Observations on delayed neurologic deficit after thoracoabdominal aortic aneurysm repair

Hazim J. Safi, MD, Charles C. Miller III, PhD, Ali Azizzadeh, MS, and **Dimitrios C. Iliopoulos, MD,** *Houston, Tex.*

Purpose: **To describe the phenomenon of delayed-onset neurologic deficit after thoracoabdominal aortic aneurysm repair and to discuss management of this type of deficit in a case series.**

Methods: **Since September 1992 we have used cerebrospinal fluid drainage and distal aortic perfusion routinely to reduce the risk of neurologic deficit in thoracoabdominal aortic aneurysm patients. All patent intercostal arteries were reattached when this was technically feasible. Delayed neurologic complications occurred in eight patients who underwent operation for thoracoabdominal aortic aneurysm between September 1992 and March 1997, between 1 and 14 days after awakening from anesthesia. All patients had immediate cerebrospinal fluid drainage on recognition of their symptoms.**

Results: **Patients were evaluated by an independent neurologist and were classified by a modified Tarlov score between 0 and 5. All eight patients improved at least two points by discharge. The mean change in Tarlov score from onset to discharge was 2.4 ± 1.1 (p = 0.008).**

Conclusions: **Cerebrospinal fluid drainage significantly improved late-onset neurologic deficit that occurred between 1 day and 2 weeks after operation in our series. Immediate drainage should be considered when signs of neurologic deficit first begin to appear.**
(J Vasc Surg 1997;26:616-22.)

The phenomenon of delayed neurologic deficit after thoracoabdominal aortic aneurysm repair was first documented fairly recently.[1] The treatment of this entity, like the treatment of immediate neurologic deficit, used blood pressure stabilization, steroids, and intensive physical therapy, but these measures met with limited success. Delayed neurologic deficit, it was observed at the time, was beyond the surgeon's control.[1] A later randomized prospective study of intraoperative cerebrospinal fluid drainage by this same group did little to disprove this statement.[2]

Combined with distal aortic perfusion, we have found cerebrospinal fluid drainage to significantly lower the overall incidence of neurologic deficits, whether early or delayed.[3] Since publishing this prospective study, we have combined the traditional supportive therapies for delayed neurologic deficit with the free drainage of cerebrospinal fluid. Delayed neurologic deficit is generally regarded as a complex phenomenon, related to arterial blood pressure control and postoperative bleeding. The rationale for the use of cerebrospinal fluid drainage after operation is to decrease spinal cord edema, which may be an important cause of delayed neurologic deficit. As we know from experimental and clinical studies, the effect on the spinal cord of clamping the aorta is twofold.[4-7] First, spinal artery pressure falls, resulting in decreased spinal cord perfusion; and second, cerebrospinal fluid pressure goes up, leading to further deterioration of blood flow to the spinal cord. Because the spinal cord is housed in a rigid structure, we propose that the condition is similar to postoperative brain edema and compartmental syndrome and that it requires urgent decompression.

There are two anecdotal reports in the literature that address the phenomenon of delayed neurologic deficit and reversal using cerebrospinal fluid drainage.[8,9] These earlier reports suggest that an adjunct exists that may have a beneficial impact on delayed neurologic deficit. We reviewed our experience in managing patients who had delayed neurologic deficit and examined the impact of cerebrospinal fluid

From the Methodist Hospital, Baylor College of Medicine.
Reprint requests: Hazim J. Safi, MD, Baylor College of Medicine, 6550 Fannin, Suite 1603, Houston, TX 77030.

