The influence of cranioplasty on postural blood flow regulation, cerebrovascular reserve capacity, and cerebral glucose metabolism

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The indications for cranioplasty after decompressive craniectomy are cosmetic repair and, mainly, restoration of cerebral protection. Although neurological improvement after cranioplasty is repeatedly noted, the reasons for this still remain unclear. Few observations concerning the impact of CSF hydrodynamic and/or atmospheric pressure were published during the last decades. Relevant data concerning the cerebrovascular reserve capacity and cerebral glucose metabolism before and after cranioplasty have been lacking until now. To gain further insight, the present study was undertaken to investigate the impact of cranioplasty on indices of cerebral blood flow regulation and metabolism.

Thirteen patients in whom extensive craniectomies had been performed underwent a meticulous study of blood flow velocities in the middle cerebral artery (MCA) and extracranial internal carotid artery (ICA), as assessed by transcranial Doppler (TCD) ultrasonography during postural maneuvers (supine and sitting positions) and during stimulation with 1 g of acetazolamide for the interpretation of cerebrovascular reserve (CVR) capacity. Twelve patients underwent 18-fluorodesoxyglucose positron emission tomography. These measurements were made before and 7 days after cranioplasty.

Cranioplasty improved preoperative differences in MCA blood flow velocities when comparing the injured with the noninjured hemisphere. Similarly, cranioplasty resolved decreases in extracranial ICA blood flow in the injured hemisphere that were induced by postural changes, which was a constant finding prior to this procedure. More strikingly, however, the CVR capacity, which was severely impaired in both hemispheres, significantly increased after the procedure. Metabolic deficits, which were observed in the injured as compared with the noninjured hemisphere, were found to improve after reimplantation of the skull bone flap.

Cranioplasty appears to affect postural blood flow regulation, CVR capacity, and cerebral glucose metabolism markedly. Thus, early cranioplasty is warranted to facilitate rehabilitation in patients after decompressive craniectomy.

KEY WORDS • cranioplasty • cerebrovascular reserve capacity • postural blood flow regulation • cerebral glucose metabolism • “syndrome of the trephined” • “syndrome of the sinking bone flap”

Cushing was the first to offer a systematic description of decompressive craniectomy for relief of intracranial pressure, and since that time, surgical decompression has been advocated as a treatment for severe brain edema associated with high intracranial pressure. Some authors have reported that decompressive craniectomy reduces the risk of death in patients who sustain severe cerebral edema after head injury. Recently we observed an increasing number of patients who underwent a replacement of the bone flap. The indications for repair of large cranial bone defects are usually protective or cosmetic. To our knowledge, the first reported use of the cranioplasty technique to repair skull defects dates to 1670, when J. van Meekren successfully used a bone of a dog to repair a cranial defect in man. Although the graft was successful, it was removed because the church took great offense to using an animal bone in man and viewed it as “marring God’s image.”

Curative effects have been noted following cranioplasty. These procedures are usually performed months after the initial injury or operation. A well-known indication for cranioplasty is the ST—severe headache, dizziness, undue fatigability, poor memory, irritability, convulsions, mental depression, and intolerance to vibration. In 1977 Yamaura and Makino coined the term “syndrome of the sinking skin flap” and underlying tissue, and they reasoned it was secondary to the action of atmos-

