Postcraniotomy Temporalis Muscle Atrophy: A Clinical, Magnetic Resonance Imaging Volumetry and Electromyographic Investigation

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Aims: *To evaluate both cosmetic and functional effects of temporalis muscle atrophy, by means of clinical examination, magnetic resonance imaging (MRI), and electromyographic (EMG) activity in patients who underwent craniotomy in order to treat refractory mesial temporal lobe epilepsy (MTLE).* ***Methods:*** *A total of 18 controls and 18 patients who underwent surgery for MTLE were investigated. The temporalis muscle volume of the patients was assessed by a 3D reconstruction. The image analysis software (ITK-SNAP) was used for the 3D reconstruction. In addition, the amplitude of the EMG signal during a maximum voluntary clench was recorded from both temporalis muscles by surface electrodes. The presence of temporomandibular disorder (TMD) signs was assessed by clinical examination that was performed only after surgery. Data were analyzed statistically by means of the Mann-Whitney* U *test, paired* t*-test, Pearson* χ^2 *and linear regression.* ***Results:*** *The volume of the temporalis muscle of the operated side was significantly reduced (*P *= .004). The EMG results confirmed the presence of muscle atrophy, the amplitude of the EMG signal being significantly decreased on the operated side (*P *< .05). Also the patients' maximum mouth opening after surgery was significantly reduced compared to that of the controls (*P *< .0001). Patients presented facial asymmetry, signs of TMD (pain, disc displacement, and joint sounds), and masticatory abnormalities.* ***Conclusion:*** *These preliminary results showed that, despite the good control of seizures, some patients may experience cosmetic and functional abnormalities of temporalis muscle secondary to atrophy and fibrosis.* J OROFAC PAIN 2010;24:391–397

Key words: craniotomy, electromyography, epilepsy surgery, magnetic resonance imaging, temporal muscle atrophy, volumetry

Surgical treatment for refractory mesial temporal lobe epilepsy (MTLE) has a better outcome compared to antiepileptic drugs.[1,2] Different surgical approaches (standard temporal lobectomy or selective transsyilvian amygdalohippocampectomy) may result in a similar surgical outcome.[3] Craniotomy can be performed according to different techniques, including the pterional,[4] pretemporal,[5,6] or other frontotemporal approaches. Besides different craniotomy options, the surgical management of the temporalis muscle and the facial nerve branches can also be carried out in different ways, ie, interfascial,[4] subfascial or submuscular,[7] and retrograde dissection.[8]

Considering the good results in seizure control, less attention has been directed to evaluate cosmetic side effects and masticatory impairment that may occur after craniotomy, such as temporalis muscle atrophy, as well as temporomandibular disorders (TMD) that can lead to pain and masticatory impairment, delaying the patient's return to previous activities.[8] In addition, some patients may even suffer from pseudoankylosis of the mandible after temporal or frontotemporal craniotomy, with severe reduction of mouth opening, increasing the difficulty in intubation in case of future surgeries.[9–11] These postoperative side effects are not exclusive of epilepsy surgery; they can arise after pterional and frontotemporal craniotomy that may be necessary in treatment procedures for other intracranial pathologies such as brain tumors or cerebral aneurysms.[12,13]

The aim of this study was to evaluate both cosmetic and functional effects of temporalis muscle atrophy, by means of clinical examination, magnetic resonance imaging (MRI), and recording of electromyographic (EMG) activity in patients who underwent craniotomy in order to treat refractory mesial temporal lobe epilepsy.

Materials and Methods

A total of 18 patients (13 female, 5 male, age 37.6 ± 11.5 years) and 18 controls (13 female, 5 male, age of 37 ± 11.7 years) were included. All patients were selected for surgery according to the authors' published protocol[2] and underwent surgical treatment for refractory MTLE in the authors' institution between 2002 and 2004. The control group was matched for age and gender and consisted of the hospital's employees who did not suffer epilepsy, headache, or TMD. The control group was used to obtain the maximal mouth opening value in the normal population, as the extent of mouth opening of the patients was not recorded preoperatively. Postoperative EMG recording and Research Diagnostic Criteria for TMD (RDC/TMD)[14] examination were carried out for each individual on the same day, and the postoperative MRI scans were acquired during the same month as these examinations. The study included a paired analysis between preoperative and postoperative MRI scans and analyzed exclusively postoperative EMG and RDC/TMD data.

