HYDROCEPHALUS COMPLICATING CEREBRO-SPINAL FEVER, AND ITS TREATMENT.

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In cerebrospinal fever two types of hydrocephalus are met with: (1) Generalized Hydrocephalus; (2) Internal Hydrocephalus.

I. Generalized Hydrocephalus.—Generalized hydrocephalus is that type in which an increased amount of cerebrospinal fluid is present throughout the subarachnoid space, both in the ventricles and externally, without obstruction of the foramina of Magendie and Luschka.

In addition to that occurring early in the disease, generalized hydrocephalus may develop at any time subsequent to the first week. The characteristic symptoms are practically those of internal hydrocephalus but in a somewhat less severe form—headache, lethargy, vomiting, tremulousness, and dilated pupils; the temperature usually rises, but may remain at its previous level. When there is a considerable accumulation of cerebrospinal fluid under increased pressure throughout the subarachnoid space, and no meningeal adhesions interrupt the continuity of the latter, lumbar puncture yields a large quantity of cerebrospinal fluid. The evacuation of this affords considerable relief, but it may be necessary to repeat the operation every twelve hours before the hydrocephalus has definitely passed off. In other instances, however, in spite of repeated lumbar puncture, death may result from hydrocephalus, generalized as regards the cranial cavity, and due to a blockage occurring either at the foramen magnum or in the spinal canal above the region of lumbar puncture. For
example, the following case proved fatal owing to fibrinopurulent exudate producing almost complete obstruction at the upper extremity of the spinal theca, in the region of the foramen magnum.

Case 1.—Generalized Hydrocephalus of the Cranial Subarachnoid Space, due to Obstruction at the Foramen Magnum.—For a month prior to coming under our observation, the patient, age 18, had been in hospital elsewhere suffering from pyrexia of obscure origin which was apparently ascribed to 'rheumatism'. The history given by the patient was that he complained of severe headache and 'pain all over'. The only record of his illness obtainable was the temperature chart, which showed an irregularly intermittent pyrexia varying between subnormal and 103°—extremely suggestive of cerebrospinal fever. The rises in temperature had persisted for sixteen days; following this the pyrexia subsided. Throughout this period the pulse-rate had remained below 96 per minute, varying between this figure and 60. The patient had then been kept in bed for a further five days (21st day of illness), and was finally discharged from hospital eleven days later (32nd day).

The patient stated that on leaving the hospital he still felt far from well. Two days later he commenced shivering, complained of headache during the afternoon, and vomited in the evening. He was then sent to hospital, being admitted as a case of influenza. No improvement in his condition was noticed, however, and two days later (3rd day of 'second attack' and 38th day of total illness) he came under my observation. The temperature was then 101°, pulse 80, and all the usual signs of meningitis were well marked; he answered questions well, but at times was inclined to become incoherent. Lumbar puncture yielded a slightly turbid fluid containing meningococci.

Daily lumbar puncture and serum administration were instituted, fair quantities (40 to 60 c.c.) of cerebrospinal fluid being obtained; the colour of the latter, however, tended to become an increasingly deep yellow (xanthochromia). For the first two days little or no change occurred in the patient's general condition. His subsequent progress was as follows:—

Sixth day (41st day of total illness).—The patient was distinctly delirious and exhibited incontinence of urine. On being roused, however, he spoke fairly rationally, stating that he had absolutely no headache. As he was inclined to be tremulous, hydrocephalus was feared to be developing; lumbar puncture was therefore performed both morning and evening.
11 a.m. L. puncture: 60 c.c. Slightly turbid yellowish fluid.
11 p.m. L. puncture: 60 c.c. Slightly turbid yellowish fluid.
(No increased pressure was apparent.)

Seventh day (42nd day of total illness).—Delirium persisted, but incontinence was absent. Occasional floccitation was present. Profuse sweating was a marked feature, being so intense as to necessitate changing the bedclothes several times. The pupils were dilated and the arms tremulous.
11 a.m. L. puncture: 60 c.c. Slightly turbid yellowish fluid.
11 p.m. L. puncture: 40 c.c. Slightly turbid yellowish fluid.
The fluid trickled only very slowly through the needle; on each occasion the needle was withdrawn and re-inserted in a different interspace, with no better result. 30 c.c. of antimeningococcal serum were administered at the morning puncture.
Eight day (43rd day of total illness).—Dellirium increased, and incontinence again made its appearance. No sweating occurred, but the pupils were very dilated and tremulousness was pronounced. Definite strabismus was observed.

