Preoperative and operative predictors of delayed neurologic deficit following repair of thoracoabdominal aortic aneurysm

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Purpose: Delayed neurologic deficit has been recognized in recent years as a source of morbidity following thoracic and thoracoabdominal aortic repair. We wanted to find risk factors specifically significant for delayed neurologic deficit. In this initial study we looked at preoperative and operative risk factors.

Methods: We performed 854 thoracoabdominal aortic repairs between February 1991 and May 2001. For this study we excluded 26 patients who died before postoperative neurologic status could be evaluated and 38 who had immediate neurologic deficit on initial postoperative evaluation, leaving 790 consecutive patients. We evaluated a wide range of demographic, preoperative physiological and intraoperative data, using univariate and multivariable statistical analyses.

Results: Twenty-one of 790 (2.7%) patients had delayed neurologic deficit. Significant univariate predictors included preoperative renal dysfunction (odds ratio 5.9; $P < .006$), acute dissection (odds ratio 3.9; $P < .05$), extent II thoracoabdominal aorta (odds ratio 3.0; $P < .03$), and use of adjuncts (cerebrospinal fluid drainage and distal aortic perfusion; odds ratio 7.7; $P < .03$). The use of the adjuncts dropped from the multivariable model but all other factors remained. No other significant risk factors were identified. Twelve of 21 (57%) patients recovered neurologic function with optimization of blood pressure and cerebrospinal fluid drainage.

Conclusion: Preoperative renal dysfunction, acute dissection, and extent II thoracoabdominal aorta are significant predictors of delayed neurologic deficit. Previous studies have demonstrated that the use of adjuncts protects against immediate neurologic deficit. The findings of this study are consistent with the hypothesis that adjuncts reduce ischemic insult enough to prevent immediate neurologic deficit but that a period of increased spinal cord vulnerability persists several days postoperatively.

Although different surgical adjuncts, advanced anesthesia, and improved critical care have contributed to improved results, neurologic deficit following repairs of the thoracic and thoracoabdominal aorta (TAAA) remains a devastating complication. The incidence of postoperative neurologic deficit, as high as 16% to 25% in the era of clamp-and-go,¹⁻³ currently ranges between 5% and 16%, depending on the use of adjuncts and the extent of the aneurysm.⁴⁻⁶

Factors that have been identified as part of the multifactorial etiology of neurologic injury include aortic ischemic period, extent of the aneurysm, and use of adjuncts.^{1,7-10}

Neurologic deficit following TAAA repair can be categorized by the degree of injury (paraplegia or paraparesis) and by the time of onset. Immediate neurologic deficit refers to paraplegia or paraparesis upon awakening from anesthesia. Delayed neurologic deficit (DND) occurs after a patient has recovered from anesthesia and has been evaluated as neurologically intact yet develops paraplegia or paraparesis several hours or days later. The distinction between immediate and delayed onset is important because the etiology involved may be different.¹¹ In addition, the prognosis for immediate neurologic deficit is poor with unlikely neurologic recovery and an increased risk of early death.^{2,8,11,12} However, DND may be reversed if identified early and treated promptly with therapeutic measures such as cerebrospinal fluid (CSF) drainage and optimization of blood pressure and oxygen delivery.^{8,11,13,14}

DND was identified as a postoperative complication in the late 1980s and has been reported to occur in 1% to 12% of patients following TAAA graft replacement.^{10,14-17} Cases of DND have also been reported after endovascular repair.¹⁸⁻²⁰ We previously reported the positive impact of CSF drainage on recovery from DND.^{11,12} Few reports, however, have analyzed the risk factors associated with DND. The purpose of this study was to analyze the preoperative and operative predictors of DND following TAAA repair.

Material and Methods

Between February 1991 and May 2001, 854 patients underwent graft repair of the descending thoracic or TAAA. Thirty-eight (4.4%) cases of immediate neurologic deficit were excluded from the analysis. In addition, 26 (3.0%) patients who died intraoperatively before neurologic assessment were also excluded. We performed a review of the prospectively collected data of 790 patients, studying the entire series to test our clinical impression that the incidence of DND has been increasing with greater adjunct usage.

