



## POINT OF VIEW

# Does decompressive craniectomy improve other parameters besides ICP? Effects of the decompressive craniectomy on tissular pressure?

S. Lubillo,<sup>a,\*</sup> J. Blanco,<sup>b</sup> P. López,<sup>a</sup> J. Domínguez,<sup>c</sup> C. Ruiz,<sup>a</sup> I. Molina,<sup>a</sup> J. Morera<sup>d</sup>

<sup>a</sup>Unidad de Medicina Intensiva, Hospital Universitario Nuestra Señora de Candelaria, Sta. Cruz de Tenerife, Spain

<sup>b</sup>Unidad de Medicina Intensiva, Hospital Universitario Dr. Negrín, Gran Canaria, Spain

<sup>c</sup>Servicio de Neurocirugía, Hospital Universitario Nuestra Señora Candelaria, Sta. Cruz de Tenerife, Spain

<sup>d</sup>Servicio de Neurocirugía Hospital Universitario Dr. Negrín. Gran Canaria, Spain

Received 12 August 2010; accepted 29 September 2010

### KEYWORDS

Decompressive  
Craniectomy;  
Intracranial  
hypertension;  
Brain tissue oxygen  
pressure

**Abstract** Second level therapeutic maneuvers for controlling intracranial hypertension (ICH) proposed by the European Brain Injury Consortium and the American Association of Neurological Surgeons include barbiturates, moderate hypothermia and more recently the decompressive craniectomy (DC). In most patients, ICP can be maintained below 25 mmHg after a DC. However, the exact effect of DC on brain oxygenation (PtiO<sub>2</sub>) still unclear. From our point of view the PtiO<sub>2</sub> monitoring with the probe located in the healthy area of the most severely damaged cerebral hemisphere is not only a important tool for timing craniectomy in the future but also for evaluating the therapeutic effectivity of DC.

© 2010 Elsevier España, S.L. and SEMICYUC. All rights reserved.

### PALABRAS CLAVE

Craniectomía  
Descompresiva;  
Hipertensión  
intracraneal;  
Presión tisular de  
oxígeno cerebral

**¿Mejora la craniectomía descompresiva otros parámetros además de la PIC? Efectos de la craniectomía descompresiva en la presión tisular**

**Resumen** Las medidas terapéuticas de segundo nivel para el control de la hipertensión intracraneal que propone el European Brain Injury Consortium y la American Association of Neurological Surgeons son los barbitúricos, la hipotermia moderada o más recientemente la craniectomía descompresiva (CD). En la mayoría de los pacientes la Presión intracraneal se mantiene por debajo de 25 mmHg tras la CD. Sin embargo, el efecto de la CD sobre la monitorización de la presión tisular de oxígeno cerebral (PtiO<sub>2</sub>) no está claro. Desde nuestro punto de vista, la monitorización de la PtiO<sub>2</sub> con el catéter colocado en área aparentemente sana del hemisferio más dañado no solo es una herramienta útil para la indicación del momento de la CD sino también para evaluar la efectividad terapéutica de la misma.

© 2010 Elsevier España, S.L. y SEMICYUC. Todos los derechos reservados.

\*Corresponding author.

E-mail address: lubimon@gmail.com (S. Lubillo).

Intracranial hypertension (ICH) without space-occupying lesions remains the most frequent cause of death and disability following a serious brain condition, particularly (but not exclusively) among trauma patients.<sup>1,2</sup> Approximately 10-15% of all patients with severe cranioencephalic traumatism (CET) suffer ICH refractory to the maximum clinical management measures.<sup>1,3-6</sup> In this situation, the intensivist and neurosurgeon face the dilemma of having to decide which second-level therapeutic options can be used: high barbiturate doses, moderate hypothermia or decompressive craniotomy (DC), as proposed by the European Brain Injury Consortium<sup>7</sup> and the American Association of Neurological Surgeons (AANS).<sup>8</sup>

Despite the recommendations of the Brain Trauma Foundation<sup>9</sup> on the use of barbiturates, the updated Cochrane review<sup>10</sup> concludes that although such treatment is able to reduce intracranial pressure (ICP), there is no evidence that such reduction implies lesser mortality or improved outcome among the survivors. Moreover, the decrease in ICP was associated to a drop in arterial pressure in at least one out of every four cases.

