Traumatic pneumocephalus

By R. T. Ramsden and J. Block (Dundee)

Pneumocephalus is a condition which is only occasionally encountered in otorhinolaryngological practice. When it does occur, however, it may often be missed, owing no doubt to its very rarity. It is the purpose of this paper to alert those who may deal with fractures of the paranasal air sinuses to this complication. A case is reported which illustrates how extensive cerebral destruction may result if the presence of intracranial air is not suspected early. In addition, the anatomical and pathophysiological factors which influence the development of pneumocephalus are discussed.

Case report

A thirty-four-year-old man was admitted to another hospital on 18 November 1970 with multiple injuries. For some years the patient had been under psychiatric care for a manic-depressive illness and had apparently fallen from a fourth floor window. His injuries included multiple comminuted fractures of the skull (Fig. 1), with neurological evidence of associated injuries to the left cerebral hemisphere. He also had multiple fractures of his ribs on the right side, fractures of his pelvis and right lateral malleolus, compound fractures of both forearms and a dislocation of the right wrist with fractures of several of the carpal bones. He showed evidence of intra-abdominal injuries and an immediate laparotomy disclosed a ruptured bladder and lacerated liver. His post-operative recovery was complicated by renal and respiratory difficulties, the latter necessitating tracheostomy.

During his convalescence he was noted to be slightly depressed, with poor concentration, memory disturbance and difficulty in 'finding the right word'. By the middle of January 1971 he was considered fit for discharge to his home, although he was still gravely disabled as a result of his orthopaedic injuries.

His psychological state then rapidly deteriorated and within a few days it was necessary to admit him again under psychiatric care with severe depression, apathy and marked debility. Shortly afterwards he developed septicaemia related to a large pressure sore and became extremely ill. Antibiotic therapy was instituted and he improved considerably. However, although fully orientated, he had become totally apathetic, making no effort to feed himself, and was incontinent of urine and faeces. Slight nominal dysphasia was observed. A Consultant Psychiatrist considered that this state was one of a severe depressive of catatonic type and considered a course of electro-convulsive therapy. However, he felt that prior to instituting this treatment an organic lesion such as a subdural haematoma should be excluded and he was referred to the Department of Surgical Neurology at the Dundee Royal Infirmary.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

