Özet

Bulgular: Dokuz hastaya dekompresif kraniektomi tedavisi uygulanmıştır. Bu hastaların ilk başvurularındaki intrakranial basınç (ICP) değerleri ortalaması 22.6 ± 6.7 mmHg; ortalama ameliyat öncesi ICP değeri 40.8 ± 16.3 mmHg ve cerrahi tedavi sonrası ICP ortalama değeri 9.3± 3.6 mmHg olarak ölçülmiştir. İki hastaya bilateral ve kalan yedi hastaya unilateral frontotemporoparietal kraniektomi uygulanmıştır. Kraniektomi kemik flebinin tekrar yerine koyulması için ortalaması 25.75±10.0 gün beklenmiştir.

Subaraknoid kanama ile takip edilen bir hasta cerrahi tedavi sonrası ölümürtür (mortalite oranı %11.1). Otuz altı aylik takip sonrası hastaların ortalaması Glasgow iyileşme skala puanı yaklaşık 4 olarak hesaplanmıştır. Sonuç: Eğer zamanında ve dikkatli bir şekilde uygulanırsa bu cerrahi teknik akut ya da gecikmiş dirençli intrakranial hipertansiyon tedavisinde başarılı sonuçlar verebilir.

Anahtar Kelimeler
Intrakranial Basınç Yükselişi; Sonuç Değerlendirmesi

Abstract
Aim: This retrospective study was established to analyse the effects of the decompressive craniectomy on patients with different causes of increased intracranial pressure. Material and Method: Nine patients at risk of developing malignant cerebral edema aged between 18 and 75 years were included in this study. Four patients suffered from severe traumatic brain injury; two patients from subarachnoid haemorrhage (SAH) and vasospastic ischemia, and 3 patients from malignant infarction of the middle cerebral artery. Results: Nine patients underwent decompressive craniectomy as the last therapeutic choice. At first admission mean intracranial pressure (ICP) was 22.6 ± 6.7 mmHg; mean preoperative ICP value was 40.8 ± 16.3 mmHg; and mean postoperative ICP value was 9.3± 3.6 mmHg. In two patients bilateral; and in seven patients unilateral frontotemporoparietal craniectomy was preferred. Mean time of the re-implantation of the bone flap was 25.75±10.0 days. One patient with SAH died postoperatively and eight patients survived (mortality rate 11.1%). Mean value of the Glasgow Outcome Scale score as evaluated at 36 months after the decompression was approximately 4. Discussion: This surgical procedure is successful for treatment of the acute or delayed intractable intracerebral hypertension with a low rate of complication if it is performed timely and carefully.

Keywords
Intracranial Pressure Increase; Outcome Assessment

DOI: 10.4328/JCAM.676

Corresponding Author: Bulent Bakar, Kirikkale University Faculty of Medicine, Department of Neurosurgery, 71100 Kirikkale, Turkey.
T: +90 3182252485 E-Mail: bulentbaars@yahoo.com
Introduction
Intractable intracranial hypertension (IIHT) is an additional risk for patients who already have suffered from brain damage and therefore remains a challenge for the treating physician [1]. In these patients the initial strategy is to repair or to stabilize the primary cause and to decrease the intracranial pressure (ICP) levels to minimize the alterations of both local and total cerebral perfusion [2]. Intracranial hypertension which could not be managed with maximal medical treatment occurs in up to 10 to 15% of these patients. Studies show a significant relationship between an ICP greater than 25 mmHg and poor outcome [3]. Numerous recent clinical reports provided data that decompressive craniectomy improves O2 delivery to brain cells when cerebral ischemia is at its peak levels [4].

There are yet few reports in the medical literature that compare the effect of decompressive craniectomy in different neurosurgical diseases. Therefore this retrospective study was established to analyse the effects of the decompressive craniectomy on patients with different disease groups.

Material and Method
Patients characteristics
On admission, cerebral pathology was revealed by neuroradiological studies (CT, MRI, MRA) and neurological status was recorded as the pre-intubation Glasgow Coma Scale (GCS) score. Penetrating head injury or systemic injury with head injury was an exclusion criterion. None of the chosen patients needed to be operated on admission for an intracranial hematoma or mass lesion. Patients with primary fatal brainstem failure as indicated by GCS score of 3 did not undergo surgical intervention.

