Transmastoid Repair of Temporal Meningoencephaloceles and Cerebrospinal Fluid Otorrhea

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ABSTRACT

This paper reviews the diagnosis and treatment of temporal bone meningoencephaloceles, defined as the herniation of meninges or brain tissue into empty spaces within the temporal bone, i.e. tympanic or mastoid cavity, through the tegmen tympani or antri respectively. It also describes the current methods of control of cerebrospinal fluid (CSF) leaks, which commonly present as serous otorrhea or rhinorrhea in addition to a variety of symptoms, such as conductive hearing loss. Imaging is the mainstay of the diagnostic process. Management of the condition is surgical, and this review outlines the surgical options with special emphasis on the transmastoid approach and the materials applicable for repair of the bony dehiscences.

Keywords: Temporal bone, Meningoencephalic hernia, Cerebrospinal fluid leak, Transmastoid.

INTRODUCTION

Defects in the bony floor of the lateral skull base, i.e. in the upper surface of the temporal bone can lead to cerebrospinal fluid (CSF) leaks, meningoencephaloceles and meningitis. These worrisome complications can ensue after a congenital, traumatic, neoplastic or iatrogenic lesion of the skull base (Table 1). The defects are conventionally repaired through a limited subtemporal craniotomy with elevation of the temporal lobe or through a combined transmastoid and middle cranial fossa (MCF) approach. A more conservative intervention through the mastoid alone has been proposed by some authors in order to reduce the morbidity of the procedure. Combined cranioplasty techniques are otherwise used to treat larger bone defects.

Several materials have been used to reconstruct bony defects of the lateral skull base: Autologous and homologous cartilage or bone, lyophilized homologous dura mater, preformed hydroxylapatite (HA) blocks, titanium or carbon fiber meshes and myofascial pedicled or distant free flaps. Each material has unique advantages, but each of them also has disadvantages that can lead to failure of the implantation, adverse reactions or complications.

This article will review the available surgical methods and materials to repair temporal bone defects, with special emphasis on the transmastoid route.

Table 1: Etiology of skull base defects

<table>
<thead>
<tr>
<th>Iatrogenic</th>
<th>Neoplastic</th>
<th>Traumatic</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Planned partial surgical removal of middle fossa dural plate</td>
<td>Glomus tumor</td>
<td>Temporal bone fracture</td>
<td>Langerhans cells hystiocytosis</td>
</tr>
<tr>
<td>– Transpetrous approach</td>
<td>Meningioma</td>
<td>Suppurative ear disease</td>
<td>Temporal bone fibrous dysplasia</td>
</tr>
<tr>
<td>– Subtemporal ± transmastoid approach</td>
<td>Middle ear carcinoma</td>
<td>Temporal bone osteitis</td>
<td>Congenital dehiscence</td>
</tr>
<tr>
<td>Accidental drilling during mastoidectomy</td>
<td>Middle ear carcinoma</td>
<td>Invasive cholesteatoma</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Middle ear tuberculosis</td>
<td></td>
</tr>
</tbody>
</table>

Temporal Bone Defects, CSF Leaks and Meningoencephaloceles

The pathogenic mechanisms of production of a meningoencephalic hernia through a bony defect in the skull base have been mainly related with increased intra-abdominal or intrathoracic pressure, leading to increased central venous pressure and subsequent benign intracranial hypertension, a condition also known as pseudotumor cerebri. A long-standing increase in intracranial pressure might produce a meningoencephalocele, as seen in obese patients.
A CSF leak is frequently associated with a meningoencephalocele. It occurs when direct communication of the subarachnoid space to the extracranial compartment, usually a paranasal sinus or the middle ear cavity, is established through an osseous and dural defect at the skull base.

