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Endaural cerebral hernia

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Introduction

Herniation of brain tissue into the middle ear and mastoid is a problem which rarely faces the present day otologist. This has not always been the case. In the first forty years of this century protrusion either of temporal lobe or cerebellum into the middle-ear cleft was a well-recognized entity. The condition occurred most commonly as a complication of trepanation for an otogenic temporal lobe or cerebellar abscess, and raised intracranial pressure and local infection were essential predisposing factors. Better health care and the introduction of antibiotics have reduced the incidence of intracranial otogenic infection to very low levels. Furthermore, improved surgical technique, particularly as a result of the introduction of the operating microscope and the drill, has reduced the likelihood of iatrogenic hernia from trauma to the dura mater, particularly in the region of the tegmen tympani. The literature abounds with colourful and inaccurate names for the condition, the most popular in early years being 'Brain Fungus' which must surely finally be expunged. The present authors prefer the term 'Endaural Cerebral Hernia', proposed by Schurr (1960).

Historical review

The first important date in the story is 1727 when Vander Wiel reported the first case of a clear watery discharge from the ear following head trauma, this long before the discovery of cerebrospinal fluid. The first authoritative discourse on brain hernia was presented in 1902 by Caboche in the French literature. An excellent account of the problem with a good historical review is given by Colby Hall in 1939. The great majority of endaural cerebral hernias in those early days were complications of otogenic abscess or lateral sinus thrombosis, and were classified as immediate, which occurred as soon as the dura was opened, in search of the abscess; and secondary, occurring some days after incision of the dura.

Immediate hernias were evidence simply of raised intracranial pressure and brain volume. They more commonly followed cerebellar abscesses than temporal lobe abscesses because of the tendency for a posterior fossa abscess to cause a much greater increase in intracranial pressure. Hall (quoting Caboche) states that an immediate hernia was usually small, and reduced after drainage of the abscess. Secondary hernias developed in the presence of persisting infection with necrosis of both brain and dura. Softened brain could pass through the defect and become strangulated, with further oedema and necrosis. The literature of the post-antibiotic era contains relatively few references to the condition. Notable contributions have come from Schurr (1960), Dedo and Sooy (1970), Paparella *et al.* (1978) and Glasscock *et al.* (1979). All writers agree that this now uncommon condition requires prompt surgical treatment if the

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problems of meningitis and cerebrospinal fluid fistula are to be avoided, but on the subject of how this repair should be effected—tot homines, quot sententiae—fascia, either intradural, extradural or intramastoid, free or pedicled temporal muscle, bone or non-biological materials all have their champions.

Anatomy

The dura mater is a tough bilaminar membrane, the visceral layer of which envelopes the brain, and the peripheral layer of which is closely applied to the inside of the bones of the skull. In children and in the aged this layer of dura is particularly adherent to bone. The bony roof of the middle ear and mastoid, the tegmen tympani, is very thin. Ahren and Thulen (1965), in a series of cadaver dissections, found dehiscence of the tegmen in 21 per cent of cases and a very thin bony plate in 16 per cent. Similar findings have been reported by Lang (1983). Normal dura is capable of supporting brain over quite large bony defects.

Another important anatomical point which has received little attention in the literature is the fact that the floor of the middle cranial fossa and thus the dura is lower laterally, over the attic and aditus, than it is more medially, as it sweeps up towards the arcuate eminence. The otologist may thus often have difficulty drilling between the tegmen and the superior meatal wall, and may often expose middle fossa dura in this region.

The subarachnoid space between the inferior surface of the temporal lobe and the tegmen is narrow. Alberti and Dawes (1961) point out that, because of this, the weight of overlying brain may be sufficient to plug any defect in the dura, and prevent more than a very transient CSF leakage. In the relatively rare case of erosion in the region of Trautman's triangle into the posterior cranial fossa, the lateral cistern would be opened and profuse CSF otorrhoea would ensue.

Pathogenesis

Schurr (1960) restated the requirements for the development of an endaural brain hernia. There must be a defect in the dura, and in the bone, through which brain can herniate; and there must be raised intracranial pressure or infection or both.

