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Clinical Article

Relevance of ICP and $ptiO_2$ for indication and timing of decompressive craniectomy in patients with malignant brain edema

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Summary

Background. The exact effects of decompressive craniectomy on intracranial pressure (ICP) and cerebral tissue oxygenation ($ptiO_2$) are still unclear. Therefore, we have monitored ICP and $ptiO_2$ intraoperatively and correlated these values to different operative steps during craniectomy.

Methods. ICP and $ptiO_2$ values have been monitored both, simultaneously and continuously, in 15 patients with cerebral edema due to posttraumatic or postischemic brain swelling. Indications for craniectomy were an increase in ICP above 25 mmHg or a decrease in $ptiO_2$ below 10 mmHg resistant to conservative treatment (e.g. mannitol, hyperventilation, adequate arterial blood oxygenation, etc.). In all cases, we performed a fronto-temporo-parietal craniectomy (15×12 cm) and dura enlargement with galea-periosteum. During craniectomy, monitoring of ICP and $ptiO_2$ in the affected hemisphere was continued. Values were recorded and correlated with the different operative steps.

Findings. We performed craniectomy according to our treatment protocol in 5 patients. Prior to surgery, mean ICP values were 25.6 mmHg (range: 23–29 mmHg), mean $ptiO_2$ values were 5.9 mmHg (range: 2.4–9.5 mmHg), and mean CPP values were 66 mmHg (range: 60–70 mmHg). After removing the bone flap, ICP values dropped to physiological values (mean: 7.4 mmHg), whereas $ptiO_2$ values increased only slightly (mean: 11 mmHg). Opening of the dura resulted in a further decrease of ICP (mean 4.8 mmHg) and an increase of $ptiO_2$ to normal limits (mean: 18.8 mmHg). After skin closure, mean ICP was 6.8 mmHg and mean $ptiO_2$ was 21.7 mmHg, respectively. We found a significant decrease of ICP after craniectomy ($p < 0.042$) and after dura enlargement ($p < 0.039$) as well as a statistically significant increase in $ptiO_2$ after craniectomy ($p < 0.043$) and after dura enlargement ($p < 0.041$).

Conclusion. As a large bone flap in decompressive craniectomy is essential for adequate ICP reduction, the results of the presented cases suggest that dura enlargement is the crucial step to restore adequate brain tissue oxygenation and that $ptiO_2$ monitoring could be an important tool for timing craniectomy in the future.

Keywords: Intracranial hypertension; craniectomy; $ptiO_2$; indication.

Introduction

Brain edema is a clinical problem especially in patients with head trauma or cerebral ischemia and can lead to a profound rise in intracranial pressure (ICP). Dependent on the underlying pathology, edema may be vasogenic or cytotoxic and, furthermore, both forms of edema may affect each other. Additionally, the increase in ICP can be aggravated by cerebral blood congestion. Despite various conservative management strategies such as osmodiuretics, hypothermia, barbiturates, hyperventilation, free-radical scavengers, or ventriculostomy, ICP does not respond in each case, so that decompressive craniectomy may represent the ultimative therapeutic approach. The positive effect of bifrontal craniectomy on both, the ICP and the clinical outcome of head injured patients, has recently been demonstrated by Polin [18].

However, the ideal point of time for craniectomy and the exact effects of decompressive craniectomy on ICP and cerebral tissue oxygenation ($ptiO_2$) are still unclear. Therefore, we performed craniectomy according to pathological ICP or $ptiO_2$ values that were resistant to conservative treatment options. Intra-operatively, we proceeded to monitor ICP as well as $ptiO_2$ and correlated these values to different operative steps during craniectomy.

Patients and methods

Patient characteristics

Fifteen patients at risk of developing malignant cytotoxic brain edema aged between 18 and 65 years were included in this study.

On admission, cerebral pathology was confirmed by neuroradiological studies (cranial computed tomography [CCT] and cerebral panangiography). Seven patients suffered from subarachnoid hemorrhage (Hunt and Hess grade IV or V), three patients from severe head injury, and five patients from complete infarction of the middle cerebral artery. After evacuation of a mass lesion or operative treatment of a cerebral aneurysm, patients were sedated and mechanically ventilated.

