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Decompressive craniectomy for severe head injury in patients with major extracranial injuries

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Summary

Neurosurgical therapy aims to minimize secondary brain damage after a severe head injury. This includes the evacuation of intracranial space-occupying hematomas, the reduction of intracranial volumes, external ventricular drainage, and aggressive therapy in order to influence increased intracranial pressure (ICP) and decreased $P(ti)O_2$. When conservative treatment fails, a decompressive craniectomy might be successful in lowering ICP.

From September 1997 until December 2004, we operated on 836 patients with severe head injuries, of whom 117 patients (14%) were treated by means of a decompressive craniectomy. The prognosis after decompression depends on the clinical signs and symptoms at admission, patient age, and the existence of major extracranial injuries. Our guidelines for decompressive craniectomy after failure of conservative interventions and evacuation of space-occupying hematomas include: patient age below 50 years without multiple trauma, patient age below 30 years in the presence of major extracranial injuries, severe brain swelling on CT scan, exclusion of a primary brainstem lesion or injury, and intervention before irreversible brainstem damage.

Keywords: Head injury; edema; craniectomy; intracranial pressure.

Introduction

Bergmann described decompressive craniectomy in 1880 and Cushing published a case report about a subtemporal decompressive craniectomy for relief of intracranial pressure in 1908. There is still a controversy going on about the value of operative decompression after severe head injuries with traumatic brain edema [1, 2, 5, 6].

The aim of neurosurgical therapy after severe head injuries is the minimization of secondary brain damage. General principles of neurosurgical therapy are the evacuation of space-occupying hematomas, the reduction of intracranial volume, the drainage of hematocephalus, and conservative therapy focused on intracranial pressure (ICP), cerebral perfusion pressure, and brain tissue PO_2. In intractable intracranial hypertension that is refractory to conservative interventions, a decompressive craniectomy is indicated in a few patients. Indications for decompressive craniectomy, course of disease, and prognostic criteria are analyzed and compared with the literature [16–18].

Patients and methods

All patients with a severe head injury at the Unfallkrankenhaus are treated by the neurosurgical service in the interdisciplinary intensive care unit. Standard management includes an initial computed tomography scan in the emergency room. Patients were included in our prospective study when we saw an indication for a neurosurgical operation.

Patients with severe head injuries ($n = 836$) were operatively treated in the Department of Neurosurgery of the Unfallkrankenhaus Berlin between September 1997 to December 2004. The average age of the 674 male and 162 female patients was 41 years. Decompressive craniectomy was performed in 117 patients (14%). In 74 patients, craniectomy was performed in addition to removal of a space-occupying hematoma (subdural or epidural hematoma, contusion hemorrhage) because generalized brain edema was found intraoperatively. The second group included 43 patients in whom conservative treatment in the intensive care unit was therapy-resistant when neuro-monitoring with ICP, cerebral perfusion pressure, mean arterial pressure, and $P(ti)O_2$ measurement revealed intractable brain edema. The average age of the decompression group of patients was 35 years and the male/female ratio was 3:1 (87 male, 30 female). In 105 patients we performed a unilateral craniectomy (right side 48, left side 57), and in 12 patients a bilateral craniectomy was necessary.

Results

In accordance with the results of other groups [16], the majority of head injuries with indication for cra-

niectomy was due to motor vehicle accidents (58%), followed by falls (28%), attempted suicide (6%), and violence (8%). Fifty-five percent of patients had a craniectomy after diffuse brain injury, 27% after an acute subdural hematoma, 9% after an acute epidural hematoma, and 9% after an open head injury. In patients with a diffuse brain injury, the primary operative intervention was performed for evacuation of a space-occupying contusion hemorrhage.

Forty-seven patients (40%) died despite decompression, 16 patients (14%) remained in a vegetative state, 24 patients (20%) had persistent severe neurological deficits, and 30 patients (26%) reached a Glasgow Outcome Score (GOS) of 4 to 5. The course of disease in patients who were decompressed is illustrated in Fig. 1.

A comparison of patient age and postoperative results is not possible because of the small number of patients (Fig. 2). Altogether, patients younger than 40 years have a better prognosis than the older ones. In the fourth and fifth decades, satisfactory results are obtainable in 80%. According to our results, a decompression is less favorable in patients over 60 years of age, and our patients in this group remained in a vegetative state or died.

