

Effect of spinal cord compression on local vascular blood flow and perfusion capacity by Alshareef M, Krishna V, Ferdous J, Aishareef A, Kindy M, Kolachalama VB, et al.

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Received: 28 July 16 Accepted: 29 July 16 Published: 22 September 16

Abstract

Background: Different degrees of blood flow/vascular compromise occur with anterior, posterior, or circumferential spinal cord compression/spinal cord injury (SCI). SCI is also divided into primary and secondary injury. Primary SCI refers to the original neurological damage to tissues, whereas secondary injury reflects interruption of normal blood flow leading to further inflammatory response/ other local changes which contribute to additional neurological injury.

Methods: The authors developed a quantitative “3-D finite element fluid structure interaction model” of spinal cord blood flow to better document the mechanisms of secondary ischemic damage occurring in the spinal cord anteriorly, posteriorly, or circumferentially. This included assessment of the anterior spinal artery (ASA) and five arterial branches (L1, L2, L3, R1, R2), but excluded the microvasculature.

Results: Different locations of cord compression resulted in alternative patterns of spinal cord ischemia. Anterior spinal artery (ASA) flow was substantially reduced by direct anterior compression, but resulted in the least vascular compromise. Alternatively, posterior compression resulted in a significant and critical reduction of distal ASA blood flow and, therefore, correlated with the greatest susceptibility to acute ischemia. Counterintuitively, they concluded “at equivalent degrees of dural occlusion, the loss of branch blood flow under anterior posterior compression was intermediate to predictions for purely posterior or anterior loading.”

Conclusion: Utilizing a computational three-dimensional model, Alshareef *et al.* observed that anterior cervical cord compression resulted in the least severe compromise of ASA blood flow to the spinal cord, whereas posterior cord compression/SCI maximally reduced distal ASA blood flow potentiating acute ischemia. Therefore, the latter warranted the earliest surgical intervention.

Key Words: Anterior, anterior spinal artery, circumferential, cord compression, distal compromise, posterior, spinal cord injury, vascular compromise

Access this article online

Website:

www.surgicalneurologyint.com

DOI:

10.4103/2152-7806.191077

Quick Response Code:



COMMENTARY

The authors evaluated the extent of vascular compromise attributed to anterior, posterior, or circumferential spinal cord compression/injury (SCI).^[1] SCI related ischemia is typically divided into primary and secondary injury. Primary injury refers to the original neurological damage to tissues related to trauma, whereas secondary injury reflects

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How to cite this article: Epstein NE. Effect of spinal cord compression on local vascular blood flow and perfusion capacity by Alshareef M, Krishna V, Ferdous J, Aishareef A, Kindy M, Kolachalama VB, et al. *Surg Neurol Int* 2016;7:S682-5. <http://surgicalneurologyint.com/Effect-of-spinal-cord-compression-on-local-vascular-blood-flow-and-perfusion-capacity-by-Alshareef-M,-Krishna-V,-Ferdous-J,-Aishareef-A,-Kindy-M,-Kolachalama-VB,-et-al/>

an interruption of the normal blood/brain/spinal cord barrier resulting in an inflammatory response/other local changes, which lead to further neurological injury. The resultant cord ischemia then contributes to increased damage of neurological tissues: “progressive spinal cord ischemia, which can cause necrosis and irreversible tissue damage if perfusion falls below a critical level or vascular threshold.”

Development of three-dimensional quantitative model of spinal cord blood flow

The authors have attempted to develop a quantitative model of spinal cord blood flow to better understand the mechanism of secondary ischemic cord damage. They evaluated how continued mechanical compromise of the cord alters/compromises local blood flow resulting in differing degrees of ischemia. This required the development of a “3-D finite element fluid structure interaction model” to evaluate the impact of different degrees of spinal cord compression and to assess the subsequent extent of blood flow compromise (e.g. through the anterior spinal artery and “5 of its braches (L1, L2, L3, R1, R2)”^[1]

Vascular anatomy of the spinal cord

There is no clear discussion in this paper as to what L1-L3 and R1, R2 represent nor of the presence/role of the posterior spinal artery. Anatomically, the anterior spinal artery (ASA) and its distal branches and/or microvasculature constitute the arterial supply to the anterior two-thirds of the spinal cord, whereas the posterior spinal arteries/branches supply the posterior one-third of the cord. Thron further described the vascular anatomy of the spinal cord as consisting of the ASA feeding the bilateral anterior radicular arteries, and bilateral transverse/longitudinal interconnections.^[6] The posterior spinal artery feeds the bilateral posterolateral spinal arteries and the posterior radicular arteries. Ultimately, both the anterior and posterior radicular arteries join laterally to form the nervomedullary artery.

Compression models assess anterior and posterior cord blood flow

The authors evaluated blood flow through the anterior and posterior cord utilizing the 3 (anterior, posterior, and 360°) compression models. They concluded; “the percent dural occlusion alone is an insufficient predictor of the ischemia potential following SCI.” Surgical intervention was warranted following SCI depending upon both the “extent of occlusion” and “orientation of the cord compression.”^[1]

Anterior and/or posterior, compression differently impact cord blood flow

Anterior compression

Different types of compression resulted in alternative

patterns of ischemia, some resulting in adverse events sometimes difficult to discern (e.g. present on subclinical levels). They noted that anterior compression of ASA flow could be substantially reduced; “aberrant ASA mechanics give rise to a potential for chronic complications following anterior compression.”^[1] Specifically, they observed with 6.5% dural compromise, a 67% reduction in ASA blood flow.

