SHORT REPORT

Effects of decompressive craniectomy on brain tissue oxygen in patients with intracranial hypertension

M Jaeger, M Soehle, J Meixensberger

This report examined the intraoperative course of partial pressure of brain tissue oxygen (PtiO2) and intracranial pressure (ICP) during surgical decompressive craniectomy for medically intractable intracranial hypertension due to diffuse brain swelling in three patients after severe subarachnoid haemorrhage and aneurysm coiling. The mean ICP decreased from 59 mm Hg to 10 mm Hg in a two step fashion, relating to bone flap removal and dural opening. Simultaneously, PtiO2 increased rapidly from 0.8 kPa (6 mm Hg) to 3.07 kPa (23 mm Hg). PtiO2 and ICP remained at non-critical ranges postoperatively. Despite these beneficial effects on ICP and PtiO2, the patients’ clinical status remained poor with two in a persistent vegetative state and one dead.

Control of increased intracranial pressure (ICP) remains an important challenge in the treatment of patients with severe post-stroke or post-traumatic brain oedema. Decompressive craniectomy has been proposed as an effective treatment as beneficial effects on outcome have been reported in clinical trials on patients with traumatic brain injury and middle cerebral artery stroke.14 Anecdotal reports and small series suggest decompression may also be successful in other diseases associated with high ICP, such as encephalitis, metabolic encephalopathy, and subarachnoid haemorrhage (SAH).4–9 There exists little information about the pathophysiological changes induced by the cranial decompression. To gain more information about these effects, we investigated the intraoperative course of ICP and partial pressure of brain tissue oxygen (PtiO2) during surgical decompression in three patients with medically intractable intracranial hypertension, occurring after severe aeurysmal SAH. The monitoring of PtiO2 with the polarographic Clark-type probe has been proven to be a reliable tool for detecting cerebral hypoxic events after severe cerebral insults.10–12 The estimated threshold for significant cerebral tissue hypoxia is reported to be at about 1.33 kPa (10 mm Hg). Values below this threshold indicate critical cerebral oxygenation and a high risk of secondary brain damage.

Patients
Three patients suffering from severe cerebral oedema and intracranial hypertension after aneurysmal SAH were studied. Clinical data are given in table 1. External ventricular drains for haemorrhagic hydrocephalus were placed in all patients after admission and the aneurysms were coiled within two days of SAH. Thereafter, all patients developed increased intracranial pressure because of diffuse brain swelling refractory to medical treatment, including analgesia, sedation, mannitol, hypertonic saline, TRIS buffer (THAM), moderate hyperventilation (Paco2 about 4.67 kPa (35 mm Hg)) and barbiturate coma.

ICP probes (Codman and Shurtleff, Raynham, MA, USA) and PtiO2 probes (LICOX Systems, GMS mbH, Kiel, Germany) were inserted into the cerebral white matter via a double lumen bolt located about 15 mm lateral to midline and 20 mm anterior to the coronal suture. PtiO2 probes were placed into CT visible tissue at a depth of 22–27 mm in the anterior cerebral artery vascular territory, as this was initially considered to be tissue at risk for development of symptomatic cerebral vasospasm. Neuroradical monitoring started 4 hours, 46 hours, and 33 hours after the haemorrhage. To observe the immediate effects of decompression, data collected intraoperatively of ICP and PtiO2 were stored on a computer with a rate of 6/min. Mean arterial pressure was monitored via a radial artery catheter referenced to the foramen of Monro.

Decompressive fronto-temporo-parietal craniectomy (diameter about 12 cm) was performed after medical treatment failed to keep ICP values below 30 mm Hg. Preoperatively, the implanted probes were meticulously covered with a sterile dressing to avoid contamination of the surgical field. After the removal of the bone flap, the dura was opened to provide maximum reduction of ICP and duraplasty with periosteum was performed.

Results
Before removal of the bone flap patients exhibited hypoxic mean PtiO2 values of 0.8 kPa (6 mm Hg) and mean ICP of 59

Abbreviations: ICP, intracranial pressure; PtiO2, partial pressure of brain tissue oxygen; SAH, subarachnoid haemorrhage

Table 1  Clinical data of three patients undergoing intraoperative monitoring of ICP and PtiO2 during decompressive craniectomy

