Measurement of Spinal Cord Ischemia During Operations upon the Thoracic Aorta

Initial Clinical Experience

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Paraplegia has been an unpredictable, devasting complication following operations upon the thoracoabdominal aorta for over 30 years. The frequency ranges from 0.5% with operations for coarctation to as high as 15% following surgery for thoracoabdominal aneurysms. Both uncertainty and controversy exist about the value of different protective methods during aortic crossclamping (AXC): heparinized shunts, partial bypass, and reimplantation of intercostal arteries. This report describes the authors' initial clinical experience with a highly sensitive indicator of spinal cord ischemia, somatosensory evoked potentials (SEP) in an attempt to prevent paraplegia associated with surgical procedures on the thoracoabdominal aorta. Seven consecutive patients (one coarctation, five thoracic aneurysms, one thoracoabdominal aneurysm) underwent continuous operative monitoring of SEP. Cortical response to simultaneous electrical stimulation (20 mAmps, 0.6 mSec., 2.3 cps) of both the right and left posterior tibial nerves was recorded before, during, and after AXC, and following operation. When ischemic changes were detected by SEP, increasing distal circulation by different maneuvers (heparinized shunt, femoral-femoral bypass, reimplantation of intercostal arteries) reversed these changes. In two patients with thoracic aneurysms, ischemic changes appeared within three minutes after AXC and all potentials disappeared in nine minutes. Rapid insertion of a graft (AXC 28 and 37 minutes) resulted in SEP return 40 minutes following restoration of flow. These changes were prevented by a heparinized shunt in two patients, femoral/femoral bypass in one, and T_8–T_9 intercostal reimplantation in one. No SEP changes occurred in the patient with coarctation. No postoperative neurologic complications occurred. Continuous operative monitoring of SEP has exciting possibilities for preventing paraplegia. It is simple, highly sensitive, and seems to provide a precise measurement of adequacy of circulation to the spinal cord.

SINCE THE INCEPTION of surgical therapy for lesions of the descending thoracic and thoracoabdominal aorta in the early 1950s, paraplegia or paraparesis has been reported as a frequent, devastating, and unpredictable complication of otherwise successful surgical procedures.[1,2] As a result, numerous improvements in the preoperative and anesthetic management as well as operative technique have evolved.[3-6] In addition, various approaches to maintaining adequate spinal cord blood flow including the use of temporary shunts[7-14] or partial bypass[15-20] have been employed, all with the intent of preventing postoperative paraplegia. Presently, the incidence of postoperative neurologic injury related to spinal cord ischemia has remained remarkably constant at 1 to 10% in all major series despite implementation of a variety of surgical techniques to protect the spinal cord during aortic crossclamping and graft replacement of the descending thoracic or thoracoabdominal aorta.[3-10,15-21] Current efforts to reduce the incidence of postoperative paraplegia are generally directed at reduction of aortic occlusion time, removal of minimal segments of aorta, and avoidance of hypotension.[3-5] In addition, preservation of critical intercostal vessels as first proposed by Spencer[22] has become an important concept in the reduction of the incidence of postoperative paraplegia.[18,23].

Although it is currently recognized that interruption of critical intercostal vessels, as well as other factors, play an important role in the etiology of ischemic spinal cord injury, no attempt has heretofore been made on the part of thoracic surgeons to determine or measure intraoperatively the adequacy of spinal cord blood flow during crossclamping of the thoracic aorta. This report summarizes the authors' initial clinical experience using somatosensory evoked potentials (SEP) as a simple, noninvasive, and highly sensitive method for the detec-

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TABLE 1. *Summary of Preoperative Patient Data*

Number	Patient	Age	Sex	Etiology	Extent of Lesion
1	S.D.	73	M	Atherosclerotic	Left subclavian origin-T_6
2	M.S.	82	M	Traumatic (old)	Left carotid origin-T_8
3	P.C.	15	M	Congenital-coarctation	Postductal-T_6
4	E.A.	76	M	Atherosclerotic	Left subclavian origin-T_{11}
5	J.D.	78	M	Atherosclerotic-ruptured	Left subclavian origin-T_7
6	A.G.	52	M	Atherosclerotic	Left subclavian origin-T_8
7	E.B.	60	M	Mycotic	T_9-T_{10}

tion and prevention of intraoperative spinal cord ischemia during surgery on the thoracoabdominal aorta.

Materials and Methods

Preoperative Patient Data

Seven consecutive patients undergoing surgical procedures for lesions of the descending thoracic aorta between October and December 1981 form the basis of this report. Detailed preoperative information for all patients is illustrated in Table 1. As is shown, the etiology of thoracic aortic lesions was variable including atherosclerotic, traumatic (old), mycotic, and congenital (postductal coarctation). The origin of the carotid and/or left subclavian arteries was involved in five patients and thoracoabdominal disease encontered in two patients.

Operative Data

Detailed operative data for each patient is shown in Table 2. Aortic crossclamp placement varied accordingly with the extent of disease and crossclamp intervals ranged from 27 to 65 minutes.

Dacron tube grafts were inserted in four patients using the graft inclusion technique of Crawford.[3] Standard end-to-end anastomoses using 3–0 running Prolene were performed proximally and distally. In one patient a Dacron roof-top patch was employed after excision of a sacular post-traumatic aneurysm, and a congenital coarctation in another patient was excised with primary end-to-end anastomosis. In a final patient, a right axillo-femoral bypass was performed first due to the presence of a mycotic thoracoabdominal aneurysm at the level of T-9–T-10. Upon completion of this procedure a left thoracotomy was performed, the infected segment of aorta removed, and three large critical intercostal arteries effectively reimplanted into the aortic stump.

