LOCAL bulging of the skull is an uncommon manifestation of an intracranial space-occupying process. It is commonest in children and the usual causes are chronic subdural hematomas and cerebral tumors. The skull changes in cases of juvenile chronic subdural hematomas have been described by Davidoff and Dyke and Bull. Hardman reported a case in which similar changes in the skull were associated with what was believed to have been a chronic subdural hygroma—a closely related condition. Like cases have been recorded, and they will be reviewed later. We have had such instances and from a study of them fresh suggestions as to their nature will be made.

CASE REPORTS

Case A (NS 5234). A boy aged 8 years was admitted on July 14, 1953 with a 3 months' history of swelling of the head above the left ear. It was thought that this had increased in size. There were rare headaches. He had no other complaints and he was in the normal class for his age.

Examination. The sole physical sign was a painless swelling above the left ear. Roentgenograms of the skull showed thin bulging bone in the left temporal region above and in front of the left ear (Fig. 1). The middle fossa was not expanded forwards. Left carotid arteriogram showed that the middle cerebral artery was elevated from its origin and there was a slight shift of the anterior cerebral artery to the right (Fig. 1). No tumour circulation was seen. EEG showed excessive slow activity in the left temporoparietal leads. The pre-operative diagnosis was of a temporal glioma.

Operation. A temporal bone flap was raised. The bone was thin and bulged outwards. A normal-looking dura mater was opened. At the site of the temporal pole was a bluish transparent cyst. The arachnoid swept off the temporal and frontal lobes to make up the outer wall of the cyst (Fig. 2). There were some small vessels running in the wall to the middle cerebral vessels. The cyst was opened and contained clear fluid. The medial part of the cyst communicated with the subarachnoid space over the free edge of the tentorium. Cerebrospinal fluid welled up into the cyst during the operation. The anterior part of the temporal lobe was absent and such lobe as was present had a normal gyral pattern. There did not appear to be any arachnoid over the brain that walled the cyst. Beside the tentorium, the olfactory, optic and oculomotor nerves and the carotid artery and its bifurcation were visible without retracting the brain. The arachnoid wall was stripped away. In closing the wound, the bone flap was wired into place.

The postoperative course was uneventful and he was well a year later.
**Case A.** Left carotid arteriogram (anteroposterior view) showing elevated course of middle cerebral artery and slight shift of anterior cerebral artery to right. The bulging of the left temporal area can be seen.

**Case B** (NS 6522). A boy aged 14 years was admitted on April 9, 1954 with a story that at the age of 4 years he had been hit on the head with a toy pistol. Since then his parents had noticed that the left temple had been swollen. There had been a dubious increase in size. There were no other complaints and he was in the normal class for his age.

**Fig. 1.** *Case A.* View of operation showing the cyst at the temporal pole. This is covered with arachnoid.
**Fig. 3.** Case B. Left carotid arteriogram (anteroposterior view) showing elevated course of middle cerebral artery and the space between brain and skull. Some bulging of the skull can be seen in the left temporal area.

**Examination.** There was considerable protrusion of the head in the left temporoparietal region. There were no other physical signs. Roentgenograms of the skull showed marked bulging of the left temporoparietal area with thinning of the bone.

**Fig. 4.** Case B. View of operation, showing the temporal pole to be absent. The insula can be seen. There is no arachnoid covering to the underlying brain.
There was forward bulging of the middle fossa that was best seen on base views. Left carotid arteriography gave no filling of the anterior cerebral artery. The middle cerebral artery was elevated throughout its course. There was an avascular area between the brain and the skull (Fig. 3). EEG showed low voltages in the left frontoparietal leads.

**Operation.** A temporoparietal bone flap was elevated. The bone was very thin and bulged outwards in its centre. The dura mater was bluish and on opening it there was a large collection of clear fluid in the anterior part of the middle fossa. Inspection showed that the arachnoid had been opened with the dura mater. It was reflected for a considerable distance from the underlying frontal and temporal lobes. The arachnoid was thickened where it resumed its normal relations with the pia mater. The temporal pole of the brain was absent. The gyri were free of arachnoid and had a normal pattern, and the vessels stood out clearly. The insula was visible and the temporal operculum was absent (Fig. 4). Cerebrospinal fluid welled into the cyst from over the tentorium. The internal carotid artery, the middle cerebral artery and the oculomotor nerve were free and ran in the medial wall of the cyst. Some of the peripheral dura mater and arachnoid were taken for examination. The wound was closed and the bone flap was wired into place.

**Postoperative course** was satisfactory and he was well 9 months later.

**Histological Examination.** The membranes showed a normal dura mater and an inner membrane similar to arachnoid.

**Comment.** The essential feature of these 2 cases was the asymmetry of the skull without other symptoms. On inspection, the anterior part of the middle fossa was occupied by a collection of clear fluid in the subarachnoid space. The temporal pole of the brain was absent and there was no evidence of compression of the brain. Case B had the insula uncovered.