10 a.m. L. puncture: 10 c.c. Deep yellow fluid.
10 p.m. L. puncture: 5 c.c. Deep yellow fluid.

On each occasion the fluid was obtained only with difficulty; several interspaces were entered, the flow being very slow.

Ninth day (44th day of total illness).—The patient died somewhat suddenly during the early hours of the morning.

Autopsy.—The dura mater was found tense, and on incising it a large quantity of slightly turbid fluid escaped. Over the convexity of the cerebrum there was practically no meningitis. At the base, however, tough fibrinopurulent exudate was found over the pons, medulla, and cerebellum, extending as far forwards as the optic chiasma. The exudate also extended downwards to the cervical region of the spinal cord, the spinal canal at the foramen magnum being almost completely occluded. The lateral ventricles were only moderately distended with slightly turbid fluid.

Occasionally, adhesions between the parietal and visceral arachnoid of the spinal cord may bring about a generalized hydrocephalus similar to that described above. In a case mentioned by Foster and Gaskell death resulted from this cause.

In chronic cases of cerebrospinal fever, generalized hydrocephalus is occasionally found to persist even after apparent recovery from meningitis. This result, no doubt, is due to organized exudate and adhesions of the pia-arachnoid diminishing the total capacity of the subarachnoid space, and also, possibly, to some extent limiting the absorption of cerebrospinal fluid. Cases, usually in children, may come under observation and, on examination, suggest a condition of hydrocephalus. A history may be obtained of an obscure illness, accompanied by headache and vomiting, some weeks or even months previously, from which the patient was supposed to have recovered. On lumbar puncture a clear fluid will usually be obtained. Such a condition is really a chronic meningitis following an acute attack of cerebrospinal fever from which the patient merely appears to have recovered; some of the so-called relapsing cases are probably of this nature.

II. Internal Hydrocephalus.—Under normal conditions, the cerebrospinal fluid secreted by the choroid plexus into the lateral ventricles passes from there into the fourth ventricle, and thence to the subarachnoid space through the median foramen of Magendie and the two lateral foramina of Key and Retzius or Luschka. According to Merkel, the lateral ventricles also communicate with the subarachnoid space at the apices of their descending horns.

When, owing to inflammatory changes, complete or partial occlusion of the foramina of Magendie and Luschka occurs, the
cerebrospinal fluid, being unable to escape through its usual channels, accumulates in excess throughout the ventricular system. Consequently, since normal absorption is only possible from the subarachnoid space, a condition of dilatation of the ventricles results.

Internal hydrocephalus is not invariably due to the mechanical closure of the foramina of Magendie and Luschka. It may arise, even in posterior basic types, without occlusion of these orifices, owing to the lack of mechanical resistance offered by the ventricular walls to the total increase of fluid and internal pressure. The softening of the tissue surrounding the ventricles by oedema, and often by actual inflammation, facilitates such a procedure to a considerable extent. This factor, however, is probably of greater importance in patients where hydrocephalus occurs comparatively early in the course rather than in chronic cases.

Dandy and Blackpan have shown clinically that phenolsulphophthalein, when injected intraventricularly, is excreted in the urine in small amounts where internal hydrocephalus exists. Experimentally, the same observers have produced internal hydrocephalus by (1) the mechanical blockage of the aqueduct of Sylvius, and (2) low ligation of the vena Galeni magna; high ligation had no such effect. Clinically, therefore, it is possible for either blockage of the Sylvian aqueduct by purulent exudate, or thrombosis of the vena Galeni, to produce internal hydrocephalus; as far as I am aware, however, the latter condition, originally suggested by Carr in 1897, has not been demonstrated at autopsy, but Foster and Gaskell mention one case exhibiting, on post-mortem examination, well-marked hydrocephalus of the lateral and third ventricles, the iter being completely blocked.