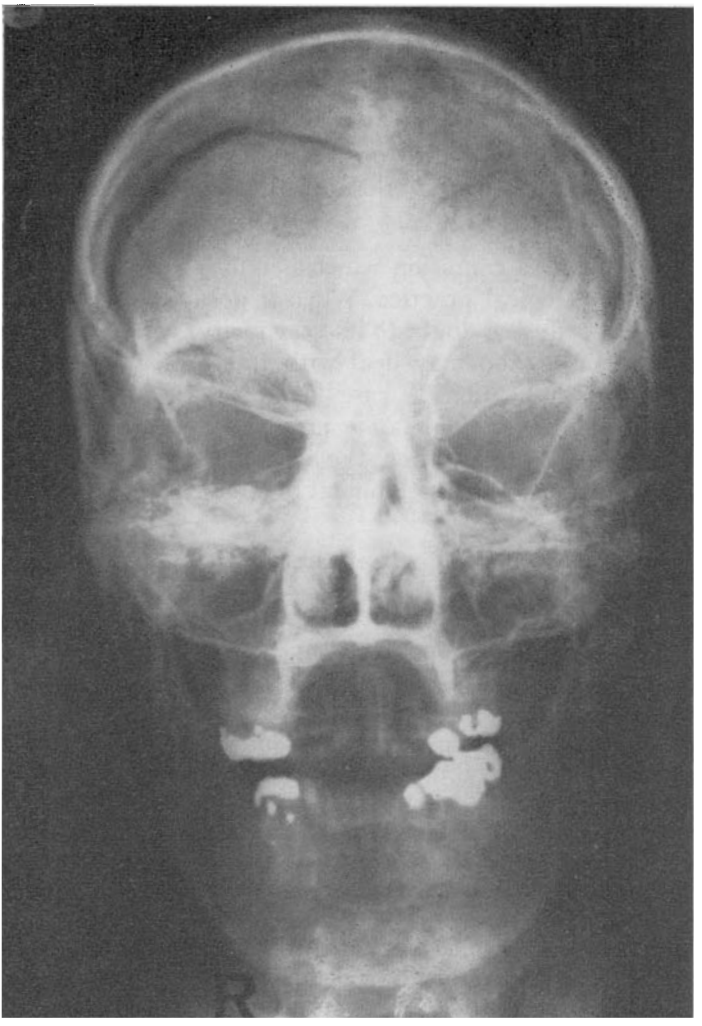


FIG. 1.

Taken immediately following the injury. Multiple comminuted fractures are seen in the frontal region, more marked on the right side.

On admission to this department the patient was co-operative and although he was able to give a reasonable account of his illness it soon became apparent that he was mildly confused, with evidence of some residual nominal dysphasia. He was apathetic and totally indifferent to his faecal incontinence. Although neurological examination was somewhat restricted by his mental state, there was no evidence of raised intracranial pressure and no other neurological abnormality was found. (Subsequently, when his condition had improved, he was shown to have bilateral anosmia.) Radiographs of his skull were then taken and these showed the striking appearance of bilateral frontal intracerebral

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

pneumatocoeles (Figs. 2 and 3). These were extensive in size and clearly communicated with the ventricular system, but there was no indication of a lateral shift on the antero-posterior projection. The patient was then turned into the prone position and there was an immediate flow of about 20 ml. of cerebrospinal

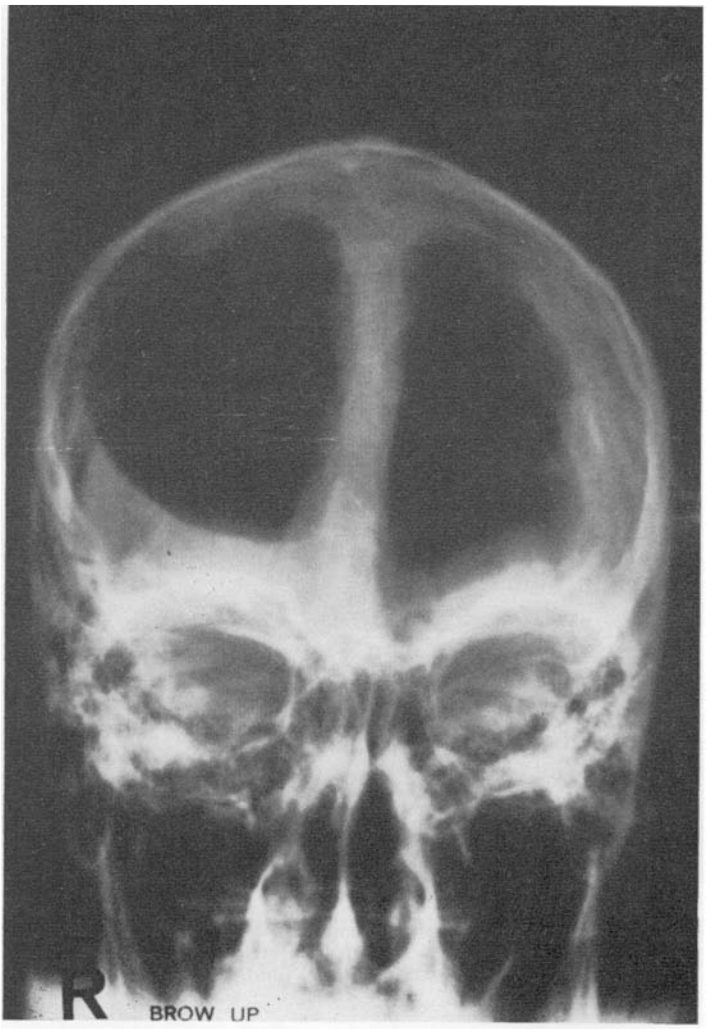


FIG. 2.

