A polymorphonuclear leucocytosis in the blood is usually found in cases of cerebral abscess, but this, too, is apt to be variable—sometimes slight in cases in which one might expect it to be more pronounced, depending upon factors such as the rate of increase of the abscess in size and its spread, its degree of encapsulation, and the nature of the infecting organisms.

A note may be added here as to certain findings not usually recorded in “official” descriptions of examinations of the cerebro-spinal fluid. In the centrifuged deposits of lumbar-puncture specimens, I have not infrequently encountered squamous cells from the skin-surface, together with their accompanying staphylococci, etc., and occasionally even a little cylindrical fragment of skin punched out by the exploring needle—an argument in favour (1) of the *thorough* preliminary sterilisation of the skin, e.g. with 1 in 500 biniodide spirit, and not a mere rapid and perfunctory “dab” with spirit or ether, or even tincture of iodine: and (2) of rejecting the first few drops of fluid flowing from the needle, as these usually also contain contaminating red blood-corpuscles.

On one occasion several years ago, whilst examining a specimen of fluid, I was puzzled to find, in the preliminary fresh-wet films from the centrifuged deposit, certain “unusual” cells, until, in toluidin-blue and Leishman-stained films, I recognised them as myelocytes, accompanied by nucleated red corpuscles from the bone-marrow, due evidently to the over-enthusiastic penetration of one of the vertebral bodies (Fig. 6A).

In a similar “over-energetic” specimen I once found a bone-corpuscle along with myelocytes and erythroblasts (Fig. 6B); and, as a complementary experimental investigation, at my next autopsy, I passed a lumbar-puncture needle, attached to a syringe, through the fourth lumbar interspace, pushing it *well forward* (!) into the intervertebral disc, and then slowly withdrawing it under negative pressure, collecting some cerebro-spinal fluid on its backward journey—with the interesting result of finding several little groups of cartilage-cells in the centrifuged deposit of the specimen so obtained (Fig. 6C).

THE ANATOMY OF THE MENINGES

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The meninges constitute the coverings of the brain and spinal cord, and, unlike the central nervous system itself, they are mesodermal in origin. They are usually described in three layers, of which the outer or dura mater is mainly protective in function and relatively avascular, whereas the inner two layers, comprising the leptomeninges or pia-arachnoid, are mainly nutritive in function and are highly vascular. It is the leptomeninges which are the site of generalised meningitis or leptomeningitis.

Pachymeninx or Dura Mater.

This is a strong fibrous sheet which lines the skull and vertebral canal, in which it extends down to the second sacral vertebra.

Embryologically it arises as two layers which fuse in the vertebral canal and become closely united in the cranial region. The layers are only distinct at the sutures and where they separate to form the venous sinuses.

The dura mater is continuous with the orbital periosteum and, through the sutures, with the pericranium. It also accompanies most of the cranial nerves and all the spinal nerves a short distance, fusing with their respective sheaths. Inside the skull the dura forms the periosteum, encloses the intracranial venous sinuses between its layers, and gives rise to four septa:—

(1) One of these, the *falx cerebri*, separates the cerebral hemispheres.

(2) The *tentorium cerebelli* intervenes between the cerebellum and the occipital lobes of the cerebrum, and is supported in the midline by its attachment to the falx cerebri. From the under surface of the tentorium (3) the *falx cerebelli* descends to separate the cerebellar hemispheres.

(4) The *diaphragma sellae*, the smallest of the septa, forms a roof over the pituitary fossa. The side walls of the pituitary fossa are also composed of dura mater and are formed on either side by the anterior ends of the lateral attachments of the tentorium cerebelli. The tentorium resembles a crescent, and together with the basi-sphenoid surrounds the brain stem, which lies in the central opening or *incisura tentorii*. The cerebro-spinal fluid, in its normal and necessary passage from the posterior fossa to the middle fossa, must flow through the incisura.

At the lines of junction of these septa with each other and with the skull lie the main *venous sinuses*.