0741-5214/97/$5.00 + 0 **24/1/84438**

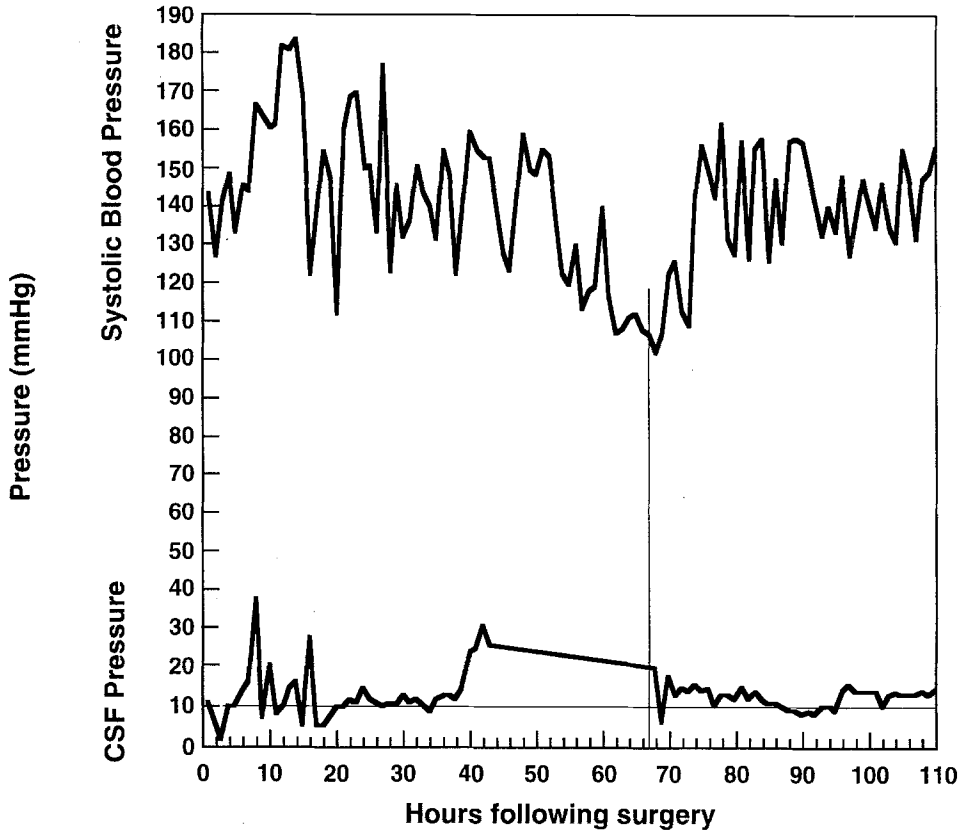

Fig. 1. Systolic arterial blood pressure and cerebrospinal fluid pressure tracings for patient #1 in Table I. *Vertical reference line* shows when neurologic deficit was first noted. *Horizontal reference line* at 10 mm Hg represents target postoperative cerebrospinal fluid pressure.

Table I. Descriptive statistics

Patient no.	*Age (yr)*	*Sex*	*Type*	*Dissection*	*Clamp time (min)*	*Intercostal arteries*		*Tarlov | Day*	
						Reattached	*Ligated*	*Onset*	*Discharge*
1	75	F	TAAAII	Chronic	32	10-12	None	1|3	5|14
2	63	F	TAAAI	None	38	10-11	4-9	0|2	5|7
3	40	F	TAAAII	Chronic	73	8-12	None	1|3	3|17
4	71	M	TAAAII	Chronic	91	7-12	None	0|3	2|8
5	35	M	TAAAI	Chronic	52	12	8-10	2|1	4|13
6	82	M	TAAAI	None	19	9-12	4-7	0|4	2|8*
7	72	M	TAAAI	Acute	36	9, 11-12	6-8,10	0|4	2|6
8	75	F	TAAAI	None	29	11-12	None	0|14	2|26

Patient number corresponds to figure number. Intercostal artery numbers under the headings "reattached" and "ligated" refer to the intercostal spaces in which the arteries are found. Intercostal arteries not listed were not patent and did not require management. Tarlov | day for onset and discharge is read as follows: The number to the left of the pipe (|) is Tarlov score. The number to the right of the pipe is the postoperative day on which the Tarlov was recorded. The left-hand column reports Tarlov scores on the day the deficit was discovered. The right hand column shows Tarlov scores on the day the patient was discharged.
*This patient returned to normal function (Tarlov score 5) 6 months after discharge after intensive rehabilitation.

drainage combined with supportive therapies on postoperative neurologic outcome.

