Spinal cord ischemia after elective stent-graft repair of the thoracic aorta

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Objectives: **Neurologic deficit after endovascular treatment of the thoracic aorta is a complication reported with variable frequency that may be associated with severe morbidity and mortality. The mechanism of spinal cord ischemia appears to be multifactorial and remains ill-defined. We reviewed our experience to investigate the determinants of paraplegia after stent-graft repair of the thoracic aorta, identify patients at risk, and assess the effectiveness of ancillary techniques.**
Methods: **Over a 5-year period (June 1999 to December 2004), 103 patients underwent elective endovascular repair of the thoracic aorta at a university referral center. Indications for treatment were atherosclerotic aneurysms in 88 patients, chronic type B dissection in 10 patients, and penetrating aortic ulcer in 5 patients. Four of the 103 patients affected with thoracoabdominal aortic aneurysms had hybrid procedures and were excluded from the cumulative analysis. Twelve patients with zone 0 and zone 1 aortic arch aneurysms were operated on with synchronous or staged surgical aortic debranching. Preoperative cerebrospinal fluid (CSF) drainage was instituted in seven selected patients. Neurologic deficits were assessed by an independent neurologist and classified as immediate or delayed. Patient demographics and perioperative factors related to the endovascular procedure were evaluated by using univariate statistical analyses.**
Results: **A primary technical success was achieved in 94 patients (94.9%). At a mean follow-up of 34 ± 14 months, a midterm clinical success was obtained in 90 patients (90.9%). Four patients (4.04%) had delayed neurologic deficit that completely resolved after the institution of CSF drainage, steroids administration, and arterial pressure pharmacologic adjustment. None of the four patients who underwent hybrid procedures for thoracoabdominal aortic aneurysms had paraplegia or paraparesis. Univariate analyses identified only a perioperative lowest mean arterial pressure (MAP) of <70 mm Hg as a significant risk factor (*P* < .0001).**
Conclusion: **Perioperative hypotension (MAP <70 mm Hg) was found to be a significant predictor of spinal cord ischemia; hence, careful monitoring and prompt correction of arterial pressure may prevent the development of paraplegia. When the latter occurred, reduction of the CSF pressure by drainage was useful. Patients with a previous or synchronous abdominal aortic repair may also benefit from CSF drainage as a perioperative adjunct.** (**J Vasc Surg 2005;42:11-7.**)

Endovascular treatment of thoracic aortic pathologies is emerging as a less invasive alternative to open surgical graft replacement. Recent series have reported encouraging results in terms of technical success and midterm outcomes compared with traditional open repair.[1-3] On the other hand, the use of thoracic stent grafts has introduced a new pattern of complications related to their delivery and deployment, as well as to the grafts themselves.

Endoluminal repair allows the avoidance of aortic cross clamping and its sequelae[4]; however, the intercostal arteries covered by the stent graft cannot be reimplanted. The reported incidence of both immediate and delayed paraplegia in patients undergoing endovascular procedures can be as high as 12% of cases[1-8] compared with 2% to 21% after open repair.[9-15] The mechanism underlying the occurrence of spinal cord ischemia (SCI) after thoracic endovascular aortic repair (EVAR) has yet to be completely understood and represents a key issue for further developments of thoracic stent grafts, because paraplegia results not only in severe physical disability but is also associated with decreased survival rates.[16]

In this study, we analyzed our most recent 5-year experience with repairs of thoracic aortic pathologies to evaluate the incidence and investigate the determinants of spinal cord ischemia in endovascular procedures, identify patients at risk, and assess the role and efficacy of prophylactic adjuncts and therapeutic measures.

METHODS

This study was designed as a single-center experience. We conducted an analysis of data prospectively collected on a computerized database of all patients undergoing elective thoracic EVAR at our center. Over a 5-year period (June 1999 to December 2004), 103 patients underwent thoracic EVAR. There were 86 men and 17 women with a mean age of 70.1 ± 7.7 years. Four of the 103 patients underwent hybrid procedures for thoracoabdominal aortic aneurysms (TAAs). Because of the presence of possible confounding factors related to the different nature of the disease and to the complexity of each of these tailored surgical and endovascular procedures, these cases were excluded from the cumulative analysis.