Three patients underwent procedures without use of shunts or bypass. One of these patients with a coarctation exhibited significant evidence of "self-shunting" with mean distal aortic pressure remaining greater than 70 mmHg throughout the crossclamp interval. In the remaining four patients, a temporary shunt or bypass procedure was employed. Adequate function of shunts and/or bypass was determined by placement of femoral and radial arterial pressure lines for documentation of proximal and distal aortic pressures in all patients. In addition, an electromagnetic flow meter coated with TD MAC® (Gould/Statham Corporation, Oxnard, California) was inserted in all shunts for intraoperative assessment of shunt flow.

Evaluation of Neurologic Status and Somatosensory Evoked Potentials

All patients underwent a thorough preoperative and postoperative neurologic examination performed by the same neurologist. In all patients, baseline somatosensory evoked potentials (SEP) were performed 24 hours

TABLE 2. *Summary of Operative Patient Data*

		Aortic Crossclamp Placement					
Number	Patient	Proximal	Distal	Duration (min.)	Procedure	Shunt/Bypass	SEP Response Type
1	S.D.	Proximal to left subclavian	T_8	29	Dacron tube graft	0	I
2	M.S.	Proximal to left carotid	T_9	37	Dacron patch	0	I
3	P.C.	Distal to left subclavian	T_7	27	Primary anastomosis	0	II
4	E.A.	Proximal to left subclavian	T_{12}	65	Dacron tube graft	Gott	II
5	J.D.	Proximal to left subclavian	T_8	31	Dacron tube graft	Femoral vein-femoral artery	II
6	A.G.	Proximal to left subclavian	T_9	35	Dacron tube graft	Gott	II
7	E.B.	T_5	L_1	40	Extra-anatomic bypass, aortic resection, intercostal reimplant	Extra-anatomic graft	III

before surgery. Intraoperatively, SEP were determined serially in all patients prior to aortic crossclamping, throughout the duration of aortic crossclamp interval, and for 2 hours following reperfusion. Postoperative recordings of SEP were also obtained 24 hours following completion of the surgical procedure. Intraoperative and postoperative SEP tracings were compared with preoperative baseline tracings for determination of any significant changes.

A clinical evoked potential system (TN-3000, TRACOR Analytic Inc., Oak Grove Village, Illinois) was used to monitor spinal cord perfusion by generation of SEP. SEP traces were generated by bilateral stimulation of posterior tibial nerves with two bipolar input channels. After conduction of impulses via the dorsal spinal columns, the cortical response to 200 consecutive stimuli was recorded from needle electrodes inserted at the nasion and 55% of the distance from the nasion to the inion in the midline of the scalp. The potentials were amplified 10,000 times and processed with a 10-H LoPass and 250-H HiPass filter. To improve the signal to noise ratio of these small potentials, 200 consecutive responses activated by supramaximal stimuli to the nerves (4 × the motor twitch threshold, 10–20 milliamps, 0.6 mSec. duration pulses, 2.3/sec.) were averaged for each SEP trace (Fig. 1). A separate grounding electrode was placed in the upper thorax of each patient, and in addition, a no stimulus control trace was recorded in each patient for establishment of the background noise level.

A typical SEP trace is shown in Figure 2. Two parameters, latency of onset and amplitude of the generated response, are serially monitored. Ischemia of the spinal cord is indicated by increases in latency and/or decreases in amplitude of the SEP trace. Since a new trace can be generated every one and a half minutes for comparison with baseline, any change in these parameters caused by ischemia are immediately recognized. SEP traces are stored on a floppy disk recorder for subsequent retrieval and comparison with baseline if needed. The picture of the complete monitoring system is shown in Figure 3. As is shown, the system is compact and highly portable, allowing for easy positioning of the apparatus in the operating room at a spot distant from the surgical procedure itself.

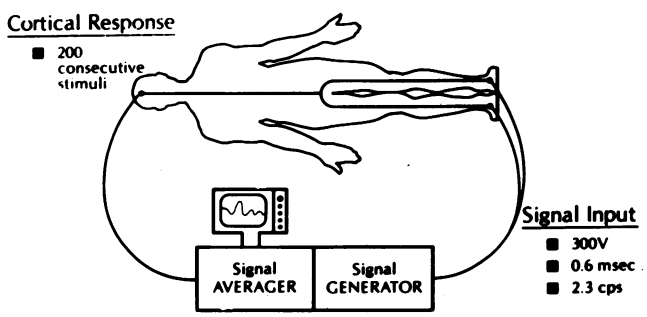

FIG. 1. Technique of intraoperative monitoring of somatosensory evoked potentials. Signal input is via bilateral posterior tibial nerves. The cortical response to 200 consecutive stimuli is averaged for the generation of a single SEP trace.

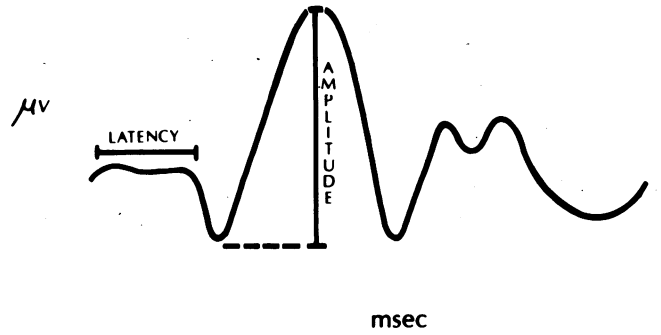

FIG. 2. Typical SEP trace. Latency (in milliseconds) and amplitude (in microvolts) are clearly marked. Serial traces are compared for determination of changes in these parameters.

