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TRAUMATIC DILATATION OF THE CEREBRAL VENTRICLES

BY

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CASES of traumatic dilatation of the ventricles may be separated into three groups although any one case may belong to more than one group.

1. Local bulging of the ventricle which in extreme degrees presents the appearance of a diverticulum. This condition will not be discussed, because the atrophy of the brain and the formation of a cerebral or meningo-cerebral cicatrix which may result from focal contusion and laceration of the brain rest upon a firm foundation of fact, of unequivocal observation.

2. The enlargement involves the whole of one lateral ventricle; it may occur on that side of the head which received the blow, or on the opposite side. These cases may provide a link connecting the processes leading to local bulging, and those causing general symmetrical dilatation. It is tempting to assume that a large focal contusion can lead to œdema of the whole hemisphere. If this were proven, and if the œdema alone, as opposed to any vascular disturbances which cause œdema were sufficiently severe and enduring, then doubtless gliosis and atrophy would lead to ventricular enlargement. Although such an explanation is accepted by many, an œdema of this extent has not yet been satisfactorily proven. The occurrence of contralateral enlargement seems related to contrecoup effects, but the precise mechanism of dilatation is not thereby explained. Raised intracranial pressure by itself cannot be the cause, unless a softened state of one hemisphere allows its ventricle to dilate.

3. General symmetrical enlargement of the lateral ventricles; rarely the third and fourth ventricles are involved, although not to any considerable extent. Two questions immediately present themselves. What is an abnormally large ventricle, and what is the incidence of the condition? Davidoff and Dyke (1937) measure the distance between the outermost limits of the bodies at the lateral angles as seen in the antero-posterior projection and regard 3.5-4.5 cm. as normal. Robertson (1941) proposed

virtually the same diameter, and his figures are comparable. Evans (1942) proposes the ratio of this diameter to the transverse diameter of the skull. These methods of measurement are of help when used with identical film-target distances, but they do not take into account variations in size according to age, nor do they give due weight to those lesser degrees of enlargement in which there may be only slight increase of the lateral diameter, but considerable rounding of the angles and deepening of the body of the ventricle. The incidence of ventricular dilatation is unknown, and will remain so until encephalography is performed on every case of head injury; such a thorough investigation is not likely to be attempted. Every case would need to be done or at any rate cases with symptoms persisting more than twenty-four hours, in order to get a correct perspective, for personal observations suggest that even in clinically mild injuries slight dilatation may occur. Such figures as are available relate only to selected cases.

Peter Bielschowsky (1928) described a series of 106 cases of head injury investigated in Foerster's Clinic at periods after the accident of from several months to years. They were highly selected by reason of abnormalities of the cerebrospinal fluid system which were present to a varying degree in all. Of 48 cases of the so-called post-contusion syndrome in which there were no focal signs of cerebral damage, 77 per cent. showed abnormal encephalograms comprising for the most part various degrees of dilatation. In 38 cases in which focal signs were also present—and in those the injury was probably more severe—there were 87 per cent. of abnormal encephalograms. In a third group of 30 cases of traumatic epilepsy there were also 87 per cent.

In my own cases of head injury, no attempt has been made to gauge the frequency of ventricular dilatation as the utilization of encephalography was somewhat haphazard, but a few cases have been selected to illustrate certain points.

Case 1.—B. aged 34 years: Canadian soldier. 28th March, 1942, rendered immediately unconscious by a fall down two flights of stone stairs. Half an hour later, at 11.10 p.m. B.P. 140/100. Plantars $\nearrow \nwarrow$. Right pupil dilated and fixed. Laceration of occiput; X-rays showed fissure fracture left occipital bone. At 1.0 a.m. stertorous breathing. B.P. 245/120. Almost unresponsive; pupils, right larger than left, no reaction; moves limbs; spasms of extensor rigidity; tendon jerks all exaggerated, plantars $\nearrow \nwarrow$. C.S.F. pressure 200 mm. bright red fluid (2½ million red cells per cu. mm.).

2.0 a.m. Operation: exploration for right extradural hæmorrhage—none found. Tense brain—subdural and subpial bleeding; right ventricle tapped with difficulty (i.e. compressed) but left ventricle easily entered; blood in ventricles. Right subtemporal decompression performed. Gradual recovery.

