Management of severe spinal cord injury following hyperbaric exposure

There is an increasing body of evidence that drainage of lumbar cerebrospinal fluid (CSF) improves functional neurological outcome after reperfusion injury to the spinal cord that occasionally follows aortic reconstructive surgery.[1,2] This beneficial effect is considered owing to lowering of the CSF pressure thereby normalising spinal cord blood flow and reducing the 'secondary' cord injury caused by vascular congestion and cord swelling in the relatively confined spinal canal. Whilst lacking definitive proof, there are convincing randomised controlled trials (RCTs), cohort data and systematic reviews supporting this intervention. The therapeutic window for lumbar CSF drainage requires further elucidation; however, it appears to be days rather than hours post insult.[3,4] We contend that the same benefit is likely to be achieved following other primary spinal cord injuries that cause cord swelling and elicit the 'secondary' injury.

Traditionally the concept of CSF drainage has been considered more applicable to the brain as contained in a 'closed box' by lowering intracranial pressure (ICP) to improve cerebral perfusion pressure (CPP). The control of CPP is intended to limit 'secondary' brain injury and is a key concept of brain injury management. Using microdialysis in the spinal cords of trauma patients, it has been shown that intraspinal pressure (ISP) needs to be kept below 20 mmHg and spinal cord perfusion pressure (SCPP) above 70 mmHg to avoid biochemical evidence of secondary cord damage.[5] Vasopressor have also been used in spinal cord injury to improve perfusion, however complications are common, typically cardiac in nature, and require very careful monitoring; the evidence supporting this approach is notably less convincing.

Decompression illness (DCI) of the spinal cord is treated with recompression, hyperbaric oxygen, various medications designed to reduce the inflammatory response and fluid administration to normalise blood pressure and haematocrit.[6] These management protocols are based largely on anecdote and transferred evidence from conventional cord trauma, as the low numbers and sporadic nature of DCI in divers makes RCTs nigh on impossible. Unfortunately even with best management, some patients are left with significant neurological deficit.

The 'iceberg phenomenon', occurs when patients with DCI of the cord make a good neurological recovery but actually have profound cord damage as revealed in one case some four years later at post mortem and another example in a diver who developed late functional deterioration due to loss of neuronal reserve.[7,8] This clinical evidence, together with animal study data, support the notion that even a modest preservation of spinal cord axons is associated with significant improvement in neurological outcome.[9]

In the light of the positive level two evidence in the vascular literature that CSF drainage limits 'secondary' injury thereby improving neurological outcome, we propose that centres with appropriate clinical experience consider using lumbar CSF drainage to normalise SCPP, as an adjunct to the conventional treatment of severe spinal cord DCI. Divers with severe spinal cord DCI are generally in the most productive years of their lives and, given the potentially devastating impact of this condition, should be given the benefit of any possible adjuvant treatment that may serve to improve long-term outcome.

References

1 Sobel JD, Vartanian SM, Gasper WJ, Hiramoto JS, Chuter TA, Reilly LM. Lower extremity weakness after endovascular aneurysm repair with multibranched thoracoabdominal stent grafts. *J Vasc Surg*. 2015;61:623-8.
2 Cina CS, Abouzahr L, Arena GO, Lagana A, Devereaux PJ, Farrokhyar F. Cerebrospinal fluid drainage to prevent paraplegia during thoracic and thoracoabdominal aortic aneurysm surgery: a systematic review and meta-analysis. *J Vasc Surg*. 2004;40:36-44.
3 Safi HJ, Campbell MP, Miller CC, 3rd, Iliopoulos DC, Khoynezhad A, Letsou GV, et al. Cerebral spinal fluid drainage and distal aortic perfusion decrease the incidence of neurological deficit: the results of 343 descending and thoracoabdominal aortic aneurysm repairs. *Eur J Vasc Endovasc Surg*. 1997;14:118-24.
4 Richards JM1, Hayward I, Moores C, Chalmers RT. Successful management of both early and delayed-onset neurological deficit following extent II thoracoabdominal aneurysm repair. *Eur J Vasc Endovasc Surg*. 2008;35:593-5.
5 Phang I, Saadoun S, Papadopoulos M. High intraspinal pressure and low spinal cord perfusion pressure are detrimental after traumatic spinal cord injury: a microdialysis study. *B J Nsurg*. 2015;29:129.
6 Broome JR. Aspects of neurological decompression illness: a view from Bethesda. *J R Nav Med Serv*. 1995;81:120-6.
7 Palmer AC, Calder IM, McCallum RI, Mastaglia FL. Spinal cord degeneration in a case of "recovered" spinal decompression sickness. *Br Med J (Clin Res Ed)*. 1981;283(6296):888.
8 Dyer J, Millac P. Late deterioration after decompression illness affecting the spinal cord. *Br J Sports Med*. 1996;30:362-3.
9 Bradbury EJ, McMahon SB. Spinal cord repair strategies: why do they work? *Nat Rev Neurosci*. 2006;7:644-53.

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