MATERIALS AND METHODS

From September 1992 to the present, cerebrospinal fluid drainage and distal aortic perfusion have been used for all of our thoracoabdominal aortic aneurysm patients. Between September 1992 and March 1997, delayed neurologic deficit developed in eight patients after surgery, between 1 and 14 days after awakening from anesthesia. Descriptive statistics are shown in Table I. Delayed neurologic deficit

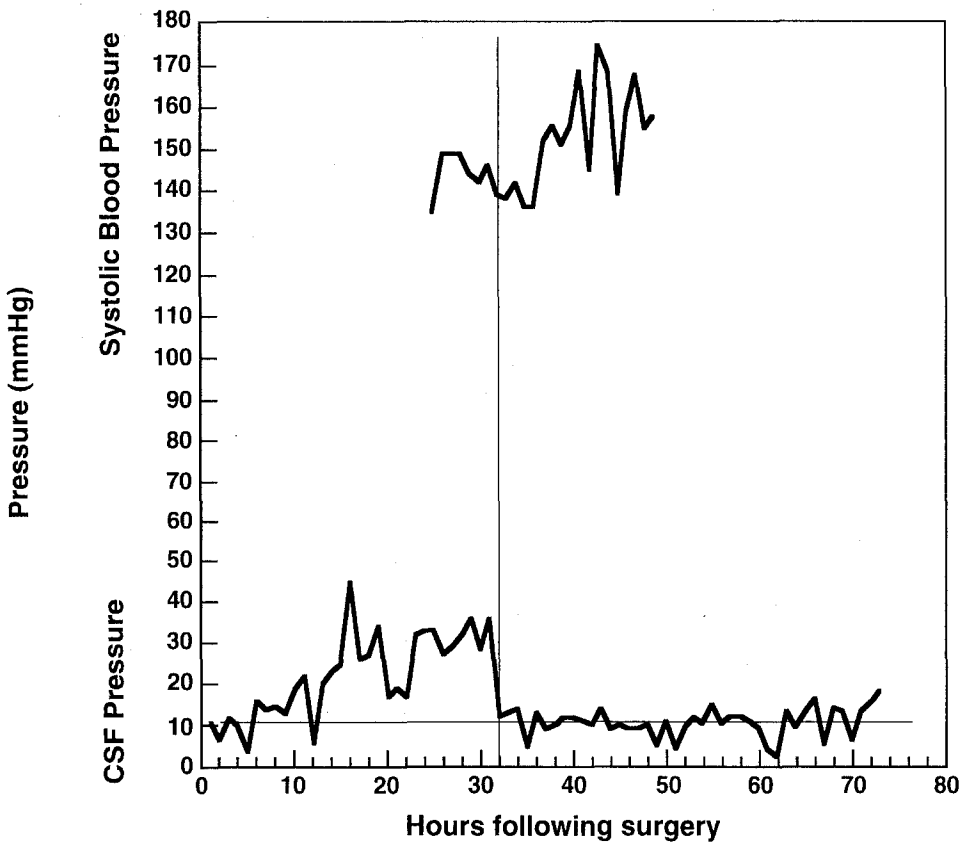

Fig. 2. Systolic arterial blood pressure and cerebrospinal fluid pressure tracings for patient #2 in Table I. *Vertical reference line* shows when neurologic deficit was first noted. *Horizontal reference line* at 10 mm Hg represents target postoperative cerebrospinal fluid pressure.

occurred in equal numbers among male and female patients. The patients' median age was 71.5 years (range, 35 to 82 years). Associated diseases were hypertension in seven patients, pulmonary disease in two, and renal disease in two. Five patients had type I thoracoabdominal aortic aneurysms that extended from just below the left subclavian artery to above the renal arteries, and three patients had type II thoracoabdominal aortic aneurysms that extended from immediately below the left subclavian artery to below the renal arteries. Five patients had aortic dissection: two chronic type B, one acute type B, and two chronic type A. Previous operations were repair of the ascending aorta and aortic valve, ascending aortic graft replacement and kidney transplant, abdominal aortic aneurysm repair, and graft replacement of the descending thoracic aorta. Three patients were symptomatic on presentation.

