Sphenoidal defects—a possible cause of cerebrospinal fluid rhinorrhoea

A. C. HOOPER

Department of Anatomy, University College, Dublin, Ireland

SUMMARY In a study of 138 adult sphenoidal bones, 27 defects which could cause cerebrospinal fluid rhinorrhoea were found. Fourteen of these defects were situated at the site of the superior opening of the transient lateral craniopharyngeal canal. The remaining 13 defects were situated along the pathway of the internal carotid artery. The gross and microscopic appearance of the defects is consistent with their production by a process of 'focal atrophy' caused by the pressure of the internal carotid artery and other adjacent structures.

For cerebrospinal fluid (CSF) rhinorrhoea to occur a fistulous track involving arachnoid and dura mater, cranial bone, and nasal mucosa must be established, the cranial bone presenting the greatest obstacle to the leakage of CSF. In some cases of non-traumatic CSF rhinorrhoea raised intracranial pressure is the precipitating cause (Ommaya, di Chiro, Baldwin, and Pennybacker, 1968; Schechter, Rovit, and Nelson, 1969), but frequently no such precipitating cause can be found.

In some cases no definite site of leakage can be found. CSF has leaked through an apparently normal lamina cribrosa (Coleman and Troland, 1947), while a bony defect in this region has also been the cause of CSF leakage (O'Connell, 1964; Nussey, 1966). CSF rhinorrhoea has occurred as a result of fistulae involving the sphenoidal sinus (Ommaya *et al.*, 1968) and a persistent craniopharyngeal canal has also been implicated (Johnston, 1926).

The present paper describes a study of adult sphenoidal bones undertaken to determine the incidence of bony defects which might be the site of CSF leakage in cerebrospinal fluid rhinorrhoea.

MATERIALS AND METHODS

The number of adult sphenoid bones examined was 138. The bodies of the sphenoids were examined for bony defects allowing communication between the sphenoidal sinuses and the intracranial cavity. Bony defects were examined by low power microscopy using incident light. Defects were measured by vernier calipers and for each defect the mean of the largest and smallest diameters was considered the mean diameter. The presence or

absence of a persistent craniopharyngeal canal was noted.

FINDINGS

A total of 41 defects were seen in 31 sphenoid bones. The margin of 14 of these defects appeared jagged and irregular under the microscope and these were considered to be post-mortem artefacts (Fig. 1). This left 16 skulls in which there was one defect, four in which there were two, and one in which there were three defects. Under the microscope the margins of these 27 defects appeared smooth and regular (Fig. 2). These defects are considered to have been present during life and are described below.

None of the defects seen was situated in a mid-line position and it is thus possible to consider the defects as occurring in 276 half-sphenoids. The

FIG. 1. Post-mortem artefact showing jagged irregular margins, $\times 22$.

FIG. 2. Two-part defect showing smooth regular margins, $\times 22$.

defects occurred at four sites (Table 1, Fig. 3). They were all situated in areas where the bone was abnormally thin. This was especially noticeable in the case of defects at the anterior end of the carotid groove underneath the anterior clinoid process. Seven of the defects comprised more than one opening (Fig. 2). One of these was situated at the anterior end of the carotid groove and had nine minute openings in an area of extremely thin bone. The mean diameter of the defects ranged from less than 0.1 mm to 2.5 mm (Fig. 4). The overall mean diameter was 0.9 mm.

FIG. 3. Sphenoid bone: the sites where defects were found are indicated by circles.

TABLE I
SITE OF DEFECTS

Site	Number	Percentage of total defects	Percentage of half-sphenoids examined
Immediately behind superior orbital fissure just below posterior root of lesser wing of sphenoid	14	52	5.1
At anterior end of carotid groove under anterior clinoid process	7	26	2.5
At mid-point of carotid groove	4	15	1.4
At posterior end of carotid groove immediately above foramen lacerum	2	7	0.7
Total	27	100	9.7

A persistent craniopharyngeal canal was seen in two (1.4%) of the sphenoids examined. In one of these the canal had two openings in the hypophyseal fossa, 2 cm apart (Fig. 5).