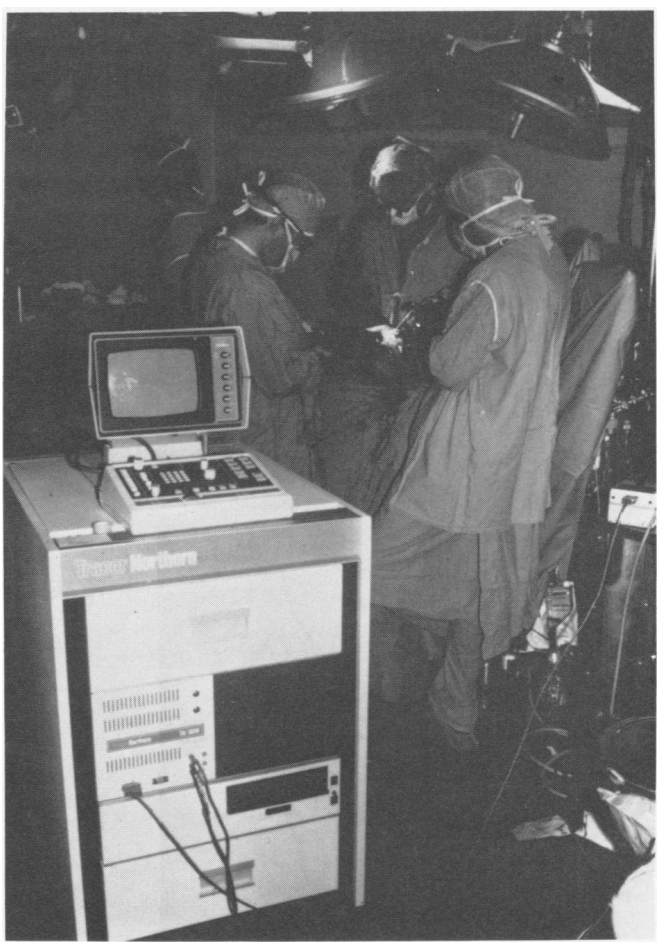

FIG. 3. Photograph of TN-3000 system in clinical use during an operative procedure on the thoracoabdominal aorta.

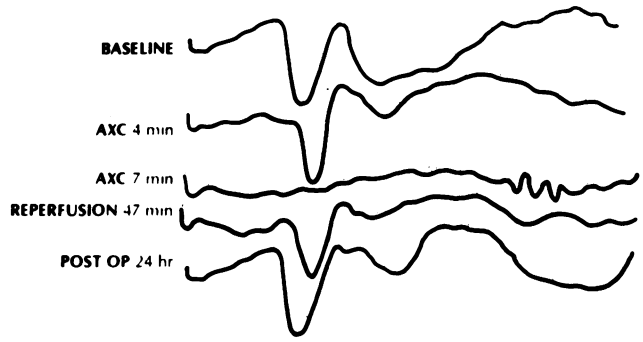

FIG. 4. Type I SEP response. Note early changes in latency as early as four minutes following AXC with progression to cessation of spinal cord conduction within seven minutes of aortic crossclamping. Return of spinal cord conduction occurred 47 minutes following distal aortic reperfusion, with return to normal spinal cord conduction within 24 hours after operation.

Results

One patient died intraoperatively following unsuccessful resuscitation from cardiogenic shock subsequent to a massive myocardial infarction occurring during the procedure. The remaining six survivors exhibited no evidence of postoperative spinal cord dysfunction. Comparison of preoperative, intraoperative, and postoperative SEP tracings allowed identification of three clear-cut patterns of response that followed aortic crossclamping in this group of patients (Table 2).

Type I SEP Response (Rapid Loss After Crossclamping—No Distal Perfusion)

Rapid loss of SEP following aortic crossclamping without distal perfusion by either a shunt or partial bypass was observed in two patients. Neither of these patients had evidence of significant collateralization around the area of crossclamping as evidenced by the maintenance of mean distal aortic pressures of less than 25 mmHg throughout the entire crossclamp interval. Figure 4 depicts the characteristic changes in SEP in these patients after crossclamping. Changes in latency and amplitude uniformly began three to four minutes following proximal clamping and signify changes in physiologic spinal cord function secondary to ischemia. These changes progressed to the extent that SEP traces became completely flat eight to nine minutes following aortic crossclamping, thereby signaling cessation of spinal cord impulse conduction via the dorsal columns secondary to inadequate distal perfusion. Fortunately ischemic intervals in both of these patients were brief (29 and 37 minutes, respectively) and critical intercostal arteries were not ligated because of the location of the aneurysms in the upper thoracic aorta. Evoked potentials remained absent during the entire period of proximal clamping with the first evidence of return of spinal cord conduction occurring at 40 and 47 minutes, respectively, following unclamping and reperfusion of the distal aorta after graft implantation. Posterior column impulse conduction, as determined by SEP, progressively returned toward normal over the ensuing 24-hour period in both patients (Fig. 4).

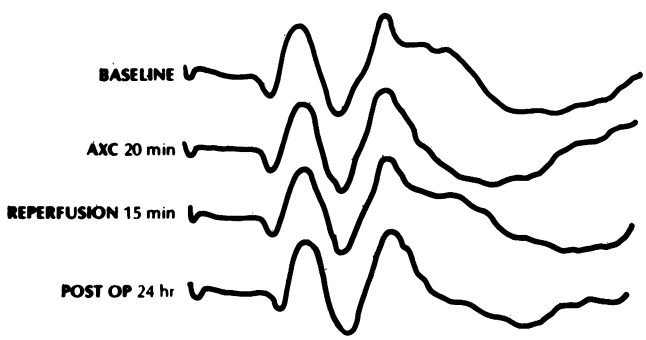

FIG. 5. Type II SEP response. Note maintenance of baseline latency and amplitude throughout the aortic crossclamp interval. Normal distal aortic perfusion pressures following AXC were maintained by either shunt or bypass procedures in these patients.