Recognition of the source of the leakage and its appropriate treatment are necessary to avoid rhinorrhea or otorrhea, low-pressure headaches and meningitis, which are known complications of CSF leak.3

Patients with meningoencephaloceles are either asymptomatic or complain of mild symptoms, such as aural fullness, hypoacusis, tinnitus. In case of chronic elevation of the intracranial pressure, patients may describe pressure type headaches, pulsatile tinnitus, balance problems and visual disturbances.4 If a CSF leak ensues, orthostatic5 or exertional headaches6 are frequent, and sudden sensorineural hearing losses are sometimes reported.7

Assessment of the existence and extension of a meningoencephalocele depends on computerized tomography (CT) and magnetic resonance imaging (MRI).8 Current imaging techniques include multidetector thin section CT with axial and coronal scans of the temporal bone.9 It allows to assess the extent of bony resorption and helps in planning the reconstruction. However, in cases in which the tegmen is thinned, CT alone can be misleading because tissue averaging may hide an intact bony plate. Patients suspected of having a meningocele must then undergo an MRI of the brain in order to better define the pathology and to differentiate among granulation tissue, cholesteatoma and brain hernia.10 In this respect, T2-weighted coronal images clearly visualize brain and CSF entering the mastoid or middle ear11 (Fig. 1).

Localization of the site of a CSF leak is a more challenging task, and multiple imaging methods are required, such as digital subtraction myelography,12 intrathecal gadolinium MRI13 or MR cisternography.14 Confirmation of the presence of CSF in the middle ear requires a myringotomy and positive sampling for β₂-transferrin or the more specific β-trace protein.15,16

Bone dehiscences in the lateral skull base are either congenital, post-traumatic or related with bone resorption caused by chronic infection/inflammation of adjacent structures (i.e. the middle ear and mastoid) or to neoplasms (Table 1), or to the otosurgical procedure performed to cure the pathology.

### Congenital Defects

Submillimetric defects in the upper surface of the petrous pyramid are frequent,17 but the formation of a meningoencephalic hernia is a rare entity.18 Therefore, it is argued whether there is a real need to repair such small skull base dehiscences, because they are usually asymptomatic throughout a person’s life. However, CSF leaks and meningitis have been associated with microscopic fissures in the petrous bone.19 Most patients reported in the literature presented with a lesion that was overt at birth or early in childhood.20-24

Kuba et al25 describe the case of a young lady who had undergone ventriculoperitoneal (VP) shunt placement, a shunt operation for aqueductal stenosis in her infancy and who suffered a massive subdural pneumocephalus after a minor whiplash injury due to multiple (congenital) defects in the lateral skull.

Conversely, Wills et al26 describe an old male presenting with a 2-month intermittent watery discharge from his left ear and a negative otological history, but with an episode of bacterial meningitis 10 years earlier. A high-resolution CT scan revealed full-thickness erosion of the tegmen tympani bilaterally, and an MRI showed soft tissue masses plunging into the mastoid on both sides.2

In a rare cadaveric study addressing the issue, the site of the defects in the temporal bone roof corresponded to the areas of greater thinness of the tegmina, i.e. over the mastoid antrum and the attic (Fig. 2).27 Occurrence of meningoencephaloceles in areas of weakened bony structures have also been confirmed by Mokri et al6 in a retrospective review of patients admitted at the Mayo Clinic in Minnesota.

![Fig. 1: Coronal (left panel) and axial (right panel) T2-weighted MRI of the brain showing a meningocele plunging in the right mastoid cavity (personal observation). CSF (confirmed at surgery) is filling the mastoid cavity. Encephalomalacia of the temporal lobe is recognizable (asterisk).](image1)

![Fig. 2: Rostral intracranial view of locus minoris resistentiae in the petrous bone roof](image2)
In a clinical study, Nahas et al. reported 15 patients with surgically confirmed spontaneous meningoencephalocele admitted during 7 years at a single institution. CSF effusion in the middle ear occurred in 13 patients; a history of adult-onset recurrent acute otitis media with intermittent otorrhea was present in four, and one case was complicated by brain abscess. One or more full thickness defects of the tegmen associated with thinning of the middle fossa floor were identified at surgery and correlated well with high-resolution CT. The authors recommend to consider the possibility of a meningocele in all adult-onset recurrent otitis media relating to the obstruction of the aditus ad antrum by the meningoencephalic tissue.

