

## Decompressive Craniectomy in Large Hemispheric Infarction

### Hemisferik Geniş Enfarktlarda Dekompresif Kraniektomi

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**Abstract: Objective:** Many cases of large hemispheric infarction are associated with early death due to massive edema, increased intracranial pressure, and subsequent uncal herniation. If medical therapy fails to lower intracranial pressure in a stroke patient, surgical decompression by large craniectomy may be indicated. The aim of this study was to determine the clinical characteristics of this fatal situation, and the results of surgical decompression.

**Methods:** We report a series of nine patients with large hemispheric ischemia who were treated by decompressive craniectomy in a 14-month period during 1999-2000.

**Results:** The five male and four female patients ranged in age from 55 to 76 years (mean, 64.4 years). Five patients had dominant, and four patients had non-dominant middle cerebral artery infarctions. Two patients also had anterior cerebral artery infarctions. The preoperative Glasgow Coma Scores ranged from 5 to 10 (mean, 7). Four patients had anisocoria, and all nine exhibited significant midline shift. After the operation, eight patients were alive at the end of the first month, and seven were alive after 6 months. At 6 months post-surgery, three of the survivors had Rankin Score 3, and the remaining four had Rankin Score 4 or 5.

**Conclusion:** Decompressive craniectomy effectively reduces mortality rates in patients with medically uncontrolled brain edema caused by large hemispheric infarction. However, functional outcome is still poor in these cases, and there is a high probability of lifetime dependence. This risk is particularly high in individuals older than 60 years who have low preoperative coma scores. Early surgery and careful patient selection may improve functional outcome of surgical management for large hemispheric infarction.

**Key Words:** Cerebral infarction, craniectomy, decompressive surgery

**Özet: Amaç:** Hemisferik geniş enfarktlı bir çok hasta yoğun ödem, artmış kafa içi basıncı ve bunu izleyen unkal herniyasyonla kaybedilir. Stroklu bir hastada kafa içi basınç medikal tedavi ile düşürülemezse geniş bir kraniektomi yoluyla cerrahi dekompresyon yapılabilir. Çalışmamızın amacı bu ölümcül durumun klinik özelliklerini ve cerrahi dekompresyon sonuçlarını ortaya koymaktır.

**Yöntemler:** 1999-2000 yıllarını kapsayan 14 aylık bir sürede dekompresif kraniektomi ile tedavi edilen dokuz hastalık bir seri sunulmaktadır.

**Bulgular:** Grup, yaşları 55 ile 76 arasında değişen (ort. 64,4) beş erkek, dört bayan hastadan oluşmaktadır. Beş hastada dominant, dört hastada non-dominant hemisferde orta serebral arter (MCA) enfarktı vardı. İki hastada ise ön serebral arter (ACA) enfarktları MCA enfarktına eklenmişti. Ameliyat öncesi Glasgow Koma Skorları 5 ile 10 (ort. 7) arasında değişmekteydi. Tüm hastalarda belirgin orta hat şifti, dördünde anizokori mevcuttu. Hastaların sekizi ilk ay, yedisi ise altıncı ay sonunda hayattaydı. Altıncı ayın sonunda sağ kalan hastaların üçü Rankin Skorlamasına göre 3, diğer dördü ise 4 veya 5 puan aldı.

**Sonuç:** Dekompresif kraniektomi hemisferik geniş enfarktlarda görülen ve medikal olarak kontrol edilemeyen yoğun ödeme bağlı mortalite oranlarının düşürülmesinde etkindir. Fakat fonksiyonel çıkış durumu halen kötüdür ve özellikle 60 yaşın üstündeki koma skorları düşük hastalarda bakıma muhtaç kalma olasılığı yüksektir. Erken operasyon ve dikkatli hasta seçimi cerrahi uygulanan hastalarda fonksiyonel sonuçları iyileştirebilir.

