NON-TUBERCULOUS POSTERIOR BASIC MENINGITIS IN INFANTS

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So large a proportion of all the cases of meningitis in children are tubercular that we are apt almost to ignore the existence of other forms; nor is this so surprising when we find that, whereas the tubercular disease is one running a tolerably definite course, and having a very well defined pathological anatomy, the other and rarer forms of meningitis present the greatest diversity, alike in course, symptoms, and morbid appearances. Some cases are very acute, lasting only a few days; others extremely chronic. In some the vertex is mainly affected, in some the base, whilst in others the affection is uniformly distributed over the whole of the brain and perhaps the spinal cord also. Even more diverse is the reputed causation of these varied conditions—injury, ear disease, rheumatism, exposure to the sun, congenital syphilis, the acute specific fevers, influenza, pneumonia, septic conditions, &c., are all assigned as possible causes, and even then some cases remain which we have to be content to term idiopathic. Amid this chaos I wish to single out a

certain number of cases which present a fairly definite course and symptomatology, and tolerably uniform pathological changes, and which are therefore probably due to some one definite cause, though this as yet eludes our observation. The cases referred to are those of non-tubercular meningitis, more or less chronic in character, and affecting a definite region at the base of the brain in young children.

So far as I know, this disease has only been at all fully described previously in a paper contributed by Dr. Gee and Dr. Barlow to the 'St. Bartholomew's Hospital Reports' for 1878 (vol. xiv), entitled "The Cervical Opisthotonos of Infants;" but, as the title of the paper indicates, they regarded the condition from the standpoint rather of the chief symptom, the opisthotonos, and described twenty-five cases in which this was present, although in all those in which an autopsy was obtained basic meningitis was found. Several of their cases were seen only once or twice as out-patients, and the ultimate result was not ascertained. On the other hand, I wish, if possible, to describe this basic meningitis as a definite pathological condition, with more or less characteristic symptoms and course, basing the description on a few cases under observation in hospital, and followed out to an autopsy.

The cases to be recorded—except perhaps No. 11—may, I think, be regarded as fairly typical ones; others showing less characteristic symptoms or less definite morbid changes have been met with, but, in describing a comparatively rare and not very commonly recognised condition, it seems well to limit our attention first to the typical cases, and once having noted the usual manifestations we are in a better position for dealing with less definite examples of the disease. Such irregular, atypical, and border-line cases are sure to be met with,—in what disease do they not occur? Little good, however, is, I think, to be gained from dwelling upon them at first, although they may prove interesting later, for we have to begin by establishing, if possible, the identity of the disease, then to ascertain its relations with other

morbid conditions, and afterwards to learn how to recognise it in its less characteristic manifestations.

All the cases occurred in the Victoria Hospital for Children, Chelsea. I am indebted to my colleagues Dr. Ridge-Jones and Dr. Drewitt for the use of the notes of the cases during life, and to Mr. Holmes Spicer, ophthalmic surgeon to the Hospital, for most of the reports upon the ophthalmoscopic appearances.

Both the clinical and pathological records of the later cases are more complete than those of the earlier ones; this is owing to the fact that it was not till I had met with several examples of the disease that I became specially interested in it, and so led more fully to investigate the symptoms and morbid changes.

[In each case the age given is that of the child when admitted to the hospital.]

CASE 1.—Sidney K—, aged 8 months, admitted March 5th, 1890. Six other children living and well; one miscarriage. No previous illness, but said to have had snuffles. Fed on breast only. Stated to have been ill since a fall on the top of the head on December 20th, 1889; had convulsions and vomiting next day, and occasional convulsive attacks after, but had had none for a fortnight until the day before admission, when he had a severe attack followed by sickness. On admission, head greatly retracted; fontanelle rather bulged; slight rigidity of limbs and twitchings of face; occasional squint; pupils equal; pulse about 100, rather irregular; severe vomiting; bowels open. Temperature on admission 96° , rose to 102° , but fell again before death, which, without any change in symptoms, occurred on March 8th.

Autopsy (five hours after death).—Body extremely emaciated; slight beading of ribs, and very slight craniotabes in cerebellar fossæ. Brain: convolutions much flattened, dry, and bloodless. At the base all the structures from the posterior border of the cerebellum to the optic commissure firmly matted together by a very thick layer of

lymph; also much matting together in both Sylvian fissures. Lateral and third ventricles and aqueduct of Sylvius enormously distended with fluid, but showed no sign of inflammation. Brain substance almost diffuent. Left middle ear healthy, right full of inodorous pus; membrana tympani intact. Cranial bones and dura mater healthy. No thrombosis in sinuses. Other organs normal; no sign of tubercle anywhere.