Treatment protocols
Patients were treated in the neurosurgical intensive care unit (NICU). Each patient was fully resuscitated, intubated, and anaesthesized with pentothal sodium or sedated with propofol and remifentanyl hydrochloride after first admission. In the NICU, patients were kept in a supine position with 30 degree head elevation. Basic monitoring (six lead ECG, invasive arterial blood pressure measurement, pulse oxymeter, continuous rectal temperature assessment), and frequent (2-4 hourly) blood gas analyses were performed; and all basic blood biochemistry parameters (sodium, potassium, albumine, creatinine, etc.) were followed twice daily. Neurologic monitoring included an ICP probe parenchymal type device (Camino, Integra Neurosciences Corporation, USA), the temporal muscle was treated by bilateral wide F-T-P skin flaps. The burrholes are connected to the frontal, temporal and parietal lobes extensively (Figure 1). Cortical resection was not performed in any patients. After insertion of the ICP probe, a standard treatment protocol was used. Ventilation parameters were managed to an optimum PaCO₂ according to ICP; and a rising in ICP over the 20 mmHg was managed by milder hyperventilation (PCO₂= 30-32 mmHg), intermittent 4 hourly application of mannitol (25-50 g). Dopamine was titrated when cerebral perfusion pressure (CPP) was less than 65 mmHg for more than 15 minutes.

Operative procedures
Unilateral oedema or swelling was treated by a large hemispheric decompressive craniectomy over the swollen hemisphere, whereas bilateral diffuse oedema/ swelling were treated by bilateral fronto-temporo-parietal (F-T-P) bony decompression. If the neurological status and/or ICP levels were better than the above mentioned surgical indications, supportive and antioedema treatment was continued.

ICP probe insertion is preferably done on the side of planned or predicted surgery. This is important because a contralateral placement may cause difficulty in positioning the patient for surgery or may cause unnecessary pressure over the Camino bolt. From this point of view there is no problem with bilateral decompression yet overturning of bicoronal flap anteriorly will be limited because of the bolt.

Unilateral decompressions are performed using wide F-T-P central skin flap whereas bilateral decompressions are performed by bilateral wide F-T-P skin flaps. The burrholes are connected using pneumatic drill, with subsequent removal of a 12x15 cm free bone flap from each side. Additional temporal craniectomy towards the floor of middle fossa is done using a large rongeur. For bilateral decompressions, a parasagittal 3 cm wide bony strip is preserved both to avoid damage to the sagittal sinus and to serve as a frame for a late calvarial reconstruction. The dura is usually opened with stellate incision in the areas involving the frontal, temporal and parietal lobes extensively (Figure 1). Cortical resection was not performed in any patients. After duraplasty using galea greft or artificial meninges (Duragen, Integra LifeSciences Corporation, USA), the temporal muscle was loosely reapproximated to the healthy dura and the skin flap was then closed with interrupted prolene sutures. The bone flap was usually re-implanted within 1-2 months after craniectomy, having been stored under sterile conditions at -80 OC.

Postoperative management
Postoperatively, a CT scan was obtained for checking brain-bone relationship at craniotomy borders and change in the midline shift. After the decompressive surgery, conventional medi-
cal management including sedational anaesthesia, intermittent mannitol, antiepileptic agents were continued at least two more days (48 hours). A ventriculoperitoneal shunt was performed if it was necessary.

**Assessment of neurological outcome**
The neurological outcome was scored according to the Glasgow Outcome Scale (GOS). When GOS was:
1. death
2. persistent vegetative state
3. severe disability
4. moderate disability
5. mild or no disability [5]

**Results**

**Patients characteristics**
All patients were treated during the time period of February 1, 2005 to April 1, 2009. Nine patients at risk of developing malignant cerebral oedema aged between 18 and 75 years (mean age was 48.1 ± 18.8 years) were included in this study. Four patients suffered from severe traumatic brain injury (STBI), two patients from subarachnoid haemorrhage (SAH) (Hunt Hess grade IV or V), and 3 patients from complete infarction of the middle cerebral artery (CVA-cerebrovascular accident) (Table 1).