**Anahtar Kelimeler:** Serebral enfarkt, kraniektomi, dekompresif cerrahi

## INTRODUCTION

Ischemic stroke is not only the most prevalent condition in patients with disease of the brain, but also the most common cause of death in these patients. In large hemispheric infarction, massive edema is a major problem. Many of these cases are associated with early death due to raised intracranial pressure, significant midline shift, and, finally, uncal herniation. This phenomenon has been well documented in clinical observations and autopsy series (2,5,6,8,11,12,28,36). Hacke coined the term "malignant MCA [middle cerebral artery] infarction" for this condition (17). Although the reported mortality rates for unselected groups of patients with MCA infarction range from 30% to 60% (23,27,37), the death rate in patients who develop malignant hemispheric infarction is as high as 80%, even when maximum conservative intensive care is administered (4,6,17,32).

Decompressive craniectomy is a procedure in which the bone on one side of the skull is removed, and the dura is opened. Almost all studies have stressed the life-saving nature of this procedure, but there is no consensus on functional outcome. The series reported to date have been relatively small, and, because of the non-uniform nature of the studies, the information related to patients is insufficient. There is still debate about when, how, and in which cases to use this invasive therapeutic procedure for large hemispheric infarctions. There are dramatic policy differences among neurosurgical clinics around the world concerning the question of operating on these patients.

In this study, we report on nine patients who underwent decompressive craniectomy as treatment for medically uncontrolled edema due to large cerebral infarction. The aim is to contribute the clinical characteristics of this fatal situation, and the results of surgical decompression. Special emphasis was placed on the relationship between preoperative status and functional outcome in order to ascertain patient selection criteria that may help ensure better surgical results in future.

## PATIENTS AND METHODS

We performed decompressive craniectomy in nine patients with large hemispheric infarctions at Trakya University Medical School Hospital during the 14-month period from August 1999 to October 2000. Both dominant and non-dominant hemispheric

infarctions were included in the series. Each patient was observed and treated by a team of neurologists and neurosurgeons. Ischemic stroke was diagnosed by history, clinical examination, and computed tomographic (CT) scanning. Subsequent CT scanning was used to assess neurological deterioration and post-surgical developments in all cases.

### Patient Selection

As each patient's condition worsened with decreased level of consciousness, intravenous mannitol, diuretics, and steroids were administered. If the medical therapy failed, decompressive craniectomy was considered in consultation with the family. In all cases, the decision to perform decompressive craniectomy was based on the presence of a large space-occupying hemispheric infarction with midline shift and compression of the basal cisterns on CT, and on further neurological deterioration despite maximum medical management.

### Surgical Technique

The surgical procedure involved a reverse question mark-shaped skin flap based on the ear. A wide craniectomy was performed removing the frontal, temporal, and parietal bones. The dura was opened in stellate fashion to the extent of the craniectomy. A large pericranial graft was used to expand the dura. The bone flap was placed in a subcutaneous pocket overlying the abdomen for preservation until subsequent cranioplasty. All of the operations were performed by one of the authors (CK) alone, or under this author's supervision.

### Clinical and Neuroradiological Data

The patients' records were retrospectively reviewed, and data for age, sex, infarct etiology, and neurological and radiological findings were collected. Stroke etiology was determined according to TOAST criteria (1).

CT scans were reviewed by an independent neurologist (NT) who was blinded to follow-up imaging and clinical details. Particular attention was paid to early (first 12 hours) signs of infarction, specifically early hypodensity, lateral ventricle compression, hyperdense MCA sign (26), decreased corticomedullary contrast (CMC) (39), and effacement of the sylvian fissure and sulci.

For all cases, we recorded the time period between the stroke and operation, preoperative Glasgow Coma Scale (GCS) score (38), presence of

anisocoria, distribution of the infarct, hemorrhagic transformation, and midline shift at the septum pellucidum level on subsequent CT scanning.

At 4 weeks and 6 months after surgery, clinical outcome was assessed with the modified Rankin Score (RS) to rate physical disabilities in areas of mobility and self-care.

