REVIEW ARTICLE

Spinal Cord Infarction in Disease and Surgery of the Aorta

R.T. Ross

ABSTRACT: Diseases of the aorta and surgery of the aorta can produce spinal cord damage. There are major variations in blood supply to the spinal cord between individuals. The spinal cord may be tamponaded by increased spinal fluid pressure subsequent to clamping the aorta. Both of these factors may contribute to spinal cord infarction. The available methods and procedures to protect the spinal cord during surgery are discussed.

RÉSUMÉ: Infarctus de la moelle épinière lors des maladies et de la chirurgie de l'aorte Les maladies de l'aorte ainsi que la chirurgie de ce vaisseau peuvent causer des lésions de la moelle épinière. Il existe de fortes variations dans l'irrigation sanguine de la moelle entre les divers individus. Après clampage de l'aorte, il est possible d'observer une tamponnade de la moelle consécutive à l'augmentation de la pression du liquide céphalorachidien. Ces deux facteurs peuvent donc contribuer à l'infarctus de la moelle. Nous décrivons les méthodes et procédures de prévention de ces complications pendant la chirurgie.

Can. J. Neurol. Sci. 1985; 12:289-295

The incidence of spinal cord infarction accompanying disease or trauma of the aorta, or following surgery of the aorta is unpredictable and may be at an irreducible minimum. Permanent paraplegia can occur, is a devastating complication, and if it follows surgery the medical-legal implications are obvious.

This is a brief review of the blood supply of the spinal cord and its more common variations. Diseases, trauma, and surgery of the aorta are discussed in relationship to transient or permanent spinal cord damage. The possible mechanisms of cord infarction subsequent to clamping of the aorta and various protective methods are reviewed. Mention is also made of neural axis compliance to increased volume and pressure. This may significantly contribute to the integrity of the spinal cord when the aorta is clamped.

Spinal Cord Blood Supply

A spinal cord infarct from a diseased aorta or during surgery of the aorta may depend more on the individual variation in spinal cord blood supply than any other single factor. The blood supply of the cord can be considered in three divisions: proximal, radicular, and terminal.[1,2,3] The proximal division consists of the spinal branches of the thyrocervical trunk, vertebral, costocervical, intercostal, and lumbar arteries. The anterior and posterior radicular arteries arise from the spinal branches and enter the spinal canal along the ventral and dorsal nerve roots. These vessels contribute to the formation of long anastamotic channels of the cord. There are significant differences between the anterior and posterior cord circulations.

The anterior radicular arteries are usually small and most of them end in the ventral nerve roots or in the pial circumferential plexus of the cord. A small variable number (4 to 9) lower cervical, lower thoracic and upper lumbar are larger and reach the anterior median sulcus of the cord. They then divide into ascending and descending branches to form a single or paired vessel of uneven calibre along the anterior median sulcus.[4] The upper end of the anterior spinal artery is a direct branch from the two (or sometimes one) vertebral arteries. It is not a functionally continuous, uninterrupted vessel from the cervical region to the lumbar area.[2] It either terminates or becomes substantially reduced in size between C3 and C4.

The posterior spinal arteries arise from the vertebral or posterior inferior cerebellar artery and descend as two branches, one anterior, the other posterior to the dorsal nerve root. Effectively, there are four posterior spinal arteries. They are segmentally reinforced by posterior radicular collaterals from the vertebral, cervical, and posterior intercostal arteries. Although variable in size and number, these are more numerous than the anterior radicular arteries. The vertical continuity of the posterior spinal arteries is better than the anterior spinal artery and they are

From the Section of Neurology, Department of Medicine, University of Manitoba, and the Health Sciences Centre, Winnipeg, Canada
Received April 30, 1985. Accepted in revised form August 8, 1985.
Reprint requests to: R.T. Ross MD, GF543-700 William Avenue, Winnipeg, Manitoba, Canada R3E 0Z3

less vulnerable to interruption of a radicular branch. Finally, the posterior spinal arteries receive an important contributing loop from the anterior spinal artery at the conus medullaris. The radicular artery of Adamkiewicz contributes a branch to both the anterior spinal artery and to the division of the posterior spinal artery lying anterior to the posterior nerve root.

About two-thirds of the cross sectional area of the cord is supplied by the anterior spinal artery. The grey and white posterior columns and the periphery of the anterior and lateral white columns receive multiple small radial vessels from the posterior spinal arteries and from the circumferential pial plexus.

The variations in this anatomy are considerable. Embryonically, every segment of the spinal cord receives paired radicular arteries, but many of these segmental branches atrophy or disappear.[3] The ultimate arrangement, which is important in determining cord infarcts, consists of only one or two cervical, two or three thoracic, and one or two lumbar radicular arteries persisting into adult life. There are less than five radicular segmental arteries in 45% of the population.[5] The variation in number and origin of radicular arteries may prevent the anterior spinal artery from functioning as a collateral. Occlusion of an intercostal vessel may be harmless to one patient and a disaster to another.[2]

The middle and lower thoracic regions of the spinal cord usually have the poorest segmental vessels. The wide spacing of the radicular arteries leaves large segments of the spinal cord with a precarious inflow. Interruption of any important radicular branches could have serious ischemic consequences.

