

Decompressive Hemicraniectomy in Patients With Transtentorial Herniation

Orhan BARLAS^{1,4}, Selhan KARADERELER¹, Yakup KRESPI^{2,5},
Nilüfer YEŞİLOT³, Sara BAHAR³, Rezzan TUNCAY³,
Reha TOLUN^{2,5}

Şişli Florence Nightingale Hospital Departments of Neurosurgery¹, Neurology²,
Istanbul Medical Faculty Departments of Neurology³, Neurosurgery⁴,
Istanbul Bilim University Medical Faculty Department of Neurology⁵

✓ **Objective:** To present the results, technique and indications of decompressive hemicraniectomy in patients with unilateral transtentorial herniation.

Methods: Ten consecutive patients with intracranial hypertension refractory to medical management and transtentorial herniation were treated with decompressive hemicraniectomy over a 3-year period with the following diagnoses: malignant infarction (3 patients), subarachnoid hemorrhage and vasospasm (2 patients), traumatic brain injury (3 patients), spontaneous intracerebral hemorrhage, and venous thrombosis. Neurological status was assessed by the Glasgow Coma Scale (GCS) and by the modified Rankin Scale (mRS) on admission, and by the mRS on discharge, and on follow-up examinations. Computerized tomography (CT) scans performed preoperatively and on the first, 7th postoperative days, and at one month were assessed for transtentorial herniation and midline shift. A decompressive hemicraniectomy, at least 14 by 11 cm was performed in all patients.

Results: There were three male and 7 female patients, ages 23 to 57 years. Neurological assessment preoperatively showed a median GCS score of 5.5, and hemiplegia in all patients, ipsilateral 3rd nerve palsy in eight of ten patients. Median mRS score on admission was 5. CT showed radiological signs of unilateral transtentorial herniation in every patient. Mean midline shift was 11.2 mm in preoperative CT scans and 3.2 mm on the first postoperative day. Median mRS score at the last follow up examination was 1.5 The overall mortality rate was 10 %. Six patients had favorable outcome (mRS <3).

Conclusion: The results of this series suggest that decompressive hemicraniectomy is a safe and effective technique that can be a life saving measure in patients with intractable intracranial hypertension and unilateral transtentorial herniation due to various causes.

Key words: Brain edema, craniectomy, decompressive surgery, intracranial pressure, transtentorial herniation

J Nervous Sys Surgery 2008; 1(3):161-168

Transtentoryal Herniasyondaki Hastalarda Dekompresif Hemikranyektomi

✓ **Amaç:** Unilateral transtentoryal herniasyonu olan hastalarda dekompresif hemikranyektominin sonuçlarını, tekniğini ve endikasyonlarını ortaya koymak.

Yöntem: Üç yıl içinde, medikal tedaviye dirençli intrakranyal hipertansiyonlu ve transtentoryal herniasyonda, habis infarct (3 olgu), subaraknoid kanama ve vazospazm (2 olgu), travmatik beyin hasarı (3 olgu), spontan intraserebral kanama ve venöz tromboz tanıları olan 10 ardışık hastaya dekompresif hemikranyektomi yapıldı. Nörolojik durum girişte Glasgow Koma Skalası (GKS) ve modifiye Rankin Skalası (mRS) ile, çıkışta ve kontrollerde mRS ile değerlendirildi. Operasyon öncesinde, 1. ve 7. postoperatif günlerde ve 1. ayda yapılan bilgisayarlı tomografi (BT) incelemeleri değerlendirildi. Hastaların hepsinde en az 14x11 cm'lik dekompresif hemikranyektomi yapıldı.

Bulgular: 23 ila 57 yaşları arasında üç erkek, yedi kadın hasta vardı. Operasyondan hemen önceki nörolojik değerlendirmeye göre medyan GKS skoru 5.5, mRS skoru 5'ti. Hastaların hepsinde hemipleji, sekiz tanesinde ipsilateral okülomotor felci vardı. BT incelemesi hastaların hepsinde unilateral transtentoryal herniasyon bulguları gösterdi. Ortalama orta hat itilmesi operasyon öncesi BT'lerde 11.2 mm iken, operasyon sonrası 1. gün BT'lerde 3.2 mm'ydı. Altı hastada iyi sonuç alındı (mRS <3), son kontrol muayenesinde medyan mRS 1.5'ti. Serinin mortalitesi % 10 oldu.