**RESULTS**

Tables I, II, III, and IV highlight the patient characteristics and outcomes in this series. The group consisted of five male and four female patients whose ages ranged from 55 to 76 years (mean, 64.4 years). The etiology of stroke was cardioembolism (in most cases associated with atrial fibrillation) in six patients, and atherothrombosis in three. Seven of the patients had complete MCA territory infarction, and two had internal carotid artery (MCA+ anterior cerebral artery) infarction. The stroke affected the dominant hemisphere in five patients, and the non-dominant

hemisphere in four. All the patients with dominant hemispheric infarction were global aphasic. All nine patients presented with severe hemiparesis, four had forced eye and head deviation, and two had nausea/ vomiting.

Early CT scans (first 12 hours) were obtained in seven cases. Effacement of cerebral sulci was the most frequent sign on these scans (six of the seven cases). Other findings were early hypodensity in more than 50% of the MCA territory (five of seven), effacement of the sylvian fissure (four of seven), lateral ventricle compression (three of seven), and decreased CMC (one of seven). None of these patients exhibited the hyperdense MCA sign.

Concerning the timing of the operation, seven of the nine patients deteriorated and underwent operation between the 24<sup>th</sup> and 48<sup>th</sup> hour of ictus, and only one did not begin to deteriorate until the 6<sup>th</sup> day. The preoperative GCS scores were between 5 and 10 (mean, 7), and four patients had unilaterally fixed

Table I: Patient characteristics

Patient No	Age	Sex	Etiology of Stroke	Distribution of Infarct	Dominant/ Non-dominant Hemisphere
1	69	M	Cardioembolism	MCA	Non-dominant (R)
2	67	M	Cardioembolism	MCA	Non-dominant (R)
3	55	M	Cardioembolism	MCA	Dominant (L)
4	61	F	Atherothrombosis	MCA	Dominant (R)
5	65	F	Atherothrombosis	MCA	Dominant (L)
6	76	F	Cardioembolism	MCA + ACA	Dominant (L)
7	67	M	Cardioembolism	MCA	Non-dominant (R)
8	63	F	Cardioembolism	MCA + ACA	Dominant (L)
9	57	M	Atherothrombosis	MCA	Non-dominant (R)

MCA: middle cerebral artery, ACA: anterior cerebral artery

Table II: Early CT signs of hemispheric infarction

Patient No	Early Hypodensity	Lateral Ventricle Compression	HMCA Sign	Decreased CMC	Effacement of sulci	Effacement of sylvian fissure
1	Yes	Yes	No	Yes	Yes	Yes
2	No	No	No	No	Yes	No
3	n/a*					
4	Yes	No	No	No	No	No
5	Yes	No	No	No	Yes	Yes
6	Yes	No	No	No	Yes	Yes
7	n/a*					
8	No	Yes	No	No	Yes	No
9	Yes	Yes	No	No	Yes	Yes

n/a\*: not applicable. Early (first 12 hours) CT scans were not obtained in these cases, HMCA: Hyperdense middle cerebral artery, CMC: Corticomedullary contrast.

Table III: Preoperative clinical and neuroradiological signs

Patient No	Preoperative GCS	Anisocoria	Hemorrhagic transformation	Midline shift (mm)	Time of Operation
1	7	No	No	4	48 <sup>th</sup> hour
2	7	No	Yes	12	48 <sup>th</sup> hour
3	8	No	No	15	6 <sup>th</sup> day
4	7	Yes	No	8	24 <sup>th</sup> hour
5	5	Yes	No	15	24 <sup>th</sup> hour
6	5	Yes	No	12	24 <sup>th</sup> hour
7	10	No	Yes	10	24 <sup>th</sup> hour
8	5	Yes	No	16	48 <sup>th</sup> hour
9	9	No	Yes	10	72 <sup>nd</sup> hour

GCS: Glasgow Coma Score

Table IV: Functional outcomes

Patient No	Time to Cranioplasty (week)	RS at 1 month post-op	RS at 6 months post-op
1	12	4	4
2	12	5	5
3	23	3	3
4	14	4	4
5	12	4	4
6	n/a	6	6
7	16	3	3
8	n/a	5	6
9	14	3	3

RS: modified Rankin Score, n/a: not applicable.

and dilated pupils. All showed significant midline shift (mean, 11 mm) at the septum pellucidum level. Three of the patients exhibited hemorrhagic transformation in the infarcted area.