The most important radicular artery is the great radicular artery of Adamkiewicz (GRA). When it arises at its common level of origin, T11 or T12 on the left, there is usually another important radicular artery of lesser size at the L1 or L2 level. Either of these two major radicular arteries in the thoracolumbar region may become dominant.[1]

In man, the GRA enters the spinal cord at the level of T5 to T8 in 12-15% of cases, T9 to T12 in 60%, L1 in 14%, L2 in 10%, L3 in 1.4%, and L4-5 in 0.2%.[5] The location of the GRA may be the decisive factor in spinal cord damage during aortic clamping. If it arises within the area of aortic clamping, spinal cord damage will be due to inadequate collateral circulation irrespective of shunts and distal perfusion pressure. The cord may survive this situation if there is an infrarenal lumbar radicular artery of substantial size. If the GRA rises inferior to the clamped area and the collateral circulation is adequate, a protective procedure is not necessary nor is it necessary if the GRA arises above the clamp.

The effectiveness of the collateral and anastamotic circulation will be reduced by hypotension, blood loss, narrowing and occlusive disease of anastamotic vessels, and possibly intraspinal pressure (see below).

Another reason (see below) why a shunt may not prevent infarction is because its proximal attachment may disrupt a major normal source of blood to the spinal cord, i.e. the thyrocervical trunk or the internal thoracic artery. Thus, there is a net reduction in peripheral blood flow to the cord in spite of the shunt.

In spite of the importance of the arrangement of vessels to the spinal cord, pre-operative aortography to demonstrate them is not a practical consideration. The information obtained does not justify the added delay and risks and cannot negate the indications for surgery in what is often a life-threatening situation.

Disease, Trauma, and Surgery of the Aorta

Occlusion

This is the least common disease of the aorta. In 3000 patients with peripheral vascular disease, DeMedeiros[6] found 36 occlusions of the aorta and in 1047 consecutive autopsies on patients with no neurological symptoms Lueth found one occlusion.[7] It may present with intermittent, effort-related neurological signs and symptoms in the legs. Dejerine[8] described this intermittent cord ischemia and differentiated it from the claudication described by Charcot. Paraplegia lasting 12 hours,[9] or several months,[10] or permanent[11,12] may occur with occlusion of the aorta.

Aortic Surgery

The incidence of spinal cord damage depends on the nature and location of the aortic lesion, the urgency of the surgery, and a host of other factors.

It is important to put the risks of intra-operative and post-operative cord infarction into perspective and to illustrate the inevitability of some morbidity and mortality with diseases of the aorta and their treatment.

Of 655 non-traumatic paraplegics admitted to a spinal injury unit over 30 years, only five had become paraplegic following surgery for coarctation, aneurysm, or traumatic rupture of the aorta.[13] This does not represent the true incidence of cord damage, as from 38 to 70% of post-operative paraplegics die in the immediate or early post-operative period and never reach a spinal injury unit.[1]

Coarctation of the Aorta

Spinal cord infarction occurs with coarctation unrelated to surgery, although rarely.[2]

In the 12,532 cases of surgical repair of coarctation collected by Brewer et al,[2] the incidence of post-operative spinal cord damage was 0.41%. The infarct was not always related to the sacrifice of intercostal arteries or the duration of the aortic clamp time. The critical factor appeared to be the anomalous and variable system of radicular arteries contributing to the anterior spinal artery. In some patients, the system was so tenuous that cross-clamping was not tolerated for even brief periods in spite of the use of protective measures, i.e. aortic pressure measurements above and below the coarctation before and after clamping, and where indicated, the use of hypothermia, left heart bypass, or jump grafts. This suggests the cord infarction incidence of 0.41% in coarctation may be irreducible and not predictable.

Aneurysm

Paraplegia as a presenting complaint in aortic aneurysm is uncommon.[14,15,16] It has followed the destruction of the lumbosacral spine by the aneurysm.[17] Seventy percent of abdominal aneurysms are small and symptom-free, and have an operative mortality of less than one percent.[18,19,20]

There are greater hazards with a ruptured aneurysm. The mortality before surgery in all ruptured abdominal aneurysms is 35%, during or after emergency surgery is 55% (and 10% worse in the presence of shock), while during or after elective surgery it is 11%.[21] When the operation is an emergency but rupture is not found, the mortality is 20-25%.[20] The prognosis is worse in the presence of pre-operative or intra-operative cardiac arrhythmia, anuria, intraperitoneal rupture,[22] and delay in diagnosis.[23]

Paraplegia complicating resection of abdominal aortic aneurysms is uncommon.[20,24,25,26] Keye et al reported a single case and found 36 similar patients in the literature up to April 1980.[27] Szilagyi et al[1] found spinal cord damage in 0.25% of 3164 patients following abdominal aortic surgery for a variety of lesions. All the spinal cord complications were in patients with aneurysm and were 10 times more common in ruptured aneurysm. The commonest neurological lesion was a flaccid paraplegia (5 of 8), of whom three died. Partial recovery occurred in two of eight patients, and complete recovery in one. In spite of the hazards associated with the treatment of a ruptured or impending ruptured aneurysm, surgery is the only treatment. The risk of paraplegia is small compared to the usual outcome of an untreated rupture.