Indications for thoracic EVAR included atherosclerotic aneurysms in 84 patients, chronic type B dissections in 10,

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Competition of interest: none.
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0741-5214/$30.00

doi:10.1016/j.jvs.2005.04.016

and penetrating aortic ulcers in 5 patients. The site of proximal endograft deployment is shown in the Fig according to the "aortic arch map" proposed by Ishimaru.[2] Patients categorized as zone 0 and zone 1 received a synchronous or staged open surgical procedure for aortic debranching, as previously described.[17] In patients with zone 2 aneurysms, a revascularization of the left subclavian artery was performed prophylactically in the first three patients[17] and then only selectively in one patient with previous coronary artery bypass grafting (CABG) that consisted of a left internal thoracic artery-to-left anterior descending coronary artery. Four patients underwent thoracic aorta stent-graft repair and synchronous open abdominal aortic aneurysm (AAA) repair.

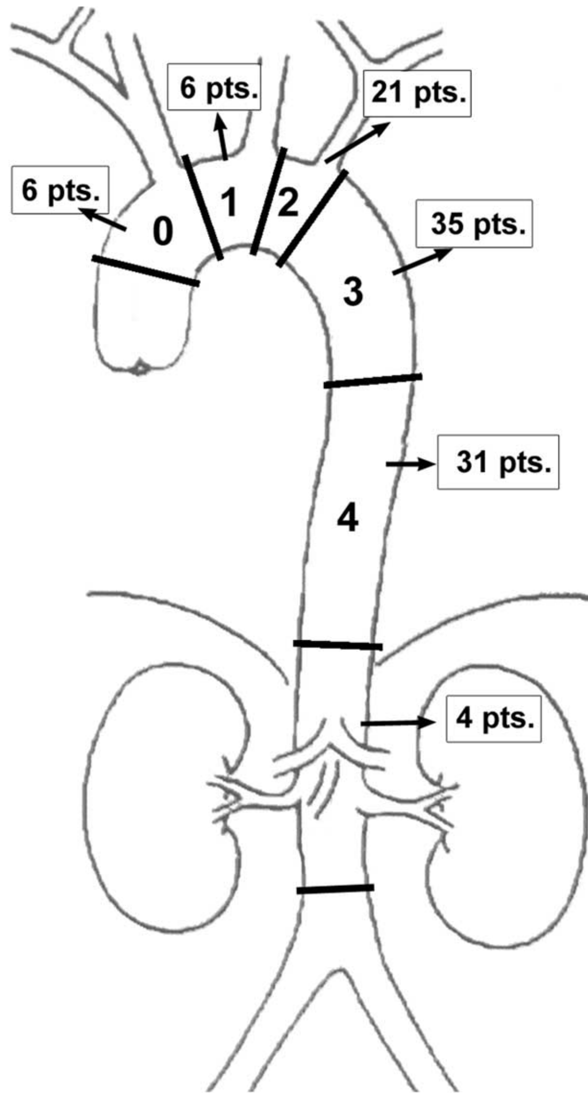
The feasibility of the endoluminal intervention and sizing of stent grafts was determined with preoperative computed tomography (CT) scans and aortography. The endografts implanted were Gore Excluder TAG (WL Gore and Assoc, Flagstaff, Ariz) in 38 cases (28 old devices and 10 new devices), Talent (AVE/Medtronic Inc, Santa Rosa, Calif) in 4 cases, Endofit (Endomed Inc, Phoenix, Ariz) in 11 cases, and Zenith TX1 (46 cases) and TX2 (4 cases) devices (William Cook Europe Aps, Bjaeverskov, Denmark).

Patients were operated on under spinal anesthesia (47 cases), local anesthesia (6 cases), or general anesthesia (50 cases). Preoperative cerebral spinal fluid (CSF) drainage was instituted in seven selected patients. Based on data previously reported in the literature and our experience in open surgical repair of TAA, we used CSF drainage in patients with an aneurysm involving critical intercostal arteries (T8 to L2), in those requiring the coverage of a long descending thoracic aortic segment (≥20 cm), and in patients with a previous AAA repair. Intraoperative transesophageal echocardiography monitoring was used for aortic dissection.