Most authorities consider that internal hydrocephalus, once definitely established, is invariably fatal, apart from operative procedures. When the occlusion of the ventricular outlets is complete and permanent, death of course would be the only possible termination. Naturally, a diagnosis of internal hydrocephalus is always open to suspicion in cases recovering, as the condition is not confirmed by autopsy. Nevertheless, a case may present all the typical signs of internal hydrocephalus, including ‘dry punctures’, and yet recover. Post mortem, as already mentioned, complete obliteration of the foramina is rarely found. It is fair to assume, therefore, that in the majority of cases of internal hydrocephalus, the interference with the outlets stops just short of complete occlusion. Early recognition of hydrocephalic symptoms and treatment by repeated lumbar puncture may often prevent the hydrocephalus from becoming entirely internal. As Foster and Gaskell point out, the effect of the puncture may gradually lessen the maximum tension below a
certain critical point at which normal drainage is able to take place. Following a period of serum administration, treatment by repeated daily lumbar puncture, as described elsewhere, tends to avert the development of internal hydrocephalus. When, however, the amount of cerebrospinal fluid obtained progressively diminishes and reaches the stage of 'dry punctures', other methods of treatment, which will be described later, have to be considered.

**TREATMENT.**—The generalized hydrocephalus occurring in the early stages of meningitis, which is responsible for such symptoms as headache, vomiting, stupor, and, in children, bulging of the anterior fontanelle, is relieved by the lumbar puncture preliminary to the first intrathecal injection of serum. As much cerebrospinal fluid as possible should be withdrawn. The importance of the early recognition of hydrocephalic symptoms developing later in the course cannot be sufficiently insisted upon. Speedy and repeated lumbar puncture, performed twice daily if necessary, will often avert a chronic condition. Continued daily lumbar puncture, following the period of serum administration, largely counteracts any tendency towards the development of hydrocephalus; nevertheless, symptoms of this complication occasionally occur. Continued lumbar puncture, however, will often bring about complete relief, as, for example, in the following case:

**Case 2.**—A subacute case, age 29, and not received until the fourteenth day of illness, was given antimeningococcal serum in 30-c.c. doses each day for eight days. For two days following this, only small amounts (5 to 10 c.c.) of cerebrospinal fluid were obtainable on lumbar puncture; as a result, the patient developed symptoms of hydrocephalus—apathy, vomiting, incontinence, tremulousness, dilated pupils, and marked head retraction. No organisms were visible in the cerebrospinal fluid or obtainable on culture. With repeated daily lumbar puncture, however, larger quantities of fluid (50 to 70 c.c.) were gradually obtained, and the hydrocephalic symptoms disappeared. The patient was punctured on nine successive days, following the termination of serum administration, before a perfectly clear cerebrospinal fluid was withdrawn. The total course of illness amounted to thirty days.

In a few cases symptoms of hydrocephalus may appear in spite of the fact that fair quantities of cerebrospinal fluid are withdrawn daily. When this occurs, lumbar puncture should be performed both morning and evening until the hydrocephalic symptoms have disappeared; if the cerebrospinal fluid is only slightly turbid and no organisms have reappeared, serum administration need not necessarily be resumed.

Occasionally a hydrocephalic case of chronic meningitis may come under observation in which it is impossible to find any organisms in the cerebrospinal fluid, either on direct examination or on culture.
The fluid may be clear to the naked eye, and the cytological charac-
ters resemble those found in tuberculous meningitis, viz., the number
of mononuclear cells far exceeding that of the polymorphonuclears.
If the case, for some reason or other, does not appear to be typical
of tuberculous meningitis, e.g., owing to the history of onset or the
appearance of well-marked muscular rigidities, the exhibition of anti-
meningoenceal serum, administered intrathecally for twelve daily doses,
followed by continued lumbar puncture for some days, is well worthy
of trial. I have met with two such examples. One case of chronic
meningitis, that of a man, age 35, who had been given a practically
hopeless prognosis, made a complete recovery, and has been recorded
elsewhere. 6

The details of the second case are as follows:—

Case 3.—History prior to Admission.—The patient, a man, age
39, was first seen on May 5. The history was that during the previous
February he had a bad attack of influenza, and after about four weeks
was able to get up and go about in a very listless fashion. During April
he became steadily worse, being apathetic and at times losing consciousness.
He had in turn been regarded as a case of encephalitis lethargica,
cerebrospinal syphilis, and (?) tuberculous meningitis.

Condition on Admission.—The patient was profoundly emaciated
and appeared very deaf; he was apathetic, but resentful of interference,
and answered questions incoherently. The limbs were kept in an attitude
of flexion, and he exhibited general hyperesthesia. Temperature, 99·6°;
pulse-rate 82. Pupils were dilated, but reacted sluggishly to light; beyond
the deafness, the other cranial nerves were normal. Neck rigidity well
marked, but without definite occipital retraction. Kernig's sign was posi-
tive on the right side but negative on left; on this latter side, however,
there was pronounced rigidity of the iliopsoas muscle. Both knee-jerks
brisk; right ankle-jerk brisk, left sluggish. The plantar reflexes were
flexor, and the abdominals sluggish but equal. Incontinence of urine and
feces.

