Intraoperative electrophysiologic monitoring in thoracoabdominal aortic aneurysm surgery

Present by Hung Nguyen, MD, CNIM
Disclosure

- None
Introduction

- Immediate or delayed paraplegia or paraparesis remain complications.
- Intraoperative spinal cord monitoring with SomatoSensory Evoked Potentials (SSEPs) and Motor Evoked Potentials (MEPs) will give Anesthesiologist and Surgeons an estimate of spinal cord perfusion, implement aggressive intervention before treatable spinal cord injury evolves into irreversible neuronal ischemia.
- Reperfusion injury, apoptosis or new postoperative spinal ischemic events may still lead to delayed paraplegia.
Axons of somatic sensory ascending tract travel through the dorsal column white matter of the spinal cord.
SSEPs
SomatoSensory Evoked Potentials

Evaluate the integrity of the somatosensory pathways
Provides information about Lower extremities and Dorsal column of spinal cord perfusion
Recording obtained from
- B - popliteal fossa (Pop)
- C - Cervical spine (Cerv)
- D - Cortical (Cpz)
- A - following stimulation of the Posterior Tibial nerve.
Axons of the upper motor neuron corticospinal tract travel through the anterior lateral white matter and synapse with lower motor neuron in the anterior horn gray matter of the spinal cord.
TcMEPs
Transcranial Motor Evoked Potentials

Evaluate the integrity of the motor pathways
Provides information about grey matter and ventral white matter of spinal cord and extremities perfusion
CMAPs (Compound Muscles Action Potentials) Recording obtained from
- Hand muscle: Abductor digiti minimi (ADM)
- Leg muscles: Tibialis Anterior (TA)
  Extensor Digitorum Brevis (EDB)
  Abductor Hallucis (AH)

Transcranial stimulation
SPINAL CORD ISCHEMIA

- Early detection of spinal cord ischemia is important as it permits early intervention before ischemia evolves into irreversible neuronal ischemia.
- Decreased amplitude and increase latency of Evoked Potentials has proved to neuronal tissue ischemia.
- FP, FN, The Sensitivity, Specificity, Negative predictive value and Positive predictive value of SSEPs and MEPs! …
Left leg ischemia
Right Leg ischemia
Peripheral ischemia, Spinal cord ischemia
Anterior spinal ischemia
Anterior and Posterior Spinal ischemia:
Time of loss response to complete ischemia

- Cortex: 20-30 seconds (EEG)
- Spinal cord sensory white matter: 7-17 minutes (SSEP)
- Spinal cord motor white matter: 5-17 minutes (MEP)
- Spinal cord gray matter: 2-3 minutes (MEP)
- Peripheral nerve (Limb ischemia): 20-25 minutes (SSEP-MEP)
Anatomy and Physiology of spinal cord circulation

- A better understanding of anatomy and physiology of the spinal cord circulation have in recent years led to a reduction risk of postoperative spinal cord ischemia.
- The extensive network of extra- and intraspinal anastomoses protects the spinal cord against ischemia due to segmental arterial occlusion.
- The extensive collateral network and The arterial basket of the conus medullaris allow compensatory flow to the spinal cord when some of the direct inputs to the ASA are compromised during Aortic cross-clamping.
Vascular of the spine and spinal cord

- 31 pairs segmental arteries and their anastomoses: Supply to the spinal column, paraspinal muscles, dura, nerve roots and spinal cord.
- Anastomose extensively across the midline and between levels above and below
- Extraspinal longitudinal system connects the neighboring segmental arteries longitudinally
- Intraspinal extradural system has transverse anastomosis and longitudinal interconnections
- Retrocorporeal and prelaminar arteries interconnect with neighboring and contralateral segmental arteries, provide an excellent collateral circulation.
- The extensive network of extra- and intraspinal anastomoses protects the spinal cord against ischemia due to segmental arterial occlusion
Fig. 2. Drawing showing the artery of the lumbar enlargement and its variations. 1 = superior end of the artery; 2 = inferior end of the artery; 3 and 5 = small arteries of the aortal spinal cord. Percentages refer to the occurrence of the artery of lumbar enlargement with specific spinal nerves in the vertebral canal.