The four main causes of bony defect are: (1) surgical trauma; (2) infection; (3) skull base fracture; and (4) congenital defect.

Most mastoid surgery is carried out for chronic suppurative otitis media, whether associated with cholesteatoma or with mucosal disease alone. Healthy dura can be fairly widely exposed without much danger of herniation. In fact, Alberti and Dawes (1961) go so far as to state that, as long as one leaves a buttress of bone along the line of the superior petrosal sinus, the risks of prolapse are not great. If, on the other hand, the surgeon penetrates the dura with the drill, an immediate repair of the fistula must be carried out using usually fascia or muscle. Cholesteatoma commonly exposes dura, usually in the tegmen region and not infrequently in Trautman's triangle or round the lateral sinus. Such exposed dura may be weakened by infection and is often rather granular in appearance.

Skull base fractures frequently involve the roof of the middle ear—indeed the only evidence may be CSF otorrhoea or rhinorrhoea, the radiology often being uninformative. The majority of such fistulae heal spontaneously within two weeks, but the residual defect may become the site of a herniation, particularly if, as in our first case, a growing fracture develops. Some hernias develop in ears which have not been traumatized either by previous surgery or by skull fracture, and have never harboured a cholesteatoma. Such lesions may present in the external meatus, or sometimes as a chance finding to the surgeon carrying out a simple tympanoplasty. One assumes that in such ears there has been a widespread congenital dehiscence of the roof of the middle-ear cleft and that the exposed dura has been weakened by repeated middle-ear infections.

Whatever the cause, the herniated brain expands into the middle ear and mastoid. It does not have a dural covering, and the arachnoid sheath is confined to the neck. If arising in a mastoid cavity, it may push before it the epithelial lining and may project at the external meatus as a smooth skin-lined cyst which

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may be mistaken for a chocolate cyst or blue-domed cyst.

Case reports

Case I

This boy was involved in a road traffic accident at the age of ten. He suffered severe injuries to the right temporal region, as a result of which he had brain extruding and CSF leaking through the temporal wound. This was dealt with by the neurosurgeons. About two years after this, he was found to be deaf in his right ear and examination of the ear showed marked stenosis of the external canal. Microscopic examination revealed some cholesteatoma deep in the stenosed meatus and the audiogram showed a 60 dB conductive hearing loss. There was no previous history of ear disease prior to the accident. The mastoid was explored and a smooth blueish cyst was found to occupy the antrum with a large pearl of cholesteatoma deep to it. During the removal of the cholesteatoma CSF began to pour from the cyst. The mastoid cavity was packed and the patient was transferred to the neurosurgical unit at Manchester Royal Infirmary. X-rays of the petrous bones showed a large defect in the tegmen on the right side (Fig. 1). The left side was normal. Computer tomography showed an extensive irregular region of low attenuation in the right temporal region connecting with the posterior part of the body and with the temporal horn of the right lateral ventricle. The ventricles were otherwise normal in shape and size (Fig. 2). No useful information was provided as to the state of the temporal bone. The defect in the floor of the middle fossa was explored through a temporal craniotomy incision. The dura was found to be torn and widely open in two distinct areas. The larger of the two bone defects measured approximately 2 cm × 1 cm, the long axis running antero-medially in the line of the middle-ear cleft from the sino-dural angle. There was a small defect in the roof of