**Abbreviations used in this paper:**
- CBF = cerebral blood flow
- CSF = cerebrospinal fluid
- CVR = cerebrovascular reserve
- ICA = internal carotid artery
- MCA = middle cerebral artery
- PET = positron emission tomography
- ST = “syndrome of the trephined”
- TCD = transcranial Doppler
- 18-FDG = 18-fluorodesoxyglucose
phasic pressure. In 1968 Langfitt experimentally showed that the CSF pressure in the upright position is higher in a model with a large bony defect than that in one with a closed skull, whereas in 1976, Magnaes reported that the 0 CSF pressure level and the hydrostatic indifferent point were cranially shifted in five patients with large flaccid skull defects, in whom the parameters returned to normal levels after a cranioplasty. Neurological improvement is repeatedly noted, possibly caused by improvement of cerebral blood flow after cranioplasty. A controversial indication for cranioplasty is the prevention or amelioration of postoperative epilepsy. However, in more recent studies the authors demonstrate that cranioplasty does not alter the frequency or incidence of postoperative epilepsy. In only a few reports is a neurological amelioration demonstrated in patients after the bone flap replacement. The personal case of one patient, who recovered dramatically (his capacity for language was restored immediately after cranioplasty), stimulated us to conduct this prospective study to obtain more insight in the pathophysiological background of this condition (Werani, Radau, and Winkler, manuscript in preparation).

Some information about the hydrodynamic studies of CSF before and after cranioplasty is available, and previous investigations were published by Fodstad, et al. Understandably, skull defects after decompressive craniectomy might have greater impact on dynamic CBF regulation than on resting blood flow, thus contributing to the impairment of normal brain function and metabolism. Because this issue has not yet been previously addressed, the present prospective study was designed to investigate the influence of cranioplasty on indices of CBF as determined by TCD ultrasonography during postural maneuvers. From the TCD findings the CVR reserve capacity (as a percentage value) was calculated. Furthermore, the effect of cranioplasty on cerebral metabolism was investigated using 18-FDG PET, which also served as an indicator of functional improvement.

All tests were performed by highly qualified professionals. The first author (P.A.W.) was responsible for establishing the TCD ultrasonography as a routine procedure at this institution in 1986. The PET studies were supervised by the senior author (K.T.).

**CLINICAL MATERIAL AND METHODS**

This study was approved by the ethical committee of the Ludwig Maximilian University.

**Surgical Protocol**

Thirteen patients were included in this study. Data on the clinical diagnoses and the time between exploration and reimplantation of bone flaps are presented in Table 1. In 10 (77%) of 13 cases the removed autologous bone flap was reimplanted in an average of 11.1 weeks (range 6–20 weeks), and in three cases (23%), a stereolithographic method was used to perform a three-dimensional reconstruction of the skull defect due to a long duration between explantation of the bone flap and reconstruction of the defect (the time lapse ranged from 3 years to an unknown period of time). Computerized tomography scanning was performed in all patients both pre- and postoperatively on a routine basis (Fig. 1).

**Measurement of Cerebrovascular Reserve Capacity**

We used TCD ultrasonography to acquire information on the CBF and thereby the CVR capacity. Transcranial Doppler ultrasonography (TC-2-64; EME, Überlingen, Germany) to determine the blood flow velocities in the MCA and extracranial ICA was performed in all patients pre- and postoperatively. Measurements were obtained prior to and 7 days after cranioplasty. Supine and sitting positions were considered for postural changes in CBF by using the same technique. The CVR capacity was calculated after flow stimulation with 1 g of intravenously administered acetazolamide as: where \( V_v \) is the stimulated TCD flow velocity and \( V_r \) is the resting TCD flow velocity.

**Positron Emission Tomography Studies**

The clinical condition in 12 of the 13 patients permitted PET for assessment of cerebral metabolism prior to and 7 days after cranioplasty. Patients fasted for at least 6 hours prior to the examination (blood glucose levels 88–137 mg/dl precranioplasty and 78–124 mg/dl postcranioplasty). Static PET studies were performed for 30 minutes, starting 30 minutes after the intravenous administration of 18-FDG (range prior to cranioplasty 343–405 MBq; range after cranioplasty 325–406 MBq) by using a tomograph (Siemens HR +; Knoxville, TN) in two-dimensional mode, permitting simultaneous acquisition of 63 contiguous transverse sections of 2.46-mm thickness covering an axial field of view of 15.5 cm. Tissue attenuation of 511-keV gamma radiation was measured for 15 minutes with three 68-Ge rotating rod sources. The emission data were reconstructed by a filtered back projection reconstruction algorithm in which a Hanning filter at a cut-off frequency of 0.5 Nyquist was used. Positron emission tomography examinations were performed in the calm atmosphere of a room with dimmed lighting, no conversation, and only ambient noise from the scanner gantry fans.