Technique. The operative technique of thoracoabdominal aortic aneurysm repair is described in detail elsewhere.[10] Left atrial–to–left femoral bypass grafting using a BioMedicus pump (BioMedicus, Minneapolis) and perioperative cerebrospinal fluid drainage accompanied all operations. A Cordis lumbar drain kit (Cordis Corp., Miami, Fla.) was used for cerebrospinal fluid drainage. The patient was placed in a lateral position, and a 14-gauge Tuohy needle was introduced in the intervertebral space between L3 and L4. The position of the Tuohy needle in the subarachnoid space was confirmed by free flow of cerebrospinal fluid from the needle. Pressure was then recorded and monitored continuously for the remainder of the procedure, and was drained if it rose above 10 mm Hg.

Operative data. The median aortic clamp time was 37 minutes (range, 19 to 91 minutes). The median pump time was 63 minutes (range, 35 to 113 minutes). The median lowest rectal temperatures for moderate hypothermia, used for six patients, was 33.8° C (range, 33.2° to 34.7° C). All patent intercostal arteries between T7 and T12 were reimplanted in all patients.

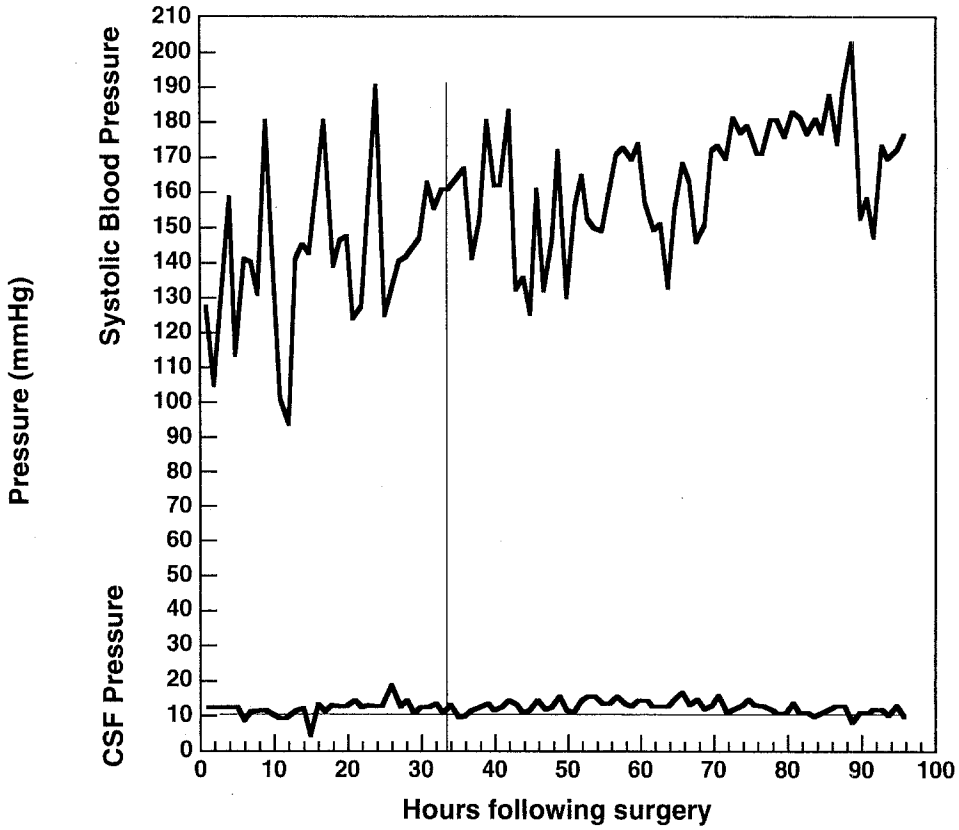

Fig. 3. Systolic arterial blood pressure and cerebrospinal fluid pressure tracings for patient #3 in Table I. *Vertical reference line* shows when neurologic deficit was first noted. *Horizontal reference line* at 10 mm Hg represents target postoperative cerebrospinal fluid pressure.

