THE PRINCIPLES OF PHYSIOLOGY INVOLVED IN THE MANAGEMENT OF INCREASED INTRACRANIAL PRESSURE

WILDER PENFIELD, M.D.

MONTREAL, CANADA

FROM THE DEPARTMENT OF NEUROLOGY AND NEUROSURGERY, MCGILL UNIVERSITY AND MONTREAL NEUROLOGICAL INSTITUTE, MONTREAL, CANADA

EVERY surgeon must think about his problems physiologically but it has been difficult for me on invitation to precipitate out of the medium of my own clinical thinking principles of physiology which are appropriate for publication. Exchange of ideas is so free among neurosurgeons that I can no longer recognize my own contributions and must therefore lay little or no claim to originality for the statements which follow.

The circulation of the cerebrospinal fluid which Cushing has aptly termed the third circulation may be obstructed at any point in its course from the ventricles where the fluid is formed to the fissure of Sylvius and subarachnoid spaces where it is absorbed. Such obstruction results in ventricular dilatation due to increase of ventricular pressure from the maximum normal of 100 Mm. of water to between 400 and 700 Mm. From a practical point of view it is my opinion that there is no such thing as a hypersecretive hydrocephalus, nor have I ever been able to recognize a hydrocephalus due to obliteration of the vein of Galen.

The result of blockage of the cerebrospinal fluid pathway is a ventricular dilatation which follows a certain pattern. The dilatation begins far proximal to the site of the block and works downward toward it. Thus blockage of the interpeduncular cistern at the base of the brain (Fig. 1, A) causes dilatation of the lateral and third ventricles initially and later on the aqueduct of Sylvius, the fourth ventricle, cisterna magna and finally the pontine cisternae more or less in that sequence.*

When the dura is left open over an obstructed ventricle that ventricle enlarges with no decrease in the intraventricular pressure and the intervening portion of brain is destroyed with surprising rapidity. This destruction occurs whether the decompression be placed under the temporal or occipital muscle or elsewhere.

Thus after a negative exploration has been carried out in the presence of ventricular dilatation it is wiser to close the dura tightly unless it can rea-

* It should be pointed out that an occasional exceptional case is encountered in which the cerebrospinal fluid spaces are closed and the ventricles progressively enlarge without the measured intraventricular pressure rising above 150 to 200 Mm. of water. Such cases lead one to wonder what may be the effect upon the cerebral hemispheres of adequate closure of the subarachnoid space. Is the supposed circulation of fluid between perivascular space and subarachnoid space essential to normal metabolism of the cerebral tissue? Is it not possible that closure of that space may have a directly destructive effect upon the hemispheres?

sonably be hoped that the decompression may give to an underlying tumor enough added "elbow room" to free the cerebrospinal channels at the point of compression or unless it is desired to thin out the brain in a certain area so as to prepare the field for a second stage tumor removal.

In the case of a suboccipital craniotomy, if the tumor is found to extend so far forward that the surgeon cannot remove it completely the blockage of the basal cisternae is not relieved. Under such circumstances the result of leaving the dura open over the cerebellum would be the rapid destruction

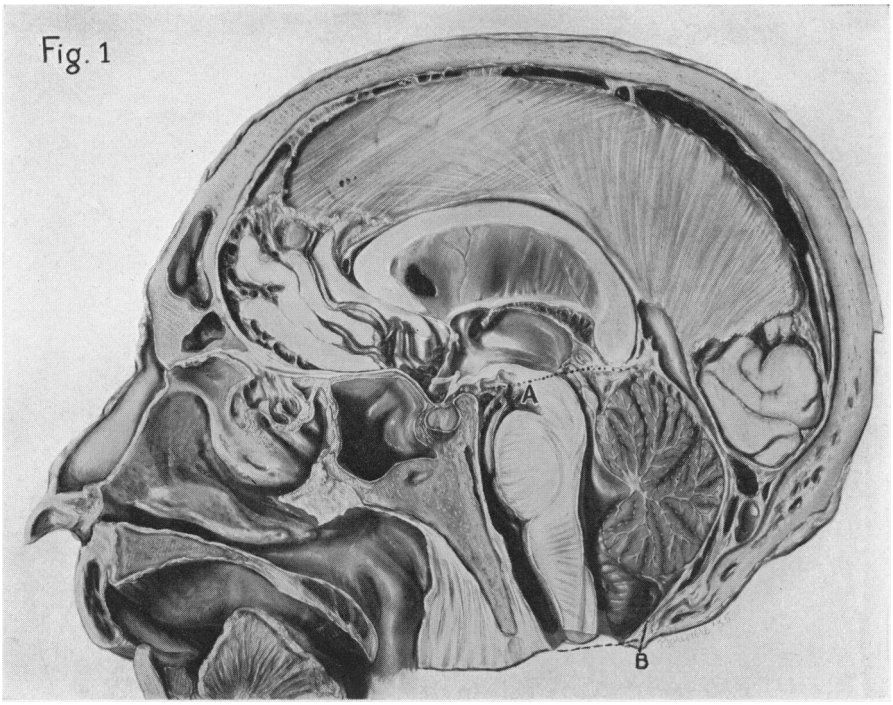

FIG. 1.—Sagittal section of skull. (A) Interpeduncular cistern. (B) Cisterna magna. Dotted line—Outlet for supratentorial structures. Broken line—Outlet of posterior fossa.