**Analysis of PET Data**

Volumes of interest were defined for the quantitative assessment of FDG uptake in the cortex of both hemispheres. To achieve comparability, the results of all PET studies were coregistered to a standardized volume map, adjusting nine parameters, three each for rotation, translation, and anisotropic scaling. The mean FDG uptakes in both hemispheres were established before and after cranioplasty. Asymmetry between homologous regions was calculated, and the influence of cranioplasty on cerebral glucose metabolism was estimated as:

\[
\text{ipsilateral} - \text{contralateral} \times 200\%
\]

\[
\text{ipsilateral} + \text{contralateral}
\]
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TABLE 1
CLINICAL DATA OBTAINED IN 13 PATIENTS IN WHOM CRANIOPLASTY WAS PERFORMED AT DIFFERENT TIMES AFTER CRANIECTOMY *

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)†</th>
<th>Reason for Craniectomy</th>
<th>Time Lapse</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>34</td>
<td>SAH; SDH; rt ICH</td>
<td>12 wks</td>
<td>rt hyperreflexia; anxiety; lack of drive; reduced alertness, cooperation &amp; learning ability</td>
</tr>
<tr>
<td>2</td>
<td>31</td>
<td>rt subtotal MCA infarction</td>
<td>20 wks</td>
<td>lt hemineglect; lt hemiplegia; UMN facial paralysis</td>
</tr>
<tr>
<td>3</td>
<td>63</td>
<td>rt acute SDH</td>
<td>18 yrs‡</td>
<td>It severe paraparesis</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>Arnold-Chiari malforma-</td>
<td>3 yrs‡</td>
<td>ataxia, lt hyperreflexia; functional nystagmus; reduced cognition</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tion; syringomyelia; in childhood rt SDH; down- ward displacement of thalamic structures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>69</td>
<td>rt acute SDH (trauma)</td>
<td>12 wks</td>
<td>gait disturbance; severe ataxia</td>
</tr>
<tr>
<td>6</td>
<td>56</td>
<td>lt frontal ICH</td>
<td>11 wks</td>
<td>rt hemiparesis; severe motoric aphasia</td>
</tr>
<tr>
<td>7</td>
<td>52</td>
<td>rt basal ganglia hemato-</td>
<td>3 yrs‡</td>
<td>lt spastic hemiparesis</td>
</tr>
<tr>
<td>8</td>
<td>64</td>
<td>rt ICH</td>
<td>13 wks</td>
<td>lt hemiplegia; disorientation; dysphagia</td>
</tr>
<tr>
<td>9</td>
<td>52</td>
<td>rt ICH</td>
<td>9 wks</td>
<td>hemianopsia; hyperreflexia; lt hemiparesis</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>EDH; rt frontoparietal</td>
<td>6 wks</td>
<td>retrograde amnesia; gait disturbance; blurred vision</td>
</tr>
<tr>
<td>11</td>
<td>69</td>
<td>subtotal MCA infarction; rt frontoparietotemporal; coma</td>
<td>7 wks</td>
<td>lt hemiparesis; lt facial paralysis; lt hemineglect; dyspraxia lt hand</td>
</tr>
<tr>
<td>12</td>
<td>50</td>
<td>lt acute SDH</td>
<td>11 wks</td>
<td>difficulty in finding words aphasia; hemiparesis rt slight</td>
</tr>
<tr>
<td>13</td>
<td>49</td>
<td>frontoparietal meningio- ma; bleeding during embolization procedure; midbrain compression &amp; lt herniation</td>
<td>10 wks</td>
<td>neurologically symptom free normalization of all neurological symptoms</td>
</tr>
</tbody>
</table>

* EPH = epidural hematoma; ICH = intracerebral hematoma; SDH = subdural hematoma; SAH = subarachnoid hemorrhage; UMN = upper motor neuron.
† At the time of cranioplasty.
‡ Stereolithographic three-dimensional reconstruction was used.