Outcome Variables

Paraplegia or paraparesis upon awakening, regardless of the severity, defined immediate neurologic deficit. Paraplegia or paraparesis after a period of normal neurologic function classified DND. Aortic dissection accompanying aortic aneurysms was considered acute if surgery was performed within less than 14 days from the onset of pain and chronic if after 14 days. Cerebrovascular disease was defined by a history of stroke, transient ischemic attack, or intervention for carotid artery disease. A history of chronic bronchitis and emphysema, or <60% of predicted forced expired volume in 1 second, identified chronic obstructive pulmonary disease. A serum creatinine level of greater than 2.0 mg/dL or the need for dialysis characterized renal dysfunction. Surgery was considered emergent when performed for symptomatic, contained, or free ruptured TAAA. Previous aneurysm repair referred to any

previous replacement of the ascending, transverse arch, descending thoracic, thoracoabdominal, or abdominal aorta.

Technique

Descending thoracic and thoracoabdominal aortic repair was performed according to our standard surgical protocol, as described previously.⁵ In September 1992, based on our experience with animal studies and on reports that were beginning to appear in the literature, we changed our surgical protocol to incorporate distal aortic perfusion and CSF drainage. This technique allows increased time to evaluate and manage the intercostal arteries. Prior to September 1992, we utilized the clamp-and-go technique.

All patent lower thoracic intercostal arteries (level T8-T12) were reattached when possible (about 75% of all extent I and II cases, 60% of extent III and V cases, and about 9% of type IV cases). Extent IV and descending thoracic aortic aneurysms of the upper portion of the aorta seldom warrant intercostal artery reattachment. Occluded arteries and aortic dissections also rule out the feasibility of reimplantation. Active visceral and renal cooling was performed in addition to moderate systemic hypothermia.

The patient was anesthetized and intubated using a double lumen endotracheal tube. An arterial line and a pulmonary artery catheter monitored patient hemodynamics through central venous access. A CSF catheter (NMT, Neurosciences, Duluth, Ga) placed in the third or fourth lumbar space provided CSF drainage and monitoring of CSF pressure. The CSF pressure was maintained at less than 10 mm Hg by allowing passive CSF drainage. Mean arterial pressure (MAP) was maintained between 60 and 85 mm Hg by manipulating cardiac preload and afterload. During cross-clamping, MAP was controlled by adjusting the flow rate of distal aortic perfusion and the administration of preload as required. Care was taken to prevent the temperature from drifting below 34°C. After completion of the repair the patient was warmed to 36°C core body temperature and distal aortic perfusion was discontinued.

Since September 1992 we have used CSF drainage for all elective and urgent repairs of TAAA and descending thoracic aortic aneurysms. Contraindications for CSF drainage included cases of free rupture, hypotension, sepsis or active bacteremia, recent history of intracerebral hemorrhage, or previous spinal surgery. CSF pressure was recorded at the insertion of the CSF drain, prior to initiation of distal aortic perfusion, at bypass, crossclamping, 10-minute intervals during crossclamping, release of clamp, and at closure when the patient was warmed.

The CSF drain was kept in place postoperatively while the patient was in the intensive care unit and removed after 3 days if no neurologic deficits were encountered. CSF was drained to maintain pressure below 10 mm Hg. Neurologic function was monitored on an hourly basis while the CSF drain was in place. The surveillance was reduced to once every 2 to 4 hours if normal function was documented.

If a new neurologic deficit was encountered after discontinuation of the drain, the CSF drain was reinserted by the anesthesiologist and gravity drainage was initiated. The patient was placed in the recumbent position to improve drainage. Hemodynamics and oxygen delivery were optimized by increasing serum hemoglobin to greater than 10 mg/dL and by improving oxygen saturation and cardiac output. Volume status was optimized and MAP

TABLE 1. Modified Tarlov scoring scale

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

maintained at greater than 90 mm Hg. Magnetic resonance imaging of the spine was obtained to exclude the possibility of spinal hematoma or other causes of cord compression and to identify spinal cord infarction.