Other congenital causes of CSF leak associated with meningoceles or meningoencephaloceles are due to developmental skull base anomalies, such as large arachnoid granulations or cysts, or congenital inner ear abnormalities (Fig. 3).9

Chronic Otitis Media and Iatrogenic Lesions

Brain herniation into the middle ear cavities is a potential, although rare, life-threatening complication of chronic otitis media (COM) or of the surgical procedures to eradicate the disease.28

Early in the 20th century, it was common practice to treat otogenic brain abscesses with trephinations performed through the mastoid and dura. This often led to a delayed herniation of brain and created a chronically draining mastoid cavity. With the advent of the operating microscope and antibiotics, the incidence of iatrogenic encephaloceles was greatly reduced. It can still rarely occur nowadays, while drilling the middle cranial fossa bony floor during a mastoidectomy: a surgeon can expose or even tears the dura mater of the MCF. In the latter instance, a CSF leak can suddenly manifest during the surgical procedure and its immediate treatment becomes mandatory. When a simple bone dehiscence caused by the drill leaves the meninges intact, most surgeons tend to underscore the problem and do not take any immediate measure. This attitude potentially leads to delayed development of an herniation of the meninges and/or the brain into the middle ear cavities, especially if the size of the defect is wide or the intracranial pressure is repeatedly increased, as in patients exerting weightlifting or other physically straining activities.

Greenberg and Manolidis,29 reviewing 66 patients operated upon for COM found 17 patients (19%) who had a dural dehiscence larger than 1 cm. In one out of three, there was an encephalocele associated with the dehiscence.

In most case series, the majority of brain herniation occur as a complication of previous mastoid surgery,30 direct iatrogenic injury of the tegmina by inadvertent drilling was the single most reported cause of revision mastoidectomy in the USA. A simple bony defect alone is probably not sufficient to lead to herniation, unless the size of the defect is huge, or increased intracranial pressure is not present.

Mosnier et al.28 operated upon 50 patients with a tegmen defect associated with COM over a 15 years time span. Fifteen of them presented a brain hernia associated with the bony defect, and 14 had undergone previous mastoid surgery; five patients showed neurological symptoms at admission.

McMurphy and Oghalai31 treated three patients with residual cholesteatoma and meningoencephalocele who had underwent previous canal-wall-down surgery. They repaired the temporal floor defect using a three-layer closure via a middle fossa craniotomy without obliterating the cavity. All patients recovered without further CSF leak, signs of infection or residual cholesteatoma.

Post-traumatic Defects

Temporal bone fractures do not have the capacity of self-repair by neo-osteogenesis, but are usually closed by scar tissue. Again, a large size of the defect or an increased intracranial pressure can lead to CSF leak or slow development of a brain hernia into the middle ear (Fig. 4).

Nishiike et al.32 reported of a young man who developed CSF leakage following traumatic temporal bone fracture. At surgery via a middle cranial fossa approach, they found intracranial penetration of bony fragments of the tegmen and torn brain tissue between the incus and lateral semicircular canal.
These include a middle cranial fossa craniotomy alone, a mini-middle cranial fossa approach, a transmastoid approach, and a combined middle cranial fossa-transmastoid approach. (Table 2).

The choice of surgical approach is driven by the etiology of the defect, by the site and extent of the bony defect and of the brain hernia, by the type and degree of hearing loss, the presence of chronic infection in the middle ear, and by the intraoperative finding of active CSF leakage. In general, the herniated tissue can be either resected or pressed back into the intracranial compartment. If a middle cranial fossa approach is selected, the herniated cerebral tissue can be sectioned from above and left in the middle ear. The remnants left in the middle ear and/or mastoid cavity progressively shrink, becoming part of the scar tissue.

