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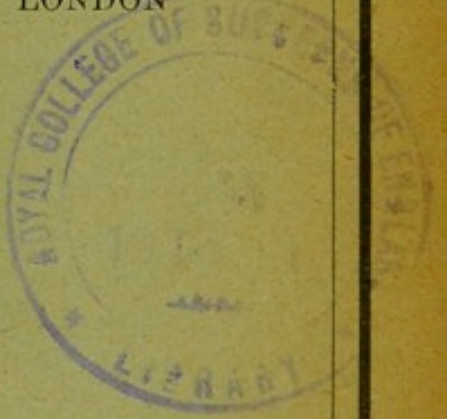

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ON DIFFUSE ENCEPHALITIS: WITH AN ACCOUNT OF A CASE[1] IN WHICH THE PATIENT SURVIVED.

BY R. LAWFORD KNAGGS, M.C.
AND
R. CONYNGHAM BROWN, M.B.

James C——, age 10, was admitted into the Huddersfield Infirmary, on October 3, 1891, under the care of Mr. Samuel Knaggs, for spastic paralysis of the left arm and leg and suffering from fits.

He was the son of healthy parents, and one of a healthy family.

He had perfect health till five years before, when he fell from a wall seven or eight feet high. He was taken indoors, and, though apparently none the worse for the fall (?) it was thought advisable to put him to bed. The following morning he could not be roused, and a medical man was sent for, who "said he was suffering from compression." The greater part of that day he lay unconscious, and then gradually recovered; and in a few days was going about as usual. A month later, without any apparent cause, he fell down in a fit; he was unconscious, and "twitched his left arm and leg." After an interval of two months he had a similar fit, and, for a period of two years, the fits recurred every other month. Between them the boy appeared all right, but for slight loss of power on his left side, which was noticed towards the close of that time.

Nearly three years before admission he had a second fall; and though it was only from a height of two or three feet he was taken up insensible. For two weeks he lay unconscious and perfectly quiet, the only change being slowly increasing emaciation; and then, though gradually regaining strength, he was unable to rise from his bed for eight months.

[1] The writers are indebted to the kindness of Mr. Samuel Knaggs for permission to publish this case, and to Dr. F. L. Mackenzie for some valuable notes about the early history of the illness.

Dr. F. L. Mackenzie, who was called in to see the patient somewhere about this time, has kindly supplied us with the following notes of his condition. On January 9, 1889, he saw the patient for the first time, and "found him in an extremely weak and emaciated condition. He lay on his right side, his eyes half open, and without any other characteristic facial condition. He was coiled up, his head being flexed upon his chest, and both thighs acutely flexed on the abdomen. When shouted at, or shaken somewhat roughly, he responded with a howl and again became quiescent. There was no evidence of hemiplegia. The emaciation was so extreme that unilateral wasting could hardly have been detected. His pupils were equal, and only slightly responded to light. The conjunctival reflex was very imperfect, and so was the knee tendon reflex." "Urine and fæces were passed in bed. Respiration was regular, and not indicative of cerebral disease. P. 120, small. Temperature 97.8." "In the course of a week the patient began to move in bed, and take more nourishment."[1] By the middle of April, his general condition was greatly improved, and he was moderately intelligent, but "as movement of the limbs returned, evidence of hemiplegia showed itself—flattening of the left lip, slight contracture of the left hand, and flexion of the left leg" (which last was corrected by extension). The condition of spastic paralysis, which the arm presented on admission, gradually developed, the flexion being first noted at the wrist, and then at the elbow.

During the eight months the patient was in bed, he had no fits, but shortly after getting up he had one, in which he is stated to have squinted and worked the left side. These fits continued with increasing frequency till his admission. Dr. Mackenzie was again consulted on September 7, 1889, and describes "attacks of sudden jerking, especially of the left leg"—"a sudden clonic spasm sometimes causing the patient to fall." "There was no loss of consciousness, no aura and no drowsiness after the attack." At first, these attacks occurred two or three times a week, but they practically disappeared under treatment by the end of November. In March, 1891, Dr. Mackenzie saw the boy again for "similar attacks affecting the upper extremity more markedly than formerly." They occurred now as often as two or three times a day, and treatment had no effect upon them.

With the increase in number of the fits, the loss of power on the left side became more marked, and for 18 months previous to

[1] Mercury was given during this period, and its value seems to have been very marked.

admission his parents stated that he had become peculiar in manner and difficult to control.

On admission (October 3, 1891) the patient was a powerfully-made well-nourished, healthy-looking boy. His bright eyes gave him an intelligent expression, but his slowness in answering questions, deliberate enunciation, and eccentric behaviour evidenced an impaired intellect.