Surgery

All patients underwent pretemporal craniotomy[5] with transsylvian selective amygdalohippocampectomy,[15] performed by two neurosurgeons (HT and EO). Temporalis muscle dissection was carried out according to Yasargil's interfascial dissection in order to maximize the visibility as well as preserve the facial nerve[4] without any additional incision of muscle. The temporalis muscle was reattached to a cuff of fascia-periostium complex on a free bone flap along the superior temporal line.

The patients were operated on between 2002 and 2004, and the mean follow-up period was 25.5 ± 10.8 months. After surgery, patients were classified in terms of seizure control according to Engel's classification of postoperative outcome:[16] Class I, free of disabling seizures; Class II, rare disabling seizures; Class III, worthwhile improvement; Class IV, no worthwhile improvement.

MRI Analysis

All patients underwent the same protocol for 3D acquisition on a 2T scanner (Elscint Prestige). T1-weighted images (TR = 22 ms, TE = 9 ms, flip angle = 35°, matrix = 256 × 220, field of view = 25 x 22 cm, sagittal acquisition) with 1 mm isotropic voxel. The same protocol was used for acquiring both preoperative and postoperative scans. The MRI evaluation was performed both preoperatively and postoperatively.

The images were acquired in DICOM format transformed to ANALYSE by MRIcro software. After format conversion, image analysis software (ITK-SNAP) was used to segment the temporalis muscle. ITK-SNAP is an interactive image segmentation software developed to implement an active contour segmentation of anatomical structures, allowing regional segmentation by employing user-initialized deformable implicit surfaces that evolve to the most appropriate border between neighboring structures.[17] Segmentation is the process by which appropriate image points (voxels) are assigned to be part of a specific anatomic structure.

The three views (coronal, sagittal, and axial) were used to define the boundaries of the temporalis muscle. Manual segmentation was performed with a computer mouse by one neurosurgeon (CLY) drawing a line around the muscle borders, enclosing the whole structure, on every single MRI slice. The operator selected the points to produce a visually appropriate tracing of the surface contour, following careful realignment of the region of interest. The segmentation of the temporalis muscle included both the superficial portion and the deep belly according to Geers et al.[18] The high-resolution images allowed the investigators to clearly identify and segment the temporalis muscles (bilateral) in preoperative and postoperative scans. Subsequent to the manual segmentation, a 3D-graphical rendering of the volumetric object allowed navigation between voxels in the volumetric image, enabling one to scroll through the data (Fig 1). The software provided the volume of those selected voxels in cubic mm. In order to assess whether surgery led to an ipsilateral atrophy of temporalis muscle, the ratio of the volume between the affected/nonaffected side was calculated.

EMG Recordings

The EMG recordings were performed by means of a Neuropack 2 Electromyographer (Nihon Kohden) during a maximum voluntary clench lasting 5 seconds. Surface electrodes were placed over the middle belly of the temporalis muscles (active) and malar prominence.[19] High and low filters were set to 5 kHz and 10 Hz, respectively. For each individual, analysis time was 200 ms. The amplitudes of the electrical activity were expressed in microvolts (μV). The degree of asymmetry in the EMG signal of both temporalis muscles was expressed as the ratio between the highest peak amplitude of the affected/nonaffected side. In normal individuals, this ratio is close to 1, since no significant differences exist between the EMG signal amplitude of the right and left side.[20]

Clinical Examination

After surgery, all patients were asked about TMD symptoms and evaluated by a trained and calibrated dentist according to a standardized clinical protocol (RDC/TMD axis I)[14] in order to assess the presence of TMD signs. The RDC/TMD examinations allow the diagnosis of the following: muscle disorders, disc displacement, arthralgia, osteoarthritis, and osteoarthrosis.[14] Facial asymmetry was checked visually. These evaluations were performed only after surgery, so the results are only the description of findings, as the authors could not compare the preoperative and postoperative conditions.