One patient died on the third day post-surgery due to transtentorial herniation (Case 6), and another died in the third postoperative month due to cardiac problems (Case 8).

Overall neurological improvement varied among the seven survivors. Six months after the operation, three patients had RS 3, three had RS 4, and one had RS 5.

None of the patients who underwent craniectomy for stroke in the speech-dominant hemisphere were left with global aphasia. These individuals could not talk, but they were able to understand and communicate in a limited fashion.

The seven surviving patients returned for elective cranioplasty, most within 3-4 months of the original procedure.

## DISCUSSION

Severe life-threatening brain edema occurs in approximately 10% of patients with large hemispheric infarction (29). In these cases of malignant hemispheric infarction, the typical clinical course is that a space-occupying mass effect develops rapidly, and the patient deteriorates within the first 2-4 days (35). Most of the patients in our series deteriorated during the first 1-2 days, and four had signs of uncal herniation before the operation. Other symptoms of large hemispheric infarction including forced head/eye deviation and nausea/vomiting, were present in four and two of the individuals, respectively.

In accord with the literature, the majority (six) of our cases of large hemispheric infarction were associated with cardioembolism, and atherothrombosis was the next most frequent cause (three cases) (19,35). There are a number of characteristic early CT signs after large vessel occlusion (18,25,26,39,41). Research has identified early hypodensity in more than 50% of the MCA territory and local brain swelling (sulci

effacement, lateral ventricle compression) as strong predictors of unfavorable functional outcome (7,22,40). All of our patients had at least one of these early CT signs of infarction, the most frequent being effacement of sulci.

Frank hypothesized that hyperventilation and mannitol administration could potentially have a selective effect, lowering pressure to a greater extent in the uninjured hemisphere than in the injured hemisphere due to the intact blood-brain barrier on the undamaged side (16). If this is true, then the problems of pressure differential and midline shift would be augmented by conservative treatments. This may explain why patients with large hemispheric infarctions who have received maximum medical treatment still have high mortality.

Surgical decompression has been attempted in patients with elevated intracranial pressure from a variety of neurological disorders, including subdural hematoma, head trauma (3,30), cerebellar infarction (10,21), encephalitis (34), and space-occupying hemispheric infarction (9,13,31). The aims of this procedure are to allow the edematous tissue to expand away from the midline structures; reduce intracranial pressure; increase perfusion pressure; and preserve cerebral blood flow by preventing further compression of the collateral vessels. These factors may help to increase cerebral blood flow in areas surrounding ischemic regions, thereby preventing further brain tissue infarction. Some animal studies have shown that decompressive craniectomy for cerebral infarction significantly reduces mortality, improves clinical outcome (14,15), and even reduces infarct size if surgery is done early (14). In a non-randomized, controlled trial of decompressive craniectomy for large hemispheric infarction, in-hospital mortality was 76.2% in the non-surgically treated group and 34.4% in the surgically treated group (31).

The literature describes different techniques and anatomical limits for decompressive craniectomy. Some authors find that a limited craniectomy, including the frontal and temporal bones, is adequate (24). Others carry out a full hemicraniectomy that extends to the superior sagittal sinuses. In all our cases, we performed a large craniectomy that included the frontal, temporal, and parietal bones but spared the occipital bone; thus, we prefer the term "decompressive craniectomy" to "hemicraniectomy." We used pericranial fascia for duraplasty. This helps prevent cerebral spinal fluid leakage, and also facilitates subsequent cranioplasty by preventing adhesions

between the brain tissue and the subcutaneous layer. In each case, the removed bone was preserved in a pocket of abdominal subcutaneous tissue. We find this to be an easy, economical, and safe method of preservation for subsequent cranioplasty.

