

## Decompressive craniectomy for acute subdural haematoma: An overview of current prognostic factors and a discussion about some novel prognostic parameters

Murat Kalayci,<sup>1</sup> Erol Aktunç,<sup>2</sup> Sanser Gül,<sup>3</sup> Volkan Hanci,<sup>4</sup> Nurullah Edebalı,<sup>5</sup> Ferda Çağavi,<sup>6</sup> Bektas Açıköz<sup>7</sup>

### Abstract

**Objective:** To identify specific factors that can be used to predict functional outcome and to assess the value of decompressive craniectomy in patients with acute subdural haematoma.

**Methods:** The retrospective study was done at the Zonguldak Karaelmas University Practice and Research Hospital, Turkey, and included 34 trauma patients who had undergone decompressive craniectomy for acute subdural haematoma from 2001 to 2009. At the 30th day of the operation, the patients were grouped as survivors and non-survivors. Besides, based on their Glasgow Outcome Scale, which was calculated 6 months post-operatively, the patients were divided into two functional groups: favourable outcomes (4-5 on the scale), and unfavourable outcomes (1-3 on the scale). The characteristics of the groups were compared using SPSS 15 for statistical analysis.

**Results:** One-month mortality was 38.2% (n=13) and 6-month total mortality reached 47% (n=16). Patients with higher pre-operative revised trauma score, Glasgow coma scale, partial arterial pressure of carbon dioxide, arterial oxygen pressure, Charlson co-morbidity index score, blood glucose level, blood urea nitrogen, and lower age had a higher rate of survival and consequently a favourable outcome. Higher platelet values were only found to be a determinant of higher survival at the end of the first month without having any significant effect on the favourable outcome.

**Conclusion:** In patients of traumatic acute subdural haematoma whose Glasgow coma scale on arrival was  $\leq 8$ , a massive craniectomy along with the evacuation of the haematoma, may be considered as a treatment option for intra-operative and post-operative brain swelling. But in patients with a score of 3 on arrival and bilaterally fixed and dilated pupils, decompressive craniectomy is unnecessary.

**Keywords:** Acute subdural haematoma, Trauma, Decompressive craniectomy, Outcome. (JPMA 63: 38; 2013)

### Introduction

Traumatic acute subdural haematoma (SDH) continues to have high morbidity and mortality rates despite the advent of rapid transportation, computed tomography (CT) scanning, intracranial pressure monitoring and intensive care management.<sup>1,2</sup> Outcome for these patients may be influenced mainly by the underlying brain injury than the SDH itself.<sup>3</sup>

Despite studies on the impact of early craniectomy (EC) in traumatic acute SDH,<sup>4,5</sup> the value of primary decompressive craniectomy (DC) remains uncertain. To our knowledge, there are only two studies comparing mortality rates between normal craniotomy and DC.<sup>3,6</sup> In a group of 180 patients, one study reported a higher mortality rate in DC than EC.<sup>3</sup> However, the study had some pitfalls, as the two groups were not adjusted for age and Glasgow coma scale (GCS), or the presence of the

signs of herniation. Consequently, subjects undergoing DC have had a higher rate of herniation and mortality than the subjects in the other group.<sup>3</sup>

The other study reviewed a group of 34 patients having acute SDH.<sup>6</sup> The study performed DC in severe patients having significant brain oedema, while craniotomy was performed in less severe patients without any clinically devastating brain oedema.<sup>6</sup> The study failed to find any favourable neurological outcome in patients undergoing DC seemingly due to a randomisation pitfall.<sup>6</sup>

Our aim in the present study was to assess the value of DC in acute SDH. We sought to assess whether the surgical procedure conferred any increase in short-term survival rates and long-term favourable outcomes defined by the Glasgow outcome scale (GOS) score in the study population. Special emphasis was placed on the relationship between patient characteristics and functional outcome to ascertain patient selection criteria that may ensure better surgical results in the future.

### Patients and Methods

In this retrospective cross-sectional study, records of 34

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<sup>1,3,5,7</sup>Departments of Neurosurgery, <sup>2</sup>Department of Family Medicine,  
<sup>4</sup>Department of Anesthesiology, Zonguldak Karaelmas University Medical  
 School, <sup>6</sup>Department of Neurosurgery Kocaeli Acibadem Hospital, Turkey.

**Correspondence:** Murat Kalayci. Email: drkalayci@yahoo.com

patients were analysed who had undergone DC for acute SDH from 2001 to 2009 at the Zonguldak Karaelmas University Practice and Research Hospital in Zonguldak, Turkey. Following the establishment of airway patency and appropriate fluid resuscitation, patients having significant acute SDH in CT were immediately operated for decompression.

Data about the demographical characteristics such as age and gender, the history and clinical findings such as mechanism of injury, the time duration from the traumatic event until surgical decompression, the hemispherical location of the haematoma, the presence of extracranial injury, systolic and diastolic blood pressure, the pupillary reactivity and the signs of herniation (unilateral or bilateral pupil dilatation) were recorded.

Laboratory data such as the presence of midline shift at the level of septum pellucidum in CT, complete blood count, full biochemistry and arterial blood gas measurements were also recorded at admission to the emergency department.

Complete blood count tests included haemoglobin (normal value 12-18 mg/dl), white blood cell count (normal value 4800-10800/mm<sup>3</sup>) and platelet (PLT) count (normal value 130.000-400.000/mm<sup>3</sup>).

Full biochemistry included blood glucose level (GLU) (normal value 70-110 mg/dl), blood urea nitrogen (BUN) (normal value 10-50 mg/dl), creatinine (CRE) (normal value 0.5-1.2 mg/dl), sodium (Na) (normal value 135-157 mEq/l), potassium (K) (normal value 3.5-5.5 mEq/l), calcium (Ca) (normal value 8.4-10.2 mEq/l), chlorine (Cl) (normal value 98-110 mEq/l), aspartate aminotransferase (AST) (normal value <40 IU.L-1), alanine aminotransferase (ALT) (normal value <40 IU.L-1), alkaline phosphatase (ALP) (Normal value 40-129 IU.L-1), gamma glutamil transferase (GGT) (Normal value <73 IU.L-1 in males; <38 IU.L-1 in females), lactate dehydrogenase (LDH) (normal value 120-146 U.L-1), creatine kinase (CK) (normal value 32-294 U.L-1 in males; 33-211 U.L-1 in females), creatine kinase-MB (CKMB) (normal value 0.6-6.3 ng/ml), total protein (Normal value 6.4-8.3 gr/dl), and albumin (normal value 3.2-4.8 gr/dl).

