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Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with posttraumatic brain swelling treated by surgical decompression

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Object. The use of decompressive craniectomy has experienced a revival in the previous decade, although its actual benefit on patients' neurological outcome remains the subject of debate. A better understanding of the intracranial pressure dynamics, as well as of the metabolic and hemodynamic brain processes, may be useful in assessing the effect of this surgery on the pathophysiology of the swollen brain. The aim of this study was to use transcranial Doppler (TCD) ultrasonography to examine the hemodynamic changes in the brain after decompressive craniectomy in patients with head injury, in addition to examining the relationship between such hemodynamic changes and the patient's neurological outcome.

Methods. Nineteen patients presenting with traumatic brain swelling and cerebral herniation syndrome who had undergone decompressive craniectomy with dural expansion were studied prospectively. The TCD ultrasonography measurements were performed bilaterally in both the middle cerebral artery (MCA) and in the distal portion of the cervical internal carotid artery (ICA) immediately prior to and after surgical decompression.

After surgery, the mean blood flow velocity (BFV) rose to 175 $\pm$ 209% of preoperative values in the MCA of the operated side, while rising to 132 $\pm$ 183% in the contralateral side; the difference between the mean BFV increase in in the MCA of both the decompressed and the opposite side reached statistical significance ($p < 0.05$). The mean BFV of the extracranial ICA increased to 91 $\pm$ 119% in the surgical side and 45 $\pm$ 60% in the opposite side. Conversely, the MCA pulsatility index (PI) values decreased, on average, to 33 $\pm$ 36% of the preoperative value in the operated side and to 30 $\pm$ 34% on the opposite side; the MCA PI value reductions were significantly greater in the decompressed side when compared with the contralateral side ($p < 0.05$). The PI of the extracranial ICA reduced, on average, to 37 $\pm$ 23% of the initial values in the operated side and to 24 $\pm$ 34%, contralaterally. No correlation was verified between the neurological outcome and cerebral hemodynamic changes seen on TCD ultrasonography.

Conclusions. Decompressive craniectomy results in a significant elevation of cerebral BFV in most patients with traumatic brain swelling and transtentorial herniation syndrome. The increase in cerebral BFV may also occur in the side opposite the decompressed hemisphere; the cerebral BFV increase is significantly greater in the operated hemisphere than contralaterally. Concomitantly, PI values decrease significantly postoperatively, mainly in the decompressed cerebral hemisphere, indicating reduction in cerebrovascular resistance.

Key Words • brain swelling • head injury • intracranial hypertension • decompressive craniectomy • transcranial Doppler ultrasonography • cerebral hemodynamics

Posttraumatic brain swelling is a frequent cause of intracranial hypertension and of brain herniation syndrome in head-injured patients. Despite modern advances in neurosurgery and intensive care medicine over the previous decades, the mortality rate for these patients remains high,[31] reaching values of 87.2%.[30] Decompressive craniectomy is one of the therapeutic options for malignant brain swelling associated with instances of high ICP or brain herniation refractory to conventional conservative management, and may be indicated in extreme cases.[4,7,13] This surgical modality has experienced a revival since the previous decade,[2,6,9,14,17,24,34,36,39,43,44,49,52,55] although its actual benefit on patient neurological outcome remains unclear.

To date, few studies have addressed the effect of decompressive craniectomy on intracranial hemodynamics.[15,16,40,41,54] To the best of our knowledge, no authors have demonstrated the effect of decompressive craniectomy on cerebral BFV by means of TCD ultrasonography in patients with head injury.

The purpose of this study was to use TCD ultrasonography to investigate prospectively the cerebral hemodynamic changes in patients with severe traumatic brain swelling

Abbreviations used in this paper: BFV = blood flow velocity; CBF = cerebral blood flow; CPP = cerebral perfusion pressure; CT = computerized tomography; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICA = internal carotid artery; ICP = intracranial pressure; MABP = mean arterial blood pressure; MCA = middle cerebral artery; PI = pulsatility index; SDH = subdural hematoma; TCD = transcranial Doppler.