Type II SEP Response (Maintenance of SEP—Adequate Distal Perfusion)

Four patients exhibited no change in evoked potentials following total aortic occlusion because of maintenance of adequate distal perfusion during the interval of aortic crossclamping (Fig. 5). In this particular group of patients, distal aortic pressures (beyond the lowest crossclamp) were maintained at 60 to 70 mmHg throughout the interval of aortic occlusion. Adequate distal aortic perfusion was achieved by use of a heparinized shunt in two patients, femoral-femoral bypass in one patient, and as a result of "self-shunting" through physiologic collaterals in a single patient with a postductal coarctation. The absence of any significant changes in SEP during the aortic crossclamp interval in this group of patients assured the surgeon intraoperatively that ligation of various intercostal vessels in the excluded aortic segment would not result in permanent ischemic damage to the cord. In particular, in the patient undergoing repair of aortic coarctation, temporary occlusion of several intercostals near the area of constriction was not associated with any change in SEP. This provided the surgeon with vital information that allowed him to interrupt these particular intercostals without fear of producing postoperative paraplegia.

The high degree of sensitivity of SEP monitoring for detecting spinal cord ischemia is illustrated in Figure 6. This series of tracings illustrates the subtle changes

in SEP amplitude following left thoracotomy in the patient undergoing repair of postductal coarctation. As depicted, minimal amplitude changes in the SEP response occurred following ligation of collaterals during thoracotomy, indicating that even subtle changes in distal perfusion following such routine maneuvers are reflected by constant SEP monitoring. These minor amplitude changes disappeared, and SEP returned to normal following reanastomosis of the aorta after resection of the coarctation.

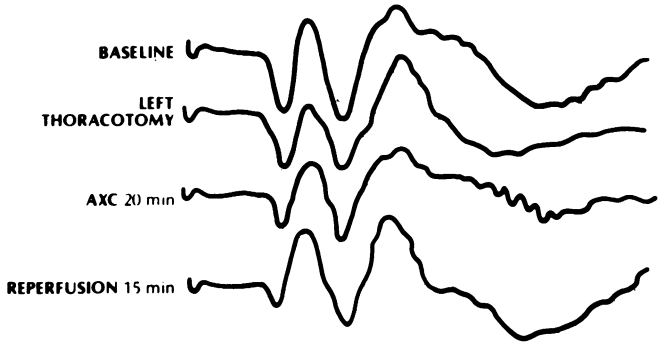

FIG. 6. Type II SEP response. This is the SEP record of a patient undergoing coarctation repair. Minimal changes in amplitude, but not latency of the SEP trace are seen following left thoracotomy. Note the lack of changes in latency and amplitude following aortic crossclamping. This illustrates the sensitivity of this technique in protecting even minimal disruption of blood flow to the distal spinal cord. These changes are reversed following excision of the coarcted segment and reperfusion of the distal aorta.

Type III SEP Response (SEP Loss After Interruption of "Critical Intercostals")

Figure 7 displays the loss and subsequent return of SEP in a patient in whom critical intercostal vessels were initially interrupted and then subsequently reimplanted. In this patient with a mycotic aneurysm involving the thoracoabdominal aorta, maintenance of normal SEP was initially achieved by implantation of an extra anatomic axillo/femoral bypass graft. Following aortic occlusion above and below the mycotic aneurysm, evoked potentials disappeared. The infected aneurysmal sac was inspected, and three large intercostals were noted in the aortic cuff at the T-9 to T-10 level. The proximal and distal aortic stumps were fashioned in such a manner as to effectively "reimplant" these apparently important intercostals. Restoration of flow of these three vessels resulted in almost immediate return of SEP tracings toward normal. Ischemic spinal cord dysfunction (as evidenced by SEP deterioration) in this patient was undoubtedly caused by critical intercostal interruption since distal aortic perfusion was adequate by virtue of the previously implanted extra-anatomic graft and since no other cause of deterioration in impulse conduction could be found.

Discussion

The exact etiology of spinal cord ischemia and paraplegia following surgical procedures on the thoracoabdominal aorta remains controversial. Four major causes have been proposed: (1) increases in cerebrospinal fluid pressures associated with proximal hypertension secondary to aortic crossclamping,[24] (2) duration of aortic crossclamping,[25] (3) intraoperative hypotension,[3,4] and (4) inadvertent permanent interruption of critical intercostal arteries.[3-5,18,22,25] Without question, improvements in anesthetic and pharmacologic management during surgery have decreased the likelihood of spinal cord injury secondary to intraoperative hypertension or hypotension. The use of heparinized shunts or partial bypass techniques to insure adequate perfusion of the distal cord beyond the lowest crossclamp would seem on a physiologic basis to offer a safe approach to aneurysm repair even if prolonged aortic crossclamping were required. Unfortunately, however, despite all of these interventions, the incidence of permanent ischemic spinal cord injury following thoracoabdominal aortic aneurysectomy remains as high as 10%.[3-10,15-21] The occurrence of such injury appears to be almost unpredictable, does not correlate well with the duration of aortic crossclamping, and most strikingly does not correlate with the use of shunt or no-shunt techniques in most series.[3-6,18] It is quite obvious that the etiology of ischemic spinal cord injury during thoracic aortic crossclamping is a multifaceted phenomena. It is more likely related to a combination of factors such as intraoperative hypertension or hypotension, duration of aortic crossclamping, and quite importantly the interruption of critical intercostal arteries. Unfortunately, efforts to

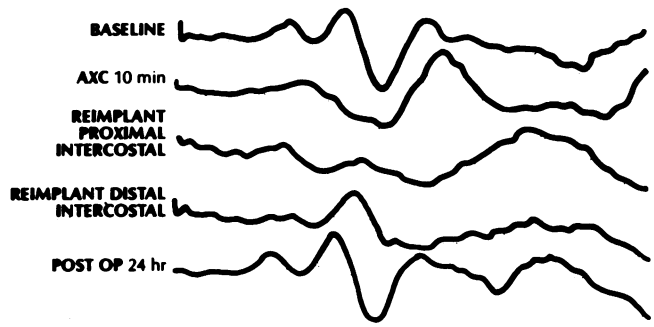

FIG. 7. Type III SEP response. Ischemic spinal cord dysfunction is noted following exclusion of the aneurysmal aortic segment despite adequate distal perfusion pressure provided by an extra-anatomic graft. This response signifies interruption of critical intercostal arteries in the excluded aortic segment and deterioration in the SEP trace is reversed by appropriate intercostal reimplantation. Normalization of the SEP trace 24 hours after operation is evident.