Maximum mouth opening, including overbite, was recorded from the patients and controls. The mouth opening was assessed between the edges of the upper and lower central incisor at the middle line.[21] The controls provided the mean value of maximal mouth opening of a normal population, as the maximum mouth opening of the patients was not recorded before surgery.

Statistical Analysis

Nonparametric Mann-Whitney *U* test compared data (age and maximum mouth opening) between patients and controls, and paired *t*-test compared preoperative and postoperative ratios obtained with manual segmentation of the temporalis muscles. Categorical variables were analyzed by means of Pearson χ^2. Pearson correlation analyzed the relationship between time after surgery (months) and the mouth opening degree (in millimeters). The statistical analysis was performed using SYSTAT 12 (Systat Software). The results are described as mean ± standard deviation; P values < .05 were considered statistically significant.

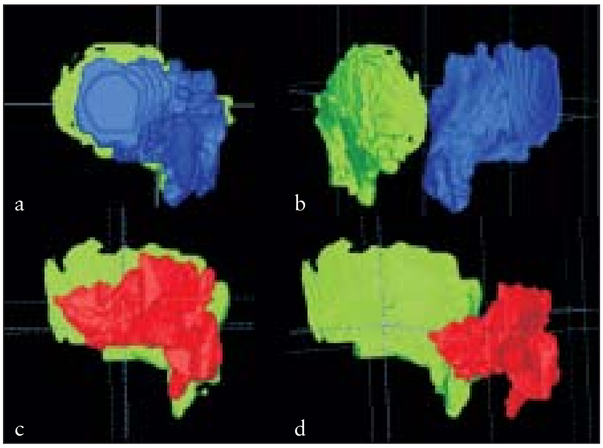

Fig 1 A 3D view of both temporal muscles of one patient, with presurgery view (*a* and *b*, the muscle in blue is the affected side before the procedure) and postsurgery view (*c* and *d*; red is the operated muscle after surgery).

Results

There were no significant differences between patients and controls as far as age (P = .89) and gender (P = 1). The MRI, clinical (RDC/TMD), and EMG evaluations were performed within 25.5 ± 10.8 (mean ± standard deviation) months after surgery. Sixteen patients were classified as Class I according to Engel's postoperative outcome scale, one patient as Class II, and one patient as Class III.[22] These results showed that 89% of these patients presented excellent control of seizures after surgery. None of the patients had facial palsy secondary to facial nerve injury.

The volume of temporalis muscle was significantly reduced after surgery. The ratio before surgery was 0.99 ± 0.07, and the ratio after surgery was 0.86 ± 0.15 (P = .004) (Table 1, Fig 2).

The EMG findings supported the volumetric data. Temporalis muscle atrophy was so severe in five individuals that it was impossible to record EMG activity with surface electrodes. In the remaining 13 patients, the ratio was 0.55 ± 0.20, indicating that the EMG activity of the operated side was nearly half that of the normal side (Table 1).

Table 1 Data from Patients

Patients	EMG (ratio)	Temporalis muscle ratio (presurgical)	Temporalis muscle ratio (post-surgical)	Time of evaluation (months after surgery)	Postsurgical mouth opening (mm)
1	0.545	0.89	0.99	40	42
2	0.333	1.14	0.77	14	39
3	0.333	0.94	0.71	24	36
4	0.281	1.0	0.67	24	39
5	0.75	1.1	1.0	11	36
6	0.5	0.91	0.99	39	55
7	0.583	0.97	0.69	34	40
8	0.384	0.99	1.01	24	30
9	NA	0.95	1.08	36	38
10	0.705	0.99	0.65	24	35
11	0.6	0.98	0.84	12	36
12	NA	0.95	0.68	36	38
13	NA	1.0	0.67	36	39
14	0.9	0.92	1.03	27	42
15	NA	0.98	0.85	9	34
16	0.83	1.0	0.95	36	39
17	0.416	1.0	0.94	9	37
18	NA	1.1	0.89	24	42