Sonuç: Bu çalışmanın sonuçları dekompresif hemikranyektominin çeşitli nedenlere bağlı dirençli intrakranyal hipertansiyonu ve unilateral transtentoryal herniasyonu olan hastalarda güvenilir ve hayat kurtaran etkili bir teknik olduğunu göstermektedir.

Anahtar kelimeler: Beyin ödemi, kranyektomi, dekompresif cerrahi, intrakranyal basınç transtentoryal herniasyon

J Nervous Sys Surgery 2008; 1(3):161-168

Refractory elevated intracranial pressure leading to transtentorial herniation is a life threatening condition with very high mortality. Increased intracranial pressure caused by expanding supratentorial mass lesions push the medial temporal lobe over the tentorial edge, compressing the oculomotor nerve, the brain stem, and the posterior cerebral artery, and if unchecked, lead to the death of the patient. Ipsilateral pupillary dilatation, decreased consciousness and coma, hemiplegia, and respiratory arrest are clinical signs of unilateral transtentorial herniation. Refractory edema leading to increased intracranial pressure and transtentorial herniation may result from a number of conditions including trauma, tumor, infarction, subarachnoid hemorrhage, intracerebral hemorrhage, and various inflammatory processes. When edema progression occurs despite medical measures and cerebrospinal fluid drainage, decompressive hemicraniectomy may improve survival. In this study we present our clinical experience with decompressive hemicraniectomy in patients with transtentorial herniation in various conditions including malignant middle cerebral artery (MCA) infarction, subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), and traumatic brain injury.

PATIENTS and METHODS

Patients

Ten consecutive patients with increased intracranial refractory to medical management treated with decompressive hemicraniectomy over a 3-year period (2005 to 2007) comprise this series. History was obtained from the patient, relatives, or medical personnel. Time of onset of symptoms were recorded. Medical history with particular attention to hypertension, antiplatelet use and anticoagulation was recorded. Neurological status was assessed by the Glasgow Coma Scale (GCS) and by the modified Rankin Scale (mRS) on admission, discharge and during follow-up examinations.

Computerized tomography (CT) in all cases and magnetic resonance (MR) images when possible were performed and the cause and extent of edema as well as signs of herniation were recorded. Midline shift was calculated by measuring displacement of the septum pellucidum from a line drawn between the most anterior and most posterior portions of the falx cerebri at the level of maximum displacement. Midline shift was measured and recorded preoperatively, and on the first, 7th postoperative days, and at one month. Unilateral transtentorial herniation was defined by the presence of the following five findings: i. ipsilateral effacement or obliteration of the suprasellar cistern, ii. brainstem displacement, iii. widening of the ipsilateral crural subarachnoid space, iv. effacement or obliteration of the contralateral perimesencephalic cisterns, and v. enlargement of the contralateral temporal horn (10,19).

Medical management included mannitol % 20 in all patients, dexamethasone in selected cases. Patients on anticoagulants and prolonged prothrombin times received 10 mg vitamin K and 15 ml/kg fresh frozen plasma. All patients received cephazoline intraoperatively.

Outcome at discharge and at follow up examinations was assessed by the mRS score.

Surgical Technique

A large question mark skin incision, beginning at the level of the zygoma, extending well behind the ear, then curving anteriorly over the sagittal suture, and crossing the midline to end at the hairline, three cm lateral to the midline, was made. The superficial temporal artery was protected to preserve the blood supply to the flap. The incision was carried through the subcutaneous layers and the temporalis muscle to the bone, and the muscle was reflected along with the skin flap. Six bur holes were then placed: 1. Inferiorly, posterior to the pterion, 2. Anterolaterally, lateral to the insertion of the

temporalis muscle, 3. Anteriorly 1 cm lateral to the midline, as low on the frontal bone as possible without entering the frontal sinus, 4. Medially,