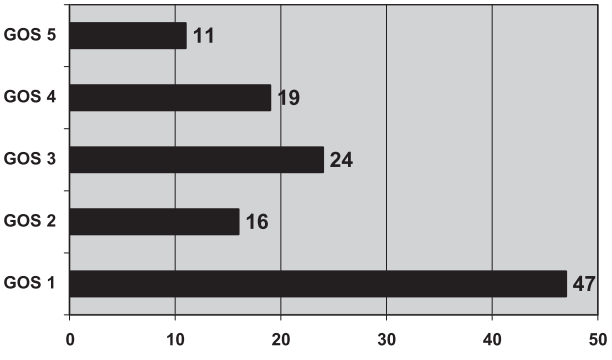

Fig. 1. Prognosis in severe head injuries with decompressive craniectomy

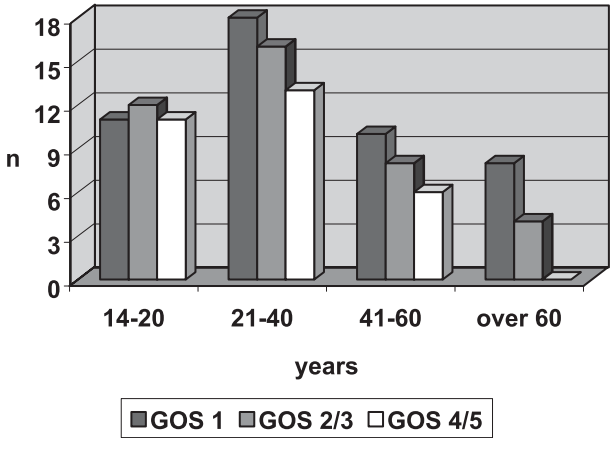

Fig. 2. Outcome versus patient age

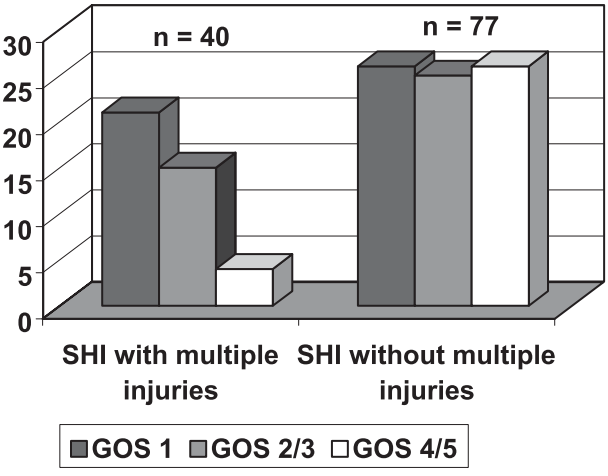

Fig. 3. Comparison between severe head injury (*SHI*) patients with and without major extracranial injuries

There is evidence for an important influence on the outcome when major extracranial injuries are present in contrast to an isolated head injury (Fig. 3). We found increased lethality of 53% when there was multiple trauma, in contrast to 34% with an isolated head injury. Furthermore, the outcome is influenced by the existence of an extracranial injury, with positive results in only 10% compared to 34% in isolated head injuries. In our small series, we could not find a difference in the time span between accident and surgical decompression. This is not contradictory for early decompression when indicated.

Discussion

Prognosis after severe head injury depends on the clinical status at admission, intracranial lesions, patient age, and the existence of accompanying injuries. Patients with epidural hematomas and open head injuries have a better prognosis than those with acute subdural hematomas. A poor prognosis is given in patients with brain contusions, diffuse brain injuries, traumatic injuries of the venous sinuses, less than 8 points on the Glasgow Coma Scale, and advanced age [16–18].

The pathophysiology of posttraumatic, primarily

vasogenic and cytotoxic, cerebral edema is mainly a disturbance of the functional entity consisting of capillary, astroglia, and neuron. The driving force for the formation of edema is the pressure gradient across the injured capillary with loss of blood-brain barrier function and an accumulation of extracellular fluid. Fluid accumulation is not caused by diffusion, but by a hydrostatic pressure gradient in the interstitium of the white matter. This global, mostly hemispherical blood-brain barrier disturbance induces generalized brain edema, which can only be marginally influenced by conservative treatment. To a degree, an intracranial volume increase can be compensated for by shifts in cerebrospinal fluid or blood volume. According to the intracranial pressure-volume relationship, when the intracranial reserve areas are exhausted, any further increase in volume is responsible for a considerable change in pressure. When the ICP increase decompensates, a decompressive craniectomy must be considered. Decompression changes the pressure-volume relationship and causes an increase in the compliance and, therefore, a shift to the right on the pressure-volume curve [1, 2, 6, 8, 10, 15].