Mean pre-intubating GCS score of all patients was 7.3±3.1. At initial insertion of ICP probe, mean ICP level of overall patients was 22.6 ± 6.7 mmHg.

Overall, 9 patients underwent decompressive craniectomy between days 1 and 12 post-admission (mean day 5.2 ± 4.6). At initial insertion of ICP probe, mean ICP level was 22.6 ± 6.7 mmHg; preoperative mean ICP value was 40.8 ± 16.3 mmHg; and mean postoperative ICP value was 9.3 ± 3.6 mmHg. In two patients bilateral; and in seven patients unilateral F-T-P craniectomy was preferred. Mean time of the re-implantation of the bone flap for calvarial reconstruction was 25.7±10.0 days. The mean hospital stay was 56 days (range 13 to 148 days).

**Complications related to decompressive surgery**
There was no intracranial and/ or wound infection; nor any CSF leakage in any patient. In one patient, a subdural effusion occurred at the surgery side; and after the bone flap re-implantation, the effusion had disappeared (Figure 2). One patient who had sigmoid sinus thrombosis occurring after blunt head trauma required ventriculoperitoneal shunt for late hydrocephalus. In two patients, posttraumatic epilepsy occurred.

**Follow-up and outcome**
The clinical outcomes were evaluated at 36 months after the decompressive surgery. One patient died postoperatively (day 13), and seven patients survived (mortality rate 11.1%). GOS score values of the operated patients were as follows: 4/9 full recovery (GOS score, 5); 1/9 moderate recovery (GOS score, 4); 2/9 severe disability (GOS score, 3); 1/9 vegetative state (GOS score, 2); 1/9 died (GOS score, 1).

Postoperative fatality was associated with delayed basilar artery spasm caused by SAH in a patient with coiled basilar tip aneurysm. In the STBI group, one patient recovered fully, two patients were left with severe disability, and another patient remained in vegetative state. Additionally, each of three CVA patients had moderate/ full recovery; and one SAH patient had full recovery. Mean GOS score value for operated patients was approximately 4±1.2.

<table>
<thead>
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<th>Table 1. Descriptive table of the patients</th>
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<td>IM: male, F: female, STBI: severe traumatic brain injury, SAH: subarachnoid haemorrhage, CVA: cerebrovascular accident, f-GCS: Glasgow Coma Scale score on admission, a-ICP: intracranial pressure on admission, f-ICP: pre-operative ICP level, p-ICP: post-operative ICP level, d-GCS: Glasgow Coma Scale score while discharge from the hospital, GOS: Glasgow Outcome Scale score as evaluated at 36 months after the decompression</td>
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Discussion

The basic pathophysiological mechanism of brain oedema associated with brain injury is an increase in the net water content of the brain. This causes an increase in tissue pressure which increases cerebrovascular resistance, compromises the microcirculation, and results in reduced cerebral blood flow [4]. Various neurological conditions which cause an increase in intracranial pressure have been treated successfully with uni- or bilateral decompressive techniques. The effectiveness of this surgical procedure in reducing ICP has been proven by recent reports or studies that come out in a steadily increasing manner [3]. Decompressive craniectomy together with dura expansion is intended to increase the volume of the intracranial compartment, and to create space for oedematous brain tissue. The procedure thus not only prevents the event cascade leading to impaired tissue oxygenation but also the secondary injuries resulting from ischemia [3, 4]. Recent studies have shown that early decompression after development of refractory intracranial hypertension almost always brings about an improved outcome [6]. Mortality figures had also dropped from as high as 80% to 20%. Most of the reported investigations suggested that patients who underwent an early surgery (within 6 hours from the injury) have a more favourable outcome compared with those who underwent surgical intervention beyond 6 hours [7]. Kening et al. (2009) have suggested that hing craniotomy may be widely applicable for cerebral decompression, and it appears to be as good as decompressive craniectomy in providing postoperative ICP control. They also have shown that their results are in equivalent early clinical outcomes [8]. However this study has not contained the results of the tissue oxygenation levels, and has required long term outcome analysis.