Arterial blood gas measurements included pH (normal value 7.35-7.45), partial arterial pressure of carbondioxide (PaCO<sub>2</sub>) (normal value 35-45 mmHg), partial arterial pressure of oxygen (PaO<sub>2</sub>) (normal value 80-100 mmHg), and arterial oxygen saturation (SO<sub>2</sub>) (normal value >90%).

Table-1: Revised Trauma Score.

Glasgow Coma Scale (GCS)	Systolic Blood Pressure (SBP)	Respiratory Rate (RR)	Coded Value
13-15	>89	10-29	4
9-12	76-89	>29	3
6-8	50-75	6-9	2
4-5	1-49	1-5	1
3	0	0	0
$RTS=0.9368 \text{ GCS} + 0.7326 \text{ SBP} + 0.2908 \text{ RR}$			

Table-2: Charlson Co-morbidity Index Scoring System.

Score	Condition
1	Myocardial infarction (history, not ECG changes only) Congestive heart failure Peripheral vascular disease (includes aortic aneurysm $\geq 6$ cm Cerebrovascular disease: CVA with mild or no residua OR t?a Dementia Chronic pulmonary disease Connective tissue disease Peptic ulcer disease Mild liver disease (without portal hypertension, includes chronic hepatitis) Diabetes without end-organ damage (excludes diet-controlled alone)
2	Hemiplegia Moderate or severe renal disease Diabetes with end-organ damage (retinopathy, neuropathy, or brittle diabetes) Tumor without metastases (excludes if >5 y from diagnosis) Leukaemia (acute or chronic) Lymphoma
3	Moderate or severe liver disease
6	Metastatic solid tumour AIDS (not just HIV positive)

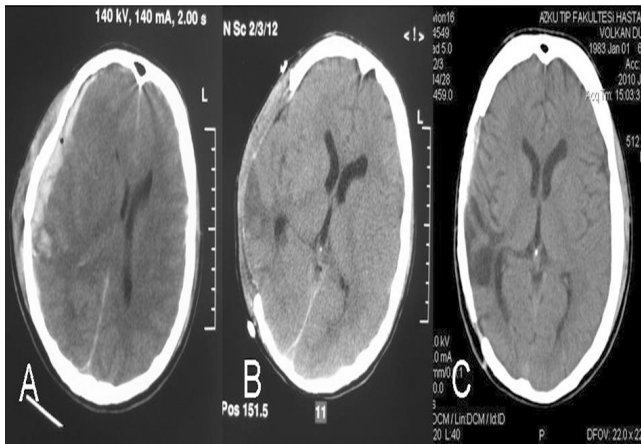
Note: For each decade > 40 years of age, a score of 1 is added to the above score.

ECG: Electrocardiogram; CVA: Cerebrovascular accident; TIA: Transient ischaemic attack; AIDS: Acquired immunodeficiency syndrome; HIV: Human immunodeficiency virus.

Patients were preoperatively evaluated for GCS, Revised trauma score (RTS) (Table-1),<sup>7</sup> Charlson co-morbidity index score (CCIS) (Table-2)<sup>8</sup> and GOS.<sup>9</sup>

Therapeutic measures such as the duration for intravenous infusion of mannitol solution, the need and the duration for thiopental infusion and mechanical ventilation were recorded.

A unilateral or bilateral fronto-temporo-parietal craniectomy was performed depending on the extent and location of SDH with a question mark-shaped skin flap based on the ear. The bone flap was approximately 15cm in diameter. The temporal bone was craniectomised upto the base of the skull to maximise the extent of decompression at the level of the perimesencephalic cisterns. The dura was opened in



**Figure-1:** A- Cerebral axial computed tomographic scan of a 22-years-old patient, depicting an acute subdural haematoma due to traffic accident with a 12-mm shift to the left, obliterating the basal cisterns. B- He had immediate decompressive craniectomy. C- The patient was neurologically intact at 6th month following discharge.

stellate fashion and then haematoma was evacuated. The cortical surface of the swollen brain was protected against the sharp edges of the cut dura. Following the decompression, autologous pericranial layer was spread over the brain instead of a watertight duraplasty. The removed bone flap was then placed in a subcutaneous pocket overlying the abdomen for subsequent cranioplasty. The continuous sedation was tapered according to the needs of each patient and withheld on day five or six of the surgical decompression. Post-operatively, patients had at least one CT. If and when the patient recovered sufficiently, the preserved bone flaps were replaced or methyl methacrylate cranioplasties were performed (Figure-1).

At the 30th day of the operation, the patients were grouped as survivors and non-survivors and were compared for their demographical, history-related and clinical parameters as well as laboratory test results. The factors affecting the survival rate were evaluated.

At the end of the 6th month, the surviving patients were re-classified into favourable and unfavourable outcome groups according to their GOS in order to disclose the factors affecting long-term physical disabilities. The favourable outcome was defined as GOS 4 (moderate disability) and 5 (good recovery including independent daily living activity), while unfavourable outcome was defined as GOS 1 (death), 2 (vegetative state) or 3 (severe disability).

Statistical analysis was carried out using SPSS 15. Continuous variables were presented as means  $\pm$  standard deviation, while categorical variables were

presented in terms of frequency and percentage. P values less than 0.05 were considered to be significant. Mann Whitney U test was used to compare the continuous variables, whereas the Chi-square test was used for comparing categorical variables. Pearson's correlation analysis was used to disclose factors affecting survival and favourable outcome.

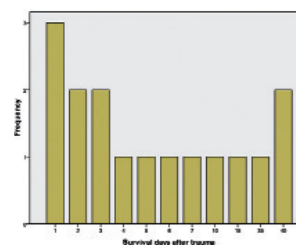
Life table analysis was performed using 'Survival days following surgery' as the dependent variable. The maximum life span was determined as 180 days (6 months) with an interval of 10 days. The status variable was selected as GOS at the end of the 6th month.

A cox regression analysis was conducted in order to disclose any indicators affecting the mortality rate. The relative risks were calculated.

## Results

The study group consisted of 26 (76.5%) males and 8 (23.5%) females, with the mean age being  $37.2 \pm 24.3$  years, ranging between 2 and 81 years. The mechanisms of injury were traffic accidents in 17 (50.0%); fall in 13 (38.2%) and blunt injury due to occupational accidents in 4 (11.8%) patients. Left hemispheric injury was found in 18 (52.9%); right hemispheric injury in 11 (32.4%); and bilateral hemispheric injury in 5 (14.7%) patients.

Acute SDH was the only injury in 26 (76.5%) patients, while the remaining 8 (23.5%) had accompanying extracranial injuries. The extracranial injuries were classified as intra-abdominal bleeding in 2 (5.9%); pneumothorax in 2 (5.9%); traumatic enucleation of the eye in 2 (5.9%); cervical vertebral fracture in 1 (2.9%); and thoracic vertebral fracture in 1 (2.9%) patient. Pre-operative median GCS was 7, ranging from 3 to 13, and



**Figure-2:** The distribution of survival time for the cases.

Table-3: Comparison of the demographical and clinical parametres between the survivors and the non-survivors at the 30th day of the operation.