Supine, antero-posterior projection. Two intracerebral pneumatocoeles are clearly visible, with no significant shift from side to side.

fluid from the left nostril. A radiograph taken in this position (Fig. 4) shows the free communication with the ventricular system, which is somewhat dilated, with the anterior horns of the lateral ventricles displaced backwards. He was

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

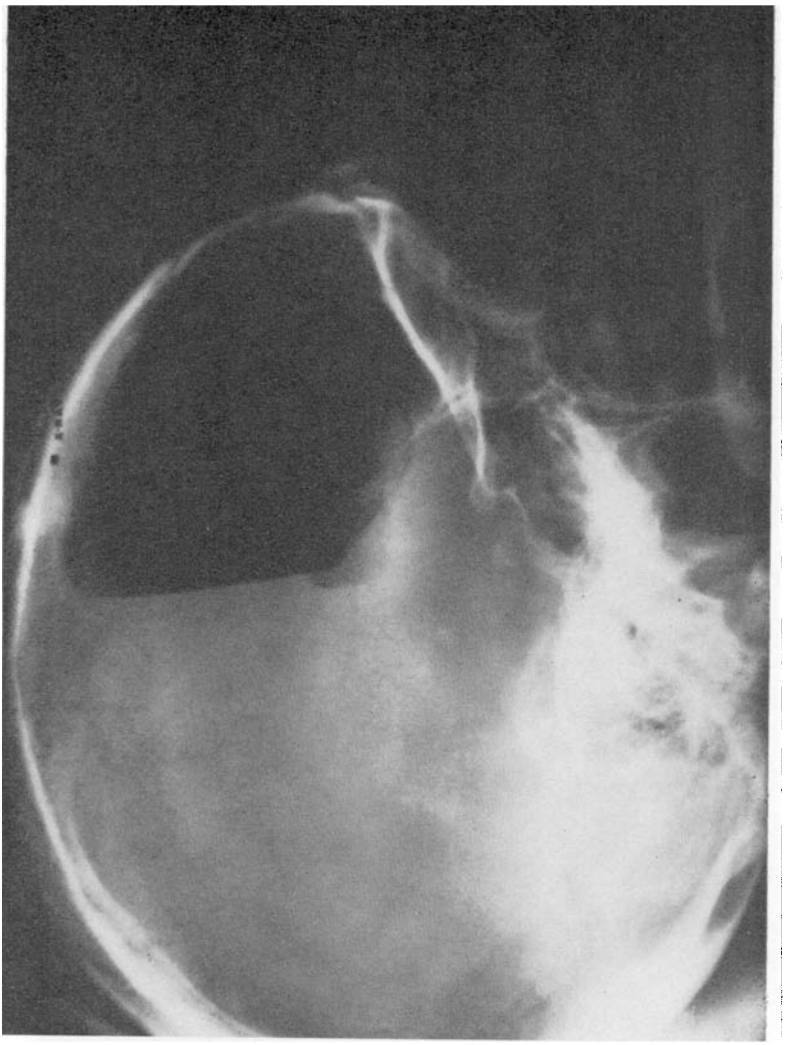


FIG. 3.

Supine, lateral. The two pneumatocoeles are superimposed with a fluid level clearly visible on one side. A small amount of air is seen in one lateral ventricle. A fracture is seen to involve the posterior wall of the frontal sinus.

again turned on to his back and a further radiograph showed that whereas one pneumatocoele had largely emptied into the ventricular system and through the foramina of the fourth ventricle into the spinal theca, the size of the other pneumatocoele remained unchanged. Although sinus X-rays at the time of the accident had not revealed any injury to the frontal sinuses, tomography of the fronto-ethmoidal area carried out in the light of the discovery of air in the skull did show fractures of both right and left frontal sinuses (Fig. 5).