**Table 2: Review of literature**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>Etiology</th>
<th>No. of cases</th>
<th>Surgical procedure</th>
<th>Recurrence (N%)</th>
<th>Follow-up (months)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feenstra et al</td>
<td>1985</td>
<td>Netherlands</td>
<td>26 iatrogenic, 4 COM, 4 post-traumatic, 1 spontaneous</td>
<td>35</td>
<td>29 TM, 6 Comb</td>
<td>2.8%</td>
<td>27.8</td>
<td>Hernia reduced in 31 cases, amputated in 4</td>
</tr>
<tr>
<td>Lundy et al</td>
<td>1996</td>
<td>USA</td>
<td>11 spontaneous, 4 chronic otitis media, 2 iatrogenic, 2 post-traumatic</td>
<td>19</td>
<td>16 Comb, 2 MCF, 1 TM</td>
<td>100%</td>
<td>31</td>
<td>Fascia-bone-fascia graft (extradurally)</td>
</tr>
<tr>
<td>Jackson et al</td>
<td>1997</td>
<td>USA</td>
<td>27 iatrogenic, 4 spontaneous, 3 chronic otitis media, 1 acute otitis media</td>
<td>35</td>
<td>4 MCF, 15 TM, 16 Comb</td>
<td>8.5%</td>
<td>48.7</td>
<td>23 extradural repairs, 15 intradural repairs, 4 obliteration procedures</td>
</tr>
<tr>
<td>Souliere et al</td>
<td>1998</td>
<td>France</td>
<td>2 post-traumatic, 2 chronic otitis media, 2 iatrogenic</td>
<td>6</td>
<td>5 Comb, 1 TM</td>
<td>100%</td>
<td>3-48</td>
<td>Extradural repair with fascia and bone</td>
</tr>
<tr>
<td>Mosnier et al</td>
<td>2000</td>
<td>France</td>
<td>9 chronic otitis media, 5 cholestatoma, 1 iatrogenic</td>
<td>15</td>
<td>11 Comb, 4 MCF</td>
<td>100%</td>
<td>24</td>
<td>Extradural repair with fascia and bone</td>
</tr>
<tr>
<td>Wootten et al</td>
<td>2005</td>
<td>USA</td>
<td>Iatrogenic</td>
<td>12</td>
<td>7 TM, 5 Comb</td>
<td>16.6%</td>
<td>48</td>
<td>Brain herniation + CSF leaks</td>
</tr>
<tr>
<td>Gubbels et al</td>
<td>2007</td>
<td>USA</td>
<td>Spontaneous</td>
<td>16</td>
<td>MCF</td>
<td>100%</td>
<td>13</td>
<td>Hernia amputated in 11, reduced in 5</td>
</tr>
<tr>
<td>Scurry et al</td>
<td>2007</td>
<td>USA</td>
<td>Spontaneous</td>
<td>8</td>
<td>5 Comb, 2 TM</td>
<td>25%</td>
<td></td>
<td>Obese patients</td>
</tr>
<tr>
<td>Nahas et al</td>
<td>2008</td>
<td>US</td>
<td>Spontaneous</td>
<td>15</td>
<td>Comb</td>
<td>6.6%</td>
<td></td>
<td>Combined repair with hydroxyapatite paste + bone graft</td>
</tr>
<tr>
<td>Sanna et al</td>
<td>2009</td>
<td>Italy</td>
<td>61 iatrogenic, 33 spontaneous, 29 middle cranial fossa, 10 post-traumatic</td>
<td>143</td>
<td>55 middle ear obliteration, 37 TM, 37 MCF, 4 Comb</td>
<td>3%</td>
<td>38.4</td>
<td></td>
</tr>
</tbody>
</table>

**Table Notes:***

COM – chronic otitis media; CSF – cerebrospinal fluid; TM – transmastoid; MCF – middle cranial fossa; Comb – combined approach
The defect is then sealed with autologous, heterologous or alloplastic materials, or their combinations. Each of these solutions depends on surgeon experience, size of defect and volume of herniated brain.