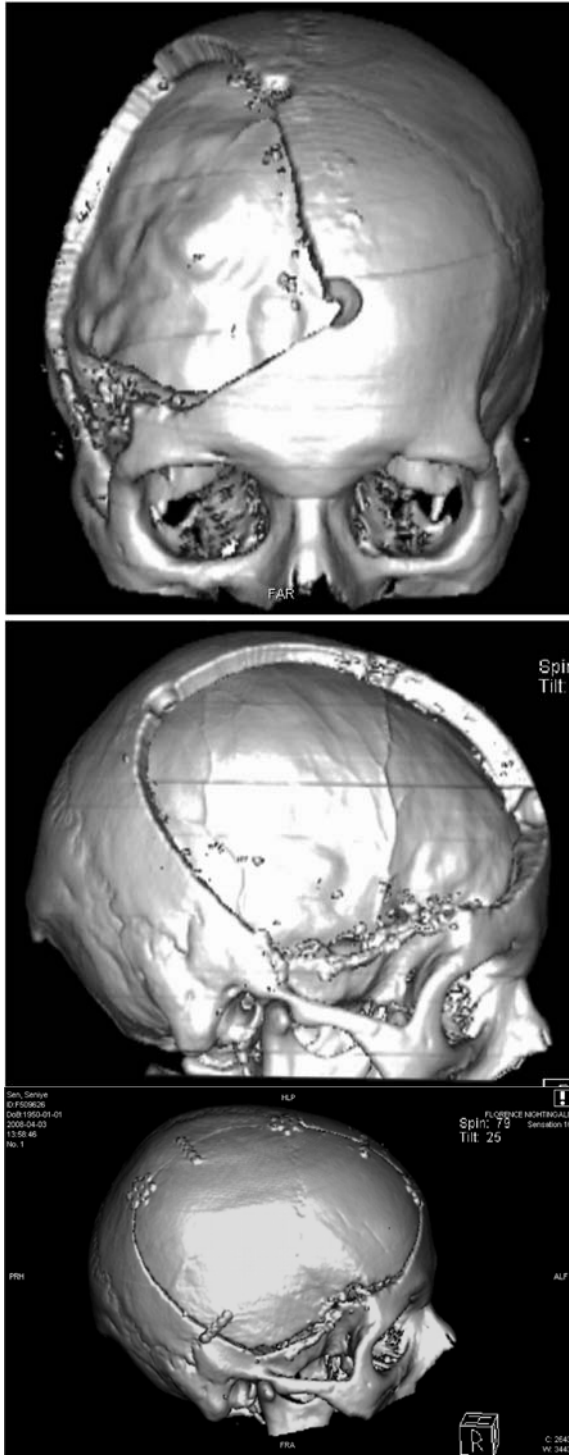


Fig 1. Three dimensional computerized tomography images showing hemicraniectomy. The lateral aspect (A), and anterior aspect (B), after reconstruction of the bone flap (C).

one cm lateral to bregma, 5. Posteriorly, posterior to vertex one cm lateral to the midline, and 6. four cm above and behind the external ear canal. A free bone flap that was at least 14 cm by 11 cm in dimensions was created with a power drill or Gigli saws. Laterally, the temporal bone was rongeuired down to the floor of the middle cranial fossa. Medially the edge of the craniotomy ran just off the midline assuring that the convexity was revealed. Anteriorly the bur hole low on the frontal bone assured that the anterior cranial fossa was adequately exposed. Medially and laterally, draining veins were protected by cottonoids. The dura is opened by multiple radial incisions, to provide maximal relaxation to the brain. Associated hematomas were evacuated when present. In two patients early in the series, the temporal pole and part of the mesial temporal lobe, including the herniated uncus, were removed. The durotomy was left open, and the cortex overlayed with collagen matrix or a dural substitute graft without suturing in all patients. The removed bone flap was kept frozen at -700 Celcius in nine patients, and embedded between the abdominal fat and muscle in one patient ⁽¹⁾. The bone flap was restored during the second postoperative month (Fig 1). Time of reconstruction was indicated by the relaxation of the brain as determined by inspection and palpation. At the time of reconstruction the temporalis muscle needs particular attention. It was dissected carefully from the skin flap and adhesions below, and attached to tension sutures over the bone. Dissection of the skin and muscle could usually be achieved without injuring the arachnoid, obviating the use of dural grafts. When the arachnoid was opened and there was free flowing cerebrospinal fluid, dural substitutes and fibrinogen glue were used to reconstruct the dura. The bone flap was fixated by plates and screws.

RESULTS

There were three male and seven female pati-

Table 1. Summary of clinical findings in 10 patients.