DISCUSSION

The incidence of persistent craniopharyngeal canal in the present study (1.4%) is somewhat higher than the incidence (0.42%) found in a review of 8,333 skulls (Arey, 1950). Skulls displaying this interesting anatomical feature would however tend to be retained in an anatomical collection.

Non-traumatic CSF rhinorrhoea can occur as a result of extremely small fistulae. Ommaya (1970) had a patient with a fistula 2 mm in diameter, while O'Connell (1964) describes CSF rhinorrhoea as a result of a fistula 1 mm in diameter. Ten of the defects seen in the present study have a mean diameter greater than 1 mm and could therefore be the site of CSF leakage.

The 14 defects situated just below the posterior root of the lesser wing of the sphenoid are at the site of the superior orifice of the lateral craniopharyngeal canal. This little-known canal, described by Sternberg (1888) and Cruveilhier (1877), is a space between the component parts of the developing sphenoid and has normally disappeared by the 10th year. Remnants of this canal in the adult skull were seen in 142 (28.4%) of 500 half-sphenoids (Radoievitch and Jovanovitch, 1956) and 50 (18.1%) of 276 half-sphenoids (Hooper, 1970). Defects allowing communication between the sphenoidal sinus and intracranial cavity in this position were seen in 6.1% of the half-sphenoids described by Radoievitch and Jovanovitch (1956), an incidence comparable with the 5.1% seen in the present study. The other

FIG. 4. Mean diameter of defects.

13 defects were all situated on the path of the internal carotid artery along the body of sphenoid.

The defects seen were all surrounded by an area of extremely thin bone. When viewed under the microscope it appeared as if the bone had been gradually eroded until one or more openings were created. Ommaya *et al.* (1968) suggested that the pressure of CSF in an abnormal arachnoid pouch could cause erosion of the thin sellar floor in a manner similar to the formation of cranial vault excavations by the arachnoid granulation. This 'focal atrophy', as they termed it, could eventually result in the creation of a bony defect, fistulation and CSF rhinorrhoea. O'Connell (1964) advanced a similar hypothesis in two patients who had non-traumatic CSF rhinorrhoea due to a fistula in the lamina cribrosa over which lay a pulsating pocket of CSF. The appearances of the defects seen in the present study are consistent with their production by a process of 'focal atrophy'.

However pressure of CSF in an arachnoid pocket may not be the only cause of this phenomenon. The internal carotid artery is situated in a groove, presumably formed as a result of the pressure of

FIG. 5. Upper end of persistent craniopharyngeal canal. Two openings can be seen in a distorted sellar floor.

the artery as it passes forward along the side of the sphenoid. Similarly the venous sinuses and diploic veins groove the inside of the calvarium. An abnormal internal carotid artery may erode the lateral margins of the dorsum sellae and by erosion undercut the posterior clinoid processes (McLachlan, Williams, and Doyle, 1968). In view of the effect of these vessels on cranial bones, focal atrophy as a result of the pressure of the internal carotid artery would seem a likely cause for the defects seen along the course of that artery, particularly as the largest defects were seen under the anterior clinoid process where the artery loops sharply upwards to take part in the formation of the *circulus arteriosus*.

The cavernous sinus extends as far forward as the superior orbital fissure. It could thus be a cause of focal atrophy resulting in the defects seen beside the posterior root of the lesser wing of sphenoid, as could the pressure of CSF in the sleeve of arachnoid mater surrounding the cranial nerves passing through the superior orbital fissure. Defects in this position might also be created during the development of the sphenoidal sinuses. The sinuses do not form until some time after birth and it is generally agreed that bone is resorbed under the influence of the expanding nasal mucosa (Arey, 1965). Normally this resorption halts just short of the edge of the bone leaving the thin bony wall of the sinus. At the site of the lateral craniopharyngeal canal the bone would present less resistance than elsewhere to this resorption and a defect might thus be created.

The defects seen in the present study might also be congenital in origin. This possibility cannot be entirely ruled out but it poses the question as to why they should occur in these particular sites. The position of the defects seen would more readily support the hypothesis that they were formed by a process of 'focal atrophy'.

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