		Survivors (n = 21)	Nonsurvivors (n = 13)	p
Age		28.6±18.5	51.2±26.9	0.013
Gender	Female	7 (33.3%)	1 (7.7%)	0.094
	Male	14 (66.7%)	12 (92.3%)	
Mechanism of injury	Fall	9 (42.9%)	4 (30.8%)	0.559
	TA	9 (42.9%)	8 (61.5%)	
	Occupational	3 (14.3%)	1 (7.7%)	
Time duration until surgery (hours)		5.1±2.4	5.5±2.5	0.701
Localisation of injury	Left hemispheric	11 (52.4%)	7 (53.8%)	0.457
	Right hemispheric	8 (38.1%)	3 (23.1%)	
	Bilateral	2 (9.5%)	3 (23.1%)	
Extracranial injury	Present	6 (28.6%)	2 (15.4%)	0.328
	Absent	15 (71.4%)	11 (84.6%)	
SAP**		111.2±30.1	88.5±54.7	0.065
DAP**		67.8±22.6	51.8±36.4	0.089
Pupillary reactivity	Bilaterally dilated and fixed	3	9	0.002
	Unilaterally dilated	9	0	
	Isochoria	9	4	
Preoperative GCS***		7 (3-13)	5 (3-12)	0.193
RTS***		8 (4-12)	7 (1-11)	0.042
CCIS***		3 (0-12)	5 (0-12)	0.002
PLT (total/mm <sup>3</sup> X10 <sup>3</sup> )		277.3±97.5	197.5±102.0	0.022
GLU (mg.dl <sup>-1</sup> )		145.2±51.4	266.3±106.7	<0.001
BUN		29.3±9.7	37.8±6.5	0.005
PaO <sub>2</sub>		159.7±60.5	72.0±16.6	<0.001
SO <sub>2</sub>		95.9±4.4	91.1±4.5	0.005

\*p<0.05, \*\*mean ±SD, \*\*\*median (Range).

TA: Traffic accident; SAP: Systolic arterial pressure; DAP: Diastolic arterial pressure; GCS: Gasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson comorbidity index score; PLT: Platelet; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

the mean midline shift in CT was 8.7±4.2mm, ranging from 2 to 20mm.

By the 30th day of the operation, 13 (38.2%) patients were dead and 21 (61.8%) had survived (Figure-2). The survival rate at this point of time did not differ according to gender, mechanism of injury, the time duration from the traumatic event until surgery, hemispheric location of the injury, the presence of extracranial injury, or the level of systolic and diastolic blood pressure at admission to the emergency department. However, the age and the presence of the signs of herniation differed significantly between the survivors and the non-survivors (Table-3).

All of the patients with unilaterally dilated pupils (n=9) survived. Pre-operative GCS did not differ among the survivors and the non-survivors at the 30th day of the surgery. All the 3 patients scoring 3 in pre-operative GCS were dead. Four of the 5 patients scoring 4 in pre-operative GCS were dead and the remaining 1 patient scored GOS 2 at the 6th month (Tables 4 and 5). However, the survivors scored significantly higher RTS

and lower CCIS.

Among the laboratory values at admission, PLT, GLU, BUN, PaO<sub>2</sub> and SO<sub>2</sub> values differed significantly between the survivors and the non-survivors. The rest

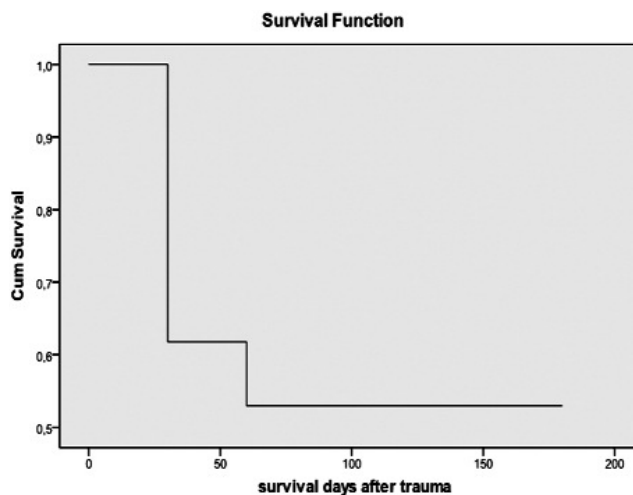


Figure-3: The survival function by days following trauma.

Table-4: Patient characteristics in the favourable outcome group of patients at the end of the 6th month.

Patient	Age (years)	Gender (M/F)	Survival Days After Trauma (Days)	Pre-operative GCS	Mechanism of injury	Hemispheric location of the injury	Midline Shift (MM)	Time between trauma and operation (hours)
23	21	M	180	6	TA	Right	8	6
24	40	M	180	12	TA	Left	2	3
25	6	F	180	9	Fall	Left	6	2
26	27	M	180	8	BT	Left	3	8
27	22	F	180	7	Fall	Right	10	3
28	33	M	180	11	Fall	Both	3	5
29	41	M	180	6	Fall	Left	12	3
30	52	M	180	5	TA	Right	10	4
31	3	F	180	13	Fall	Right	11	3
32	22	M	180	6	BT	Left	14	4
33	21	M	180	10	Fall	Right	6	12
34	2	F	180	8	Fall	Right	15	4

TA: Traffic accident, BT: Blunt trauma due to occupational accidents, GCS: Glasgow Coma Scale. M=Male, F=Female.

Table-5: Patient characteristics in the unfavourable outcome group of patients at the end of the 6th month.

Patient	Age (years)	Gender (M/F)	Survival Days After Trauma (Days)	Pre-operative GCS	Mechanism of injury	Hemispheric location of the injury	Midline Shift (MM)	Time between trauma and operation (hours)
1	77	F	1	4	Fall	Left	14	4
2	48	M	10	4	TA	Left	10	5
3	43	M	1	5	TA	Left	9	3
4	44	F	180	7	TA	Left	8	5
5	27	M	180	4	TA	Right	10	5
6	27	M	6	4	Fall	Right	4	10
7	25	M	1	3	TA	Both	4	4
8	4	M	2	4	BT	Left	12	4
9	20	M	40	6	BT	Left	12	6
10	23	M	180	8	TA	Both	9	8
11	72	F	180	8	Fall	Left	8	4
12	33	M	180	6	Fall	Left	8	6
13	79	M	7	11	Fall	Right	6	5
14	49	M	2	3	Fall	Left	9	3
15	30	M	180	5	TA	Right	4	5
16	2	F	40	7	Fall	Left	14	3
17	14	M	5	7	TA	Both	4	2
18	69	M	4	12	TA	Left	6	7
19	59	M	35	3	Fall	Left	12	8
20	74	M	3	7	TA	Right	20	8
21	81	M	3	8	TA	Left	10	8
22	75	M	15	9	TA	Both	4	8

TA: Traffic accident, BT blunt trauma due to occupational accidents, GCS: Glasgow Coma Scale. M=Male, F=Female.

of the CBC, biochemistry and arterial blood gas analysis along with the midline shift in CT remained insignificant between the groups.