Age, Sex	Etiology	Neurological Examination	GCS	mRS			
		3. Nerve palsy	Hemiplegia	Initial	Initial	Discharge	Last FU
32, F	Venous thrombosis	-	+	8	4	2	0
54, F	SAH, vazospasm	+	+	4	5	4	3
38, F	Traumatic brain injury	+	+	9	5	3	1
23, M	Traumatic brain injury	+	+	4	5	5	4
36, M	Traumatic brain injury	+	+	6	5	2	1
49, F	SAH, ICH, vazospasm	-	+	10	5	4	1
52, M	Malignant infarction	+	+	5	5	4	4
57, F	Spontaneous ICH	+	+	6	5	3	0
57, F	Malignant infarction	+	+	5	5	6	6
59, F	Malignant MCA infarction	+	+	5	5	4	4
Mean 47				median 5.5	median 5	median 4	median 1.5

Abbreviations: FU, follow up examination; GCS, Glasgow coma scale; MCA, middle cerebral artery; mRS, modified Rankin scale; ICH intracerebral hematoma; SAH, subarachnoid hemorrhage

ents, ages 23 to 57 years (mean 45 years). The series included three patients with malignant infarctions (two patients with MCA and one patient with MCA and anterior cerebral artery infarction), two patients with SAH one of which had ICH and the other ischemia in the MCA territory, three patients with traumatic brain injury, one patient with spontaneous ICH, and one pregnant patient with antiphospholipid antibody syndrome and venous thrombosis.

Neurological assessment at the time of surgery showed a median Glasgow Coma Scale (GCS) score of 5.5 (range 4 to 10), and median mRS score of 5. All patients had clinical signs and symptoms of transtentorial herniation (eight of 10 patients had ipsilateral third nerve palsy and all patients had contralateral hemiplegia) (Table 1). Preoperative CT showed signs of unilateral transtentorial herniation in every patient (Fig 2). Mean midline shift was 11.2 mm in the preoperative CT scans.

CT examination on the first postoperative day showed a mean midline shift of 3.2 mm and resolution of uncal herniation in eight of 10 patients. On the seventh postoperative day midline shift persisted in three patients (mean 3 mm). Mean dimensions of hemicraniectomy as measured at CT was 14 by 10 cm (Fig 3).

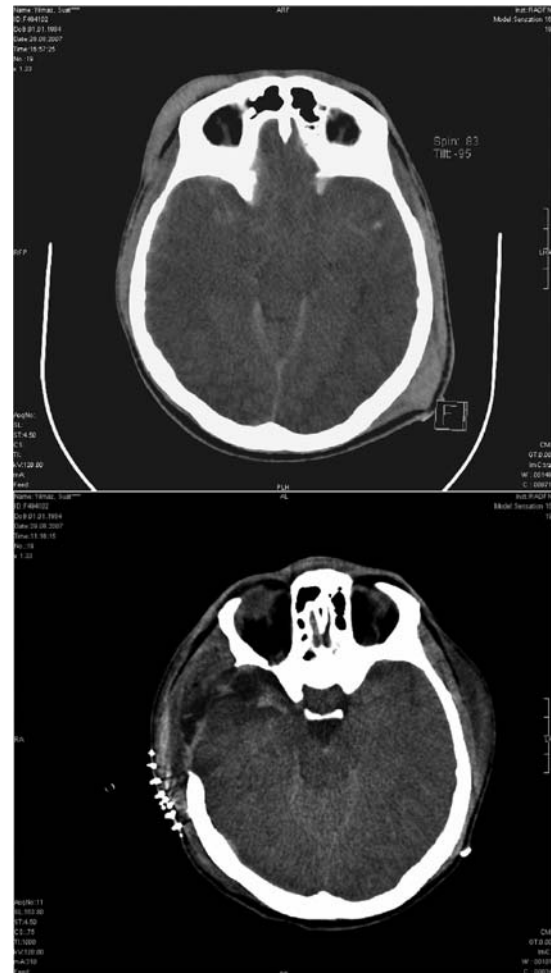


Figure 2. A. Preoperative computerized tomography shows transtentorial herniation. Note obliteration of the basal cisterns, medial displacement of the medial temporal lobe, distortion of the brain stem, and dilatation of the contralateral temporal horn. B. Postoperatively the basal cisterns become visible, the mesial temporal lobe and the brainstem have returned to their normal positions.

