renal perfusion for renal protection during thoracoabdominal aortic aneurysm repair. *J Vasc Surg* 2009;**49**:11–9.

22 Miller III CC, Villa MA, Achouch P, Estrera AL, Azizzadeh A, Coogan SM, et al. Intraoperative skeletal muscle ischemia contributes to risk of renal dysfunction following thoracoabdominal aortic repair. *Eur J Cardiothorac Surg* 2008;**33**:691–4.

23 Elbardissi AW, Wiegmann DA, Henrickson S, Wadhera R, Sundt III TM. Identifying methods to improve heart surgery: an operative approach and strategy for implementation on an organizational level. *Eur J Cardiothorac Surg* 2008;**34**:1027–33.