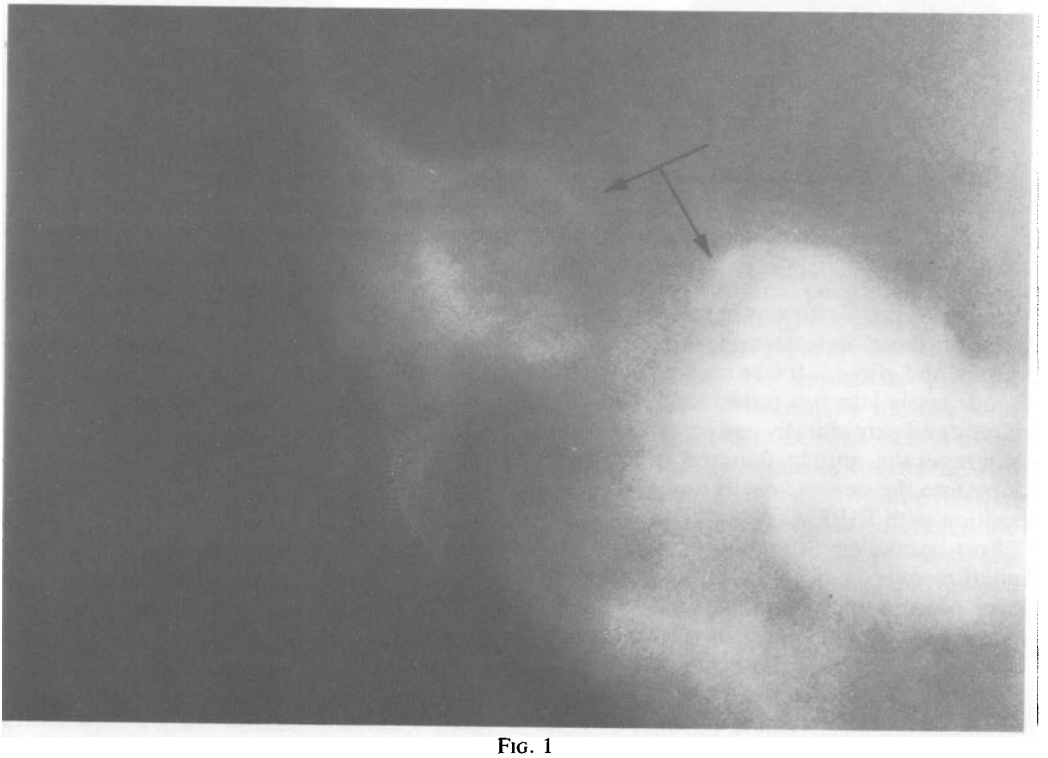


FIG. 1

Case I. AP tomograph of right temporal bone showing large defect in tegmen tympani (arrowed).

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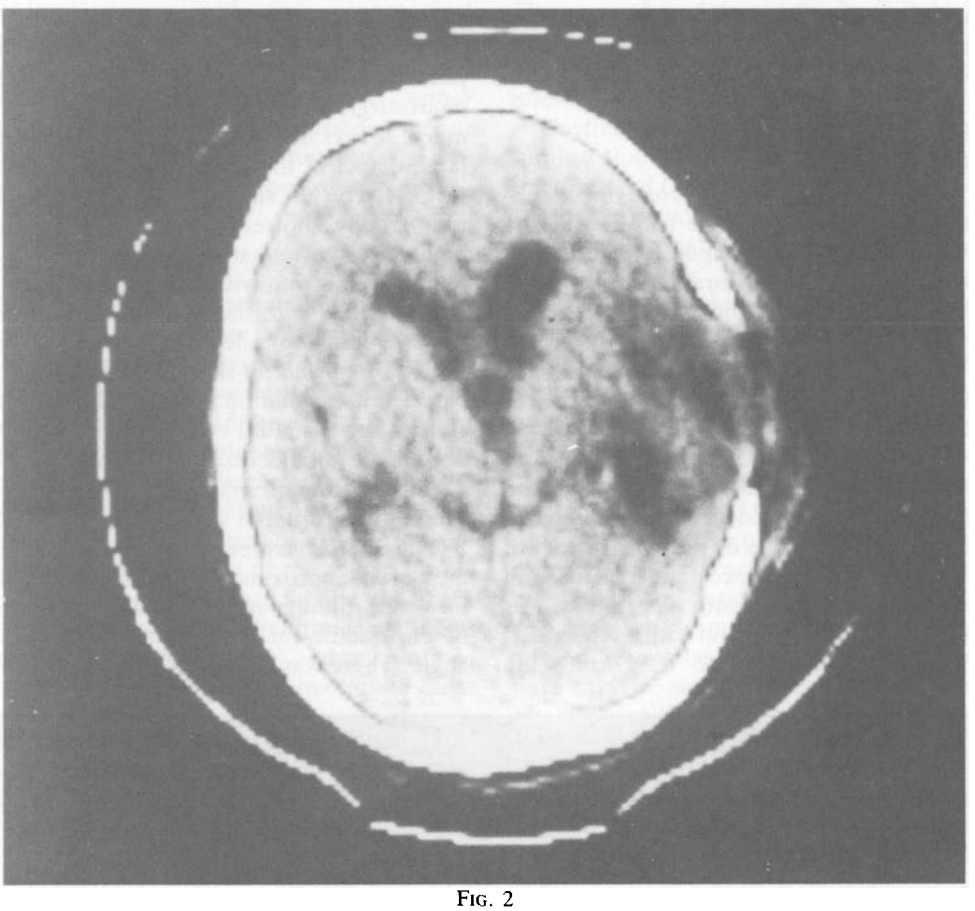


