INTRACRANIAL INFECTIONS OF RHINOGENOUS ORIGIN*

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In this paper I discuss certain aspects of intracranial infections arising from or related to infection of the paranasal sinuses. These sinuses may be either the primary focus of infection, or they may become infected as a result of local trauma such as compound fracture or—more rarely—neoplastic disease causing disruption or distruction of their thin bony walls, that is to say, the posterior wall of the frontal sinus or the maxilla.

Intracranial infections of this kind have certain peculiarities which make them interesting and important for the neurologist as well as the neuro-surgeon, and even for the psychiatrist because of the close relationship to the frontal lobe of the brain. A striking change in behaviour or personality may be the first and in fact the only sign of a unilateral frontal abscess; at a later stage the question not infrequently arises whether such patients should be certified and sent into a mental institution—even after radical excision of a frontal abscess or repair of a dural defect resulting in rhinorrhoea.

There are 25 cases in the present series, which excludes the many war injuries involving the paranasal sinuses in the main, detailed case records of injuries due to high velocity missiles seen during the war are not available at present.

In Table I the cases presented here are classified according

* Paper read at annual general meeting of National Group of Neurologists, Psychiatrists and Neuro-Surgeons, Durban, September, 1958.

to their underlying pathology. It is now commonly accepted that in most of these cases the infection spreads into the cranial cavity by way of an intermediary *thrombophlebitis* of the diploic veins, mainly the vena diploica frontalis at the upper margin of the orbit, and perhaps the anterior temporal diploic veins. Courville[1] calls attention to retrograde extension along the venous channels. I wish to stress this point because septic thrombophlebitis and sinus thrombosis may be a serious complication of any form of intracranial infection, especially of brain abscesses.[2, 3] To this mode of extension is also possibly related the multiplicity and the peculiar dumb-bell shape of some of these brain abscesses; they are aptly also called hour-glass abscesses.

TABLE I. CLASSIFICATION OF CASES

Rhinogenous frontal abscess	8
Pott's puffy tumour	6
C.S.F. leak	8
Frontal subdural empyema	2
Frontal encephalocele (case 4) ..	1
Total	25

Case 1. *Boy aged* 17. *Right frontal rhinogenous abscess. Rupture during radical excision followed by meningitis.*

This boy had no localizing signs on admission. The diagnosis of a rhinogenous cerebral abscess was made for the following reasons: Some 6 weeks before admission a subcutaneous abscess in the right upper eyelid had been incised as was evidenced by a surgical scar at this place. Following this, the boy developed frontal headache and failing vision and eventually became drowsy. High papilloedema was seen on both sides. X-rays showed opacity of the frontal sinus, the outlines of which were indistinct. The sedimentation rate was markedly increased. A right carotid angiogram revealed a mass in the right frontal lobe.

At the operation, a frontal flap was raised on the right and the frontal lobe of the brain was excised, including two well-encapsulated abscesses, one the size of a peach, the other that of a walnut. In the course of excision the abscess ruptured. The wound was cleaned and closed up, including the dura, without a drain. Within the following 8 days the patient developed a purulent meningitis despite heavy doses of sulphonamides (at that time—1942—penicillin was not available) and from the lumbar fluid *Streptococcus pyogenes haemolyticus* was cultured. The craniotomy wound also became septic. On the request of his parents the patient was discharged in a hopeless condition. Fig. 1 shows the operative specimen with one of the abscesses visible on the cut surface. A tangle of thrombophlebitic veins can be seen as an appendage on the right.

Fig. 1. Operative specimen of a rhinogenous frontal abscess of some 8 weeks' duration. The appendage on the right consists of thrombosed veins (case 1).

Case 1 brings us to a short discussion of *osteitis of the frontal bone* related to infection of the adjacent frontal sinus. Locally, this condition usually presents with a pitting oedema of the scalp over the frontal region, frequently giving the impression of diffuse or localized fluctuation, and extending to the soft parts of the orbit and the upper part of the face. Percival Pott was the first to describe this sign in 1760, and after him it is called 'Pott's puffy tumour'.[4] Pott found it in cases of extradural abscess complicating frontal osteomyelitis due to sinus infection or compound fracture. However, it may be found also in a variety of other lesions closely related to those under consideration here.

The clinical syndrome associated with Pott's tumour may be a very acute one; the following salient features emerge from my own experiences with 6 cases which came under my care during recent years:

1. The condition usually sets in rather acutely, with frontal headache, a puffy swelling over the forehead, stiffness of the neck, and pyrexia; the patient may become increasingly drowsy.