His gait was peculiar and jerky, owing to the spastic condition of the left leg, which was advanced without bending the knee. The jerkiness was communicated to the left arm.

There was slight left facial paresis and a spastic paralytic condition of the left arm and left leg.

The left arm was always held close into the side. The hand was bent at right angles to the fore-arm, and the phalangeal joints were flexed. The fore-arm was pronated and bent almost to a right angle with the arm.

The hand was smaller than the other, and the limb slightly thinner than the right.

The deltoid was particularly flabby. The arm could be abducted nearly to the horizontal by rotation of the scapula, but the deltoid was inactive. With effort he could partially extend the elbow, but, once extended, it was flexed without difficulty. There was hardly any power of movement in the wrist and finger-joints. There was considerable rigidity to passive movement, which was most marked in extension.

The left leg was extended. It was of equal length, but slightly thinner than the right, and the power of voluntary movement was only slightly inferior. The greatest difficulty was shown in extending the foot.

Rigidity to passive movement was present in flexion and extension, and especially in abduction.

On the left side there was slight ankle clonus and an exaggerated knee jerk. Sensation was normal on both sides. The fundus in both eyes was healthy, and no evidence of an old papillitis could be detected.

The heart, lungs, and abdomen were normal, and the urine had a sp. gr. 1020, and contained no albumen.

The boy was kept under observation for a fortnight, during which time he had at least three or four fits every day. They were of an epileptiform character, and showed a distinct tendency to become more frequent.

He had a stiff feeling in his left arm when they were coming on. During the fit he became unconscious and fell. The mouth

was drawn to the left side, and a stiffening of the left arm and leg was succeeded by clonic spasms; these, though most marked on the left side, were never entirely limited to it. A fit lasted about two minutes, and on recovering he became pale and faint for a few seconds and then said he felt well again.

The convulsive movements did not begin in any particular muscle or group of muscles. At other times the left arm would be involuntarily jerked forward once. This was of frequent occurrence and was unaccompanied by any other symptom. The temperature was normal, but when taken in the left axilla it usually registered nearly a degree lower (subnormal) than when taken in the right.

The history, and the unilateral and progressive character of the paralysis and of the convulsions pointed to some lesion of a traumatic character implicating the R. motor area, and after consultation it was decided to explore over that part of the brain.

Operation. On October 16 (the boy's head having been previously shaved and rendered aseptic), the situation of the fissure of Rolando was marked out on the right side of the scalp, a large semi-lunar flap reflected down, and two half-inch discs of bone removed above and below a point situated over the arm centre. The dura mater did not pulsate very evidently at this stage, and it did not bulge. A third disc in front of, and intermediate between the other two was removed, the intervening bone marked out by Hey's saw, and clipped away with bone forceps, and in this way a triangular aperture was made whose sides measured from 2 to 2½ inches. The exposed dura concealed the lower $\frac{2}{3}$ of the fissure of Rolando, and the centres for the face and arm, and part of the leg centre.

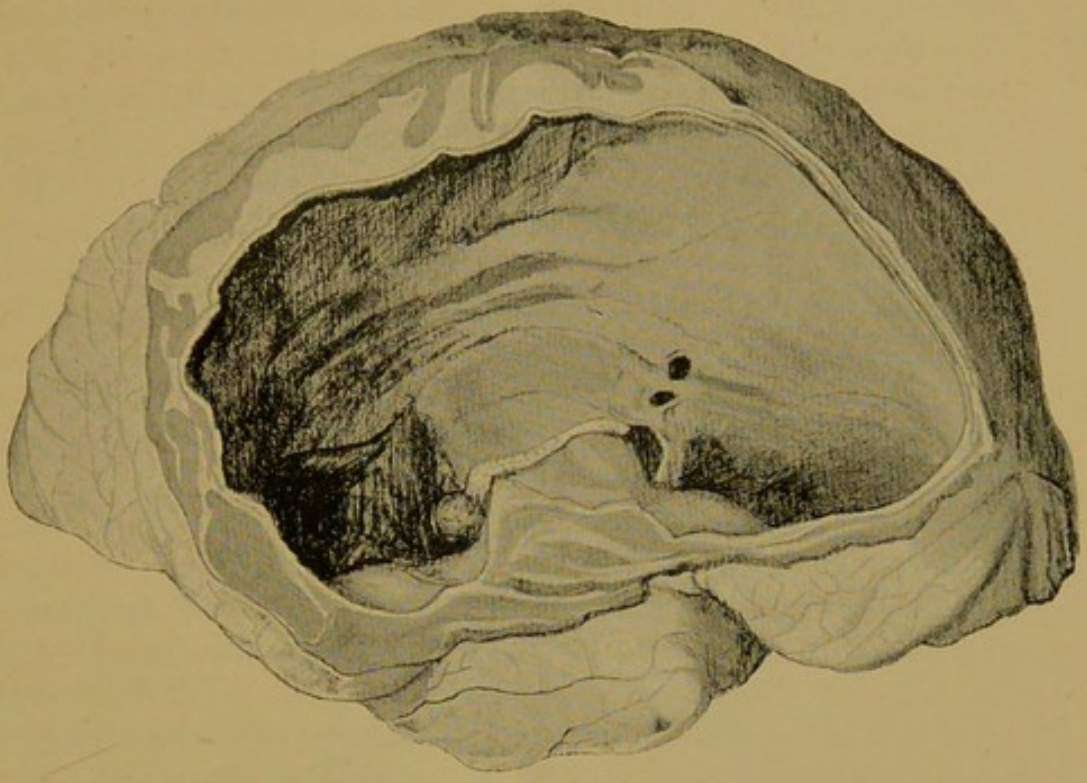
The dura did not bulge at all into the opening, and there was no pulsation, but the child's condition had rapidly failed and the absence of pulsation was thought to be connected with that fact. The flap was re-adjusted and further exploration postponed.