On lumbar puncture, the cerebrospinal fluid was under increased
pressure, yellowish in colour, and, on examination by Dr. Braxton Hicks,
exhibited 1100 cells per c.mm., 50 per cent being polymorphonuclears and
50 per cent mononuclears. The globulin reaction was markedly positive,
and the Wassermann and glucose reactions were negative; no organisms
were visible on microscopical examination, and cultures remained sterile.

Treatment and Progress.—The patient was given ten consecutive
daily doses of antimeningeal serum (Types I, II, and III pooled) and
polyvalent vaccine, in increasing doses from 250 million to 2500 million
organisms, every four days. The cerebrospinal fluid examined on the
tenth day of treatment showed only 112 lymphocytes per c.mm. Follow-
ing the cessation of serum administration, lumbar puncture was resumed
daily for ten further days. The patient rapidly put on flesh, sphincteric
control was regained, neck rigidity gradually diminished, and Kernig's sign
became negative. By May 26, the day following the last lumbar puncture
and that on which the last dose of vaccine (2500 million) was adminis-
tered, the deafness had greatly improved; the patient's mental condition
was normal, and physical signs were practically absent. He got up from

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bed on June 12, and on June 28, a few days before leaving hospital, lumbar puncture was performed and the cerebrospinal fluid examined, with the following result: Fluid clear to the naked eye, 5 lymphocytes per c.mm., glucose reaction positive, globulin negative, cultures sterile.

If symptoms of internal hydrocephalus appear—lethargy, incontinence, persistent vomiting, tremulousness, general hyperesthesia, dilated pupils, and possibly nystagmus—and no fluid can be obtained on lumbar puncture, treatment can only be effective if directed to the drainage of the subarachnoid space above the site of obstruction. A 'dry tap' on lumbar puncture should not be diagnosed unless the spinal canal has been entered in at least three different intervertebral spaces. The obstruction may occur in the dorsal region of the spinal canal, the upper portion of the spinal subarachnoid space being thus shut off from the lower; under these circumstances, dorsal puncture may be successful, but if no fluid is obtained, cervical puncture should be tried. Unfortunately, above the level of the 11th dorsal vertebra there can be no absolute certainty of the presence of a posterior subarachnoid space. In a series of 11 dissections, Lusk found that in only 3 was there present a complete posterior subarachnoid space above the level of the conus medullaris; even in these 3 instances interrupting transverse septa occurred at intervals, so that the posterior channel was not continuous. Free circulation of cerebrospinal fluid, however, was allowed by means of the lateral communications with the anterior part of the space through the ligamentum denticulatum. In the remaining 8 dissections, adhesions existed at various sites between the spinal cord and the posterior portions of the arachnoid. The adhesions extended downwards to a distance varying from the level of the 5th thoracic vertebra to two inches above the conus medullaris.

It will be seen, therefore, that even if the obstruction be present in the spinal canal, dorsal or cervical puncture will not necessarily give the desired result. Cervical puncture is usually more successful than dorsal. The technique is as follows: The patient is maintained in the left lateral position, with rounded spine, and the interspace between the 6th and 7th cervical vertebrae is defined; in the case of dorsal puncture, one of the mid-dorsal interspaces is selected. The needle is then directed in the mid-line from below upwards, following the axis of the interspinous spaces. As soon as the skin is penetrated, the stylet is removed from the needle in order that the cerebrospinal fluid can escape directly the point has reached the subarachnoid space, injury to the spinal cord thus being avoided.

Cases in which cervical puncture was performed and serum injected in the same situation, followed by recovery, have been reported by Cantas and Gerard. Ravaut and Kranlitski applied
the same methods in suitable cases, but experienced difficulty in withdrawing a sufficient quantity of cerebrospinal fluid from the dorsal region; also, each fresh injection of serum was followed by epileptiform seizures. In the cervical region, however, fluid was readily obtained.