of the cerebellum with little or no relief of the hydrocephalus. True decompression and relief of the block could then be secured only by splitting the tentorium from its free margin outward (Fig. 2, broken line). This maneuver which has been recommended by Naffziger may well relieve both the aqueduct of Sylvius and basal cisternae so that the fluid may once again circulate freely.

Ventricular drainage into the tissues anywhere in the body results in absorption of fluid for a short time only. There forms rapidly an endothelium-like covering which seems to be an effective barrier to further fluid absorption. Such short lived absorption takes place into the scalp after many operations as evidenced by frequent postoperative pitting edema of the scalp and swelling about the eyes. Repeated puncture of the ventricles relieves the pressure temporarily but is followed in many instances by a higher rise

in the pressure as though the fluid formation had been increased by the temporary drop in the pressure of the fluid that bathes the choroid plexus.

The effect of expanding lesions above the tentorium demonstrates the fact that the brain is not a fluid structure and pressure is not conducted intracerebrally according to the laws which obtain in a closed fluid-filled space. An expanding lesion in one hemisphere produces a generalized increase of intracranial pressure, it is true, but the falx and tentorium offer enough resistance so that taken with the brain's elasticity the intrahemispheral pres-

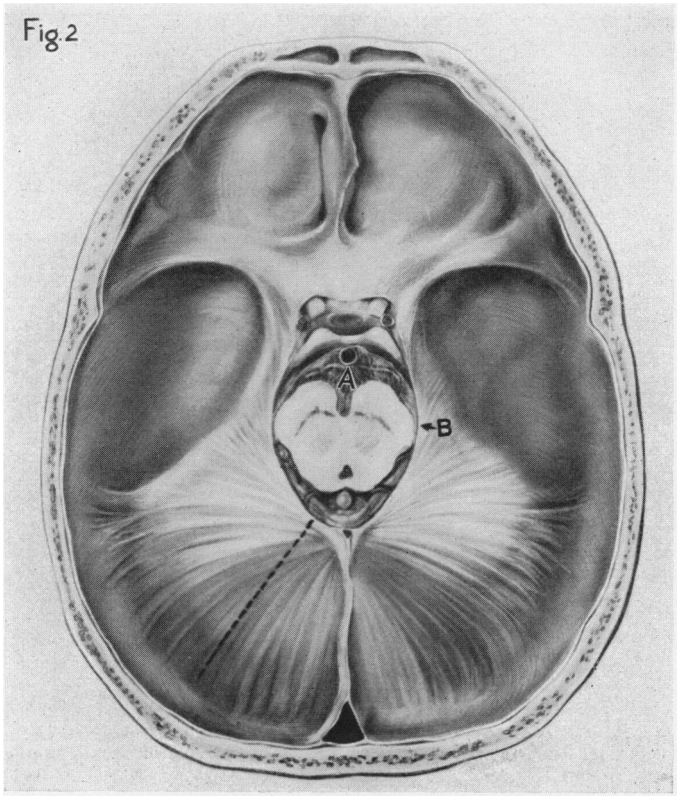

FIG. 2.—Base of skull with tentorium intact and brain stem transected at level of the incisura of the tentorium. (A) Interpeduncular cistern.

sure on the same side is greater than on the other side. Consequently when bilateral trepanation is performed in such a case cerebral tissue herniates through a small dural opening under greater pressure on the ipsilateral than it does on the contralateral side.

An expanding lesion in or upon one hemisphere usually produces a decrease in the size of the ventricle of the same side and an increase in the size of the ventricle of the opposite side. This increase very often amounts to dilatation. The mechanism of this contralateral hydrocephalus varies with the position of the expanding focus but for the most part pressure is exerted indirectly against the midbrain which cannot be displaced because it is ringed

about by the falciform edge or incisura (Fig. 2). Thus, caught between the expanding hemisphere and incisura, the aqueduct of Sylvius is closed sufficiently so that fluid can escape through it only when the ventricular pressure becomes very high. This back pressure must act equally upon ipsilateral and contralateral ventricle, but the higher pressure within the ipsilateral hemisphere counteracts the tendency to dilatation on that side while the lower intracerebral pressure permits dilatation on the opposite side.