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

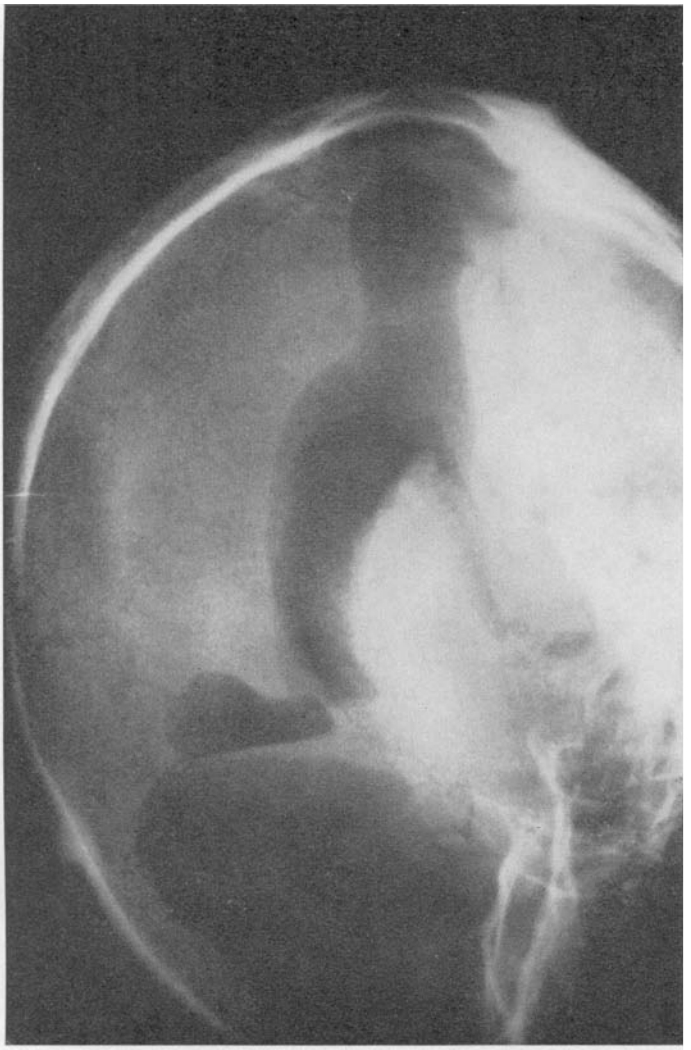


FIG. 4.

Prone, lateral. Both lateral ventricles are clearly visible as well as a small amount of residual air in the pneumatocoele on one side. The size of the other pneumatocoele is largely unchanged, indicating that it may not communicate with the ventricular system.

Operation—Anterior cranial fossa exploration (24:2:71):

A hole approximately 5 mm. in diameter was found in the roof of the horizontal part of the left frontal sinus, i.e. the anterior part of the left anterior fossa, and attached to the edges of this hole there was a corresponding defect in the dura. Intradural exploration revealed adhesions between the inferior surface of the frontal lobe and the edges of this dural defect. On the right side an essentially similar situation existed, the fracture of the roof of the frontal sinus

