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Cerebral Perfusion Pressure for Prediction of Recurrent Intracranial Hypertension after Primary Decompressive Craniectomy

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Summary
Background: Decompressive craniectomy (DC) with dural grafting may be performed in patients with moderate (Glasgow-Coma-Scale [GCS] score 9-12 points) or severe traumatic brain injury (TBI; GCS score ≤ 8 points) and threatening herniation. However, its effectiveness especially after primary craniectomy is still discussed due to missing evidence of improved outcome. The objectives of this study were to show the incidence of recurrent intracranial hypertension after primary DC, to identify predictive parameters for secondary DC, and to evaluate the long-term neurological performance 12 months after TBI.
Methods: Between 01/1997 and 06/2001 all consecutive patients admitted with moderate or severe isolated TBI were enrolled in this study. They were treated according to the guidelines of the European Brain Injury Consortium, and the American Association of Neurosurgical Surgeons (AANS) for the management of severe TBI. Process and clinical data as well as every intervention were registered prospectively. The long-term neurological status was reassessed using the Glasgow Outcome Score (GOS) 12 months after TBI. Statistical comparison was performed using Mann-Whitney-U test, and multivariate testing by means of logistic regression analysis.
Results: Fifty-one (43 males, 8 females; median age 51.4 years) of 119 isolated TBI patients were included. Ten patients (8 males, 2 females; median age 38.4 years) underwent secondary extended or contralateral DC in their clinical course. Three of them (30%) died at a median of 1 day after revision respectively 6 days after TBI. According to univariate analysis, secondary DC significantly correlated with arterial hypotension ($p = 0.020$) and otorrhagia at admission ($p = 0.041$), skull base fracture ($p = 0.011$) and decreased maximum cerebral perfusion pressure (CPP; $p = 0.006$) after primary surgery. Multivariate analysis identified decreased maximum CPP as the only independent predictive parameter ($p = 0.036$) for secondary DC and unfavourable GOS after 12-months follow-up.
Conclusion: Arterial hypotension, otorrhagia at admission and skull base fractures are negatively influencing the mortality and morbidity of patients with isolated moderate or severe TBI. However, only decreased maximum CPP may independently indicate secondary DC after primary craniectomy in case of recurrent intracranial hypertension.

Key words: Traumatic brain injury; decompressive craniectomy; brain oedema; ICP; CPP; outcome

Introduction

Severe traumatic brain injury (TBI; Glasgow Coma Scale [GCS] score < 8 points) is one of the leading causes for disability and mortality in previously healthy adults. In the United States, about 200.000 of 1.5 million TBI patients per year suffer from severe TBI, which is survived by 80.000 to 90.000 patients with different degrees of physical handicap [37]. Mortality rates of severe TBI patients are reported between 37% and 60% despite improved non-operative treatment [25, 36].

Brain swelling may rapidly occur following moderate (GCS score 9-12 points) or severe TBI, which leads to a sudden increase of brain volume and an elevated intracranial pressure (ICP). Regions that were not directly traumatized are secondarily damaged by the negative effects on the cerebral blood flow [10, 20, 33]. The resulting decrease of the cerebral perfusion pressure (CPP) leads to further circulatory impairment with hypoxia of the brain tissue and increase of brain edema.

Sustained intracranial hypertension becomes clinically remarkable by dilated pupils, elevated blood pressure, bradycardia or respiratory insufficiency. Increased ICP values and constriction of the basal cisterns in the cerebral computed tomography (CCT) may indicate escalating brain swelling [19]. Several non-operative treatment options are recommended in case of increasing intracranial hypertension [19, 35]: intubation, mechanical ventilation, mild hyperventilation ($paCO_2$ ~ 35 mmHg); adequate analgesia sedation; 30°-head-up tilt; osmotic therapy with mannitol; volume therapy as well as use of vasopressors and/or positive inotropic substances for raising mean arterial pressure (MAP); maintenance of patient´s normothermia; blood glucose levels < 150 mg/dl; drainage of cerebrospinal fluid (CSF) via ICP catheter; use of barbiturates (methohexital, pentobarbital) and trometh-amine buffer.