Postoperative course. The Tuohy needle remained in position for 3 days after the operation. Patients were evaluated after the operation by an independent neurologist. All eight patients were neurologically intact on awakening, but within 1 to 14 days neurologic deficit developed. The patients were classified according to the following modified Tarlov score, ranging between 0 and 5: 0, no motion; 1, motion without gravity; 2, motion against gravity; 3, able to stand with assistance; 4, able to stand and walk with assistance; and 5, normal. A Tarlov score of 0 to 2 was defined as paraplegia, and a score from 3 to 4 was defined as paraparesis. Paraplegia developed in five patients, who were unable to move one or both legs (score, 0), and a slightly less severe deficit developed in three, able to move limbs only while supported (score, 1).

Of the six patients for whom on-line pressure data were available, three had acutely unstable blood pressure episodes (pneumothorax in two, hypotension as a result of bleeding in one) before onset of the neurologic deficit. At the onset of symptoms, all had a rise in cerebrospinal fluid pressure. Figs. 1 through 5 illustrate the cerebrospinal fluid pressure and hours after surgery at which late neurologic deficit developed for six patients. Figure numbers correspond to the patient numbers in Table I. All eight patients were treated in an identical fashion on recognition of their symptoms, with immediate cerebrospinal fluid drainage and no pharmacologic intervention. Cerebrospinal fluid pressure was checked hourly and fluid drained freely after discovery of neurologic deficit. After a 2- to 3-day period of rest, patients gradually mobilized. When cerebrospinal fluid pressure and blood pressure were stabilized the catheter was withdrawn, and at this time patients were placed on a regimen of intense physical therapy. The postoperative period of drainage was no more than 4 days for any of these patients. On discharge, patients #1 and #2 had complete recovery (score, 5), patient #5 near recovery (score, 4), patient #3 was able to stand with assistance (score, 3), and three patients (#6, #7, #8) after complete paralysis were able to move lower limbs (score, 2). Although recovered to score #2