There is some controversy about the timing for this operation. Decompressive craniectomy is a life-saving procedure, but needs to be done before the brainstem sustains irreversible damage. It has been suggested that early decompressive craniectomy (that is, surgery prior to signs of herniation) improves outcome (35). On the other hand, surgery should not be performed too early in patients who have a good chance of recovering with conservative therapies. Of our nine patients, four exhibited preoperative anisocoria, and the only patient who suffered early death due to transtentorial herniation had this sign (Case 6). In contrast, the patients who had independent-outcome scores (RS 3) did not have anisocoria preoperatively (Cases 3, 7, and 9). These latter individuals also had higher preoperative GCS scores (GCS 8, 10, and 9, respectively) than the others. All the surviving patients who had poor Rankin results (RS 4 and 5) had low preoperative GCS scores (one GCS 5, three GCS 7), and two of them had anisocoria preoperatively.

Many prior studies on decompressive craniectomy have only investigated surgery on the non-dominant hemisphere. However, one report noted that, in patients with speech-dominant hemispheric infarction who were treated by decompressive surgery, the aphasia partially resolved, and follow-up studies revealed only mild to moderate aphasia (35). In our series, five of the patients had dominant hemispheric infarction, and all of these individuals had global aphasia preoperatively. At 6 months post-surgery, three of these five patients were alive, were able to understand verbal messages, and had some communication skills. These improvements are in line with previously published findings.

Reports in the literature differ with respect to the functional outcome of decompressive surgery. Many clinical studies have emphasized that this procedure reduces mortality, and some have concluded that it improves outcome as well (9,13,31,33,35). Age is an important predictor of functional outcome. Carter stated that the best recoveries were observed in patients younger than 50 years, and that recovery to a state of near-independence is possible in these patients (9). However, other authors have concluded that most patients will require extensive rehabilitative therapy

and lifelong assistance (13). Holtkamp et al. performed 12 decompressive craniectomies in patients over the age of 55, and reported that none of the eight survivors had a RS below 4 (20). All of the patients in our series were older than 55 years. At 6 months post-surgery, three of the survivors patients were RS 3, indicating independent status, but the other four patients were RS 4 and 5. The former group was slightly younger (mean age 59.7 years) than the latter (mean age 65.5 years).

### CONCLUSION

Assessing the outcome of decompressive craniotomy in our nine elderly (older than 55 years) patients, seven individuals were alive at 6 months post-surgery, and three of the seven were functionally independent. These results are significantly superior to those associated with conservative treatment. On the other hand, four of the surviving patients were functionally dependent at the 6-month stage, and were likely to remain so for life. We view decompressive craniectomy as a life-saving procedure that sometimes yields good functional outcome. However, since there is significant likelihood of lifetime dependence in these cases, and since it is not possible to obtain informed consent, there is a moral issue associated with operating on these patients. Our series was too small to provide statistically significant results, but the numbers suggest that age older than 60 years, presence of anisocoria, and GCS below 8 are positive predictors of a poor functional outcome. Early surgery and careful patient selection may improve the functional outcome of surgical management for large hemispheric infarction.