There had been a good deal of hæmorrhage from the flap and the bone, but not to such an extent as to raise alarm.

Before the operation $\frac{1}{6}$ of a grain of morphia was to have been injected, but owing to a defective syringe less than that quantity was probably administered.

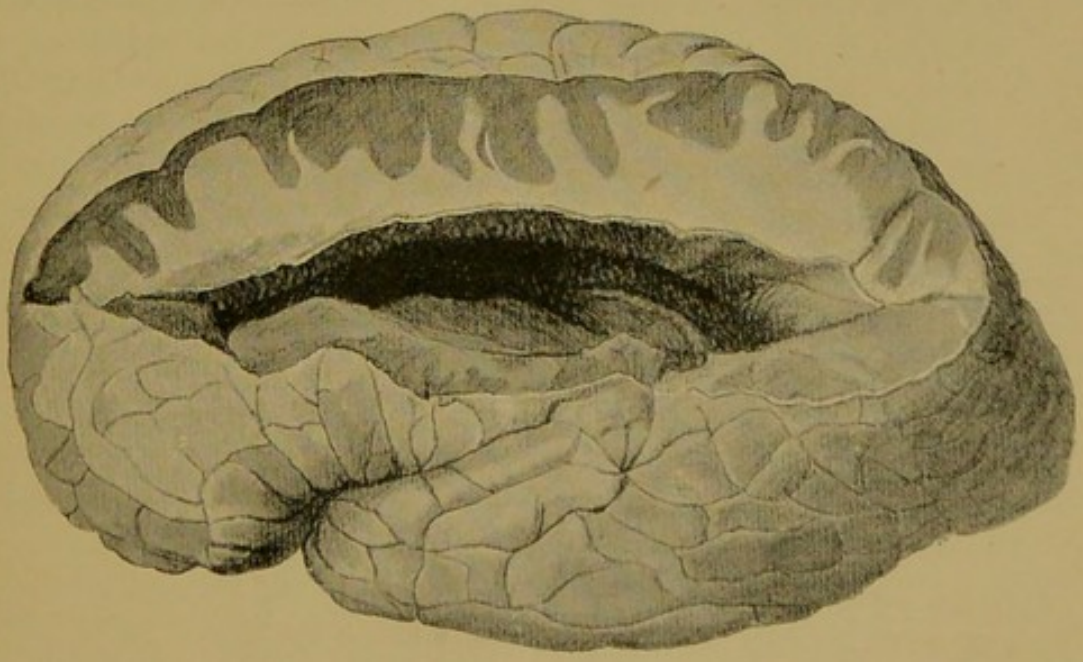
At the beginning of the operation the child's condition was good, but no chloroform was given after the removal of the second disc.

When Hey's saw was being used the anæsthetist found it necessary to inject ether, and though the operation was concluded



DRAWING OF THE RIGHT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]



DRAWING OF THE LEFT LATERAL VENTRICLE.

(Case of J. C.) [*R. C. Brown, fecit.*]

with all despatch and means adopted to lessen shock, the pulse could not not be felt at its conclusion, and the patient's condition was very serious. He was placed in bed with the head and shoulders depressed, and for twenty minutes or half-an-hour respiration had to be kept going by slight artificial aid. Then gentle breathing became established, and it was just possible to detect the pulse. During the operation, and till death occurred, some four hours later, the pupils remained contracted and very small.

