EXPANDING LESIONS OF THE TEMPORAL FOSSA

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Brain-stem Compression

Expanding lesions in the temporal fossa readily displace the brain-stem towards the opposite side where it is forced against the edge of the tentorium. At the same time a wedge formed by the uncus and hippocampal gyrus is forced downward between the midbrain and the edge of the tentorium on the side of the lesion (Fig. 1). These two mechanisms produce the various stages of midbrain compression. The downward herniation of the uncus and hippocampal gyrus also compresses the homolateral oculomotor nerve. Rarely the nerve is at first stimulated and therefore the pupil is constricted, but as pressure on the nerve increases paralysis ensues and the pupil becomes dilated. Usually, however, the pupil dilates as soon as the nerve is compressed. If the patient is conscious, ptosis and paralysis of the external ocular muscles supplied by the oculomotor nerve may be observed. The posterior cerebral artery prolapses through the tentorial notch with the herniated brain and occasionally a contralateral homonymous hemianopia is produced by kinking of the artery or its calcarine branch over the free edge of the tentorium.

Increased intracranial pressure is produced by expansion of the lesion itself, pressure on the mid-brain and consequent obstruction of the aqueduct of Sylvius, and often also by associated cerebral oedema. The increasing pressure, if not too rapid, may produce papilloedema. It also causes descent of the brain-stem through the tentorial opening sometimes resulting in sufficient traction on one or both abducens nerves to cause unilateral or bilateral external rectus palsy. If descent of the brain-stem continues, 'coning' of the cerebellar tonsils occurs at the foramen magnum and compression of the medulla oblongata results.

Midbrain compression may be roughly classified into the following stages:

Stage 1. Clouding of consciousness, normal pupils or occasionally contraction of the pupil on

FIG. 1.—Temporal lobe herniation. The groove made by the free edge of the tentorium on the under surface of the left temporal lobe is indicated by arrows. The enlargement of the temporal lobe was caused by a glioblastoma multiforme. (From 'Basic Surgery' by kind permission of the publishers, H. K. Lewis, London.)

the side of the lesion, and contralateral or homolateral hemiparesis with hypertonia, hyperreflexia and a positive Babinski sign. (Homolateral pyramidal signs are caused by displacement of the brain-stem away from the side of the lesion and resulting indentation of the contralateral cerebral peduncle by the edge of the tentorium—the Kernohan-Woltman phenomenon.) Occasionally contralateral or homolateral Parkinsonian tremor and rigidity, presumably due to ischaemia of the substantia nigra or its efferent pathways.

Stage 2. Stupor, dilatation and paralysis of the

pupil on the side of the lesion (Hutchinson's pupil), and contralateral or homolateral hemiparesis, hypertonia, and hyperreflexia with unilateral or bilateral Babinski responses. Occasionally Parkinsonian manifestations in the limbs. Increasing pulse rate (the classical slow pulse, although of considerable diagnostic importance, is found less often than a fast one).

Stage 3. Coma, dilatation and paralysis of the pupil on the side of the lesion or on both sides, decerebrate rigidity with increased tendon jerks and bilateral Babinski responses. Cheyne-Stokes respiration. Tachycardia (if cerebellar coning is causing compression of the medulla oblongata, the pulse may be slow and associated with increased blood pressure and slow respiration).

Stage 4. Absence of response to all external sensory stimuli, dilatation and paralysis of both pupils, flaccidity of all voluntary muscles, absent tendon reflexes, bilateral Babinski responses or absent plantar reflexes. Rapid weak pulse. Shallow slow respirations.

Patients may pass from one stage to another in either direction. Rapidly expanding lesions in the temporal fossa may lead immediately to stages 3 or 4. Thus there are often no lateralizing or localizing signs with acute lesions.

Visual Field Defects

The optic radiation sweeps widely forward in the upper posterior part of the temporal lobe before passing backward to the occipital cortex. Thus lesions within the temporal lobe may invade the optic radiation from below, and as light rays from above strike the lower part of the retina and light rays from one side strike the opposite side of the retina, contralateral homonymous upper quadrantic field defects are produced. This is in contrast with lesions within the parietal lobe, for they invade the optic radiation from above and cause contralateral homonymous lower quadrantic field defects. A lesion arising in either situation may eventually cut across the whole of the optic radiation and produce a complete contralateral homonymous hemianopia. Involvement of the optic radiation usually, though not invariably, indicates a destructive lesion within the cerebrum rather than compression of its surface.