29th March, 1942. C.S.F. pressure 400 mm. and reduced to 120 mm. fluid bloody, decompression tense.

30th March, 1942. C.S.F. pressure 210 mm. and reduced to 100 mm.

1st April, 1942. Responding to voice. Lumbar puncture difficult to perform, thought to be due to low C.S.F. pressure.

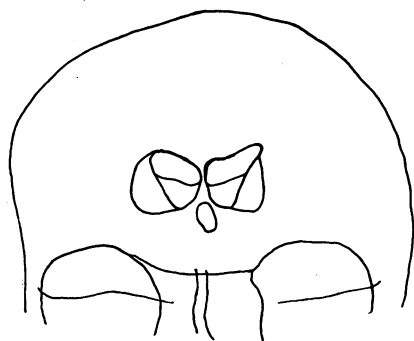
2nd April, 1942. Decompression only slightly bulging.

6th April, 1942. C.S.F. pressure 130 mm. 60 mgm. per cent. of protein.

6th May, 1942 transferred to Canadian Hospital, and subsequently graded category C. Slight increase of tendon jerks on right side. Right pupil larger than left with poor reaction and limited upward movement.

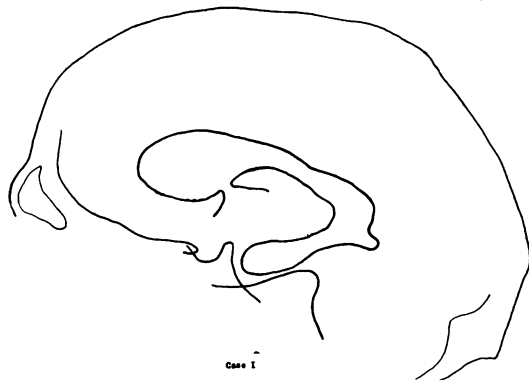
Diagnosis: contusion of right hemisphere and mid-brain.

Encephalogram: enlargement of lateral ventricles, especially right frontal and temporal horns (Figs. 1a and b).



Case 1

FIG. 1 (a).



Case 1

FIG. 1 (b).

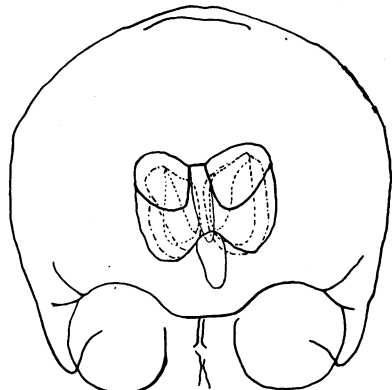
Case 2. encephalogram on 24 Jan., '41 ————
" " 4 Jun., '41 ————
" " 25 Jul., '42 ————

FIG. 2 (a).

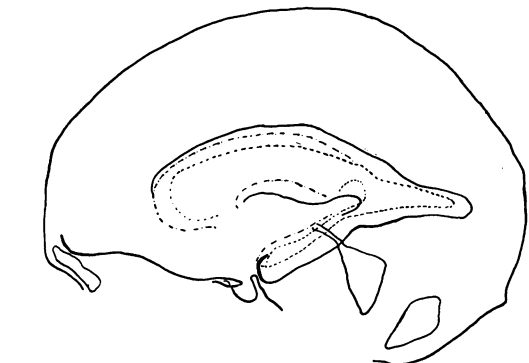
Case 2. encephalogram on 24 Jan., '41 ————
" " 4 Jun., '41 ————
" " 25 Jul., '42 ————

FIG. 2 (b).

(I am indebted to Colonel Botterell, R.C.A.M.C., for the subsequent history of this man and for permission to publish the tracing of the encephalogram.)

The clinical findings pointed unmistakably to a mid-brain contusion and operation yielded good evidence of a right hemisphere contusion. But encephalography demonstrates considerable general cerebral atrophy, especially of both frontal lobes. His intellectual capacity is unimpaired, for he is back in the Service as an electrical engineer and he has no anosmia. The blow struck the back of the head. Thus there are good grounds for postulating a bifrontal contusion, but no unequivocal clinical evidence that this actually occurred.