Post-mortem.—The brain only was examined. The dura mater was quite normal, and there was no opacity or thickening of the arachnoid. There was œdema of the pia mater on the convexity of the R. hemisphere over the frontal and parietal lobes. This, no doubt, was due to the operation. The surface of the R. hemisphere was fluctuant and tremulous from within an inch of the anterior extremity of the frontal lobe as far back as the occipital fissure, and laterally from half an inch from the longitudinal fissure to the sylvian fissure. The same condition was marked in the middle temporo-sphenoidal convolution. It was plain that the R. hemisphere contained a cavity full of fluid, and that its walls were very thin. Eight ounces of fluid were removed with an aspirator, and the brain was placed in a bichromate preservative solution.

The fluid was clear, of an alkaline reaction, and had a sp. gravity of 1010. It contained a trace of albumen and sugar, and a considerable quantity of salt. Microscopical examination gave no information.

Examination of the hardened brain.—The R. hemisphere was decidedly smaller than the left. This difference was not noticed at the necropsy, and was probably due to the preparation of the brain and the removal of some of the fluid contents.

In order to expose the R. ventricle, its roof was removed by a vertical incision 6½in. long parallel to the median fissure and half an inch external to it. The extremities of this incision were joined by a horizontal cut which divided the sylvian fissure near its bifurcation. The roof so removed was 6½in. long by 3in. wide at its broadest part.

The dimensions of the R. ventricle were :

Length	4⅝in.
Breadth in ant. cornu	1⅛in.
,, in centre of ventricle	1 1/16in.
,, behind descending cornu ...	1¾in.

Depth, everywhere about 1¾in., except at the anterior part, where it was 2⅛in.

In shape it resembled the hollow cast of a large kidney, the dilated anterior cornu corresponding to the upper extremity, the posterior and part of the descending cornu, dilated into a single hollow, represented the lower extremity, whilst the concavity of the hilus was suggested by the rounded elevation formed by the corpus striatum and optic thalamus.

The *thickness* of the wall of the ventricle measured less than $\frac{3}{16}$ of an inch anteriorly, $\frac{1}{2}$ an inch posteriorly, and $\frac{14}{16}$ of an inch externally at its thickest part, where the section had traversed the temporo-sphenoidal lobe.

The anterior and posterior cornua were dilated beyond recognition, and formed part of the general kidney-shaped cavity. The descending cornu was enormously enlarged, and when the little finger was introduced to the end, its tip could be moved about as if inside the finger of a much too large thick glove.

The lining membrane of the ventricle was thick and hard, and could be partially peeled away here and there; the thickening was common to both ventricles, but the readiness with which it could be separated in the right as compared with the left was due to the firmness of the brain tissue surrounding the right ventricle.

The corpus striatum was so compressed and atrophied that its position could only be guessed at. The optic thalamus was visible in front of the choroidal plexus as an eminence $\frac{1}{2}$in. long by $\frac{3}{16}$in. wide. In front of this eminence was the foramen of Munro—an irregular opening having a diameter of $\frac{5}{16}$ of an inch. Reaching from this foramen and curving into the descending cornu was the choroidal plexus. The septum lucidum was thinned and expanded, and anteriorly two rounded apertures with smooth edges (diameter about $\frac{3}{16}$in.), and a third minute one communicated with the left ventricle. They were situated just above the foramen of Munro. The walls of the right ventricle looked, and were firm, and when the roof was removed the ventricle maintained its shape and showed no tendency to collapse. The part removed could be handled and raised by one end without any fear of damage resulting. This formed a striking contrast to the soft and succulent condition of the left hemisphere, which resembled fresh brain substance, although it had been in hardening fluids for some months.

The left ventricle was very different.—The roof was removed as on the right side, but on account of the thickness and softness of the walls it was difficult, and when it was lifted off precautions had to be taken to prevent the sides of the ventricle falling together.

The extreme length of the L. ventricle was ... 4in.
The width ,, ,, ,, about ... 1in.
The depth ,, ,, ,, about ... ½in.

These measurements cannot be considered exact, because, in consequence of the softness of the walls and the necessity for handling the parts in order to inspect them, the cavity could be made to appear either larger or smaller, but when care was taken to keep the structures as far as possible in their natural position, the cavity seemed to be approximately of the above dimensions.

The walls were nowhere less than an inch thick, and in some places as much as 1½inches.

During manipulation there was a tendency for the lining membrane with a layer of brain substance nearly ⅛in. thick, to separate from the surrounding tissue.