Repair of thoracic aneurysms carries a greater threat to the cord. The incidence of paraplegia has been reported as high as 6%.[28,29] Repair of thoracoabdominal aneurysm is even more hazardous, the incidence of cord damage being 10% or higher.[21,30] This is due to the disruption of important radicular arteries including the major radicular artery (Adamkiewicz), which arises from the aorta between T1 and L1.

Dissection of the Aorta

Neurological signs are common at the onset of this disorder. Twenty percent of patients have a reduced level of consciousness, probably due to cerebral hypoperfusion. Three to six percent present with paraplegia or monoplegia and half this number with hemiplegia.[31,32] Both proximal and distal dissections may cause spinal cord ischemia.[32,33,34] Dissection is nearly always fatal if not treated surgically[35] as only 10% of patients treated by medical means were alive three months after onset.[36] The patency of the carotid, subclavian, and coronary arteries is compromised in 50% of ascending aorta dissections.[35] The disease presents with a combination of chest pain, heart failure, acute abdomen, transient or permanent limb ischemia, and neurological signs. The mortality in the first 24 hours in untreated ascending dissections is 20-40%.[31,36]

The aim of medical treatment is reduction of blood pressure,[37] which may allow an acute situation to become chronic with the expectation of better surgical mortality. Medical treatment alone resulted in 22% mortality of 90 patients with descending lesions and 83% mortality of 76 patients with ascending lesions.[38] The overall surgical mortality is between 15% and 30%[32,35,39] and ranges from 38% of 170 patients with acute lesions to 18% of 74 patients with chronic lesions.[38] In dissections of the descending aorta, the surgical mortality ranges from 5% to 25% in uncomplicated chronic cases[38,39] to as high as 36% of 121 patients with acute lesions.

In 527 patients with dissections of all types, seen over a 20 year period, 21 presented with strokes and 10 with paraplegia. Post-operatively there were 19 paraplegic, 18 paraparetic, and four patients with neurogenic bladder only. Thus, 41 patients or 7.7% had post-operative ischemic spinal cord damage.[32]

Traumatic Rupture of the Aorta

Most traumatic ruptures of the aorta follow deceleration injuries. The injury can also contuse or lacerate the cord and this may account for the paraplegia even though the aorta is clearly ruptured. Patients may present with anuria and paraplegia[40] either permanent or transient.[41] Kirsh et al had one post-operative paraplegia and reported overall survival of 70% of 43 patients treated over 10 years.[42] They used a bypass without the aid of extracorporeal circulation, plus antihypertensive therapy.

In the treatment of traumatic rupture of the aorta there is a relationship between spinal cord damage and the duration of the clamp time. The damage is compounded by the absence of a shunt or bypass. Although a shunt does not provide complete protection, the probability of cord damage is less in a group of shunted patients at all times after about 30-35 minutes of clamping. The risk factors are less obvious when the clamp time is less than 30 minutes.[43] This suggests that most spinal cords can survive almost total ischemia for some minutes (?10-15); but after 30 minutes, shunting plus effective collateral circulation are needed.

Aortography

Spinal cord damage is fortunately rare after this examination. Szilagyi et al reported an incidence of 0.01% (2 of 17,494).[1] After selective angiography to demonstrate spinal cord arteriovenous malformations, Riché et al reported no complications in 38 patients.[44]

Protective Measures

Clamping the aorta below the subclavian artery produces profound physiological changes.[45,46,47,48,49] There is an abrupt and severe rise in blood pressure proximal to the clamp and an abrupt drop and hypoperfusion distal to the clamp. Reduced systemic perfusion pressure even without cord vascular disease or clamping the aorta can infarct the spinal cord. Silver and Buxton described 11 patients with spinal stroke in the anterior spinal artery territory at the lower dorsal and upper lumbar segments. All were secondary to a drop in systemic arterial pressure and reduced cord perfusion.[50]

Estimating the length of time the cord may tolerate interruption of blood flow from a clamp placed above the renal arteries and below the diaphragm is speculative and may be pointless. Because of the variation in the blood supply of the lumbar spinal cord, the length of time of aortic occlusion and the subsequent development of neurological signs do not correlate well, particularly if the occlusion is greater than 30 minutes. Complete freedom from neurological damage is the invariable rule when the interruption is less than 10 to 15 minutes.[1] Interruption of the aorta below the diaphragm and above the renal vessels for 20 to 25 minutes with no damage has been observed in a large number of cases.

Adams and VanGeertruyden[3] and Cunningham et al[51] suggest that the safe time limit for complete cross-clamping is perhaps 18 minutes or less. If the positions of the clamps compromise major radicular vessels the safe time is probably 12 to 18 minutes.[2,3,51] Reversible ischemia becomes irreversible infarction after this. Under these anatomical and clamp conditions it is unlikely that shunts, bypass, hypothermia, or other supportive measures can extend this time.

