INTRACRANIAL PNEUMOCŒLE

AN UNUSUAL COMPLICATION FOLLOWING MASTOID SURGERY

By M. Horowitz (Liverpool)

History

A woman aged 32 was transferred to Liverpool Regional Neurosurgical Centre on 12.12.61, with a history of continuous left occipital headaches for 2 weeks. She had undergone a left radical mastoidectomy for a chronic left suppurative otitis media at the end of November. At operation, a note was made of accidental trauma to the middle fossa dura, but, until the onset of the headaches two weeks later, recovery had been uneventful.

A lumbar puncture report at this time was as follows: Cells 195 (90 per cent. lymphocytes), Protein 80 mgm. per cent., Sugar 51 mgm. per cent. A provisional diagnosis of secondary otitic cerebral abscess was made.

Examination on admission revealed remarkably few signs. Apart from slight neck stiffness there were no other generalized or localizing signs in the Central Nervous System.

During a left carotid angiogram, a large intracerebral air cavity was discovered in the left temporal lobe area, connecting inferiorly with the mastoid cavity by a small track, outlined with B.I.P.P., and internally with an air track leading into the ipsilateral ventricle which was also partly outlined with air.

It was decided to re-explore the left mastoid cavity postaurally.

Operation

The dura was exposed in the tegmen and in the sino-dural angle. In the latter area there were multiple granulations, in the midst of which a small track led upwards into the cranium. After exposure of healthy dura all round this granular area, a temporal pedicle muscle flap was turned down as a seal, and the post-aural incision was closed.

Progress

There was no recurrence of the headache and the intracranial air resorbed spontaneously; a final serial X-ray picture 6 weeks later showed complete resolution.

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PLATE I.

Head Erect Position. Intracerebral Pneumocœle of Mastoid Origin.

Discussion

This is an excessively rare complication following mastoidectomy, but by no means uncommon following an injury to the vault of the nose. Dandy (1926) has discussed this syndrome in detail and has pointed out that an essential feature for the formation of a traumatic pneumocœle is the presence of a ball valve mechanism which allows air to enter but not leave the dural defect. In many cases the air percolates into the subarachnoid space, but where there is trauma to the overlying brain an air sack may form within the brain itself.

The precipitating factor in nasal cases is the act of coughing or sneezing. This raises the presure in the vault of the nose sufficiently to drive air through the ball valve.

This explanation however is not applicable to the mastoid. It is most unlikely that any increase in epipharyngeal pressure could traverse the eustachian tube and set up sufficient force within the mastoid to produce a pneumocœle in the manner described above. Moreover, in the present case, even if air were driven up the eustachian tube, it could not produce any significant rise in intramastoid pressure, since the operation had already placed the mastoid cavity in free communication with the exterior.

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PLATE 2.

Brow Up Position. Intracerebral Pneumocœle of Mastoid Origin.

The problem can be resolved on hydrodynamic principles as follows:

ABCD is a rigid box filled with water. When B is vertically above D (Fig. 1) the hydrostatic pressure difference between BD corresponds to the length BD. i.e. *relative* to D the pressure of ABC is *negative*. When the box is laid flat (Fig. 2) this pressure difference disappears. Despite these pressure variations, no fluid movement occurs because the box is rigid. If however, EFD is non rigid then some slight fluid displacement will occur as in Fig. 3.

The conditions with the rigid box ABCEF are unaltered and in particular the relative negative pressure at B persists. If now the rigid box is breached, this negative pressure sucks air in and the downward bellying of EFD becomes much more *pronounced* as in Fig. 4.

In the normal animal the conditions approximate to Fig. 3 in so far as the cranium is a rigid box, whilst the spinal portion of the cerebrospinal system is capable of limited bulging. In addition, however, whilst the fluid in Fig. 3 has a fixed volume, that of the cerebrospinal system can alter for two reasons.

(*a*) Surging of the vascular tree.

(*b*) Absorption and secretion of cerebrospinal fluid.