Statistical Analysis

All values here are presented as mean ± standard deviation. The Kolmogoroff–Smirnow test was used to analyze the normal distribution, and significance of data differences was tested using the two-tailed paired t-test or Wilcoxon’s test. An error probability of p less than 0.05 was considered significant. The PET results were correlated with the results of TCD investigations and neuropsychological tests or clinical findings.

RESULTS

Clinical Outcome

The diagnosis at the time of decompressive craniotomy, the patients’ clinical symptoms before cranioplasty, and the condition there after are listed in Table 1.

An overall functional and cognitive improvement in patients could be observed after they received their implants. However, functional and cognitive improvement was not found in three patients in whom a significant time lapse had occurred between craniectomy and cranioplasty. In the patient in Case 4 with Arnold–Chiari malformation, the reconstruction of the cranial defect effected slight improvement: his cognitive function improved slightly, and he became more cooperative, but ataxia remained unchanged, not considering the longest time lapse between decompressive craniectomy and reconstruction. In the patient in Case 3, although 18 years had lapsed between decompressive craniectomy and cranioplasty, a slight improvement was observed. However, in the patient in Case 7, one of the three long-term cases, ironically the clinical condition remained unchanged. In all other cases (except Case 2) either the clinical condition completely

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improved or it reached normal levels in relation to daily social interaction. The patients became cooperative, without distress, and quite communicable after undergoing cranioplasty. Although her spastic paresis improved and she could walk without support, the arm of one patient (Case 3) remained plegic at her 2 year follow up.

The CVR Capacity

No significant difference was observed in the resting blood flow velocities in extracranial ICA when comparing ipsilateral and contralateral hemispheres and when assessing the effects of cranioplasty (Fig. 2). However, although blood flow in the MCA was significantly impaired in the ipsilateral as compared with the contralateral hemisphere prior to the procedure, following cranioplasty the impairment was only marginal, returning to almost near-normal values.

By moving to the sitting position, the blood flow velocity in the extracranial ICA significantly decreased on the side on which craniectomy had been performed as compared with the intact contralateral hemisphere. Cranioplasty resolved postural influences on extracranial ICA flow (Fig. 3).

Although the CVR capacity before cranioplasty was severely impaired in both hemispheres, after cranioplasty a striking improvement was observed in this parameter in both the ipsilateral as well as the contralateral extracranial ICA (Fig. 4).

Results of the PET Studies

Cerebral metabolism as assessed by 18-FDG PET was found to be depressed in the injured compared with the contralateral hemisphere (p < 0.01). Cranioplasty resulted in a significantly increased uptake of 18-FDG in both hemispheres (p < 0.001), indicating globally enhanced metabolism in the brain (Fig. 5 upper and Table 2).

Fig. 1. Computerized tomography scans obtained before (upper two rows) and after (lower two rows) cranioplasty in a patient who had undergone decompressive craniectomy for acute subdural hematoma 4 months previously.

Fig. 2. Bar graph depicting the influence of cranioplasty on resting TCD flow values obtained in 13 patients (averages ± SD). eICA = extracranial ICA; ipsi = ipsilateral hemisphere; contra = contralateral hemisphere. Vertical bars show the TCD values in milliliters/second.
However, the findings were more pronounced in the ipsilateral hemisphere (ipsilateral and contralateral mean increases ~ 12% and 4%, respectively). These results were neither related to the injected FDG dose nor the blood glucose level. Metabolic asymmetry decreased from 35% to 28% after cranioplasty. We found a significant correlation between the FDG uptake in the injured hemisphere and the extracranial ICA blood flow velocity assessed in supine and erect positions prior to (r = 0.870 and r = 0.860 respectively, p < 0.05) and after cranioplasty (r = 0.867, p < 0.05), but not in the sitting position. There was no dependent relationship between extracranial ICA flow and cortical FDG uptake contralaterally. Furthermore, no relationship could be established between glucose metabolism and flow stimulation studies with acetazolamide or MCA blood flow measurements in either the ipsi- or contralateral hemispheres.