CASE 2.—Alfred S—, aged 5 months, admitted May 10th, 1890. Fourth child. No evidence of syphilis in family history or in child's condition. Fed on breast only. Quite well till April 20th, when taken ill suddenly; said to have become unconscious and motionless, then constantly sick for two or three days, and had sudden screaming fits. For about a fortnight before admission had head retraction and convulsions every day. On admission, head retracted; anterior fontanelle bulging; pupils equal; slight squint; pulse 108, regular; sighing breathing; food taken well, no vomiting. Whilst in the hospital there was excessive head retraction and some general opisthotonos; anterior fontanelle tense; little or no rigidity of limbs, but twitchings and slight convulsions occasionally; squint and nystagmus at times; pulse rapid (140 to 160) and regular; breathing sometimes irregular; sickness most days; bowels open regularly; no optic neuritis. Temperature normal, save for an occasional evening rise to 100° , but during the last two days of life it rose steadily and reached 105.8° at death, which occurred on May 25th. The head retraction and the rigidity of the neck lasted about one hour after death. Total duration of illness thirty-five days.

Autopsy (twenty-five hours after death).—Body considerably emaciated; very slight cranio-tabes in cerebellar fossæ, but no other sign of rickets or of syphilis. Brain: convolutions flattened, and cortical veins rather congested; pia mater at base thickened, with matting together of parts over the central portion of the inferior surface of

the cerebellum, and over the medulla, pons, and base of the cerebrum, as far forwards as the optic commissure; also over the tip of the left temporo-sphenoidal lobe, where there was a thin layer of puriform lymph. The fold of pia mater closing the fourth ventricle between the medulla and cerebellum thickened to form a firm membrane. Lateral ventricles dilated to form two large bags filled with turbid fluid containing flakes of lymph; fourth ventricle also much dilated. Brain substance almost diffuent. Middle ears and cranial bones healthy. Other organs normal, except for a cyst in the right kidney; no sign of tubercle anywhere.

CASE 3.—Emma H—, aged 7 months, admitted November 17th, 1890. First child, no miscarriages. No previous illness; said to have had snuffles soon after birth, but no rash. Fed on breast only. Five weeks before admission had diarrhoea for a week, and a week later became dull and restless when asleep; no vomiting. Convulsions, preceded by screaming for a day or two, set in twenty-five days before admission, right side most affected; increasing retraction of head noticed after first fit. On admission, head markedly retracted; frequent slight twitchings of face; no rigidity of limbs, but hands closed; right internal squint; pupils equal; no optic neuritis. In hospital there was extreme retraction of head throughout; no note of fontanelle; no convulsions, and only very slight rigidity of limbs, except occasionally of the forearms; chewing movements, yawning, and sighing noted; pulse rapid (130 to 170), only once or twice noted to be slightly irregular; Cheyne-Stokes breathing; frequent vomiting; bowels relaxed. Temperature normal, except for one rise to 101.4° , but rose steadily before death on December 1st to 106.4° . Duration of symptoms five to six weeks.

Autopsy (thirty hours after death).—Body fairly nourished, no rickets. Brain: convolutions much flattened, dry, and bloodless. A thick layer of lymph extended over the base of the brain as far forwards as the optic

commissure, with matting together of all the parts there and along the Sylvian fissures ; lymph thickest over the pons. Thickening and opacity of the pia mater over the central part of the under surface of the cerebellum, ending on each side in a sharply-defined line. Isolated patches of lymph on the tip of each temporo-sphenoidal lobe, and a small patch also on the tip of each frontal lobe. The changes on the two sides very symmetrical. A few small isolated patches of lymph on the vertex, chiefly along the branches of the middle cerebral arteries. Pia mater closing fourth ventricle thickened, and foramen of Majendie apparently obliterated. Lateral and third ventricles and foramen of Munro much dilated, contained a few flakes of lymph (one large one on choroid plexus), and about 7 ounces of slightly turbid fluid, sp. gr. 1013, which did not coagulate spontaneously, but gave a large deposit of albumen on boiling, and contained abundant chlorides but no copper-reducing substance. Brain substance very soft. Right middle ear healthy, left contained a little non-fœtid, semi-purulent mucus. No disease of cranial bones ; no thrombosis of sinuses. Upper half of spinal cord almost diffuent, and protruded when dura mater was cut through ; lower half appeared healthy. Other organs normal ; no sign of tubercle anywhere.

CASE 4.—Alice O—, aged 3 months, admitted September 27th, 1892. Second child ; elder one healthy ; no previous illness ; no evidence of syphilis. Fed on breast only. Brought to the out-patient department on September 6th for sickness and screaming.

September 13th.—Marked head retraction.

20th.—Head retraction very marked ; constant sharp cry.

27th.—Symptoms as before. Child hardly able to take breast ; cries when head is moved.