The *superior longitudinal* or *sagittal sinus* lies in the line of attachment of the falx cerebri to the skull and extends from the cristi galli backwards to the internal occipital protuberance. Here it is joined by the *straight sinus* which lies in the midline of the tentorium along its junction with the falx cerebri. At this point both the superior longitudinal sinus and the straight sinus drain into the *right* and *left transverse (lateral) sinuses* which lie in the line of attachment of the tentorium to the skull; the flow is from the internal occipital protuberance laterally, forwards and eventually downwards to the jugular foramen on each side.

The superior longitudinal sinus receives the superior cerebral veins which enter it obliquely in a forward direction (against the stream). This unusual mode of entry reduces transmission to the cortical veins of the variations in intra-thoracic pressure associated with normal respiration. The superior cerebral veins are, however, very susceptible to a persistent rise in pressure in the superior longitudinal sinus; thrombo-phlebitis of this sinus gives rise to a syndrome of spastic diplegia of the lower limbs involving the arms only to a slight extent, and the face not

FIG. 8.—A diagrammatic representation of the region of the vertex in coronal section (see text) (from Berthe and Dickson's *Textbook of Pathology*).

at all. This is because the remainder of the motor cortex is drained inferiorly, chiefly by the superficial sylvian vein to the cavernous sinus, and the deep sylvian vein towards the great vein of Galen. Venous engorgement of the scalp in this condition, and, in infants, the nasal veins also, illustrates the extent of the extra-cranial venous drainage into the superior longitudinal sinus.

In the free lower margin of the falx cerebri runs the *inferior longitudinal* or *sagittal sinus* and where the anterior part of the base of the falx cerebri joins the anterior free margin of the tentorium, the inferior longitudinal sinus is joined by the *great vein of Galen* which drains the choroid plexuses and the interior of the brain. Together they join to form the straight sinus which lies in the roof of the tentorium, and, as already stated, joins the superior longitudinal sinus at the internal occipital protuberance to drain into the transverse (lateral) sinuses. This confluence of sinuses, termed the torcular Herophili, is subject to slight individual variations, possibly associated with an inequality of the cerebral hemispheres. It has been suggested that this, in turn, may be associated with right or left handedness.

The chief remaining venous sinuses of the dura mater are the *cavernous sinuses*, one on each side of the body of the sphenoidal bone (and the pituitary fossa). Each cavernous sinus receives ophthalmic veins, communicates with its fellow of the opposite side and drains into the *inferior* and *superior petrosal sinuses*. These in turn drain into the jugular bulb and the lateral sinus respectively. Thrombo-phlebitis of the cavernous sinus is usually secondary to cellulitis of the face or orbit, or to infection of the para-nasal sinuses, just as infection of the middle or inner ear may spread to involve the transverse (lateral) sinus.

In general, as the dural tissue is relatively avascular, infections do not usually involve this layer or extend through it. Nevertheless it may fail as a barrier, and infection spread from an extra-cranial septic focus to the leptomeninges, giving rise to meningitis. This may occur via the "emissary" veins, by direct extension through bone (osteomyelitis, periostitis, Gradenigo's syndrome), or via the sheaths and foramina of issuing nerves. The dura may also be involved in granulation tissue due to tuberculosis or syphilis, especially in the spinal cord. In the former condition, tuberculous meningitis is a possible sequel.

The Leptomeninges or Pia Arachnoid.

These are derived from the primitive mesenchyme at the head end of the embryo and arise as a single sheet. Later, at about the time when the cerebro-spinal fluid is first produced by the choroid plexus, the pia arachnoid splits into two layers between which the fluid accumulates (Weed).

The external layer or *arachnoid mater* closely lines the dura mater, the subdural space being negligible, and the inner layer or *pia mater* follows the brain surface closely, dipping into sulci, and accompanies the vessels into the substance of the brain. Fine cobweblike trabeculations traverse the subarachnoid space between the pia and arachnoid. In the deeper layers of the cortex the subarachnoid space becomes continuous with the perivascular spaces of Robin and Virchow, from which, under pathological conditions, infective and necrotic material finds its way into the cerebrospinal fluid. The perivascular spaces communicate in turn with the perineuronal spaces around the nerve cells.