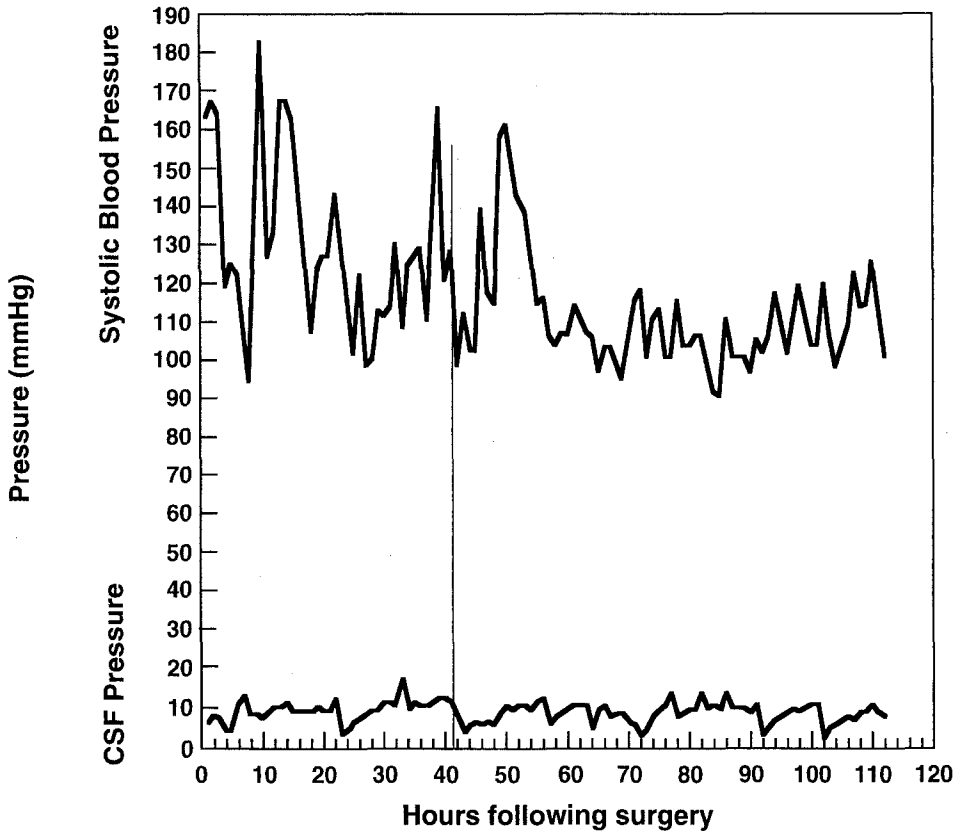

Fig. 4. Systolic arterial blood pressure and cerebrospinal fluid pressure tracings for patient #4 in Table I. *Vertical reference line* shows when neurologic deficit was first noted. *Horizontal reference line* at 10 mm Hg represents target postoperative cerebrospinal fluid pressure.

status, patient #4, who had been admitted with chronic renal failure, died in the hospital 3 months after surgery from multiple organ system failure. Patient #6, who was discharged to a physical rehabilitation institution 6 days after surgery, made a complete recovery (score, 5) within 6 months.

DISCUSSION

The infrequent but ever-dreaded neurologic complication that occurs as a consequence of thoracoabdominal aortic aneurysm repair most commonly becomes evident when the patient awakes, immediately after surgery. Less often, this sort of complication will first appear several hours or sometimes days after surgery. Why the onset is delayed is uncertain. Steroids, physical therapy, and methods of blood pressure stabilization have been ineffective in reversing this discouraging outcome. We know of no cases of spontaneous improvement.

The eight patients described here who experienced delayed neurologic deficit had aneurysms of the extent considered at highest risk for neurologic complications; five patients also had aortic dissection. Intercostal arteries were reattached in all eight patients. After awakening neurologically intact, cerebrospinal fluid pressure rose above 10 mm Hg hours or in some cases days later, and then the deficits developed.

We suggest that this complication is a result of a spinal cord compartmental syndrome that may arise from unstable blood pressure, increased cerebrospinal fluid pressure, or both. When cerebrospinal fluid pressure exceeds spinal arterial pressure, spinal perfusion may be reduced, leading to neurologic deficit. Possibly, edema set into motion by the aortic cross-clamp, but initially held at bay by intercostal artery reattachment and cerebrospinal fluid drainage (and distal aortic perfusion), was later aggravated by multifactorial causes.[11] Fig. 6 represents our hypothesis regarding the etiologic mechanism of late-onset neurologic deficit. Paradoxically, as improved operative techniques lead to reduction of the rate of immediate neurologic deficit, the proportion of late-onset cases may increase.

We know of no reports of neurologic deficit reversal using methods other than cerebrospinal fluid