The anterior, posterior, and descending cornua were larger than natural, but the last-named would not admit the little finger.

The corpus striatum and optic thalamus were normal in shape and appearance ; the foramen of Munro was enlarged, but smaller than that on the R. side, and had a more regular outline. The L. ventricle appeared uniformly and moderately enlarged, but there was no obvious atrophy of the surrounding structures.

Base of the brain.—On looking at the base the difference in size of the two hemispheres was very marked. The measurements were—

R. 6in. × 2½in.
L. 7in. × 3in.

The floor of the third ventricle formed a prominence as big as half a small walnut, and was about the thickness of a grape skin. It was divided by a median incision.

The third ventricle was dilated, and admitted the first phalanx of the index finger without risk of doing any damage. The R. wall was markedly concave—the left was flat.

Section through the pons showed the iter was not dilated. The tissue around it was slightly softened (*post-mortem* change ?). There was no dilatation of, or atrophy of the structures in the *fourth ventricle,* and the cerebellum was unaltered.

The medulla showed descending degeneration in the usual places.

We are indebted to Professor Jacob for the following description of two microscopical sections cut from corresponding positions in the R. and L. frontal lobes.

Right.—"Considerably thinned. Microscopically has lost all trace of nerve structure, showing no nerve cells, but merely condensed neuroglia infiltrated with inflammatory products. Numerous inflammatory foci are present, and the perivascular spaces are enlarged.

"Pia mater shows marked inflammatory thickening."

Left.—"The ordinary layers of the cortex can be recognised, but the nerve cells show considerable degenerative changes, and there is much dilatation of the perivascular spaces. There is no thickening of the pia mater."

There were peculiarities in the clinical history of this case, which all who saw it during life were unable to explain upon any supposition. The pathological conditions throw light upon those clinical difficulties, and in the following explanation we shall try to show how the clinical facts and the *post-mortem* appearances can be interpreted.

The first injury occurred five years ago and, after a considerable interval of consciousness, was followed by slight signs of compression which soon disappeared. That some serious lesion had resulted was clear, for a month later a fit occurred beginning in the L. arm, and this was repeated every second month.

Now this is hardly like a history of hæmorrhage, and nothing was found at the necropsy to give colour to such a suggestion. But it is not at all improbable, in the light of subsequent events, that there may have occurred at this time a transient congestion of part of the encephalon with effusion, capable of producing slight and temporary symptoms of compression; and a bruised brain, which we might expect to be the initial factor in such a traumatic congestion, would easily explain the subsequent development of epileptiform attacks.

After an interval of two years another fall occurred. There is nothing to show whether this was the result of a fit or of accident. Concussion resulted, and from the prolonged unconsciousness (about a fortnight) that ensued it is practically certain that the patient was again suffering from compression. During a very slow recovery he exhibited for a time symptoms of cerebral irritation, and signs of spastic paralysis of the L. arm and leg gradually appeared.

He was unable to leave his bed for eight months, and the medical man who saw the patient at this time has described him as having a "purely vegetative existence."

The pathological appearances suggest that there occurred during this period:

(1) A sub-acute encephalitis involving the whole or greater part of the R. hemisphere, and producing a softening of the affected area.

(2) Inflammatory effusion into the brain substance, into the ventricles, and into the lymph spaces on the surface of the brain, giving rise to increased intra-cranial pressure and to signs of compression.

(3) Dilatation of the R. ventricle with absorption of the softened and inflamed brain tissue surrounding it in consequence of the increased intra-ventricular pressure; and

(4). From the same cause, dilatation of the L. ventricle, but without absorption, because the L. hemisphere was not involved in the inflammation, and was of normal consistency.

(5) Loss of function of those parts of the brain which had been or were inflamed.

Prolonged increased pressure would lead to absorption and recession of the softened inflamed brain tissue wherever it was exposed to it, whether upon the surface of the brain or within the ventricles, and since the ventricles are in direct communication with the lymph spaces upon the surface of the brain, any increase of pressure within the ventricles would speedily equalize itself throughout the whole cerebro-spinal canal.

In order, therefore, to explain why absorption of brain tissue has occurred only at the expense of the softened parts of the ventricle walls *from within*, it is necessary to account for the intra-ventricular contents being maintained at a higher tension than normal, whilst the pressure upon the surface of the brain hardly varied from what was natural; and this can be done if a plausible reason can be suggested to show that the intra-ventricular fluid was prevented escaping by the iter.

The maintenance of an ordinary pressure upon the

surface of the brain is easily understood, for any temporary increase of fluid resulting from inflammatory effusion would speedily be drained away into the general circulation.