There are mixed opinions on most of the ancillary devices designed to protect the spinal cord during clamping of the aorta.

Left Heart Bypass and Shunts

Brewer et al[2] found that neither the sacrifice of intercostal arteries or the duration of aorta occlusion was related to cord damage in all cases of coarctation repair. They believed hypothermia, left heart bypass, or jump grafts should be pro-

vided when the pressure of the aorta distal to the clamp became significantly different from the proximal, preclamping aorta pressure. Cunningham et al state that shunting or partial bypass are necessary if sensory evoked potentials (SEP) are used as a monitor of spinal cord integrity. Without the simultaneous use of bypass, the SEP changes or attenuates for insignificant reasons (i.e. opening the chest) and the signal is then not visible when a real threat does occur.[51,52] Connolly considers the heparinless bypass and preservation of T9-T12 intercostals essential in all cases.[53]

Katz found a shunt was not totally protective in the treatment of traumatic rupture, but the probability of cord damage increased in the unshunted after 30 minutes of clamp time.[43] Kirsch et al also believe that repair of traumatic rupture of the aorta can safely be accomplished by using a bypass shunt without extracorporeal circulation.[42] However, even a blood pressure of 60 mm of Hg distal to the lower clamp did not prevent spinal cord damage of from 3.6%[54] to 17.7%.[55] Crawford and Rubio[56] reported an incidence of paraplegia of 7.8% when using a shunt and 2.2% without a shunt.

DeBakey et al reported in 1982 that since 1970 they had abandoned hypothermia, shunts, left atrial femoral bypass and femorofemoral bypass with no change in the incidence of spinal cord ischemia in the 12 year period.[32]

Crawford believes that "recommending routine distal perfusion or bypass . . . seems to be a step backward since recent experience indicates that they are not only unprotective but hazardous and associated with a mortality rate comparable to the incidence of complications that they would be employed to prevent".[57]

However, shunts and bypasses have additional benefits in ameliorating the aorta cross-clamp and declamp syndromes (see below).

In contrast to the variations of opinion on the usefulness of shunts and bypasses, there seems to be unanimity of opinion of the importance of intercostal arteries and their reimplantation.[5,30,51,58,59,60,61] However, even reimplantation will not save the cord if the perfusion pressure in the segment of aorta from which they arise is inadequate (i.e. less than 40-60 mm of Hg).

The Aorta Cross-Clamping Syndrome

There is an additional source of damage from cross-clamping the aorta and another from declamping.

Clamping the thoracic aorta in healthy dogs for 20 minutes produced a 65% increase in coronary blood flow, an 80% increase in cardiac work and, important to the survival of the spinal cord, a 68% increase in peripheral resistance relative to baseline values.[45] There is an abrupt and marked rise in blood pressure proximal to the clamp in man and animals.[45,46,47,48]

In a group of patients with clamped aorta and no bypass, marked muscle acidosis and a decrease in adenylate energy charge was found. On declamping, these metabolic changes regained their pre-clamp values although energy phosphate compound remained reduced. The same hemodynamic changes occurred as in the canine study above. There was a marked fall in mean arterial blood pressure on declamping.

In a similar group of patients with clamped aorta and a brachial-femoro-femoral bypass, there was no increase in systemic vascular resistance, mean arterial blood pressure, or left ventricular work during clamping.

On declamping, there was only a minor drop in mean arterial blood pressure. In both groups there was a transient rise in pulmonary vascular resistance suggesting pulmonary microemboli. These authors think an extracorporeal bypass is a worthwhile adjunct in corrective surgery of the aorta. It offers protection to cardiac function, prevents muscle metabolic derangement, prevents the marked rise in peripheral vascular resistance and proximal hypertension. On declamping, it obviates the serious hypotension.[48] Even if a bypass does not always prevent ischemic cord damage, these reasons justify its use.

The Declamping Syndrome

After declamping of the infrarenal aorta or common iliac arteries marked hypotension occurs.[62] This is probably due to pooling of blood in the reperfused lower limbs and represents another insult to the ischemic spinal cord at a time when it may be most vulnerable. Left ventricular systolic pressure, total peripheral resistance, and cardiac work decrease 55%, 85%, and 80% of baseline levels respectively after declamping.[45] The distribution of cardiac output to the lower extremities rises from 2.5% to 20% at the expense of renal, hepatic, mesenteric, and possibly spinal cord perfusion.[63]

Central hypovolemia with internal steal is the likely pathogenesis of declamping shock. This may be circumvented by employing moderate volume loading prior to declamping while closely monitoring mean pulmonary artery occlusion pressure.[62]

Intracranial Pressure and Spinal Cord Tamponade

Clamping the aorta is accompanied by increased intracranial pressure (ICP).[45,46,64] The mechanisms include a rise in central venous pressure,[5] a rise in arterial blood pressure,[45] and drugs used to treat the hypertension particularly sodium nitroprusside.[65,66] Sodium nitroprusside is thought to directly dilate intracranial vessels with increased blood volume and cerebral blood flow.[65,67] Cerebral autoregulation can be overcome by the hypertension and the drugs. The rise in intracranial blood volume leads to an increase in intracranial pressure.[68,69,70]

The volumes of blood and cerebrospinal fluid (CSF) in the cranium are reciprocal. As the former rises the latter decreases and more CSF is forced into the spinal theca. When the compliance of this space is exhausted, minute increases in volume have large effects on pressure.[71]

Miyamoto et al[49] believe the post-operative paraplegia was due to increased intracranial pressure during aortic cross-clamping. They thought the raised CSF pressure could tamponade the cord at a time when its arterial perfusion was low.