A couple of complications were observed on the 30th day of the operation in 7 (20.6%) of the surviving patients; 1 (2.9%) being cerebral abscess, 1 (2.9%) being hydrocephalus, and superficial wound infections in the

remaining 5 (14.7%) were observed. The surgical defect was closed using autograft in 7 (20.6%) patients and methyl methacrylate cranioplasty in 7 (20.6%) patients.

The Pearson's analysis of correlation revealed a couple of prognostic factors (Table-6). At the 30th day of the surgery, good prognosis was positively correlated with lower age, CCIS, BUN and GLU, whereas negatively

Table-6: The Correlation between clinical and laboratory admission parameters and poor prognosis (Death).

	Poor Prognosis (Death)
RTS	-0.400*
PLT	-0.374*
PaO <sub>2</sub>	-0.668**
SO <sub>2</sub>	-0.468**
Age	0.457**
CCIS	0.547**
BUN	0.441**
GLU	0.619**

\*p&lt;0,05. \*\*p&lt;0,001.

RTS: Revised trauma score; CCIS: Charlson comorbidity index score; PLT: Platelet; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

Table-7: Comparison of patient characteristics between the survivors and the non-survivors at the 30th day of the surgery.

	Survivors (n = 21)	Nonsurvivors (n = 13)	p
<b>Age (years)</b>			<b>0.007</b>
<60 (n)	20 (95%)	7 (53%)	
≥60 (n)	1 (05%)	6 (47%)	
<b>Preoperative GCS</b>			<b>0.042</b>
≤5	4 (19%)	7 (53%)	
>5	17 (81%)	6 (47%)	
<b>Midline Shift (mm)</b>			<b>0.406</b>
≤10	14 (66%)	10 (77%)	
>10	7 (34%)	3 (23%)	
<b>RTS</b>			<b>0.051</b>
>5	18 (86%)	7 (53%)	
≤5	3 (14%)	6 (47%)	
<b>CCISS</b>			<b>0.016</b>
<5	17 (81%)	5 (38%)	
≥5	4 (19%)	8 (62%)	
<b>GLU</b>			<b>&lt;0.001</b>
<180	17 (81%)	2 (84%)	
≥180	4 (19%)	11 (16%)	
<b>BUN</b>			<b>0.002</b>
≤30	13 (62%)	1 (07%)	
>30	8 (38%)	12 (93%)	
<b>PaO<sub>2</sub></b>			<b>0.003</b>
≤160	11 (52%)	13 (100%)	
>160	10 (48%)	0	
<b>SO<sub>2</sub></b>			<b>0.004</b>
≤96	9 (43%)	12 (93%)	
>96	12 (57%)	1 (07%)	

GCS: Gasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson comorbidity index score; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

Table-8: The classification of the patients according to Glasgow outcome scale score.

Outcome	GOS	n(%)
Unfavourable	1	16 (47.00)
	2	5(14.70)
	3	1(0.03)
Favourable	4	2(0.06)
	5	10(29.41)
Total		34(100.00)

correlated with lower RTS, PLT, PaO<sub>2</sub> and SO<sub>2</sub> on admission.

When 60 years of age was chosen as a cutoff point for age, the survivors at the 30th day of the operation were found to be significantly younger than the non-survivors. Similarly, pre-operative GCS>5, RTS>5, CCIS<5, glucose<180 mg.dl-1, PaO<sub>2</sub> >160 (mmHg), SO<sub>2</sub> >96 (%) were found to be the significant clinical and laboratory findings affecting the survival positively at the 30th day of surgery (Table-7).

At the end of the 6th month from the surgery, 16 (47.0%) of the patients were dead, while 18 (53.0%) had survived (Figure-2). At this point of time, the patients were re-classified into favourable (n=12; 35%) and unfavourable (n=22; 65%) outcome groups according to their GOS (Table-8).

The favourable outcome rate at this time point did not differ according to gender, mechanism of injury, the time duration from trauma until surgery, hemispheric location of the injury, the presence of extracranial injury, or the levels of systolic and diastolic arterial

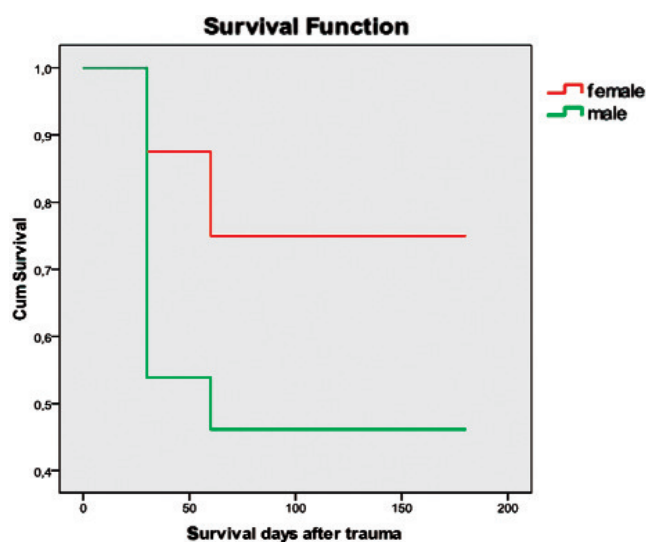


Figure-4: The survival function by gender.

Table-9: Comparison of the demographic and clinical parameters between favourable and unfavourable outcome groups at the 6th month of the operation.