FIG. 2

Case I. CT scan. Diffuse low density area in right temporal lobe. There is no ventricular dilatation.

the middle ear. Brain was herniating through both of these defects and the tissue was scarred and gliosed. It was resected and autologous fascia lata was introduced both intradurally and extradurally and sutured in place. A temporalis muscle flap was then swung down into the mastoid cavity and packed into position with BIPP.

Post-operatively, the patient made a good initial recovery but 12 days after surgery his level of consciousness deteriorated. A CT scan demonstrated enlargement of his ventricles (Fig. 3) and a diagnosis of hydrocephalus was made. A ventriculo-peritoneal shunt was inserted with improvement in his level of consciousness. However, he developed ventriculitis which required a further course of antibiotics and a shunt revision. The mastoid cavity healed uneventfully and, eighteen months following his craniotomy, there was no sign of recurrent brain hernia or CSF leak.

Case II

This boy had a right modified radical mastoidectomy carried out at the age of 12. In the operation note it was stated that the middle fossa dura was exposed but not apparently breached. One year later, at the age of 13, he was found to have a polyp in the right mastoidectomy cavity and during an attempt to remove this, a leakage of CSF was noticed and the removed tissue found to be brain. For 2 years he was treated with antibiotic packs in

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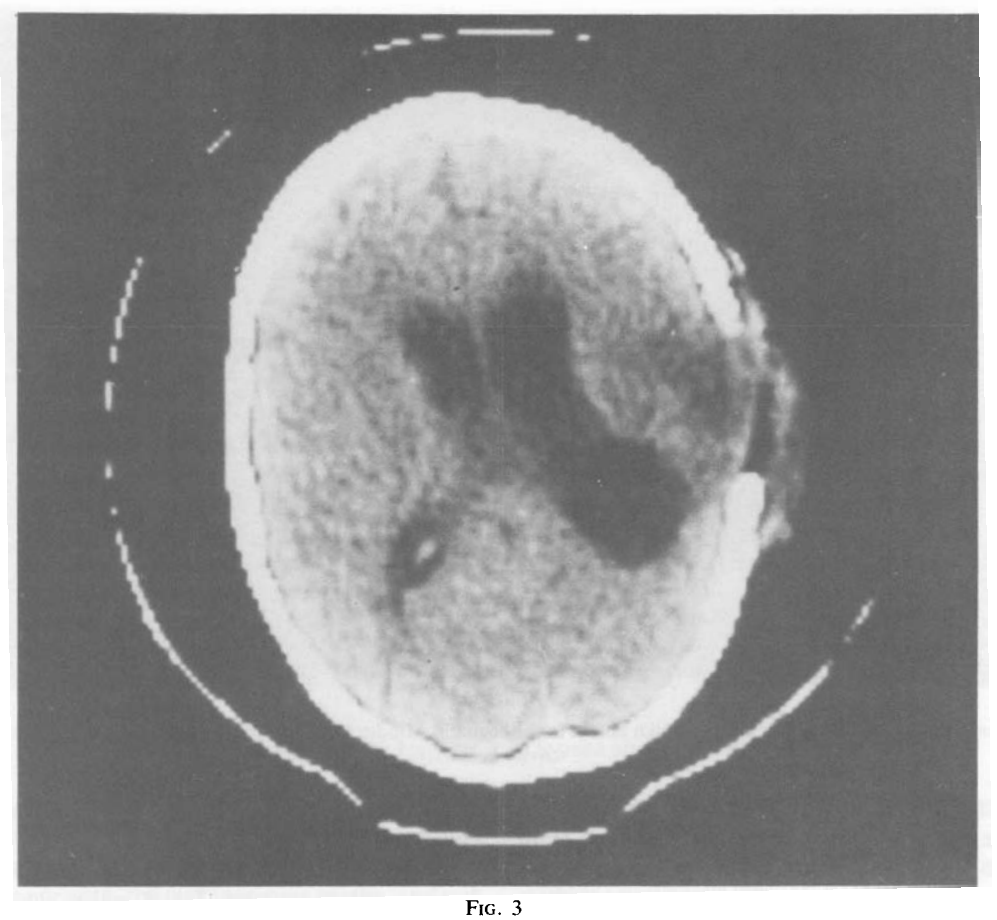