A lumbar drain is advocated by some authors\textsuperscript{31} for a few days postoperatively as a preventive measure, while it is not deemed necessary by others. In our experience, CSF drainage is warranted in large defects of the skull base or obese patients; bed rest, avoidance of straining and sneezing, fecal softeners and cough sedation are sufficient preventive measures in all minor repairs.

### The Subtemporal Approach

It was the first method proposed to repair any size of a defect in the tegmina,\textsuperscript{44} and still the most popular approach among the neurosurgical community.\textsuperscript{45} Its advantages are the better control of the anterior part of the roof of the petrous bone, the preservation of hearing by avoiding any manipulation of the ossicular chain, and the reduced risk of contamination by a possibly infected middle ear cavity. Its drawbacks include a greater invasiveness and morbidity, thus Sanna et al\textsuperscript{42} suggest to reserve it to spontaneous meningoencephaloceles with intact hearing.

If concomitant middle ear disease requires treatment, this can be postponed to a 2nd stage after complete healing of the tegmen reconstruction.

Dutt et al\textsuperscript{45} claim that the MCF approach is superior to the transmastoid route in the long-term stability of the reconstruction. Their series is limited to four patients with spontaneous CSF otorrhea who had tegmen defects repaired using an autologous bone pate slab mixed with fibrin glue with additional temporalis fascia reinforcement; no recurrences of the leaks were observed up to 3 years of follow-up.

### The Transmastoid Approach

Minor tegmen plate defects causing CSF leaks and brain hernias have conventionally been repaired by otologists by using soft tissue grafts via a retroauricular transmastoid approach (Fig. 5). It is a simple, direct way to the lateral cranial base, with minimal morbidity, usually limited by the involvement of the ossicular chain in the attic, whose handling often leads only to a mild to moderate conductive deafness.

A simple or complete mastoidectomy with preservation of the outer ear canal (OEC) wall is the preferred procedure. If a previous canal-wall-down mastoidectomy has been performed, its revision is mandatory. In case of chronic otitis media, eradication of the disease is enforced, and a tympanoplasty with reconstruction of either the eardrum or ossicular chain is performed as needed. After the bone work is complete and the pathology is removed, the bone defect is exposed. The operative field is then rinsed with an antibiotic solution. The herniated tissue is generally small, even when the dura is extensively uncovered, and it is sufficient to coagulate the herniated tissue, reduce it into the intracranial compartment and cover the bony defect with bone dust and fascia. In rare instances, suturing the dura in an extradural fashion and cutting the redundant meningocele is required (Figs 6A and B). The bone dehiscence is then sealed by any of the materials selected for the reconstruction.
which must be trimmed and fitted precisely to the size and contour of the defect through subsequent trials in situ (Figs 7A and B).

In the “Overlay” or external method, the graft or the implant is glued to the mastoid (extracranial) surface of the defect, leaving the intracranial compartment untouched. Unfortunately, if not adequately supported by other filling materials, the attached insert is prone to be dislodged into the mastoidectomy under the CSF pressure.

In the “Inlay” technique, the middle cranial fossa dura is smoothly detached 3 or 4 mm from the bony borders of the defect with a duckbill elevator (Figs 8A and B). The final dimensions of the implant are slightly larger (1 to 2 mm) than the defect itself to permit limited overlapping. If an alloplastic material is used, an autologous fascia temporalis graft is interposed in the intracranial compartment facing the dura (Figs 9A and B). The implant is placed in the intracranial compartment between the dura and the petrous bone and thus held in place by gravity and the physiologic intracranial pressure.