On admission, fairly nourished ; extreme retraction of head ; fontanelle normal ; pupils equal ; frequent sharp cry ; fed with difficulty. In hospital marked bulging of the

fontanelle developed, and coincidently with this the head retraction diminished; no convulsions; some strabismus; no note of pulse; frequent vomiting; considerable diarrhœa, perhaps due to treatment by calomel. The child had to be fed by nasal tube. The optic discs were quite normal two days before death. Temperature normal except for an occasional rise to 100° or 101° ; no ante-mortem rise. Death on October 12th. Duration of illness about six weeks.

Autopsy (twenty-five hours after death).—Body much wasted; no rickets. Examination of head only permitted. Brain: convolutions pale and much flattened. A thick layer of purulent lymph covered the medulla, pons, and under surface of the cerebellum; considerable thickening round the optic commissure, but very little lymph and no distinct adhesions in the Sylvian fissures; a very thin layer of lymph over the tip of each temporo-sphenoidal lobe, and on the under surface of the frontal lobes. The fold of pia mater closing the fourth ventricle was greatly thickened, but presented in the middle line a circular aperture a quarter of an inch in diameter, with a well-defined edge. The lymph did not extend down the cord. Lateral and third ventricles and aqueduct of Sylvius greatly dilated; contained about 6 ounces of clear fluid, sp. gr. 1010. Choroid plexuses normal; many dilated veins on walls of lateral ventricles. Brain substance rather soft. Middle ears and cranial bones healthy. No tubercles seen.

CASE 5.—William N—, aged 9 months, admitted May 16th, 1892. Five other children; two living, three said to have died in infancy from convulsions. Child fed on breast only, and said to have been quite well till April 10th, when it was taken to a medical man for “a cold on the chest,” and he noticed bulging of the fontanelle. Three days later patient had a convulsion, and two more shortly after; vomiting, head retraction, and rapid wasting supervened. On admission there was marked head retraction; circumference of head $18\frac{1}{2}$ inches; no optic neuritis. In

hospital the head retraction continued to be extreme, and the fontanelle became increasingly tense; no convulsions; nystagmus and squint were present. Pulse 100 to 120, regular; vomiting frequent; bowels freely open; temperature normal. On account of the extreme bulging of the fontanelle the lateral ventricles were tapped six times between June 6th and August 27th by a Southey's tube introduced at the side of the fontanelle, from 2 to 5 ounces of fluid being withdrawn each time, 20 ounces in all. The fluid was perfectly clear and watery in appearance, sp. gr. 1004 to 1006, contained a very faint trace of albumen, abundant chlorides, no copper-reducing substance. After the first tapping, when 4 ounces were withdrawn, the temperature rose to $100\cdot6^{\circ}$, but on no subsequent occasion was there any rise, and no ill effects resulted, even the pulse being unaffected. Attempts to apply pressure had to be abandoned owing to rapid ulceration of the scalp. Despite the tapplings the head steadily got larger, the circumference increased to 20 inches, and the interparietal suture opened throughout its entire length; nevertheless the child's condition began slowly to improve, in three months (June 29th to October 2nd) the weight increased from 12 lbs. to 15 lbs. 10 oz., the vomiting ceased, and by the beginning of August there was hardly any head retraction and no nystagmus; the child seemed more lively and took food better; bowels still rather loose; temperature normal. Iodide and bromide of potassium, a grain of each, were given three times a day.

All symptoms of active meningitis having subsided, whilst the hydrocephalus continued to increase, and would evidently in time cause permanent atrophy of the brain, the question of operative interference was considered. On the assumption that the hydrocephalus might be due to a blockage of the cerebro-spinal aperture by thickening of the pia mater between the medulla and cerebellum, a sequel to basic meningitis, it was thought that if a small opening could be re-established in this membrane the condition might possibly be relieved, especially as all active

inflammatory mischief seemed to have passed off. Accordingly on October 7th Mr. Raymond Johnson made an incision over the occipital bone, and trephined as far back as possible in the middle line, afterwards enlarging the aperture with forceps up to one third of an inch from the foramen magnum. The dura mater bulged into the wound and was opened just to the left of the middle line, several ounces of clear fluid escaped, and the cerebellum was clearly seen with its lobes rather widely separated by a deep space ; no fold of pia mater was visible. A small rubber drainage-tube was inserted, the wound sewn up, and an antiseptic dressing applied. Fluid continued to escape in large quantities, vomiting and then convulsions set in, the temperature rose to 104° , and the child died, twenty-eight hours after the operation, with a temperature of 102° .

Autopsy (forty-six hours after death).—Body wasted ; slight rickets. Brain : a good many adhesions between the dura mater and the brain, beneath the anterior fontanelle, the result doubtless of theappings. Examination of the brain was difficult owing to its almost diffuent condition, but evidences of old meningitis were found at the base in the form of numerous distinct white patches in the pia mater over the tip of each temporo-sphenoidal lobe, especially the left, and slight thickening around the optic commissure ; no distinct thickening could be made out more posteriorly, certainly not between the cerebellum and medulla. Convolutions injected and somewhat flattened. Lateral and third ventricles and aqueduct of Sylvius greatly dilated, with marked engorgement of vessels on the walls of the former ; no thickening of the ependyma ; choroid plexuses normal ; fourth ventricle apparently very little dilated. No tumour. Middle ears and cranial bones healthy. Other organs normal ; no sign of tubercle anywhere.