2. The differential diagnosis from an orbital cellulitis or a cavernous-sinus thrombosis may be difficult, and in fact the spreading oedema may result in an abscess in the eyelid or the upper part of the face, necessitating evacuation. Such patients not rarely are first seen by the ophthalmologist or the ear, nose and throat specialist. A surgical attack on the paranasal sinuses, however, may be disappointing—no pus being present. Also, the X-rays may be negative at this early stage. Later the plates usually reveal a mottled appearance of the frontal bone with confluent areas of increased density; sometimes the bone becomes truly ivory. In such chronic cases a serological search for syphilis may be instituted.

3. Finally, the condition may result in the formation of an abscess beneath the galea in the frontal region, requiring surgical drainage.

Fortunately, in some cases the condition takes a chronic course from the beginning, with recurrent acute exacerbations.

In 1955, I saw a Native boy of 13 who was admitted as an emergency case to the Pretoria Hospital with an acute frontal osteomyelitis and Pott's pitting oedema. He died within 4 days from a widespread subdural empyema covering the whole of the right cerebral hemisphere. He was in a comatose state throughout and eventually developed jacksonian fits resulting in a true status epilepticus. Surgical attempts and chemotherapy were unsuccessful.

The following case is an example of Pott's tumour of the classical type.

Case 2. *African male, aged* 37. *Repeated attacks of purulent frontal sinusitis. Evacuation of an extradural frontal abscess by craniotomy. Recovery.*

This man had suffered from recurrent attacks of frontal-sinus empyema, and fair quantities of pus were removed by the ENT specialist on several occasions. On admission, the patient complained of a throbbing pain in his forehead. The scalp there was thickened, of doughy consistence, giving the impression of diffuse fluctuation. There were no other neurological signs. X-rays revealed an opaque frontal sinus and a thickened, mottled frontal

bone. On the cisternal air pictures both anterior horns were equally displaced downward. Likewise, the carotid angiogram evinced a characteristic displacement of the anterior cerebral group.

Bilateral frontal craniotomy was carried out and a quadrangular piece of ivory bone excised, underneath which an extradural abscess with a smooth pyogenic membrane was found. More than 1 fl. oz. of thick pus was evacuated in one stage. The patient was restored to full working capacity within 4 weeks from the operation. He has remained well since.

In the rare cases of *frontal encephalocele* an intracranial infection may be introduced by misdirected surgical procedures, as exemplified by the following case.

Case 3. *African boy, aged* 14. *Surgical attempt to remove a 'lipoma' between the eyes resulting in a CSF leak from the incision. Craniotomy and repair of a frontal encephalomeningocele. Recovery.*

In this boy a tumour between the orbits was misinterpreted as a lipoma and an attempt was made in a country hospital to remove it. This was followed by a persistent leak of CSF through the skin incision, and mild meningitic attacks. A bifrontal Souttard flap was raised and the dura incised on either side of the sagittal sinus, which was ligated and cut. A peculiar tongue-like extension of both frontal lobes was found stretching forward and occupying a roundish defect in the dura and the anterior cranial fossa on either side of the crista galli; it looked exactly like a cerebellar pressure cone. Ingraham and Matson[5] gave a fine picture of this condition. The defect in the anterior cranial fossa was filled with cancellous bone, and the dural defect was grafted from the fascia lata. Fig. 2 shows the patient at the time of his discharge from the King Edward VIII Hospital, Durban.

Fig. 2. Surgically repaired frontal encephalocele in a boy of 14. Note craniotomy scar illustrating right half of a bilateral Souttard flap. Through the stellate scar between the eyes there was C.S.F. leak after a surgical attempt to remove the encephalocele (case 3).

TRAUMATIC GROUP

The cribriform plate is one of the most vulnerable parts of the base of the skull, and the so-called 'indirect' fractures of this plate occur more frequently than it is generally thought. This applies equally to all sorts of blunt injuries to the skull and to injuries caused by high-velocity missiles irrespective to which part of the calvarium the force has been applied. Klaue[6] has rightly drawn attention to this. It follows that the olfactory area is among the most frequent sites of contrecoup lesions of the brain. At necropsy one may find the olfactory tracts completely torn off or heavily contused, and the same applies to the orbital surface of the frontal lobes.

It is useful, therefore, as Spatz[7] suggested, to establish the diagnosis of *anosmia* in all patients with head injury at the very first neurological examination, provided the patient is cooperative and intelligent enough to give reliable answers. The finding of olfactory impairment may give a valuable hint of damage to the cribriform plate with all its consequences, viz. CSF leakage and intracranial infection.

The criteria of paranasal sinus involvement are as follows:

1. CSF leak through the nose or the wound (if any), or into the nasopharynx (which is easily overlooked especially with the patient in the supine position), or, in rare instances, into the orbit.