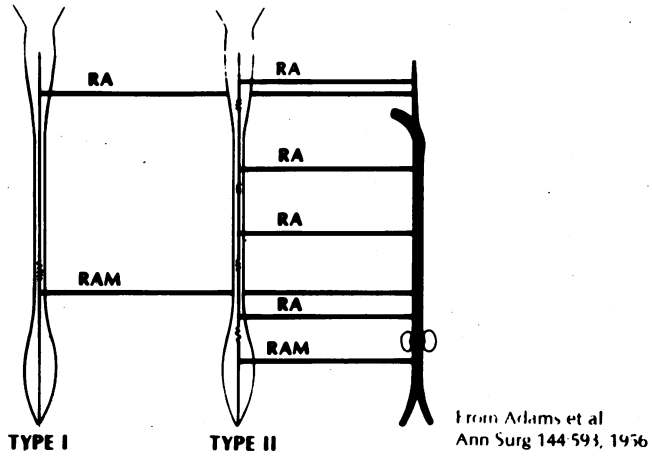

FIG. 8. Illustration of the anatomical variations in the blood supply to the spinal cord.

identify early and to further decrease the incidence of intraoperative spinal cord ischemia have been hampered by the lack of application of any reliable method for monitoring the adequacy of spinal cord perfusion during surgery. In addition, no method of effectively identifying "critical intercostal" vessels has heretofore been reported. The results of the authors' early clinical experience indicate that monitoring of somatosensory evoked potentials during proximal thoracic aortic occlusion provides such a reliable method for monitoring the adequacy of spinal cord perfusion.

Variations in Spinal Cord Blood Supply

Without question, the varying incidence of paraplegia following surgery on the thoracoabdominal aorta is most certainly related to the unpredictable anatomical variations in spinal cord blood supply in different individuals (Fig. 8). The majority of 62 radicular arteries present in the embryo ultimately regress to a point where only a small number of radicular arteries (ranging from 2–5) remain in the adult.[25,26] Seventy-five per cent of spinal cord blood supply is derived from the anterior spinal artery which often becomes extremely narrow in caliber and distribution in the mid and lower portions of the spinal cord. If blood flow through this segment of the anterior spinal artery is insufficient to maintain the integrity of the lower spinal cord, then ischemic injury to the cord and paraplegia are likely to result if the segmental blood supply to the dorsolumbosacral region is permanently interrupted by ligation of critical intercostals.[18,25] The dorsolumbosacral region extends approximately from T-8 to the conus terminalis and generally derives its blood supply from a single large radicular artery arising from a left intercostal vessel in approximately 80% of individuals. Usually this vessel reaches the cord accompanying a nerve root between T-8 and L-4, while in a smaller number of patients (approximately 15%), it arises at a higher level between T-5 and T-8.[25,27] Such a high origin of this "artery of Adamkewicz" is generally associated with supplemental blood supply to the lower cord arising from additional vessels in the lower dorsolumbar region. In such an instance, it is unlikely that division or ligation of the intercostal arteries above T-8 will result in paraplegia, and most assuredly cord injury in this anatomic situation can only result from extremely long proximal aortic crossclamp times or interruption of the critical lower intercostal vessels.[18,25]

The importance of such large, isolated intercostal vessels cannot be under-estimated since the integrity of the lower spinal cord is directly related to their continuity. This has been borne out by both experimental and clinical evidence that interruption of such intercostal arteries results in a high incidence of paraplegia.[22,23,28–37] Spencer and Zimmerman made an early recommendation for reimplantation of intercostal vessels in extensive lesions of the thoracoabdominal aorta based on their observations following experimental ligation of intercostal arteries and production of paraplegia in dogs.[22] Crawford has reported his extensive experience with thoracoabdominal aneurysms and has repeatedly suggested that "blind" reimplantation of distal thoracic and lumbar vessels significantly decreases the incidence of postoperative paraplegia in such lesions.[23]

Clinical and Experimental Use of Somatosensory Evoked Potentials

Dawson was the first to describe changes in electrical potentials (SEP) that were recorded on the scalp after stimulation of peripheral nerves in the extremities. He determined that these changes in evoked potentials were, in fact, cerebral action potentials arising from the central or postcentral cerebral cortex.[38] Clinical use of SEP gained strong impetus with the work of Perot in 1972. His technique and findings served as the basis of much of the clinical application of this method, and it was he who first suggested that SEP might be of significant use as a predictive tool for detection of impending injury during operations on or near the spinal cord.[39] Further work by D'Angelo and associates has shown that conduction of stimuli via the spinal cord might be inhibited not only by mechanical distortion or disruption of spinal cord fibers, but also by ischemia, hypoxia, or neurochemical depressors.[40] Since conduction of stimuli to the cortex has been shown to occur via the dorsal columns, the physiologic integrity of these pathways in the spinal cord can be readily ascertained

using the technique of SEP measurement.[41] Although the anterior spinal artery supplies primarily the anterior half of the cord, interruption of flow in this artery (as would be the case with intraoperative hypotension, poor collateral spinal cord flow during crossclamping, or interruption of critical intercostal blood vessels supplying the cord) results in ischemia and subsequent infarction of almost the entire transverse diameter of the cord including both sensory as well as motor pathways.[25,42]

Growing interest in the development and application of techniques for intraoperative monitoring of spinal cord function through the use of continuous monitoring of somatosensory evoked potentials has recently been popularized by both neurosurgeons and orthopedists.[43-51] Utilization of this methodology for continuously monitoring the integrity of the spinal cord now permits the safe performance of both elective and emergent operations on or near this vital organ, for example, spinal cord tumors, spinal trauma, scoliosis.[43-51]

Recently, characteristic changes in SEP latency and amplitude have been demonstrated in dogs following experimentally induced ischemia of the spinal cord. Such ischemic changes were seen to occur following either episodes of severe hypotension or in association with spinal cord ischemia secondary to crossclamping of the thoracic aorta.[46,52] These experimental findings support well our current clinical data and hypotheses that constant monitoring of SEP during surgery on the thoracoabdominal aorta provides a specific means for early detection of spinal cord ischemia during such procedures.