In the “sandwich” technique, further watertight sealing on the mastoidectomy side is achieved by means of another layer of temporalis muscle fascia and fibrin glue added to the inlay procedure (Figs 10A and B).
Another option after repairing the bony defect is to exclude or “ablate” the mastoid cavity. This implies:
1. Obliterating the mastoidectomy cavity with soft tissues (fat graft, local or revascularized free flaps) or hard materials (bone chips, bone paté, hydroxyapatite)
2. Sealing the Eustachian tube and
3. Closing the outer ear canal as a “cul-de-sac”, according to the standardized Rambo technique. This procedure provides the ultimate solution to a recurrent CSF leak but also leads to a full conductive hearing loss and has the potential for hiding retained squamous epithelium that might result in an occult recurrence of cholesteatoma.

Some authors argue that the results of transmastoid repairs have been less than satisfactory than with the MCF approach. In Ramalingam et al series, 10 out of 13 mastoidectomy revision procedures were effective in repairing a temporal lobe hernia developing years after primary COM surgery; in one patient only, a combined approach was necessary and in two others a blind sac closure of the middle ear was needed.

Sanna et al treated and solved the meningoencephalohernia with a transmastoid approach alone in 93/122 patients (combined with a total obliteration of the middle ear cavities in 55 cases); they reported an absence of complications, while they had one meningitis and one epidural hematoma with the MCF approach.

In a personal study, we treated seven patients with a petrous bone dehiscence less than 3 cm in diameter, either iatrogenic or caused by COM or temporal bone trauma, by a transmastoid route. The defect was repaired with a new composite alloplastic material (Hydroxylapatite and caprolactone) and a connective tissue graft. Outcome was evaluated in terms of anatomical integrity of the tegmina, absence of cerebrospinal fluid leaks, side effects, extrusion and complication rates. None of the patients suffered from immediate side effects related to the implant or the operation. With a minimum follow-up of 18 months (maximum, 62 months), neither extrusion nor a foreign body reaction occurred. Postoperative CT confirmed a satisfactory anatomic contour and all patients were free from CSF leak.

**Combined Approach**

In Mosnier et al series, the encephalic hernia was repaired using a combined, MCF craniotomy associated with a transmastoid approach in 11 out of 50 patients. The combined approach was used in cases where a large hernia, and/or inflammatory tissues were present in the mastoid. The herniated brain tissue was resected in all cases, and the dural and bony defects were closed with fascia and bone. No complication or recurrence occurred during a mean follow-up of 2 years.

In Nahas et al series, all patients first underwent mastoidectomy to confirm the presence and location of herniated meningeal tissue and an associated CSF leak. Then a mini-craniotomy (3×4 cm) of the middle fossa was located with inferior edge at 5 to 10 mm of the tegmen to optimize visualization of the defects while minimizing the temporal lobe retraction.

McMurphy and Oghalai perform a mini temporal craniotomy facing the revised mastoidectomy cavity with a canal-wall-down procedure. After identifying circumferentially the edges of tegmen defect, they elevate the temporal lobe, they cut and remove the stalk of the hernia and repair the dura by suturing a graft of the deep layer of the superficial temporalis fascia. The bone defect is then fixed with a calvarial bone split graft interposed intracranially. A pedicled temporalis muscle flap is rotated between the dura and bone graft and sutured to the dura medial to the defect.

**THE MATERIALS FOR RECONSTRUCTION**

While the future of bone defects reconstruction will probably stand on the road of growth factors promoting autogenous bone regeneration, and applicable to the affected site with gels or other vehicles, many biological and synthetic...
material are currently used for the purpose of obtaining a waterproof seal of bone defects in the lateral skull base. They include autologous and homologous bone or cartilage grafts, local pedicled soft tissue flaps, \textsuperscript{36,41} or distant free revascularized flaps, \textsuperscript{35,39} as well as many alloplastic materials, such as bone substitutes, cements, ceramics, metals, polymers, acrylics and their composites. \textsuperscript{33,34,37}

Dural repair can be accomplished by means of direct primary suturing if the defect is minimal, or by suturing a connective tissue graft, such as autologous peristium or fascia temporalis, homologous or heterologous dura mater, fascia lata and soft allogenic tissues.