TABLE 1

*Demographic, clinical, and imaging characteristics of patients with severe, predominantly hemispheric swelling who underwent decompressive craniectomy**

Case No.	Age (yrs), Sex	Cause of Injury	Traumatic Lesions Operated at Admission	Interval Btwn Admission & Craniectomy	Preop GCS Score	Preop Pupils	Midline Shift on CT Scan (mm)	Side of Craniectomy	6-Mo GOS Score
1	63, F	VPA	none	6 hrs	6	unequal	12	rt	1
2	18, M	MVA	none	4 hrs	6	unequal	16	rt	1
3	28, M	VPA	none	6 days	6	unequal	9	lt	5
4	39, F	MVA	none	7 hrs	6	unequal	11	rt	3
5	28, M	MVA	none	3 hrs	7	unequal	15	rt	4
6	25, M	VPA	none	4 days	6	unequal	12	rt	3
7	19, F	MVA	ICH	24 hrs	6	unequal	NM	rt	4
8	28, F	VPA	none	2 hrs	6	unreact	9	lt	4
9	22, M	MVA	none	2 hrs	6	unequal	14	rt	2
10	25, F	MVA	none	6 days	5	unequal	8	rt	1
11	30, M	VPA	ICH	2 days	6	unequal	17	rt	1
12	27, M	VPA	SDH	8 days	6	unequal	12	rt	5
13	17, M	MVA	SDH	3 days	10	unequal	5	lt	4
14	39, M	assault	EDH	3 days	12	unequal	NM	rt	4
15	61, F	VPA	ICH, SDH	3 days	9	unequal	11	lt	2
16	51, M	fall	ICH	4 days	9	unequal	19	lt	3
17	43, M	VPA	SDH	3 days	6	unequal	20	lt	1
18	46, M	MVA	SDH	9 days	4	unequal	10	rt	2
19	23, M	VPA	ICH	24 hrs	6	unreact	15	lt	3

* EDH = epidural hematoma; ICH = traumatic contusional intracerebral hemorrhage; MVA = motor vehicle accident; unequal = at least one side reactive; unreact = unreactive bilaterally; NM = not measured; VPA = vehicle–pedestrian accident.

who underwent decompressive craniectomy with the dural expansion technique. Furthermore, we attempted to verify the relationship between cerebral hemodynamic changes identified via TCD ultrasonography and the patient's long-term clinical outcome.

Clinical Material and Methods

This study was approved by the Research Ethics Committee of the University of São Paulo Medical School, São Paulo, Brazil. The patients eligible for this study were those presenting with severe posttraumatic brain swelling for whom decompressive craniectomy with dural augmentation was indicated and in whom TCD examinations had been performed immediately pre- and postoperatively. Patients excluded from this protocol were those who had undergone decompressive craniectomies and sustained penetrating head injuries or nontraumatic brain swelling, and those for whom it was impossible to compare pre- and postoperative TCD measurements due to significant variations in physiological factors that could affect intracranial BFV such as hematocrit, arterial blood carbon dioxide and oxygenation, body temperature, and systemic arterial blood pressure.

Patient Population

In our prospective study we included 19 patients with the diagnosis of severe posttraumatic brain swelling treated by decompressive craniectomy at our institution between January 1999 and September 2002. The ages of the 13 male and six female patients ranged from 17 to 63 years, with a mean age of 33 years (standard deviation 14 years). Demographic, clinical, and imaging features were collected for every patient and have been summarized in Table 1. Two patient subgroups were noted. The first consisted of nine patients without focal lesions in whom massive posttraumatic brain swelling associated with clinical deterioration had required a decompressive craniectomy. The second included 10 patients who presented with intracranial space-occupying hematomas (cerebral parenchymal hemorrhages and epidural or SDHs) that had been operated on previously. Postoperative severe brain edema developed later, however, leading to clinical deterioration and the need for a decompressive craniectomy.