J. Neurol. / Volume 35 / September, 1971

Fig. 9. Radiogram, lateral view, showing the lumbar enlargement of the human spinal cord. Note, on the left, the pathway of the anterior spinal artery and the anterior branches of the lumbar enlargement (1) and the spinal artery (3) from the roots of the cauda equina. Note also, on the bottom, the anastomotic loop of the conus medullaris (4).
Vascular of the spine and spinal cord

**Arteries of the spinal cord:**
- The intrinsic arteries: Central and peripheral system
- The ASA supplies two-thirds of the ventral of the spinal cord
  The anterior gray matter, anterior portion of the posterior gray matter and inner half of the anterior and lateral white matter (Descending motor tract)
- The pairs of PSAs and pial arterial plexus supplies the outer portion of the anterior and lateral white matter and the posterior portion of the posterior gray matter and dorsal columns white matter.
- Their terminal branches overlap, because blood flows away from the center in the central system and toward the center in the peripheral system, their relationship is not truly compensatory
Vascular of the spine and spinal cord

- Capillaries: The density of the capillary bed is 5 times greater in gray matter than in white matter.
- The capillaries beds in white matter stretching longitudinally in the direction of the axon fibers.
- Within the gray matter, the density of the beds depends on the location of the neuron cell bodies. This arrangement reflects the greater metabolic requirements of the cell bodies compared with axons.

Fig. 2 Capillary density of the spinal cord of a baboon (dorsal above, ventral below). The average number of capillaries per square millimetre in the grey and the white matter is indicated in figures. The blood flow in the grey matter is approximately 15 times greater than in the white matter, in response to metabolic demands. Because of the metabolic demands the intraneural plexus of small vessels compels ‘absolute protection and preservation’ (Domnisse (11)) section approximately 500 nm, prepared by the Spalteholz technique for clearing. (Reproduced by courtesy of Professor D P Knobel, Head, Department of Anatomy, University of Pretoria.)
Watershed areas

- The watershed area, border-zone infarct occurs at the junction of two arteries territories and is precipitated by a hemodynamic impairment, although it cannot be excluded by specifically precipitating from micro-embolic etiology.
- 3 watershed zones.
- The first is along the longitudinal axis of the thoracic spinal cord between the arteries of the cervical and lumbar enlargements. At the union of a radiculomedullary artery and the ASA, the blood courses upward and downward from the entry point. Therefore, in the area of the ASA between neighboring radiculomedullary arteries, there is a dead point where blood flows in neither direction, that is, a watershed area.
- The second is over the anterolateral surface of the cord between circumferential pial branches of the anterior spinal artery and the posterior spinal arterial arcade.
- The third is along the gray/white junction between the intramedullary territories of the central arteries and the pial plexus. There is overlap between the pial plexus and central arteries, which produces a watershed zone.
THE BLOOD SUPPLY OF THE HUMAN SPINAL CORD

BY

B. BOLTON

From the Research Unit and Pathological Department, National Hospital, Queen Square, London

(Received 6th March, 1939)

Figure 2. The unique configuration of the anterior spinal artery—medullary artery anastomosis. In addition, an area where flows rise from two adjacent anterior medullary arteries is illustrated. This is an area at potential risk for ischemia.

Anterior
Posterior

Schematic drawing of cord showing source of supply and direction of flow of arterial blood in the spinal cord.
Blood flow currents in spinal cord arteries

Giovanni Di Chiro, M.D., and Larry C. Fried, M.D.

The introduction of selective technique has established spinal cord angiography as a reliable diagnostic tool for the study of the spinal cord vessels and blood flow therein. Very early in our clinical radiological experience dealing with the angiographic aspects of spinal cord vascular disease, we were confronted with the problem of the direction of blood flow in the anterior spinal artery. The concept of descending and ascending blood flow currents in the anterior spinal artery—what we refer to as the anterior spinal arterial axis—has been controversial. On the basis of anatomical observations in man and anatomical and experimental injection studies in animals, two conflicting theories have been advanced. A group of authors—Kadysa, Bolton, Mettler, Mitchell, and Klotz—suggests the theory of a blood flow current from above downward. On the basis of our angiographic observations, we are in agreement with the investigators—Adamkiewicz, Rub and Alexander, Woolam and Miller, Szabo, and Feidner et al.—supporting the other theory that the blood flow from the two adjacent anterior arteries takes place in opposite (converging) directions. Hence, several "watersheds" can be found at points equidistant from the bifurcations of the anterior arteries.