However, irreversible collapse of brain circulation often appears very fast, so that no time remains for extended diagnostics and therapy [23]. If non-operative treatment fails in case of threatening constriction, early decompressive craniectomy (DC) with evacuation of epidural (EDH) or subdural hematoma (SDH) may be performed. But its effectiveness is controversially discussed, in particular after primary surgery. In contrast to non-operative treatment, DC did not show an improved long-term outcome in case of intracranial hypertension so far [1, 8, 11]. Definitions for indication and timing of secondary DC are still missing, since they were often determined by the surgeon due to personal experiences. Thus, the objectives of this study were to evaluate the incidence of increasing brain edema after primary trepanation, to identify independent predictors for urgent secondary DC, and to reexamine the neurological performance 12 months after trauma by means of Glasgow Outcome Score (GOS) in patients with moderate or severe TBI.

Patients and Methods

Study Design and Subjects

Between January 1997 and June 2001 all consecutive patients who were admitted with moderate (GCS score 9-12 points) or severe isolated TBI (GCS score ≤ 8 points) to our level 1 trauma center were enrolled in this study. Primary stabilization, operative treatment and postoperative care were provided according to the guidelines of the American Association of Neurosurgical Surgeons (AANS), the European Brain Injury Consortium and the German Society of Trauma Surgery for the management of severe TBI [2, 22, 30]. All subjects were initially examined by CCT, which was applied at admission or before surgery as well as 24 hours after admission or following every single operation. Reexaminations were performed due to increasing ICP values, neurological degradation or before ICU discharge. The following data were registered in a standardized protocol:

- process data, i.e. time of accident; time of admission; time of intubation; time of initial and repeated CCT; time of primary surgery and every additional operation; time of discharge; time of death.
- clinical data, i.e. GCS score; rate of respiration (RR); heart rate (HR); medium arterial pressue (MAP); systolic blood pressure (BP); central venous pressure (CVP); intracranial pressure (ICP); cerebral perfusion pressure (CPP) = difference between MAP and ICP; blood glucose level; CO2 saturation; otorrhagia; reaction of the pupils; CCT evidence of skull base fracture.
- interventions, i.e. intubation; initial and repeated CCT; CCT-guided ventriculostomy; operations; pentobarbital application; tromethamine application.

Indications and Operation Techniques

All patients were assigned to three different groups according to the initial CCT findings (Table 1):

- patients *without* operation, who were provided with a CCT-guided ventriculostomy for ICP monitoring and CSF drainage.
- patients *with* operation, but without ICP monitoring.
- patients *with* operation, who were provided with CCT-guided ventriculostomy for ICP monitoring and CSF drainage.

Relative indications for primary DC were defined for: SDH or EDH greater than the full thickness of the adjacent calvarium; midline shift greater than the clot thickness or volume; intraventricular hemorrhage; isolated intracerebral hemorrhage (ICH); small petechial bleedings with diffuse axonal injury; impression fractures (open or with substantial mass shift, without sinus participation). *Absolute indications* for primary and secondary DC were: clinical degradation and/or dilation of one or both pupils still responsive to light; diffuse, uni- or bilateral increasing brain edema in CCT; midline shift > 5 mm; volume of the hematoma > 10 ml in case of extracerebral hemorrhage, > 25-30 ml in case of intracerebral hemorrhage; obliteration of the basal cisterns or the third ventricle; elevated or therapy-resistant ICP increase > 25 mmHg and/or CPP reduction < 45 mmHg.

In case of abnormal unilateral findings, DC was performed in terms of a wide frontotemporoparietal craniectomy using a curvilinear skin incision. With the exception of EDH without intraoperative signs of brain edema an intraventricular catheter was usually inserted at the contralateral side for permanent ICP monitoring and/or CSF drainage. In case of bilateral pathologies, DC was performed on both sides after an arcuated skin incision dorsally of both parietal tubers leaving a bony rim on top of superior sagittal sinus.