Dysphasia

Nominal dysphasia may occur with lesions of the superior temporal convolution of the dominant hemisphere. Lesions which spread upwards and backwards from the dominant temporal lobe may eventually cause total aphasia by destroying the speech centres of the parietal lobe.

Personality Changes

The temporal cortex is concerned with the reception, directly or indirectly, of association-fibre systems of the other areas of the cerebral cortex, and may therefore be expected to play an important part in the total function of the cerebral cortex. Nevertheless, personality changes are rarely associated with expanding lesions of the temporal fossa, but when they do occur they take the form of anxiety, irritability and occasionally aggressiveness. This is in marked contrast with the apathy often produced by expanding lesions of the anterior fossa.

Temporal Lobe Epilepsy

About a quarter of all cases of epilepsy have their focus of discharge in one or other temporal lobe. Although atrophy of part of the temporal lobe is the most commonly occurring underlying pathological process, tumours account for a significant proportion of cases of temporal lobe epilepsy. Often the discharge is confined to the temporal lobe, giving rise to characteristic phenomena, but it may spread over the whole of the cerebral cortex to produce grand mal attacks.

There are several modes of origin of temporal epilepsy and each reflects one of the functions of the temporal lobe. The commonest is a sensation in the abdomen, chest or throat (visceral aura) and is related to autonomic function. The classical attack as originally described by Hughlings Jackson, begins with a sensation of a bad smell or taste accompanied by smacking of the lips and a 'dreamy state'; the origin of such attacks is in the uncus, hippocampus or amygdala, the location of olfactory and gustatory function. Other patients complain of unpleasant noises or a disturbance of equilibrium, sometimes so severe that the patient is thrown to the ground (gyratory epilepsy); sounds may seem unduly loud or soft; these attacks are related to the auditory and equilibratory functions of the superior temporal convolution. Some patients experience sudden fear, anger or pleasure. There may be a sudden feeling of familiarity with the surroundings, or the patient may feel that what is happening has taken place before (*déjà vu* phenomenon). Sometimes objects seem small and receding (*micropsia*), or large and near (*macropsia*). A vivid recollection of the past with its associated emotions may force its way into consciousness. Patients may carry out complicated acts unrelated to present events but sometimes related to the sensory phenomena and tending to be aggressive (not to be confused with post-epileptic automatism). Depersonalization (a feeling of complete detachment from self), and sometimes the related phenomenon of auto-scopy (seeing an image of oneself in the external

environment), are sometimes experienced. These curious effects are related to the highest functional levels of the temporal lobe.

Neighbourhood Effects

Expansion of temporal lesions upward often causes contralateral facial paresis due to involvement of the facial area of the frontal cortex or its efferents. Expansion upwards and medially causes contralateral facial paresis and hemiparesis by interference with the homolateral pyramidal fibres.

Haematomas

Extradural Haematoma

The temporal fossa is the commonest site for this type of haematoma. The haemorrhage arises from damage to the middle meningeal artery or its branches, severed diploic veins in the line of fracture, or sometimes from a torn transverse or superior petrosal sinus. The haematoma is usually, but not always, associated with a fracture of the temporal bone and there is nearly always an overlying visible scalp lesion.

Brain-stem compression (*vide supra*) rapidly develops, usually within 24 hours of the injury, and in about half the cases there is no improvement in the level of consciousness before the onset of brain-stem compression. In childhood an initial period of coma seldom occurs; more often children are momentarily dazed and later become unconscious from the effects of the expanding haematoma. They sometimes have convulsions. Also, wide separation of the sutures or fracture lines sometimes allows much of the blood to escape under the elastic scalp producing some degree of spontaneous decompression and delaying the onset of compression of the brain. In infants, enough blood may be lost from the circulation in the formation of an extradural haematoma to cause severe anaemia and shock.