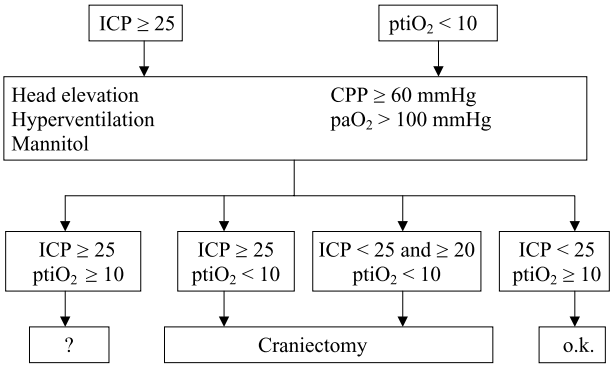
ICP and $ptiO_2$ monitoring

We used the LICOX microcather (GMS mbH, Kiel, Germany) for monitoring brain tissue oxygenation. Catheter placement was guided by a specific three-lumen-introducer which was fixed on a special skull screw. The depth of insertion of the probe from the dura to the catheter tip was standardized to 35 mm. Brain $ptiO_2$ values were continuously adjusted to the actual brain temperature by a brain temperature probe placed in the second lumen. The intraparenchymal pressure probe for ICP monitoring (Codman) was introduced in the third lumen of the introducer and adjusted to a depth of 40 mm. Monitoring probes were placed in the tissue at risk and the correct position was verified with CCT within 48 hours.

Treatment protocol

After insertion of the monitoring probes, a standardized treatment protocol was used (Table 1). A rise in ICP equal to or over 25 mmHg was managed by head elevation (30°), mild hyperventilation ($pCO_2 = 30–35$ mmHg), and bolus-application of mannitol (125 ml of mannitol 20% in 20 minutes). When a decrease in brain tissue oxygenation below 10 mmHg occurred, CPP was raised to a minimum of 60 mmHg by continuous catecholamine infusion and arterial blood oxygenation was maintained above 100 mmHg. Conservative treatment was continued when ICP was decreased below 25 mmHg and $ptiO_2$ increased equal or over 10 mmHg by these measurements. Indications for craniectomy were a decrease in $ptiO_2$ below 10 mmHg which had to be coupled with ICP values over 20 mmHg or a progradient increase in ICP of at least 25 mmHg resistant to conservative treatment. We defined no standard regime for patients with an increase in ICP equal or over 25 mmHg with a concurrent increase in $ptiO_2$ over 10 mmHg. In these cases the clinical condition of the patients as well as additional investigations like neurophysiological recordings should be used to confirm indications for craniectomy.

Table 1. *Algorithm for patient's management*



Operative procedure

As soon as craniectomy was indicated, a wide fronto-temporo-parietal craniectomy (15×12 cm) was performed on the side with the major pathology. The dura was widely opened in a stellate fashion to provide additional space for brain swelling. Subsequently, an autologous galea-periosteal flap was used as dura graft and a watertight dura-closure was performed over the affected hemisphere. Monitoring devices were left in place, draped in a sterile manner and monitoring of ICP and $ptiO_2$ in the affected hemisphere was continued during craniectomy. Values were recorded and correlated with the different operative steps. Statistical significance was determined using the Wilcoxon test.

Results

In 10 out of 15 patients, pathological changes in ICP and $ptiO_2$ were successfully treated by conservative management. We observed in no patient an increase in ICP equal or over 25 mmHg with a concurrent increase in $ptiO_2$ over 10 mmHg. Craniectomy was indicated in 5 out of 15 patients according to the treatment protocol. An isolated cerebral hypoxemia was the rationale for craniectomy in two patients, whereas a combined increase in ICP and decrease in $ptiO_2$ was the indication for craniectomy in three patients (Table 2). Prior to surgery, mean ICP values were 25.4 mmHg (range: 23.0–29.0 mmHg) and mean $ptiO_2$ values were 5.9 mmHg (range: 2.4–9.5 mmHg) respectively. After removing the bone flap, ICP values dropped to physiological limits (mean: 7.4 mmHg) whereas $ptiO_2$ values increased only slightly (mean: 11 mmHg). Opening of the dura resulted in a further decrease of ICP (mean: 4.8 mmHg) and a progressive increase of $ptiO_2$ up to normal limits (mean: 18.8 mmHg) after dura enlargement. After skin closure, mean ICP was 6.8 mmHg and mean $ptiO_2$ 21.7 mmHg, respectively (Fig. 1a and b).