The following example illustrates a case of my own in which cervical puncture and the injection of serum was successful, the patient recovering:—

Case 4.—The patient, age 27, was taken ill suddenly with headache, a feeling of soreness in the throat, and vomiting. He was admitted to hospital, but apparently showed some improvement by the following day, as it was considered that he was suffering merely from ‘tonsillitis’. He remained in bed, however, and continued to complain of headache and general malaise; a record of the temperature and pulse-rate during the earlier period was unobtainable. Seventeen days after his admission to hospital he suddenly became very restless, and within a few hours was profoundly delirious; it was then that the case came under our observation. On examination the condition was as follows:—

Mental condition one of stupor with occasional delirium. Temperature 102.8°F, pulse-rate 92, respirations 36. Pupils somewhat dilated, and reacting sluggishly to light. Well-marked neck rigidity, with slight occipital retraction; Kernig’s sign positive; knee- and ankle-jerks slight, and abdominal very sluggish.

Lumbar puncture was performed, and 50 c.c. of turbid cerebrospinal fluid evacuated; 30 c.c. of polyvalent antimeningococcal serum were at once injected intrathecally. It was considered that the case was one of cerebrospinal fever of the recrudescent type, the day of the recrudescence being the 17th day of total illness. Subsequent examination of the turbid cerebrospinal fluid removed, showed the presence of numerous polymorphonuclear leucocytes, and both intracellular and extracellular meningococci. Cultivations yielded a meningocoecus corresponding with Gordon’s Type III.

The daily intrathecal injection of serum was continued, and the patient progressed satisfactorily up till the fifth day of treatment (21st day of total course), when great difficulty was experienced in obtaining fluid by lumbar puncture, no less than three interspaces being entered before 18 c.c. could be removed; this quantity was replaced by an equivalent amount of Type III serum (as by this time the type had been identified).

On the following two days (22nd and 23rd days of illness and sixth and seventh days of treatment) the patient was inclined to be delirious, and exhibited floecitation and carphology; on lumbar puncture, cerebrospinal fluid escaped very slowly, drop by drop. On the 24th day (eighth day of treatment) the patient was considerably worse, being quite delirious and exhibiting incontinence of urine and faeces. No fluid could be obtained on lumbar puncture (three interspaces being entered) or dorsal puncture (two interspaces). Consequently, cervical puncture was resorted to and 40 c.c. of slightly turbid yellowish fluid were easily removed; 80 c.c. of Type III serum were injected very slowly by the gravity method. Next day (25th) the patient was somewhat better, being less delirious and not exhibiting carphology. Cervical puncture was again performed, and 30 c.c. of serum injected. Both this fluid and that of the previous day showed a
few meningococci. On the 26th day the patient was considerably better, answering questions fairly well, exhibiting slight delirium only at intervals, and incontinence was absent. Ten c.c. of fluid were obtained on lumbar puncture, but as this quantity was considered insufficient, cervical puncture was performed, resulting in the removal of 40 c.c. further of fluid; 30 c.c. of serum were injected in the same situation. Neither sample of fluid showed meningococci, either on direct examination or in culture. On the following day (27th day of illness and eleventh day of treatment) 35 c.c. of slightly turbid yellowish fluid were obtained on lumbar puncture but no serum was injected; next day (28th) the patient’s mental condition, was apparently normal, and 35 c.c. were again removed by the lumbar route. From this time onwards the patient rapidly improved, the temperature reaching normal on the 31st day; lumbar puncture was repeated daily up till the 33rd day (seventeenth day of treatment and seventh day after cessation of serum administration), when the cerebrospinal fluid was quite clear and all symptoms were absent.

Vaccine was administered from the third day of treatment (19th day of illness) every fourth day in increasing doses from 500 million organisms to 2500 million; at the third dose, polyvalent vaccine was replaced by Type III vaccine.

In the above case, it is probable that inflammatory adhesions were forming in the spinal subarachnoid space in the dorsal region; the intrathecal injection of serum apparently led to the inhibition of the adhesion formation and the ultimate subsidence of the inflammatory changes.