The mortality of extradural haematoma is still about 50 per cent., for it is not sufficiently realized that it is one of the most rapidly lethal conditions in surgery. Immediate diagnostic burr-holes are indicated in traumatic coma if (a) the level of consciousness does not rapidly improve, (b) the level of consciousness declines, or (c) any new abnormal neurological signs appear, e.g. hemiparesis, pupillary dilatation.

Subdural Haematoma

There are three varieties of haematoma occurring between the dural and arachnoid membranes, acute, subacute and chronic. An acute subdural haematoma is frequently found in head injuries but it is usually small and associated with severe

brain damage. Sometimes, however, an acute subdural haematoma is large and clinically indistinguishable from an extradural haematoma. Its bulk tends to be in the temporal fossa although it extends well beyond the temporal boundaries and may sometimes envelop the whole hemisphere. Acute subdural haematomas arise from damage to cortical vessels or venous sinuses. They have no capsule (cf. subacute and chronic subdural haematomas).

A subacute subdural haematoma reveals itself a few weeks after a minor head injury although in many cases no history of injury is obtained. This type of haematoma is thought to be caused by tearing of one of the veins which pass from the cerebral cortex to the venous sinuses. The blood tends to gravitate to the temporal region but extends well beyond its limits. A thin capsule forms around the haematoma. In adults subacute haematomas are frequently bilateral; in early childhood they are almost always bilateral. The diagnosis is made when evidence of increased intracranial pressure and perhaps contralateral, homolateral or bilateral pyramidal signs develop a few weeks after a head injury. In the absence of a history of trauma a clinical diagnosis of intracranial tumour is made and the haematoma revealed by subsequent investigations.

A chronic subdural haematoma reveals itself months or even years after a minor head injury although again a history of trauma is often lacking. In most cases the clinical diagnosis is intracranial tumour. The capsule tends to be thick; occasionally the whole of the haematoma becomes replaced by fibrous tissue and the presence of blood pigment may be the only remaining evidence of trauma.

In early childhood, birth trauma is thought to be a common cause of subdural haematoma. In the majority of cases there is no characteristic clinical picture. Thus infants may show nothing more than restlessness and bad temper or there may be attacks of generalized convulsions, the commonest manifestation of the condition in early life. Sometimes vomiting occurs. There is pyrexia in more than half the cases and bulging of the fontanelle in somewhat less than half. The circumference of the head may be 2 to 3 in. more than average. Retinal haemorrhages are quite common although papilloedema is rare owing to the decompressive effect of separation of the sutures of the skull. The tendon reflexes may be exaggerated but paresis of the limbs occurs in only a small proportion of cases. The manifestations in early life are thus vague. Therefore a subacute or chronic subdural haematoma should be one of the conditions considered when a child is not thriving.

Subdural Hygroma or Hydroma

Subdural collections of colourless or yellow fluid of raised protein content may occur with or without a history of head injury. Some follow meningitis, especially that caused by *H. influenzae*. They are frequently bilateral and although the temporal region is usually involved they tend to be widespread. The clinical manifestations are the same as those found with subdural haematomas and long-standing collections are surrounded by a thin capsule.

Intracerebral Haematoma

The temporal lobe is the commonest site for a spontaneous intracerebral haematoma. Such a haematoma may arise from rupture of an intracranial aneurysm or, less often, a vessel forming part of a vascular malformation. In either case there is commonly an associated subarachnoid haemorrhage. Often there is no discoverable cause for the occurrence of a spontaneous intracerebral haematoma and in these cases subarachnoid haemorrhage is exceptional. Rarely an intracerebral haematoma develops in an area of softening caused by a previous head injury (late post-traumatic apoplexy). An expanding haematoma in the temporal lobe usually causes very acute brain-stem compression.

Arachnoid Cyst

The outer wall of an arachnoid cyst is formed by arachnoid and the inner wall by pia. It is an excessively rare type of congenital cyst which is usually found between the frontal and temporal lobes in the Sylvian fissure. It contains clear yellow fluid of greatly raised protein content. As the cyst occupies part of the temporal fossa it tends to cause acute and sometimes fluctuating brain-stem compression. The importance of arachnoid cysts is that their effects can be permanently relieved by excision of the outer arachnoid wall.