Case 2.—G.J. aged 45 years: night watchman. 6th October, 1940 admitted with "signs of cerebral irritation" to Middlesex Hospital, having been found at bottom of flight of stone steps. B.P. 100/85. X-ray: fracture left parietal running into sagittal suture and right occipital suture with separation. There was a period of mental confusion and of euphoria and he was discharged on the 18th January, 1941. He stated that he was a watchman in building in Holborn which was bombed. There was a history of fits but details were not forthcoming, except that the woman he lived with stated that he seemed well until the bombing.

22nd January, 1941 fell unconscious in street—admitted to London Hospital and transferred next day to Chase Farm Hospital. He was then drowsy and dysarthric, with ptosis, impaired upward movement of eyes, and slight left-sided spastic weakness. Plantars $\nearrow \nwarrow$. Slight headache.

C.S.F. sitting, pressure 70 mm. (fluid could not be obtained in lateral position), pink; pale yellow supernatant fluid; 9 white cells, with red cells and 60 mgm. per cent protein.

24th January, 1941 encephalogram—left ventricle larger than right, query right-sided subdural hæmatoma (Figs. 2a and b).

19th February, 1941 headaches; dysarthria; nystagmus; left plantar now flexor.

20th February, 1941 Encephalogram—slight exaggeration of former appearances. C.S.F. yellow, still high protein. *Small right subdural hæmatoma evacuated*, less than 1 oz. Slow recovery; with dysarthria and residual left-sided weakness.

May, 1941 delusions of grandeur; confabulation; eccentric but reads paper. Orientated in space and time.

21st May, C.S.F. pressure 110 mm. 1 cell and 50 mgm. per cent. protein.

4th June, 1941 Encephalography—generalized dilatation of ventricles including the fourth ventricle (Figs. 2a and b).

25th July, 1942 Encephalography—great degree of ventricular dilatation (Figs. 2a and b).

Now: euphoric. No relatives or friends and could hardly with safety be sent out to fend for himself. Helps in wards and goes errands. B.P. 120/80. Vessels soft. No retinal arteriosclerosis; slight weakness left upper limb; all tendon jerks exaggerated, bilateral extensor plantar responses. C.S.F. 1 cell. 30 mgms. per cent. of protein.

Although satisfactory details of the accident are lacking, this case is of importance because of the radiological evidence of slowly progressive ventricular dilatation, associated with prolonged mental defect which has latterly improved. The available information as to his former mental acuity is scanty but he seems to have earned his own living which he could not do now. The small subdural hæmatoma was incidental and can hardly have influenced the main clinical picture.

The next case is of a somewhat similar type.

Case 3.—S.S. aged 48 years: boiler repairer and scaler. 6th October, 1940 knocked down by car and transferred from another hospital without any clinical records six days later.

12th October, 1940 varying degree of traumatic delirium and stupor. Skin injuries around right temple and orbit. Neurological examination normal, except brisk tendon jerks. B.P. 140/85. Moderate thickening and tortuosity radial vessels. No retinal arteriosclerosis. Tongue dry. No fracture of skull.

14th October, 1940 C.S.F. pressure 60 mm. 2 cells and 100 mgm. per cent. of protein. W.R. negative. Fluids taken poorly.

27th October, 1940 C.S.F. pressure too low for satisfactory recording—faintly bloody.

28th October, 1940 C.S.F. pressure 195 mm. query bleeding due to lumbar puncture.

14th November, 1940 one month after accident mental state improving but much confabulation and disorientated in space and time.

9th January. Three months after accident encephalography demonstrates large ventricles and capacious subarachnoid spaces (Figs. 3a and b); protein in C.S.F. still high; 60 mgm. per cent.