FIG. 3
Case I. CT scan after closure of aural-CSF fistula, showing hydrocephalus.

the hope of minimizing the herniation but this did not help and he was referred to Manchester Royal Infirmary in 1980 for repair.

He was found to have a large hernia filling the whole of the mastoid segment and causing a secondary infection in the mastoid cavity. A CT scan was normal and tomograms of the temporal bones were uninformative. A combined trans-mastoid and middle fossa approach was carried out. A tongue of brain was found to be herniating through the dura and into the mastoid through a circular defect approximately 1 cm in diameter in the roof of the mastoid (Figs. 4 and 5). There was a further smaller defect in the tegmen tympani. A two-layer repair was carried out, using autologous fascia lata sutured both intradurally and extradurally. The mastoid cavity was then cleaned up and a pedicled temporalis muscle flap turned into the mastoid and held in position with a BIPP pack.

Post-operatively his immediate progress was very good but eventually the temporal muscle flap formed adhesions between the facial ridge and the tegmen, interfering with the natural drainage of the cavity, and this flap had to be removed at a second operation some months later. Thereafter his progress has been satisfactory and the cavity has healed nicely.

Case III

This 31-year-old woman had a left modified radical mastoidectomy in 1974 for chronic suppurative otitis media with cholesteatoma.

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FIG. 4.

Case II. Cerebral hernia passing through defect in the tegmen. Middle fossa and temporal lobe above; mastoid cavity below.

After remaining symptom-free for four years she began to develop pain and discharge from the left ear and a feeling of pulsation in the ear.

On examination there was a smooth pulsatile mass taking origin in the roof of the mastoid cavity. The mass was covered with skin. It increased in size and the pulsations stopped when the patient carried out Valsalva's manoeuvre. The mass was insensitive to touch and felt soft and cystic. Petrous tomograms were normal and a high resolution CT scan also failed to reveal any convincing abnormality.

A clinical diagnosis of brain hernia was made and a combined middle fossa and transmastoid approach was made to repair this. A hernia was defined and its neck found to be passing through a bony defect in the roof of the mastoid antrum. A small mass of necrotic brain was found within a flimsy sac of arachnoid. The brain was excised and the dural defect was repaired with two layers of autologous fascia lata, one intradurally and one extradurally. A temporalis flap was not employed in this case.

Post-operatively, she had a transient left lower motor neurone facial palsy which recovered completely over two weeks. She complained of headache at the site of operation and a CT scan revealed a thin extradural haematoma. Since her level of consciousness remained satisfactory, she was treated expectantly and serial CT scans showed that the haematoma resolved. The mastoid cavity healed perfectly and she is well three years after her operation.