As the contours of the brain are irregular the subarachnoid space is wider in some places than in others, and contains larger amounts of cerebro-spinal fluid; these places are known as *cisterns*. The most important are the *cisterna interpeduncularis* between the cerebral peduncles, and the main site of basal meningitis; the *cisterna chiasmatis*, where chronic inflammation ("arachnoiditis") can give rise to optic atrophy; and the *cisterna magna* between the cerebellum and the medulla, from which cerebro-spinal fluid can be obtained, and into which dyes and opaque media can be injected by "cisternal puncture."

Histologically, the pia arachnoid is composed of fine elastic and fibrous connective tissue with a single layer of flattened polygonal mesothelium. With irritants these arachnoidal cells become rounded into free macrophages, and they represent the fixed cells of the reticulo-endothelial system in the pia arachnoid. These cells are markedly stimulated by malarial infection as in the treatment of general paresis.

The spinal pia mater is tougher and less vascular than the cerebral pia mater, and gives rise to a septum on either side known as the *ligamentum denticulatum* because of its intersegmental attachments to the dura. These septa incompletely divide the spinal subarachnoid space into anterior and posterior compartments, the relative sizes of which depend on the position of the

spinal cord. This varies considerably at different levels, and the posterior subarachnoid space is virtually absent in most subjects above the level of the eleventh thoracic vertebra where the cord is in close apposition to the dura (Worster-Drought). The space may be correspondingly shallow below this point, making the withdrawal of cerebro-spinal fluid virtually impossible above the safe level without passing the needle through the spinal cord.

The spinal cord terminates just above the second lumbar vertebra in the adult, but in infants may extend as far as the third lumbar vertebra. A line joining the highest points of the iliac crests usually passes between the third and fourth lumbar spinous processes, with the spine in the fully flexed position. From the termination of the spinal cord the pia mater extends downwards as the *filum terminale* through the large *lumbar subarachnoid space* in company with the nerve roots known as the *cauda equina* to fuse with the dura at the level of the second sacral vertebra, and join, together with the dura, an attachment further down to the posterior aspect of the first segment of the coccyx.

The sacral canal, below the termination of the subarachnoid space, forms the greater part of what is termed the *epidural space*, which extends upwards outside the dura forming a potential space around the issuing nerve roots after they have passed through the dura.

The Cerebro-spinal Fluid.

It was known by Galen that a colourless fluid filled the ventricles, but only recently have we had ample proof, mainly due to the researches of Weed, that this fluid is produced by the choroid plexuses: vascular tufts of pial blood vessels projecting into all the ventricles. Intravenous fluorescein injections have shown that a small amount also arises from the superficial pial blood vessels. And pathological changes in disease indicate that products of degeneration and reaction (protein and cells) within the brain find their way into the cerebrospinal fluid, probably via the perivascular spaces of Robin and Virchow.

The fluid produced in the ventricles escapes through the foramina in the roof of the fourth ventricle to enter the subarachnoid space in the cisterna magna. From the subarachnoid space it is reabsorbed into the blood stream mainly through the *arachnoid villi*: projections of the pia arachnoid through the dura into the dural venous sinuses (Fig. 8). It is probable that as much as four-fifths of it is absorbed into the superior longitudinal sinus where most of the arachnoid villi are situated. Some of them are very large and are termed *pacchionian granulations*. They project not only into the superior longitudinal sinus itself, but also into irregular extensions of the sinus on either side known as *lacunae* (Fig. 8). The pressure relations and rate of flow are such that in life these lacunae contain not blood but cerebro-spinal fluid. Another route of absorption of much less importance is into the lymphatic system via the perineural lymphatics.