All the procedures were performed in the operating room, and a portable digital C-arm image intensifier with subtraction angiography and roadmap capabilities was used. No temporary or permanent prosthetic conduits were used to access the arterial system. The preferred entry sites were the common femoral arteries. Access was also through the common iliac artery in three cases, through the left branch of a previous aortobiiliac graft in one case, and through the abdominal graft in the four patients who had synchronous AAA surgical repair.

Neurologic deficits were defined as paraplegia or paraparesis according to the Tarlov scale[18] (Table I) and assessed by an independent neurologist. Paraplegia or paraparesis observed immediately or upon awakening were defined as immediate neurologic deficits. Those occurring after a period of normal neurologic function were classified as delayed deficits.

We investigated the influence of the following demographic factors as possible predictors of postoperative SCI: age, gender, cardiac risk factor (previously documented myocardial infarction, ongoing angina pectoris, previous coronary bypass surgery, percutaneous transluminal coronary angioplasty, or a combination), diabetes mellitus (hyperglycemia requiring diet, oral medication, or insulin treatment), hypertension (medication for hypertension or arterial pressure >160/95 mm Hg), cerebrovascular disease (history of stroke or transient ischemic attacks), pulmonary disease (chronic obstructive pulmonary disease or



Anatomic location of proximal landing zone of thoracic stent graft in 103 patients according to the aortic map.

Table I. Modified Tarlov scoring scale

Scale	*Motor function*	*Deficit*
0	No lower extremity movement	Paraplegia
1	Lower extremity motion without gravity	Paraplegia
2	Lower extremity motion against gravity	Paraplegia
3	Able to stand with assistance	Paraparesis
4	Able to walk with assistance	Paraparesis
5	Normal	Normal

asthma), renal failure (serum creatinine >150 μmol/L), smoking (current smoker), and surgical AAA repair.

The following perioperative factors related to the endovascular procedure were also analyzed: indication for EVAR (aneurysm, chronic dissection, penetrating aortic ulcers), anatomic location of proximal landing zone (0 to 4), length of devices, intraoperative device migration, aortic occlusion during graft deployment (including ballooning), type I or type II endoleak at intraoperative control, deliberate covering of the left subclavian artery by the stent graft, and intraoperative and postoperative (≤48 hours after the procedure) lowest mean arterial pressure (MAP) <70 mm Hg. A systematic assessment of stent-graft occlusion of the intercostal or lumbar arteries between T8 and L2 was not performed because of difficulties related to significant aortic tortuosity and intraluminal thrombosis.

Univariate analyses were conducted for patient demographics and considered perioperative risk factors as shown in Tables II and III. Student's *t* test and the χ^2 test or Fisher's exact test were performed as appropriate. All analyses were run on the Statistical Package for the Social Sciences (SPSS) PC+ version 9.0 software (SPSS Inc, Chicago, Ill).

RESULTS

According to the reporting standards for endovascular aortic aneurysm repair,[19] a primary technical success was achieved in 94 (94.9 %) of the 99 subjects included in the cumulative analysis. One patient died intraoperatively because of graft migration,[20] and four patients had a residual type Ia endoleak that was left untreated because the aortic proximal neck was deemed inadequate for further endovascular procedures. A type II endoleak was also observed in eight patients.

Stroke occurred in one patient who had endovascular repair of a zone 3 penetrating aortic ulcer and in two patients treated for a zone 0 aortic aneurysm by means of synchronous surgical aortic debranching and stent-graft repair of the aortic arch with intentional coverage of the supra-aortic vessels. No positive conclusions can be drawn regarding the cause of this complication being related to possible ischemic or embolic events that were secondary to surgical and endovascular procedures. Postoperative complications also included acute renal failure reversed without dialysis in two patients, acute respiratory failure in one, and seizure in one patient.

An initial (30-day) clinical success[19] was obtained in 91 patients (91.9%). We recorded two postoperative in-hospital deaths caused by stroke in one patient and acute respiratory failure in the other. One patient electively underwent a successful surgical conversion 2 weeks after the procedure because of a total collapse of the graft.[20]

A short-term clinical success[19] was achieved in 91 patients (91.9%). Two patients who suffered a perioperative stroke died, and the complete spontaneous resolution of a Type Ia endoleak was observed in two patients.