An independent neurologist performed a comprehensive neurologic examination to identify the level of deficit. This involved the assessment of the patient's motor skills classified according to a modified Tarlov scale²¹ (Table 1). Paraplegia was classified by a Tarlov score of 0, 1, or 2; paraparesis by a score of 3 or 4. An increase in the Tarlov score of 2 or more upon discharge or transfer denoted improvement in the neurologic deficit.¹¹

Statistical Analysis

Data were collected from chart reviews done by a trained nurse abstractor and were entered into a dedicated Microsoft Access database. Analysis was retrospective. Data were exported to SAS for data analysis, and all computations were performed using SAS version 6.12 running under Windows NT. Univariate categorical data were analyzed using contingency table analyses. For 2×2 tables, common odds ratios with test-based confidence intervals were computed, and chi-square statistics are reported for hypothesis tests. For tables greater than 2×2 , chi-square statistics were computed for hypothesis tests, and univariate logistic regression estimates were also computed keeping data in their native continuous distribution. *P* values and confidence intervals for continuous data are based on maximum-likelihood estimates.

Results

Patient characteristics and significant intraoperative data are illustrated in Table 2. The overall incidence of neurologic deficit following repair of descending thoracic and TAAA was 6.9% (59/854), immediate neurologic deficit was 4.4% (38/854), and DND was 2.7% (21/790). In the patients with DND, the 30-day mortality rate was 38.1% (8/21). In-hospital mortality in these patients was 47.6% (10/21).

Of the 21 patients who suffered DND, 12 were men and nine were women. The mean age of patients was 66 years (range, 35-82 years). In the DND group, 9 (42.6%) patients were extent II TAAA, 7 (33.3%) extent I, 2 (9.5%) extent III, 2 (9.5%) descending thoracic, and 1 (4.8%) extent V.

DND occurred at a median of 2 days postoperatively with a range of 1 to 14 days.

Univariate analysis identified extent II TAAA (OR, 2.98; 95% CI, 1.23-7.18; *P* < .03), preoperative renal insufficiency (OR, 5.87; 95% CI, 2.35-14.62; *P* < .006), acute dissection (OR, 3.97; 95% CI, 1.09-14.19; *P* < .05), and adjuncts (OR, 7.71; 95% CI, 1.03-57.82; *P* < .03) as associated factors for DND (Table 2). Independent predictors of DND as identified by multiple logistic regression were extent II (OR, 3.21; 95% CI, 1.29-7.97; *P* < .012), acute dissection (OR, 5.25; 95% CI, 1.42-19.47; *P* < .013), and preoperative renal dysfunction (OR, 18.10; 95% CI, 3.09-106.00; *P* < .0013; Table 3). Distal aortic perfusion time, aortic crossclamp time, and intercostal artery reattachment were not associated with DND.

Overall improvement of neurologic function in patients with DND was 57% (12/21). With regard to the use of CSF drainage, 19 of the 21 cases of DND had a drain in place or reinserted at the time of identification of the deficit. In patients that had the CSF drain still in place when the neurologic deficit developed, 75% (9/12) noted a significant improvement in neurologic function upon discharge (Figure 1). In patients who required reinsertion of the CSF drain when the DND occurred, 43% (3/7) noted an improvement. Neither of the 2 patients who did not have CSF drainage upon discovery of the delayed deficit recovered significant function.

Discussion

Neurologic deficit, or paraplegia and paraparesis, remains one of the most dreaded complications of TAAA surgery. Disconcerting is the fact that after successful repair and a normal initial recovery with no evidence of neurologic dysfunction, the spinal cord remains vulnerable to ischemia and patients may still suffer neurologic insults. The complication of DND was first reported to have an incidence from 8% to 12% during thoracoabdominal repair.¹⁶ Others have also recognized and reported this complication.^{8,13,18,20,22,23}

In the clamp-and-go era of TAAA repair, the incidence of neurologic deficit ranged from 15% to 25%, depending on the extent of aneurysm,¹⁻³ with rates as high as 31% in extent II TAAA.² With adjunctive strategies to protect the spinal cord from ischemia the incidence has improved, falling to between 5% and 10%.⁴⁻⁶ Despite the use of adjuncts, the incidence of DND remains significant, comprising 36% (21/59) of overall neurologic deficits after repair in this series and comparable to a previous prospective study that identified delayed deficits to be one third of all neurologic deficits.¹⁷