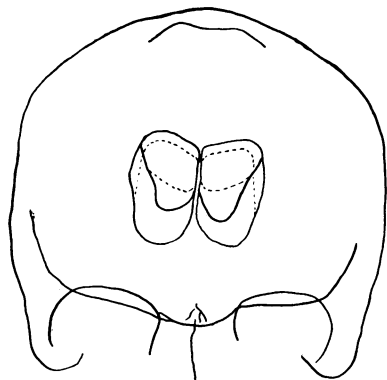
May, 1941 euphoric; no initiative; poor memory; sits about vacantly (wife's description). Has started light employment, sweeps floors and gets tea, etc. for his "mates." No physical stamina. Mental arithmetic fair.

20th July, 1942. Encephalogram—pressure 100 mm. (taken while lying in lateral position). Still high protein in C.S.F. 70 mgm. per cent. Mental impairment. Great amount of air in subarachnoid spaces over convexity; greater degree of ventricular dilatation (Figs. 3a and b).

Like the previous case there has been a slow increase in the size of the ventricles over a period of eighteen months, the dilatation is fairly symmetrical and diffuse with an enormous amount of cortical air. The injury was insufficient to fracture the skull and was apparently applied to the right lateral frontal region. Although there is some thickening of the radial arteries, the retinal vessels are healthy and the blood pressure is normal. There was a prolonged period of traumatic delirium and confusion and there is still a moderate mental defect, but the man can just earn his living. Headache was rarely experienced, and the measured pressure of the cerebrospinal fluid never raised.

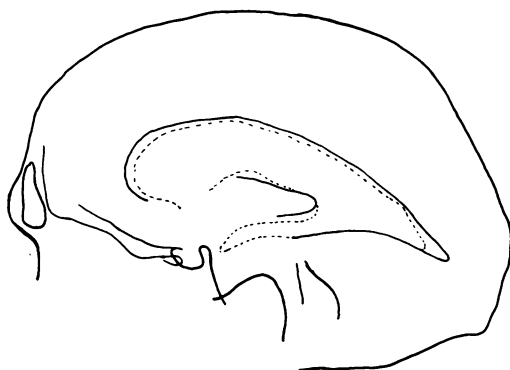
The next case demonstrates the difficulty of deciding whether the ventricles are of normal size or not. Although measurements and proportions are approximately normal as judged by the criteria given above (the transverse diameter is 4 cms. and the ratio 0.26), yet to an accustomed observer, the appearance is one of slight dilatation.

Case 4.—M.M. a big woman of 35 years fell heavily on her buttocks. She was not apparently rendered unconscious, but had severe headache shortly afterwards



case 3. encephalogram on 8 Jan. '41 - - - - -
"20 Jul. '42 - - - - -

FIG. 3 (a).



case 3. encephalogram on 8 Jan. '41 - - - - -
"20 Jul. '42 - - - - -

FIG. 3 (b).

and was kept in bed. Some days later her headaches increased and she showed transient weakness of the left limbs. These symptoms cleared and then headache returned. A subdural hæmatoma was suspected and the encephalogram was performed some five weeks after the accident; the pressure was not raised and the fluid was of normal content. After a further period of rest the headaches disappeared and she resumed her normal home life, but she remained susceptible to bouts of headaches of a migrainous type.

It is a common experience, and supported by published cases, that the degree of ventricular dilatation is usually greatest in those patients who have sustained severe injuries, as judged by such criteria as prolonged unconsciousness, presence of focal signs of cerebral damage, temporary traumatic dementia, etc. and the presence of blood in the cerebrospinal fluid. Not only is the cerebral damage severe, but the prolonged disturbance of consciousness and mental function may mean a widespread or diffuse disturbance of neuronal function. The widespread nature of neuronal paralysis, and its longer duration when the force producing it was increased, was emphasized by Denny-Brown and Russell (1941) in their experiments on acceleration concussion. Confusion of the lesion of concussion with that of contusion is not intended, but a parallel may be drawn; namely that the process leading to dilatation of the ventricles may well be due to a generalized effect on the brain, although its microscopic pathology has not yet been identified.

Broadly speaking the possible mechanisms which may lead to dilatation of the ventricles after injury can be divided into two groups, although in practice it is possible that both may be operative in any given case. The ventricles may dilate as a result of some disturbance of the cerebrospinal fluid system; or they may dilate as a result of some change taking place primarily in the brain tissue, leading to its shrinkage, i.e. hydrocephalus ex vacuo.