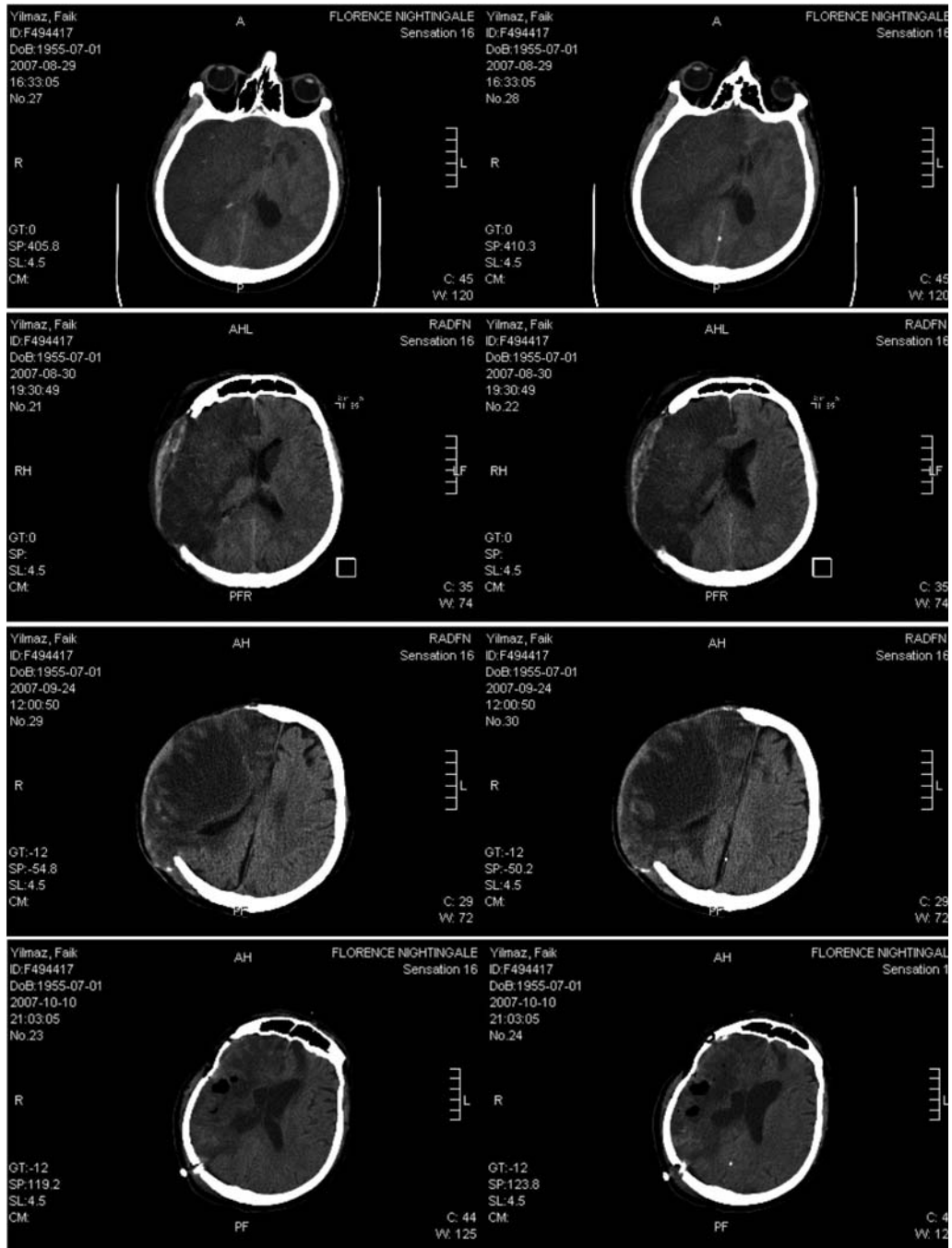


Figure 3. Computerized tomography images showing middle cerebral and anterior cerebral artery infarctions causing midline shift (A), postoperative scans show return of midline structures (B), edematous brain expands outside the skull (C), and cranium is reconstructed after resolution of edema (D).

One patient was treated for postoperative meningitis. This patient underwent bone flap resorption and cranioplasty two years postoperatively. Three patients developed subdural effusions requiring taps. One patient with malignant MCA

and anterior cerebral artery infarction developed ventricular enlargement as well as subdural effusion and required insertion of a ventriculo-peritoneal shunt. Another patient with malignant MCA infarction developed MCA occlusion in

the contralateral hemisphere and died. Surviving nine patients were followed for a mean of 14 months.

Neurological examination at discharge showed median mRS score of 4. One patient died, giving an overall mortality rate of 10%. At last follow up examination mean mRS score was 1.5. Six patients had favorable outcomes (mRS <3).