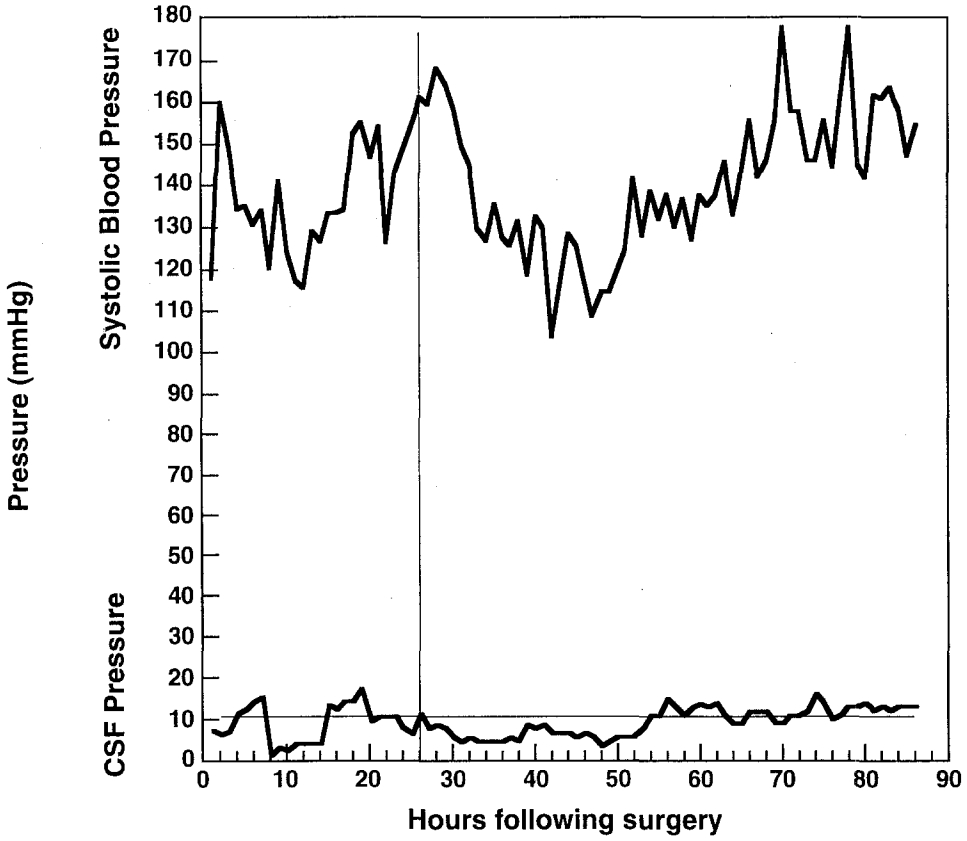

Fig. 5. Systolic arterial blood pressure and cerebrospinal fluid pressure tracings for patient #5 in Table I. *Vertical reference line* shows when neurologic deficit was first noted. *Horizontal reference line* at 10 mm Hg represents target postoperative cerebrospinal fluid pressure.

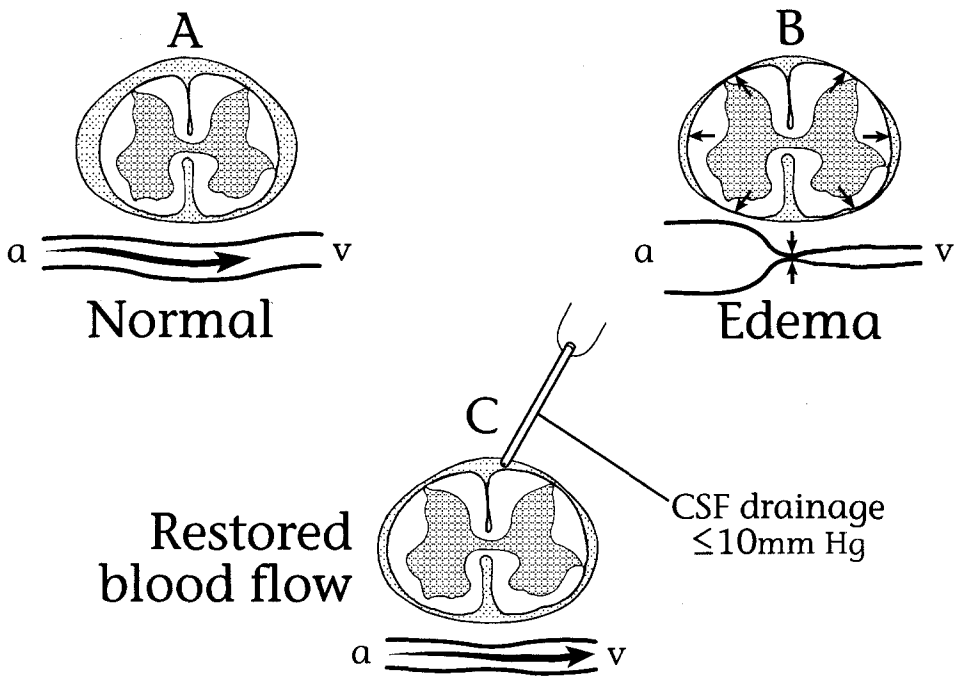

Fig. 6. A, Normal state. **B,** Spinal cord edema after surgical low-flow state and compartment-like syndrome. **C,** Cerebrospinal fluid drainage and restoration of blood flow.

drainage. Hollier and associates[8] reported the resolution of a single case of delayed paraparesis using cerebrospinal fluid drainage in 1992. More recently, two similar episodes were reported by Hill et al.[9] Of interest in these two cases, cerebrospinal fluid drainage was used only after operation, and there was no intercostal artery reattachment. Other reports have speculated on the potential of cerebrospinal fluid drainage to decompress spinal cord swelling and reverse neurologic deficit.[12]