2. Surgical emphysema in the frontal or orbital regions.

3. Plain X-rays should be taken with a special view of the posterior wall of the frontal sinus and the anterior cranial fossa. A spontaneous pneumocephaly may be missed if not specially looked for; I remember a case in Pretoria where this condition was diagnosed from a tiny air bubble within the pontine cistern.

4. During operation, a conspicuously slack dura should be noted as a sign of intracranial hypotention due to CSF loss. The importance of low intracranial pressure is mentioned here only briefly; it has various aspects which I have outlined elsewhere.[8]

All cases with paranasal sinus injury—not only depressed or comminuted fractures—where there is any suspicion of involvement of the dura call for early surgical intervention. There may or not be an open wound in the frontal region. The surgical intervention includes débridement of the wound with removal of the posterior wall of the frontal sinus, careful inspection of the dura and, if necessary immediate repair of a dural defect. This is the only means of preventing an intracranial spread of infection, which even nowadays is a serious and often fatal complication. The scalp wound should be closed completely, in two layers, if necessary, by means of a rotation flap.

At the beginning of the last war we did not realize how vital an early exenteration of a fractured or infected frontal sinus really was, and the results of inadequate surgery were very bad. I have seen gunshot wounds through the frontal sinus resulting in an encephalitic cerebral fungus with fatal outcome several weeks after the injury.* Autopsy showed that these patients died from a fresh meningitis or subdural empyema or from both. There was little doubt that a slumbering infection within the frontal sinus not properly dealt with surgically was a deciding factor right from the beginning and finally gave rise to the fatal meningitis. However, we soon learned to handle such cases more adequately along the lines mentioned above.[9, 10]

With the present resources of chemotherapy, débridement followed by primary closure can nowadays be carried out successfully even several days after injury—not only within 12 or 24 hours as was usual before the penicillin era. In 1954, I operated upon a young man of 20 who crashed with his car into a moving train. He suffered a deep laceration

* Slides illustrating one of these cases were shown at the meeting.

on his forehead and an extensive comminuted fracture of the frontal and ethmoid sinuses. An unsuccessful surgical attempt had been made on the day of the accident. There was a large defect in the dura with brain extruding, and a slumbering meningitis with polymorphs and lymphocytes but no organisms in the lumbar fluid. The ENT specialist (Dr. J. H. Hofmeyr) found a broad communication with, and a CSF leak into, the nasopharynx. The repair was carried out on the 9th day after the injury. The only neurological signs were anosmia and deafness on the right due to a fracture of the right petrous bone. The man has remained well ever since with a suggestion of a psycho-organic mentality.

At the time of débridement of such wounds the immediate cosmetic result is irrelevant. These patients are on the verge of life and death, and plastic surgery is a cura posterior. Of course the skin incision should nevertheless be planned so as later on to be visible as little as possible. In cases where the incision is not simply an enlargement of the laceration caused by the injury itself we prefer to make a sort of butterfly or cross-bow incision.

If for some reason an immediate débridement cannot be done it is advisable to leave the open wound untouched, and to apply a sterile dressing until the patient can be handed over to a neuro-surgical service. Systemic and intrathecal chemotherapy is a prerequisite. The worst thing to do is a superficial stitching-up of the skin laceration; this will certainly be followed by meningitis.

The question arises how rapidly an intracranial infection may develop after a penetrating skull injury involving the fronto-basal area. From 2 cases of which I have detailed records it is evident that this may happen within 48 hours. By this time, in one of these two, who was not operated upon because of his hopeless condition, a full-blown purulent meningitis was found at necropsy. The other case is the last to be mentioned here (case 4).

Case 4. *Injury to the frontal region involving the frontal and ethmoid sinuses. Skin laceration stitched up in the casualty service. Pneumococcal meningitis within* 2 *days. Radical débridement and closure with drain. Primary healing of the wound. Returned to work. Death from a left frontal abscess* 21 *months after the injury.*

This middle-aged man was admitted to the general surgical ward with an open wound on his forehead and a comminuted fracture involving the paranasal sinuses. The wound had been stitched up by the casualty officer. Within the next 2 days signs of a meningitis became apparent and in the lumbar fluid pneumococci were found. No rhinorrhoea was noticed. The patient was now taken without delay to the theatre and a cross-bow incision was made above the eyes. The frontal sinus was full of clotted blood and debris. There was a stellate fracture of its posterior wall. This was removed, together with several fragments from the ethmoid bone; a minute tear in the dura was closed with a stitch and sealed off with gelfoam. The wound was cleaned thoroughly and sulpha powder sprinkled into the pockets. Two soft rubber drains were inserted and the wound closed up in layers. Recovery was remarkably rapid and without neurological deficit. The wound healed practically by primary union.

The patient resumed his work as a gardener. Later on troublesome personality changes developed, obviously due to damage to the frontal lobes.