Disturbance of the cerebrospinal fluid system might arise from excessive production, delayed circulation, or impaired absorption of the fluid. An excessive production can only be of significance if the production rate is greater than its absorption rate, and from the evidence available (Greenfield, 1936) this seems highly improbable provided always that the absorptive mechanism is undamaged. There appear to be no observations on the human as to the rate of secretion in head injuries, or even in the normal. Hoff (1930) performed experiments on animals in which an inspection window was made in the skull, over a tunnel in the brain leading into the ventricle. After an intravenous injection of fluorescein, a blow was struck on the animal's head. He describes the result as of clouds of pink fluid welling up from the depths of the ventricle presumably from the choroid plexus, which, however, he could not see, and further waves emanating from along the walls of cortical vessels. In their examinations of recently injured brains, Rand and Courville (1931) observed changes in the choroid plexus which they ascribed to an excess of

fluid being passed into the ventricles. Thus there is some suggestive evidence of increased production and the problem deserves more careful attention before it can be stated as fact that increased fluid production does not occur.

Delay in the circulation and absorption of the fluid has been investigated more fully. Bagley (1928) showed that small quantities of blood introduced into the cerebrospinal fluid system of dogs produced matting of the lepto-meninges, scarring of the cortex, and in some cases dilatation of the ventricles. Fay and Winkelman (1930) are said to have demonstrated similar changes in cases of human brain injury and also changes in the Pacchionian bodies, but these observations need confirmation, especially as the granulations are not the only sites of arachnoidal villi. In infants suffering from hydrocephalus secondary to birth injury, matting of the lepto-meninges and retention of blood pigment can be demonstrated. We do not know whether such occlusion of the lepto-meningeal spaces is permanent, or whether in course of time fresh spaces are opened up, comparable with the canalization of a venous thrombosis. Clinical experience lends some support to the view that blood may act in this way, and in all three severe cases quoted, the fluid was bloody, but unfortunately for the argument, the amount of blood was greatest in the first although the ventricular enlargement was only moderate. Hydrocephalus is not a characteristic feature of recurrent subarachnoid bleeding from aneurysms. If such an obstructive element were active then the cerebrospinal pressure should be persistently raised, but in cases of head injury the pressure if raised usually remains so only for a few days after the injury, and it is not known whether the ventricles remain permanently dilated after a transient intracranial hypertension.

In the series of cases of Bielschowsky already referred to, this matter of delayed fluid circulation was carefully investigated. He stated that in 54 per cent. of all the 106 cases studied, the pressure was over 200 mm. Seeing that these measurements were made months and years after the injury, the proportion seems amazingly high. Somewhat similar results were published by Gierlich (1936). It is difficult to reconcile these findings with personal experience, and although Bielschowsky's paper came from Breslau, it is unacceptable without further confirmation. He also made tests upon the rate of absorption of the cerebrospinal fluid, using the iodide method of Foerster. There was a marked delay in 58 per cent. But this aspect of the problem becomes more complicated when we consider the result of encephalography. In some cases, the cortical subarachnoid spaces are not filled with air, and their occlusion may in fact have taken place. But in many cases a great quantity of air spreads over the cortex, so that occlusion cannot have occurred. We need to know whether there is impairment of the absorptive powers of the villi in such cases. Analysis of Bielschowsky's series shows that there were 28 cases of extensive collections of

air over the cortex and in 17 there was delayed absorption according to the test of iodide excretion.

It appears therefore that there is some evidence to support the view that disturbances of the cerebrospinal fluid system may lead to traumatic ventricular dilatation, but that the clinical and histological observations need further confirmation, and that investigation of the rates of secretion and absorption of cerebrospinal fluid in the normal and the traumatized must be obtained before the hypothesis can be accepted as fact.