EMG ratio obtained from EMG activity on operated side/contralateral side; temporalis muscle ratio (volume of affected side/volume of contralateral side). NA = no activity detected on affected side.

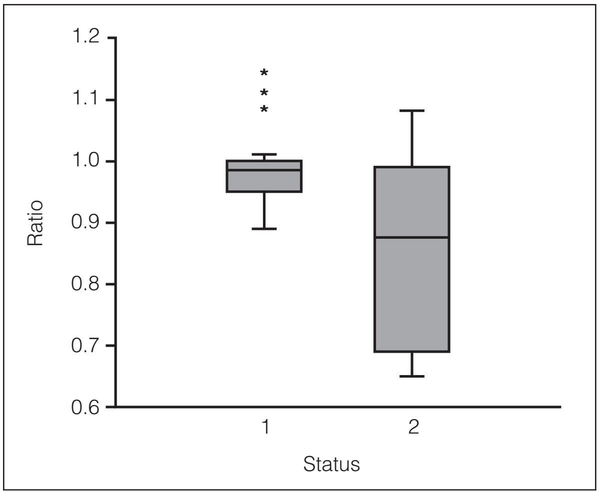

Fig 2 Box plot showing the ratio between the volumes of the normal and operated muscle. 1 = presurgery (0.99 ± 0.07); 2 = postsurgery (0.86 ± 0.15), $P = .004$.

The maximal mouth opening was significantly larger in the controls (46.6 ± 5.1 mm) than in patients (38.7 ± 6.9, $P < .0001$) (Fig 3). The overbite assessment was balanced ($P = 1$) between controls (2.22 ± 0.44 mm) and patients (2.16 ± 0.33 mm). A significant correlation ($P = .033$, $r = 0.504$) was found between the degree of mouth opening and time after surgery (Table 1, Fig 4).

Ten (55.6%) patients presented facial asymmetry due to temporalis muscle atrophy. Muscle disorders were found in 11 (61.1%) patients, including pain in response to palpation (7 patients) and spontaneous pain (4 patients). Seven patients had tenderness to palpation of the ipsilateral temporalis muscle, 5 of the ipsilateral masseter, 1 of the ipsilateral medial pterygoids, and 1 of the ipsilateral lateral pterygoids muscle; some patients presented pain in more than one muscle. Spontaneous pain was reported in the ipsilateral temporalis muscle by 2 patients and in the ipsilateral masseter muscle also by 2 patients.

Twelve patients presented joint sounds, and 11 of these also presented disc displacement with reduction ipsilateral to the surgery. Temporomandibular joint (TMJ) pain during palpation was recorded in 5 patients.

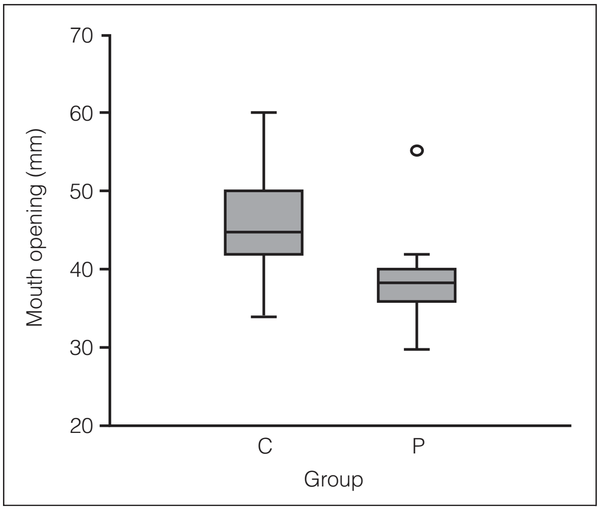

Fig 3 Box plot of the maximum mouth opening for controls (C) and patients (P). Controls = 46.6 ± 5.1 mm, patients = 38.7 ± 6.9 mm; *P* < .0001.