		Favourable (n = 12)	Unfavourable (n = 22)	p
Age		24,2±15,6	44,3±25,6	0.025
Gender	Female	4 (33.3%)	4 (18.2%)	0.279
	Male	8 (66.7%)	18 (81.8%)	
Mechanism of injury	Fall	5 (41.7%)	8 (36.4%)	0.707
	MVA	5 (41.7%)	12 (54.5%)	
	Occupational	2 (16.7%)	2 (9.1%)	
Time duration until surgery (hrs)		4,8±2,8	5,5±2,1	0.168
Localisation of injury	Left hemispheric	5 (41.7%)	13 (59.1%)	0.254
	Right hemispheric	6 (50.0%)	5 (22.7%)	
	Bilateral	1 (8.3%)	4 (18.2%)	
Extracranial injury	Present	3 (25%)	5 (22.7%)	0.598
	Absent	9 (75%)	17 (77.3%)	
SAP**		110,83±24,66	98±48,85	0.345
DAP**		67,83±17,76	58,36±33,83	0.292
Pupillary reactivity	Bilaterally dilated and fixed	0 (0.0%)	12 (54.5%)	0.004
	Unilaterally dilated	6 (50.0%)	3 (13.6%)	
	Isochoria	6 (50.0%)	7 (31.8%)	
Preoperative GCS***		8 (5-13)	6 (3-12)	0.023
RTS***		9,50 (6-12)	6.5 (1-11)	0.004
CCIS***		1 (0-4)	5 (0-15)	<0.001
CKMB		5.40±3.05	46.41±48.07	0.029
GLU (mg.dl-1)		141.29±43.40	218.90±106.85	0.023
BUN		26.91±9.19	35.60±8.34	0.015
PaO <sub>2</sub>		169.53±77.12	102.49±42.52	0.004
SO <sub>2</sub>		96.75±4.78	92.58±4.50	0.005

\*p<0.05, \*\*mean ±SD, \*\*\*median (Range).

TA: Traffic accident; SAP: Systolic arterial pressure; DAP: Diastolic arterial pressure; GCS: Glasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson comorbidity index score; PLT: Platelet; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

Table-10: GOS values in relation with the age of the patients.

GOS	Age		Total (n%)
	≤60 (n)%	>60 (n)%	
1	10	6	16 (47)
2	4	1	5 (14)
3	1	0	1 (03)
4	2	0	2 (06)
5	10	0	10 (30)
Total	27	7	34

GOS: Glasgow outcome scale.

blood pressure at admission to the emergency department (Table-9). The age and the presence of the signs of herniation differed significantly between favourable and unfavourable outcome groups.

All of the patients with bilaterally dilated and fixed pupils (n=12) were in the unfavourable outcome group at the end of the 6th month. All the patients older than 60 years of age scored GOS 1 or 2 at the end of the 6th month. All of the patients in the favourable outcome

Table-11: Correlation between clinical and laboratory on-admission parameters and favourable outcomes.

	Favourable outcomes
Age	0.436**
RTS	-0.558**
CCIS	0.539**
Preoperative GCS	-0.402*
BUN	0.413*
Glucose	0.461**
PaO <sub>2</sub>	-0.565**
Saturation	-0.398**

\*p<0.05. \*\*p<0.001

GCS: Glasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson comorbidity index score; PLT: Platelet; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

group were ≥60 years of age (Table-10).

The pre-operative GCS at the end of the 6th month of surgery was significantly higher in the favourable outcome group. All of the patients in the favourable outcome group had scored ≥5 in pre-operative GCS.

Table-12: Comparison of favourable and unfavourable outcome groups regarding selected study parameters.

	Favourable Outcomes (n = 12)	Unfavourable Outcomes (n = 22)	p
<b>Age (years)</b>			<b>0.032</b>
<60 (n, %)	12	15	
≥60 (n, %)	0	7	
<b>Preoperative GCS</b>			<b>0.030</b>
≤5	1	10	
>5	11	12	
<b>Midline Shift (mm)</b>			<b>0.502</b>
≤10	8	16	
>10	4	8	
<b>RTS</b>			<b>0.009</b>
>5	12	13	
≤5	0	9	
<b>CCISS</b>			<b>0.001</b>
<5	12	10	
≥5	0	12	
<b>GLU</b>			<b>0.002</b>
<180	11	8	
≥180	1	14	
<b>BUN</b>			<b>0.031</b>
≤30	8	6	
>30	4	16	
<b>PaO<sub>2</sub></b>			<b>0.001</b>
≤160	4	20	
>160	8	2	
<b>SO<sub>2</sub></b>			<b>0.002</b>
≤96	3	18	
>96	9	4	

GCS: Glasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson co-morbidity index score; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen; SO<sub>2</sub>: Arterial oxygen saturation.

They had also scored significantly higher RTS and lower CCIS.

Among the laboratory values at admission, significantly lower GLU, CKMB, BUN along with higher PaO<sub>2</sub> and SO<sub>2</sub> were observed in the favourable outcome group. Unlike the 30th day of the surgery, the PLT did not differ

Table-14: Transformed Variables in Cox Regression.

Variable	Category	Frequency (n)
Pre-operative GCS	0= ≤5	3
	1= >5	31
RTS	0= >5	25
	1= ≤5	9
CCIS	0= ≥5	12
	1= <5	22
GLU	0= ≥180	15
	1= <180	19
BUN	0= ≥30	20
	1= <30	14
PaO <sub>2</sub>	0= ≥160	10
	1= <160	24
Age	0= ≥60	7
	1= <60	27

GCS: Glasgow outcome scale; RTS: Revised trauma score; CCIS: Charlson comorbidity index score; GLU: Glucose; BUN: Blood urea nitrogen; PaO<sub>2</sub>: Partial arterial pressure of oxygen.

between the groups at the end of the 6th month. The rest of the CBC, biochemistry and arterial blood gas analyses along with the midline shift in CT remained insignificant between the groups at the end of the 6th month.

The Pearson's analysis of correlation revealed a couple of prognostic factors (Table-11). At the 6th month of the surgery, a favourable outcome was positively correlated with lower age, CCIS, BUN, CRE and GLU, whereas it was negatively correlated with lower RTS, PaO<sub>2</sub>, SO<sub>2</sub> and pre-operative GCS on admission.

When 60 years of age was taken as the cutoff point for age, patients older than 60 years had a significantly unfavourable outcome at the end of the 6th month of the surgery. Similarly, pre-operative GCS ≤5, RTS ≤5, CCIS ≥5, glucose ≥180 (mg.dl-1), PaO<sub>2</sub> ≤160 (mmHg), SO<sub>2</sub> ≤96 (%) were all the determinants of poor prognosis and unfavourable outcome at the end of the 6th month of the surgery (Table-12).

The number of terminal events were 13 during the first

Table-13: Life Table Analysis.

Interval Starting Time	Entering to interval (n)	Withdrawing during the Interval (n)	Patients Exposed to Risk (n)	Patients with Terminal Events (n)	Proportion Terminating	Proportion Surviving
0	34	0	34	13	0.38	0.62
30	21	0	21	3	0.14	0.86
60	18	0	18	0	0.00	1.00
90	18	0	18	0	0.00	1.00
120	18	0	18	0	0.00	1.00
150	18	0	18	0	0.00	1.00
180	18	18	9	0	0.00	1.00

Table-15: Cox Regression Analysis of Parameters regarding Glogow Outcome Scale score.

