A decompressive craniectomy and expansive duraplasty is required when medical management is unable to control malignant brain swelling due to a massive cerebral infarction or severe head injury. However, such external decompression based on opening the cranial vault to increase the volume of the cavity is limited by extradural structures, in particular the temporal muscle and fascia.

Severe brain swelling after an infarction involving the entire distribution of the internal carotid or middle cerebral artery requires maximum decompression to decrease the intracranial pressure and allow for an increase in the cerebral perfusion pressure, thereby aiding the blood flow to the ischemic penumbra. However, if external decompression by itself can obtain optimal results, then internal decompression, including resection of the infarcted brain tissue and/or temporal lobe, should not be conducted because of the difficulty in differentiating between salvageable ischemic and irreversibly infarcted brain tissue.

Thus, for the surgical treatment of a malignant hemispheric infarction, we included resection of the temporal muscle and fascia with a conventional decompressive craniectomy to maximize the external decompression. To our knowledge, this is the first report on a surgical technique for external decompression that includes temporal muscle resection on a routine basis.

**Key Words** • cerebral edema • cerebral infarction • decompressive craniectomy • mastication • temporal muscle
Methods

Fifteen consecutive patients (8 men and 7 women; age range 33–73 years [mean age 56 years]) with a large hemispheric infarction underwent resection of the temporal muscle and fascia in addition to a decompressive craniectomy and expanded duraplasty at 2 tertiary hospitals, Kyungpook National University Hospital and Dongsan Medical Center in Daegu, Republic of Korea, between October 2006 and August 2007.

Operative Technique

During the scalp elevation after a large question-mark incision had been made, care was taken to avoid the frontal branch of the facial nerve, by using an interfascial dissection technique, and to keep the deep layer of the temporal fascia and pericranial flap intact. The temporal muscle and its fascia, along with a generous pericranial flap, were elevated from the temporal fossa (Fig. 1). The muscle was then excised at the level of the floor of the middle cranial fossa (Fig. 2). During the procedure, the supply arteries to the temporal muscle (including anterior and posterior deep temporal arteries from the internal maxillary artery and middle temporal artery from the superficial temporal artery) were coagulated using a bipolar cautery.

A hemicraniectomy was performed, which involved the removal of a large frontotemporoparietal bone flap with a minimum diameter of 12 cm. The temporal squama was drilled out until the floor of the middle cranial fossa was exposed. The bone flap was made anteriorly to avoid violation of the frontal sinus, except in the case of a huge frontal sinus. The posterior limit of the bone flap was ~ 5 cm posterior to an external auditory canal, whereas the medial limit of the craniectomy was ~ 2 cm from the midline.

Following a dural incision, the infarcted brain was not resected at all, although intracerebral hematoma was removed, if present. The expansive duraplasty was then performed using a large flap of pericranial tissue and temporal fascia. Otherwise, an artificial dura mater substitute was used instead. Meticulous hemostasis with multiple dural tacking sutures and bipolar coagulation of the dural surface was crucial to prevent a postoperative epidural hematoma. After placing a closed suction drain in the epidural/subgaleal space, the scalp was closed in 2 layers. The bone flap was stored in a tissue bank at −70°C.

The cranioplasty was performed using the autogenous bone 2–3 months after the craniectomy. For reconstruction of the temporal muscle defect, a MEDPOR implant (MEDPOR FLEXBLOCK TF2; Porex Surgical, Inc.) was successfully used (Fig. 3).

Results

The volume of the maximum external herniation that was measured on the basis of a CT volumetry study ranged from 130 to 300 ml (mean ± standard deviation, 200 ± 64 ml) on postoperative Day 3.2 ± 1.5 (range 2–5 days postoperatively). The mean value represented a 2-fold volume expansion in comparison with the conventional decompressive craniectomy, and the greater the external herniation obtained by external decompression, the smaller the midline brain shift after surgery (Fig. 4).

All patients showed initial clinical improvement after surgery. However, 3 (20%) of the 15 patients died postoperatively due to rupture of an aortic dissection or repeated cardioembolism with extension of the infarction. The stroke-related mortality rate was low, at 13.3% (2 of 15 patients). At their 6-month follow-up visit, 9 (60%) of the 15 patients had a favorable outcome (1 patient with an mRS score of 2, and 8 patients with an mRS score of 3), whereas 3 (20%) of 15 had an unfavorable outcome (2 patients with an mRS score of 4, and 1 patient with an mRS score of 5).

The masticatory function was evaluated in 8 patients. Two of them reported eating problems, such as leakage and hoarding of food in the mouth, on the paralyzed side rather than on the side with the resected temporal muscle. The mouth opening was also limited (30 mm in one patient and 35 mm in the other), plus the maximal bite force on the paralyzed side was lower compared with that on the side with the resected temporal muscle (50 N vs 160 N in one patient; 150 N vs 200 N in the other).