CASE 6.—Rose L—, aged 6 months, admitted March 20th, 1893. Fed on breast only. Illness began March 7th or 8th with convulsive attacks lasting some hours ; these

recurred on succeeding days, and the child vomited each time after being fed. The child was seen as an out-patient on March 13th; the pupils were unequal, there was no optic neuritis. On admission, a week later, the child was well nourished; head much retracted, with considerable general opisthotonos; anterior fontanelle bulging; pupils unequal; discs blurred; knee-jerks exaggerated; no rigidity; pulse 160; respirations 28. The lateral ventricles were tapped through the anterior fontanelle four times between March 23rd and May 2nd, from 2 ounces to 5 ounces being withdrawn each time, about 14 ounces in all. No ill effects followed; on the contrary, the pulse seemed to improve, and after the first tapping the respiration, which had been Cheyne-Stokes in character, became more regular; fluid clear, sp. gr. 1005 or 1006, contained only a trace of albumen. Despite the treatment the head slowly increased in size, and the child became more unconscious, having to be fed by a nasal tube from shortly after admission. Head retraction was excessive to the last. There were short convulsive attacks on March 30th, April 1st and 2nd; no squint; pupils always unequal; pulse rapid (120 to 160) and regular; respiration rather irregular; vomiting frequent, especially at first; bowels rather loose; some rigidity of the limbs was noted a few days before death. Temperature normal, only twice rose to 100°; no rise before death, which occurred rather suddenly on May 5th. Duration of symptoms about eight weeks. Autopsy refused.

CASE 7.—Arthur A—, aged 3 months, admitted May 9th, 1893. Second child, the other healthy; no evidence of syphilis obtained. Brought up on breast, and had vomited frequently from birth; had otherwise been in good health until ten days before admission, when he had a fit, followed by diarrhoea, head retraction, and some rigidity of the limbs. On admission, child fairly nourished; head markedly retracted; all limbs flexed and rather rigid; slight squint; pupils equal and reacted to light; knee-jerks and superficial reflexes normal; temperature 100·6°.

In hospital there was extreme head retraction throughout until death; fontanelle full, but not markedly bulged; no convulsions; pupils equal and contracted; pulse rapid (120 to 160), only slight irregularity, dependent probably on respiration; marked Cheyne-Stokes breathing; frequent vomiting; severe diarrhoea; child had to be fed entirely by nasal tube; cried much when disturbed. On June 2nd hands clenched, but limbs not rigid; knee-jerks brisk; tendency to ankle-clonus on left side; plantar reflexes present on both sides. Shortly before death, Mr. Spicer found distinct neuritis of the right disc; the left could not be seen. Temperature rose to 101.6° the day after admission, was afterwards normal, but reached 102.6° before death on June 5th. Duration of symptoms about five weeks.

Autopsy (twenty hours after death).—Body much emaciated; very slight rickets. Brain: convolutions notably pale and flattened. A firm membrane of thickened pia mater completely closed the fourth ventricle between the medulla and cerebellum; the thickening extended along the under surface of the cerebellum for not more than half an inch on each side of the middle line, ending in a tolerably well-defined margin; slight thickening also over the pons, and as far forwards as the optic commissure. There was a thin patch of lymph over an area fully half an inch in diameter on the tip of the right temporo-sphenoidal lobe, and traces of lymph on the tip of the left, but very little elsewhere. No thickening or adhesions in the Sylvian fissures or round any of the cerebral arteries. Lateral and third ventricles much dilated, and contained over 6 ounces of clear fluid, sp. gr. 1012; moderate dilatation of veins on walls of lateral ventricles, and a few small flakes of lymph. Choroid plexuses normal; brain substance soft. Middle ears and cranial bones healthy. No thrombosis of sinuses. Spinal cord rather soft, but no pus or lymph seen in spinal canal. Other organs normal; no sign of tubercle anywhere.

CASE 8.—Herbert B—, aged 6 months, admitted March 6th, 1894. Twelfth child, seven others living; no miscarriages. No history of syphilis, ear disease, or injury obtained. Fed on breast only. Was taken ill on January 11th, and admitted to hospital January 16th, suffering from bronchitis and broncho-pneumonia. The child was noticed to have head retraction and a bulging fontanelle, with twitching of the fingers, squint, and Cheyne-Stokes breathing at times. Most of these symptoms, however, disappeared when the temperature fell to normal on January 22nd, but the head retraction persisted, and there was occasional vomiting; no optic neuritis. The child was discharged on February 7th, the lungs being clear, but he continued to have head retraction and bulging of the fontanelle, and to be very fretful, and severe vomiting having set in he was readmitted on March 6th, much wasted, drowsy, the head retraction well marked, and the fontanelle much bulged. Head measured 17 inches in circumference. In hospital child stuporous, crying only when disturbed; excessive head retraction with general opisthotonos; fontanelle bulged; no convulsions, squint, or nystagmus; pulse rapid (110) and regular; breathing regular; some vomiting at first; bowels regular; no optic neuritis; knee-jerks not obtained. Temperature 100.2° on admission, afterwards normal.