	B	SE	Wald	df	Sig.	Exp(B)
Preoperative GCS	1.822	1.175	2.406	1	0.121	6.184
RTS	-2.353	0.885	7.064	1	0.008	0.095
CCIS	-0.804	0.949	0.717	1	0.397	0.448
GLU	-0.069	0.617	0.013	1	0.911	0.933
BUN	0.864	0.557	2.405	1	0.121	2.372
PaO2	-0.390	0.785	0.246	1	0.620	0.677
Age	2.706	1.088	6.184	1	0.013	14.964

GCS, Gasgow coma scale; RTS: Revised trauma score; CCIS: Charlson co-morbidity index score; GLU: Glucose; BUN: Blood urea nitrogen; PaO2: Partial arterial pressure of oxygen.

month, and 3 during the second month of the surgery. The proportion of termination was 0.38 for the first month, being the highest, and 0.14 for the second month (Table-13).

The survival rate decreased sharply during the initial two months time following the surgery and became steady from the third month onwards (Figure-3). The survival regarding gender was apparently higher among the female patients (Figure-4).

A cox regression analysis was conducted about some selected study parameters regarding the favourable and unfavourable outcome at the end of the 6th month. These selected parameters were transformed into categorical variables in order to perform the analysis (Table-14).

The predicted change in the hazard (Exp(B)) was significant for  $RTS > 5$  and  $age < 60$  years. Only younger age and  $RTS > 5$  were found to have significant effects on the favourable outcome at the end of the 6th month (Table 15). The rest of the study parameters were insignificant regarding the favourable outcome in the study population.

## Discussion

The present study mainly comprised of male patients having acute SDH primarily due to traffic accidents. Female patients had a higher survival rate than the male patients. The survival rate of the study group decreased sharply during the initial two months. The survival rate at the end of the first month as well as the favourable outcome at the end of the 6th month of the operation was higher in younger patients without any signs of herniation at admission to the emergency department.

Along with young age patients with higher pre-operative RTS, GCS, PaO<sub>2</sub>, SO<sub>2</sub>, CCIS, GLU, and BUN readings had a higher rate of survival and consequently a favourable outcome. Higher PLT values were only found to be a determinant of higher survival at the end

of the first month without having any significant effect on the favourable outcome. Although BUN and pre-operative GCS produced lower p values than the other variables in the cox regression analysis, the sample population was perhaps too small to detect any significant difference between the groups regarding these parameters. Studies in wider sample groups may be useful to detect the effects, if any, of these parameters.

The mortality from acute SDH ranges from 55% to 79%, even with surgical treatment.<sup>1,3</sup> There may be a trend of decreasing mortality over the last 25 years. However, in the present study, the first-month mortality rate was 38.2% and the 6th months mortality rate was 47%.

One study reported the brain dialysate lactate levels to rise and glucose levels to return to normal more rapidly in rats with induced SDH when these animals were ventilated 100% oxygen.[10] Thus, higher cerebral blood oxygen levels affect the cerebral glucose metabolism positively. Concordant with this finding, higher pre-operative PaO<sub>2</sub> and SO<sub>2</sub> values produced a more favourable outcome in our sample population

Another study has shown that the vasogenic oedema in rats with intracerebral haemorrhage either in the affected hemisphere or in the contralateral part is exacerbated by hyperglycaemia. In cases with traumatic brain injury, hyperglycaemia may be considered a marker of brain injury and, when present upon admission, could reflect extensive brain damage which is associated with higher mortality and poor outcome.<sup>12,13</sup> Concomitantly, lower glucose levels in our study population was found to be associated with better survival and favourable outcome.

Trauma is well known to be associated with cytotoxic and vasogenic brain oedema, aggravating intracranial hypertension associated with brain contusion.<sup>14-16</sup> Less well recognised is that the presence of blood gives rise

to haemotoxicity-related inflammation, which acts on capillaries to further aggravate oedema.<sup>14,17,18</sup> The presence of blood in the subdural space of healthy or contused brain reduces regional cerebral microcirculation and causes oedema, ischaemia, and increased intracranial pressure (ICP).<sup>14,19,20</sup> There is evidence that DC in animals can lower ICP and improve cerebral blood flow.<sup>21</sup> A study<sup>22</sup> indicated that DC is efficacious in adding to the available intracranial volume. DC significantly reduces intractable intracranial hypertension<sup>14,23</sup> and improves brain tissue oxygenation,<sup>24,25</sup> brain compliance,<sup>26</sup> cerebral blood flow,<sup>27,28</sup> pressure reactivity<sup>29</sup> and cerebrovascular resistance.<sup>30</sup> Each of these factors may lessen the burden of ischaemic penumbra, ischaemic brain volume and ICP.<sup>14,31</sup>

Severe life-threatening brain oedema occurs in approximately 85% of patients with an acute subdural haematoma.<sup>32</sup> Thus, frequently massive intra-operative brain swelling accompanies surgery for acute subdural haematoma, especially when the thickness of the haematoma is smaller than the deviation in midline, as measured on the CT. Massive brain swelling is far more common in patients with the lowest pre-operative GCS scores and those with unilateral or bilateral dilated pupils, suggesting a relationship to prior hypoperfusion.<sup>33</sup> Most are associated with early death due to increased ICP and subsequent uncal herniation.<sup>32</sup> Our patients with herniation both had a lower survival rate and unfavourable outcome.

The precondition necessary for surgical evacuation of traumatic acute SDH has previously been well defined. An acute SDH with a thickness greater than 10mm or a midline shift greater than 5mm as determined from a CT scan should be surgically evacuated.<sup>6,34-36</sup> However, there is no consensus regarding which surgical technique should be employed for the evacuation of acute traumatic SDH. Some surgeons perform craniotomies, while others perform DC. The presence and the degree of midline shift have not had any significant effect on either the survival or the favourable outcome in our study group.

In the 1970s, one study presented encouraging results with a hemicraniectomy followed by haematoma evacuation for SDH. It reported a recovery rate of 40%, but could not confirm these results in a later study.<sup>4,37</sup> In a separate series of 15 patients, researchers found a slightly improved recovery and lower mortality in patients with hemicraniectomy and external drainage, but the results were not statistically significant.<sup>5</sup>

A 2008 study reported that 80 patients with acute SDH had early DC combined with uncusotomy and tentorial section to relieve brainstem compression.<sup>38</sup> Craniectomy threshold was abnormal pupillary response to light and ICPs greater than 20mmHg. In that study, 75% patients had good outcome and only 15% died. Younger age was significantly related to good outcome.<sup>38</sup> The association of higher survival and favourable outcome were also confirmed in our study population.