Blaisdell and Cooley[46] found 100% incidence of paraplegia in dogs when the CSF pressure equalled or exceeded the pressure in the aorta distal to the clamp. In other animals when the CSF pressure was lowered by removal of CSF or urea dehydration, the rate of paraplegia was 8% after clamping of the aorta and 50% in the control animals.

Berendes et al[64] measured CSF and femoral artery pressure in eight patients before, during, and after clamping in the course of surgical correction of aneurysm or coarctation. The only patient with CSF pressure exceeding the femoral artery pressure during clamping was paraplegic post-operatively. Intracranial pressure has also been measured before and after clamping of the aorta in a patient with pre-existing raised intracranial pressure. The CSF pressure rose more than 70% which indicates the end of the intrathecal and intracranial compliance.[47] In spite of the

https://doi.org/10.1017/S0317167100035368 Published online by Cambridge University Press

clinical evidence of raised intracranial pressure in some patients following aortic clamping,[47,64] CSF pressure measurements or drainage is not a routine measure before or during surgery.

The capacity of intracranial and intravertebral space varies from person to person as studies on spinal stenosis and cervical spondylosis have shown.[72] Nordqvist found a large range of sagittal diameters of the spinal subarachnoid space. At the level of the seventh thoracic vertebra, the space has an average sagittal diameter of about 13 mm and a range from 8 to 17 mm.[73]

A proportional increase in volume will raise the pressure less in a large distensible container than in a small one. Thus, the individual response to increased intracranial contents will be variable and unpredictable. Only one of the eight patients of Berendes et al had significantly raised ICP after clamping of the aorta.[64] If there is to be an improvement in the small and apparently irreducible incidence of spinal cord infarcts associated with aortic surgery, the assessment of neural axis compliance will have to become routine. When the surgery is elective a pressure volume index measurement[74] will demonstrate the patient who cannot tolerate increased volume. This patient must have spinal fluid drainage or dehydration therapy. When the urgency of the surgery will not permit the measurement, the treatment should be used anyway.

Sensory Evoked Potentials (SEP)

SEP provides a method of assessing spinal cord ischemia during operation. The stimulus is applied to both posterior tibial nerves and the latency and amplitude of the response is recorded from the scalp. The details of the technique are well established.[75] The afferent signals which generate the SEP are conducted in the posterior columns,[76] and normal recordings have been obtained from patients with infarcts in the anterior spinal artery territory.[77] Spinal cord ischemia related to disease or surgery of the aorta is generally in the territory of the anterior spinal artery and therefore the test may be anatomically inappropriate. A further weakness is that a clinically complete spinal cord lesion may not be neurophysiologically complete.[78]

In animal studies, after 12.4 minutes of aortic occlusion, the SEP signal vanished. Where perfusion was immediately re-established there was complete recovery with no clinical or histological sequelae. In another group in which the occlusion was extended for 15 minutes after the loss of the SEP, two-thirds sustained spinal cord infarction.[79]

Another animal study monitored SEP and measured spinal cord blood flow to determine the safe level of distal aorta occlusion and critical vessel exclusion. The method appeared to be informative and sensitive.[60,61]

SEP have been used as a continuous monitor on patients undergoing thoracoabdominal aortic surgery. When ischemic changes were detected by SEP, distal circulation was increased by a heparinized shunt, femoro-femoral bypass, or reimplantation of intercostal arteries.[51] One of the major criticisms of SEP monitoring[57] is the necessity of using a shunt or bypass in conjunction. It is necessary because from the thoracotomy alone or after cross-clamping the aorta for eight to nine minutes the SEP response will vanish. This is due to ischemia but does not indicate infarction; however it prevents further monitoring. The object is to maintain sufficient distal perfusion to allow persistent baseline SEP recordings. Removal of important intercostal arteries can then be quickly recognized.

In addition to the criticisms that SEP are too sensitive or not sensitive enough, or monitor the function of the posterior cord when the anterior aspect is at risk, the potentials can be attenuated by halothane, hypothermia, and hypotension. Furthermore, technical problems and operating room noise may invalidate the recordings in 20% of patients. There may be inordinate requests to halt the surgical procedure to obtain clean recordings and the time lapse needed for this is unacceptable.[53]

Although there are arguments for and against the use of SEP, the balance of evidence suggests they are helpful and likely to become more so as techniques and equipment improve. Anything that can herald the onset of a cord infarct at a time when the direction or form of the surgery can be modified and the infarct prevented cannot be abandoned. The obligatory shunt needed with the SEP has ample justification on its own merits as described above.