Cerebral Abscess

Abscesses occur more frequently in the temporal lobe than elsewhere in the brain owing to the proximity of the middle ear and mastoid process. A temporal lobe abscess is more likely to arise from chronic infection of the middle ear than from the original acute infection. A metastatic abscess, usually from infection in the lungs, may develop anywhere in the brain including the temporal lobe. A cerebral abscess may also be caused by a penetrating head injury. Most cerebral abscesses mature in from one to two weeks and therefore there is usually time for localizing and lateralizing signs to be observed. A large abscess in the temporal lobe may produce a contralateral

homonymous upper quadrantic field defect, contralateral facial paresis sometimes associated with hemiparesis, and, when the abscess is on the dominant side, nominal dysphasia or aphasia may occur. If untreated, the abscess with its accompanying cerebral oedema causes progressive brain-stem compression.

Gliomas

The commonest glioma encountered in the temporal lobe is the glioblastoma multiforme. It is a very malignant tumour which, because of its rapid growth, its tendency to haemorrhagic infarction and the surrounding cerebral oedema, commonly causes acute effects as the result of midbrain compression, and may be mistaken for a stroke.

Meningiomas

Medial sphenoid ridge meningiomas produce a characteristic syndrome. Patients complain of unilateral loss of vision and sometimes unilateral exophthalmos. There is homolateral optic atrophy, diminished visual acuity, and, in the early stages, a central scotoma. There may be papilloedema on the other side. (Optic atrophy with central scotoma on one side and papilloedema on the other is known as the Foster-Kennedy syndrome.) When the optic tract is involved instead of the optic nerve, there is an homonymous hemianopia. Ophthalmoplegia is present in the majority of cases, and results from occlusion of the orbital fissure. The ophthalmic division of the trigeminal nerve is affected in some cases; patients then complain of 'pins and needles' over the forehead where there is diminished or absent sensation. Temporal lobe epilepsy may occur, and is sometimes the presenting symptom. These deeply placed tumours tend to envelop the internal carotid artery and to adhere to the optic nerve.

Middle sphenoid ridge meningiomas usually grow to large dimensions before revealing themselves. Manifestations of raised intracranial pressure may be associated with personality changes produced by involvement of the frontal and temporal lobes. Sometimes the presenting symptom is unilateral exophthalmos (Figs. 2 and 3). Plain radiography may show hyperostosis or erosion of the lesser wing of the sphenoid. Occasionally the hyperostosis is widespread, involving much of the orbit and middle fossa (Fig. 2).

Lateral sphenoid ridge meningiomas produce few or no localizing signs. Those on the dominant side may cause memory defects or nominal dysphasia. Radiography may reveal hyperostosis or erosion of bone. Upward angulation of the

FIG. 2.—Radiograph showing thickening of the roof and lateral wall of the right orbit produced by a meningioma arising from the middle of the lesser wing of the sphenoid and growing downward into the middle fossa and forward into the anterior fossa.

FIG. 3.—Proptosis of the right eye caused by the changes in the wall of the orbit shown in Fig. 2.

middle cerebral artery may be seen in angiograms with meningiomas arising along the sphenoid ridge.

Meningiomas *en plaque* are tumours which lie like a carpet over the dura mater, and are characterized by an increase in thickness of the adjacent bone and lack of neurological effects. The tumour most often grows in relation to the sphenoid bone and runs a chronic course. The presenting symptom is unilateral exophthalmos

which is produced by thickening of the bony walls of the orbit.

Sylvian fissure meningiomas. These tumours are attached to the dura mater overlying the posterior ramus of the Sylvian fissure, and penetrate between the frontal and temporal lobes. Localizing evidence may be found, and includes personality changes, contralateral facial paresis, Jacksonian attacks beginning in the face, and

nominal dysphasia with tumours on the dominant side.

Investigations

Lumbar Puncture

This procedure should not be carried out in the presence of raised intracranial pressure due to an expanding lesion, for it may precipitate or aggravate brain-stem compression, sometimes with a fatal result. Furthermore, lumbar puncture is unlikely to yield worth-while information.

Plain Radiography

Fractures may be confirmed or revealed. In extradural haematoma, a fracture may be shown running across the markings of the middle meningeal vessels, although some extradural haematomas occur without a fracture.