Such indirect pressure upon the midbrain may produce another effect of practical clinical importance. It causes the cerebral peduncle of the opposite side to be cut by the sharp edge of the incisura of the tentorium. This may result in a pyramidal defect on the ipsilateral side of the body including grave paresis.

For example, a patient whom I saw recently was found to have a right homonymous hemianopsia and paralysis of the left arm and leg. In the presence of increased intracranial pressure this indicated an expanding lesion of the left occipital lobe and a lesion of the pyramidal tract from the right hemisphere. Ventriculography showed the expanding lesion to be in the left occipital lobe as surmised and the right lateral ventricle to be dilated. An intracerebral blood clot was evacuated from this lobe and the patient rapidly recovered from the left hemiplegia. Thus the expansion within the left occipital lobe had affected the left motor cortex not at all but had compressed the midbrain against the opposite edge of the tentorium at B in Fig. 2, thus closing the aqueduct and injuring the opposite peduncle against this sharp edge.

In any case, of course, the cause of expansion should be removed radically but when this is impossible or ill-advised subtemporal decompression may be of the greatest help. The decompression should be on the side of the lesion and it may thus result not only in giving more "elbow room" for the brain but it may free the aqueduct of Sylvius from its compression between expanding lesion and tentorium and thus abolish the contralateral hydrocephalic pressure which can so seriously complicate the situation. Decompression on the opposite side would be of no help whatever and as we have pointed out above would only result in destruction of the portion of brain exposed.

Small lesions near one foramen of Monroe may, of course, produce a unilateral hydrocephalus on the same side of the cranial cavity. If an expanding lesion does not produce ventricular dilatation on either side then it may be temporarily relieved by subtemporal decompression on either or both sides.

As the result of an expanding lesion anywhere in the cranial cavity two pressure cones may be produced. First the temporal lobes may be crowded into the incisura of the tentorium (A, Fig. 1, and B, Fig. 2, dotted line), even to the extent of producing a bilateral pyramidal tract compression and probably also loss of consciousness. Secondly a pressure cone may be formed at the foramen magnum (Fig. 1, B), the outlet of the posterior fossa. A portion of the cerebellum is crowded down into this foramen producing

evidence of bulbar compression, Cheyne-Stokes breathing, and respiratory failure.

An expanding lesion in the posterior fossa presents certain characteristic difficulties. It causes local pressure and closes the aqueduct of Sylvius, thus producing hydrocephalus above the incisura of the tentorium. This results in a secondary pressure downward through the exit of the middle fossa (Fig. 1, A) against the already embarrassed structures in the posterior fossa which include the medulla oblongata. Such a lesion also tends to close the pathway for fluid through the basal cisternae as seen in Figs. 2 and 3. This in turn must further increase the pressure due to fluid formation by the choroid plexus within the fourth ventricle.

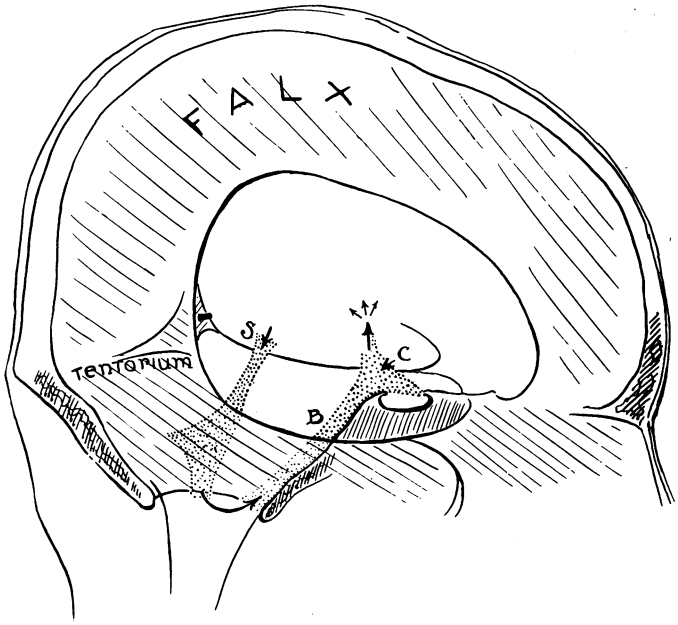

FIG. 3.—Schematic drawing of skull with dural reflections in place to show the relation of aqueduct of Sylvius (S), and basal cisternal passage (B) to the incisura of the tentorium. (C) Interpeduncular cistern.