Conclusions

The number of spinal cord infarcts associated with disease or surgery of the aorta may be at an irreducible minimum. However, the tragedy of paraplegia is so great that the subject requires continuing scrutiny.

There are two unassessable factors partially determining the prognosis. They are both individualistic and unique. The first is the number and location as well as the effectiveness of the radicular arteries contributing to the anterior and posterior spinal arteries. The second is the size of the patient's subarachnoid space and the intraspinal volume/pressure relationships in response to brain swelling.

The efficacy of protective intra-operative devices is debatable and there is no consensus on sensory evoked potentials. The latter have the attraction of being a physiological measurement, are applicable in an almost continuous way and may anticipate spinal cord infarction. Their intra-operative time consumption, hypersensitivity, and the necessity of using a shunt at the same time has produced some strong negative opinion from some very experienced cardiovascular surgeons.[57]

References

1. Szilagyi DE, Hageman JH, Smith RF et al. Spinal cord damage in surgery of the abdominal aorta. Surgery 1978; 83(1): 38-56.
2. Brewer LA, Fosburg RG, Mulder GA, Verska JJ. Spinal cord complications following surgery for coarctation of the aorta. J Thorac Cardiosvas Surg 1972; 64(3): 368-381.
3. Adams HD, VanGeertruyden HH. Neurological complications of aortic surgery. Ann Surg 1956; 144: 574-610.
4. Gray's Anatomy - 35th Edition, Edited by Warwick and Williams, Longman Group Limited, 1973, Edinburgh, p. 839.
5. Wadouh F, Lindemann EM, Ardnt CF. The arteria radicularis magna anterior as a decisive factor influencing spinal cord damage during aortic occlusion. J Thorac Cardiovasc Surg 1984; 88: 1-10.
6. DeMedeiros A, Kramer DW, Perilstein PK. Diseases of the Abdominal Aorta. JAMA 1958; 166: 1711-1717.
7. Lueth HC. Thrombosis of the Abdominal Aorta. Ann Int Med 1940; 13: 1167-1173.
8. Dejerine J. Semiologie Des Affections du Systeme Nerveux. Masson et Cie, Paris, 1914, 267.
9. Hoffman TG, Harati Y, Witaker SH. Transient paraplegia caused by aortic occlusion. Arch Neurol 1981; 38: 668-669.
10. Mills RP, Robbs JV, Duncan HJ. Paraplegia following abdominal aortic surgery. South Afr Med J 1983; 63: 930-932.