DISCUSSION

The Monro-Kellie doctrine states that the skull is a rigid compartment. It follows that expansion of the contents of the rigid skull, or addition of a mass lesion, leads to elevation of intracranial pressure (ICP). This, in turn impedes cerebral blood flow, leading to secondary ischemia and edema, further elevating the ICP, thus producing a vicious cycle, where increase in edema leads to increase in ICP and increased ICP leads to edema. The process terminates in herniation and death if allowed to propagate. When treatment with osmotic agents and CSF diversion, both directed to lower ICP by reducing intracranial contents, fail to control intracranial hypertension, removal of the rigid skull may break the vicious cycle. The brain can then expand without the untoward consequences anticipated by the Monro-Kellie doctrine, and without causing herniation^(6,15). Increased ICP may be due to various causes, such as tumor, trauma, hemorrhage, infarction, etc., but it always leads to the same process of herniation.

Medical management of transtentorial herniation attempts to reduce contents of the intracranial compartment mainly by the use of hyperosmolar agents such as mannitol and hypertonic saline, by drainage of cerebrospinal fluid and by hyperventilation. Studies evaluating the results of aggressive medical management of transtentorial herniation in intensive care facilities have shown that although these measures can reverse herniation, long term outcomes are bleak, with

only a small number of patients achieving good functional outcomes^(2,9,12).

The results of this series, as well as others, confirm that refractory edema and unilateral transtentorial herniation can be reversed with timely and judicious use of decompressive hemicraniectomy, allowing good functional outcomes. This series of patients with life-threatening transtentorial herniation includes patients with various pathologies, including malignant MCA infarction, ICH, SAH, venous infarction, and trauma.

The standard technique of hemicraniectomy employed in this series of patients requires that the bone flap be large, at least 14 by 10 cm, and extend down to the floors of the middle and anterior cranial fossae. The flap must also run close to the midline in order to effectively relieve bridging veins draining to the sagittal sinus. The dura was left open in every case in this series, without untoward consequences. Partial temporal lobectomy and uncalsectomy was performed early in the series. Clinical and radiological observation of reversal of herniation and return of midline structures in patients without uncalsectomy in later patients suggested that temporal lobectomy may not be necessary in most cases, and this practise was abandoned.

In this series, timing of surgery was determined by the appearance of clinical and radiological signs and symptoms of transtentorial herniation. Every patient in this series had contralateral hemiplegia and CT signs of herniation, and eight of 10 patients had pupillary dilatation. It has been noted that appearance of CT signs of herniation usually precede development of full clinical signs and symptoms⁽²⁰⁾. Once signs of herniation became evident, surgery was initiated as soon as possible. Timing of surgery is critical in that delay will lead to irreversible brainstem damage or infarction in the posterior cerebral artery area, as occurred in our patient no.2. On

the other hand, initiation of decompressive surgery in patients with MCA infarction or vasospasm before the appearance of signs of herniation may lead to otherwise avoidable surgical decompression in some patients. Age is another critical consideration in the decision to perform decompressive surgery. There is consensus among authors that results of decompressive surgery in malignant MCA infarction in the elderly are not satisfactory^(4,8,18). Most authors have chosen to offer surgery to patients younger than 60 years of age.

Decompressive surgery was first proposed by Kocher⁽¹⁵⁾ more than a century ago. Since then traumatic brain injury has been the most frequent indication for decompressive surgery^(4,7). Hemicraniectomy, bifrontal craniectomy and holo-craniectomy are performed to decompress posttraumatic intracranial hypertension^(3,11,16). All three of our patients with traumatic brain injury made excellent recoveries. Although good results for decompression after traumatic brain injury are reported by many centers, a prospective randomised study of is lacking⁽¹³⁾. On the other hand, many retrospective series of decompressions performed for MCA occlusion have reported good results, and recent randomised studies have established that in patients with malignant MCA infarction, decompressive surgery reduces mortality and increases the number of patients with a favorable functional outcome^(8,16-18). In a pooled analysis of three prospective randomised studies, the mortality rate of conservatively treated malignant MCA infarction in patients younger than 60 years was 71 %, whereas decompressive surgery reduced this to 22 %⁽¹⁸⁾. Several studies have suggested that decompressive craniectomy is also associated with good outcomes in SAH patients with ICH and vasospasm^(6,14,15).

In conclusion, our experience suggests that decompressive hemicraniectomy can reverse unilateral transtentorial herniation and lead to

good neurological outcomes when employed without delay in patients with unilateral transtentorial herniation.