The improvement of the glucose metabolism demonstrated in the injured hemisphere after cranioplasty was a good predictive value for a patient’s clinical outcome. We found an increase of FDG uptake in 10 of 12 patients. During follow up, all these patients presented with an
overall improvement and a reduction of the symptoms. The PET results and clinical condition remained unchanged only in the patient in Case 7. Normalization of all neurological symptoms was observed in the patient in Case 13, whereas the mean cortical FDG uptake remained stable.

Nevertheless, in the latter case we found obvious changes in the regional glucose metabolism, particularly in the margins of the defect.

DISCUSSION

Several authors have described the ST the appearance of symptoms weeks and months after craniectomy, with aggravation of symptoms when the patient changes body position and relief or reduction of symptoms after cranioplasty. In the literature there are very few case reports in which the ST is described. Large concave cranial defects were present in all reported cases. A variety of theories have been developed regarding the role of the bony defect of the cranium in the development of the neurological symptoms. The size and location of the cranial defect may be important.

Derangement of CSF hydrodynamics as well as regionally impaired CBF are well documented.

In 1979 Fodstad and colleagues reported their study of CSF hydrodynamics in 18 patients (13 men and 5 women aged 23–59 years [average age 40.5 years]. In 15 cases the large cranial defect was caused by decompressive craniectomy that had been performed to treat various conditions (eight trauma, five subarachnoid hemorrhage, two tumors, and one encephalitis) and in two cases because of postop-

Fig. 5. Upper: Positron emission tomography scans demonstrating the influence of cranioplasty on cerebral metabolism. Upper Row: Scans obtained prior to cranioplasty. Lower Row: Scans obtained after cranioplasty. Lower: Bar graph showing quantitative evaluation of effects of cranioplasty on cerebral metabolism in 12 patients (means ± SD).
In patients in whom large defects were present before and after cranioplasty the constant-pressure infusion method was used to study CSF hydrodynamics. The results of the CSF hydrodynamic studies before and after cranioplasty were somewhat varied and not easy to interpret. In eight of the patients normal CSF hydrodynamic values were observed before and after cranioplasty; all of these patients had flat and rigid skull defects. In seven other patients with low and one with high resting pressure and sinus pressure preoperatively, normal values were demonstrated after cranioplasty. These patients had large defects with flaccid concavity of the skin flap. In 11 patients the investigators measured the CSF pressure both in the supine and the sitting position. They found that the CSF pressure tended to change in the sitting position after cranioplasty. At the same time the pulse amplitude became larger. There was also a striking change in elastance and pulse-related CSF pressure variations in some patients after cranioplasty. In five patients the electroencephalograph findings were improved after cranioplasty, and many of the patients with changed CSF hydrodynamics preoperatively claimed that they experienced fewer episodes of headache and vertigo after the operation. The authors concluded that despite the fact that their series was small and their findings somewhat confusing, some conclusions could be inferred from their data. The change in CSF hydrodynamics with an improved clinical picture in patients with skull defects could be explained by the stretching or a distortion of the dura and the underlying cerebral cortex due to the atmospheric pressure with possible shifting of the intracranial contents. This is supported by the fact that the patients who benefited most from cranioplasty were those in whom there were large defects near the dural sinuses with concave deformity of the skin flap that allowed transmission of the atmospheric pressure directly to the cerebral cortex. Such cranial defects should therefore be repaired as early as possible.

In a second published series, Fodstad, et al., described cranial bone defects in 40 patients after craniectomy in whom extensive CSF hydrodynamic investigations were performed using CSF infusion test before and after cranioplasty. The results of these investigations were shown to be related to the clinical signs of the patients before and after cranioplasty and to the size and location of the skull bone defect. After cranioplasty the patients were classified into the following four groups.