At a mean follow-up of 34 ± 14 months, including only 1-month survivors, midterm clinical success[19] was obtained in 90 patients (90.9%), including eight with type II endoleak in the absence of aneurysm expansion. One type Ia endoleak resolved completely without any further intervention. One patient underwent a successful surgical conversion 43 months after EVAR because of a late fracture of the longitudinal support wire of a Gore Excluder TAG endoprosthesis. Aneurysm expansion was observed in one patient. Five patients died of unrelated causes. One patient was lost to follow-up.

Spinal cord ischemia. Four patients (4.04%) had delayed neurologic deficit. The first, a 70-year-old man who previously had a surgical AAA repair, underwent endovascular exclusion of an aneurysm of the descending thoracic aorta (zone 4) with a Zenith TX1 42- × 139-mm device. The procedure was performed under spinal anesthesia. No intraoperative complications occurred. His postoperative course was characterized by wide fluctuations in MAP, with a lowest value of <70 mm Hg. A left lower extremity neurologic deficit (Tarlov score, 2) developed 48 hours after EVAR. It completely resolved within 72 hours after pharmacologic adjustment to keep MAP >90 mm Hg, intravenous steroids administration (dexamethasone, 24 mg/day), and institution of CSF drainage. Cerebrospinal fluid was allowed to freely drain with gravity when intrathecal pressure was >10 mm Hg. The highest registered CSF pressure was 15 mm Hg, and a total of 280 mL of CSF was drained. A CT scan disclosed no signs of spinal cord infarction. The remainder of his postoperative course was uneventful, and the patient was discharged on postoperative day 7.

In the second case, a 68-year-old-man underwent endovascular repair of a zone 4 thoracic aortic aneurysm under general anesthesia. A Gore Excluder TAG 30- × 150-mm endograft was successfully deployed without any technical complications. Upon awakening, the patient showed no neurologic deficits. During his immediate postoperative course, the patient had significant blood pressure instability, and 24 hours after the procedure, paraplegia (Tarlov score, 1) developed after a hypotensive episode (MAP value was 60 mm Hg). The neurologic deficit was completely reversed ≤12 hours by means of maintenance of MAP of >90 mm Hg, CSF drainage (highest CSF pressure, 16 mm Hg; total CSF drained, 190 mL), and steroids administration. The patient was discharged on postoperative day 5.

The third patient was a 70-year-old man who previously had a left internal thoracic artery-left anterior descending coronary artery CABG. He underwent synchronous left carotid-subclavian bypass and endovascular exclusion of a zone 2 aortic arch aneurysm with a Cook Zenith TX1 (36 ×156 mm) under general anesthesia. The procedure was uneventful. The early postoperative period was characterized by hemodynamic instability, with the lowest MAP <70 mm Hg. On postoperative day 4, paraparesis developed (Tarlov score, 4) associated with ataxia. The neurologic deficit completely resolved ≤72 hours after institution of the treatment described previously, including drainage of 230 mL of CSF (highest CSF pressure value, 14 mm Hg). The patient was discharged well 7 days after the procedure.

Table II. Univariate analysis of the association between patient demographics and paraplegia

Variables	*No paraplegia n (%)*	*Paraplegia n (%)*	P*
Age (mean ± SD)	70.0 ± 7.8	72.0 ± 5.6	.62†
Gender (M/F)	78 (82.1)/17 (17.2)	4 (100)/0 (0)	1.0
Cardiac risk factor	46 (48.4)	1 (25.0)	.61
Diabetes mellitus	10 (10.5)	0 (0)	1.0
Hypertension	66 (69.5)	4 (100)	.31
Cerebrovascular disease	11 (11.6)	0 (0)	1.0
Pulmonary disease	47 (49.5)	3 (75.0)	.61
Renal failure	13 (13.7)	1 (25.0)	.46
Smoking	63 (66.3)	3 (75.0)	1.0
Indication to EVAR			.68
Aneurysm	80 (84.2)	4 (100)	—
Chronic dissection	10 (10.5)	0 (0)	—
PAU	5 (5.3)	0 (0)	—
AAA surgical repair	11 (11.6)	2 (50.0)	.08

M, Male; *F,* female; *EVAR*, endovascular aortic repair; *PAU,* penetrating aortic ulcers; *AAA,* abdominal aortic aneurysm.
*χ^2 statistics or Fisher's exact test.
†Student's *t* test.