Variations in SEP Response Following Aortic Crossclamping

To date, three clear-cut patterns of SEP response following aortic crossclamping have been identified by virtue of preliminary clinical observations.

Type I SEP Response—this characteristic and reproducible response was seen clinically in this survey and has been corroborated experimentally by subsequent animal experimentation.[53] Within three minutes following complete aortic crossclamping at the level of the left subclavian artery, changes in SEP latency and amplitude begin to occur. Complete loss of SEP is routinely noted around eight to nine minutes following cross-clamping. These observations seem to strengthen the prediction made by Adams over 20 years ago that the safe limit of complete aortic crossclamping might, in fact, be much less than had previously been expected, perhaps as little as 18 minutes.[25] Such changes with brief crossclamping undoubtedly represent ischemia, not infarction and the safe upper limit of proximal aortic crossclamping as mentioned earlier has yet to be defined. The definition of the safe upper limit of such crossclamping awaits further experimental studies and will obviously vary from patient to patient based on the nature of collateral circulation to the cord. It would appear, however, from the authors' observations as well as numerous clinical reports over the past several years that in the absence of interruption of critical intercostal arteries, the upper thoracic cord (above T-5 to T-8) will tolerate significantly greater lengths of ischemia than that observed with more extensive crossclamping or interruption of critical intercostal vessels. Within the limits of tolerance, the ischemic changes and loss of SEP that follow upper thoracic aortic crossclamping appear to be completely reversible within several hours following unclamping.

The disadvantages of inducing a state of ischemia created by simple aortic crossclamping in the upper thoracic aorta are twofold. First, as mentioned, the duration of spinal cord tolerance to temporary ischemia is unknown and varies considerably between patients. Therefore, the duration of aortic crossclamping does become a critical factor in a certain unpredictable number of cases. Secondly, the state of ischemia induced by such crossclamping precludes further SEP monitoring, therefore preventing intraoperative recognition of critical intercostal interruption should it occur. Herein lies a very strong argument for the use of techniques involving heparinized shunts or partial bypass to ensure adequate perfusion of the distal spinal cord during crossclamping. Such perfusion allows for maintenance of baseline SEP tracings and recognition of the need to implant vital intercostal vessels when needed.

It is of importance to record the fact that aortic crossclamping at any level eventually induces peripheral nerve ischemia which also precludes adequate SEP monitoring. The loss of evoked potentials following peripheral nerve ischemia can be delineated from that following spinal cord ischemia by the temporal relationship of the two. It has been observed that SEP loss following spinal cord ischemia begins to occur as early as three minutes and is complete by nine minutes. On the other hand, SEP loss secondary to peripheral nerve ischemia following aortic crossclamping has generally occurred at approximately 20 minutes following cessation of aortic flow (Fig. 9). Further identification of peripheral nerve ischemia can be obtained by recording the reduced conduction velocity in such ischemic peripheral nerves, a finding not seen in pure spinal cord ischemia.

Type II SEP Response—in general, SEP can be maintained at normal levels following crossclamping providing that adequate distal aortic and spinal cord perfusion is achieved and with the qualification that the

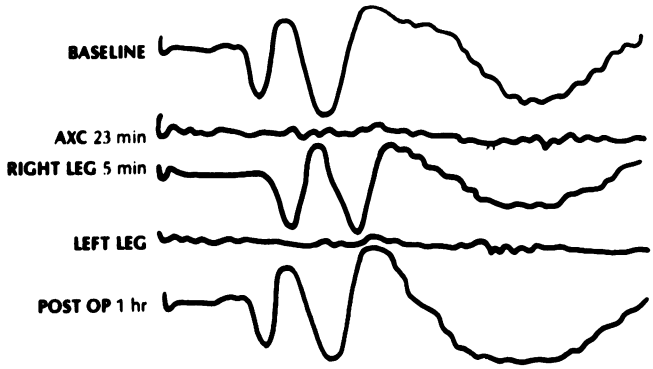

FIG. 9. Effect of peripheral nerve ischemia on the SEP response during aortoiliac graft implantation. Note that ischemic SEP changes due to peripheral nerve ischemia occur much later following crossclamping (23 minutes *vs.* nine minutes) than changes due to spinal cord ischemia. Following right leg reperfusion peripheral nerve conduction is quickly re-established while conduction of the SEP trace from the left leg remains absent. Return of the trace to normal after operation occurs much more quickly than that seen in spinal cord ischemia.

lowest aortic crossclamp does not interfere with critical intercostals supplying the lower cord. By maintaining normal evoked potentials via a shunt or partial bypass after placement of the proximal aortic crossclamp in the upper thorax, the surgeon should be able to accurately ascertain the lower limit of safe aortic crossclamp. As long as SEP are maintained following application of the crossclamp in the lower thoracic or upper abdominal aorta, there appears to be good evidence that interruption of vital intercostal arteries will not be affected by graft interposition.

Type III SEP Response—this particular change in evoked potentials is characterized by loss of SEP following placement of the distal aortic crossclamp. This particular response can be seen only if normal evoked potentials have been maintained by virtue of a shunt or partial bypass technique before the application of the crossclamp. The loss of cortical response to peripheral stimuli in this particular setting signals the interruption of critical intercostal vessels supplying the lower cord and serves as an indicator for immediate and expeditious reimplantation of specific intercostal vessels rather than "blind" reimplantation in an unguided fashion. It is important to note once again that such a response will otherwise go unnoticed unless measures are taken to maintain normal SEP tracings by virtue of perfusion of the distal spinal cord before exclusion of the entire segment of aorta involved. The authors' clinical impression and early experience indicate that distal perfusion pressures of 60 mmHg or greater are necessary in order to insure adequate distal flow following crossclamping. For this reason, radial and femoral artery pressure monitoring is routinely carried out and, as an additional monitoring device, an electromagnetic flow probe is now used routinely to record the adequacy of flow in heparinized shunts.