General Management Protocol

Patients were treated in a standard fashion according to the guidelines of the American College of Surgeons (Advanced Trauma Life Support) and of the Brazilian Society of Neurosurgery,[13] the latter being based on the guidelines of the American Association of Neurological Surgeons.[4] After the stabilization of respiratory and systemic hemodynamic parameters, all patients who had developed neurological deterioration and signs of transtentorial herniation underwent urgent CT scans of the brain; these disclosed predominantly unilateral brain swelling. Monitoring of ICP was not considered in this protocol.

Indications for Surgery

Surgical decompression was indicated in cases of predominantly unilateral diffuse brain swelling associated with mass effect, a midline shift greater than 5 mm, and/or obliteration of perimesencephalic cisterns noted on CT scans, along with correlating neurological deterioration such as worsening of GCS score, and/or dilation of pupils that were unresponsive to light. Patients with a seriously injured brainstem, as suggested by an initial and persisting GCS score of 3 and/or bilaterally fixed and dilated pupils, did not undergo surgery.

Surgical Procedure

Large hemicraniectomies with dural augmentation over the swollen cerebral hemisphere were performed. The surgical approach consisted of a skin incision that began anterior to the external auditory canal and curved occipitally to the midline and up to the hairline frontomedially so that a large scalp flap shaped like a reversed question mark could be made to expose the cranial convexity unilaterally as much as possible. A large frontotemporoparietooccipital craniectomy included, inferiorly, the floor of the middle fossa to relieve the pressure from the herniating mediobasal temporal lobe, and superiorly to midline to avoid the compression of bridging veins.[53] Care was taken to avoid the sigmoid and superior sagittal sinuses to prevent the possibility of sinus thrombosis. The objective of performing large hemicraniectomies was to reduce the risk of small craniectomy–related brain lesions.[51] The dura mater was opened, allowing its expansion. Subsequently, the dura defect was covered with a dural graft of periosteum from the bone flap or the temporal muscle fascia. The graft was sutured in such a way as to prevent cerebral hemisphere compression and cerebrospinal fluid leakage. Brain tissue resections were not performed.

Transcranial Doppler Ultrasonography Studies

All TCD measurements presented in this study were done by the senior author (E.B.S.S.). The neurosurgical team was oriented to maintain their routine practice, without waiting for the arrival of the senior author to avoid any delay in the surgical treatment. In practice, preoperative TCD measurements were obtained while the patient waited to go into the operating room (one case) or while the anesthesiologist prepared the patient in the operating room (18 cases); postoperative TCD examinations were performed soon after the completion of wound closure and dressing. In seven patients, a third TCD examination was performed within 48 to 72 hours after surgery.

The MCA and the distal portion of the extracranial ICA were insonated bilaterally using a portable 2-MHz pulsed TCD device (Pioneer TC 2020 EME; Nicolet Biomedical, Madison, WI), through temporal and posterior submandibular windows, respectively, as previously described by Aaslid, et al.[1] When possible the anterior and posterior cerebral arteries, the carotid siphon, and the vertebral and basilar arteries were insonated via temporal, orbital, and suboccipital/foraminal windows, respectively.

The blood flow parameters analyzed with TCD ultrasonography were the BFV (mean, end diastolic, and peak systolic velocities), and the PI, which was calculated by dividing the difference between peak systolic and end diastolic BFV by the mean BFV.

During the TCD examinations, physiological data such as MABP, pulse oximetry, expired CO_2 tension, and body temperature were documented, and blood samples were analyzed for blood gases and hematocrit levels.

Data Collection

Patient data noted included: age, sex, date of the accident, time intervals between the accident and hospital admission, and between hospital admission and decompressive craniectomy, mechanism of injury, neurological status (GCS score and pupil response) at hospital admission and prior to surgical decompression, midline shift and associated intracranial lesions noted on brain CT scan, duration of postoperative coma, length of hospital stay, and outcome. The patient's neurological outcome was determined using the GOS score at 6 months after injury.[20] Patients with severe disability (GOS Score 3), patients in a vegetative state (GOS Score 2), or those who died (GOS Score 1) were considered as presenting poor outcome; patients with good recovery (GOS Score 5) or moderate disability (GOS Score 4) were defined as having favorable outcomes.