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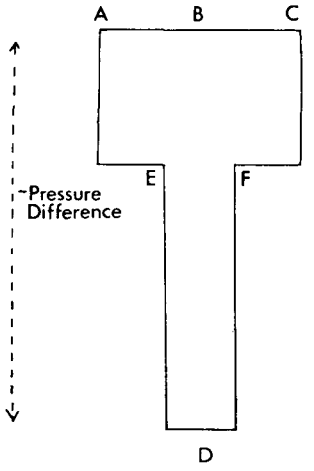


FIG. 1.

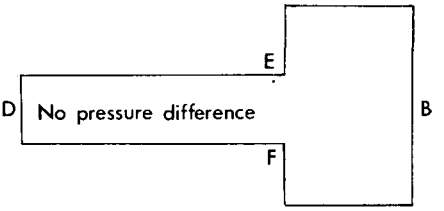


FIG. 2.

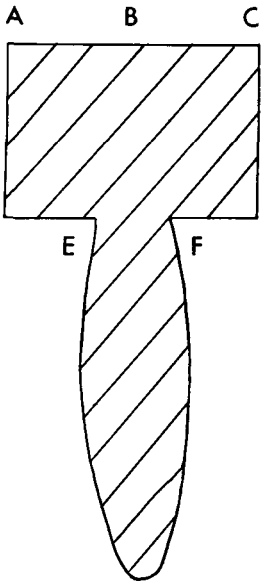


FIG. 3.

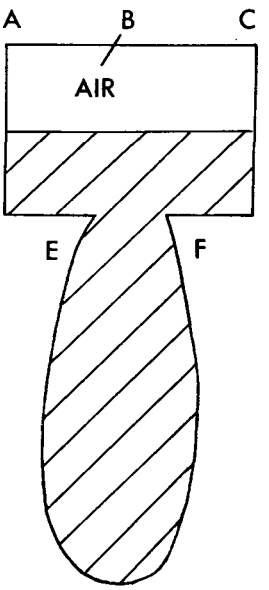


FIG. 4.

The former occurs with every physiological or pathogical alteration in the volume of intracranial blood. Clearly this must produce an equal and opposite change in the volume of the other two fluid components (i.e. the cerebrospinal fluid and the brain itself), since the total volume of the three constituents corresponds to the absolute capacity of the cranial box, which is constant. This is the so-called Monro-Kelly doctrine.

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The effect of postaural changes on the normal animal is more complex. In passing from the horizontal to the vertical position both the cerebrospinal fluid and venous systems develop a pressure gradient, compare Figs. 2 and 3. This again does not necessarily imply any displacement of fluid *out of* the cranium, unless some compensatory flow of fluid *into* the cranium can occur. Under normal circumstances such a flow might occur in the arterial tree although no definite experimental evidence is available.

The case herein described indicates how this can occur, under pathological circumstances.

The process may be envisaged as follows: First the rigid box is breached. Secondly changes of posture from horizontal to vertical produce negative pressures within the cranial box, which tend to suck in a small quantity of air. During the vertical posture this air becomes trapped by the ball valve mechanism and produces a raised intracranial pressure in the horizontal posture. This however evokes a compensatory absorption of the cerebrospinal fluid, so that the horizontal pressure returns to normal. Consequently, when next the patient stands up, a further bubble of air is sucked in, and so the process continues remorselessly until the gradual replacement of cerebrospinal fluid by air, with its associated effect on the brain and meninges, evokes symptoms.

Thus in the formation of this type of intracranial pneumocœle three factors are involved.

(1) The break in the rigid box.
(2) The ball valve mechanism consequent on the laceration of soft tissue.
(3) The absorption of cerebrospinal fluid.

Theoretically any of the spaces can be involved. Thus if the dura alone is damaged, air accumulates between it and the pia arachnoid to form a subdural pneumocœle.

It is well known that cerebral tissues, being semi-fluid, present little resistance to external pressure and, as the present case shows, the comparatively slight pressure changes involved in postural variation by their cumulative effect can finally drive the air column right through into the ventricle itself.

Attention may be drawn to the silent period following the actual trauma. In this case no less than 2 weeks elapsed before headache drove the patient to seek assistance once more. In less fortunate cases, violent infection may supervene with meningitis and encephalitis. The silent period is easily understood in view of the minute air transfer which accompanies each change of posture. It follows that wherever any suspicion of such trauma occurs, the surgeon should not be lulled into a feeling of false security by the apparent absence of symptoms. Air in the cranial cavity is easily demonstrated by X-ray and this examination should be carried out as often as necessary until all danger is past.