In animal experiments, it was found by other groups [1, 2, 4, 11] that after decompression the injured brain exhibits an ICP decrease, an increase in the pressure-volume index, and a decrease in interstitial fluid accumulation. There are contradictory statements in the literature concerning an improvement in the regional cerebral blood flow and cerebrovascular resistance [11, 24]. In clinical trials with PET, Xenon-CT, and MR-spectroscopy, Yamakami *et al.* [24] and Yoshida *et al.* [25] found a hemispheric CBF increase as well as an increase in the cerebral metabolism after the craniectomy in comparison to the preoperative results. This requires early decompression and the craniotomy must be wide enough and include an opening and prolongation of the dura. Craniectomy should be performed when conservative treatment fails to influence an ICP between 25 to 40 mmHg and before the cerebral perfusion is irreversibly disturbed [2].

There are different methods described in the literature. Clark *et al.* [3] prefer circumferential craniotomy, and other groups [13, 20, 23] advise bifrontal craniotomy. Gerl *et al.* [6] recommend bilateral craniotomy, and Gaab *et al.* [5, 12] recommend wide unilateral or bilateral frontotemporal-parietal craniectomy with dura opening and duraplasty. According to the recommendations of Gaab *et al.* [5, 9] we have performed the wide frontotemporal-parietal craniectomy with duraplasty, for which we used the temporal muscle and its fascia or a Neuropatch (Braun, Melsungen, Germany) as a graft. The bone flap is preserved by subcutaneous implantation by storage under sterile conditions [19]. In accordance with Yoshida *et al.* [25], who found that regional cerebral blood flow and cerebral metabolism is reduced in the area of craniectomy, we performed the reimplantation after the regression of cerebral edema and when physiological ICP was present. Other than decompressions in patients with diffuse brain injuries, we performed a craniectomy more often in patients with acute subdural hematomas when the development of cerebral edema appeared during the operation and was resistant to conservative interventions.

The course of disease after craniectomy depends on the clinical status at admission, which was also found by other authors [5, 9, 21, 22]. In the literature, the postoperative results differ. In an analysis of the literature, there are between 11% and 71% dead despite decompression, 8% to 27% were in a vegetative state, 8% to 27% survived with severe neurological deficits, 0% to 21% survived with mild neurological deficits, and 0% to 41% had good recovery [5–7, 9, 12, 14, 20]. In another large analysis, Guerra *et al.* [9] found 49% of patients died, 13% survived with severe neurological deficits corresponding to a GOS of 2 and 3, and 32% had a favorable outcome with a GOS of 4 or 5. Our results are in accordance with those reported above, but we had a slightly larger number of patients in a vegetative state (Fig. 1). Polin *et al.* [20] found significantly better results after decompression, a finding that may be due to fewer patients with diffuse brain injuries who did not have surgery. In our comparable group, all were operatively treated patients after severe head injuries.

The influence of patient age on outcome after severe head injury and craniectomy (Fig. 2) was also found by other authors [5, 9, 14, 20, 21]. We need to critically re-evaluate the indication for decompressive craniectomy in patients over 60 years of age. Postoperative results in patients with a GOS of 3 is one reason for our higher rate of patients in a vegetative state, and we postulate that there should be an age restriction. Like Karlen *et al.* [12], the prognosis was worse when there were major extracranial injuries present (Fig. 3).

Under the influence of the results of Gaab *et al.* [5, 9, 21], we conclude with the following guidelines as an indication for decompressive craniotomy after severe head injuries:

1. Patient age below 50 years without multiple trauma.
2. Patient age below 30 years in the presence of major extracranial injuries.
3. Severe brain swelling on CT scan.
4. Exclusion of a primary brainstem lesion/injury.
5. Intervention before irreversible brainstem damage.
6. ICP increase up to 40 mmHg and unsuccessful conservative therapy.
7. Primarily for space-occupying hematomas with hemispheric brain edema.
8. Rising ICP and falling tissue oxygenation $P(ti)O_2$ before irreversible brain damage has occurred.

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