11. Cook AW. Occlusion of the abdominal aorta and dysfunction of the spinal cord. Bull NY Acad Med 1959; 35: 479-489.
12. Rudar M, Urbanke A, Radonic M. Occlusion of the abdominal aorta with dysfunction of the spinal cord. Ann Int Med 1962; 56: 490-494.
13. Watson N. A survey of non-traumatic paraplegia. Paraplegia 1981; 19: 107-110.
14. Brindley P, Schwab EH. Aneurysms of the aorta, with a summary of pathological findings in 100 cases at autopsy. Texas St J Med 1930; 25: 757-760.
15. Heggtveit HA. Syphlitic aortitis: a clinico-pathological study of 100 cases 1950-1960. Circulation 1964; 29: 346-355.
16. Lucke B, Rey MH. Studies on aneurysms. II. Aneurysm of the aorta. JAMA 1923; 81: 1167-1170.
17. Rothschild BM, Cohn L, Aviza A et al. Aortic aneurysm producing back pain, bone destruction and paraplegia. Clin Orthop & Relat Res 1982; 164: 123-125.
18. Baird RJ, Gurry JF, Kellam JF et al. Abdominal aortic aneurysms. Can Med Assoc J 1978; 118: 1229-1235.
19. Erikson I, Hallen A, Simonsson N et al. Surgical classification of abdominal aortic aneurysms. Acta Surg Scand 1979; 145: 455-458.
20. Søreide O, Lillestøl J, Christensen O et al. Abdominal aortic aneurysms: Survival analysis of four hundred thirty-four patients. Surgery 1982; 91(2): 188-193.
21. Pilcher DB, Davis JH, Ashikaga T et al. Treatment of abdominal aortic aneurysm in an entire state over 7½ years. Am J Surg 1980; 139(4): 487-494.
22. Ottinger LW. Ruptured arteriosclerotic aneurysms of the abdominal aorta; reducing mortality. JAMA 1975; 233: 147-150.
23. Yashar JJ, Indeglia RA, Yashar J. Surgery for abdominal aortic aneurysms. Factors affecting survival and long term results. Am J Surg 1972; 123: 398-405.
24. Shannon R. Abdominal aortic aneurysm. A review of 12 years experience. Med J Aust 1979; 2: 578-580.
25. DeBakey ME, Crawford ES, Cooley DA et al. Aneurysm of abdominal aorta analysis of results of graft replacement therapy one to eleven years after operation. Ann Surg 1964; 160: 622-639.
26. Wakefield TW, Whitehouse WM, Wu S-C et al. Abdominal aortic aneurysm rupture: Statistical analysis of factors affecting outcome of surgical treatment. Surgery 1982; 91(5): 586-596.
27. Keye G, Bulger J, Shanik G. Paraplegia following abdominal aneurysmectomy. Irish J Med Sci 1980; 149(4): 165-166.
28. Golden GT, Sears HF, Wellons HJ et al. Paraplegia complicating resection of aneurysms of the infrarenal abdominal aorta. Surgery 1973; 73: 91-96.
29. Crawford ES. Thoraco-abdominal and abdominal aortic aneurysms involving renal, superior mesenteric, and coeliac arteries. Ann Surg 1974; 179: 763-772.
30. Crawford ES, Snyder DM, Cho GC, Roehm JOF. Progress in treatment of thoraco-abdominal and abdominal aortic aneurysms involving coeliac, superior mesenteric, and renal arteries. Ann Surg 1978; 188: 404-421.
31. Hirst A, Johns V, Kime S. Dissecting aneurysm of the aorta, a review of 505 cases. Medicine 1958; 37: 217-279.
32. DeBakey ME, McCollum CH, Crawford ES et al. Dissection and dissecting aneurysms of the aorta: Twenty year follow up of five hundred twenty-seven patients treated surgically. Surgery 1982; 92(6): 1118-1134.**
33. Slater E, DeSactis R. The clinical recognition of dissecting aortic aneuryms. Am J Med 1976; 60: 625-633.
34. Waltimo O, Karli P. Aortic dissection and paraparesis. Eur Neurol 1980; 19: 254-257.
35. Dalen JE, Pape LA, Cohn LH et al. Dissection of the aorta: pathogenesis, diagnosis, and treatment. Prog Cardiovasc Dis 1980; 23(3): 237-245.
36. Anagnostopoulos CE, Prabhakar MJS, Kittle CF. Aortic dissections and dissecting aneurysms. Am J Cardiol 1972; 30: 263-273.
37. Wheat MW. Treatment of dissecting aneurysm of the aorta: current status. Prog Cardiovasc Dis 1973; 16: 87-101.
38. Miller DC, Stinson EB, Oyer PE et al. The operative treatment of aortic dissections: experience with 125 patients over a 16 year period. J Thorac Cardiovasc Surg 1979; 78: 365-382.
39. Lindsay J. Aortic Dissection. Clinical Vascular Disease. ed. John A Spittel, F A Davis, Philadelphia 1983, 103-119.
40. Gschaedler R, Dollfus P, Loeb JP, Mole JP, Huber B, Adli G. Traumatic rupture of the aorta and paraplegia. Paraplegia 1978; 16: 123-127.
41. Herendeen TL, King H. Transient anuria and paraplegia following traumatic rupture of the thoracic aorta. J Thorac Cardiovasc Surg 1968; 56: 599-602.
42. Kirsh MM, Behrendt DM, Orringer MD et al. The treatment of acute traumatic rupture of the aorta. Ann Surg 1976; 184(3): 308-315.
43. Katz NM, Blackstone EH, Kirklin JW, Karp RB. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. J Thorac Cardiovasc Surg 1981; 81: 669-674.
44. Riche ML, Modenosi-Frietes J, Djindjian M, Merland JJ. Arteriovenous Malformations (AVM) of the Spinal Cord in Children: A review of 38 cases. Neuroradiol 1982; 22: 171-180.
45. Brusoni B, Colombo A, Merlo L, Marchetti G, Longo T. Hemodynamic and metabolic changes induced by temporary clamping of the thoracic aorta. Eur Surg Res 1978; 10: 206-216.
46. Blaisdell FW, Cooley DA. The mechanism of paraplegia after temporary thoracic occlusion and its relationship to spinal fluid pressure. Surgery 1962; 51: 361-355.
47. Hantler CB, Knight PR. Intracranial hypertension following cross clamping of the thoracic aorta. Anaesthesiology 1982; 56: 146-147.
48. Neglen P, Eklof B, Thomson D. Temporary incomplete ischemia of the legs induced by aortic clamping in man. Scand J Thor Cardiovasc Surg 1983; 17: 289-258.
49. Miyamoto K, Keno 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 1960; 16: 188-199.
50. Silver JR, Buxton PH. Spinal stroke. Brain 1974; 97: 539-550.
51. Cunningham JN, Laschinger JC, Merkin HA et al. Measurement of spinal cord ischemia during operations upon the thoracic aorta. Ann Surg 1982; 196: 285-296.
52. Cunningham JN - In discussion of Reference 79; Coles JG et al.
53. Connolly JE - In discussion of Reference 51; Cunningham JN et al.
54. Bloodwell RD, Hallman GL, Cooley DA. Partial cardiopulmonary bypass for pericardiectomy and resection of descending thoracic aortic aneurysms. Ann Thorac Surg 1968; 6: 46-56.
55. Kahn DR, Vathayanon S, Sloan H. Resection of descending thoracic aneurysms without left heart bypass. Arch Surg 1968; 97: 336-340.
56. Crawford ES, Rubio PA. Reappraisal of adjuncts to avoid ischemia in the treatment of aneurysms of descending thoracic aorta. J Thorac Cardiovasc Surg 1973; 66: 693-704.
57. Crawford ES - In discussion of Reference 51; Cunningham JN et al.
58. Spencer FC, Zimmerman JM. The influence of ligation of intercostal arteries on paraplegia in dogs. Surg Forum 1958; 9: 340-342.
59. Wakabayashi A, Connolly JE. Prevention of paraplegia associated with resection of extensive thoracic aneurysms. Arch Surg 1976; 111: 1186-1189.
60. Laschinger JC, Cunningham JN, Nathan IM, Krieger K, Isom OW, Spencer FC. Intraoperative identification of vessels critical to spinal cord blood supply - Use of somatosensory evoked potentials. Current Surg 1984; 41(2): 107-109.
61. Laschinger JC, Cunningham JN, Isom OW, Nathan IM, Spencer FC. Definition of the safe lower limits of aortic resection during surgical procedures on the thoracoabdominal aorta: Use of somatosensory evoked potentials. J Am Coll Cardiol 1983; 2(5): 959-965.