For the other 6 patients, no change in their chewing ability was reported. The maximal mouth opening was also within a normal range (42 ± 2 mm [mean ± standard deviation]), plus the maximal bite force on the side with the resected temporal muscle (180 ± 41 N) was less than that on the paralyzed side (217 ± 63 N; p = 0.044, Wilcoxon signed-rank test) (odds ratio 0.85, 95% confidence interval 0.69–1.00). Only 2 patients showed a slight lateral deviation of the mandible on opening their mouth.
Decompressive craniectomy including temporal muscle resection

The cases in which the MEDPOR FLEXBLOCK TF2 implant was used produced the best cosmetic results with no temporal hollow, although the disadvantages included the cost and a hard consistency on palpation.

Discussion

The temporal muscle is a large, fan-shaped muscle that fills the temporal fossa and is externally covered by the temporal fascia, which is a tough aponeurosis. After a craniectomy, the muscle becomes swollen, thereby limiting the external herniation of the brain with its tough fascia. Then, a limited external herniation of the temporal lobe reduces brainstem decompression. In particular, hemorrhagic suffusion of the temporal muscle can sometimes cause inadequate decompression (Fig. 5). Accordingly, the proposed temporal muscle resection technique is intended to increase the elasticity of the extradural soft tissue to allow free external herniation. In addition, it eliminates one of the important risk factors for epidural/subgaleal hematomas, a hemorrhagic temporal muscle, and also technically allows better subtemporal bone decompression without hindrance of the temporal muscle.

The temporal muscle originates from the bone surface of the temporal fossa and passes medial to the zygomatic arch, inserting into the coronoid process of the mandible. Thus, it is a powerful elevator of the mandible, retracts the mandible using the horizontal fibers of the posterior part of the muscle, and coordinates the closing movements of the mandible as a positioning muscle. The masticatory muscles that produce the bite force include the masseter and medial pterygoid muscles in addition to the temporal muscle. The masseter and medial pterygoid form a muscular sling that supports the mandible at the mandibular angle and provides the force necessary to chew efficiently.\(^3\)

Mobilization of the temporal muscle is common in neurosurgical procedures, such as pterional and orbitozygomatic craniotomies. Damaging the temporal muscle in such procedures causes muscle atrophy with disfigurement, contracture of the muscle with limited mouth opening, and pain. Thus, every effort is made to preserve the temporal muscle during the surgical procedures.\(^4\) In the present technique, although the resection of the temporal muscle produced a temporal hollow as a cosmetic sequela, it did not cause limited mouth opening or pain.

The unilateral weakness of masticatory muscles in patients with hemiplegia is clinically inconspicuous or limited to the acute period because the masticatory nucleus receives bilateral hemispheric innervation. Although the control of the contralateral hemisphere may be more dominant, as reported in a previous study by Cruccu et al.,\(^1\) in which electromyography studies demonstrated that the level at maximum strength was 25% lower on the paralyzed side compared with the contralateral side, the maximal bite force seems not to be considerably affected by a hemispheric infarction. When Kemppainen et al.\(^2\) evaluated the maximal bite force of 16 patients (mean age 53 years) after a hemispheric infarction and hemiplegia, the brain infarction did not induce any difference in the
maximal bite force between healthy and the paralyzed sides; the mean maximal bite force was 292 N on the paralyzed side and 296 N on the intact side.

In the present study, 2 of the 8 patients whose masticatory function was examined demonstrated a reduced bite force on the paralyzed side. In contrast, the other 6 patients demonstrated a somewhat lower maximal bite force on the side with the resected temporal muscle compared with that on the paralyzed side, yet had no problems with chewing, possibly because the grinding phase of the closure stroke only requires one-third of the maximal bite force. The force on teeth when chewing meat and carrots is only 70 N and 130 N, respectively.4 Meanwhile, the maximal bite force was lower on both the side with the resected temporal muscle and the paralyzed side in the present study, compared with the results of Kemppainen et al.2 One of possible reasons was a shorter postictal period, and thus less exposure to a diet containing tough foods for the patients in this study.

To prove the clinical benefits of the present technique clearly, a large prospective, randomized study that includes intracranial pressure, cerebral perfusion pressure, and cerebral blood flow measurements is necessary. In addition, further studies are needed to devise distinctive indicators for the proposed technique as opposed to conventional external decompression.
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Conclusions

Although preliminary, this study supports the suggestion that resection of the temporal muscle and fascia is an effective procedure to maximize external herniation in a decompressive hemicraniectomy. The proposed procedure would also seem to produce better clinical outcomes, with an acceptable cost of minimal masticatory dysfunction and cosmetic disfigurement.

Disclaimer

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Acknowledgment

The authors thank Dong-Ju Cho for her illustration (Fig. 2).

References


Manuscript received February 26, 2008. Accepted April 23, 2008. Please include this information when citing this paper: published online October 3, 2008; DOI: 10.3171/2008.4.17540.

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