While prophylactic moderate hypothermia was discarded after the study published by Cifton,<sup>11</sup> there are data suggesting that it could be used as a second level measure for the control of ICH,<sup>12,13</sup> though there are no definitive conclusions in this regard.

Thus, DC possibly may represent a reasonable second level option which both the intensivist and neurosurgeon may prescribe for the treatment of refractory ICH in critical neurological patients when there are no space-occupying lesions.

Although over 240 articles related to DC have been published in the last 10 years (100 of them in the last two years), no randomized, prospective and controlled study (class I evidence) has been made to date in adults, demonstrating the benefit of DC in relation to final patient outcome. Nevertheless, recently many articles offering class III evidence have been published,<sup>14-17</sup> together with some non-randomized, prospective single-center studies,<sup>18-21</sup> and the results regarding the control of refractory ICH were found to be better with DC than in its absence.<sup>22-25</sup>

Considering the above, neurosurgeons and intensivists have shown renewed interest in cranial decompression and aperture of the dura mater for controlling ICH refractory to first-level management measures, despite the fact that the Cochrane review conducted by Sahuquillo et al.<sup>26</sup> on the use of DC in ICH refractory to treatment among brain injury patients concluded that there are no data to either confirm or refute the effectiveness of such treatment in adults.

In most of the analyzed studies on decompressive craniotomy, the authors monitored post-decompressive craniotomy ICP in the decompressed hemisphere. In this region the brain expands, shifting the pressure-volume curve to the right. This would explain the decrease in ICP recorded in the majority of the studies made.<sup>14-17,27</sup> In our experience, commented in a recent review published in 2009,<sup>28</sup> where ICP was monitored in 28 patients, the median ICP prior to DC was 33 mmHg (range 28-45). In 22 of these patients (78.6%) the pressure dropped to < 25 mmHg after DC, and the outcome proved unfavorable in the 6 patients in which the ICP remained > 25 mmHg.

Due to the increase in brain blood volume after DC, and since the brain is no longer contained within a closed space, some reviews on the management of ICH<sup>29</sup> propose that the threshold for the management of ICP should be lowered to 15 mmHg in these patients. In addition, the Cambridge group<sup>30</sup> has suggested the consideration of lower values of mean arterial pressure and cerebral perfusion pressure (CPP), for although cerebral adaptability improves after DC, vasoconstrictive blood vessel reactivity to increased transmural pressure is affected - thus causing increases in pressure to imply progressive increases in ICP.

The measurement of local brain oxygenation using a Clark-type polarographic catheter implanted in the brain (PtiO<sub>2</sub>) is common practice in many critical neurological patient units, according to the latest guidelines published by the Brain Trauma Foundation.<sup>7</sup> Since 1999 in our hospital we have systematically monitored PtiO<sub>2</sub> together with ICP in all patients with severe CET. We have analyzed such monitorization in over 300 patients with severe CET, and always attempt to position the catheter in the apparently healthy area of the most damaged hemisphere in the case of evacuated expansive lesions; in the most affected hemisphere in the case of diffuse lesions; and in the right frontal lobe in the case of diffuse lesions without apparent differences in damage between the two hemispheres.<sup>31</sup>