** Personal communication with Dr. M. DeBakey confirmed that the incidence of postoperative spinal cord ischemia is overstated in reference 32. Table VIII on page 1125 counts some patients more than once and erroneously increases the complication rate.

62. Reiz S, Peter T, Rais O. Hemodynamic and cardiometabolic effects of infrarenal aortic and common iliac artery declamping in man - an approach to optimal volume loading. Acta Anaesth Scand 1979; 23: 579-586.
63. Rutherford RB - Discussion of Bush HL et al. Assessment of myocardial performance and optimal volume loading during elective abdominal aortic aneurysm resection. Arch Surg 1977; 112: 1301-1306.
64. Verendes JN, Bredee JJ, Schipperheyn JJ, Mashour YAS. Mechanisms of spinal cord injury after cross-clamping of the descending thoracic aorta. Circulation (Supp. 1) 1982; 66: 112-116.
65. Turner JM, Powell D, Gibson RM, McDowall DG. Intracranial pressure changes in neurosurgical patients during hypotension induced with sodium nitroprusside or trimetaphan. Br J Anaesth 1977; 49: 419-426.
66. McDowall DG. The influence of anaesthetic drugs and techniques on intracranial pressure; A Basis and Practice of Neuroanaesthesia, ed. E Gordon, Elsevier, Amsterdam 1975, 135.
67. Ivankowitch AD, Miletich DJ, Albrecht RF, Zahed B. Sodium nitroprusside and cerebral blood flow in the anesthetized and unanesthetized goat. Anaesthesiology 1976; 42: 21-26.
68. Adolph RJ, Fukusumi H, Fowler NO. Origin of cerebrospinal fluid pulsations. Am J Physiol 1967; 212: 840-846.
69. Ryder HE, Espey FF, Kimbell FD et al. Modification of effect of cerebral blood flow on cerebrospinal fluid pressure by variations in craniospinal blood volume. Arch Neurol & Psych 1952; 68: 170-174.
70. Bruce DA, Berman WA, Schut L. Cerebrospinal fluid pressure monitoring in children: physiology, pathology, and clinical usefulness. Adv Pediat 1977; 24: 233-290.
71. Fishman RA. Cerebrospinal fluid in disease of the nervous system. W B Saunders, Philadelphia, 1980.
72. Payne EE, Spilanne JD. The cervical spine: An anatomico-pathologic study of 70 specimens with particular reference to the problem of cervical spondylosis. Brain 1957; 80: 571-596.
73. Nordqvist L. The sagittal diameter of the spinal cord and subarachnoid space in different age groups. Acta Radiol Suppl 227, 1964; 5-92.
74. Shapiro K, Marmarou A, Shulman K. Characterization of clinical CSF dynamics and neural axis compliance using the pressure-volume index I: The Normal Pressure volume index, Ann Neurol 1980; 7: 508-514.
75. El-Negamy E, Sedgwick EM. Properties of a spinal somatosensory evoked potential recorded in man. J Neurol Neurosurg Psychiat 1978; 41: 762-768.
76. Thomas PK, Jefferys JGR, Smith IS, Loulakakis D. Spinal somatosensory evoked potentials in hereditary spastic paraplegia. J Neurol Neurosurg Psychiat 1981; 44: 243-246.
77. Zalis AW, Kahsar DF, Lamb CR, Kaller BS. Verification of the pattern of anterior spinal artery syndrome by use of brain evoked potentials. Electromyogr Clin Neurophysiol 1981; 21: 223-227.
78. Sedgwick EM, El-Negamy E, Frankl H. Spinal cord potentials in traumatic paraplegia and quadriplegia. J Neurol Neurosurg Psychiat 1980; 43: 823-830.
79. Coles JG, Wilson GJ, Sima AF, Klement P, Tait GA. Intraoperative detection of spinal cord ischemia using somatosensory cortical evoked potentials during thoracic aortic occlusion. Ann Thorac Surg 1982; 34(3): 299-306.