The adverse relationship between elevated ICP and outcome has been recognized for more than 30 years [9]. In our study, mean GCS score on admission was approximately 7; and preoperative mean ICP level was 40.8 ± 16.3 mmHg. Despite these unfavourable preoperative data, the mean GOS score of the operated patients was approximately 4, and the mortality rate was 11.1%. Salvatore et al. (2008) postulated that in STBI patients, the preoperative neurological status and pupillary reactivities has not effect on the favourable outcome [7]. In our study, mean time between occurrence of primary injury and decompressive surgery for IHIT was 5.2±4.6 days; and mean GOS score value of these operated patients was approximately 4. The mean GOS score value of STBI group was nearly 3, but the SAH and CVA group had GOS score of 4. These results suggest that both surgery timing and aetiology of the IIHT is much more important for the favourable outcome.

On the other hand even after sufficient decompression, the brain will still maintain a high demand of oxygen and previously compressed cerebral vessels will fill with blood; and this may cause a further increase in net brain volume and a paradoxical rise in ICP in early postoperative period [4]. In our study, this event occurred during intra- and early post-operative course which had to be aggressively managed by medical treatment. Decompressive craniectomy is usually considered technically simple and it has a low rate complication. With current armamentarium of operating rooms lifting a bone flap and incision of the dura to relieve the pressure will not take more than 20 minutes. The cumbersome part is duraplasty. As for bony decompression, almost all the authors have suggested an adequate size of the craniectomy to prevent self-strangulation of edematous brain and local pressure of bony corners on cortex of bulging brain. Within few hours after injury or ischemia pressure of edematous temporal lobe on brain stem will cause necrotic changes within the stem and along the cranial nerves and vascular structures [7] therefore addition craniectomy along the temporal fossa is a crucial part of decompression technique in order to relieve pressure on temporal lobe as a whole hence on brain stem. A large bony decompression also reduces the incidence of delayed intracranial haematoma, repeated surgical intervention, postoperative CSF leakage, traumatic seizure(s), and/or intracranial infections [10]. In our series three patient had minimal brain stranuglation: one patient (patient #2) underwent bilateral F-T-P craniectomy; and he had full recovery (GOS score, 5). The other one (patient #1) undergoing bilateral cranietomy ended up was vegetative status (GOS score, 2); and another one (patient #3) performed unilateral craniectomy was severe disability (GOS score, 3).

In several studies, age appeared as a negative predicting factor for favourable outcome. Several authors concluded that the mean age of the group with unfavourable outcome was 56.6 years [6, 9, 11, 12]. Our study did not support this data. In STBI group, all patients' ages were below the 55 years, and mortality rate was 0%; however in CVA group the mean age was 65 years, and they had mild disability with full survival. In SAH group there was one mortality who was 38 year-old-woman.

STBI is a major cause of death and disability in the population; and this morbidity and mortality is produced by cerebral oedema which contributes to elevation of ICP and reduction of cerebral blood flow. These processes manifest as progressive intracranial hypertension refractory to medical therapy with subsequent brain herniation and death. The literature data stress the importance of age, severity of injury, presence of extracranial injuries as main predictors of outcome. Initial GCS score has not been a reliable marker for development of marked ICP elevation [13]. Our study also showed that initial GCS score did not have a predictive value for the outcome. Age too did not correlate with outcome; but severity of the injury and initial ICP values were important prognostic factors in this study (See Table 1).

Most of patients with hemispheric infarctions had unilateral brain swelling and they have 80% mortality rate. Decompressive craniectomy has been shown to reduce this mortality to 20-35% [2, 14,15]. Few recent studies on malignant ischemic stroke demonstrate that early surgery even before the pupil dilates may increase the number of patients with a favourable functional outcome [6, 16, 17]. In our study, although the mean time between the onset of ischemic stroke and the operation was approximately 60 hours, these CVA patients still had mild disability with full survival.