TABLE 2

*Physiological parameters immediately before and after decompressive craniectomy**

Physiological Parameter	Before Brain Decompression	After Brain Decompression	p Value
MABP	89 ± 16	77 ± 11	0.002
$PaCO_2$	33 ± 5	34 ± 5	0.587
Ht (%)	32 ± 7	31 ± 6	0.289

* Ht = hematocrit.

Statistical Analysis

Results are reported as the means ± standard deviations. Statistical comparisons used the paired Student t-test, the Mann–Whitney U-test, the Wilcoxon rank-sum test, and the Fisher exact test. Spearman correlation coefficients were calculated when appropriate. The level of statistical significance was set at a probability value less than or equal to 0.05.

Results

Physiological Parameters Prior to and Following Brain Decompression

The pre- and postoperative physiological parameters, such as blood pressure, blood CO_2 concentration, and hematocrit, are presented in Table 2. A decrease in MABP was observed postoperatively ($p < 0.05$).

Mean BFV Before and After Decompressive Craniectomy

After surgery, the mean BFV increased from 53 ± 38 to 94 ± 33 cm/second in the MCA of the decompressed cerebral hemisphere ($p < 0.001$) and from 29 ± 15 to 44 ± 9 cm/second in the ipsilateral extracranial ICA ($p < 0.008$). On the opposite side, the mean BFV increased from 51 ± 26 to 76 ± 16 cm/second ($p < 0.001$) and from 35 ± 11 to 44 ± 10 cm/second ($p < 0.015$) in the MCA and extracranial ICA, respectively (Table 3).

Postoperatively, the mean BFV rose to 175 ± 209% of preoperative values in the MCA of the decompressed cerebral hemisphere, while rising 132 ± 183% in the contralateral MCA. The difference between mean BFV increase in the MCA seen in both the decompressed and the opposite side reached statistical significance ($p < 0.05$). After the surgery, the BFV of the extracranial ICA increased on average 91 ± 119% in the treated side and 45 ± 60% on the opposite side (Table 4).

TABLE 3

Preoperative and postoperative TCD blood flow velocity of patients with brain swelling who underwent surgical decompression

Artery	Side	No. of Patients	Preop BFV (cm/sec)	Postop BFV (cm/sec)	p Value
MCA	decompressed	19	53 ± 38	94 ± 33	<0.001
cervical ICA	decompressed	16	29 ± 15	44 ± 9	<0.008
MCA	opposite	17	51 ± 26	76 ± 16	<0.001
cervical ICA	opposite	15	35 ± 11	44 ± 10	<0.015

TABLE 4

*Percentage change in TCD hemodynamic parameters of 19 patients before and after surgical decompression**

TCD Parameter	Artery	Operated Side	Opposite Side	p Value
flow velocity	MCA	175 ± 209%	132 ± 183%	<0.05
flow velocity	cervical ICA	91 ± 119%	44 ± 59%	NS
PI	MCA	−33 ± 36 %	−30 ± 34%	NS
PI	cervical ICA	−37 ± 23%	−24 ± 34%	NS

* NS = not statistically significant.

Preoperative and Postoperative PI

The PI values for the MCA in the decompressed cerebral hemisphere diminished from 1.85 ± 1.56 to 0.81 ± 0.18, before and after surgery, respectively ($p < 0.001$), whereas the PI values for the ipsilateral extracranial ICA decreased from 1.48 ± 0.94 to 0.75 ± 0.27 ($p < 0.001$). On the opposite side, the pre- and postoperative PI values for the MCA decreased from 1.73 ± 1.36 to 0.86 ± 0.22, respectively ($p = 0.001$), and the PI values for the ipsilateral extracranial ICA decreased from 1.29 ± 0.92 to 0.72 ± 0.15 ($p < 0.05$; Table 5).