Bulging of the temporal fossa and/or elevation of the lesser wing of the sphenoid may be present with a long standing lesion, e.g. chronic subdural haematoma, chronic subdural hygroma, arachnoid cyst.

If calcified, the pineal gland is usually seen to be displaced away from the side of a space-occupying lesion.

In early childhood, the presence of raised intracranial pressure is revealed by separation of the sutures and exaggerated convolitional markings.

Increased width and tortuosity of vascular channels, occasionally with enlargement of the foramen spinosum, may be seen with meningiomas. Sometimes there are adventitious vascular channels converging towards the site of a meningioma.

Increase in thickness of the lesser and greater wings of the sphenoid, the roof and lateral wall of the orbit, occurs with most meningiomas of the lesser wing of the sphenoid and meningiomas *en plaque* (Fig. 2). Rarefaction or total destruction of the posterior clinoid processes may be caused by long-standing raised intracranial pressure or directly by the growth of adjacent meningiomas. Increased size and density of one posterior clinoid process may be seen with meningiomas of the medial end of the lesser wing of the sphenoid.

Calcification can be demonstrated radiologically in some meningiomas, some astrocytomas and most oligodendrogliomas.

Carotid Angiography

This is the most valuable investigation for the demonstration of space-occupying lesions in the temporal fossa. It is preferable to ventriculography which dangerously increases intracranial pressure and aggravates or produces brain-stem compression. Angiography is seldom indicated to demonstrate the presence of an extradural haematoma, for the march of events is usually too rapid in this condition.

FIG. 4.—Arteriogram showing the branches of the middle cerebral artery folded medially towards the midline by a subdural haematoma. There is also some displacement of the anterior cerebral artery towards the opposite side.

In the A-P projection, subdural space-occupying lesions show medial displacement of the vascular tree on the side of the lesion, with the branches of the middle cerebral artery folded medially towards the main trunk, and there is an avascular space lateral to the vessels (Fig. 4).

Meningiomas of the sphenoid ridge tend to produce localized and sharply angulated upward displacement of the middle cerebral artery and its branches. Meningiomas overlying the posterior ramus of the Sylvian fissure produce the same type of displacement as subdural lesions, but in some cases the tumour gives rise to a visible vascular pattern.

Space-occupying lesions within the temporal lobe produce a characteristic upward curve of the whole of the middle cerebral artery and its branches, best seen in lateral projections (Fig. 5).

Ventriculography

This investigation is avoided whenever possible in the diagnosis of expanding lesions in the temporal fossa for the reasons already given, but may be required when lateralizing signs are not obtained or if angiography is unsatisfactory. A-P views show displacement of the ventricles towards the opposite side with angulation of the

FIG. 5.—Arteriogram showing the characteristic unfolding of the carotid 'syphon' and the upward curving of the middle cerebral artery produced by temporal tumours (in this case a glioblastoma multiforme).

third ventricle on the septum lucidum (Fig. 6). Lateral views taken after manoeuvres to fill the temporal horns may demonstrate absence of filling, deformity or displacement of the temporal horn.

Electroencephalography

Because of the rapidity with which brain-stem compression is produced by some expanding lesions of the temporal fossa (e.g. extradural haematoma, intracerebral haematoma, some gliomas), there may not be time for this investigation to be done. Subdural haematomas cause electrical 'silence' or waves of diminished potential over the compressed hemisphere. Meningiomas or intracerebral lesions change the normal alpha rhythm (average 10 cycles per sec.) to delta rhythm (average 2 cycles per sec.) in the vicinity of the lesion. The larger the amplitude of the waves, the larger is the mass of abnormally functioning brain; and the slower the rate of the waves the more acute is the lesion. Thus an intracerebral haematoma or abscess produces the most striking changes, the average glioma moderate changes, and slowly growing meningiomas minimal changes.

FIG. 6.—Drawing of ventriculograms (A-P views) showing the normal relationship of the third ventricle to the septum lucidum on the left and the characteristic angulation of these structures produced by tumours of the temporal lobe. (From 'Basic Surgery' by kind permission of the publishers, H. K. Lewis, London.)