It is still a controversial point that in patients who have undergone craniectomy, cerebral oedema and the risk of additional cerebral damage due to the displacement of the brain increases.<sup>39</sup> Although significant reduction of ICP and secondary ischaemia can be achieved, the procedure may allow herniation of brain tissue through the bone defect. This frequently results in the compression of arteries and veins by dural margin, causing further congestion, oedema and ischaemia in the herniated tissue.<sup>40-42</sup> In order to prevent this complication, studies<sup>41,42</sup> have suggested that the dura be opened in stellate fashion and the main cortical vessels to be left under the margins and not the corners of the dural opening. They have also emphasised the importance of generating a vascular tunnel by the placement of haemostatic sponges on both sides of the main vessels. The technique, as shown by them, decreased mortality and morbidity. We also followed this procedure in our study population.

When considering the treatment options, one should always keep in mind that DC, although not an obligation, entails certain risks for the patient. Blood loss, not an unusual complication in large wounds, during and after the operation and impaired blood coagulation, a frequently seen problem, can lower the cerebral perfusion pressure (CPP) dramatically.<sup>14,22</sup> One study reported the risk of a new or expanded haemorrhagic contusion to be 58% in patients undergoing DC.<sup>43</sup> The expansion of the haemorrhagic contusion was not life-threatening in our study group.

Moreover, DC for traumatic brain injury may incite a new mass lesion, contralateral or remote to the decompressed hemisphere.<sup>44,45</sup> A study reported a 7% incidence of new contralateral haematomas, over half occurring within the initial 24-hour period following DC.<sup>45</sup> Decompressive surgery may relieve the tamponade effect on a contralateral bleeding site and predispose the patient to an epidural haematoma.<sup>44</sup> A study has reported new contralateral epidural haematomas in 71% of its population. We observed no

such complications in our study group.

Decompressive craniectomy alters the dynamics of cerebrospinal fluid circulation. This may exacerbate the occurrence of subdural hygromas and hydrocephalus.<sup>14,22,44</sup> Researchers reported a 50% rate of subdural hygroma and hydrocephalus following DC, regressing spontaneously over weeks to months.<sup>47</sup> Others have reported a 21% rate of subdural hygroma, 87% resolving spontaneously without having any resultant neurological deficit.<sup>45</sup> Only one patient developed hydrocephalus in our study group who was treated through placement of a ventriculoperitoneal shunt.

Infectious complications may emerge either following the surgery or the replacement of the bone flap.<sup>14,22,44</sup> Our study group had one patient with cerebral abscess which deceased by the 5th month, while superficial wound infections were treated successfully in five patients.

## Conclusion

The concept of performing decompressive craniectomy in traumatic acute subdural haematoma patients seems to be attractive. In SDH patients whose GCS on arrival is  $\leq 8$ , a massive craniectomy along with the evacuation of the haematoma may be considered as a treatment option for intra-operative and post-operative brain swelling. But in patients whose GCS is 3 on arrival and who have bilaterally fixed and dilated pupils, decompressive craniectomy is unnecessary. Patients over 60 years of age, or those with signs of herniation, male gender, pre-operative GCS $<5$ , RTS $<5$ , CCIS  $\geq 5$ , GLU  $\geq 180$ mg.dl-1, BUN  $\geq 30$ mg.dl-1, PaO<sub>2</sub>  $\leq 160$ mmHg, SO<sub>2</sub>  $\leq 96\%$  were found in our study to have poor prognosis. In order to assess the efficacy of DC on acute SDH and to provide solid criteria for the necessity of this type of surgical decompression of the brain, it is essential to perform studies with larger population size.

## References

1. Servadei F. Prognostic factors in severely head injured adult patients with acute subdural haematomas. *Acta Neurochir* 1997; 139: 279-85.
2. Koc RK, Akdemir H, Oktem IS, Meral M, Menku A. Acute subdural hematoma: outcome and outcome prediction. *Neurosurg Rev* 1997; 20: 239-44.
3. Woertgen C, Rothoerl RD, Schebesch KM, Albert A. Comparison of craniotomy and craniectomy in patients with acute subdural haematoma. *J Clin Neurosci* 2006; 13: 718-21.
4. Ranshoff J, Benjamin MV, Gage EL, Epstein F. Hemicraniectomy in the management of acute subdural haematoma. *J Neurosurg* 1971; 34: 70-6.
5. Shigemori M, Syojima K, Nakayama K, Kojima T, Ogata T, Watanabe M, et al. The outcome from acute subdural haematoma following decompressive hemicraniectomy. *Acta Neurochir* 1980; 54: 61-9.
6. Wong GK, Hung YW, Chong C, Yeung J, Chi-Ping Ng S, Rainer T, et al. Assessing the neurological outcome of traumatic acute subdural hematoma patients with and without primary decompressive craniectomies. *Acta Neurochir Suppl* 2010; 106: 235-7.
7. Champion HR, Sacco WJ, Copes WS, Gann DS, Gennarelli TA, Flanagan ME. A revision of the trauma score. *J Trauma* 1989; 28: 623-9.
8. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987; 40: 373-83.
9. Jennett B, Bond M. Assessment of outcome after severe brain damage: a practical scale. *Lancet* 1975; 1: 480-4.
10. Reinert M, Alessandri B, Seiler R, Bullock R. Influence of inspired oxygen on glucose-lactate Dynamics after subdural hematoma in the rat. *Neurol Res* 2002; 24: 601-6.
11. Song EC, Chu K, Jeong SW, Jung KH, Kim SH, Kim M, et al. Hyperglycemia exacerbates brain edema and perihematomal cell death after intracerebral hemorrhage. *Stroke* 2003; 34: 2215-20.
12. Melo JR, Di Rocco F, Blanot S, Laurent-Vannier A, Reis RC, Baugnon T, et al. Acute hyperglycemia is a reliable outcome predictor in children with severe traumatic brain injury. *Acta Neurochir* 2010; 152: 1559-65.
13. Kreutziger J, Schlaepfer J, Wenzel V, Constantinescu MA. The role of admission blood glucose in outcome prediction of surviving patients with multiple injuries. *J Trauma* 2009; 67: 704-8.
14. Aarabi B, Hesdorffer DC, Simard JM, Ahn ES, Aresco C, Eisenberg HM, et al. Comparative study of decompressive craniectomy after mass lesion evacuation in severe head injury. *Neurosurgery* 2009; 64: 927-40.
15. Marmarou A. Traumatic brain edema: an overview. *Acta Neurochir Suppl* 1994; 60: 421-4.
16. Simard JM, Kent TA, Chen M, Tarasov KV, Gerzanich V. Brain oedema in focal ischaemia: Molecular pathophysiology and theoretical implications. *Lancet Neurol* 2007; 6: 258-68.
17. Chen-Roetling J, Regan RF. Effect of heme oxygenase-1 on the vulnerability of astrocytes and neurons to haemoglobin. *Biochem Biophys Res Commun* 2006; 350: 233-7.
18. Hoff JT, Xi G. Brain edema from intracerebral hemorrhage. *Acta Neurochir Suppl* 2003; 86: 11-5.
19. Fujisawa H, Maxwell WL, Graham DI, Reasdale GM, Bullock R. Focal microvascular occlusion after acute subdural haematoma in the rat: A mechanism for ischemic damage and brain swelling. *Acta Neurochir Suppl* 1994; 60: 193-6.
20. Sawauchi S, Beaumont A, Signoretti S, Tomita Y, Marmarou C, Marmarou A. Diffuse brain injury complicated by acute subdural hematoma in the rodents: The effect of early or delayed surgical evacuation. *Acta Neurochir Suppl* 2002; 81: 234-44.
21. Rinaldi A, Mangiola A, Anile C, Maira G, Amante P, Ferraresi A. Hemodynamic effects of decompressive craniectomy in cold induced brain oedema. *Acta Neurochir Suppl* 1990; 51: 394-6.
22. Münch EC, Horn P, Schürer L, Piepgras A, Paul T, Schmiedek P. Management of severe traumatic brain injury by decompressive craniectomy. *Neurosurgery* 2000; 47: 315-23.
23. Schneider GH, Bardt T, Lansksch WR, Unterberg A. Decompressive craniectomy following traumatic brain injury: ICP, CPP and neurologic outcome. *Acta Neurochir Suppl* 2002; 81: 77-9.
24. Jaeger M, Soehle M, Meixensberger J. Effect of decompressive craniectomy on brain tissue oxygen in patients with intracranial hypertension. *J Neurol Neurosurg Psychiatry* 2003; 74: 513-5.
25. Stiefel MF, Heuer GG, Smith MJ, Bloom S, Moloney-Wilensky E,