After decompressive craniectomy, the PI values for the MCA decreased, on average, to 33 ± 36% of the presurgical value in the treated side and to 30 ± 34% on the opposite side. On the other hand, the mean PI values for the extracranial ICA reduced to 37 ± 23% of the initial values in the treated side, and to 24 ± 34% on the opposite side (Table 5).

Comparison Among Cerebral Hemodynamic Changes

The hemodynamic parameters studied using TCD ultrasonography were BFV and PI. No correlation was found between cerebral hemodynamic changes revealed through TCD ultrasonography that were related to surgical decompression and other variables such as preoperative GCS score, the presence of a midline shift on a preoperative brain CT scan, neurological recovery at 6 months postinjury (according to the GOS score), or neurological recovery based on favorable (good recovery or moderate disability) or unfavorable neurological outcome (severe disability, vegetative state, or death).

Discussion

Since ancient times, many attempts have been made to treat massive posttraumatic brain swelling, a condition that can cause refractory intracranial hypertension and brain herniation. When nonsurgical management fails to control ICP and cerebral herniation, decompressive craniectomy may be indicated as a last resort.[4,7,13] Despite many publications on the subject,[2,6,9,12,14,17,24,34,36,39,43,44,49,52,55] the actual benefit of this treatment modality on neurological outcome in patients remains unknown. The hemodynamic changes in the brain in response to decompressive craniectomy have not been systematically studied in patients with traumatic brain swelling. To the best of our knowledge, this is the first time that an effort has been made to investigate the effects of surgical decompression on hemodynamic variables in such patients prospectively through TCD ultrasonography. Although prospective, randomized, well-controlled trials are necessary to define the true effectiveness of this surgery, we believe that in addition to the neurological outcome parameters, a better comprehension of ICP dynamics as well as metabolic and circulatory brain processes may be useful in assessing the effects of decompressive surgery on the brain.

The results of this investigation reveal that surgical decompression for most patients with posttraumatic brain swelling and evidence of brain herniation syndrome is associated with a significant increase in cerebral BFV, not only in the decompressed cerebral hemisphere, but also in the opposite side. Moreover, the increase in cerebral BFV is significantly greater in the operated hemisphere than in the contralateral side. At the same time, surgical decompression is also associated with a significant decrease in postoperative PI values in both cerebral hemisphere arteries.

In interpreting these results, some principles regarding TCD ultrasonography should first be discussed. One point that must be constantly remembered is that this modality measures the velocity of blood flowing through the vessel (expressed in centimeters per second) and not the blood flow rate (expressed in milliliters per minute) or tissue perfusion (expressed in milliliters per 100 grams of tissue per minute).[32,38] Therefore, TCD ultrasonography can only be used to assess relative changes in blood flow in an intracranial artery over a period, providing a qualitative rather than a quantitative analysis of CBF. Because the diameter of the large basal brain arteries remain constant during decompressive craniectomy, any variation in BFV correlates well with change in CBF in the territory of the insonated vessel.[23,32,38] Taking all of this into account, it is reasonable to consider that the increase in cerebral BFV, as demonstrated by this study, may correspond to CBF elevation following surgical decompression. This possibility may be reinforced by the simultaneous finding of BFV increase in the distal segment of the extracranial ICAs of our patients, because the BFV in the extracranial ICA may be considered an index of CBF; the extracranial ICA diameter is believed to be constant over time, so BFV changes in this vessel result from changes in CBF rather than from changes in its arterial diameter.[33]