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

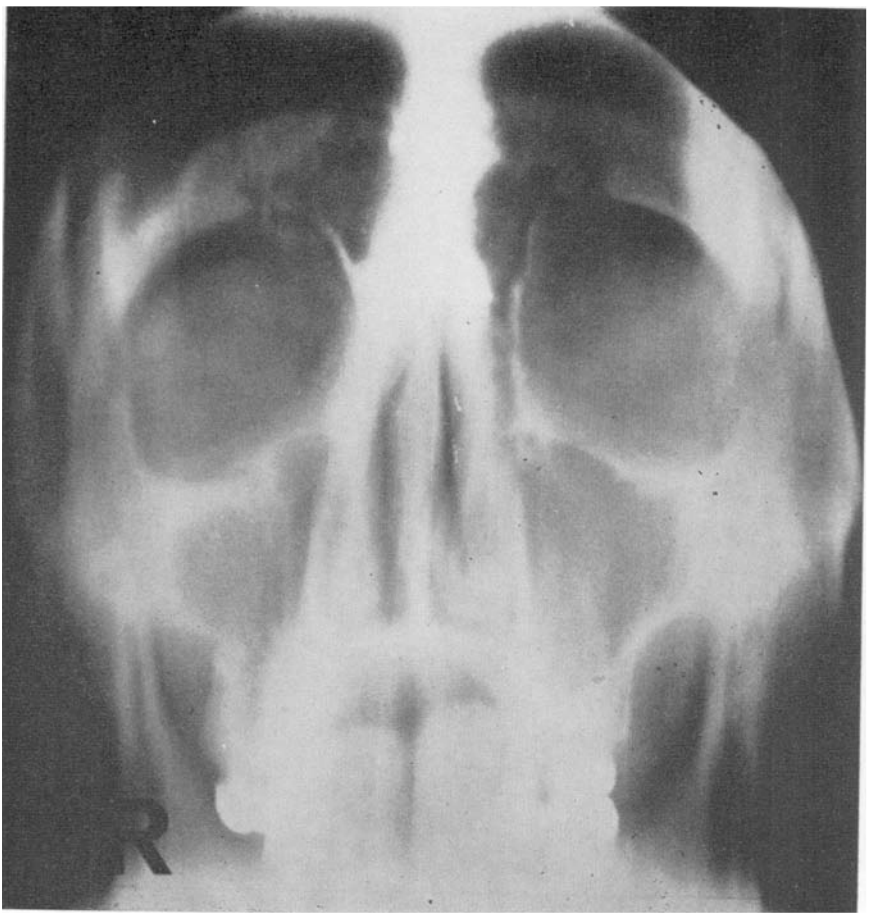


FIG. 5.

Tomogram demonstrating clearly fracture of the right frontal sinus. The fracture involving the left frontal sinus is less well shown on this cut. There is also evidence of a healed fracture of the right malar complex.

being more extensive and comminuted, and the adhesions between the frontal lobe and dura, and the dura and bone, being more marked. The dural defect on the right side was a 7 mm. longitudinal slit. The cribriform plate was intact but the crista galli had been avulsed from the remainder of the ethmoid complex. The olfactory bulbs were not found and some areas of necrotic brain were visible on the inferior surfaces of the frontal lobes. The dural defects were closed with silk sutures and free muscle grafts, and further pieces of muscle inserted into the bony defects.

Following the operation the patient had no further CSF rhinorrhoea and radiological examination on the eighth post-operative day showed that the intracranial air had disappeared almost completely. Mentally he improved gradually during the next three weeks, particularly in regard to his confusion.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

An initial period of disinhibition with continuous talking and singing gradually lapsed into a state of mild depression. He remained incontinent for about four months. Subsequently he had one grand mal seizure and was put on a regular anti-epileptic drug regime.

When reviewed approximately eighteen months after the operation, the patient had had no recurrence of CSF rhinorrhoea and denied all headache. He still had bilateral anosmia. He was no longer incontinent but remained under psychiatric care as an out-patient.

Discussion

The most striking feature of this case is that such gross disruption of both frontal lobes should have progressed unsuspected over a period of almost three months. In the period immediately following the patient's admission to hospital, attention was of necessity directed towards resuscitative measures and care of his orthopaedic injuries, and the possibility of his developing pneumocephalus was not considered, especially as a sinus X-ray on admission had not revealed a frontal sinus fracture. Subsequently the clues to the presence of an expanding intracranial space-occupying lesions were not apparent. The 'silent' nature of the frontal lobes and the consequent difficulty in recognizing damage to them is well known. The apathy, depression and slight dysphasia in this case were the only manifestations of the extensive disruption that had occurred. Because the pneumatocoeles were bilateral, a rare occurrence in itself, no significant lateral shift of cerebral contents resulted, so that in terms of cerebral displacement the pathological state was not greatly different from that obtaining in bilateral subdural haematoma—and no doubt this accounted for the similar clinical picture, with no lateralizing signs. Due to the ready displacement of cerebrospinal fluid, there was no increase in intracranial pressure, hence no drowsiness, vomiting, papilloedema or headache (which is often severe with pneumocephalus).

