OUR knowledge of intracranial hypertension has been greatly increased in the last few years both by the application of instruments of precision to its diagnosis and by attempts to clear up certain debated points in its etiology. Owing to the general use of a manometer in lumbar punctures we have a fairly accurate knowledge of the normal pressure of the cerebrospinal fluid within the cranium and, in consequence, can say with some degree of certainty when the intracranial pressure is increased. The border line between normal and increased pressure is put by most observers at 200 mm. of water in the horizontal position. It has been observed, for example, that cases of cerebral tumour begin to show evidence of increased intracranial pressure soon after this level is passed. It is true that the diagnosis of cerebral tumour may be made in the absence of any increase in cerebrospinal fluid pressure when evidences of a focal lesion make their appearance in a progressive manner. But such cases present none of the three cardinal signs of cerebral tumour, viz.: headache, vomiting or papilloedema. The degree of pressure at which these signs appear cannot be stated with certainty as other factors than pressure play some part in them, but when the pressure is below 250 mm. it is unusual to find papilloedema, and headache and vomiting are not usually prominent symptoms. Nor are pressures of this order dangerous to life. In fact the pressure must probably go beyond 300 mm. before it threatens life directly.

Another instrument which is probably of considerable value in estimating intracranial pressure in a more indirect manner is Bailliart's\(^1\) ophthalmodynamometer. With it a comparison is made of the pressure in the arteries of the retina and in the brachial artery, and an indication is thereby obtained of the pressure in the smaller arteries inside the cranium. This instrument, although less accurate than the manometric lumbar puncture needle, is free from the risks attendant on tapping the subarachnoid space in cases of intracranial hypertension. There is no doubt that the use of the manometer considerably reduces the risk of lumbar puncture in such cases, but it is well known that sudden alterations of intracranial pressure in cases of cerebral tumour are extremely

\(^1\) *Annales d'ocul.*, 1922, clix, 785.
dangerous. The ophthalmodynamometer is only useful before
the onset of papilloedema, for after this has made its appearance
the readings obtained become fallacious. It is, therefore, of
little use in distinguishing papilloedema from papillitis.

From a combined use of the manometric lumbar puncture
needle and the ophthalmodynamometer we may look forward
to the solution of certain vexed problems. For example, (1)
What is the relationship between hyperpiesis and intracranial
hypertension? and (2) what is the ratio in normal and patho-
logical cases between the arterial pressure inside the skull
and cerebrospinal fluid pressure? Does reduction of pressure
round a leaking aneurysm by lumbar puncture or craniotomy
increase the leakage, or does it rather reduce the pressure within
the bleeding artery, and so favour hæmostasis? These are
questions to which a clear answer is urgently needed.

The etiology of intracranial hypertension appears at first
to be self-evident; it is clearly due to compression of the contents
of the skull by the growth of a tumour, or by an increase in the
amount of cerebrospinal fluid within it. But a glance below
the surface shows us that the problem is less simple than that.
For since blood is constantly passing in and out of the cranium
the increase of pressure must be largely dependent on the blood.
It has also been shown that the intracranial tension found in
cases of cerebral tumour depends more on the amount of
cerebrospinal fluid than on the size of the tumour. It is well
known that a small tumour may raise intracranial pressure
much more than a large one if it is so situated as to cause
hydrocephalus, but it is perhaps less generally realised that
practically every case of cerebral tumour is associated with
some degree of hydrocephalus. The first problem that arises,
therefore, is: Why does cerebrospinal fluid continue to be
formed when its formation is harmful to the balance of physical
forces within the cranium? If, as is now generally held, the
cerebrospinal fluid is dialysed from the blood capillaries as
through a semi-permeable membrane, why does the increase of
pressure of the fluid side not abolish the formation of fluid from
the blood? The answer appears to be that, unless grave
anæmia is to ensue, the capillary pressure must be kept above
the general intracranial pressure. Consequently the formation
of cerebrospinal fluid must continue so long as the patient is
alive. Other factors contribute to the vicious circle thus begun.
An analysis of these has recently been made by Stopford, who