Conclusions

Utilization of continuous intraoperative monitoring of somatosensory evoked potentials in these preliminary clinical studies has demonstrated exciting possibilities for prevention of paraplegia in the large number of patients undergoing surgery on lesions of the thoracoabdominal aorta.

In order to use appropriately the methodology of monitoring somatosensory evoked potentials to determine spinal cord ischemia, it is necessary to initially insure adequate distal perfusion of the aorta and spinal cord by use of either a heparinized shunt or a partial bypass technique. Adequacy of distal perfusion must be determined after placement of the most proximal aortic crossclamp in the upper thoracic aorta by accurate measurements of distal pressure, shunt or bypass flow, and on-line somatosensory evoked potential determination. After adequacy of distal perfusion is thus determined, total exclusion of the diseased portion of the aorta may be carried out. Maintenance of normal SEP following total aortic exclusion insures that graft implacement without intercostal reimplantation will result in no permanent neurologic sequelae. However, loss of SEP following proximal and distal aortic crossclamping signals the need for subsequent reimplantation of "critical" intercostal vessels in the excluded aortic segment in order to avert paraplegia.

Temporary shunting or partial bypass to insure adequate distal aortic perfusion, although sometimes cumbersome, are necessary adjuncts to obtaining accurate measurements of somatosensory evoked potentials during aortic interruption for surgery on the thoracoabdominal aorta. The use of such measures to insure adequate distal flow in order to allow maintenance of accurate SEP monitoring and identification of critical intercostal vessels should provide the surgeon with adequate information to perform operations intelligently on the thoracic aorta without incidence of postoperative neurologic sequelae related to spinal cord ischemia or infarction.

References

1. Bahnson HT. Definitive treatment of saccular aneurysms of the aorta with excision of the sac and aortic suture. Surg Gynecol Obstet 1953; 96:383.
2. DeBakey MD, Colley DA. Successful resection of aneurysms of the thoracic aorta and replacement by graft. JAMA 1973; 152:673.
3. Crawford ES, Fenstermacher JM, Richardson W, Sandiford F. Reappraisal of adjuncts to avoid ischemia in the treatment of thoracic aneurysms. Surgery 1970; 67:182–96.
4. Crawford ES, Waler HS, Saleh SA, Normann NA. Graft replacement of aneurysm in descending thoracic aorta: results without bypass or shunting. Surgery 1981; 89:73–85.
5. Najafi H, Javid H, Hunter J, et al. Descending aortic aneurysectomy without adjuncts to avoid ischemia. Ann Thor Surg 1980; 30:326–35.