On March 12th, the circumference of the head having increased to 18 inches, Mr. Johnson trephined half an inch to the right of the middle line of the occipital bone, as close to the foramen magnum as possible. The dura mater bulged and did not pulsate; it was incised and the cerebellum protruded; on introducing a director towards the middle line, clear fluid escaped freely. The dura mater was left open and the bone not replaced, a horsehair drain inserted, and the external wound stitched up. The fluid was examined by Dr. Frank Blaxall, and streptococci in pure culture were found in it and also in the blood.¹

¹ Dr. Blaxall tells me that the streptococcus he found was similar in morphological and biological characters to the ordinary *Streptococcus*

The pulse was 120 to 130 both before and after the operation, breathing quite regular, but became very shallow. At the end of the operation the fontanelle was depressed and the bones overlapped; firm pressure was applied over the dressing. Twitchings came on very soon, but no general convulsions; the fluid soaked through in a few hours, and some packing was applied. The temperature rose in a few hours to 104.6° , and remained high, once reaching 105.8° , but was readily, though only temporarily, lowered by sponging.

On March 14th the child took more notice, its cry was more natural, and there was hardly any head retraction, but severe sickness set in, with Cheyne-Stokes breathing and more head retraction. On March 16th the wound was dressed and seemed to have ceased draining; fontanelle level but not bulging; pulse 150, regular. After this the temperature still continued high, the wound broke down, there was a little head retraction, the child gradually got weaker, and died on March 20th.

Autopsy (two hours after death).—Body moderately wasted; no rickets. Fontanelle admitted four fingers; interparietal suture loose, but not open; no bossing; no craniotabes. Trephine hole about an inch from margin of foramen magnum; wound completely broken down. Dura mater healthy. Brain: two changes observable over the surface, a chronic and very marked thickening of the pia mater, chiefly at the base, and a more diffuse recent purulent inflammation. Parts at base, from lower end of medulla to optic commissure, extremely thickened and matted together; on the under surface of the cerebellum the thickening ended in a fairly defined margin about three-quarters of an inch from the middle line. Cerebellum and pons firmly glued together, but in the middle line there was an opening in the thickened membrane about the size of a pin's head, through which fluid passed freely. A good

pyogenes and the *Streptococcus erysipelatis* (Fehleisen), except that this particular streptococcus curdled milk, which the others do not, and continued to do this for nine successive generations.

deal of lymph on the tip of the right temporo-sphenoidal lobe, and a little on the left. In the Sylvian fissures only slight adhesions due to recent puriform lymph, which extended all over the thickened part of the pia mater, and a little way on to the vertex. Lateral ventricles much dilated, and contained semi-purulent fluid; walls intensely injected and thinly covered with lymph; third and fourth ventricles also greatly dilated. Choroid plexus much swollen; veins of Galen apparently recently thrombosed. Brain substance rather soft, and posterior part of right lobe of cerebellum (beneath trephine wound) quite diffuent. Middle ears and cranial bones healthy. Some recent clot in right lateral sinus, but otherwise all the sinuses normal. Spinal cord appeared perfectly firm and healthy; central canal not dilated; no thickening of membranes. All other organs healthy; no sign of tubercle anywhere. Dr. Blaxall examined some of the thickened pia mater from the base of the brain for tubercles, but none were found.

CASE 9.—Julia C—, aged 12 months, admitted December 24th, 1894. Parents healthy; three other children alive and well. Soon after birth child said to have had snuffles and a rash, which soon got well. Never any otorrhœa. Had been mainly breast fed till ten months old, and was healthy until two months before admission, when she had measles; afterwards, for about six weeks before admission, she had head retraction, was very fretful, took food badly and wasted; no vomiting; became drowsy day before admission. There was an indefinite history of a fall on the head about two weeks before illness began.

On admission child pale, wasted, and drowsy; head moderately retracted; pupils much contracted. In hospital had marked head retraction throughout; stuporous, but could be roused, and would take food to the last; no convulsions, twitchings, rigidity, squint, or nystagmus; no slowing or irregularity of pulse, and no Cheyne-Stokes breathing noted; had attacks of vomiting and diarrhœa at times; no optic neuritis two weeks before death. Tempe-

perature rose at times to 101° (? due to bronchitis), and at death on January 31st to 104° . Probable duration of symptoms about eleven weeks.