Dural soldering with \textit{CO\textsubscript{2}} laser has been successfully tested in animals, \textsuperscript{51} but its watertight resistance to CSF leaks in humans remains questionable. In another animal study, \textsuperscript{52} methyl methacrylate and \textit{CO\textsubscript{2}} laser techniques were inadequate for stopping dural leakage and had harmful effects on brain tissue. CSF leak occurred less easily with \textit{n-butyl-cyanoacrylate} than with fibrin glue, but lipid peroxidation levels showed that it was not safe for the brain. Biological sealants, such as fibrin glue or collagen, are thus considered helpful adjuncts, \textsuperscript{53} while acrylic adhesives have been abandoned due to neurotoxicity.

**Organic Tissues**

Autologous bone grafts are the oldest and most preferred material to reconstruct any size of defect in the petrous bone roof. \textsuperscript{54} Bone strips are obtained by cutting the cortical bone with a chisel or a bone saw from the mastoid or by splitting the squama without the need for a craniotomy. The bone grafts are then inserted from a free margin inside the bone defects, taking care to avoid tearing the dura mater.

Homologous bone grafts have almost been abandoned worldwide because they are prone to infection and difficult to harvest and fit to the defect, leading to instability and a lack of osseointegration. The process of remodeling usually reduces their size 15 to 25\%, thus impairing the outcome of the reconstruction.

Bone dust obtained during the drilling of the mastoid cortex can be mixed with fibrin glue or blood to provide a malleable paste termed “bone pate”. It has been commonly utilized by otologists to obliterate wide mastoidectomy cavities in chronic ear surgeries. Dutt et al.\textsuperscript{45} recommend the use of autologous bone pate along with soft tissue for the repair of temporal bone defects in order to achieve a secure seal. Bone pate can effectively seal multiple defects in the tegmen plate without any risk of migration and with minimal recurrence rates.

Cartilage grafts are sufficiently elastic but offer little resistance to deformation. They are not osteogenic and are often reabsorbed to a variable extent or converted to fibroid tissue one year after implantation.

Both cartilage and bone homografts and xenografts share the common drawbacks of availability, resorption and infection. Furthermore, they are unavailable in some countries because of governmental regulations and medicolegal implications. In fact, the use of homologous or heterologous tissues undergoes the restrictions of different policies applied in each country. Moreover, cadaveric bone and cartilage grafts carry the risk of contamination by human retrovirus (HIV) or prions with the risk of diffusion of Kreutzfeld-Jacob disease.

Commercially available, certified graft materials such as dried bone, bone matrix, cartilage, lyophilized dura and other harvested tissue are considered safe because of the strict rules of preparation (de-antigenization, purification, etc.) and sterilization (e.g. gamma-rays), but there are few experiences in skull base reconstruction.

Extensive defects occurring after resection of malignant tumors involving the skull base justify the use of microvascular free flaps for reconstruction, in order to improve the quality of life and survival. \textsuperscript{55,56} They represent an efficient barrier to CSF leakage and provide a tailored filling of the operative defects, reducing the cosmetic consequences. \textsuperscript{57,58}

**Alloplastic Materials**

Current cranioplasty materials synthesized in the laboratory include metal mesh, ceramics, fluoroplastics and methyl methacrylate, either alone or in combination.