Recent articles have described improved brain oxygenation (PtiO<sub>2</sub>) after cranial decompression.<sup>17,27,32-34</sup> In our experience with 28 patients,<sup>28</sup> PtiO<sub>2</sub> monitoring was carried out before and after DC. Of these subjects, 19 underwent PtiO<sub>2</sub> monitorization with the electrode placed in the apparently healthy area in the CAT scan of the most damaged hemisphere (i.e., the side of the craniotomy), while in two cases monitoring of the side opposite the hemicraniotomy was performed, and in the remaining three patients bilateral monitorization was carried out. After decompression, a significant increase in PtiO<sub>2</sub> was recorded ( $9.7 \pm 7.06$  mmHg) with the catheter placed in the apparently healthy area of the hemicraniotomy hemisphere versus the PtiO<sub>2</sub> recorded on the contralateral side, which even dropped to  $1.4 \pm 3.8$  mmHg on average ( $p < 0.001$ ). Three patients remained with initial PtiO<sub>2</sub> values in the range of brain ischemia and did not increase to the normal levels of PtiO<sub>2</sub> measured on the craniotomy side after DC. These patients all suffered brain death. Other studies,<sup>27,34,35</sup> have described multiple monitorization involving ICP, CPP, microdialysis and PtiO<sub>2</sub> simultaneously before and after DC - with improvements not only in ICP, CPP and PtiO<sub>2</sub>, but also in the parameters obtained at microdialysis, such as glucose concentration and the lactate / pyruvate ratio. In addition, while involving a limited number of patients ( $n = 5$ ), the study published by Reitmeier et al.<sup>27</sup> found that although ICP decreased in all subjects after DC, only those showing normalization of PtiO<sub>2</sub> and of the biochemical parameters as a result of microdialysis presented a favorable outcome. These studies do not specify the precise positioning of the PtiO<sub>2</sub> catheters or of microdialysis. At present, we are analyzing the changes in PtiO<sub>2</sub> before and after DC only when the catheter was positioned before and after DC on the craniotomy side in 33 patients, and the preliminary results presented at the XI Congress of the Euroacademy of Multidisciplinary Neurotraumatology ([www.emn2010.com](http://www.emn2010.com)) indicate that the patients showing a greater persistent PtiO<sub>2</sub>

increment above the brain hypoxia threshold (20 mmHg) and after DC showed an improved Glasgow Outcome Scale (GOS) score 6 months after trauma compared with those individuals with significantly lesser increments or with persistent values below the brain hypoxia threshold.

The indication of DC has always been centered on refractory ICH, independently of the presence or absence of brain hypoxia. However, once the brain pressure has been freed as a result of the craniotomy, and particularly after aperture of the dura mater, hyperemia develops in the areas affected before decompression and which have a strong oxygen demand. Furthermore, the blood vessels that were compressed now fill again, and dilatation occurs secondary to vessel paralysis as a result of the metabolic changes. This increase in regional brain blood volume may increase the edema and necrosis in the zone despite normal ICP values.

Such decompressive hyperemia was studied by Yamakami et al.<sup>36</sup> using SPECT technology. These authors found perfusion to increase in the decompressed brain region after a few minutes, reaching a maximum after one week; posteriorly, after approximately one month, perfusion normalized in coincidence with patient improvement. The authors added that hyperperfusion occurred particularly when DC was performed too late. In this same line, Yoshida et al.<sup>37</sup> reported that such hyperperfusion may protect the brain from hypoxemic cell damage, as demonstrated by improvement in brain lactate and potassium clearance via xenon (Xe) CT and spectroscopic magnetic resonance imaging. This in turn would result in a decrease in cytotoxic edema, thereby avoiding the drop in cerebral blood flow, which posteriorly would give rise to elevations in ICP.

Therefore, and independently of the reductions in ICP, the normalization of  $Pt_iO_2$  after DC described above could afford another beneficial effect in relation to patient outcome.

Then why not indicate DC in cases of brain hypoxia with moderately elevated ICP (20-25 mmHg) instead of exclusively waiting for ICH > 25-30 mmHg when the first level management options have been exhausted?

In our experience, DC could be a therapeutic option in the case of brain hypoxia ( $Pt_iO_2 < 10$  mmHg in the apparently healthy area on the CAT scan) with moderate intracranial hypertension, when CPP has been optimized, with hemoglobin levels > 11 g/dl, and once all possible systemic causes of cerebral hypoxia have been discarded (hypoxemia, hypotension, sepsis, hypocapnia, etc.).