Follow-up 12 months after Trauma and Statistical Analysis

Twelve months after trauma the postoperative course, complaints and neurological status were recorded for each patient according to the GOS score: 1 = "dead", 2 = "vegetative state", 3 = "severe disabilitiy", 4 = "moderate disability", and 5 = "good recovery" [6, 18].

All demographic data are represented as median and interquartile ranges (25% and 75% percentiles). Statistical comparison was performed by means of Mann-Whitney-U test (SPSS version 11.5, SPSS GmbH, Munich, Germany). Non-parametric correlations were established using the Spearman rank analysis. Independent parameters were determined by means of logistic regression analysis. Statistical significance was set at P values less than 0.05.

Results

Demographic Characteristics of the Study Group

Sixty-five of 119 consecutive patients (54,6%) suffering from isolated moderate or severe TBI underwent uni- or bilateral DC with insertion of an intraventricular catheter (Table 1). Statistical analysis was not possi-

Table 1. Interventions and operations of 119 patients with moderate and severe traumatic brain injury in relation to initial CCT diagnosis.

	Total (n = 119)	**Sub-arachnoid hemorrhage (n = 10)**	**Epidural hematoma (n = 11)**	**Subdural hematoma (n = 44)**	**Intracranial hemorrhage (n = 22)**	**Depressed skull fracture (n = 3)**	**Cerebral edema (n = 29)**
ICP monitoring without operation	52	10	0	0	19	1	22
Operation without ICP monitoring	2	0	0	2	0	0	0
Operation with ICP monitoring	65	0	11	42	3	2	7
Study group with	**51**	**0**	**9**	**33**	**2**	**2**	**7**
Primary DC	41	0	9	28	2	2	2
Primary and secondary DC	10	0	0	5	0	0	5
Not assessed	14	0	2	9	1	0	2

ICP = intracranial pressure; DC = decompression craniectomy; CCT = cerebral computed tomography.

Table 2. Demographic data and time course in patients with primary (n = 41) and primary + secondary decompression craniectomy (DC; n = 10).

Parameter	**Study group (n = 51)**	**Primary DC (n = 41)**	**Primary + secondary DC (n = 10)**	**P value**
Age [years]	51.4 (34.0 - 60.4)	54.1 (30.2 - 64.4)	38.4 (29.2 - 56.3)	0.125
Gender [male : female]	43 : 8	36 : 5	8 : 2	0.820
ASA classification	2 (1 - 3)	2 (1 - 3)	1 (1 - 2)	< 0.01
Time course:				
Trauma - Admission [min]	**45.0 (27.0 - 66.4)**	**34.0 (24.3 - 56.6)**	**80.0 (60.0 - 100.0)**	**0.007**
Admission - CCT [min]	18.0 (15.0 - 28.0)	17.0 (13.0 - 31.5)	19.5 (9.5 - 25.5)	0.388
Trauma - CCT [min]	71.0 (48.4 - 90.3)	67.5 (47.0 - 86.4)	94.2 (66.8 - 120.5)	0.060
CCT - Start of operation [min]	61.0 (38.0 - 96.0)	59.5 (39.0 - 101.3)	64.0 (49.0 - 95.0)	0.745
Trauma - Death [days]	6.0 (1.5 - 9.0)	6.0 (0.0 - 10.0)	6.0 (5.0 - 8.5)	0.820
Trauma - Discharge [days]	**18.0 (7.0 - 28.0)**	**12.0 (7.0 - 22.5)**	**30.0 (20.0 - 38.0)**	**0.032**

DC = decompression craniectomy; CCT = cerebral computed tomography; P value = significance according to Mann-Whitney-U test between patients with primary DC and primary + secondary DC.

ble in 14 patients (10 males, 4 females; median age 45.0 years [32.0-58.0]) due to incorrect data acquisition or non-accessibility for reexamination.