One year and 9 months after the injury he was readmitted to another ward, where I had the opportunity of seeing him again. He was drowsy and disorientated, and had an expressive aphasia and a right hemiparesis. The surgical scar on his forehead was solid, and was somewhat drawn in. No CSF leak was present. A lumbar air encephalogram was attempted, which resulted in deepening coma with all signs of tentorial coning. It was obvious that the man had a late traumatic frontal abscess on the left. Unfortunately, operation was delayed and the patient died 5 days after admission. Necropsy revealed an old trephine opening in the frontal bone and a brain abscess in the left frontal lobe. No meningitis was present.

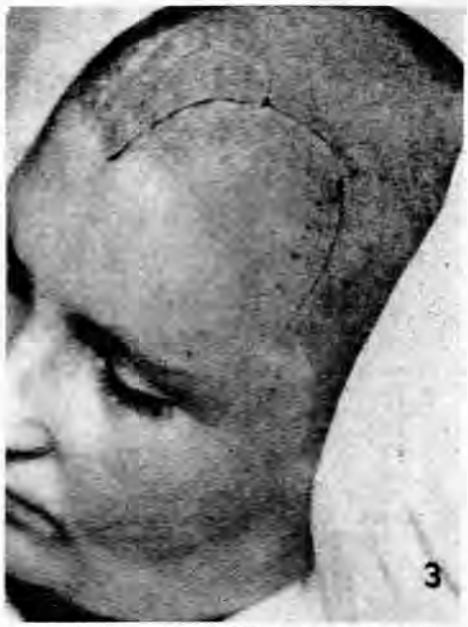

Fig. 3. Dandy's and Olivecrona's standard incision for the approach to the pituitary, the optic chiasma and the cribriform plate. Appearance of scar on the 7th day after craniotomy (from 'Allgemeine Operationslehre' by F. J. Irsigler, in: Handbuch der Neurochirurgie, edited by H. Olivecrona and W. Tönnis, vol. IV. Berlin-Heidelberg-Göttingen: Springer Verlag).

This case demonstrates the necessity of early débridement with inspection of the dura; once a patient has had an attack of meningitis the clinician should constantly be on the look-out for a late traumatic abscess to be attacked without lengthy and unnecessary diagnostic preliminaries.

Remarks on operative technique of dural repair in post-traumatic rhinorrhoea

American authors have advocated a bilateral frontal flap and ligation of the sagittal sinus.[11] This is in most cases unnecessary. A relatively small frontal flap, about the same as the standard incision in use for operations on the pituitary, is sufficient (Fig. 3). An intracerebral aërocele is tapped with the brain cannula, whereupon the air escapes under pressure; there is no difficulty in differentiating it from a frontal abscess. After dissecting the herniated parts of the dura and (not infrequently) the brain, the dural defect is closed with a free graft from the fascia temporalis or fascia lata. Reconstruction of a bony defect on the floor of the anterior fossa is unnecessary. In 1952 Tönnis and Frowein[12] reported on the operative results in 31 cases of traumatic rhinorrhoea and pneumocephaly; the mortality rate in their series was 6·3%.

CONCLUSIONS

The points emerging from the present series are as follows:

1. Even with modern chemotherapy the intracranial

spread of rhinogenous infections cannot be entirely obviated; naturally, chemotherapy is an integral part of any surgical procedures in such conditions.

2. Early diagnosis of sinus involvement is all-important, especially in the traumatic cases. Personality changes may be diagnostic of a unilateral extension into the anterior cranial cavity, and they may persist or even become worse after successful surgical intervention.

3. In cases of so-called Pott's puffy tumour neuro-surgical advice should be called in early because of the great possibility of an intracranial complication such as subdural empyema or brain abscess.

4. Early elimination of the primary focus, be it an infected sinus or a fracture involving its bony walls, is essential. The significance of rhinorrhoea here is quite different from that in CSF-leak through the middle ear, where the great majority of the cases heal up spontaneously.

5. Radical excision of encapsulated rhinogenous frontal abscesses is advocated with the view to preventing late post-traumatic epilepsy[13] and *recurrent thrombophlebitic* abscesses.[14] It has been found that under adequate 'cover' of antibiotics and sulpha drugs primary closure of the craniotomy wound is warranted even if the abscess should rupture during its excision.

My sincere thanks are due to Mr. M. J. Joubert, F.R.C.S., Durban, with whom I had the pleasure of seeing and operating upon two cases of traumatic rhinorrhoea recently; to the colleagues who sent in cases for surgery; and to the medical superintendents of the General Hospital, Pretoria, and the Addington Hospital and King Edward VIII Hospital, Durban, for permission to include cases treated at these hospitals.

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