Pneumocephalus results from the entry of air into the cranial cavity, in contradistinction to the production of gas by organisms introduced at the time of a penetrating injury (Slaughter and Alvis, 1944; Hixon, 1915). In most instances air passes into the skull through a fracture line following trauma. Less frequently the breach is due to erosion from a fronto-ethmoidal osteoma (Cushing, 1927), a postnasal carcinoma (Raider, 1951), or from cholesteatoma in chronic suppurative otitis media or surgery aimed at its removal (Horowitz, 1964).

Although Chiari (1884) is generally credited with first recognizing pneumocephalus at postmortem, it was Luckett (1913) who first demonstrated intracranial air radiologically. Since then the condition has been recognized with increasing frequency, and several comprehensive surveys exist, notably by Dandy (1926) and Jelsma and Moore (1954).

Dandy classified pneumocephalus as follows: 1. Subarachnoid; 2.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

Subdural; 3. Intracerebral; 4. Intraventricular; and to these Jelsma and Moore (1954) added extradural pneumocephalus, and described one case. The commonest variety of pneumocephalus is the collection of air occurring in the substance of the brain (Dandy, 1926). It is nearly always unilateral, although a few bilateral cases have been recorded (Lewis, 1928; Tönnis and Frowein, 1952; Eaglesham, 1945).

Most cases of traumatic intracranial pneumocephalus have had fractures involving the paranasal air sinuses. Dandy (1926) maintained that air could only be forced into the cranium through an opening in the wall of an air sinus, or from the mastoid air cells, because only in these sites could air pressure be built up, either by coughing or sneezing, until it became greater than the intracranial pressure. If this mechanism is responsible, it would seem to imply a valvular mechanism which allowed air to pass into the sinus in question (usually the frontal via the frontonasal duct), but prevented its subsequent escape.

The occurrence, however, of intracranial air in a few cases where the fracture line involved the calvarium alone demands an alternative explanation. Horowitz (1964) proposes a hydrodynamic theory based on the Monro-Kelly principle to explain the entry of air. Changes in posture produce pressure changes inside the skull, sucking in air through the fracture line. A valve mechanism, for example a flap of dura, prevents the air from escaping and compensatory resorption of CSF allows more and more air to enter as the cycle is repeated.

Knowledge of the anatomy of the meninges in the anterior and middle cranial fossae assists in the appreciation of the way in which a pneumocephalus develops. Over the inner wall of the frontal sinus and the cribriform plate the dura is very closely applied to the bone, and in the latter situation is thin (Cairns, 1937). In addition, the arachnoid is very closely applied to the undersurface of the frontal lobe, especially at the frontal pole. In a fracture of the frontal or ethmoidal sinuses, a fragment of bone may tear the dura without opening the subarachnoid space, allowing a subdural pneumatocoele to collect. Alternatively the arachnoid may be torn, but if it is, the underlying brain tissue is likely to be traumatized too, especially over the frontal pole. Local and generalized cerebral oedema act to block off the meningeal opening with brain tissue, followed by adhesions between the traumatized brain, the edges of the meningeal tear and the fracture line. Liquefaction of the injured parenchyma may ensue. Air may now be forced into the substance of the frontal lobe, causing progressive separation and compression of brain tissue until eventual rupture into the lateral ventricle occurs, an event which may be heralded by the commencement of CSF rhinorrhoea.