Fifteen similar cases were found in the literature (Table 1).

**ANALYSIS OF MATERIAL**

Analysis of the 15 cases from the literature with the 2 cases presented herein shows the common pattern.

**Age and Sex.** Seven cases were in the first decade, 6 in the second decade and 4 patients were over 21 years of age. There were 13 males and 4 females.

**Symptoms.** The left side of the head was involved in 12 of the 17 cases. In most patients the bulging of the temporal region of the skull had been evident for many years and other symptoms were trivial. Neurological signs were mostly absent or trivial. There were 4 cases (Cases 1, 7, 11 and 14) in which the symptoms were unusual. This was because of the superimposition of other factors. Cases 1 and 11 had had recent head injuries and Case 7 had malignant changes in the lining membrane. The cause of symptoms in Case 14 is uncertain. There was often a distant minor head injury and this seems to have drawn attention to the asymmetry of the skull.

**Radiography.** There was thinning and bulging of the temporal squama of the skull and the adjacent parts of the frontal and parietal bones were often affected. Only in Case 1 was the skull normal. In most instances the middle fossa was enlarged forwards and the lesser wing of the sphenoid was elevated.
## INTRACRANIAL COLLECTIONS OF FLUID

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Author</th>
<th>Sex</th>
<th>Side</th>
<th>Symptoms and Signs</th>
<th>Radiology</th>
<th>Operative Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Cohen</td>
<td>M</td>
<td>L</td>
<td>Head blow boxing 6 wks. before; Then headaches, vomiting.</td>
<td>Skull normal. Air studies by direct injection: large frontal cyst.</td>
<td>Trephine: 500 cc. brown fluid under dura mater. Frontoparietal flap; 5 cm. incision of anterior fossa and ant. part middle fossa; II and III nerves seen, recovery.</td>
</tr>
</tbody>
</table>
In cases in which air studies of the cerebral ventricles were done (Cases 3, 4, 5, 14 and 15) there was a minimal shift of the ventricles to the side opposite the bulge. The temporal horn was truncated or dorsally displaced. In none was the cyst filled with air. Carotid arteriography (Cases A, B, 7 and 11) showed that the middle cerebral vessels were displaced upwards and medially while the ipsilateral anterior cerebral artery was shifted to the opposite side.

Electroencephalography. This was done in Cases A, B, 6 and 7 and in 3 of the 4 there were focal features.

Fluid Contents. In 5 cases (Cases 2, 8, 10, 14 and 15) the cyst fluid was analysed for protein content and it was similar to the cerebrospinal fluid findings; in no instance was the protein content raised.

Operative Appearances. The common feature was that the anterior part of the middle fossa was occupied by a large collection of clear fluid—up to 125 cc.—so that a gap appeared between the frontal and temporal lobes. The sylvian fissure was opened up. The insula was uncovered in 3 cases (Cases B, 6 and 9) and probably so in Case 7. Cases 10 and 11 had absence of the polar portions of the frontal lobe as well, and Case 1 had a frontal polar cyst but the state of the temporal lobe was not known. The free edge of the tentorium was often seen and a variety of cranial nerves from the olfactory nerve to the gasserian ganglion were seen without retraction of the brain. The internal carotid artery and its major branches were exposed.

From many of the operative accounts it is difficult to decide what were the exact relations of the fluid collections to the containing membranes. The trend of description is to suggest that the fluid was in the subdural space. The fluid was subarachnoid in Cases A and B. There was a separate lining to the cyst in Cases 6, 10, 13 and 14 and there was arachnoid between the cyst wall and the brain.

Results. Case 5 was not operated upon. The rest of the patients recovered from their operations and were well for periods up to 7 years. No mention was made of any alteration in bulging of the skull.

DISCUSSION

Penfield provided 2 cases for Childe’s series and in the subsequent discussion he said, “In the past I, like some others, have assumed that the fluid was subdural but I am beginning to wonder whether in these cases that he has presented the collection may not be subarachnoid instead.” No further evidence was offered. This point is exemplified by the literature. Surgeons have usually believed that no large collection of fluid could occur in the subarachnoid space because of its trabeculation. Any large fluid mass would be deemed to be in the subdural space which has a great potential capacity. It is not easy to determine the exact relations of the membranes when a limited exploration of the intracranial contents is made. We were sure of the subarachnoid location of the fluid in our cases and there were no additional membranes.

These collections of fluid have been called subdural hygromas by most
authors. This has carried with it the implication that they are related to the subdural hygromas that follow a closed head injury. A minor head injury was believed to have initiated the condition in several cases. However in others the bulging of the skull had been noticed at an early age and an injury served to accentuate the asymmetry of the skull. It is probable that this condition is determined prenatally and any subsequent injury is a matter of coincidence.