Table III. Univariate analysis of the association between perioperative risk factors and paraplegia

Variables	*No paraplegia n (%)*	*Paraplegia n (%)*	P*
Proximal landing zone			.89
0	6 (6.3)	0 (0)	—
1	6 (6.3)	0 (0)	—
2	20 (21.0)	1 (25.0)	—
3	34 (35.8)	1 (25.0)	—
4	29 (30.5)	2 (50.0)	
Length of device (mean ± SD)	149.3 ± 38.4	162.7 ± 26.1	.49†
Intraoperative device migration	5 (5.3)	0 (0)	1.0
Aortic occlusion during graft deployment	14 (14.7)	1 (25.0)	.48
Type I endoleak‡	4 (4.2)	0 (0)	1.0
Type II endoleak‡	7 (7.4)	1 (25.0)	.29
Left subclavian artery occlusion	29 (30.5)	0 (0)	1.0
Lowest MAP <70 mm Hg	4 (4.2)	4 (100)	<.0001

MAP, Mean arterial pressure.
*χ^2 statistics or Fisher's exact test.
†Student's *t* test.
‡Intraoperative control.

The last patient was an 80-year-old man with synchronous abdominal aortic and zone 3 thoracic aortic aneurysms. He underwent an abdominal aortic surgical repair with 16-mm Dacron (DuPont, Wilmington, Del) graft interposition followed by the insertion through the abdominal graft, by means of a pursestring suture, of a Zenith Cook TX1 40- × 200-mm endograft that was successfully deployed distal to the subclavian artery. The patient recovered from general anesthesia without neurologic deficit. On postoperative day 1, he experienced persistent hypotension for about 30 minutes, with lowest levels of MAP of <60 mm Hg, and lower left hemiparesis (Tarlov score, 4) developed. The patient began to recover as the hypotension was pharmacologically reversed, and the deficit completely regressed ≤24 hours after the institution of CSF drainage (highest CSF pressure, 15 mm Hg; total CSF drained, 200 mL) and steroids administration.

Spinal cord evaluation through magnetic resonance imaging (MRI) was not performed in any of these patients. In three patients, a Cook device had been implanted and MRI was contraindicated by the manufacturer because of the presence of steel components.

None of the four patients who had hybrid procedures for TAA had neurologic deficits. The analysis of the impact of patient characteristics and perioperative variables are summarized in Tables II and III. None of the patient demographics was found to be a predictor of paraplegia; however, patients with previous (9 cases) or synchronous (4 cases) AAA repair had a trend of an increased risk of neurologic deficit ($P = .08$). Univariate analysis identified only a perioperative lowest MAP of <70 mm Hg as a significant risk factor ($P < .0001$).

DISCUSSION

Postoperative neurologic deficit is a dreaded and devastating possible complication of thoracic and thoracoabdominal aortic repair. The risk of SCI also remains significant after endovascular procedures and appears to be related to a complex interaction of several different mechanisms.

Spinal cord perfusion pressure depends on changes of the gradient between arterial pressure and CSF pressure. Aortic cross clamping performed during open repair of thoracic aortic pathologies causes a decrease in distal arterial pressure that may alter spinal cord blood perfusion. To reduce the risk of neurologic complications, a number of ancillary techniques have been developed and used (Table IV).[21-30] No method has been ultimately successful in the prevention of paraplegia, however.

Endovascular repair does not require aortic cross clamping and therefore generally reduces hemodynamic-related derangements. This may not be always the case, however, because errant stent-graft deployment can result in a period of aortic occlusion that leads to paraplegia.[31] Moreover, restoration of aortic patency after camp release or completion of device deployment may prime a spinal cord reperfusion injury whose magnitude is related to the severity and duration of the ischemic insult. As a prophylactic measure during EVAR, CSF drainage is most commonly used only in selected patients, including those with synchronous or previous abdominal aortic repair.[32] As a treatment for postoperative paraplegia of delayed onset, CSF drainage often appeared to be useful[33-35] although not always effective.[32]

Another important issue is the great variability of the spinal cord blood supply. The significant role of the intercostal arteries between T8 and L2 is supported by the direct correlation between the risk of adverse neurologic events and TAA extent. Furthermore, the reimplantation of critical intercostal arteries has been shown to significantly lower the risk of late neurologic deficit[11] even though a low incidence of paraplegia (2%) was also reported in a series with no intercostal arteries reattachment.[15]