6. DeBakey ME, McCollum CH, Graham JM. Surgical treatment of aneurysms of the descending thoracic aorta. Long-term results in 500 patients. J Cardiovas Surg 1978; 19:571-6.
7. Wolfe WG, Kleimman LH, Weschler AS, Sabiston DC Jr. Heparin coated shunts for lesions of the descending thoracic aorta. Arch Surg 1977; 112:148.
8. Lawrence GH, Hessel EA, Sauvage LR, Krause AH. Results of use of the TDMAC-heparin shunt in surgery of aneurysms of the descending thoracic aorta. J Thorac Cardiovasc Surg 1977; 73:393-8.
9. Donahoo JS, Brawley RK, Gott VL. The heparin-coated vascular shunt for thoracic aortic and great vessel procedures: a ten-year experience. Ann Thor Surg 1977; 23:507-13.
10. Connors JP, Ferguson TB, Roper CL, Weldon CS. The use of the TDMAC-heparin shunt in replacement of the descending thoracic aorta. Ann Surg 1975; 181:735-41.
11. Frantz PT, Murray GF, Shallal JA, Lucas CL. Clinical and experimental evaluation of left ventriculoiliac shunt bypass during repair of lesions of the descending thoracic aorta. Ann Thor Surg 1981; 31:551-7.
12. DeMeester TR, Cameron JL, Gott VL. Repair of a through-and-through gunshot wound of the aortic arch using a heparinized shunt. Ann Thor Surg 1973; 16:193-8.
13. Murray GF, Young WG., Thoracic aneurysectomy utilizing direct left ventriculofemoral shunt (TDMAC-heparin) bypass. Ann Thor Surg 1976; 21:26-9.
14. Cukingnan RA, Carey JS. Repair of lesions of the thoracic aorta with the TDMAC-heparin shunt. J Thorac Cardiovasc Surg 1978; 75:227-31.
15. Mag IA, Ecker RR, Iverson LIG. Heparinless femoral venoarterial bypass without an oxygenator for surgery on the descending thoracic aorta. J Thorac Cardiovasc Surg 1977; 73:387-92.
16. Hilgenberg AD, Rainer WG, Sadler TR. Aneurysm of the descending thoracic aorta. Replacement with the use of shunt or bypass. J Thorac Cardiovasc Surg 1981; 81:818-24.
17. Connolly JE. Wakabayashi A, German JC, et al. Clinical experience with pulsatile left heart bypass without anticoagulation for thoracic aneurysms. J Thorac Cardiovasc Surg 1971; 62:568-76.
18. Wakabayashi A, Connolly JE. Prevention of paraplegia associated with resection of extensive thoracic aneurysms. Arch Surg 1976; 111:1186-9.
19. Wakabayashi A, Connolly JE. Heparinless left heart bypass for resection of thoracic aortic aneurysms. Am J Surg 1975; 130:212.
20. Reul GJ, Cooley DA, Hallman GL, et al. Dissecting aneurysms of the descending aorta. Improved surgical results in 91 patients. Arch Surg 1975; 110:632.
21. Kouchoukos NT, Lell WA, Karp RB, Samuelson PN. Hemodynamic effects of aortic crossclamping and decompression with temporary shunt for resection of the descending thoracic aorta. Surgery 1979; 85:25-30.
22. Spencer FC, Zimmerman JM. The influence of ligation of intercostal arteries on paraplegia in dogs. Surg Forum 1958; 9:340-2.
23. Crawford ES, Snyder DM, Gwen CC, Roethan JOF Jr. Progress in treatment of thoracoabdominal and abdominal aortic aneurysms involving celiac, superior mesenteric and renal arteries. Ann Surg 1978; 188:404-22.
24. Blaisdell FW, Collely DA. The mechanism of paraplegia after temporary thoracic aorta occlusion and its relationship to spinal fluid pressure. Surgery 1962; 51:351-5.
25. Adams HD, Van Geertruyden HH. Neurologic complications of aortic surgery. Ann Surg 1956; 144:574-610.
26. Dijundian R, Hurth M, Houdart M, et al. Arterial supply of the spinal cord. In: *Angiography of the Spinal Cord.* Baltimore: Baltimore University Park Press, 1970; 3-13.
27. Adamkiewicz A. Die Blutfegasse des menschlichen Ruckenmarkesoer flache sitz Acad. Urss. Wien. Math Natur Klass 1882; 85:101.
28. Fried LD, DiChiro G, Doppman JL. Ligation of major thoracolumbar spinal cord arteries in monkeys. J Neurosurg 1969; 13:608-14.
29. Hill CS, Vasquez JM. Massive infarction of the spinal cord and vertebral bodies as a complication of dissecting aneurysm of the aorta. Circulation 1962; 25:997-1000.
30. Weisman AD, Adams RD. The neurological complications of dissecting aortic aneurysm. Brain 1944; 67:69.
31. Scott RW, Sancetta SM. Dissecting aneurysm of aorta with hemorrhagic infarction of the spinal cord and complete paraplegia. Am Heart J 1949; 38:747.
32. Moerscu FP, Sayre GP. Neurological manifestations associated with dissecting aneurysm of the aorta. JAMA 1950; 144:1141.
33. Schwarz GA, Shorey WK, Anderson NS. Myelomalacia secondary to dissecting aneurysm of the aorta. Arch Neurol Psychiat 1950; 64:401.
34. Thompson GD. Dissecting aortic aneurysm with infarction of the spinal cord. Brain 1956; 79:111.
35. Kalischer O. Aneurysma Dissceans der aorta mit paraplegic (Demonstration eines Praparates). Berl klin Wehnschr 1914; 51:2.
36. Reitter K. Aneurysma Disseans und Paraplegic. Zugleich ein Beitrag zur Pathologie der Blutzirkulation in Kuckenmark. Deutsches Arch. klin. Med. 1916; 119:561.
37. Case Records of the Massachusetts General Hospital (Case 38351). N Engl J Med 1952; 247:326.
38. Dawson GD. Cerebral responses to electrical stimulation in man. J Neurol Neurosurg Psychiat 1947; 10:137-40.
39. Perot PL. The clinical use of somatosensory evoked potentials in spinal cord injury. Clin Neurosurg 1972; 20:367-81.
40. D'Angelo CM, VanGilder JC, Taub A. Evoked cortical potentials in experimental spinal cord trauma. J Neurosurg 1973; 38:332-6.
41. Cohen AR, Young W, Ransohoff J. Intraspinal localization of SEP. Neurosurgery 1981; 9:157-62.
42. Fried LC, Aparicio O. Experimental ischemia of the spinal cord. Histologic studies after anterior spinal artery occlusion. Neurology 1973; 23:289-93.
43. Spielholz NI, Benjamin MV, Engler G, Ransohoff J. Somatosensory evoked potentials and clinical outcome in spinal cord injury. In: Popp AJ, ed. Neural Trauma. New York: Raven Press, 1979.
44. Engler GL, Spielholz NI, Bernhard WN, et al. Somatosensory evoked potentials during harrington instrumentation for scoliosis. J Bone Joint Surg 1978; 60A:528-32.
45. Spielholz NI, Benjamin MV, Engler GL, Ransohoff J. Somatosensory evoked potentials during decompression and stabilization of the spine. Spine 1979; 4:500-5.
46. Nash CL, Lorig RA, Schatzinger LA, Brown RH. Spinal cord monitoring during operative treatment of the spine. Clin Orthop 1977; 126:100-105.
47. Croft TJ, Brodkey JS, Nulsen FE. Reversible spinal cord trauma: a model for electrical monitoring of spinal cord function. J Neurosurg 1972; 36:402-6.
48. Nash CL, Schatzinger L, Lorig RA. Intraoperative monitoring of spinal cord function during scoliosis spine surgery. J Bone Joint Surg 1974; 56A:1765.
49. Nash CL, Schatzinger L, Brown RH, Brodkey J. The unstable thoracic compression fracture. Its problems and the use of spinal cord monitoring in the evaluation of treatment. Spine 1977; 2:261-5.
50. Vauzelli C, Stagnara P, Jouvinrous P. Functional monitoring of spinal cord activity during spinal cord surgery. Clin Orthoped 1973; 93:173-8.
51. Donaghy RM, Numoto M. Prognostic significance of somatosensory evoked potentials in spinal cord injury. Presented at the 17th spinal cord injury conference. VA Hospital, Bronx, New York, 1969.
52. Coles, JG, Wilson GJ, Sima AF, et al. Intraoperative detection of spinal cord ischemia using somatosensory cortical evoked potentials. Ann Thor Surg (In press).
53. Laschinger JC, Cunningham JN Jr, Spencer FC. Unpublished data.