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### REFERENCES

- Adams HP, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL, Marsh EE: Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke* 24: 35-41, 1993
- Adams JH, Graham DI: Twelve cases of fatal cerebral infarction due to arterial occlusion in the absence of atheromatous stenosis or embolism. *J Neurol Neurosurg Psychiatry* 30: 479-488, 1957
- Andrews B, Pitts L: Functional recovery after traumatic transtentorial herniation. *Neurosurgery* 29: 227-231, 1991
- Berrouschot J, Sterker M, Bettin S, Koster J, Schneider D: Mortality of space-occupying ('malignant') middle cerebral artery infarction under conservative intensive care. *Intensive Care Med* 24(6): 620-623, 1998
- Berry RG, Alpers BJ: Occlusion of the carotid circulation: pathological consideration. *Neurology* 7: 233-237, 1957
- Bounds JV, Wiebers DO, Whisnant JP, Okazaki H: Mechanism and timing of deaths from cerebral infarction. *Stroke* 12: 474-477, 1981
- Buttner T, Uffmann M, Gunes N, Koster O: Early CCT signs of supratentorial brain infarction: clinico-radiological correlations. *Acta Neurol Scand* 96(5): 317-323, 1997
- Carter AB: *Cerebral Infarction*. McMillan Publishing Co, New York, 1964, pp 245-268
- Carter BS, Ogilvy CS, Candia GJ, Rosas HD, Buonanno F: One-year outcome after decompressive surgery for massive nondominant hemispheric infarction. *Neurosurgery* 40: 1168-1176, 1997
- Chen HJ, Lee TC, Wei CP: Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke* 23(7): 957-961, 1992
- Clarke E, Harris P: Thrombosis of the internal carotid artery simulating an intracranial space-occupying lesion. *Lancet* 1: 1085-1089, 1958
- Cooper ES, Ipsen J, Brown HD: Determining factors in the prognosis of stroke. *Geriatrics* 18: 3-9, 1963
- Delashaw JB, Broaddus WC, Kassell NF, Haley EC, Pendleton GA, Vollmer DG, Maggio WW, Grady MS: Treatment of right hemispheric cerebral infarction by hemicraniectomy. *Stroke* 21: 874-881, 1990
- Doerfler A, Forsting M, Reith W, Staff C, Heiland S, Schäbitz WR, von Kummer R, Hacke W, Sartor K: Decompressive craniectomy in a rat model of "malignant" cerebral hemispheric stroke: experimental support for an aggressive approach. *J Neurosurg* 85: 853-859, 1996
- Forsting M, Reith W, Schäbitz WR, Heiland S, von Kummer R, Hacke W, Sartor K: Decompressive craniectomy for cerebral infarction. An experimental study in rats. *Stroke* 26: 259-264, 1995
- Frank JI: Large hemispheric infarction, deterioration, and intracranial pressure. *Neurology* 45: 1286-1290, 1996
- Hacke W, Schwab S, Horn M, Spranger M, De Georgia M, von Kummer R: "Malignant" middle cerebral artery infarction: clinical course and prognostic signs. *Arch Neurol* 53: 309-315, 1996
- Haring HP, Dilitz E, Pallua A, Hessenberger G, Kampfl A, Pfausler B, Schmutzhard E: Attenuated corticomedullary contrast: An early cerebral computed tomography sign indicating malignant middle cerebral artery infarction. A case-control study. *Stroke* 30(5): 1076-1082, 1999
- Heinsius T, Bogousslavsky J, Van Melle G: Large infarcts in the middle cerebral artery territory. Etiology and outcome patterns. *Neurology* 50(2): 341-350, 1998
- Holtkamp P, Buchheim K, Unterberg A, Hoffmann O, Schielke E, Weber JR, Masuhr F: Hemicraniectomy in elderly patients with space occupying media infarction: Improved survival but poor functional outcome. *Journal of Neurology Neurosurgery and Psychiatry* 70(2): 226-228, 2001