Thus, 51 patients (43 males, 8 females; median age 51.4 years [34.0-60.4]) were included into the present study (Table 2). There were no significant differences between the non-assessed patients and the study group concerning age, gender, initial GCS score as well as type and severity of intracranial lesion. Twenty-two patients were involved in a traffic accident, 11 patients in a fall, and 2 patients in a street fight. The mechanism of accident could not be determined in 16 cases.

Pre- and In-hospital Course of the Study Group

On arrival of the EMS team, the median GCS score was 8 points (3-12), systolic BP 115 mmHg (100-140), MAP 100 mmHg (90-110), HR 90 beats/ min (72-99), RR 12 respirations/min (10-16) and the O_2 saturation 95% (85-99; Table 3). Twenty-one patients had to be intubated on scene. Their pupils were circular, central and equal in size. Only 7 patients showed an adequate constriction and equal accommodation of the pupils to light. Three these 25 patients exhibited anisocoric or dilated pupils with poor response to light stimulation.

The study patients were admitted to our hospital 45.0 min (27.0-66.4) after trauma. Ten patients were intubated immediately after admission, the remaining 20 patients before surgery. The median GCS score at admission was 4 points (3-10), MAP 100 mmHg (90-115).

Eighteen minutes later, patients were transferred to the CCT (Table 2). Skull base fractures occurred in 22 of 23 cases with otorrhagia. An isolated EDH was di-

Table 3. Pre- and in-hospital data of patients with primary (n = 41) and primary + secondary decompression craniectomy (DC; n = 10).

Parameter	Study group (n = 51)	Primary DC (n = 41)	Primary + secondary DC (n = 10)	P value
On scene:				
GCS score [points]	8 (3 - 12)	6 (3 - 12)	8 (5 - 12)	0.425
ystolic BP [mmHg]	115 (100 - 140)	120 (100 - 140)	125 (105 - 130)	0.560
Maximum MAP [mmHg]	100 (90 - 110)	105 (90 - 110)	100 (95 - 125)	0.650
Heart rate [1/min]	90 (72 - 99)	85 (70 - 100)	95 (80 - 105)	0.384
Rate of respiration [1/min]	12 (10 - 16)	12 (10 - 15)	12 (8 - 20)	0.675
O2 saturation [%]	95 (85 - 99)	94 (85 - 98)	95 (96 - 99)	0.910
Intubation [yes : no]	21 : 30	17 : 24	4 : 6	0.588
At admission:				
Otorrhagia [yes : no]	**23 : 28**	**15 : 26**	**8 : 2**	**0.041**
Anisocoria [yes : no]	3 : 48	1 : 40	2 : 8	0.058
Irregular pupils [yes : no]	2 : 49	1 : 40	1 : 9	0.510
Fixed pupils [yes : no]	7 : 44	5 : 36	2 : 8	0.704
GCS score [Punkte]	4 (3 - 10)	4 (3 - 10)	5 (3 - 10)	0.784
Skull base fracture in CCT [yes : no]	**22 : 29**	**13 : 28**	**9 : 1**	**0.014**
Systolic BP [mmHg]	**120 (105 - 140)**	**130 (100 - 160)**	**90 (95 - 120)**	**0.011**
Maximum MAP [mmHg]	100 (90 - 115)	100 (90 - 120)	85 (78 - 105)	0.098
After surgery:				
Maximum MAP [mmHg]	106 (90 - 115)	108 (98 - 117)	100 (80 - 120)	0.255
Maximum CVP [mmHg]	8 (6 - 10)	8 (6 - 11)	7 (5 - 14)	0.940
Postoperative ICP [mmHg]	**10 (7 - 16)**	**8 (5 - 14)**	**18 (12 - 30)**	**0.016**
Maximum ICP [mmHg]	22 (16 - 30)	22 (16 - 26)	24 (20 - 30)	0.682
Maximum CPP [mmHg]	**76 (70 - 88)**	**90 (86 - 110)**	**67 (60 - 78)**	**0.006**
Pentobarbital [yes : no]	**12 : 39**	**3 : 38**	**9 : 1**	**<0.001**
Tromethamine buffer [yes : no]	**5 : 46**	**1 : 40**	**5 : 5**	**0.014**