Posterior compression

With posterior compression, there was an increased susceptibility to acute ischemia attributed to the greater potential for maximal compromise of distal but not proximal ASA blood flow. In fact, alternations of distal blood flow were proportional to the “vessel distance from the loading surface of the dura mater.”^[1]

Anteriorposterior compression

With anteriorposterior compression, blood flow was compromised both through the ASA and its distal branches. Specifically, with a 9.65% degree of dural compromise, there was a 50% loss of blood flow most severely impacting the ASA. Nevertheless, and certainly counterintuitively, they concluded: “at equivalent degrees of dural occlusion, the loss of branch blood flow under anterior posterior compression was intermediate to predictions for purely posterior or anterior loading.”^[1]

Monkey models of cord ischemia

Two studies, one by Doppman et al, and the other by Ramsey et al., utilized a monkey model to assess “controlled epidural compression via balloons introduced percutaneously both anterior and posterior to the spinal cord.”^[2,5] With spinal angiography, they could determine the extent/adequacy of perfusion.

Spinal epidural abscesses

When evaluating spinal epidural abscesses (SEA), the authors noted in the literature that posterior compressive changes resulted in maximal early ischemic injury vs. anterior SEA. In one study involving a posteriorly located SEA, patients exhibited an increased incidence of severe paraplegia or quadriplegia (30.6% of the time) vs. for those with anteriorly located SEA (7.3%).^[3] These data led to the recommendation for early surgery for posterior spinal cord SEA to avoid the increased susceptibility to irreversible neurological ischemic damage.

Cervical spine jumped facets (SCI)

For patients with cervical spine injuries attributed to jumped facets, early decompression (e.g., within 4 hours) resulted in better outcomes vs. those performed after 25 hours (e.g. better neurological recovery/reversible ischemia with early vs. later surgery).^[4,7]

Spinal cord compression models and timing of surgery

This study indicates that posterior cord compression/SCI contributes to more significant distal compromise of

ASA branches and a heightened risk of acute cord ischemia warranting early surgery. What was counterintuitive was the finding that “the loss of branch blood flow under anteriorposterior compression was intermediate to predictions for purely posterior or anterior loading.” Nevertheless, here, clinical judgment should also prompt early surgery as circumferential compression, either leading to overt myeloradiculopathy or “in its sub clinical state” can contribute to cord ischemia, irreversible cell loss, and axonal degeneration.

Import of modeling spinal cord vascular ischemia

This study provided a new, scientific model of spinal cord ischemia. Furthermore, it correlated progressive compressive/ischemic anterior, posterior, and anteriorposterior blood flow changes in the cervical cord with the potential for increased and irreversible cord damage/myelopathy resulting from SCI. The major conclusion was that the posterior cord was most susceptible to ischemic damage, and that, therefore, early surgery was warranted. For anteriorposterior cord compression/damage, it would seem that the extent/degree of anterior and posterior cord compromise should best determine the optimal timing for operative intervention. Of interest, although an ischemic model of the central cord syndrome was not within the scope of this paper, future studies should analyze the unique vascular compromise contributing to these lesions and perhaps shed light on the optimal timing of their surgical management (e.g., most studies now promote early within 24 hours).

CONCLUSION

Utilizing a computational 3D model, Alshareef *et al.*

Author Response

The above commentary reaffirms the importance of computational modeling in the understanding of spinal cord injury. Current imaging and diagnostic tools are limited in their ability to assess spinal cord perfusion. Therefore, it is necessary to come up with theoretical models that provide a framework for observed clinical phenomena. We believe this commentary is a great example of clinical validation of a model that is difficult to directly prove, given the current clinical limitations. Furthermore, the current model suggests consideration of perfusion as a new variable in both acute and chronic cervical spinal cord compression. As noninvasive imaging sequences such as arterial spin labeling (ASL) and vascular space occupancy (VASO) are enhanced for smaller voxel spaces, correlation of spinal cord perfusion and outcomes can aid in decision-making for the necessity and timing of surgical intervention.

documented that anterior spinal cord injury resulted in the least reduction of spinal cord blood flow, while posterior cord injury contributed to the greatest reduction of blood flow (e.g. with ASA distal branch occlusion). Models such as this should prompt clinicians to intervene earlier with posterior spinal cord compressive lesions/injuries, as these are the most likely to result in early, permanent, and irreversible ischemic deficits. Although beyond the scope of this study, ischemic models like this one may in the future, more clearly prompt early surgery for central cord injuries and other SCI-related syndromes.

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Recent literature shows the utility of spinal cord perfusion (SCP) along with intraspinal pressure in providing guidance for modifications of surgical management. Varsos *et al.* developed a subdural pressure monitor for a group of patients with spinal cord injury, and trialed several chemical and surgical interventions. Although a small study, they noted that most patients who underwent laminectomy alone had no improvement in spinal cord perfusion pressure (SCPP), measured indirectly from intraspinal pressure and mean arterial pressures.^[2] In comparison, the group published a follow-up study in which patients who underwent a duroplasty following traumatic spinal cord injury had lowering of their intraspinal pressure, and increased SCPP.^[1] They also found correlation between a rise in SCPP and

improved outcomes in several patients, although with low statistical power. Larger studies are needed to assess outcomes following interventions aimed at maintaining adequate SCP. We concluded that SCP is an essential variable for incorporation in clinical decision-making. Moreover, clinical validation of our computational model can yield more insight on the correlation between perfusion (both clinical and sub-clinical) and patient outcomes following spinal cord injury.

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