21. Hornig CR, Rust DS, Busse O, Jauss M, Laun A: Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke* 25(2): 372-374, 1994
22. Horowitz SH, Zito JL, Donnarumma R, Patel M, Alvir J: Clinical-radiographic correlations within the first five hours of cerebral infarction. *Acta Neurol Scand* 86(2): 207-214, 1992
23. Kalia KK, Yonas H: An aggressive approach to massive middle cerebral artery infarction. *Arch Neurol* 50: 1293-1297, 1993
24. Kondziolka D, Fazl M: Functional recovery after decompressive craniectomy for cerebral infarction. *Neurosurgery* 23: 143-147, 1988
25. Krieger DW, Demchuk AM, Kasner SE, Jauss M, Hantson L: Early clinical and radiological predictors of fatal brain swelling in ischemic stroke. *Stroke* 30(2): 287-292, 1999
26. Moulin T, Cattin F, Crepin-Leblond T, Tatu L, Chavot D, Piotin M, Viel JF, Rumbach L, Bonneville JF: Early CT signs in acute middle cerebral artery infarction: predictive value for subsequent infarct localization and outcome. *Neurology* 47: 366-375, 1996
27. Moulin DE, Lo R, Chiang J, Barnett HJM: Prognosis in middle cerebral artery occlusion. *Stroke* 16: 282-284, 1985
28. Ng L, Nimmannitya J: Massive cerebral infarction with severe brain swelling: a clinicopathological study. *Stroke* 1: 158-163, 1970
29. Plum F: Brain swelling and cerebral edema in cerebral vascular disease. *Res Publ Assoc Res Nerv Ment Dis* 41: 318-348, 1961
30. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, Jane JA: Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. *Neurosurgery* 41: 84-94, 1997
31. Rieke K, Schwab S, Krieger D, von Kummer R, Aschoff A, Hacke W: Decompressive surgery in space occupying hemispheric infarction: results of an open, prospective study. *Crit Care Med* 23: 1576-1587, 1995
32. Ropper AH, Shafran B: Brain edema after stroke: clinical syndrome and intracranial pressure. *Arch Neurol* 41: 26-29, 1984
33. Sakai K, Iwahashi K, Terada K, Godha Y, Sakurai M, Matsumoto Y: Outcome after external decompression for massive cerebral infarction. *Neurol Med Chir (Tokyo)* 38(3): 131-135, discussion 135-136, 1998
34. Schwab S, Jünger E, Spranger M, Dörfler A, Albert F, Steiner HH, Hacke W: Craniectomy: an aggressive approach in severe encephalitis. *Neurology* 48: 412-417, 1997
35. Schwab S, Steiner T, Aschoff A, Schwarz S, Steiner HH, Jansen O, Hacke W: Early hemicraniectomy in patients with complete middle cerebral artery infarction. *Stroke* 29: 1888-1893, 1998
36. Shaw CM, Alvord EC, Berry GR: Swelling of the brain following ischemic infarction with arterial occlusion. *Arch Neurol* 1: 161-177, 1959
37. Steiger HJ: Outcome of acute supratentorial cerebral infarction in patients under 60: development of a prognostic grading system. *Acta Neurochir (Wien)* 111: 73-79, 1991
38. Teasdale G, Jennett B: Assessment of outcome after severe brain damage. *Lancet* 1: 480-484, 1991
39. Tomura N, Uemura K, Inugami A, Fujita H, Higano S, Shishido F: Early CT finding in cerebral infarction: obscuration of the lentiform nucleus. *Radiology* 168: 463-467, 1988
40. Turgut N, Utku U, Ekuklu G, Kılınçer C: Geniş supratentorial enfarktlerde erken dönem klinik ve BT bulguları. *Türk Beyin Damar Hastalıkları Dergisi* (In Press)
41. Ying KS, Pang KK, Huang JK, Lin JC: Early CT findings of acute cerebral artery territory. *Chung Hua I Hsueh Tsa Chih (Taipei)* 49(4): 223-30, 1992

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*What is effective in malignant middle cerebral artery infarction: reperfusion, craniectomy, or both? An experimental study in rats.*

*Engelhorn T, von Kummer R, Reith W, Forsting M, Doerfler A.*

Early reperfusion and craniectomy at 1 hour are both effective in large MCA infarction. While reperfusion later than 1 hour was not beneficial, late craniectomy at 4 and 12 hours still resulted in significant improvement of neurological score and reduction of infarction size. Combined treatment at different time points yields no significant additional benefit compared with 1 treatment at a time.

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*Hemicraniectomy in elderly patients with space occupying media infarction: improved survival but poor functional outcome.*

*Holtkamp M, Buchheim K, Unterberg A, Hoffmann O, Schielke E, Weber JR, Masuhr F.*

Craniectomy in elderly patients with space occupying MCA infarction improves survival rates compared with medical treatment alone. However, functional outcome and level of independence are poor. Craniectomy in elderly patients should not be performed unless a prospective randomised trial proves beneficial.