Some operative accounts suggested that the brain was displaced by the fluid to such an extent that the tentorium and its related structures were visible. Yet clinical disturbances were trivial. It is not likely that such displacements would be compatible with life. In our cases the brain was not displaced or compressed and the gyral pattern was normal. Our impression was that the temporal pole was absent.

Cunningham has described the development of the brain in man. The temporal lobe is formed from a caudal bulge of the hemisphere that grows in a downwards and forwards direction. At first there is a sylvian depression between the temporal lobe and the rest of the hemisphere and this deepens to become the sylvian fissure. At about 6 months the sulci and insula appear. In the next 3 months the insula is covered by the opercula growing over from the frontal, frontoparietal and temporal regions. The insula is opercularised by birth.

If it is postulated that this condition is caused by failure of the temporal lobe to come forwards in its development then the temporal operculum will be absent and the lower part of the insula uncovered. This was the state of affairs in Cases B, 6 and 9 and probably in Case 7. Air studies showed the lack of forward position of the temporal horn of the lateral ventricle. The ease with which the structures on the medial side of the temporal lobe can be seen, can be best accounted for by a temporal lobe agenesis. A grosser degree of the deformity would be when the frontal pole fails to develop as well.

It is suggested that this condition of a fluid collection in the region usually occupied by the temporal pole of the brain and associated with enlargement of the boundaries of the middle cranial fossa is caused primarily by agenesis of the temporal lobe. It may co-exist with agenesis of the frontal lobe. The fluid is cerebrospinal fluid. It cannot be denied that this agenesis could be determined by a pre-existing cyst at this site. Our evidence points to a subarachnoid location for the fluid but in some cases there seems to have been a thin extra membrane. It is not considered that this condition is related to the arachnoidal cysts that occur elsewhere in the nervous system. These act as progressive space-occupying processes with corresponding clinical effects. With the material under discussion there is some exceedingly slow expansion of the skull that stabilises at adult life but there are no other symptoms in the uncomplicated case.

It is less easy to account for the expansion of the skull. It is well known that most types of agenesis of the brain are associated with a corresponding lack of development of the skull on that side. Intracranial tumours and
hematomas tend to bulge the skull over them in patients under the age of 15 years, but in most of these cases there is the added factor of raised intracranial pressure. Pascal's law states that in a fluid, the pressure applied at any point is transmitted equally in all directions. Yet with these pathological conditions there is some local escape from this law. Connolly has suggested that the digital impressions of the skull in adolescence and in hydrocephalic states result from a closer contact of the brain surface with the inner table of the skull than previously. Close tissue proximity certainly occurs with most hematomas and tumours, and produces, perhaps, what are no more than gigantic digital impressions. Somehow or other the fluid collections in temporal lobe agenesis act similarly although the dynamic factors are far from clear.

DIAGNOSIS AND TREATMENT

Other causes of localised bulging of the skull in childhood and adolescence are skull tumours, chronic subdural hematomas and cerebral neoplasms. The skull tumours can be readily distinguished by their radiological appearances. The chronic subdural hematomas have neurological manifestations that they share with cerebral tumours. The usual radiological appearances of the subdural hematomas are elevation of the lesser wing of the sphenoid, bulging out of the greater wing of the sphenoid and bulging forwards of the middle fossa. Similar changes may occur with a temporal lobe glioma and are present in temporal lobe agenesis. Air studies and electroencephalography may be inconclusive. Carotid arteriography will be helpful. There will be an avascular area between the brain and the skull in cases of hematomas and in many cases of temporal lobe agenesis, but the upwards course of the middle cerebral artery will probably be absent with hematomas. With a temporal lobe neoplasm the middle cerebral artery would be elevated but there would be no avascular space between the brain and the skull. The upwards course of the middle cerebral artery is most noticeable in its first part with the agenesis and greater than with a tumour.

It is suggested that the patient in the first two decades of life who has bulging of the temporal region of the skull that is not associated with any other obvious clinical features, have the skull radiographed and nothing further done. A short period of observation will soon confirm the simple nature of the lesion. There is no evidence that surgical exploration influences the course of the condition. When there are additional clinical signs then full investigation and perhaps a craniotomy are needed.

SUMMARY

Two cases have been presented in which there was bulging of the skull without other physical signs. At operation the temporal pole of the brain was absent and its place was occupied by a large amount of cerebrospinal fluid.

Fifteen similar instances have been found in the literature under the designation of subdural hygromas.
It is suggested that these are cases of temporal lobe agenesis. Agenesis of the frontal lobe may exist as well.

The diagnosis is considered but no treatment is advised.

I am grateful to Dr. A. C. Begg and Dr. N. R. Jefferson for the radiographs, to Dr. E. K. Macleod for the electroencephalograms, and to Miss D. Marshall for the photography.

REFERENCES