Changes of $ptiO_2$ and ICP between the different operative steps were analyzed with the Wilcoxon test for statistical significance. Statistical analysis revealed a significant decrease of ICP after craniectomy ($p < 0.042$) and after dura enlargement ($p < 0.039$) as well as a significant increase in $ptiO_2$ after craniectomy ($p < 0.043$) and after dura enlargement ($p < 0.041$). ICP and $ptiO_2$ measurement was continued during the postoperative period in two patients with subarachnoid hemorrhage for 48 hours and 92 hours, respectively and in one additional patient with brain trauma for 12 hours. During the whole postoperative monitoring time, neither critical decreases of $ptiO_2$ (below 10 mmHg) nor critical increases of ICP (above 20 mmHg) were observed. Three months after craniectomy Glasgow outcome score (GOS) was three in three patients and four in two patients.

Table 2. *Characteristics of patients treated with craniectomy*

Patients	Disease	Before craniectomy		After craniectomy		Time interval between start of monitoring and craniectomy	GOS
		ICP	$ptiO_2$	ICP	$ptiO_2$		
1	head trauma III	23	4.2	7	13.2	72 h	3
2	infarction of the MCA	23	9.5	6	29.2	24 h	4
3	infarction of the MCA	25	2.4	3	18.2	4 h	3
4	SAH Hunt and Hess IV	29	6.3	12	19.5	72 h	4
5	SAH Hunt and Hess IV	28	8	6	28.2	46 h	3

GOS Glasgow outcome scale; *MCA* middle cerebral artery; *SAH* subarachnoid hemorrhage.

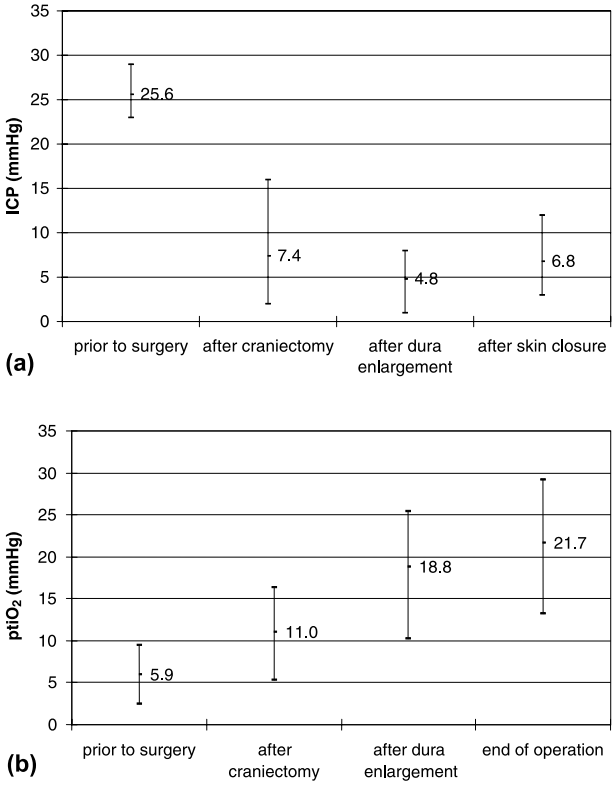


Fig. 1. (a) Intra-operative changes of ICP. (b) Intra-operative changes of $ptiO_2$

Discussion

The basic pathological mechanism in the development of brain edema is an increase in the net water content of the brain. In cytotoxic brain edema, intracellular water increases due to a failure or inadequate function of the Na^+/K^+ pump in the glial membrane. This disturbance results in intracellular retention of H_2O and Na^+ [17]. In vasogenic brain edema, extracellular water increases because of an interruption of the blood-brain barrier that allows plasma or a filtrate to enter the extracellular space of the brain [23].