Experimental and clinical research concerning the effects of surgical decompression on intracranial blood circulation in traumatic brain swelling are currently lacking in the literature. Rinaldi and colleagues[40] investigated the hemodynamic effects of decompression with craniectomy and dura opening in rabbits with brain edema due to cold-induced lesions, demonstrating that in noncraniectomized animals, ICP increased progressively up to 40 to 60 mm Hg within 2 to 3 hours. This elevation in ICP occurred at the same time

as a reduction in the diastolic BFV of the cervical ICA, and an increase in the ratio between the systolic and diastolic values. The diastolic BFV later reached a value of zero, thus indicating a remarkable rise in cerebrovascular resistance. The electrocortical activity evolved to electrocortical silence. In animals who underwent craniectomy, decompression was immediately followed by a dramatic decrease in ICP to basal levels and a simultaneous increase in the BFV of the cervical ICA to normal values, thus indicating a decrease in cerebrovascular resistance. A partial electrocortical activity recovery was later observed. Following this study, Yamakami and Yamaura[54] used single-photon emission computerized tomography with 99 m technetium-hexamethyl-propyleneamine oxime to investigate regional CBF. Five patients with severe head injury (GCS score < 8) underwent large unilateral decompressive craniectomy for hemispheric brain swelling. The authors observed that a focal CBF increase occurred in the decompressed brain within 24 hours after surgery. This hyperperfusion region exhibited an increase in extent and intensity within the first week after surgery, and its attenuation and disappearance was observed 1 month postoperatively. Yamakami and Yamaura concluded that decompressive craniectomy might have caused a focal CBF increase in the decompressed brain which was able to protect the traumatized brain tissue from ischemia caused by intracranial hypertension. The study by Rinaldi, et al.,[40] disclosed indirect evidence of intracranial hemodynamic improvement in rabbits because it demonstrated an elevation of BFV in the cervical ICA after surgical decompression, whereas our work revealed an increase in BFV directly in human intracranial arteries. Yamakami and Yamaura[54] demonstrated blood hyperperfusion in decompressed brain areas using single photon emission CT, which may support our data in terms of the CBF increase following this operation.

The use of decompressive craniectomy in the treatment of refractory intracranial hypertension may be associated with a significant improvement in levels of brain tissue oxygenation. Cerebral oxygenation increased an average of 114% after decompressive surgery performed in patients presenting with severe brain injury and intracranial hypertension, as demonstrated by Stiefel and coworkers.[48] These data can be explained by our results, because the increase in cerebral BFV may be one of the mechanisms that allows an increase in cerebral oxygenation. In a recent study on the measurement of ICP, CPP, jugular venous oxygen saturation, and laser Doppler flowmetry in patients with traumatic acute SDHs, it was shown that ICP decreased and CPP increased significantly during the removal of the bone flap, and before opening of the dura mater, resulting in a marked increase in jugular venous oxygen saturation and laser Doppler flow.[50] When the dura was opened, further significant decreases in ICP occurred. Although this work was done on patients with acute SDHs, we agree with the author's belief that these findings might explain the effect of decompressive craniectomy in patients who suffer from posttraumatic increased ICP that is refractory to medical therapy. Our data are in line with the results of this work, because they indicate an increase in CBF after surgical decompression. It is noteworthy that the increase in CBF after decompressive craniectomy may be explained by the well-demonstrated effect of this surgical technique in reducing ICP.[39,43,52,55]

TABLE 5

Preoperative and postoperative TCD PI of patients who underwent surgical decompression

Artery	Side	No. of Patients	Preop PI	Postop PI	p Value
MCA	decompressed	19	1.85 ± 1.56	0.81 ± 0.18	0.001
cervical ICA	decompressed	16	1.48 ± 0.94	0.75 ± 0.27	<0.001
MCA	opposite	17	1.73 ± 1.36	0.86 ± 0.22	0.001
cervical ICA	opposite	15	1.29 ± 0.92	0.72 ± 0.15	0.013