This is, of course, the explanation of many of the sudden deaths that occur in such patients when they are straining, as at stool. This explains why a patient with a tumor of the posterior fossa who complains of transient attacks of dizziness or blackness before the eyes should be looked upon as a candidate for emergency operation.

Lumbar puncture should usually be avoided but if carried out should be done with a small needle and the pressure as shown by manometer never reduced by more than one-half its initial value. Encephalography by the lumbar route should usually be avoided but if carried out the pressure should be kept high, oxygen used instead of air and the spinal pressure at the close left at least as high as it was found initially.

If the ventricles are tapped oxygen should be used for ventriculography.

Instead of producing the dangerous secondary rise of pressure that follows air, oxygen seems to cause a fall of pressure, apparently being absorbed from the ventricles progressively, as pointed out to me by my associate, Doctor Cone. Here again, however, much less disturbance is produced if the gas is injected under a pressure equal to that of the preexisting ventricular fluid and not under a lower pressure. But, in spite of all this and however such injections are done, radical operation if indicated at all is best carried out the same day, for the least disturbance may upset the pressure balance and cause respiratory failure.

When bulbar ischemia has resulted in Cheyne-Stokes breathing or in arrest of respiration, oxygen by nasal catheter or tracheal tube is obviously indicated, and five or ten per cent of CO_2 might be added without damage, as indicated by Carl Schmidt's recent work. After the bulb has been deprived of oxygen too long suboccipital decompression and opening of the foramen magnum and arch of atlas are of no avail. No tissue in the body is more certainly destroyed by ischemia of a few minutes' duration than the brain, as shown by Cobb and others, and the neurosurgeon who decompresses the medulla oblongata under artificial respiration only a few minutes too late is forced to watch his patient die a few hours later.

Diffuse cerebral edema is a baffling complication which follows cerebral trauma whether due to head injury or to operation. The question of its mechanism would lead us into a pathological discussion not called for in this address.

Following the work of Weed and Wegefath, hypertonic solutions were used intravenously by clinicians as well as magnesium sulphate by mouth or by rectum in order to lessen such swelling. It has been shown that this shrinking effect is transitory, disappearing often within the hour as measured by a needle left in the spinal canal, and some observers believe a greater pressure follows after the temporary relief. Consequently these procedures have recently become somewhat less frequently used. Lumbar puncture takes off the added weight of the pressure of ventricular fluid, the absorption of which is temporarily blocked by the superficial edema. Repeated lumbar punctures do not remove enough blood to be of any importance from that point of view for Sprong showed that however full of blood the spinal fluid may be, and even though the lumbar puncture needle be wielded by the most persistent of picadors not more than from two to five cc. of whole blood are actually thus removed in a whole series of punctures.

After operation, drainage of spinal fluid through a stab wound in the scalp is sufficiently safe for some days and will relieve the secondary complication of fluid pressure during the period of initial cerebral edema. At the time of closure a surgeon must decide whether he will leave the bone flap floating and thus allow strain to be exerted upon the scalp incision by cerebrospinal fluid, or whether he will wire the flap back into place. In making the former choice he is forced at times to defend the suture line by

aspiration beneath the flap or by drainage. In case the bone flap and dura are closed he is more often driven to lumbar puncture. In any case, whatever the technic, the intracranial pressure is lowered by any means that can be employed to drop the venous pressure of the large veins of the neck and the head should therefore be higher than the thorax and there must be no constriction of the neck after operation and when venous hemorrhage is feared most.

In conclusion, intelligent surgical therapy demands an understanding of the principles of physiology involved in cases of increased intracranial pressure. These principles include the mechanisms of hydrocephalic dilatation of the ventricles, the rôles of the falx, tentorium and foramen magnum under such circumstances and the uses and dangers of decompression.

DISCUSSION.—DR. WILLIAM J. MIXTER (Boston, Mass.).—It seems to me in the past we have done decompression without knowledge of what we were doing physiologically.

It has been a great pleasure to hear Doctor Penfield. He has given us reasons pro and con concerning decompression and I feel sure that the acceptance of these principles will be of great value.

I would like to ask Doctor Penfield why it is that a certain number of patients in the past who presumably had brain tumors which were never proven were markedly relieved for a considerable period of time by subtemporal decompressions.

DR. WILDER PENFIELD (Montreal, Canada).—I find it difficult to answer the question. I think there are a certain number of cases of pseudotumor which are in reality, of course, not tumor at all, but actually cases of collection of subdural fluid either secondary to trauma or secondary to some primary inflammatory process in one of the sinuses.

I think in those cases there is a collection of a fluid, high in protein, within the subdural space, which cannot escape. Consequently decompression allows the fluid to drain into the muscles and it does not reform, so that in some cases there have been permanent cures from decompression or trepanation.