As little information exists regarding DND, we sought to identify the preoperative and operative factors that may predispose patients to this complication. Using multivari-

TABLE 2. Univariate analysis of preoperative and operative factors for delayed neurologic deficit

Variable	No. patients (%)	No. delayed (%)	Odds ratio*	95% CI†	P‡
All patients	790 (100)	21 (2.7)			
Age					
8-59	201 (25.4)	4 (2.0)	1.01	0.98-1.05	.23
60-68	237 (30.0)	4 (1.7)			(0.52)
69-74	173 (21.9)	8 (4.6)			
75-91	179 (22.7)	5 (2.8)			
Gender					
Female	299 (37.8)	9 (3.0)	1.24	0.52-2.98	.66
Male	491 (62.2)	12 (2.4)	1		
COPD	245 (31.0)	3 (1.2)	0.37	0.11-1.24	.15
No COPD	545 (69.0)	18 (3.3)	1		
Cerebrovascular disease	80 (10.1)	3 (3.8)	1.50	0.43-5.20	.46
No cerebrovascular disease	710 (89.9)	18 (2.5)	1		
Emergency	44 (5.6)	0 (0.0)	0.38	0.02-6.36	.63
No emergency	746 (94.4)	21 (2.9)	1		
TAAAI	187 (23.7)	7 (3.7)	1.63	0.65-4.12	.30
Other	603 (76.3)	14 (2.3)	1		
TAAAI	164 (20.8)	9 (5.5)	2.97	1.23-7.18	.03
Other	626 (79.2)	12 (1.9)	1		
TAAAI	63 (7.9)	2 (3.2)	1.22	0.28-5.37	.68
Other	727 (92.1)	19 (2.6)	1		
TAAAI	112 (14.2)	0 (0.0)	0.14§	0.01-2.26	.06
Other	678 (85.8)	21 (3.1)	1		
TAAAV	34 (4.3)	1 (2.9)	1.12	0.15-8.56	.61
Other	756 (95.7)	20 (2.7)	1		
Descending	195 (24.7)	2 (1.0)	0.31	0.07-1.36	.13
Other	595 (75.3)	19 (3.2)	1		
Acute dissection	34 (4.3)	3 (8.8)	3.97	1.09-14.19	.05
No acute dissection	756 (95.7)	18 (2.4)	1		
Chronic dissection	200 (25.3)	8 (4.0)	1.85	0.76-4.53	.20
No chronic dissection	590 (74.7)	13 (2.2)			
Renal insufficiency	81 (10.3)	8 (9.9)	5.87	2.35-14.62	.006
No renal insufficiency	709 (89.7)	13 (1.8)	1		

COPD, Chronic obstructive pulmonary disease; TAAAI, thoracoabdominal aortic aneurysm.

* For dichotomous variables, the odds ratio represents a test against a reference category whose referent odds ratio is equal to 1. For continuous data, the odds ratio refers to the increase in odds associated with a 1-unit increase in the variable value. Although continuous data are presented in quartiles, the odds ratios are against the continuous variable.

† 95% CI = 95% confidence interval. This reflects the units against which its companion odds ratio is computed. Confidence intervals are test-based.

‡ P = probability of type I statistical error (common P value). Values without parentheses are Pearson chi-square probabilities. Probability values in parentheses are univariate logistic regression likelihood ratio P values.

§ Odds ratio computed using the logit method with a zero-cell correction.

TABLE 2. Continued

Variable	No. patients (%)	No. delayed (%)	Odds ratio*	95% CI†	P‡
Intercostal artery reattachment	411 (52.0)	13 (3.2)	1.51	0.62-3.70	.36
No intercostal artery reattachment	379 (48.0)	8 (2.1)	1		
Clamp time (min)					
Not available	14 (1.8)	0 (0.0)	1.01	0.99-1.03	.73
7-28	190 (24.1)	4 (2.1)			(.13)
29-39	185 (23.4)	5 (2.7)			
40-55	205 (25.9)	5 (2.4)			
56-154	196 (24.8)	7 (3.6)			
Pump time (min)					
Not used	172 (21.8)	3 (1.7)	1.01	1.00-1.03	.09
7-39	152 (19.2)	3 (1.9)			(.05)
40-54	150 (19.0)	2 (1.3)			
55-73	161 (20.4)	4 (2.5)			
74-196	155 (19.6)	9 (5.8)			
Adjunct	575 (72.8)	20 (3.5)	7.71	1.03-57.82	.03
Adjunct not used	215 (27.2)	1 (0.5)	1		
Prior aneurysm repair	334 (42.3)	6 (1.8)	0.54	0.21-1.40	.27
No prior aneurysm repair	456 (57.7)	15 (3.3)	1		