Regarding the posterior spinal arteries, Bolton and Zülch have postulated, and on the basis of our angiographic experience we support this concept, that in the cervical area the blood flows from the posterior area to the anterior area, thereby alternating anterior and posterior flow as the posterior spinal arteries branch off the vertebral arteries. In the thoracic area, blood flow is posterior to anterior to anterior to posterior, and in the lumbar area it is anterior to posterior to posterior to anterior to anterior to posterior. This pattern may be altered by anatomical variations, vascular disease, and other factors such as age, sex, and body position.

Fig. 1. Concept of blood flow direction in spinal cord arteries

Fig. 2. Contrast medium injected by selective arteriography into artery of Adamkiewicz (right arrow) flows in anterior spinal artery dividing into larger descending (lower left arrow) and thinner ascending currents (upper left arrow).

Fig. 3. Contrast medium from radicular branch of coccygeal trunk divides into two diverging currents of equal size (arrows) in cervical segment of anterior spinal artery.
Fig. 7. Possible flow reversals in spinal cord arteries due to intrinsic or extrinsic obstructive vascular disease. [A] Normal; [B] obstruction of vertebral arteries; [C] obstruction of thoracic anterior spinal artery; [D] obstruction of artery of Adamkiewicz; and [E] obstruction of anterior spinal artery below artery of Adamkiewicz. X = site of obstruction.

Fig. 4. [Top] Contrast medium flows down into lower anterior spinal artery to divide into rami craniales and reach posterior lateral spinal arteries where flow direction is upward. [Bottom] Sketch of the direction of flow.

Direction: blood flow of the ASA and anastomotic loop at the Conus

Giovanni Di Chiro, et al.; Neurology; Vol 21, 1971
Fig 3. Two-dimensional image and Doppler ultrasonography. Arrows indicate aortic lumen and the proximal part of the largest segmental artery close to the celiac trunk. The direction of intercostal blood flow is shown in relation to proximal aortic crossclamping.
A through D, Illustration of disrupted spinal cord supply following ligation of a key segmental artery (shown in gray) (A) with three possible compensatory mechanisms for reconstitution of the anterior spinal artery (ASA). Without direct supply to the ASA via the typical flow from the segmental artery to the radiculomedullary artery (RMA), the ASA may be reconstituted by collaterals emanating from an adjacent segment radicular artery (B), communication between the posterior spinal arterial system and the ASA system via the pial plexus and areas of spinal cord parenchymal overlap (C), or compensatory dynamic reversal of flow in the ASA itself using supply from distant RMAs or the anastomotic loop of the conus (D).
The posterior third of the spinal cord is the site of a vascular lesion less frequently than the anterior two-thirds. The reason for this is not clear, but perhaps it may be because of the plexiform character of all arteries on the posterior surface and a greater number of medullary arteries. When there is involvement of

Reversal of blood flow currents in the anterior and posterior spinal arteries may occur in pathological conditions due to hemodynamically active lesions (e.g., arteriovenous malformations in and outside the cord) or due to stenotic or obstructive vascular disease located either in the spinal cord arteries or in the major extraspinal tributaries. Reversals of blood flow direction in the spinal cord arteries may cause steal phenomena with resulting degrees of cord ischemia (Jellinger, 1972).


However, in addition to arterial disorders, disturbances of the venous drainage are important to note. The low pressures that produce circulatory disturbances speak in favor of the view that in the setting of slowly progressing compressive lesions, an impairment of the venous drainage is primarily responsible for the deficient circulation. In a considerable proportion of spinal cord lesions, the pattern suggests an impairment of the venous drainage (Levy and Strauss, 1942; MacNalty and Horsley, 1909; Na et al., 2007).