DC = decompression craniectomy; CCT = cerebral computed tomography; P value = significance according to Mann-Whitney-U test between patients with primary DC and primary + secondary DC.

agnosed in 5 cases, SDH in 11 cases and ICH in 4 cases, partially with a depressed skull fracture. In each case, a significant midline shift > 5mm was determined. Combined injuries were present in the remaining 31 patients (Table 1). Primary DC followed by insertion of a ventricular catheter for ICP monitoring started 61.0 min (38.0-96.0) after CCT. Forty-three patients were provided with dural grafting and bone flap removal, 8 patients with bone flap replacement. Bone fragments were elevated and dura lesions were closed in 3 cases with depressed skull fracture. At the end of the operation, the median ICP of the study group amounted to 10 mmHg (7-16).

Postoperative Course of the Study Group

During postoperative treatment of intracranial hypertension, the study patients presented a maximum ICP of 22 mmHg (16-30), CPP of 76 mmHg (70-88), and MAP of 106 mmHg (90-115). Twelve subjects required additional pentobarbital application for lowering ICP (Table 3).

At a median of 6.0 days (1.5 - 9.0) after trauma, 16 of 51 study patients (31.4%) died due to irreversible intracranial hypertension, which was regularly confirmed by autopsy (Table 2). Initial CCT diagnosis of these patients was EDH and extensive brain edema in 2 cases, respectively, SDH in 14 cases. Thirty-five patients were discharged from the hospital 18.0 days (7.0-28.0) after the primary surgery. At this time, 2 patients offered a GOS of 2 points, 8 patients of 3 points, 13 patients of 4 points and 12 patients of 5 points. Twelve months later, neurological reexamination revealed an improved GOS in most cases. One patient exhibited still 2 points, 4 patients 3 points, 6 patients 4 points and 24 patients even 5 points.

Pre- and Intraoperative Course of Patients Undergoing Secondary DC

Ten (8 males, 2 females; median age 38.4 years [29.2 - 56.3]) of 51 patients (19.6%) underwent secondary either ispilateral or contralateral DC (Table 2). Primary cause was a traffic accident in 9 cases; the mechanism of TBI could not be clarified in one case. On scene, there were no significant differences concerning GCS score, MAP, systolic BP, HR, RR, the CO2 saturation and the rate of intubation in comparison those subjects with primary DC (Table 3).

These patients were admitted significantly later (80.0 min [60.0 - 100.0]) after trauma than those with primary DC (Table 2). With exception of systolic BP ($p = 0.011$), otorrhagia ($p = 0.041$) and skull base fracture ($p = 0.014$) at admission, there were no significant differences between both groups concerning GCS score and MAP (Table 3). In all cases, initial DC was followed by dural grafting, bone flap removal and contralateral insertion of intraventricular catheter for ICP monitoring and CSF drainage. The immediate postoperative ICP at 18 mmHg (12 - 30) was significantly higher ($p = 0.016$) than in those patients with primary DC.

Postoperative Course of Patients Undergoing Secondary DC

Secondary DC had to be performed at a median period of 2 days (1 - 8) after primary surgery. Three patients underwent ipsilateral, 7 patients contralateral DC. Maximum ICP level prior to secondary DC was not substantially higher than in those patients with primary DC (Table 3). However, the CPP level at a median of 67 mmHg (60 - 78) was significantly decreased ($p = 0.006$) despite catecholamine application. Moreover, pentobarbital ($p = <0.001$) and tromethamine ($p = 0.014$) were used more frequently than in those cases with primary DC.