TABLE 3. Multiple logistic regression model for preoperative and operative predictors of delayed neurologic deficit

Variable	Parameter estimate	Adjusted odds ratio	95% CI	P
Intercept	-4.1991			
TAAAI	1.1660	3.21	1.29-7.97	.0120
Renal insufficiency	2.8961	18.10	3.09-106.00	.0013
Acute dissection	1.6587	5.25	1.42-19.47	.0131

TAAAI, Extent II thoracoabdominal aortic aneurysm; renal insufficiency, preoperative creatinine >2.0 mg/dL or dialysis-dependent.

TABLE 4. Recovery by drain status

Variable	No. patients (%)	No. recovered (%)	Odds ratio	95% CI	P
In place	12 (63.2)	9 (75.0)	4.00	0.55-29.18	0.33
Reinserted	7 (36.8)	3 (42.9)	1		

able analysis, we found acute dissection, extent II TAAA, and preoperative renal dysfunction to be independent predictors of DND. These risk factors are similar to the risk factors that are associated with immediate neurologic deficit.⁵ This suggests that patients at risk for immediate neurologic deficit may also be at risk for DND. Thus, even after successful TAAA repair, patients with these risk factors require close follow-up to guard against the development of DND.

Other researchers as well as ourselves have found that the use of the adjuncts (distal aortic perfusion and CSF drainage) reduces the overall incidence of neurologic deficit.^{4,5,24} In this study univariate analysis showed us that adjuncts were associated with an increased risk of DND ($P < .03$). This suggests a patient who would have developed immediate neurologic deficit previous to the use of adjuncts might now recover from repair with intact but vulnerable neurologic function. That is, intraoperative ischemic insult

is mitigated but not eliminated by the adjuncts. In the postoperative period this patient then suffers an insult such as an episode of hypotension that leads to further ischemia, resulting in the delayed deficit. So although distal aortic perfusion and CSF drainage may minimize spinal cord ischemia during the operative repair, the adjuncts do not appear to prevent DND.

Postoperative factors that may incite DND include hypotension, systemic inflammatory response syndrome, sepsis, cardiac dysrhythmias or cardiac failure, and diminished oxygen delivery caused by anemia, hypoxia, and low cardiac output. The importance of maintaining adequate blood pressure postoperatively for collateral circulation of the spinal cord has been emphasized previously.^{2,25} With regard to prognosis, the distinction between immediate and delayed neurologic deficit is important. The prospects for a patient with immediate neurologic deficit are poor since subsequent recovery is rare and early death likely.¹⁶ If DND goes unresolved the patient fares no better. However, DND may be reversed if identified early and if immediate countermeasures are undertaken (ie, CSF drainage, increase of blood pressure, and improvement of oxygen delivery).^{8,11,13,14} This was demonstrated in this study with a 57% (12/21) improvement in neurologic function if the CSF drain was utilized. When the CSF drain was in place at the time of the DND, 75% (9/12) of patients recovered function. When the CSF drain was reinserted at the time of the DND due to previous removal or malfunction, 43% (3/7) recovered neurologic function (Table 4). Reinsertion of the drain takes time. It requires assembly of the anesthesia team, preparation of the area, and so on, which can take an hour or more to accomplish. Our drain maintenance protocol requires that CSF pressure be kept at 10 mm Hg for 3 days postoperatively. When a delayed deficit is recognized in a patient with a drain in place, we open the device to drain freely, which can be done immediately. We think the timing of the intervention is responsible for the apparent difference between the success of CSF compartment decompression in the drain-in and drain-reinsertion groups, but the numbers are too small to test this formally at the present. The 2 patients who did not receive a CSF drain when the deficit occurred demonstrated no recovery at all. The significance of CSF drainage in immediate neurologic deficit has been established,⁴ but here again demonstrates a possible beneficial effect when used for DND. The improvement in neurologic recovery when the CSF drain was used suggests that DND may be caused, at least in part, by increased CSF pressure.