Discussion

The diagnosis of brain hernia should be suspected in all cases where a cystic mass is found on otoscopy, but particularly if there is a history of major trauma, or of CSOM and mastoid surgery. The typical middle fossa hernia will be attached superiorly so that a probe can be passed in front, below and behind it but not above it. It is usually blueish-grey and

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FIG. 5

Case II. Probe passing through bony defect in tegmen after amputation of the hernia.

feels soft to the touch. Characteristically it is pulsatile, and when the intracranial pressure is raised, on staining or carrying out Valsalva's manoeuvre, the cyst increases in size, and pulsates less. The main differential diagnosis is the blue cyst of the mastoid which contains blood and cholesterol. The appearances may be very similar, but a blue cyst does not pulsate. Baron (1969) states that CSF otorrhoea is the commonest mode of presentation, but most other authors appear to disagree. In two of our cases CSF otorrhoea occurred on manipulation of the hernia by the surgeon, but it was not the mode of initial presentation. Two of our patients presented with infection, one in a mastoid cavity and one in a previously traumatized ear; the third presented with pain and pulsation in a mastoid cavity.

Petrous tomography was of value only in our first patient (Fig. 1) when it demonstrated a large bony dehiscence in the tegmen. In our other two patients, the investigation did not contribute significantly. In all three patients CT scanning was normal pre-operatively and in particular did not show evidence of intracranial air or hydrocephalus. It should be remembered that a normal CT scan does not exclude raised intra-cranial pressure (Auer *et al.*, 1980) and the latter may be implicated in the development of endaural cerebral hernia in some patients. The importance of having CT scanning facilities available is, however, apparent when we consider the post-operative period of our patients. In our first case, closure of a CSF fistula was followed by hydrocephalus. Presumably in this patient a communicating hydrocephalus secondary to a basal meningitis caused by infection (or his previous head injury) was compensated by a CSF otorrhoea until the fistula was closed. The possibility of post-operative hydrocephalus should therefore be remembered when managing patients undergoing closure of a CSF fistula. Our third patient developed a small sub-temporal extradural haematoma. Her persisting headache at the site of operation increased clinical suspicion of this and normally would have warranted re-explora-

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tion. However, she appeared well and we were able to follow her progress closely with the CT scan and so avoid a further operation. This course was only justified because the haematoma was small and not associated with any deterioration in her level of consciousness. Our first patient had such a large defect following skull fracture that we felt he must have a 'growing fracture', the mode of development of which has recently been reviewed by Lye *et al.* (1981). If the dura is very adherent to the bone, a skull fracture may produce extensive dural tears which may be larger than the bone defect and the fibroblasts may be unable to reconstitute the dural defect. Raised intra-cranial pressure or more probably systolic pulsations of the brain may then cause erosion of the fracture margins and herniation of brain into the ear.

There can be no doubt about the desirability of a surgical repair of the dural defect to separate the cranial cavity from the upper respiratory tract. There is agreement that the herniated piece of brain can be amputated and discarded. As a rule otologists have tended to recommend closure from below, and neurosurgeons repair *via* the middle fossa. Free temporalis fascia grafts have been applied intradurally, extradurally from above or from below, or simply packed against the roof of the middle-ear cleft from below. Alberti and Dawes (1961) recommend that defects in the tegmen should be repaired *via* the middle fossa, and those in Trautman's triangle *via* the mastoid using temporalis fascia or muscle. A temporalis pedicle into the mastoid is used by George Hall *et al.* (1967) and by Dedo and Sooy (1970) if the external canal wall is intact. If there is an open cavity, the latter authors simply recommend packing of the mastoid for six weeks with iodoform after removal of the hernia flush with the dura. Stout *et al.* (1969) and Fernandez-Blasini and Logo (1977) both employ a pedicled temporalis muscle flap introduced through a temporal craniotomy. Over the years many other materials have been employed, either heterograft (dog's dura) or non-biological (bone wax, polyvinyl formol sponge, methyl methacrylate). Most authors, however, seem to favour the use of the patient's own tissues, and the temporalis muscle or fascia are probably the most commonly employed.

We feel that a double-layer repair of the dura, using fascia lata both intradurally and extradurally, is adequate. This is considerably stronger than temporalis fascia. It seems logical to repair this sort of defect intracranially so that the weight of the brain will hold the graft in place, rather than to attempt a repair from the mastoid. There were no major neurological sequelae from opening the middle cranial fossa. Very little retraction of the temporal lobe is necessary and none of our patients developed temporal lobe symptoms post-operatively. The additional use of a pedicle temporalis muscle flap is unnecessary. Indeed, it may cause problems of web formation and continued infection in the mastoid cavity.

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