^ Brain, 1928, ii, 485.
has emphasised the importance of one, viz., that the great vein of Galen may be kinked by any unilateral or general raising of the tentorium cerebelli. When a tumour grows in the posterior half of the cerebral hemispheres it will push the posterior end of the falx to one side and thus tug the tentorium upwards, whereas a tumour in the posterior cranial fossa will push the tentorium up from below. A kink is thus formed in the vein near its entry into the straight sinus and the obstruction thus caused leads to an increased formation of cerebrospinal fluid by that choroid plexus which is drained by the affected vein. Russell Brain\(^1\) found that tumours of the frontal lobes were not so likely to cause symptoms of increased intracranial pressure as those elsewhere, and Stopford accepts this as in favour of his hypothesis.

But an increased formation of cerebrospinal fluid would of itself matter little if the fluid could be absorbed as rapidly as it was produced. Many factors combine to prevent this. The tumour itself may obstruct the foramen of Munro or the third or fourth ventricles or the iter of Sylvius, and so prevent the escape of fluid from the ventricles. This occurs in tumours of the thalamus or brain stem. But a much more common event is that by its bulk the tumour, wherever it lies, reduces the space between the brain and the skull. Convolutions are flattened and sulci obliterated and the fluid is thus unable to pass to the large venous sinuses over the vertex where it is normally absorbed. Another vicious circle now comes in to increase this obstruction. In Dandy's well-known experiment it was proved that unless the fluid can flow freely round the sides of the midbrain through the incisura in the tentorium cerebelli, hydrocephalus rapidly appears. Now it is a commonplace in tumours of the cerebral hemispheres to find that the inner part of the hippocampal gyrus is herniated through this incisura, and this herniation must obstruct the upward flow of fluid past the midbrain.

A similar vicious circle often occurs also in hydrocephalus, for it is known that in the majority of cases there is a free communication between the ventricles and the lumbar subarachnoid space. When this is so the obstruction to the absorption of fluid must lie either at the level of the tentorium cerebelli or above it, and is probably chiefly due, in post-meningitic cases, to infiltration in the region of the cisterna basalis, aided by the obliteration of sulci which results from the ventricular distension.

\(^1\) Brain, 1925, xlviii, 105.
Intracranial hypertension may result from injuries to the skull in several different ways. Injuries to the middle meningeal artery, or perhaps more often to the middle meningeal vein, may cause epidural hæmorrhage, coming on rapidly or more slowly according as the artery or the vein is damaged. Still more insidious is the compression which results from laceration of the vertical veins as they pass across the subdural space from the cerebral cortex to the superior longitudinal sinus. The pressure in these veins is probably only very slightly above the general intracranial pressure and consequently bleeding from them into the subdural space probably takes place very slowly and spasmodically. In fact this form of hæmorrhage is rarely seen in healthy young subjects. It is as often bilateral as unilateral, especially when it is caused by a blow on the forehead or the back of the head.

Simple concussion, apart from either fracture of the skull or hæmorrhage, is also always associated with a rise in intracranial pressure, and, as was recently emphasized by Armour,¹ this hypertension may persist for weeks and months and be associated with headaches and dizziness. This condition has often been falsely called 'post-concussion neurosis.' Foerster² and Wartenberg³ have showed by encephalography that in it there is constantly a dilatation of the lateral ventricle or displacement of the ventricle towards the injured side. Penfield,⁴ by injecting air into the lumbar canal and by so arranging the patient's position that the air reached the cortical subarachnoid space rather than the ventricles, obtained pictures which suggested local cortical atrophy. All these observers have found that replacing cerebrospinal fluid by air has a remarkable curative effect on the symptoms. Penfield suggests as a possible rationale for this, that arachnoidal adhesions over the injured cortex may be broken down by the movement of the air bubbles. But the curative effects which have often been obtained either by lumbar puncture alone or by local trephining indicate that the symptoms are due rather to some alteration in the circulation and absorption of cerebrospinal fluid. It is probable that some vicious circle of the kind already described is broken by these relatively simple procedures. It is indeed one of the conditions in which empiricism has for the moment outrun scientific knowledge.

Editorial: INTRACRANIAL HYPERTENSION.

J Neurol Psychopathol 1929 s1-9: 347-350
doi: 10.1136/jnnp.s1-9.36.347

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