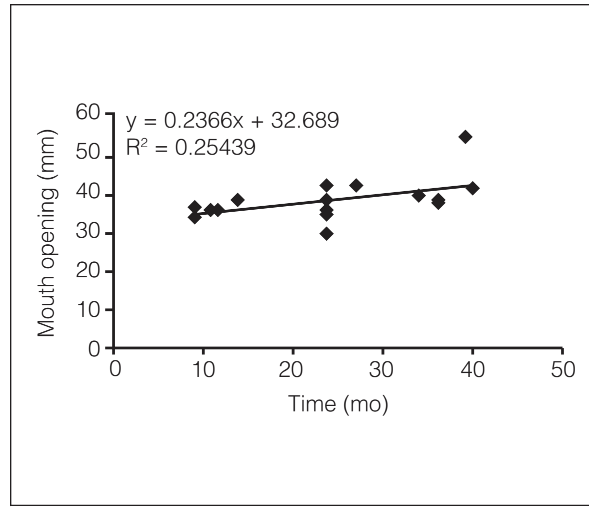

Fig 4 Relationship between mouth opening (mm) and time after surgery (months).

Discussion

This preliminary study showed that despite excellent postoperative surgical outcome (89% of patients were free of disabling seizures), some patients who underwent surgery for epilepsy developed cosmetic and even functional abnormalities due to temporalis muscle atrophy. Such effects are not exclusively related to epilepsy surgery as they are reported to result from the craniotomy procedure itself and can therefore occur also with other craniotomies performed during procedures to treat brain tumors and aneurysms.[12,23,24] Since epilepsy surgery aims to offer better quality of life through seizure control,[25] attention should be directed also to study the side effects resulting from temporalis muscle atrophy in order to avoid them.

Temporalis muscle atrophy and fibrosis secondary to craniotomy have been recognized previously,[9,11,26,27] but no volumetric or EMG evaluation has been provided. In spite of the small sample size, the present results from both volumetric and EMG data clearly confirmed the volume decrease and the functional impairment of the operated temporalis muscle, a finding that has already been reported.[12,13] In some cases, the atrophy was so intense that it was impossible to detect EMG activity. Clinically, facial asymmetry, disc displacement, and spontaneous and elicited pain during palpation were observed.

The patients' maximum mouth opening was not measured preoperatively. Comparison with controls showed that the patients' maximum opening was still significantly reduced 2 years after surgery, confirming previous findings.[11] The reduction is reported to be due to a combination of scar formation inside the muscle after incision, devascularization (due to sustained traction during the surgery) and, in some cases, the organization of hematoma.[9,11] The mouth opening reduction showed a slight tendency to improve over time, although time explained only 25% of the mouth opening increase ($r = 0.504$), meaning that other factors were also involved in this process. A longer follow-up period with a larger number of operated patients is required to determine whether or not patients may be able to recover entirely to their preoperative mouth opening.

The risk of pseudoankylosis with severe mouth opening reduction is a great concern, considering that some cases of refractory seizures need to be reoperated, mainly those secondary to cortical dysplasia.[28,29] The risk of a difficult intubation during the second or third surgery would be minimized if the maximal opening mouth could be restored to normal.