Diagnostic Burr-holes

When localization has been obtained by clinical examination and appropriate radiological investigation, a burr-hole is made over the lesion. The

diagnosis of the various haematomas can be confirmed or made by this means; pus from an abscess can be aspirated and an X-ray contrast medium injected; and small pieces of tumour can be obtained for histological examination.

Treatment

Extradural Haematoma

After the diagnosis has been established by means of a burr-hole, a temporal scalp flap is elevated and the burr-hole is rapidly enlarged into a temporal craniectomy with the aid of a double-action rongeur. The haematoma is quickly evacuated and the bleeding, usually from the middle meningeal artery or its branches, is arrested by diathermy coagulation. When oozing of blood occurs from the dura, it may be necessary to drain the extradural space for 24 hours through the posterior extremity of the scalp incision.

Subdural Haematoma

When the blood is in the fluid state, a rubber catheter is passed through the diagnostic burr-hole and incised dura mater, and the blood is washed out with isotonic saline. When large clots are present, a bone flap must be elevated and a flap of dura reflected to ensure adequate evacuation of the haematoma. In children, as in adults, the haematoma is if possible washed out through a catheter, but it is always necessary to remove the capsule at a later date if impaired development of the affected hemispheres is to be avoided.

Subdural Hygroma

A large proportion of the fluid escapes with a sudden gush when an opening is made in the dura mater (and capsule when present). The remainder is aspirated through a rubber catheter. In children, if a capsule is present it must be removed on another occasion by craniotomy.

Intracerebral Haematoma

Although it is often possible to relieve brain-stem compression by aspiration of the fluid part of the haematoma through the diagnostic burr-hole, it is usual to elevate a bone flap and evacuate the clots. Sometimes a bleeding vessel or aneurysm is discovered in the wall of the haematoma cavity and can be occluded.

Arachnoid Cyst

The cyst is revealed when a diagnostic burr-hole is made and the dura mater incised. Most of the yellow fluid escapes when the outer wall is incised. A bone flap is elevated and the arachnoid forming the outer wall of the cyst is removed. The inner wall of the cyst, formed by the pia covering the adjacent part of the cerebrum, is left undisturbed.

Cerebral Abscess

Appropriate antibiotics are administered. The

pus is aspirated through a brain cannula passed through the diagnostic burr-hole. This may have to be repeated several times. After aspiration a small volume of a solution of penicillin is injected into the abscess cavity. If this method of treatment fails, a bone flap is elevated and the capsule of the abscess is removed.

Glioblastoma Multiforme

Many neurosurgeons do not operate on this tumour, if the diagnosis has been proven by biopsy, for recurrence quickly occurs with or without post-operative radiotherapy. Partial removal, however, may be worth while for the relief of severe headache or to allow patients to put their affairs in order.

Meningiomas

These tumours are removed under induced hypotension to minimize bleeding. The tumour is incised with a diathermy electrode and the bulk of its interior removed by means of a pituitary rongeur or diathermy loop. The capsule of the tumour can then be retracted from the brain which is thereby spared from injury during removal of the capsule. Removal of medial ridge meningiomas may be dangerous because of their close relationship to the internal carotid and middle cerebral arteries which are sometimes enveloped by tumour. After removal of a sphenoid ridge meningioma, its site of origin should be well coagulated by diathermy to reduce the risk of recurrence. For the same reason, the dural attachment of a Sylvian fissure meningioma must be completely excised. Meningiomas *en plaque* extend like a carpet over the temporal fossa; as their attachment is so extensive and as these tumours do not cause raised intracranial pressure, no attempt is made to remove them. Moreover, removal would not relieve the exophthalmos which is the presenting symptom.

Brain-stem Compression

The early recognition and relief of midbrain compression are often of great urgency if irreversible damage or death is to be avoided. The only effective decompression is that obtained by appropriate treatment of the causal lesion. There is no place for the classical subtemporal decompression in which the bone of the lateral wall of the temporal fossa is removed and the dura mater widely opened.

Sometimes when the lesion has been correctly treated, midbrain compression persists. It is then necessary to re-open the wound and 'reduce' the herniation of the uncus and hippocampal gyrus. During this procedure, care is taken not to damage the posterior cerebral artery which prolapses with the herniated brain.