The consequence in both scenarios is a retention of fluid within the brain and an increase in tissue pressure, which increases the cerebrovascular resistance, compromises the microcirculation [4], and results in reduced cerebral blood flow (CBF) and CPP. Furthermore, the alteration of the cellular microgeometry results in abnormal diffusion of nutrients to the brain. This leads to a further rise in ICP and a further deterioration of cerebral blood supply with nutrients. The study of Schroder *et al.* [20] demonstrated that the reason for brain ischemia after trauma is a compromise of the microvasculature and that CBF as well as cerebral blood volume is significantly lower in ischemic areas.

Therefore, craniectomy is considered as an appropriate procedure to control intracranial hypertension resistant to conservative treatment as this procedure disrupts the events' cascade, which links primary to secondary brain damage by rebalancing the cerebral inflow-outflow regulation and by reducing the transmural pressure at the capillary bed. The effects of craniectomy are a regression of blood congestion, a reduction of edema formation, and an increase of edema absorption [19].

Different methods of decompressive craniectomy for ICP reduction have been reported in the literature. The subtemporal craniectomy had been introduced by Cushing [8] in 1905. Because this procedure alone did not lead to a better outcome in many cases, a combination of bone removal and intracranial mass reduction was favored by Gurdjian and Thomas [10] in 1964. Clark *et al.* [2] described a circumferential craniotomy but did not recommend this procedure due to fatal outcomes. Other groups advocated a bifrontal craniectomy [16, 23] as this method allows decompression of both cerebral hemispheres and avoids lateral brain shifts with the risk of midbrain compression. Gerl and Tavan [7] supported a bilateral craniectomy and Gaab *et al.* [6] recommended a wide uni- or bilateral

fronto-temporo-parietal craniectomy combined with dura enlargement.

It is now accepted that a large bone flap is essential for adequate ICP reduction [1, 3, 12] and that surgical decompression should be routinely performed when indicated before irreversible ischemic brain damage occurs [9]. However, the amount of ICP reduction achieved by craniectomy differs. Jourdan *et al.* [15] reported in their series of nine patients an ICP reduction of 15%, but opening of the dura achieved a reduction of 70%. Gower *et al.* 1988 [8] were able to reduce ICP by subtemporal decompressive craniectomy by about 34% in seven cases. In the present study, we also found a statistically significant decrease in ICP after craniectomy and dura enlargement. However the most interesting finding of the $ptiO_2$ monitoring was that dura enlargement is the most important measure to restore adequate brain tissue oxygenation. These results are similar to the findings of Jaeger *et al.* [14], who examined the intra-operative course of $ptiO_2$ and 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. However, criteria for decompressive craniectomy and especially the relevance of $ptiO_2$ monitoring remain unclear in their study. In our study, 15 patients with severe brain edema due to subarachnoid hemorrhage, brain injury, or cerebral ischemia have been monitored (ICP, CPP, $ptiO_2$) before, during, and up to 72 hours after craniectomy. Clear criteria had been defined for craniectomy with special regard to $ptiO_2$. Thus, in contrast to Jaeger *et al.*, the indication and the timing for decompressive craniectomy was based upon the monitored data. Additionally all patients in our study survived either severely or moderately disabled whereas in the study of Jaeger patients were either dead ($n = 1$) or in a vegetative state ($n = 2$) after 6 months. Therefore, in our experience decompressive craniectomy may be an additional treatment option in patients with mildly elevated ICP of at least 20 mmHg and a decrease of $ptiO_2$ below 10 mmHg. As a low brain oxygenation is the precondition for the development of a cytotoxic brain edema leading to a vicious circle with a further rise in ICP and compromise of cerebral blood flow, our concept is to restore normal brain homeostasis by performing a decompressive craniectomy as early as possible. Especially in cases with a mildly elevated ICP of at least 20 mmHg additional monitoring of $ptiO_2$ is helpful to differentiate between a cytotoxic brain edema with a further rise of ICP and a steady state condition, in which a mildly elevated ICP represents cerebral hyperemia which provides adequate brain oxygenation. Our experience that decompressive craniectomy promotes brain oxygenation is also supported by the study of Stiefel *et al.* [21] who found a significant improvement in brain oxygenation after decompressive craniectomy. However, the indication for craniectomy in their study was only based on ICP values neglecting preoperative $ptiO_2$ values that might have indicated adequate brain tissue oxygenation in some patients.