Since the temporalis muscle is involved in mastication,[18,30] it is likely that the patients may experience some masticatory impairment, especially during the first postoperative months. This, along with the risk of a permanent reduction of maximum mouth opening, points to the necessity of an early implementation of both passive and active jaw exercises, as well as of alternative surgical approaches with minimal damage to the temporalis muscle. Indeed, several techniques for muscle management other than the interfascial dissection[4] have been developed, such as submuscular or subfascial dissection[31] described by Coscarella et al,[7] and retrograde dissection[8] in an attempt to preserve deep temporal arteries and nerves and avoid muscle atrophy. Other techniques have been suggested in order to provide a better reconstruction of the temporalis muscle after the intracranial procedure, ie, a small cuff of fascia-periostium complex attached to the free bone flap for muscle closure,[26] the creation of several small holes along the superior temporal line to reattach the temporalis muscle with sutures,[31] the use of titanium microscrews (3 mm) to reattach the muscle to the bone,[32] and the utilization of methylmethacrylate cement to fill the defect where extensive temporal bone resection took place and thus preserve the contour before the muscle reposition.[12]

Although temporalis muscle atrophy was found in several patients, it is unknown why some patients developed muscle atrophy and/or TMD while others achieved total recovery without TMD, although all were operated on with the same technique. Further prospective studies, including preoperative evaluation of a larger number of patients, are necessary to identify the risk factors for developing both temporalis muscle atrophy and temporomandibular dysfunction after the surgical procedure.

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References

1. Wiebe S, Blume WT, Girvin JP, Eliasziw M. A randomized, controlled trial of surgery for temporal-lobe epilepsy. N Engl J Med 2001;345:311–318.
2. Yasuda CL, Tedeschi H, Oliveira EL, et al. Comparison of short-term outcome between surgical and clinical treatment in temporal lobe epilepsy: A prospective study. Seizure 2006;15:35–40.
3. Arruda F, Cendes F, Andermann F, et al. Mesial atrophy and outcome after amygdalohippocampectomy or temporal lobe removal. Ann Neurol 1996;40:446–450.
4. Yasargil MG, Reichman MV, Kubik S. Preservation of the frontotemporal branch of the facial nerve using the interfascial temporalis flap for pterional craniotomy. Technical article. J Neurosurg 1987;67:463–466.
5. Seoane E, Tedeschi H, de Oliveira E, Wen HT, Rhoton AL Jr. The pretemporal transcavernous approach to the interpeduncular and prepontine cisterns: Microsurgical anatomy and technique application. Neurosurgery 2000;46: 891–898.
6. de Oliveira E, Tedeschi H, Siqueira MG, Peace DA. The pretemporal approach to the interpeduncular and petroclival regions. Acta Neurochir (Wien) 1995;136:204–211.
7. Coscarella E, Vishteh AG, Spetzler RF, Seoane E, Zabramski JM. Subfascial and submuscular methods of temporal muscle dissection and their relationship to the frontal branch of the facial nerve. Technical note. J Neurosurg 2000;92: 877–880.
8. Oikawa S, Mizuno M, Muraoka S, Kobayashi S. Retrograde dissection of the temporalis muscle preventing muscle atrophy for pterional craniotomy. Technical note. J Neurosurg 1996;84:297–299.
9. Kawaguchi M, Sakamoto T, Furuya H, Ohnishi H, Karasawa J. Pseudoankylosis of the mandible after supratentorial craniotomy. Anesth Analg 1996;83:731–734.
10. Kawaguchi M, Sakamoto T, Ohnishi H, Karasawa J, Furuya H. Do recently developed techniques for skull base surgery increase the risk of difficult airway management? Assessment of pseudoankylosis of the mandible following surgical manipulation of the temporalis muscle. J Neurosurg Anesthesiol 1995;7:183–186.
11. Coonan TJ, Hope CE, Howes WJ, Holness RO, MacInnis EL. Ankylosis of the temporo-mandibular joint after temporal craniotomy: A cause of difficult intubation. Can Anaesth Soc J 1985;32:158–160.
12. Badie B. Cosmetic reconstruction of temporal defect following pterional [corrected] craniotomy. Surg Neurol 1996;45:383–384.
13. de Andrade Junior FC, de Andrade FC, de Araujo Filho CM, Carcagnolo FJ. Dysfunction of the temporalis muscle after pterional craniotomy for intracranial aneurysms. Comparative, prospective and randomized study of one flap versus two flaps dieresis. Arq Neuropsiquiatr 1998;56:200–205.
14. Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: Review, criteria, examination and specifications, critique. J Craniomandib Disord 1992;6:301–355.
15. Yasargil MG, Wieser HG, Valavanis A, von AK, Roth P. Surgery and results of selective amygdala-hippocampectomy in one hundred patients with nonlesional limbic epilepsy. Neurosurg Clin N Am 1993;4:243–261.