We believe that critical to the success of cerebrospinal fluid drainage is the unmitigated flow of cerebrospinal fluid, as first advocated by Hollier et al.[8] and used in our service since 1992.[10] Patients who have a delayed neurologic deficit must be constantly monitored because of possible sudden increases in cerebrospinal fluid pressure. In the future, we will add pressure-sensitive valves to automatically drain cerebral spinal fluid when the pressure reaches 10 mm Hg. Thus far, perioperative cerebrospinal fluid drainage combined with intraoperative distal aortic perfusion has reduced our overall incidence of neurologic complications in patients who underwent repair of type I or type II thoracoabdominal aortic aneurysm. We recommend judicious management of blood pressure by avoidance of destabilizing drugs such as nitroprusside and, given the improved outcome of these eight patients and the simplicity and safety of the technique, we recommend the routine use of cerebrospinal fluid drainage both during surgery and afterward.

Special thanks to our editor, Amy Wirtz Newland.

REFERENCES

1. Crawford ES, Mizrahi EM, Hess KR, Coselli JS, Safi HJ, Patel VM. The impact of distal aortic perfusion and somatosensory evoked potential monitoring on prevention of paraplegia after aortic aneurysm operation. J Thorac Cardiovasc Surg 1988; 95:357-67.
2. Crawford ES, Svensson LG, Hess KR, Shenaq SS, Coselli JS, Safi HJ, et al. A prospective randomized study of cerebrospinal fluid drainage to prevent paraplegia after high-risk surgery on the thoracoabdominal aorta. J Vasc Surg 1991;13:36-46.
3. Safi HJ, Hess KR, Randel M, Iliopoulos DC, Baldwin JC, Mootha RK, et al. Cerebrospinal fluid drainage and distal aortic perfusion: reducing neurologic complications in repair of thoracoabdominal aortic aneurysm types I and II. J Vasc Surg 1996;23:223-9.
4. McCullough JL, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: influence of cerebrospinal fluid drainage—experimental and early clinical results. J Vasc Surg 1988;7:153-60.
5. Miyamoto K, Ueno A, Wada T, Kimoto S. A new and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. J Cardiovasc Surg (Torino) 1988;1:188-97.
6. Grum DF, Svensson LG. Changes in cerebrospinal fluid pressure and spinal cord perfusion pressure prior to cross-clamping of the thoracic aorta in humans. J Cardiothorac Vasc Anesth 1991;5:331-6.
7. Svensson LG, Grum DF, Bednarski M, Cosgrove DM III, Loop FD. Appraisal of cerebrospinal fluid alterations during aortic surgery with intrathecal papaverine administration and cerebrospinal fluid drainage. J Vasc Surg 1990;11:423-9.
8. Hollier LH, Money SR, Naslund TC, Procter DC, Buhrman WC, Marino RJ, et al. Risk of spinal cord dysfunction in patients undergoing thoracoabdominal aortic replacement. Am J Surg 1992;164:210-4.
9. Hill AB, Kalman PG, Johnston KW, Vosu HA. Reversal of delayed-onset paraplegia after thoracic aortic surgery with cerebrospinal fluid drainage. J Vasc Surg 1994;20:315-7.
10. Safi HJ, Bartoli S, Hess KR, Shenaq SS, Viets JR, Butt GR, et al. Neurologic deficit in patients at high risk with thoracoabdominal aortic aneurysms: the role of cerebral spinal fluid drainage and distal aortic perfusion. J Vasc Surg 1994;20: 434-43.
11. Moore MM Jr, Hollier LH, The influence of severity of spinal cord ischemia in the etiology of delayed-onset paraplegia. Ann Surg 1991;213:427-32.
12. Naslund TC, Hollier LH, Money SR, Facundus EC, Skenderis BS II. Protecting the ischemic spinal cord during aortic clamping. Ann Surg 1992;215:409-16.

Submitted Apr. 10, 1997; accepted July 10, 1997.