Autopsy (twenty-one hours after death).—Body excessively emaciated; no rickets; six teeth; fontanelle nearly closed; no bossing or cranio-tabes. Brain: convolutions pale and slightly flattened. A good deal of yellowish lymph about the pons, and a little on the fold of pia mater closing the fourth ventricle, but no general thickening of this membrane, and certainly no closure of the foramen of Majendie. Very slight thickening of pia mater on under surface of cerebellum on each side of middle line. Large patch of lymph on tip of left temporo-sphenoidal lobe, and a much smaller one on right; also a few flakes about the top of the right fissure of Rolando. Sylvian fissures scarcely more adherent than normal. Lateral, third, and fourth ventricles considerably dilated, and contained about three ounces of clear fluid, which showed only a trace of albumen, probably due to a little blood accidentally mixed with it. Many dilated veins on walls of lateral ventricles. Choroid plexuses and velum interpositum apparently normal; veins of Galen not thrombosed. Brain substance of about normal consistence. Middle ears contained only a little thick mucus. Cranial bones healthy; no thrombosis of sinuses. Spinal cord and membranes apparently healthy; no lymph on surface; no softening; central canal not dilated. Other organs normal; no sign of tubercle anywhere. The thickened pia mater at the base of the brain was examined microscopically for tubercles, but none were found.

CASE 10.—Margaret F—, aged 11 months, admitted April 29th, 1895. Very little history was obtained; two other children were living and well, and there had been one miscarriage before patient was born. No history of rash, snuffles, or injury. The child had been well until three months before admission, since then had been sick at times and had gradually wasted. There are no notes of the child's

condition on admission, but on May 10th there was extreme head retraction, and the fontanelle was rather full; limbs excessively rigid, the legs and ankles being extended, the arms extended and pronated, the wrists and fingers flexed; no clonic spasm; slight external squint; no nystagmus; distinct optic neuritis; pulse rapid and regular; sick every day; child unconscious, but moaned when touched; swallowed fairly well.

May 24th.—Extreme head retraction and rigidity as before; fontanelle very full; no convulsions; breathing regular; food still taken fairly well. The coma gradually deepened, and the child had to be fed by nasal tube; the fontanelle became more bulged, the vomiting ceased. The other conditions remained unaltered, but the child became excessively emaciated; bowels freely open throughout. Temperature 97° to 100° , with an occasional rise higher, once to 103° . Death occurred on June 25th.

Autopsy (fifteen hours after death).—Body excessively emaciated; no rickets; no bossing; no craniotabes; four teeth. Head not obviously enlarged, but fontanelle admitted four fingers. Brain: convolutions pale and much flattened. Much thick lymph at base, reaching from lower end of medulla to front of optic commissure; very little lymph or matting together in Sylvian fissures. Space between medulla and cerebellum completely closed by a dense firm membrane, which extended along the under surface of the cerebellum for about half an inch on each side of the middle line, tapering posteriorly. Hardly any lymph on upper surface of cerebellum, but small patches scattered over cerebrum, chiefly in the Rolandic areas; small patches also about the tips of the temporo-sphenoidal lobes, but no distinct accumulation there. Veins of Galen not thrombosed, but considerable thickening around them in the great transverse fissure. Lateral and third ventricles much distended by about half a pint of clear fluid; some dilated veins on their walls; no lymph on choroid plexuses. Brain substance soft and cedematous. Middle ears normal, except for a little thin muco-pus in

left. Cranial bones and sinuses normal. Spinal cord : membranes normal ; slight vascularity of surface of cord ; substance fairly firm in upper half, but below mid-dorsal region almost diffuent. Other organs normal, except for some bronchitis and pulmonary collapse ; no sign of tubercle anywhere. No tubercles were found on microscopic examination of the thickened membrane from the base of the brain.

CASE 11.—Florence M—, aged 7 months, admitted May 31st, 1895. Three other children alive and well. No evidence of syphilis ; never any otorrhœa. Fed on breast only. Ailing for two months before admission, and had been treated as an out-patient for seven weeks at the West London Hospital ; cried out a good deal, frequently sick, very drowsy, squint, rigidity at times. In hospital had very few symptoms ; no head retraction or convulsions ; took very little notice, but was not unconscious till three-quarters of an hour before death. Had squint and ptosis at first, but these subsequently passed off. Vomited occasionally, and for two days before death was constantly sick and very restless. Bowels freely open every day. Temperature 97° to 100° , with an occasional rise to 103° , and once to 105° (June 16th). Death occurred rather unexpectedly on June 27th.