At a median of 1 day (1 - 4.6) after secondary DC and 6 days (5 - 8.5) after trauma, 4 of 10 patients (40.0%) died due to irreversible intracranial hypertension and constriction, which was confirmed by autopsy in each case (Table 2). Two of these deceased subjects suffered from SDH and extensive brain edema, respectively. The remaining 6 patients could be discharged from hospital to the neurological rehabilitation program 30 days (20 - 38) after trauma. Their length of hospital stay was significantly longer ($p = 0.032$) compared to those patients with primary DC. At discharge, one patient each offered a GOS of 2 or 3 points, respectively, and 4 patients a GOS of 4 points. Twelve months after trauma, neurological reexamination revealed an improved GOS of 4 points in 2 patients and 5 points in 3 patients. Only one patient with a GOS of 2 points at discharge did not tend to neurologically improve.

Correlation Analysis of Clinical Parameters for Secondary DC

On scene, there were no significant correlations between clinical findings and the urgency for secondary DC. The time between trauma and admission significantly correlated with the clinical course (Table 4). However, it could not prove as an independent parameter according to the multivariate analysis.

Systolic BP and otorrhagia at admission as well as maximum CPP and ICP levels after primary DC were found to be relevant for the prediction of recurrent intracranial hypertension (Table 4). But only maximum CPP could prove as an independent parameter according to both Spearman correlation and logistic regression analysis (score 9.366; $df = 1$; $p = 0.036$). Finally, applications of pentobarbital as well as tromethamine buffer significantly correlated with the urgency of secondary DC, but could not be confirmed as independent parameters according to the multivariate analysis (Table 4).

Discussion

Decompressive craniectomy (DC) is discussed controversially in patients with increasing intracranial hypertension. So far, surgery could not prove to be superior to non-operative management [1, 8]. Beyond that, former clinical studies are not conclusive concerning the indications to surgical treatment.

Table 4. Spearman rank analysis for significant parameters predicting secondary DC.

	Primary DC (n = 41)	**Primary + secondary DC (n = 10)**	**R**	**P value**
Trauma – Admission time [min]	34.0 (24.3 – 56.6)	80.0 (60.0 – 100.0)	0.520	0.007
Trauma – Discharge time [days]	12.0 (7.0 – 22.5)	30.0 (20.0 – 38.0)	0.380	0.032
Systolic BP [mmHg] at admission	130 (100 – 160)	90 (95 – 120)	-0.344	0.020
Otorrhagia at admission [yes : no]	15 : 26	8 : 2	0.306	0.041
Skull base fracture [yes : no]	13 : 28	9 : 1	0.468	0.011
Maximum postoperative CPP [mmHg]	90 (86 – 110)	67 (60 – 78)	-0.602	0.006
Pentobarbital administration [yes : no]	3 : 38	9 : 1	0.688	<0.001
Tromethamine administration [yes : no]	1 : 40	5 : 5	0.445	0.014

R = correlation coefficient; P value = significance between patients with primary DC and primary + secondary DC.

High mortality rates with a large variance between 13.5% and 90% seem to argue against the effectiveness of DC [8, 11]. Most studies are based on retrospectively collected data, only a few but not randomized trials are prospectively designed. Moreover, the evaluation of results is difficult because many different operation techniques, e.g. unilateral, bilateral, bifrontal and expanded frontottemporoparietal DC, are described. The patient numbers vary between 2 [9] and 533 patients [15], and the heterogeneity of the patient population within a study group [17] as well as between the individual groups raises difficulties concerning data extrapolation. In the last 10 years, only 4 studies were published about more than 10 patients undergoing primary frontotemporoparietal DC [11, 12, 14, 24]. All other studies are based on individual case reports about TBI management and ICU treatment.

Munch et al. presented the effect of DC on the outcome of TBI patients in correlation to the ICP values and the increase of intracranial space calculated from CCT scans. The distance between the bottom edge of the temporal craniectomy and the surface of the medium cranial fossa seems to have a greater influence on the clinical outcome than the size of the craniectomy [22].

In experimental studies most authors agree that DC leads to a rapid ICP decrease. In particular after opening of the dura, the oxygen partial pressure of the tissue increased without impairment of the blood-brain barrier, and the cerebral blood flow returned to normal [3, 27]. Lower mortality rates were demonstrated after DC compared to the control group [21]. Nevertheless, some experimental investigations have observed growing brain edema with hemorrhagic insults and cortical necroses after herniation due to DC as well, especially in case of simultaneous arterial hypertension [7, 13].