REFERENCES

1. **Acikgoz B, Ozcan OE, Erbeni A, et al.** Histopathological and microdensitometric analysis of craniotomy bone flaps preserved between abdominal fat and muscle. *Surg Neurol* 1986; 26:557-61.
2. **Andrews BT, Pitts LH.** Functional recovery after traumatic transtentorial herniation. *Neurosurgery* 1991; 29:227-31.
3. **Bose B.** Emergency Decompressive Craniectomy for Traumatic Malignant Intracranial Hypertension. *Neurosurgery Quarterly* 2002; 12:171-81.
4. **Coplin WM, Cullen NK, Policherla PN, Vinas FC, MD, Wilseck JM, Zafonte, Rengachary SS.** Safety and Feasibility of Craniectomy with Duraplasty as the Initial Surgical Intervention for Severe Traumatic Brain. *Injury J Trauma* 2001; 50:1050-9.
5. **Curry WT, Sethi MK, Ogilvy CS, Carter BS.** Factors associated with outcome after hemicraniectomy for large middle cerebral artery territory infarction. *Neurosurgery* 2005; 56:681-92.
6. **Fisher CM, Ojemann RG.** Bilateral decompressive craniectomy for worsening coma in acute subarachnoid hemorrhage: Observations in support of the procedure. *Surg Neurol* 1994; 41:65-74.
7. **Hutchinson PJ, Kirkpatrick PJ.** Decompressive craniectomy in head injury. *Current Opinion in Critical Care* 2004, 10:101-4.
8. **Kilincer C, Asil T, Utku U, Hamamcioglu MK, Turgut N, Hicdonmez T, Simsek O, Ekuklu G, Cobanoglu S.** Factors affecting the outcome of decompressive craniectomy for large hemispheric infarctions: a prospective cohort study. *Acta Neurochir (Wien)*. 2005; 147:587-94.
9. **Koenig MA, Bryan M, Lewin JL, Mirski MA, Geocadin RG, Stevens RD.** Reversal of transtentorial herniation with hypertonic saline. *Neurology* 2008; 70:1023-9.
10. **Osborn AG.** Diagnosis of descending transtentorial herniation by cranial computed tomography. *Radiology* 1977; 123:93-6.
11. **Polin RS, Shaffrey ME, Bogaev CA, et al.** Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. *Neurosurgery* 1997; 41:84-94.
12. **Qureshi AI, Geocadin RG, Suarez JI, Ulatowski JA.** Long-term outcome after medical reversal of transtentorial herniation in patients with supratentorial mass lesions. *Crit Care Med* 2000; 28:1556-64.
13. **Sahuquillo J, Arkan F.** Decompressive craniectomy for the treatment of refractory high intracranial pressure in traumatic brain injury. *Cochrane Database Syst Rev*. 2006;CD003983. DOI: 10.1002/14651858.CD003983.
14. **Schirmer CM, Hoit DA, Malek AM.** Decompressive hemicraniectomy for the treatment of intractable intracranial hypertension after aneurysmal subarachnoid

- hemorrhage. *Stroke* 2007; 38:987-992.
15. **Smith ER, Carter BS, Ogilvy CS.** Proposed use of prophylactic craniectomy in poor grade aneurismal subarachnoid hemorrhage patients presenting with associated large sylvian hematomas. *Neurosurgery* 2002; 51:117-24.
 16. **Toussaint CP, Oritano TC.** Decompressive Craniectomy *Neurosurg Q* 2008; 18:45-53.
 17. **Tuncer N, Elmacı İ, Afşar N, Aktan S, Pamir N.** Akut İskemik İnmede Dekompresif Cerrahi Uygulaması. *J Nervous Sys Surgery* 2008; 1:20-5.
 18. **Vahedi Hofmeijer J, Juettler E, et al.** Early decompressive surgery in malignant infarction of the MCA: a pooled analysis of three randomised controlled trials. *Lancet Neurol* 2007; 6:215-22.
 19. **Wijdicks FM, Miller GM.** MR imaging of progressive downward herniation of the diencephalon. *Neurology* 1997; 48:1456-59.
 20. **Ziai WC, Port JD, Cowan JA, Garonik IM, Anish Bhardwaj A, Rigamonti D.** Decompressive Craniectomy for Intractable Cerebral Edema: Experience of a Single Center. *Journal of Neurosurgical Anesthesiology* 2003; 15:25-32.