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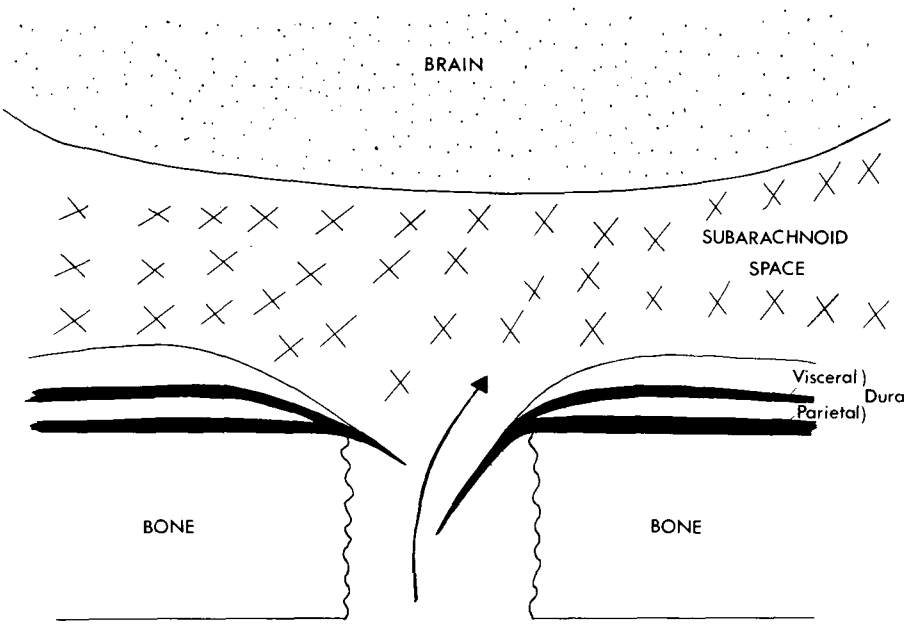


FIG. 5.

Subarachnoid Pneumocœle. This occurs when the subarachnoid space has not been sealed off by inflammatory adhesions.

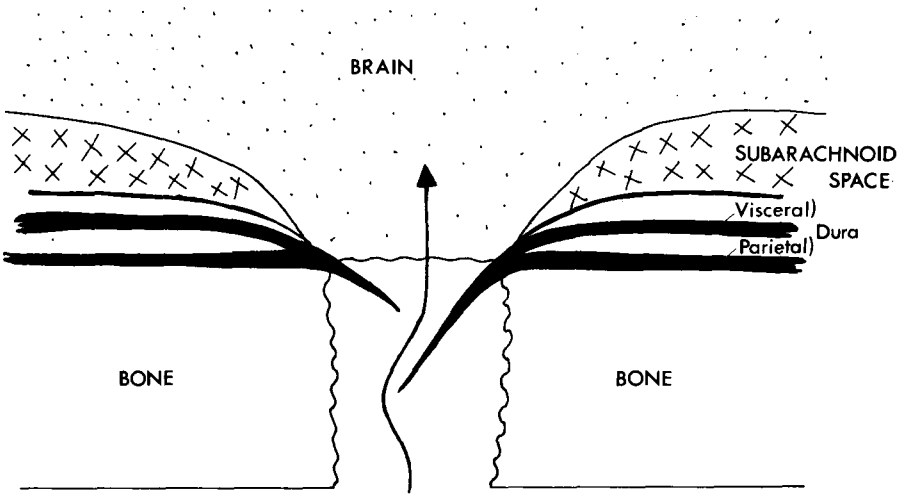


FIG. 6.

Intracerebral Pneumocœle. If the subarachnoid space is defended by adhesions and where the brain tissue has been traumatized, the pneumocœle forms within the brain substance.

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Where a pneumocœle is in fact demonstrated, operative repair should be undertaken immediately. Scrupulous debridement should be carried out and the defect sealed in the most suitable manner. The results, as this case shows, can be highly satisfactory.

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