Group I consisted of “true ST patients;” 14 patients with symptoms of ST, which was aggravated during the Vasalva maneuver or by changes in body position. Their symptoms were not present before craniectomy and were reduced or relieved after repair of the cranial bone defect. Group II was comprised of “partial ST patients;” eight patients with symptoms of ST not present before craniectomy. The symptoms were more or less relieved after cranioplasty but were unaffected by the Vasalva maneuver or positional changes. Group III consisted of “other patients;” 12 patients with neurological deficits that could be related to their primary disease or the surgical procedure. Group IV was made up of “symptom-free patients;” six patients without ST or neurological deficits.

The CSF hydrodynamic variables that were changed before and normalized after cranioplasty included the following: resting pressure; sagittal sinus pressure; buffer volume, elastance at resting pressure; and pulse variations at resting pressure. In Group I, 79% of the patients experienced relief of symptoms and 21% were improved after cranioplasty. All these patients had flaccid skin flaps that became concave while in the upright position. In 10 of

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Ipsilateral Side</th>
<th>Contralateral Side</th>
<th>Overall Clinical Improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8.8</td>
<td>4.0</td>
<td>yes</td>
</tr>
<tr>
<td>2</td>
<td>27.9</td>
<td>4.6</td>
<td>yes</td>
</tr>
<tr>
<td>3</td>
<td>18.7</td>
<td>6.0</td>
<td>yes (slight)</td>
</tr>
<tr>
<td>4</td>
<td>13.6</td>
<td>8.4</td>
<td>yes (slight)</td>
</tr>
<tr>
<td>5</td>
<td>10.3</td>
<td>2.4</td>
<td>yes (partial)</td>
</tr>
<tr>
<td>6</td>
<td>17.1</td>
<td>4.9</td>
<td>yes</td>
</tr>
<tr>
<td>7</td>
<td>−0.2</td>
<td>8.8</td>
<td>no</td>
</tr>
<tr>
<td>8</td>
<td>13.6</td>
<td>5.4</td>
<td>yes</td>
</tr>
<tr>
<td>9</td>
<td>13.0</td>
<td>−0.4</td>
<td>yes</td>
</tr>
<tr>
<td>10</td>
<td>6.7</td>
<td>4.7</td>
<td>yes</td>
</tr>
<tr>
<td>11</td>
<td>15.8</td>
<td>9.3</td>
<td>yes</td>
</tr>
<tr>
<td>12</td>
<td>−</td>
<td>−</td>
<td>yes</td>
</tr>
<tr>
<td>13</td>
<td>−0.1</td>
<td>−3.7</td>
<td>yes</td>
</tr>
</tbody>
</table>

mean improvement ± SD 12.1 ± 2.3% 4.5 ± 1.1% yes

* = results could not be interpreted.
these patients defects were located in the parietooccipital region. All patients in Group II were improved after cranioplasty. Seven of these patients had flaccid defects, and one patient had a rigid skin flap. The defects were located in the frontoparietal region in five of eight cases. In patients in Groups III and IV, rigid skin flaps were usually demonstrated, predominantly in the frontal and temporal regions. In these groups, defects extended over the midline. All patients with headache, vertigo, or a feeling of discomfort experienced improvement or complete relief of symptoms after cranioplasty, regardless of whether the symptoms were aggravated by changes in body position or not. In 10 of 17 patients with hemiparesis, improvement or relief was shown, and in the two patients with visual defects, and in five of eight patients with dysphasia improvement or relief of symptoms was also demonstrated. In this study memory disturbance was minimally affected by cranioplasty, whereas irritability and dyspraxia was not affected at all.5

It has also been suggested that the atmospheric pressure acts directly on the cerebral cortex in cases with a concave deformity.12,23,29,33,37

In a report currently being prepared by our group, the results obtained in one case stimulated us to conduct the present study. Before bone flap reimplantation this patient presented with a clinical picture of Wernicke’s aphasia with severe disturbances in all linguistic modalities. Soon after surgery, the patient improved dramatically, and 3 weeks after the bone flap replacement his spontaneous speech was totally unimpaired on all levels of observation. There remained neither disturbances in word choice and word finding nor phonemic or syntactical difficulties. The patient was able to discuss all topics without any assistance. In all other subtests of the Aachen Aphasia Test, very good results could be obtained. Compared with the initial test profile our patient’s final test showed not only a significant improvement in all linguistic modalities but also no evidence of any aphasic language disorder as such.