- Gracias VH, et al. Cerebral oxygenation following decompressive hemicraniectomy for the treatment of refractory intracranial hypertension. *J Neurosurg* 2004; 101: 241-7.
26. Timofeev I, Czosnyka M, Northje J, Smielewski P, Kirkpatrick P, Gupta A, et al. Effect of decompressive craniectomy on intracranial pressure and cerebrospinal compensation following traumatic brain injury. *J Neurosurg* 2008; 108: 66-73.
  27. Csokay A, Pataki G, Nagy L, Belan K. Vascular tunnel construction in the treatment of severe brain swelling caused by trauma and SAH (evidence based on intra-operative blood flow measure). *Neurol Res* 2002; 24: 157-60.
  28. Hlatky R, Valadka AB, Goodman JC, Robertson CS. Evolution of brain tissue injury after evacuation of acute traumatic subdural hematomas. *Neurosurgery* 2004; 55: 1318-24.
  29. Wang EC, Ang BT, Wong J, Lim J, Ng I. Characterization of cerebrovascular reactivity after craniectomy for acute brain injury. *Br J Neurosurg* 2006; 20: 24-30.
  30. Bor-Seng-Shu E, Hirsch R, Teixeira MJ, De Andrade AF, Marino R Jr. Cerebral hemodynamic changes gauged by transcranial Doppler ultrasonography in patients with posttraumatic brain swelling treated by surgical decompression. *J Neurosurg* 2006; 104: 93-100.
  31. Cunningham AS, Salvador R, Coles JP, Chatfield DA, Bradley PG, Johnston AJ, et al. Physiological thresholds for irreversible tissue damage in contusional regions following traumatic brain injury. *Brain* 2005; 128: 1931-42.
  32. Coplin WM, Cullen NK, Policherla PN, Vinas FC, Wilseck JM, Zafonte RD, et al. Safety and feasibility of craniectomy with duraplasty as the initial surgical intervention for severe traumatic brain injury. *J Trauma* 2001; 50: 1050-9.
  33. Lobato RD, Sarabia R, Cordobes F, Rivas JJ, Andoras A, Cabrera A, et al. Post-traumatic cerebral hemispheric swelling. Analysis of 55 cases studied with computerized tomography. *J Neurosurg* 1988; 68: 417-23.
  34. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of acute subdural hematomas. *Neurosurgery* 2006; 58: S2-16-S2-24.
  35. Mathew P, Oluoch-olunya D, Condon B, Bullock R. Acute subdural haematoma in the conscious patient: outcome with initial nonoperative management. *Acta Neurochir* 1993; 121: 100-8.
  36. Wong CW. Criteria for conservative treatment of supratentorial acute subdural haematomas. *Acta Neurochir* 1995; 35: 38-43:100-8.
  37. Cooper PR, Rovit R?, Ranshoff J. Hemicraniectomy in the management of acute subdural haematoma: a re-appraisal. *Surg Neurol* 1976; 5: 25-8.
  38. Salvatore C, Marco M, Antonio R, Salvatore I, Eugenio B. Combined internal unsectomy and decompressive craniectomy and decompressive craniectomy for the treatment of severe closed head injury experience with 80 cases. *J Neurosurg* 2008; 108: 74-9.
  39. Soukiasian HJ, Hui T, Avital I, Eby J, Thompson R, Kleisli T, et al. Decompressive craniectomy in trauma patients with severe brain injury. *The American Surgeon* 2002; 68: 1066-71.
  40. Cooper PR, Hagler H, Clark WK, Barnett P. Enhancement of experimental cerebral edema after decompressive craniectomy: implications for the management of severe head injuries. *Neurosurgery* 1979; 4: 296-300.
  41. Csokay A, Egyud L, Nagy L, Pataki G. Vascular tunnel creation to improve the efficacy of decompressive craniotomy in post-traumatic cerebral edema and ischemic stroke. *Surg Neurol* 2002; 57: 126-9.
  42. Csokay A, Nagy L, Novoth B. Avoidance of vascular compression in decompressive surgery for brain edema caused by trauma and tumour ablation. *Neurosurg Rev* 2001; 24: 209-13.
  43. Flint AC, Manley GT, Gean AD, Hemphill JC 3rd, Rosenthal G. Post-operative expansion of hemorrhagic contusion after unilateral decompressive hemicraniectomy in severe traumatic brain injury. *J Neurotrauma* 2008; 25: 503-12.
  44. Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. *Neurosurg Focus* 2009; 26: E7.
  45. Yang XF, Wen L, Shen F, Li G, Lou R, Liu WG, et al. Surgical complications secondary to decompressive craniectomy in patients with head injury: a series of 108 consecutive cases. *Acta Neurochir* 2008; 150: 1241-8.
  46. Su TM, Lee TH, Chen WF, Lee TC, Cheng CH. Contralateral acute epidural hematoma after decompressive surgery of acute subdural hematoma: clinical features and outcome. *J Trauma* 2008; 65: 1298-302.
  47. Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. *J Neurosurg* 2006; 104: 469-79.
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