The importance of the collateral circulation to the spinal cord has been emphasized previously^{6,10} and may be significant with respect to DND. Reattachment of important segmental arteries (intercostal arteries T8-T12) has been demonstrated to be protective.²⁵ Although it has been sug-

Figure 1. Percent recovery with delayed neurologic deficit.

gested that endovascular repair of descending thoracic aortic aneurysms may be beneficial in patients who are deemed nonsurgical candidates, significant immediate neurologic deficit has been reported.²⁰ During endovascular repair of descending aneurysms, although no aortic ischemic period is involved, reattachment of important segmental arteries is not performed. This may be responsible for neurologic deficits. In addition, DND has also been reported after endovascular descending aneurysm repair with neurologic recovery after insertion of a CSF drain.¹⁸ Collateral circulation of the spine (cord, lumbar arteries, and iliac arteries) may be more significant than previously appreciated.¹⁰

Despite its demonstrable benefits, CSF drainage is not an entirely benign procedure. Since integrating CSF drainage into our surgical protocol we have had 3 major complications in patients with CSF drainage (2 subdural bleeds and 1 case of meningitis) that may have been related to drainage. We have also had 1 case of a spinal hematoma at the insertion site that required a laminectomy, which was certainly related to CSF drainage. Clearly, judicious management of the drain is incumbent on clinicians who choose to use it.

Limitations of this study included its retrospective, non-randomized design. Although the study group was of ample size (790 cases), the group that suffered DND was small (21) and any conclusions drawn from this group must be taken with caution. A better understanding of DND will need clinical studies analyzing the postoperative events involved as well as the development of an animal model for delayed spinal cord injury.

Conclusion

This study demonstrated that preoperative renal dysfunction, acute dissection, and extent II TAAA were significant predictors of DND. Although the adjuncts distal aortic perfusion and CSF drainage may protect against immediate neurologic deficit, they did not prevent delayed deficits. Postoperatively, it appears that the spinal cord may remain vulnerable to ischemia for some time. If DND does occur,

immediate identification and reduction of the CSF pressure by drainage may be beneficial.

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Discussion

Dr Lars G. Svensson (Cleveland, Ohio). I would like to thank the Association for the invitation to comment on the interesting and vexing problem of delayed deficits presented by Dr Safi in a large series of descending and thoracoabdominal aortic repairs. Unfortunately, they did not present the postoperative causes. We have seen it occur with hypotension from things like bleeding, arrhythmia, overdiuresis, sedation, reintubation, and other causes such as pulmonary emboli, arterial embolic showers, thrombosis of the intercostal, or lumbar artery patches or bypasses, and, in some patients, for no apparent reason.

There appear to be at least 3 common pathways: first, loss of spinal cord blood flow, particularly with a tenuous and perilous collateral blood supply; second, failure of adequate spinal cord oxygenation; and third, secondary spinal cord injury from the delayed complex biochemical cascade of deleterious pathways following ischemia and reperfusion.

The preoperative risk factors presented by Dr Safi of type II thoracoabdominal aneurysm, acute dissection, and renal disease reflect the problem of maintaining collateral blood supply in some of these patients. Because you only reattach intercostals from T8 to T12, according to your manuscript, do you think reattaching a wider range of intercostals or lumbar arteries may have reduced the risk? What are you now doing differently? We have found postoperative induced hypertension and delayed extubation useful. Do you think in patients with extensive aneurysms and few intercostal or lumbar artery ostia, endarterectomies should be performed for reattaching segmental arteries?

You had more deficits occur with CSF drainage. Do you think delayed CSF hypertension may be a factor? In a previous prospective randomized study in which postoperative hypotension resulted in 32% of the deficits, CSF drainage tended to be protective. In an excellent study recently published by Coselli, there also appeared to be some possible protection against delayed deficits. In 57% of your patients, delayed deficits improved with repeat drainage. Do you think that delayed removal of the catheter may be useful or do you think this would increase the risk of complications from the catheter too much?