Autopsy (twenty-three hours after death).—Body fairly nourished ; slight beading of ribs ; no teeth. Distinct craniotabes in posterior part of parietal bones, and in occipital fossæ ; fontanelle widely open, admitting four fingers, and cranial sutures slightly separated. Brain : convolutions pale and markedly flattened. At base very slight thickening of pia mater and matting together of parts ; a few small flakes of lymph here and there, notably on tip of left temporo-sphenoidal and of right frontal lobes. Medulla drawn up to and distinctly adherent to cerebellum, but foramen of Majendie probably not obliterated. Lateral, third, and fourth ventricles greatly dilated, and contained about half a pint of clear fluid,

having sp. gr. 1009, and containing a small quantity of albumen, probably due to accidentally admixed blood. Veins on walls of lateral ventricles considerably congested. The choroid plexuses and velum interpositum appeared normal, and the veins of Galen were not thrombosed, but there was some thickening and a little lymph about the pia mater in the great transverse fissure. Brain substance soft and cedematous. Middle ears healthy. Cranial bones and sinuses normal. Spinal cord and membranes firm and healthy. Other organs normal; no sign of tubercle anywhere. On microscopic examination no tubercles were found in the thickened membrane from the base of the brain.

Course and symptoms.—Although the cases described may be too few to make any very general deductions from, yet in reviewing them one cannot but be struck by a certain general likeness in them all in the symptoms and course of the disease, and by the still greater similarity in post-mortem appearances; whilst the great resemblance alike in clinical and pathological features between my cases and those recorded by Dr. Barlow indicates that we are both describing the same condition.

All my cases occurred in infants of from three to twelve months old (Dr. Barlow had one case aged nineteen months), in whom, as a rule, there was no evidence of malnutrition or of previous illness.

The onset varied, in some being sudden, in others gradual, but in nearly all head retraction and vomiting were the most prominent early symptoms. A definitely sudden commencement seemed to be indicated by one or more convulsive attacks, but on careful questioning it appeared that in several of these cases there had been indefinite symptoms of illness for a few days before, and it is perhaps most probable that such slight symptoms are usually if not always present, but in a baby may readily be overlooked until a convulsive attack first attracts attention to the child's condition.

Head retraction was not merely an early symptom, but

was also the most marked and persistent throughout ; in only one case (11) was it absent. It was often of the most extreme character (far more than is usual in tubercular meningitis), the head being arched back to a right angle with the spine, and was sometimes accompanied by marked general opisthotonos. In two cases (4 and 5) the retraction diminished coincidently with increasing bulging of the fontanelle, and the same thing happened also in one of Dr. Barlow's cases, but there does not appear to me to be any causal relationship between the two, as in all my cases there was a large effusion of fluid in the ventricles with more or less bulging of the fontanelle, and yet head retraction usually persisted until death. Its cessation seems rather to indicate that the more active inflammation at the base of the brain is coming to an end ; this certainly was so in one of my cases (5), and Dr. Barlow's also was one in which symptoms had persisted for some months, and in which post mortem the changes were evidently of old standing.

Slight twitchings in different parts of the body were frequently noticed, but general convulsions, except at the onset, seemed to be uncommon, as would in fact be expected from the limitation of the disease to the base of the brain ; and probably for the same reason there was not often any marked rigidity of the limbs. In Case 10, however, rigidity was extreme and universal for some weeks, and in this patient many more patches of lymph were found scattered over the vertex, and especially over the Rolandic areas, than in any of the others.

At a comparatively early stage, many days or even some weeks before death, the infants usually passed through a condition of stupor into complete coma, and had to be fed entirely through the nasal tube ; this was probably attributable to the increasing intra-cranial pressure, for the fontanelle became in every case tense or actually bulging. The only case in which there was no stupor was No. 11, in whom the symptoms were in many respects aberrant, and the pathological changes correspondingly slight.

The breathing was often irregular, sighing, or markedly Cheyne-Stokes in character; the pulse on the other hand was rapid and regular, or only showed such rhythmic irregularity as might be accounted for by the great respiratory variations. Although some of the cases were under observation from a very early stage I never at any time met with the marked irregularity, and still less with the slowing of the pulse, so frequent in and so characteristic of tubercular meningitis.

Vomiting not only occurred early, but was frequent throughout the course of the disease, and constituted an important symptom in every case. Constipation was never present, on the contrary rather diarrhœa, but this may fairly be regarded as a purely accidental complication arising in young infants breathing hospital air, often treated by mercurials, and necessarily fed by hand after having been as a rule previously suckled. The same considerations may also to some extent discount the significance of the sickness, at least in the later stages.

Squint was a common symptom; nystagmus was only noticed in two instances (2 and 5). Mr. Spicer examined the eyes in most of the cases, but only found distinct optic neuritis in two (7 and 10), in one (7) a very few hours before death; in a third case (6) the discs are described as blurred. In none of Dr. Barlow's cases was optic neuritis noted.

Towards the last rapid emaciation occurred, and in all but two cases was considerable or extreme at death, which seemed frequently to result from sheer inanition from the vomiting and the difficulty in getting food swallowed.

The temperature was normal throughout in every patient, except for an occasional rise to 101° or 102°, probably of no special significance; but, as in the tubercular disease, hyperpyrexia was a frequent precursor of the end.