The cerebro-spinal fluid is almost a pure dialysate, containing no protein when first formed. The consequence is that it is markedly lacking in natural and acquired anti-bodies present in the blood stream, and has to be provided with these by the reticulo-endothelial cells of the pia arachnoid. This may be part of the explanation for the rapid spread of infection once organisms find their way into the cerebro-spinal fluid. (p. 72)

The *pressure* of the cerebro-spinal fluid is intermediate between that of the arteries and of the veins, and is a measure of the balance between production and absorption. A general increase, as measured by a lumbar puncture manometer, may be due to:—

(1) *Increased production.*

(a) Blood hypotonic. This occurs in many acute infections, and is a possible cause of "meningism" (Fremont-Smith).

(b) Increased intra-cranial venous pressure. Obstruction of the vein of Galen alone is probably not enough. Section of the vein in dogs does not produce any increase in pressure as there is a good collateral circulation (Dandy).

(2) *Defective absorption.*

(a) The factors already mentioned as increasing production also diminish absorption.

(b) Obstruction of arachnoid villi by inflammatory exudate and inflammatory reaction within them.

Apart from the generalised increase of cerebrospinal fluid pressure described above, and commonly present in meningitis, and an extreme example found in "otitic hydrocephalus, there are two other types of hydrocephalus which may occur as a result of meningitis:—

(1) *Internal hydrocephalus.*

The foramina in the roof of the fourth ventricle (see Fig. 7) may become blocked by adhesions, and the pressure rise being inside the ventricles cannot be measured by a spinal manometer. Theoretically there may be an obstruction of the narrow sylvian aqueduct, due to ependymitis. This certainly occurs apart from evident meningitis, and results in dilatation of the third and lateral ventricles only.

(2) *Communicating hydrocephalus.*

The ventricles communicate with the cisterna magna and the spinal theca, but there is an obstruction to the upward flow through the opening in the tentorium (incisura tentorii). As the greater part of normal absorption is above the tentorium a marked increase in pressure results, both in the ventricles and in the spinal subarachnoid space. Some cases of "idiopathic" hydrocephalus in children are probably of this type.

Lumbar Puncture.

The actual technique of lumbar puncture is too well known to need description here. The importance of ascertaining both the pressure and the composition of the cerebro-spinal fluid in every case suspected of meningitis or showing signs of meningism will be clear from the articles which follow. Whilst this is being taken, the patient's head should be kept extended—not flexed on the chest; breathing should be quiet and regular; there should be nothing tight round the neck (a tight pyjama collar, or even a necklace, will in a strained position, run the pressure up appreciably); and there should be no undue abdominal compression by the patient's knees.

SUPPURATIVE MENINGITIS

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The term suppurative meningitis indicates that we are basing our classification on a particular type of meningeal reaction, namely, that in which the cerebro-spinal fluid shows a pleocytosis, wholly or predominantly polymorphonuclear, and to the naked eye is turbid or frankly purulent. This type of reaction is the result of infection with pyogenic bacteria. We are therefore dealing with a number of separate diseases, but as they are similar in many essential particulars it is both practical and convenient to class them together into a group, excluding, however, meningococcal meningitis, which it is usual to consider separately.

The bacteria most usually found are streptococci, staphylococci, pneumococci and, in children, B. "influenzae" (so-called). Many others are encountered occasionally, including gonococci, B. typhosus, B. paratyphosus, B. coli, pneumobacilli and anthrax.

Suppurative meningitis as thus defined is usually secondary to some focus of infection elsewhere, but this is not invariable. Pneumococcal and so-called "influenzal" (*Hæmophilus*) cases, and occasionally others may be primary.

Source of infection.**I. *Suppurative otitis media.***

Something like 50 per cent of cases are secondary to suppurative otitis media, acute or chronic. Otologists have studied in great detail the exact paths of invasion. We need not here concern ourselves with the minutiae of this process; a broad outline will suffice. The spread may occur directly by the erosion of bone, or along vascular, usually venous channels by septic thrombosis. The direction of spread may be backward, *via* the mastoid antrum, or inward, causing a labyrinthitis, and spreading thence *via* the internal auditory meatus, or the aqueduct of the cochlea to the posterior fossa, or upward through the roof of the tympanum to the middle fossa. The process of spread, however, is not always as immediate and direct as this. An