It has been demonstrated that the Doppler waveforms, as obtained by TCD ultrasonography, are affected by both increases in ICP or decreases in CPP.[3,5,18] As ICP increases, diastolic BFV decreases, mainly at the end of the diastole, possibly as a consequence of increased resistance to the CBF. This decrease may reduce the forward flow during diastole. A decrease in diastolic BFV results in an increase in the PI, which is linearly or exponentially correlated with ICP, so that measures of PI can be used for noninvasive estimations of ICP in patients with head injury.[42] As noted in our results, a statistically significant reduction in PI occurred in arteries of both cerebral hemispheres, indicating a postoperative ICP decrease in different intracranial compartments by means of unilateral surgical decompression. Interestingly, both the increase in cerebral BFV and the decrease in PI were greater in the decompressed cerebral hemisphere (although only the former reached statistical significance), indicating more effective ICP reduction in the surgical side, at least at an early postoperative phase (among other possibilities). In cases involving high preoperative PI values, the significant reduction in postoperative PI values may be evidence of improvement in the intracranial hemodynamics, since it may translate to an effective decrease of raised ICP. This finding is important because persistently high ICP has been demonstrated to be one of the main factors related to an unfavorable outcome.[21] On the other hand, the increase in cerebral BFV does not always signify intracranial hemodynamic improvement, because it may also be caused by cerebral vasospasm or hyperemia;[19,29,33] these conditions may be associated with a poor prognosis in head-injured patients. The former, if hemodynamically significant, results in critically low CBF,[28] while the latter, when at a critical level, is related to impairment of either cerebrovascular reactivity or cerebral autoregulation, with uncoupling between cerebral blood flow and metabolism.[8,22,27,37] The increase in cerebral BFV reflects intracranial hemodynamic improvement if associated with elevation of CBF in cases of oligemia within ischemic range. In any event, the increase in cerebral BFV is of clinical interest, irrespective of the causes.

Morgalla, et al.,[35] reported on two children with severe head injury treated aggressively by repeated decompressive craniectomy according to the results of neurological monitoring. In both cases, surgical decompression was performed when simultaneously CPP dropped to less than 60 mm Hg, the ICP became uncontrollable by conservative measures, and the end diastolic BFV decreased to zero on TCD ultrasound images. After surgery, cerebral hemodynamic improvement was observed on TCD ultrasound images. The authors suggested that these criteria from neurological monitoring could be used as guidelines for

undertaking brain decompression in patients with uncontrollably raised ICP. In our series, the TCD results were not a deciding factor for surgical treatment. Our patients underwent surgery on the basis of clinical and brain CT parameters compatible with cerebral herniation syndrome. The majority of them did not present ceased cerebral end diastolic BFV, but rather presented cerebral herniation syndrome. Hence, we were afraid of waiting for the cerebral diastolic BFV to cease. At this juncture, CPP is certainly quite impaired and the indication for surgical decompression may be too late for some patients.

Experimental hemispheric stroke models in rats have revealed that decompressive craniectomy improves brain tissue perfusion through leptomeningeal collateral vessels, thereby optimizing perfusion of the penumbra area and subsequently decreasing both the infarct size and the neurological deficit.[15,16] It seems that decompressive craniectomy may increase brain perfusion and as a result protect the penumbra area in stroke patients. We are sure that these effects also occur in patients with head injuries. Although pathophysiologically the massive hemispheric swelling caused by MCA or ICA occlusion differs from that caused by brain trauma, the mechanisms leading to increased brain tissue perfusion appear to be related to decreases in ICP because of the surgical decompression in both situations.

Few studies have analyzed the hemodynamic and metabolic effects of surgical decompression in the normal brain. Interestingly, by using positron emission tomography, Schaller and colleagues[41] demonstrated for the first time that decompressive hemicraniectomy in the cat decreases CBF, and, to a lesser extent, cerebral metabolic rates of oxygen and glucose, 2 hours after craniectomy in normal brain tissue, and that the effect lasts for at least 1 day. According to these authors, it might be concluded that in acute pathophysiological conditions, decompressive hemicraniectomy results in additional perfusion and to a lesser degree, metabolic disturbances. These apparently unexpected findings require further investigation.