If lumbar, dorsal, and cervical punctures are all unsuccessful in yielding cerebrospinal fluid, it is still possible that generalized hydrocephalus exists, obstruction having occurred at the foramen magnum owing to the accumulation of fibrinopurulent exudate (e.g., Case 1). Consequently, before proceeding to tap the lateral ventricles, sphenoidal puncture, after the method of Bériel and Cazamian, should be tried. The technique is as follows:—

A pointed needle, fitted with stylet (e.g., an ordinary lumbar-puncture needle), is introduced at a point 2 mm. external to the supraorbital notch; it is pushed slightly upwards and inwards to reach the bony vault of the orbit. The sharp stylet is now withdrawn from the needle, and replaced by a blunt one reaching just beyond the point of the needle; with a little manipulation the most external portion of the sphenoidal fissure is reached and a fibrous membrane pierced with the characteristic sense of resistance. The stylet is then withdrawn, and cerebrospinal fluid escapes if the hydrocephalus be generalized in the cranial cavity. After removal of a sufficient quantity of cerebrospinal fluid, antimeningococcical serum may be slowly injected by the gravitation method. Cazamian applied this method of serum injection to three cases, two of which were in extremis and proved fatal; the third case, however, recovered.

When the above methods of puncture fail to yield cerebrospinal
fluid in the presence of hydrocephalic symptoms, the only treatment that can be successful is drainage of the ventricles. In infants, the puncture is easily performed through the anterior fontanelle, the method being as follows:

The upper part of the scalp having been shaved and the skin sterilized, the anterior fontanelle is defined and an ordinary lumbar-puncture needle with stylet placed at the lateral angle—that is, about 1 in. (2 cm.) from the mid-line. The needle is pointed in a direction downwards, slightly backwards and inwards, and is pushed in to a depth of about 1 ½ in. (3 cm.). When the ventricles are very dilated and the cerebral cortex thinned, the needle entering in almost any direction will usually strike fluid.

The operation is usually well borne, and the amount of shock small. There are two possible dangers—injury to vital centres, and hæmorrhage. The risk, however, is very slight, and the gravity of the condition warrants the procedure.

At the first operation cerebrospinal fluid should merely be withdrawn. If this fluid reveals no meningoeocci either on direct examination or culture, subsequent punctures should consist in the simple removal of fluid. If, however, the fluid proves to be infected, serum should be injected, the administration being carried out in the same way as in intraspinal injection and the same precautions observed. The amount of fluid withdrawn should, of course, be considerably less than the quantity of serum administered. Both ventricles are tapped, preferably on alternate days; if puncture be confined to one ventricle, the drainage of the other is incomplete.

In older children and adults, trephining is necessary. Two methods are available:

1. Keen's Method.—The trephine opening is made at a point 1 ½ in. (3 cm.) above the external auditory meatus; this site corresponds with the posterior end of the temporal line, and is known as Keen's point. In performing the puncture, the needle is directed towards the upper limit of the opposite pinna. At a depth of about 2 in. (5 cm.), the lateral ventricle will be entered at its widest part, that is, where the lateral and posterior horns are given off from the body at the posterior end of the optic thalamus. The most dependent part of the ventricle is tapped by this method, thus affording more adequate drainage than if it were entered from above.

W. J. Denehy found that, in nine cases coming to autopsy, the lateral ventricles having previously been tapped by Keen's method, no damage to the brain substance had occurred. In each case the puncture pierced the most dependent part of the lateral ventricle. The slightly blood-stained needle track was easily identified, but even in the case of multiple puncture there was no serious damage
to the brain. In one case a blood-clot lay extradurally immediately within the skull, due to the accidental rupture of a vessel.

2. Kocher's Method.—A point 1 in. (2.5 cm.) from the mid-line and about 1½ inches (4 cms.) anterior to the bregma, is taken as the situation for performing the trephine. In puncturing, the needle should be directed downwards and backwards; the ventricle will be reached at a depth of 1½ to 2 in. There is practically no risk of hemorrhage during the passage of the needle.

More recently, instead of trephining, I merely drilled a hole through the skull over Keen’s point and punctured the ventricle through the small opening thus made. It is possible to perform the operation under local anaesthesia, the ‘shock’ being negligible compared with that resulting from a trephine, and drainage appearing equally effective. The only risk is that of damaging one of the cortical vessels.

Whichever of the above methods be adopted, one side should be operated upon first and the other subsequently. When internal hydrocephalus is present, the dura is found tense and non-pulsating. On withdrawing the stylet from the puncture needle, the fluid usually appears with force and should be allowed to escape until the flow ceases.

Stetten and Roberts advocate incision of the corpus callosum, and the production of a wide opening which allows of prolonged drainage; a successful case is recorded.

Unfortunately, it cannot be claimed that ventricular puncture in internal hydrocephalus of adults is a striking success, since re-recoveries are relatively few. Nevertheless, excepting in very rare cases, the operation offers the only chance of avoiding a fatal issue.

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