Now when the R. hemisphere was inflamed the adjacent and continuous structures could hardly have been so fortunate as to escape entirely without feeling some effect from the mischief, even supposing that they were not directly involved; and it is not difficult to understand how so small a channel as the iter might be occluded by the pressure of inflammatory effusion or œdema in its surrounding tissue. And a further possibility may be mentioned—the orifice of the iter in the third ventricle may have been obstructed by an œdematous ependyma. In one or both of these ways in all probability the excess of fluid secreted into the ventricles was prevented escaping, the intra-ventricular pressure was raised, dilatation of both lateral and the third ventricles took place, and on the R. side the dilatation was not only more marked because this hemisphere was inflamed and soft, but was materially assisted by absorption from pressure of the damaged brain tissue, and at a later period doubtless by the contraction of the organized products of inflammation. When the encephalitis subsided the effusion into the cerebral tissues would be absorbed, the iter once more become patent, and the normal equilibrium of the cerbro-spinal fluid be restored.

If we consider how much of the substance of the R. hemisphere must have been destroyed at this period, partly from pressure and partly from the contraction of cicatricial tissue resulting from inflammatory exudation, there is no difficulty in accounting for the gradual onset of spastic paralysis in the L. arm and leg, which developed during the eight months the patient was confined to bed.

But what is the meaning of the peculiar condition of apathy in which the patient lay during the eight months following the period of compression?

It is probable that after the first two months the brain was in very much the same state as that in which it was found at the *post-mortem*, and it may be that the cause of this condition is to be sought in the loss or diminution of function that most structures are subject to during and after

inflammation. This would explain not only the tedious and very gradual recovery, but also the recurrence of fits which took place soon after he was sufficiently improved to be able to get out of bed, and which had been in abeyance from the time he met with his second fall. As the period of impaired functional activity gradually passed away the power to respond to irritation would reappear in those ganglionic and conducting structures that had not been too seriously damaged to recover.

Lastly, it must not be forgotten that at the time of the operation, and no doubt for months and even years before, the condition of parts was stationary so far as the macroscopic lesions were concerned;—one ventricle was enormously and the other moderately dilated; both were filled with normal cerebro-spinal fluid at the ordinary tension and free from all inflammatory elements. The conditions present were the result of a long past inflammation. The cerebro-spinal fluid was fulfilling its function of properly supplying the vacuum within the cerebro-spinal canal, and it was present in such large quantities because the potential vacuum had been so greatly increased.[1]

In attempting to discuss diffuse inflammation of the brain it is very difficult to find firm ground from which to make a start.

The microscopic appearances of chronic inflammation and sclerosis are apparently indistinguishable, and in doubtful cases the decision between inflammatory or purely degenerative changes can hardly fail to be arbitrary; and to those who are not experts in the pathology of the nervous system the indifferent application of terms, implying different processes of disease, to a particular morbid condition, is very puzzling.

The obscurity surrounding diffuse encephalitis is apparent in the account of inflammation of the brain given by Mr. Jonathan Hutchinson in "Quain's Dictionary of Medicine," vol. i., p. 148 (1883).

[1] The authors trust that the full description they have given of a case to which they have been unable to find a parallel may not be thought unnecessary. Full of interest to the student of medicine, it is most instructive as an illustration of one of the pitfalls that may be encountered in Cerebral Surgery.

"It may, perhaps, be doubted whether the occurrence of diffuse inflammation of the brain substance as an acute disease has as yet been proved, excepting as a result of wounds. Even as a traumatic lesion, its special features have by no means been accurately studied. It is, however, highly probable that after penetrating wounds of the brain, its substance may inflame, just as the cellular tissue of a limb may, the inflammatory processes beginning at the site of the wound, and rapidly spreading through a large part of the hemisphere. It is probably in the perivascular spaces that the process chiefly spreads, and it is in these that the microscope will detect the most abundant results. Such a condition of diffuse encephalitis may exist without there being any visible changes in the brain. It may, perhaps, be a little softened or a little congested, but very probably there is nothing about which the most experienced pathologist could feel certain until the microscope is resorted to."

"It is not possible, in the present state of our knowledge, to speak with any certainty of the symptoms of diffuse encephalitis. They will vary, of course, with the region affected; and disturbance of function, followed by, more or less, complete loss, will be the most frequent occurrences."