<table>
<thead>
<tr>
<th>Case</th>
<th>Age (y), sex</th>
<th>WFNS on admission</th>
<th>Aneurysm location</th>
<th>Side of Probes</th>
<th>Side of decompression</th>
<th>Decompression (days after SAH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60, F</td>
<td>5</td>
<td>A-com-A</td>
<td>Left</td>
<td>Left</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>36, F</td>
<td>5</td>
<td>ICA left</td>
<td>Left</td>
<td>Left</td>
<td>2</td>
</tr>
<tr>
<td>3</td>
<td>36, F</td>
<td>4</td>
<td>ICA right</td>
<td>Right</td>
<td>Right</td>
<td>2</td>
</tr>
</tbody>
</table>

A-com-A, anterior communicating artery; ICA, internal carotid artery; SAH, subarachnoid haemorrhage; WFNS, World Federation of Neurosurgeons grading scale for SAH.
mm Hg. The individual intraoperative course of P_O2 and ICP for each patient is shown in figure 1. The immediate two-step reduction of ICP to 32 mm Hg after removal of the bone flap and to 10 mm Hg after opening of the dura was accompanied by a simultaneous improvement of P_O2 above hypoxic thresholds to 3.07 kPa (23 mm Hg). During the procedures mean arterial pressure was stable between 100 mm Hg and 120 mm Hg in all three patients. P_O2 was kept at about 16 kPa (120 mm Hg) and frequently checked by arterial blood gas measurements. The postoperative course P_O2 and ICP constantly remained at non-critical ranges. Routine CT scan obtained at the first postoperative day excluded cerebral herniation and showed the maintained correct position of the implanted probes. Critical cerebral vasospasm at the time of decompression was unlikely, as preoperative and postoperative transcranial Doppler measurements revealed blood flow velocities below 120 cm/s. At six months after the haemorrhage, patients 1 and 2 remain in a persistent vegetative state and patient 3 died. No wound infections occurred in the postoperative period.

DISCUSSION

The results of these three cases of intraoperative P_O2 monitoring demonstrate that the immediate reversal of critical cerebral oxygenation is possible with the use of decompressive craniectomy. In all these patients suffering from severely raised ICP and diminished cerebral perfusion pressure, P_O2 rapidly increased to non-hypoxic levels. The simultaneous dramatic two-step reduction of raised ICP to normal values during removal of the bone flap and dural opening has been described in a similar pattern by Yoo et al., however, our data for the first time provide evidence of immediate positive effects on cerebral tissue oxygenation in humans. Previous clinical reports suggest that the decompression related increase in P_O2 is predominantly induced by simultaneous increases of cerebral blood flow. With the rapid normalisation of cerebral perfusion pressure, both transcranial Doppler and single photon emission computed tomography studies were able to show that the depressed cerebral circulation improved after the procedure. Furthermore, during evacuation of acute subdural haematomas, rapid increases of laser Doppler flow have been found, indicating improved cerebral blood flow and oxygen delivery.

Despite the demonstrated positive effects of surgical decompression on cerebral oxygen content, the rapid and successful treatment of low P_O2 and raised ICP did not translate into improved outcome with two patients remaining in a persistent vegetative state and one dead. The beneficial effects on cerebral oxygen content and ICP because of decompression were most probably offset by the devastating primary brain damage attributable to the initial haemorrhage and the natural course of such severe primary irreversible loss of neuronal function. In addition, ICP and P_O2 were in highly abnormal ranges at the time of intervention, making secondary brain damage very probable.

As well as post-SAH swelling, we are currently expanding our study to include post-traumatic patients. However, difficulties arise because the timing (early compared with "last option") of the surgical decompression may well change the pathophysiological responses. Based on the generally accepted knowledge that high ICP and low P_O2 levels are important contributors to a poor outcome, the data presented favour the early use of decompression, particular if metabolic monitoring is being carried out. Our experience suggests, however, that extended neuromonitoring with intracranial probes via a multichannel skull bolt during decompression is a feasible method for intraoperative evaluation of neurometabolic parameters.

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Litle is written in textbooks of medical history about Nansen, who is better known as the Norwegian who founded modern polar exploration. His contributions were in many spheres. Nansen was an invertebrate zoologist who in 1882 was appointed curator of zoology at the Bergen museum. He stayed in Bergen for 5 years, focusing his interests on the neuroanatomy of marine invertebrates. For one of his papers “The structure and combination of histological elements of the central nervous system” (1887), the university in Kristiana conferred upon him the degree of doctor of philosophy. His dissertation contained so many novel interpretations that the examination committee accepted it with reluctance, but the work is now considered a classic. Two days after his dissertation was accepted Nansen was on his way to Greenland. He crossed Greenland on skis during 1888–1889.

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References

L F Haas
Fridtjof Nansen (1861–1930)

L F Haas

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