Considering the above, we feel that  $Pt_iO_2$  monitorization in a healthy area of the most damaged cerebral hemisphere may be useful not only for indicating the timing of decompressive craniotomy, but also for evaluating its effectiveness.

## References

- Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, et al. Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg.* 1991;75 Suppl:S59-66.
- Clifton GL, Miller ER, Choi SC, et al. Fluid thresholds and outcome from severe head injury. *Crit Care Med.* 2002;30:739-45.
- Juul N, Morris GF, Marshall SB, Marshall LF. Intracranial hypertension and cerebral perfusion pressure: influence on neurological deterioration and outcome in severe head injury. The Executive Committee of the International Selfotel Trial. *J Neurosurg.* 2000;92:1-6.
- Marshall LF. Head injury: recent past, present, and future. *Neurosurgery.* 2000;47:546-61.
- Miller JD, Piper IR, Dearden NM. Management of intracranial hypertension in head injury: matching treatment with cause. *Acta Neurochir Suppl.* 1993;57:152-9.
- Lubillo S, Manzano JJ, Manzano JL, et al. Aspectos pronósticos y terapéuticos en el trauma craneal y torácico. *Med Intensiva.* 1983;7:33-42.
- Maas AI, Dearden M, Teasdale GM, et al. Guidelines for management of severe head injury in adults. European Brain Injury Consortium. *Acta Neurochir (Wien).* 1997;139:286-94.
- Bullock R, Chesnut RM, Clifton G, et al. Guidelines for management of severe head injury. Brain Trauma Foundation. *J Neurotrauma.* 2000;17:451-627.
- The Brain Trauma Foundation. The American Association of Neurological Surgeons. The Joint Section on Neurotrauma and Critical Care. Anesthetics. Analgesics and sedatives. *J Neurotrauma.* 2007;24(Suppl 1):S71-6.
- Roberts I. Barbiturates for traumatic brain injury. *Cochrane Database Syst Rev.* 2000;2:CD000033.
- Clifton GL, Miller ER, Choi SR, et al. Lack of effect of induction of hypothermia after acute brain injury. *N Engl J Med.* 2001;344:556-63.
- Polderman KH, Tjong Tjing JR, Peerdeman SM, Vandertop WP, Girbes AR. Effects of therapeutic hypothermia on intracranial pressure and outcome in patients with severe head injury. *Intensive Care Med.* 2002;28:1563-73.
- McIntyre LA, Fergusson DA, Herbert PC, et al. Prolonged therapeutic hypothermia after traumatic brain injury in adults. A systematic review. *JAMA.* 2003;289:2992-9.
- Albanese J, Leone M, Alliez JR, Kaya JM, Antonini F, Alliez B, et al. Decompressive craniectomy for severe traumatic brain injury: evaluation of the effects at one year. *Crit Care Med.* 2003;31:2535-8.
- Aarabi B, Hesdorffer DC, Ahn DS, et al. Outcome following decompressive craniectomy for malignant brain swelling due to severe head injury. *J Neurosurg.* 2006;104:469-79.
- Timofeev I, Kirkpatrick PJ, Corteen E, et al. Decompressive craniectomy in traumatic brain injury: outcome following protocol-driven therapy. *Acta Neurochir Suppl.* 2006;96:11-6.
- Meier U, Lemcke J, Reyer T, Grawe A. Decompressive craniectomy for severe head injury patients with major extracranial injuries. *Acta Neurochir Suppl.* 2006;96:373-6.
- Gaab MR, Rittierodt M, Lorenz M, Heissler HE. Traumatic brain swelling and operative decompression: a prospective investigation. *Acta Neurochir Suppl.* 1990;51:326-8.
- Guerra WK, Gaab MR, Dietz H, Mueller JU, Piek J, Fritsch MJ. Surgical decompression for traumatic brain swelling: indications and results. *J Neurosurg.* 1999;51:187-96.
- Coplin WM, Cullen NK, Policherla PN, Vinas FC, Wilseck JM, Zafonte RD, et al. Safety and feasibility of craniectomy with duraplasty as the initial surgical intervention for severe traumatic brain injury. *J Trauma.* 2001;50:1050-9.
- De Luca GP, Volpin L, Fornezza U, Cervellini P, Zanusso M, Casentini L, et al. The role of decompressive craniectomy in the treatment of uncontrollable post-traumatic intracranial hypertension. *Acta Neurochir Suppl.* 2000;76:401-4.
- Ransohoff J, Benjamin MV, Gage EL, Epstein F. Hemicraniectomy in the management of acute subdural hematoma. *J Neurosurg.* 1971;34:70-6.
- Cooper PR, Rovit RL, Ransohoff J. Hemicraniectomy in the treatment of acute subdural hematoma: a re-appraisal. *Surg Neurol.* 1976;5:25-8.