We have next to consider whether the ventricles dilate and whether the cortical subarachnoid spaces enlarge, as a result of shrinkage of the brain substance due to change occurring primarily in the brain tissue. Here we pass more than ever into the morass of conjecture, shibboleth, and unsubstantiated observation. The view favoured by most writers is that there is a generalized oedema of the brain, which leads to gliosis and atrophy. This may be so, but critical analysis provides few if any facts to support it. Does an oedema of the whole cerebrum, or of all of one hemisphere, ever occur as the result of trauma? No records have been discovered of any histological preparations of serial sections of the whole cerebrum, or of a sufficient number of widespread samplings of brain to substantiate this claim. On the basis of his histological studies, Courville (1942, 1937) believes that generalized oedema occurs; on the other hand Greenfield (1942) states that in 31 traumatized brains, he has never seen a generalized oedema, and that the oedema has been restricted to an area some few cms. from a focal contusion. Estimations of the water content of the brain have been made, but either the methods have been unreliable, or the samplings of the brain tissue have been too few. In a recent paper Courville (1942) disinters the descriptive terms of "wet brain" and "dry brain," used at times by surgeons and pathologists alike. These terms are picturesque but mean nothing and have no scientific value. The writer has not seen, or has not recognized, such conditions at post mortem. At operation on a case of head injury one nowadays usually sees no more of the brain than is exposed by a burr hole and an opinion of the state of the brain as a whole can be of no value at all. Very occasionally one performs a subtemporal decompression and this is usually on account of focal brain contusion in the course of exploration for hæmorrhage. It is not suggested that generalized brain oedema has no existence, but it must be recognized that its actuality has not yet been proven.

There are certain observations of a different nature which merit attention. Reference has already been made to Hoff's experimental cerebral trauma. He found that some four to six minutes after the injury, the cerebrospinal fluid pressure began to rise, and this coincided with vaso-dilatation and with efflux of clouds of fluorescein. At this stage autopsy revealed a somewhat dilated ventricle. If the experiment were prolonged the intracranial pressure gradually fell and after a

period of some twelve hours, the ventricles were found to be small, there was said to be oedema and fluorescein staining of the brain especially marked around the ventricles. The ependyma was found to present a number of fine tears and folds. In the light of the experiments of Denny-Brown and Russell (1941) these observations appear open to criticism, but Hoff's experiments may be classed as illustrative of compression-concussion and contusion, as opposed to acceleration-concussion. Denny-Brown and Russell did admit vasodilatation, and a rise of pressure in states of contusion; and the duration of their experiments was much shorter than those of Hoff's. Hoff argued that an outpouring of cerebrospinal fluid causes transient dilatation of the ventricles, and thus by leakage through a damaged ependyma might give rise to oedema. Gierlich (1936) reports 45 cases of brain injury, in which death occurred up to fourteen days after the accident; he describes defects in the ependyma, and hæmorrhage, hyperæmia and perivascular bleeding under the damaged and intact ependyma. These changes were parallel with the clinical severity of the injury. Rand and Courville (1931) also described changes in the wall of the ventricles, vacuolization of the ependymal cells, raggedness of the free cell margin and subependymal oedema. He interpreted these findings as demonstrating an outpouring of fluid into the ventricle from its wall, but the reverse may be actually the case. Finally, Greenfield (1942) has noted changes about the ventricle wall in cases of rapid hydrocephalus, which he attributes to a seepage of the fluid through the ependyma into the brain. These isolated observations seem to form a pattern, or at any rate a mosaic, and suggest that perhaps in severe trauma there is a temporary excessive outpouring of fluid from the choroid plexus, which by reason of a transient delay of circulation and absorption owing to the presence of blood, and aided by a traumatized ependyma penetrates the wall of the ventricle to enter the brain tissue, impairing its nourishment and causing oedema and thus to gliosis.

Summary

The cause of traumatic ventricular dilatation remains unidentified, and in endeavouring to solve the problem, hard facts concerning the following items need to be ascertained.

1. The normal size of the ventricles, and its variation with age; the incidence of traumatic dilatation.
2. Observations on the rate of secretion and of absorption of cerebrospinal fluid in health and after trauma to the head.
3. Whether blood in the cerebrospinal fluid leads to permanent impairment of its channels and its absorptive organ.
4. The occurrence or otherwise of a generalized form of brain oedema.
5. The histological picture of traumatic dilatation.

The weight of evidence indicates some change

occurring primarily in the brain tissue, leading to a degree of atrophy. But it is quite possible that more than one mechanism may operate.

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