16. Engel J Jr, Van Ness PC, Rasmussen TB, Ojemann LM. Outcome with respect to epileptic seizures. In: Engel J Jr (ed). Surgical Treatment of the Epilepsies, ed 2. New York: Raven, 1993:609–621.
17. Yushkevich PA, Piven J, Hazlett HC, et al. User-guided 3D active contour segmentation of anatomical structures: Significantly improved efficiency and reliability. Neuroimage 2006;31:1116–1128.
18. Geers C, Nyssen-Behets C, Cosnard G, Lengele B. The deep belly of the temporalis muscle: An anatomical, histological and MRI study. Surg Radiol Anat 2005;27:184–191.
19. Suvinen TI, Kemppainen P. Review of clinical EMG studies related to muscle and occlusal factors in healthy and TMD subjects. J Oral Rehabil 2007;34:631–644.
20. De Felicio CM, Sidequersky FV, Tartaglia GM, Sforza C. Electromyographic standardized indices in healthy Brazilian young adults and data reproducibility. J Oral Rehabil 2009;36:577–583.
21. Ikebe K, Hazeyama T, Iwase K, et al. Association of symptomless TMJ sounds with occlusal force and masticatory performance in older adults. J Oral Rehabil 2008;35: 317–323.
22. Engel J Jr. Update on surgical treatment of the epilepsies. Summary of the second international Palm Desert conference on the surgical treatment of the epilepsies (1992). Neurology 1993;43:1612–1617.
23. Spetzler RF, Lee KS. Reconstruction of the temporalis muscle for the pterional craniotomy. Technical note. J Neurosurg 1990;73:636–637.
24. Matsumoto K, Akagi K, Abekura M, Ohkawa M, Tasaki O, Tomishima T. Cosmetic and functional reconstruction achieved using a split myofascial bone flap for pterional craniotomy. Technical note. J Neurosurg 2001;94:667–670.
25. Meldolesi GN, Di GG, Quarato PP, et al. Changes in depression, anxiety, anger, and personality after resective surgery for drug-resistant temporal lobe epilepsy: A 2-year follow-up study. Epilepsy Res 2007;77:22–30.
26. Miyazawa T. Less invasive reconstruction of the temporalis muscle for pterional craniotomy: Modified procedures. Surg Neurol 1998;50:347–351.
27. Porter MJ, Brookes GB. False ankylosis of the temporomandibular joint after otologic and neurotologic surgery. Am J Otol 1991;12:139–141.
28. Gonzalez-Martinez JA, Srikijvilaikul T, Nair D, Bingaman WE. Long-term seizure outcome in reoperation after failure of epilepsy surgery. Neurosurgery 2007;60:873–880.
29. Salanova V, Markand O, Worth R. Temporal lobe epilepsy: Analysis of failures and the role of reoperation. Acta Neurol Scand 2005;111:126–133.
30. Gaudy JF, Zouaoui A, Bravetti P, Charrier JL, Laison F. Functional anatomy of the human temporal muscle. Surg Radiol Anat 2001;23:389–398.
31. Bowles AP Jr. Reconstruction of the temporalis muscle for pterional and cranio-orbital craniotomies. Surg Neurol 1999;52:524–529.
32. Webster K, Dover MS, Bentley RP. Anchoring the detached temporalis muscle in craniofacial surgery. J Craniomaxillofac Surg 1999;27:211–213.