24. Kjellberg RN, Prieto A. Bifrontal decompressive craniotomy for massive cerebral edema. *J Neurosurg.* 1971;34:488-93.
25. Lubillo S, González B, Peña V, et al. Craniectomía descompresiva en las lesiones expansivas intracraneales de origen traumático [abstract]. *Med Intensiva.* 2004;28(Suppl 1):1-90.
26. Sahuquillo J, Arian F. Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. *Cochrane Database Syst Rev.* 2006;25:CD003983.
27. Reithmeier T, Löhr M, Pakos G, et al. Relevance of ICP and PtiO<sub>2</sub> for indication and timing of decompressive craniectomy in patients with malignant brain edema. *Acta Neurochir.* 2005;147:947-52.
28. Lubillo S, Blanco J, López P, et al. Papel de la craniectomía descompresiva en el enfermo neurocrítico. *Med Intensiva.* 2009;33:72-81.
29. Sahuquillo J, Biestro A, Mena MP, et al. First tier measures in the treatment of intracranial hypertension in the patient with severe craniocerebral trauma. Proposal and justification of a protocol. *Neurocirugía (Astur).* 2002;13:78-100.
30. Timofeev I, Czosnika M, Nortje J, et al. Effect of the decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury. *J Neurosurg.* 2008;108:66-73.
31. Lubillo S, Peña V, Solsona N, et al. Monitorización de la SjO<sub>2</sub> y PtiO<sub>2</sub> en el paciente neurocrítico. In: Net A, Marruecos L, editors. *Paciente neurocrítico.* Barcelona: Ars Médica; 2006. p. 57-70.
32. Jaeger M, Soehle M, Meixensberger J. Improvement of brain tissue oxygen and intracranial pressure during an surgical decompression for diffuse brain edema and space occupying infarction. *Acta Neurochir Suppl.* 2005;95:117-8.
33. Stiefel MF, Heuer GG, Smith MJ, et al. Cerebral oxygenation following decompressive hemicraniectomy for the treatment of refractory intracranial hypertension. *J Neurosurg.* 2004;101:241-7.
34. Boret H, Fesselet J, Meaudre E, et al. Cerebral microdialysis and PtiO<sub>2</sub> for neuro-monitoring before decompressive craniectomy. *Acta Anaesthesiol Scand.* 2006;50:252-4.
35. Ho CL, Wang CM, Lee KK, Ng I, Ang BT. Cerebral oxygenation, vascular reactivity and neurochemistry following decompressive craniectomy for severe traumatic brain injury. *J Neurosurg.* 2008;108:943-9.
36. YamaKami I, Yamamura A. Effects of decompressive craniectomy on regional cerebral blood flow in severe head injuries patients. *Neurol Med Chir (Tokio).* 1993;33:13-8.
37. Yoshida K, Furuse M, Isawa A, et al. Dynamics of cerebral blood flow and metabolism in patients with cranioplastyas evaluated by Xe CT and magnetic resonance spectroscopy. *J Neurosurg Psychiatry.* 1996;61:166-71.