In the hope of learning something more definite about diffuse inflammation of the brain, we have searched through a good deal of English literature, and have collected the following cases :—

Case 1.—*Wilks and Moxon* mention, as an example of acute general cerebritis, the case of a young man, who was admitted into hospital supposed to be suffering from fever on account of the torpid state into which he had fallen ; he never spoke, but lay perfectly motionless in bed, with his eyes open, apparently watching those around him. His illness lasted a few weeks ; and after death the whole of his brain was found soft and pulpy, whilst some parts were semi-fluid.—" Pathological Anatomy," p. 237.

Case 2.—A man of 40, after frequent pain and throbbing in the L. side of the head, had convulsive paroxysms in the R. arm and leg, which were followed by giddiness, confusion of thought, and torpor of the R. side. Choreic movements were next succeeded by paralysis : speech gradually was lost, and also power over the sphincters ; but the mental faculties seemed unaffected. He often screamed, as if in pain ; and, after seven months' illness he became comatose, and died in three days.

"The L. hemisphere was soft and fluctuating throughout its whole extent, like a bag of fluid. On cutting into it there was about half-an-inch in thickness of sound cerebral substance; the remainder of the hemisphere was found nearly reduced to a fluid mass, partly consisting of purulent matter, and partly of cerebral substance in a soft pulpy state, but the greater part was purulent. From the mass of disease the ventricle was separated merely by the membrane which lines it, and contained a small quantity of serous fluid." There was a clot, the size of a walnut, in the left thalamus.—"Pathological and Practical Researches on Diseases of the Brain and Spinal Cord." 3rd edition. Abercrombie. Case 38, p. 94.

Case 3.—A complicated case. A middle-aged man one day had a fit for the first time in his life. The next day he fell in the street, and was picked up insensible, with a scalp wound. On rallying from a lengthened collapse he was very violent. For some days he suffered from intense pain in the head. On the seventh he became comatose and was trephined in consequence of an unfavourable appearance of the wound. He died the same night. *P.M.*—There was some serous effusion between the arachnoid and pia mater, especially at the base; the membranes were quite transparent; the ventricles were dilated and filled with a large quantity of fluid and the brain itself was watery throughout and soft. There was a small tubercular deposit as big as a pea, surrounded by a small area of softening and injection in one of the basal convolutions.—"Holmes' System of Surgery," vol. i., p. 623.

Case 4.—A boy, aged 13, died three weeks after he had been thrown from a donkey and fallen on the back of his head. The ventricles were enormously dilated and filled with serum and recently effused lymph, and the central white parts of the brain were very soft and broke down when slightly touched. The grey and white substance of the brain were throughout very much congested, and there was extensive inflammation of the membranes. There was a slight fissure of the occipital bone.—"Holmes' System," vol. i., p. 622.

Case 5.—A man, aged 37, died twenty-four hours after an injury of the head in a fall from a tree. The ventricles were found filled with fluid, and the fornix, remarkably soft, gave way when slightly touched. At the base of the brain around the pons and medulla oblongata ther was a large quantity of milky puriform fluid; the substance of the brain was firm, and the puncta of blood larger and more numerous than usual; the cribriform plate

of the ethmoid was broken and the corresponding part of the brain bruised.—" Holmes' System of Surgery," vol. i., 622.

Case 6.—*Wilks and Moxon* mention the case of a girl supposed to be hysterical. She was perfectly helpless, could scarcely move her arms, spoke and swallowed with difficulty, and had a stupid expression. After death the pia mater was found thickened, of a yellowish colour, and adherent to the brain, especially over the frontal lobes. There was found a general hardening of the brain, especially of the anterior lobes, which could be grasped like a hard tumour. The microscope showed the nerve fibres had almost disappeared, and their place was occupied with granule masses, vessels and fibrillated tissue.—" Pathological Anatomy," p. 238.

Case 7.—A girl of 14, in so helpless a condition of mind and body as to be at first mistaken for an idiot, had been an intelligent child a year before her death. She lay quiet in bed in a state of vacuity. At the *post-mortem* it was found she had been the subject of general meningo-cerebritis; the arachnoid was thick and opaque, and the pia adherent. The brain as a whole was much firmer than natural, and felt hard to the touch. No cause for this condition could be found.—Wilks and Moxon, " Pathol. Anat.," p. 238.

Case 8.—Dr. Bright in his medical reports relates the case of a little girl, who, for a year before her death, lay in a perfectly motionless and senseless state, with her limbs stiffly extended, and without the possibility of making the slightest movement. After death the white substance of the brain was found almost as hard as cartilage, and a stream of water washed off the grey matter, giving it the appearance of a wax model of the brain. The ventricles also looked as if they had been modelled in wax.—Wilks and Moxon, " Path. Anat." p. 238.