It remains still unclear whether the risk of additional brain damage is increased because of intracranial mass shift after DC. Obviously, postoperative brain edema will accelerate in case of irreversible brain damage prior to surgery. However, this should not be an argument against DC, but against late indication [12]. Thus, the objective of this analysis was to determine early significant criteria, which might indicate expanded ipsilateral or contralateral DC following primary operation.

The systolic blood pressure at admission proved as a significant, but not independent clinical parameter due to correlation analysis. On scene, the arterial hypotension considerably affects the morbidity of severe TBI patients [5]. Rapid elevation of the systolic blood pressure appears to be essential already during the prehospital phase [34]. Therefore, the additional administration of catecholamines should be considered for longer transportation times. However, this hypothesis must be scrutinized in a treatment study with appropriate control groups.

Initial otorrhagia turned out to be clinically relevant, but did not independently indicate recurrent intracranial hypertension after primary DC. Nevertheless, it may suggest not only a traumatic osseous injury of the skull base, but also an acute and life-threatening intracranial lesion [32].

Maximum CPP below a median threshold of 70 mmHg proved to be the only independent, prognostic parameter, which predicts the necessity of secondary DC, especially in case of pentobarbital or tromethamine administration. Application of pentobarbital may cause decreased medium blood pressure and CPP levels leading to rapidly increasing dosages of catecholamines.

Hereby, the absolute ICP value is not as important as its effect on the CPP value and its relationship to brain swelling, although the effects of postoperatively increased ICP on the cellular metabolism are to be related not only to CPP [4]. The CPP decrease and diffuse brain swelling are fundamental mechanisms of secondary brain damage following severe TBI [16, 26, 29]. The perfusion of injured brain areas can be absolutely reduced due an increase of local tissue pressure and loss of autoregulation. In these cases, the CPP as well as the ICP would overestimate the local perfusion status, since the ICP reflects the pressure of the entire cranial cavity [31]. Beyond that, elevated ICP values at adequate CPP levels may also occur with benign intracranial hypertension, but without neurological deficits.

Most protocols are tending to treat both increased ICP as well as decreased CPP values because of their effects on the clinical outcome. Decreasing CPP can initiate a cascade of vasodilative mechanisms in order to maintain the cerebral blood flow [28]. The resulting increase of the cerebral blood volume leads to a further rising of the ICP and to a reduction of the CPP. This cascade can be interrupted by raising the CPP, which subsequently leads to reduced ICP [26]. In the present analysis, only the CPP value and the administration of pentobarbital / tromethamine, but not the absolute ICP value were significantly correlated with the GOS score 12 months after trauma.

The selective composition of the study group has to be mentioned as one of the limitations of the study and the data interpretation. Fifteen of 73 patients with primary operation and ICP monitoring could not be included into this analysis for statistical reasons. However, there were no significant differences between the non-included patients and the study group concerning age, gender, initial GCS score as well as type and severity of intracranial lesion. The absence of an appropriate comparison group is another limiting factor. Finally, this analysis represents a statistical retrospective calculation of prognostic parameters predicting secondary DC during the course of a prospective study. However, the basis is established with the present data to examine the prognostic value of arterial hypotension on scene and the CPP below a threshold value of 70 mmHg in a larger number of patients with moderate or severe TBI.

Conclusions

Apart from the time between trauma and admission, arterial hypotension as well as otorrhagia at admission significantly correlated with the morbidity and mortality of patients suffering from moderate or severe TBI. The early equilibrium of arterial hypotension appears to be essential in the prehospital management of TBI

patients, but must be examined in further treatment studies.

Decreased CPP value below a median threshold of 70 mmHg proved as the only independent predictive parameter for secondary DC and unfavorable 12-months follow-up in case of recurrent intracranial hypertension after primary DC. In contrast, the type and severity of the intracranial lesion, the absolute ICP value as well as the pentobarbital and/or tromethamine buffer administration are not meaningful.

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