Overall, it appears that rather than being dependent on single segmental arteries, namely the artery of Adamkiewicz, spinal cord perfusion highly depends on a collateral circulation[36] that is extremely vulnerable to abrupt hemodynamic changes. In this respect, postoperative hypotension appears to be a major determinant of delayed-onset SCI.[37]

Thoracic endografts overlie and exclude the intercostal arteries originating from the aneurysm and the proximal and distal landing zones; thus, the coverage of a long thoracic aortic segment has been reported to be a significant risk factor for SCI.[32] Patients who have open AAA repair also appear to be prone to such a risk because of the marginal spinal cord collateral blood supply secondary to the ligation of lumbar arteries performed during the surgical procedure.[32,38] Also, proximal collateral circulation of the spinal cord may be put at risk by the occlusion of the left subclavian artery (landing zone 2) that abolishes the contribution to the blood supply provided by the anterior spinal artery, a branch of the ipsilateral vertebral artery. Finally, SCI could be precipitated by the late sealing of a type II endoleak[39] or late thrombosis of collateral pathways.

Table IV. Ancillary techniques for prevention of spinal cord ischemia during thoracic aortic repair

Distal aortic perfusion[21]
Hypothermic circulatory arrest[22]
Local hypothermia[23]
Pharmacologic agents
●steroids[24]
●naloxone hydrochloride[25]
●barbiturates[26]
●papaverine hydrochloride[27]
Spinal cord monitoring[28]
"Clamp-and-sew" technique[29]
Cerebrospinal fluid drainage[30]

To predict the risk of postoperative neurologic complications after thoracic EVAR, Kieffer et al[40] propose a preoperative angiographic assessment of intercostal arteries; however, this procedure appears to be cumbersome and carries the risk of embolization. The use of somatosensory evoked potential has also been advocated to achieve this goal.[28] The reported strategies include somatosensory evoked potential monitoring during a 20-minute deployment of a retrievable stent graft[41] or during a 15-minute segmental balloon occlusion of the aorta, with or without an external axillofemoral bypass.[42] The occurrence of ischemic changes during these tests may jeopardize the completion of the procedure, however, and the risk of embolization remains considerable.[32]

Our data showed that none of the considered preoperative risk factors were a determinant of paraplegia. These results have relevant clinical implications, because endovascular treatment has been most commonly indicated in poor surgical candidates whose severe comorbidities have the potential to influence neurologic outcome. The lack of collateral spinal cord blood supply through the lumbar arteries appears to be a relevant risk factor even though it did not reach statistical significance in our series. The intentional occlusion of the left subclavian was not a predictor of paraplegia and neither was the exclusion of an extensive thoracic aortic segment.

Among the perioperative variables, only a lowest MAP of <70 mm Hg was a significant determinant of delayed neurologic deficit. This finding is consistent with previous reports regarding thoracic and thoracoabdominal aortic open repair.[9,37]

The small number of events in our series limited the power of the statistical analyses. However, even as a merely descriptive report, our experience addresses the importance of hemodynamic control to prevent postoperative neurologic deficits and encourages aggressive, postoperative care of these patients. In our practice, we now try to maintain a perioperative MAP of >90 mm Hg[9] and use CSF drainage in patients deemed at high risk, including those who received AAA repair. In this respect, patients with synchronous thoracic and AAA, which we earlier treated simultaneously for both pathologies, currently undergo staged procedures to better allow the development of collaterals for spinal cord blood supply. In the case of delayed paraplegia, prompt CSF drainage, if not previously instituted, is also used to keep the CSF pressure <10 mm Hg and possibly reverse the deficit. Such drainage is maintained for 72 hours after open repair, as previously suggested.[30]

Our results show that elective thoracic aortic stent-graft repairs have a favorable impact on postoperative neurologic outcome, particularly with regards to severity of SCI. Ongoing technical developments of endovascular procedures, such as customized fenestrated grafts that incorporate intercostal arteries, may further reduce the risk of paraplegia that represents one of the ultimate benchmarks for thoracic EVAR as an alternative to traditional open repair.

We thank Dr Giliola Calori, Statistical Unit, Scientific Institute San Raffaele, for her help in statistical analysis of the data of the manuscript.

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Submitted Jan 20, 2005; accepted Apr 6, 2005.