Case 9.—Dr. Henry Ashby records the case of a boy who died at the age of 20 months. Three days after birth he began with fits; when eight months old he was apparently an idiot, and blind and deaf, and the R. arm and leg were flexed and more or less stiff. The rigidity increased; the symptoms of descending degeneration were more marked on the right side than on the left. He lay in bed with head retracted and face turned to the L. side. At the necropsy the arachnoid was thickened and opaque, and the pia consisted of many tortuous vessels. There was no optic neuritis or atrophy. The brain substance was remarkably hard and firm, and apparently shrunken. That between the cortical grey matter and the basal ganglia was firm

and dark in colour, and evidently sclerosed. The lateral ventricles were much dilated. There were descending changes in the cord and medulla. The microscope showed the neuroglia to be exceedingly coarse and granular, the perivascular spaces dilated, and in places an increased amount of fibre tissue accompanying the vessels in their course.

Dr. Ashby says the most plausible explanation of this condition of sclerosis is that it is due to meningo-encephalitis of the convexity, which took place during fœtal life, and which was followed by atrophy of the convolutions and descending sclerosis.—*Pathological Society's Transactions*, vol. xxxvii., p. 12.[1]

These nine cases present us with two distinct forms of encephalitis. The chief pathological feature in the one is *softening*, and in the *other hardening* of the brain tissue, but in all the changes are *diffuse*.

In the cases in which there was softening (1 to 5), and in which the inflammation was more or less acute, the changes involved either the whole brain, or one hemisphere, or central softening was accompanied by increased vascularity of the remainder.

It is, further, to be noted that in one of the cases where the latter condition was found, 24 hours only had elapsed between the injury and death, and the membranes at the base were presumably affected. In the other, after three weeks, in addition to a very marked congestion of the unsoftened parts, there was extensive inflammation of the membranes.

The *duration* of the disease varied from 24 hours to a a few weeks, but in one case, with peculiar features, it was seven months.

Traumatism was the exciting cause of the inflammation in three cases (3, 4, 5,) but in only one (5), was it probable that the membranes had been penetrated, and in that the softening was found in the central parts ; and in another (3) it is uncertain what predisposing effect a small previously existing tubercular focus may have exerted.

[1] Two cases are recorded in "Fagge's Medicine," p. 554, vol. i. One is probably a fuller account of Case 7. The other is the case of an infant who had an illness of 14 months, marked by convulsions, followed by long periods of unconsciousness. The hemispheres were indurated and the pia adherent.

In the other two cases there is nothing to suggest the cause of the disease.

In case 2, though the pathological appearances were those of a diffuse encephalitis the clinical history suggests that the disease took a course very different to what occurred in the others. The way in which additional symptoms slowly made their appearance, impairment of function preceding its abolition, point to a gradual implication of portion after portion of brain until the whole hemisphere was converted into a semi-purulent mass. Such a condition from the chronicity of its course, and the formation of pus presents some likeness to a cold abscess, and it serves to illustrate the difficulty of deciding in some instances to which category, acute or chronic, localised or diffuse, a particular case is to be relegated.

In cases 6, 7, 8, 9, *there was a general hardening* of the whole brain, and in three of them, at least, the membranes had been the subject of a chronic inflammation.

The disease in all was probably identical, and the hardening due to an increase in the fibrillated tissue throughout the organ and along the course of the vessels.

In the three cases in which the *duration* is stated or can be inferred, the disease lasted from 12 to 20 months.

In no case is there any history of traumatism, and in all the cause was obscure.

The patients were all young, probably all under 20.

In Ashby's case the disease originated as an intra-uterine condition, and this is the only instance in which the records, we have had access to, note the occurrence of convulsions.

In the only case in which the state of the optic discs is mentioned it was normal, though the infant was thought to be blind.

The most prominent symptom, and one which was present in all these cases, was the *state of vacuity and helplessness* in which the patients lay for long periods. The impairment and abolition of function was evidenced in the loss of intelligence and muscular power more or less complete. We believe that *this long continued torpor of mind and body* is the most salient feature of chronic diffuse encephalitis.

Allusion has already been made to the fact that in the chronic cases with hardening, the whole brain was affected.

In the acute form, if the patient survives long enough, the same torpor may be present when the whole brain is involved—(case 1) ; but in acute cases of short duration, this valuable symptom may never develop, as signs of concussion or compression are likely to predominate from the first.

Therefore, though this condition (torpor of mind and body) may be present in acute diffuse encephalitis, it is more especially characteristic of the chronic form in which induration is the chief pathological feature, and it is, then, usually of long duration.

The symptoms due to secondary degeneration in the cord call for no remark.