Some recent studies showed that patients with SAH still suffer from a high incidence of specific complications (such as symptomatic cerebral vasospasm); and delayed cerebral ischemia secondary to vasospasm contributes a major part of the morbidity and mortality) [18, 19]. In these patients decompressive craniectomy may enable the normalization of arterial PCO2 and reduce the co-morbidity of the 3H therapy (hypertensive, hy-
Intracranial Hypertension for Intracranial Hypertension

pervolumic and haemodilutional therapy] [18, 20]. In our study, one of the SAH patients who had symptomatic cerebral vasospasm died within 13 days; and other one who had right frontotemporal oedema caused to subfalcine herniation recovered almost fully 1 year after decompressive craniectomy. Overall, decompressive craniectomy may lead to improved outcome in a carefully selected subgroup of patients with high-grade patients (Hunt-Hess 4 and 5).

Fortunately, there are only few complications of decompressive surgery. Risk of fatal complications such as intracranial infections and contralateral intracerebral haematoma exist but rarely take place. Other complications (such as subdural effusion-hygroma, posttraumatic hydrocephalus, epilepsy) can adversely effect the patient’s outcome [3, 9, 21]. In our experience, there was no postoperative contralateral intracerebral haematoma or any intracranial infection. In two patients, postoperative epilepticus occurred, and this condition was successfully managed using antiepileptic medication. In one patient, postoperative subdural effusion recovered spontaneously after reimplantation of the bone flap. In another patient, postoperative hydrocephalus occurred 36 days after the decompressive surgery, but this complication was secondary to major dural sinus thrombosis and was not related to surgery per se. Overall, this surgical procedure can decrease the mortality and morbidity associated with acute or subacute intracerebral hypertension with a low rate complication if it is performed carefully and adequately.

This retrospective study has few pitfalls. First, because of a small and a rather heterogeneous study population, it is hard to conclude specifically on patient outcome for different etiologies. Additionally it is too small to have statistical impact and give guidelines concerning indications and timing of decompressive craniectomy. However, the patient variety shows that decompressive craniectomy does improve the survival and overall outcome for all, less in trauma more in ischemic group. Second, the study sample includes different disease groups each representing different complex mechanisms. However though the pathophysiology in these groups differs, the most important common denominator is increased ICP and cerebral oedema. All patients with CVA had mild disability and full survival, but the SAH group had one mortality associated with cerebral vasospasm. The outcome in STBI group was not uniform as would be expected. However since the secondary effects of trauma energy follow different pathophysiological steps than CVA and SAH, conditions like contusion, hematoma mass, brain stem damage, diffuse axonal injury and anoxia/hypoxia could have hampered the outcome of trauma patients. Third, there was no co-measurement of the local brain tissue oxygenation. In literature various studies demonstrate that cerebral hypoxia is common after severe brain injury and this hypoxia sometimes can occur when CPP or ICP is normal levels. This condition is also related with poor outcome. Many studies have showed that decompressive craniectomy with dura expansion restores adequate brain tissue oxygenation [4, 20]. Fourth, although the indications for decompressive craniectomy were prospectively defined, data analysis was performed retrospectively without a control group.

As conclusion, the indication for decompressive surgery is simple and straightforward and that is intractable intracranial hypertension. Choice is between unilateral and bilateral decompression and that decision is largely based on preoperative CT scans. The age, severity of injury, presence of extracranial injuries or systemic diseases should not affect patient selection.) [13].

Conclusion

1. For improving the outcome following intracranial hypertension, controlling high ICP, maintaining a close-to-normal cerebral perfusion pressure and cerebral blood flow is extremely important.

2. At present, choice of the treatment for acute or subacute intractable intracranial hypertension is unilateral or bilateral decompressive craniectomy. This surgical procedure is successful with a low rate complication if it is performed carefully and adequately.

3. A wide decompressive craniectomy with dural enlargement is essential for good outcome.

4. Both during pre- and post-operative periods, medical antiepodea treatment is recommended to decrease local pressure on cortex of herniating brain tissue.

5. Factors such as age, severity of injury, systemic diseases, systemic injuries may affect the outcome after decompressive craniectomy.

6. Finally, decompressive craniectomy is an effective procedure improving survival and overall outcome in various different neurosurgical diseases.

References