Microvasculature of the human spinal cord

IAN M. TURNBULL, M.D.
Division of Neurosurgery, University of British Columbia, Vancouver General Hospital, Vancouver, British Columbia, Canada

A

Normal

B

Edema

Fig. 5. Cross section of a normal cord (A) and a flattened cord (B). Flattening of the spinal cord (B) elongates and flattens the small arteries and veins of the lateral columns and gray matter, but merely shortens the vessels of the anterior and posterior columns.

C

Restored blood flow

CSF drainage \leq 10mm Hg

Fig. 8. Speculated mechanism of beneficial effect of cerebrospinal fluid drainage on spinal cord injury (Safi et al., 1997).
CBF (ml/100g/min)

Thresholds of ischemia
- Normal range
- Oligemia
- Ischemia
- Electrical function affected
- Electrical failure complete
- Release of potassium (and cell death)

Cerebral blood flow (ml/100 g/min)

Focal hyperperfusion:
- Mean CBF
- Normal
- Mild hypoperfusion (oligemia)

Onset of decrease in oxidative phosphorylation and increased generation of ROS
- Depression of protein synthesis
- Loss of dendritic structure & spines begins after 7 h
- Increase in lactate, decrease in phosphocreatine
- EEG slowing & decrease in amplitude of EP
- Energy metabolism disturbed, anaerobic glycolysis
- Synaptic transmission failure, EP abolished
- Terminal depolarisation and potassium efflux

Critical threshold
- Irreversible damage, infarction

Postischemic hyperperfusion

Function threshold (mild paresis)
- Penumbra
- Membrane failure

Morphological damage threshold
- Single cell necrosis
- Infarction

ROS: Reactive oxygen; EP: Evoked potentials
“There exist a very close relationship between the metabolic requirements of the nervous tissue and the final distribution of intraneural vessels in the adult, a relationship which functions in such a way as to provide the nervous system with a blood supply just adequate for its minimal needs (Feeney and Watterson; The development of the vascular pattern within the walls of the central nervous system of the chick embryo. Journal of Morphology; 1946.).”
• The blood flow and metabolic rate of the spinal gray matter be 3-5 time greater than white matter.
• The fact that when SSEPs changes suggesting the white matter would be in the ischemic penlucida or penumbra while the gray matter would have reached the stage of irreversible lesions, thereby explaining why paraplegia may occur despite SSEPs recovered.
• Aggressive intervention before treatable spinal cord ischemia evolves into irreversible neuronal ischemia.
Fig. 5. Ischemic/reperfusion injury. Two components of tissue injury contribute to the ultimate neurologic damage. The ischemic component includes the processes of tissue damage occurring during ischemia. Cell death caused by the ischemic component alone depends on the severity and duration of ischemia. The secondary consequences of ischemia include the biochemical changes that take place at the time of reperfusion and reoxygenation after ischemia. The duration of these secondary processes and the extent to which they contribute to ultimate neurologic damage determine the therapeutic window during which treatment administered after ischemia may be effective. ATP, adenosine triphosphate; FFA, free fatty acids; NMDA, N-methyl-D-aspartic acid. (From Cottrell J, Smith D. Anesthesia and neurosurgery. 3rd edition. Philadelphia: Mosby; 1994; with permission.)
Loss MEPs / TEVAR

Baseline

BP 135/70
Loss MEPs / TEVAR

2\textsuperscript{nd} stent deployed, T9 artery

2\textsuperscript{nd} stent deployed: 2 minutes
Loss MEPs / TEVAR

2\textsuperscript{nd} stent deployed : 3 minutes  

2\textsuperscript{nd} stent deployed: 5 minutes
Loss MEPs / TEVAR

Induced HTN, SBP : 146 mmHg

SBP : 187 mmHg
Loss MEPs / TEVAR

SBP: 150 mmHg

Threshold restored MEP,
SBP: 160 mmHg
Thank you for your attention!

<table>
<thead>
<tr>
<th>TOLERABLE ISCHEMIC TIMES</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>KIDNEYS</strong></td>
</tr>
<tr>
<td>GUTS</td>
</tr>
<tr>
<td>LOWER LEGS</td>
</tr>
<tr>
<td>SPINAL CORD</td>
</tr>
<tr>
<td>BRAIN</td>
</tr>
<tr>
<td>HEART</td>
</tr>
</tbody>
</table>