The influence of the regionally impaired CBF has not yet been studied, and in the present study we paid full attention to the impact of the CVR capacity and the cerebral glucose metabolism on the condition of the patients with so called ST.

All reported series have shown the impact of cranio- plasty on the influence of atmospheric pressure and CSF hydrodynamic values. In this present study the influence of cranioplasty on CVR capacity and cerebral glucose metabolism was extensively investigated.

The extracranial ICA blood flow is a global marker for the brain perfusion, whereas the MCA blood flow is representative for a large but distinct cortical area. Therefore, the impairment of MCA blood flow depends more on the locations of the brain defect. In this study we focused on the evaluation of the changes of the overall brain glucose metabolism after cranioplasty. This could be the reason why we did not find a relation between MCA blood flow and the total FDG uptake in the brain. Future studies should elucidate these aspects of CBF and metabolism. The significant correlation of ipsilateral extracranial ICA flow in supine and sitting position clearly demonstrates the influence of impaired blood flow on brain metabolism. Mielke, et al.22 have demonstrated an association between the extent of perfusion deficit and the reduction in glucose metabolism. The restitution of the percentage of CVR capacity and the resolved postural influences on extracranial ICA flow after cranioplasty corresponded well to the significant increase of cerebral glucose metabolism in both the ipsi- and contralateral hemispheres.

Furthermore, the increase of glucose metabolism not only correlates with the restitution of CBF but is a good predictive value for clinical outcome after cranioplasty. Although the location of the lesion is more important than the volume of disrupted tissue for predicting the consequences of a brain injury,15 we found that patients in whom an increasing cerebral metabolism is shown after cranioplasty, also experience improvement of their clinical symptoms.

Our data on late reconstruction with stereolithographic technique presented here are too scant to try to establish a correlation between the clinical condition and the types of material used, the time lapse between cranietomy and cranioplasty, or even the initial condition of the patient that indicated the decompressive cranietomy. Nevertheless, the unchanged state after cranioplasty in the patient in Case 7 might have resulted from irreversible changes in functionally important structures in the basal ganglia. The same speculation could be made about the patient in Case 2. She suffered occlusion of the common carotid artery caused by a massive thrombus, which might have already caused irreversible cortical changes before she underwent the cranietomy. Hence, the time lapse may not have made a significant difference in this patient. One observation that makes us connect the outcome to the initial clinical status is that all patients presenting with “less complicated” conditions, such as subdural or epidural hematoma or an acute hematoma that was promptly treated improved, and showed excellent recuperation after cranioplasty.

Analysis of our data indicates that cranioplasty after decompressive cranietomy may markedly improve cerebral glucose metabolism, as assessed with PET. Therefore, this surgical procedure not only restores cerebral protection and ensures cosmetic repair but also is associated with partial functional recovery of reversibly damaged cortical and subcortical structures of the primarily affected as well as the nonaffected hemispheres.

CONCLUSIONS

The present observations demonstrate that chronic decompressive cranietomy not only impairs postural blood flow regulation in the ipsilateral hemisphere but also CVR capacity in the brain as a whole. Cranioplasty improves both parameters, whereas only minor effects were observed regarding resting blood flow. Accordingly, cranioplasty resulted in marked improvement of metabolic activity, not only in the decompressed hemisphere but also in the contralateral hemisphere.

For functional rather than traditional reasons such as protection or cosmetic repair, our findings underscore the importance of early cranioplasty in patients who have undergone decompressive cranietomy.

Acknowledgments

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References


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