With regard to CBF management in clinical neurosurgery, the current therapeutic strategy in patients with severe head injury is to avoid states of severe cerebral hyperemia and oligemia.[32,46] The former can induce an increase in vasogenic edema and the risk of intracerebral bleeding, whereas the latter can provoke cerebral ischemia and infarction. Both states can lead to a worsening of brain swelling and an increase in ICP. Therefore, performing decompressive craniectomy as the sole treatment is insufficient. Even during postoperative phases, all efforts must be made to maintain adequate brain hemodynamics,[32,46] preferably coupled with brain metabolism[7,8] as well as to promoting pharmacological brain protection and regeneration so that some degree of brain recovery can be possible. For the future, we hope that routine TCD monitoring of both cerebral autoregulation and cerebral vasomotor reactivity may help in offering adequate intracranial hemodynamics, and consequently, adequate CBF to brain-injured patients.[10,11,25,26,28,45,47]

Recent publications have already demonstrated the association of cerebral vasoreactivity impairment with poor neurological outcome.[10,27] In addition, elevated ICP appears to affect all components of cerebral vasoreactivity, such as carbon dioxide reactivity, pressure autoregulation, and vascular reactivity to pharmacological metabolic suppression.[27] There is also work indicating that intracranial hypertension may disturb cerebral autoregulation; but, by the same token, disturbed cerebral autoregulation may aggravate intracranial hypertension.[11] Bearing these aspects in mind, the idea of reducing ICP by decompressive craniectomy becomes quite attractive in that patients with refractory intracranial hypertension.

Our preliminary results failed to demonstrate a correlation between cerebral hemodynamic changes noted on TCD ultrasonography that were attributable to surgical decompression, and between variables such as neurological outcome at 6 months postinjury according to GOS scores,[20] and assessments of neurological recovery based on favorable (good recovery or moderate disability) or unfavorable neurological outcome (severe disability, vegetative state, or death), although that does not necessarily mean that those correlations cannot exist. We are aware of the limitations of the work on the subject in question, which include the complexity of the brain's hemodynamic and metabolic phenomena, the small patient sample, the heterogeneous characteristics of the patient population, the multiplicity of prognostic factors involved in traumatic brain injury, and the difficulties of acquiring an adequate control group. Future studies concerning the influence on the patient's neurological outcome of the pre- and postoperative cerebrovascular reactivity status as well as the degree of the pre- and postoperative cerebral autoregulation impairment may shed further light on some of the roles played by decompressive craniectomy.

It is well known that various factors can affect BFV, such as hematocrit, blood gases, body temperature, blood pressure, cerebral metabolism, and anesthetic agents.[32,38] One of the main limitations of our study was that we were not able to control parameters related to cerebral metabolism and anesthetic agents. The physiological factors recorded in this protocol did not change significantly before and after surgery at the time of TCD examinations, except for the level of the MABP, which was lower, postoperatively, in relation to the preoperative values. Therefore, we believe that the changes in cerebral BFV observed in this study can be attributed to surgical decompression of the swollen brain.

Conclusions

In conclusion, our study showed that decompressive craniectomy with dural expansion results in an elevation of cerebral BFV in patients with massive posttraumatic cerebral swelling. This increase can occur not only in the decompressed hemisphere, but also in that of the opposite side, and it is significantly greater in the operated hemisphere than in that of the contralateral side. At the same time, PI values decrease significantly in the postoperative phase, chiefly in the decompressed cerebral hemisphere, suggesting reduction in ICP. It is possible that this surgery can be beneficial in patients with a viable brain (in terms of functional recovery) that has reached CBF thresholds for ischemia. A better understanding of the effect of this surgery on the brain's pathophysiology may make early decision making and proper patient selection possible, thereby achieving more favorable clinical outcomes. This understanding may allow the neurosurgeon to contribute more to the survival and better quality of life for these patients. Further studies must address these questions in a bid to cast light on the controversies surrounding this type of surgery.

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