After pressure relief by craniectomy, the injured brain has a high demand for oxygen and the previously compressed cerebral vessels fill again with blood. Furthermore, these vessels will be maximally dilated by metabolic changes and the brain soon gets into a hyperemic state. As brain edema is not primarily resolved by craniectomy and hyperemia does increase the net brain volume, a rise in ICP in the postoperative period is often observed. This postoperative increase in brain edema with the possible consequence of brain herniation and strangulation has made craniectomy questionable as a treatment modality for brain edema in the past. We also found a further increase of brain edema formation after craniectomy, on postoperative CCT, but the reduced tissue pressure – achieved by craniectomy and dura enlargement – allowed adequate reperfusion of the brain and established sufficient brain oxygenation – as confirmed by the continuous $ptiO_2$ monitoring – and thus prevented further secondary brain damage. These findings are also supported by the studies of Yamakami and Yamaura [24] and Yoshida *et al.* [25] who described an increase in regional CBF and in cerebral metabolism after craniectomy in head injured patients as compared to the preoperative results. In these patients, a marked hyperperfusion occurred in the area of the decompressed brain that may protect the brain from secondary ischemic cell damage (e.g. lactate and potassium clearance) and thus, may explain the beneficial effect of craniectomy on the outcome. Without craniectomy, the ongoing process of edema formation leads to a progressive increase in local tissue pressure which displaces cerebrospinal fluid and blood out of the brain causing an impaired cerebral microcirculation. Brain strangulation is avoided by an adequate size of the craniectomy and was not observed in our series.

As adequate brain tissue oxygenation of the tissue at risk is the aim of intensive care therapy, $ptiO_2$ seems to

represent the most important parameter in neuromonitoring these patients. Neither ICP values nor CPP values alone can adequately predict the oxygen supply to brain tissue. These findings are in line with those of Härtl *et al.* [11], who suggested that patients with a CPP above 60 mmHg can have $ptiO_2$ values below 15 mmHg and that an optimum CPP does not exclude an hypoxemic brain. Therefore, $ptiO_2$ monitoring could be an additional and important tool in timing craniectomy in patients with malignant brain edema. However, the study population was small and heterogeneous. Thus, to clearly prove the benefit of $ptiO_2$ monitoring and the effectiveness of craniectomy, a prospective, randomized multicenter study, in which homogenous groups of patients are included and clear treatment protocols are used, is necessary [13].

Conclusion

In our experience decompressive craniectomy may be an additional treatment option in patients with mildly elevated ICP of at least 20 mmHg and a decrease of $ptiO_2$ below 10 mmHg. In these patients, craniectomy is not only effective in reducing ICP but has also a positive effect on brain tissue oxygenation. The statistically significant increase in brain tissue oxygenation to normal limits after additional dura enlargement makes this step especially important when performing craniectomy. Furthermore, $ptiO_2$ monitoring could be an important tool for timing craniectomy in the future.

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Comment

Reithmeier and colleagues further confirm the finding of immediate increases of $ptiO_2$ during decompressive craniectomy in their series of five patients. The indication for surgery was based on either ICP values above 25 mmHg or $ptiO_2$ values below 10 mmHg, both refractory to conservative measures. From our experience, however, it seems very critical to base decompressive craniectomy on $ptiO_2$ below 10 mmHg in the absence of intracranial hypertension, if CPP is only moderately raised to >60 mmHg as in their study. From the pathophysiological understanding of the disease, it is more appropriate to treat hypoxic $ptiO_2$ values in the absence of high ICP by aggressively elevating the CPP as individually required to counteract cerebral hypoxia and hypoperfusion. As recently demonstrated, this approach successfully reduces hypoxic phases [1]. Nevertheless, we consider craniectomy a sound treatment option, if restricted to patients with elevated ICP and associated cerebral hypoxia.

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