

## Commentary

In the current issue of Journal of Neurosciences in Rural Practice the article by Tuzgen *et al.*, "Decompressive craniectomy in patients with cerebral infarction due to malignant vasospasm after aneurysmal subarachnoid hemorrhage" explores the issue of surgical decompression for refractory intracranial hypertension following aneurysmal subarachnoid hemorrhage. The authors report 6 patients who presented with aneurysmal subarachnoid hemorrhage and who subsequently developed vasospasm following successful surgical clipping of the ruptured aneurysm. Decompressive surgery was performed when the intracranial pressure (ICP) was persistently greater than 20 mmHg despite maximal medical management.

The surgical decompression was uneventful in each case, and outcome was adjudged "good" (mRS 0-3) in 3 cases and "bad" (mRS 4-6) in 3 cases. Overall, this study presents supportive evidence that decompressive craniectomy may be a useful method, by which intractable intracranial hypertension may be controlled, and the authors should be congratulated on a well-presented paper. However, considerable caution must be exercised before accepting the conclusion that surgical decompression is a life-saving procedure, which provides a better outcome.

As stated by the authors, this was a small study with no controls with which the intervention could be

compared, and it must be acknowledged that the role of decompressive craniectomy remains controversial. Whilst it is widely accepted that raised ICP is strongly associated with poor outcome and mortality, one of the fundamental difficulties in neurosurgery is demonstrating that therapies aimed at decreasing ICP actually improve clinical outcome.

For many years, patients were routinely hyperventilated, placed in a barbiturate coma, or more recently rendered hypothermic as these measures consistently reduce the intracranial pressure (ICP). However, numerous clinical studies have failed to demonstrate these techniques provide clinical benefit and indeed, in some instances, they may cause harm.<sup>[2-4]</sup> On face value, this may appear counterintuitive; however, the results of cerebral blood flow studies indicate a reason for treatment failure. There is little doubt that barbiturates and hypothermia have the potential to be neuroprotective due to their influence on many aspects of the cellular response to injury. In addition, hypothermia has been demonstrated to provide neuroprotection when administered early in the context of cerebral ischemia. However, the predominant mechanism by which all these therapies reduce the intracranial pressure is by cerebral vasoconstriction, and the subsequent reduction in cerebral blood flow has been clearly demonstrated by a number of studies.<sup>[4]</sup> It is here that decompressive craniectomy may have at least a theoretical advantage because, in addition to lowering the intracranial pressure, it has been shown to improve cerebral blood flow.<sup>[5]</sup>

Given these findings, the suggestion by the authors of this article, that surgical decompression should be performed early, would appear to be entirely reasonable; indeed, this was the hypothesis on which the recent DECRA study was based.<sup>[6]</sup> Unfortunately, the findings of this study were not supportive of surgical decompression. The trial compared early bifrontal decompression with standard medical therapy and at 6 month follow-up; outcome in patients in the surgical arm of the trial was worse than those patients who had received standard medical therapy. Notwithstanding some problems with randomization and crossover, it must be acknowledged that the results are unequivocal and have clearly demonstrated that early decompression for relatively mild and transient intracranial hypertension did not improve outcome and may indeed have caused more harm. Prior to the findings of the study, it was almost assumed that lowering the intracranial pressure by surgical decompression would be beneficial, and whilst many authorities remain convinced of clinical efficacy, it has to be accepted that this remains scientifically unproven. What remains to be established is at what

stage (if any) the benefit provided by decompression outweighs the morbidity of the surgery because it is becoming increasingly apparent that this morbidity is not insignificant.<sup>[7]</sup>

A final issue that is raised by the study in this publication is that of long term outcome. Three of the survivors in this study had a Modified Rankin score of 4 or less, and this represents a very poor level of neurological function with the patients by definition being heavily dependent. The authors do not comment on how acceptable that outcome is for the patients or their families; however, a recent study found that for patients that had survived after decompressive hemicraniectomy for ischemic stroke, retrospective consent would not be obtained from those patients that remained with this level of disability.<sup>[8]</sup>

Overall, it can be seen that the precise role of decompressive craniectomy in the management of intractable intracranial hypertension remains to be established. Ongoing and new randomized controlled trials must be developed and supported in order to establish clinical benefit, and long term outcome must be continually evaluated to ensure that those that survive “life-saving” surgical intervention do so in a condition that they “themselves” feel to be acceptable.<sup>[9]</sup>

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