The use of alloplastic material gained widespread acceptance during and after World War II, when many metals (aluminium, vitallium, tantalium) were introduced. Later they were abandoned in favor of less radiopaque meshes of stainless steel\textsuperscript{40} or titanium. \textsuperscript{59} At the beginning of the 1950s, acrylics were developed, and excellent cosmetic outcomes were achieved with methyl methacrylate\textsuperscript{60} because it is easily molded at the operating table. Later studies showed that acrylics had a low tensile and a tendency to crack under pressure. Improvement has been obtained by combining of titanium meshes with methyl methacrylate paste. \textsuperscript{61}

Among alloplastic materials, ceramics and metals share the common problems of biocompatibility and extrusion. \textsuperscript{52} Implants can fail immediately from exposure and infection or in a delayed fashion from graft retraction, implant fragmentation and extrusion. Most failures are related to incomplete coverage of the implant with soft tissue and limited blood supply. \textsuperscript{63,64} Possible reasons for exposure are the presence of sharp angles and failure to fit the implant exactly to the size of the defect because of the difficulty of modeling the material. The current trend is toward lightweight ceramics and other synthetic composites that promote osteogenic ingrowth at the edges of the implant.
Among ceramics, hydroxyapatite (HA) has been used extensively in other specialties as a bone substitute, since it provides a matrix on which living bone tissue can form and grow. It has optimal osteoconductive properties, primarily regulated by its porosity, that influence its speed of reabsorption.65-67 Direct and stable contact with the host’s bone stimulates osteogenesis and therefore integration of the material.68 A mucous coating usually surrounds the implant without any signs of reabsorption or foreign body reaction as occurs with organic grafts.69

The consistency and compactness of HA are responsible for its considerable resistance to compression and rigidity. Preformed rigid blocks of HA have already been used to repair petrous bone defects through a subtemporal craniotomy43,70-73 or combined transmastoid and middle cranial fossa approach.74,75 Unfortunately, presculptured HA blocks require tedious intraoperative reshaping, and the result is not always satisfactory. HA-based cements have been successfully used in reconstructions after skull base surgeries.76 HA has been used also in pediatric patients with lateral cranial bone defects.77 Mixtures of hydroxyapatite and tricalcium phosphate have been popular for skull base reconstructions in Japan.78

We used flexible HA sheets to repair minor defects in the temporal bone (less than 2 cm in diameter) through a transmastoid approach.49 The material is a composite membrane obtained in the laboratory by mixing HA powder and an organic polymer in appropriate proportions (Fig. 7). The latter adds malleability to the typical features of HA (resistance and biointegration), allowing the material to be inserted easily in an underlay fashion from the mastoid. The implant can be bent and accurately trimmed to the dimensions of the defect. There have been no adverse reactions to the material so far, and none of the patients have manifested a CSF leak after the careful reconstruction described above.

At the Neurosurgical Department of the University of New York, cranioplasties have been traditionally carried out using titanium mesh and acrylic.61 The authors do not report complications or infections. Advantages of titanium are radiolucency, nonmagnetic properties and the greatest biocompatibility among metals.

Any alloplastic material must be used in a sterile cavity, with no active disease. It is recommendable to cover it with biologic tissues and to provide antibiotic prophylaxis.

**CONCLUSION**

The occurrence of severe neurological complications as a consequence of brain herniation into the ear cavities or of CSF leakage emphasizes the necessity for recognition and appropriate management of these diseases. The hernia can be repaired using a transmastoid approach, or with an associated middle fossa craniotomy in a single stage.

Although a combined or isolated intracranial procedure (i.e. subtemporal craniotomy) is essentially extradural, the more conservative transmastoid approach alone minimizes the operative times and lowers complications, without increasing the risk of recurrence of CSF leak when properly performed. The previously reported high failure rates associated with this approach have been overcome by modifications of the surgical procedure (sandwich repair and/or mastoid obliteration).

Cartilage or alloplastic implants provide a rigid barrier to withstand the intracranial pressure; sheets of connective tissues are added to secure a watertight closure of the defect. Bone pate, adipose tissue or muscle flaps are additional materials useful to obliterate the cavity and guarantee a support for the reconstruction.

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