A frontal pneumatocoele has been observed within hours of the injury (Rand, 1930), but most cases are detected within a month of the injury, at which time the air collection is about three to four cms. in diameter

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

(Taveras and Wood, 1966). On the other hand, the disruption of the frontal lobe by air may be a very gradual process requiring up to 17 months to reach the ventricle (Dandy, 1926). It may be not so much gradual as delayed: once the conditions exist, viz. adhesions between fracture line, meninges and brain, it may be weeks or months before air is forced into the brain substance by a sneeze or similar occurrence. Occasionally sudden death follows the rapid entry of a large volume of air into the frontal lobe during a bout of sneezing (Morley and Hetherington, 1957)—the result of sudden uncompensated rise in intracranial pressure. Should, however, the force be sufficient to cause immediate rupture into the lateral ventricle, then a compensatory CSF rhinorrhoea may sometimes save the patient (Luckett, 1913).

Many writers confirm that frontoethmoidal fractures are the most commonly encountered, and the association of fractures in this site with subdural or intracerebral pneumatocoeles is well established (Eaglesham, 1945; Morley and Hetherington, 1957; Garland and Mottram, 1945). All of Eaglesham's eight cases of intracerebral pneumatocoele occurred in the frontal lobe, and all had origin in the frontoethmoidal regions. Less commonly the sphenoidal or posterior ethmoidal sinuses are the site of the fracture. In this situation the dura and the arachnoid are closely applied to one another and, in addition, there is a much larger subarachnoid space—the basal cisterns—than in the frontoethmoidal region. A dural tear in the region of the sphenoid is therefore liable to open the arachnoid space, but spare brain tissue. For these reasons CSF rhinorrhoea and meningitis are more likely to occur than an intracerebral pneumatocoele (Lewis, 1928; Morley and Hetherington, 1957).

CSF Rhinorrhoea The most valuable sign in the diagnosis of pneumocephalus is the presence of CSF rhinorrhoea (Dandy, 1926), though it is not an invariable finding in these cases. It indicates connection with either the subarachnoid space, or the ventricular system, and its significance appears to depend on its quantity and the time interval between the original injury and the appearance of the rhinorrhoea. A frontoethmoidal fracture associated with a tear in the arachnoid is likely to be associated with a scanty, short-lived rhinorrhoea (Lewis, 1928) for two reasons. Firstly, the subarachnoid space is very narrow here, and secondly the defect is likely to be occluded quickly by oedematous brain tissue (Cairns, 1937). Furthermore, if CSF rhinorrhoea commencing at the time of a frontoethmoidal fracture does not quickly dry up, it seems reasonable to conclude that plugging of the meningeal defect is not occurring and that an intracerebral pneumatocoele is unlikely to develop. Rizzoli *et al.* (1954) describe thirteen cases in whom persistent CSF rhinorrhoea complicated frontoethmoidal fracture and none of these patients developed intracerebral pneumocephalus.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

A profuse and continuous rhinorrhoea is more likely to complicate a fracture into the basal cisterns (Dandy, 1926; Jelsma and Moore, 1954), while its late commencement in association with intracerebral air must always imply a communication with the ventricular system. This occurrence may be associated with an improvement in the level of consciousness of the patient (Dandy, 1926). If the rhinorrhoea occurs only on sneezing, a valve system must be present, allowing CSF to flow out only when the tract is forced open by the pressure of the sneeze.

Pneumocephalus may often be missed by the clinician. Indeed Taveras and Wood (1966) go so far as to say that the majority of intracerebral pneumatocoeles referred to specialist hospitals have not been correctly diagnosed. One should be alerted to the possibility of its development by a history of violence to the frontonasal area, as in dashboard injuries, in particular if a fracture of the inner wall of the frontal sinus is revealed radiologically, though it is worth bearing in mind that even with tomography some fractures, particularly in the area of the cribriform plate, will not be demonstrated. CSF rhinorrhoea, if detected, is the most helpful clue. Its presence may not, however, be suspected, either by the patient or the surgeon, if there is coexistent epistaxis. In the dorsal recumbent position CSF may trickle undetected into the nasopharynx and be swallowed. Furthermore, it may be assumed to be the watery rhinorrhoea of vasomotor or allergic rhinitis, particularly if bilateral. Biochemical or radio-isotopic studies may help in differentiation. Nursing staff should be instructed in the significance of a watery discharge from the nose following head injury and to record this observation in the notes.

Not uncommonly the patient may have recovered from the immediate effects of his injury but present later with a frontal lobe syndrome due to disruption of the brain tissue by air. Because of the 'silent' nature of the frontal lobes, damage may be gross before it is detected, particularly if, as in our case, the lesion is bilateral, with no mid-line shift to produce lateralizing signs. The clinical picture of apathy and depression may be attributed erroneously to a post-traumatic psychosis. Clouding of consciousness and papilloedema may alert one, but if there is a free compensatory flow of CSF these will not be present. Other late complications of head injury, for example subdural haematoma (Dandy, 1926) or meningitis, may be suspected. A plain skull X-ray is all that is needed to confirm that air is present, and this should be a routine practice in the follow-up of those patients who are felt to be at risk.

Summary

A case is described of pneumocephalus involving both frontal lobes, following a frontoethmoidal fracture. It is noteworthy because it illustrates how extensive cerebral damage may be before it is detected.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

Traumatic pneumocephalus

Intracerebral pneumocephalus occurs most commonly in fractures involving the frontal sinus or anterior ethmoidal/cribriform plate area. These fractures are not rare, and this potential complication should be in the minds of Ear, Nose and Throat surgeons who deal with trauma to the frontonasal region.

REFERENCES

CAIRNS, H. (1937) *Journal of Laryngology and Otology*, **52,** 589.
CHIARI, H. (1884) *Zeitschrift für Heilkunde*, **5,** 383.
CUSHING, H. (1927) *Surgery, Gynecology and Obstetrics*, **44,** 721.
DANDY, W. E. (1926) *Archives of Surgery*, **12,** 949.
EAGLESHAM, E. C. (1945) *British Journal of Radiology*, **78,** 335.
GARLAND, L. H., and MOTTRAM, M. E. (1945) *Radiology*, **44,** 237.
HIXON (1915) *British Medical Journal*, **2,** 905.
HOROWITZ, M. (1964) *Journal of Laryngology and Otology*, **78,** 128.
JELSMA, F., and MOORE, D. F. (1954) *American Journal of Surgery*, **87,** 437.
LEWIS, A. J. (1928) *Brain*, **51,** 221.
LUCKETT, W. H. (1913) *Surgery, Gynecology and Obstetrics*, **17,** 237.
MORLEY, T. P., and HETHERINGTON, R. F. (1957) *Surgery, Gynecology and Obstetrics*, **104,** 88.
RAIDER, L. (1951) *American Journal of Roentgenology*, **66,** 231.
RAND, C. W. (1930) *Archives of Surgery*, **20,** 935.
RIZZOLI, H. V., HAYES, G. J., and STEELMAN, H. F. (1954) *Journal of Neurosurgery*, **11,** 277.
SLAUGHTER, H., and ALVIS, B. Y. (1944) *American Journal of Ophthalmology*, **27,** 617.
TAVERAS, J. M., and WOOD, E. (1966) *Diagnostic Neuroradiology*. Baltimore. The Williams and Wilkins Company, Vol. 1, p. 770.
TÖNNIS, W., and FROWEIN, R. (1952) *Zentralblatt für Neurochirurgie*, **12,** 323.

Depts. of Otolaryngology and Neurosurgery,
Dundee Royal Infirmary,
Dundee,
Scotland.

Present address:
R. T. Ramsden,
E.N.T. Dept.,
London Hospital,
Whitechapel,
London E1 1BB.

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press

https://doi.org/10.1017/S0022215100082165 Published online by Cambridge University Press