In the majority of cases, both mine and Dr. Barlow's, the disease terminated fatally in from five to eight weeks; but in some the course was considerably longer, extending over some months, and it seems quite possible that all

active mischief may subside and a condition of ordinary hydrocephalus be left; this question, however, can more conveniently be discussed later.

Pathological anatomy.—In nearly every case the lesion found was a very definite one, viz. effusion of lymph, thickening of the pia arachnoid, and matting together of parts over the posterior and central area of the base of the brain, from the lower end of the medulla to the optic commissure; on the under surface of the cerebellum the thickening usually ended in a rather sharply defined margin half to three-quarters of an inch on either side of the middle line. The thickening and adhesions in the Sylvian fissures, usually so marked a feature in tubercular meningitis, were in these cases either entirely absent or when present only very slight in degree. The fold of pia mater extending between the medulla and cerebellum was usually much thickened, and in several cases, though certainly not in all, the cerebral ventricles were thus completely shut off from the spinal sub-arachnoid space.

Whilst the distribution of the morbid changes was very similar in all the cases, though more extensive in some than in others, the actual appearances varied considerably; in some there was a thick layer of puriform lymph, in some a thin layer only, and in others merely thickening of the pia mater, any lymph which might have been present having probably undergone organisation into fibrous tissue.

Isolated patches of lymph were noted in nine of the cases on the tip of one or usually both temporo-sphenoidal lobes (in the first case their condition is not recorded). In two cases of general purulent meningitis in infants I have found the lymph notably abundant in this position, but I have not observed it in cases of tubercular meningitis. In three of the cases (3, 4, and 11) patches of lymph were also present on the tips of the frontal lobes. Small isolated patches were also found occasionally on the vertex, but showed no regularity in their occurrence or uniformity in their distribution.

In all the cases there was well-marked distension of the lateral and third ventricles, and generally of the fourth also, by fluid varying in quantity from a few ounces up to half a pint or more, with corresponding flattening of the convolutions.

I regret that the spinal cord was not carefully examined in more of the cases; in none did the lymph appear to extend below the medulla, and although Dr. Barlow suspected the presence of spinal meningitis in two of his cases, the spinal canal was not actually opened. In several cases in which I examined the cord, it was firm and looked quite normal; in others, parts were very soft, but possibly this was due merely to absorption of an excess of fluid in the spinal canal after death, just as in cases of hydrocephalus the excessive quantity of fluid may be one cause of the frequently almost diffuent condition of the brain. In one case (8) examined only two hours after death, the cord was firm and appeared perfectly healthy.

In no instance was there any sign of tubercle either in the cranial cavity or in any other part of the body. In four cases the thickened membrane at the base of the brain was examined microscopically for tubercle, and always with a negative result. Although in some cases of tubercular meningitis, miliary tubercles may be found only in the cerebral membranes, a caseous focus can practically always be discovered somewhere in the body, most commonly in the glands; but despite careful search no trace of caseation was found in any of my cases, nor in Dr. Barlow's. The fluid from the ventricles was examined bacteriologically in only one instance (8), and was found to contain streptococci, but no tubercle bacilli.

Pathology.—The relation between the morbid process and the symptoms calls for no special discussion. The earlier symptoms—head retraction, vomiting, convulsions, &c., may be regarded as indications of cerebral irritation; the special incidence of the mischief upon the posterior part of the base of the brain, about the medulla and pons,

explaining the persistence and extreme character of the head retraction. Squint, when present, is most likely due to special implication of one of the oculo-motor nerves by the inflammatory process. The rigidity, twitchings, or slight convulsive attacks sometimes seen during the course of the disease, are in all probability the result of extension of the inflammation to the pia mater over the Rolandic areas. In the later stages the increasing stupor, passing into complete coma, with inability to swallow, Cheyne-Stokes breathing, and sometimes hyperpyrexia at the last, are doubtless due to the increasing intra-cranial pressure; that this stage frequently lasts so long may be owing to the almost exclusive occurrence of the disease in children so young as to have extremely yielding skulls.

The most important question, however, which is raised by these cases is the wide and difficult one of the origin of the hydrocephalus. Two possible causes have been frequently suggested and discussed, viz. an inflammatory effusion, or, blocking of the exits in the fold of pia mater closing the fourth ventricle, the so-called foramen of Majendie and other smaller apertures. This latter view seems at first a very plausible one, and was strongly urged by Hilton in his well-known 'Lectures on Rest and Pain,' in which he says: "In almost every case of internal hydrocephalus which I have examined after death, I found that the cerebro-spinal aperture was so blocked up—so completely closed—that no cerebro-spinal fluid could escape from the interior of the brain, and as the fluid was being constantly secreted it necessarily accumulated, and the occlusion formed, to my mind, the essential pathological element of internal hydrocephalus."

This theory, attractive though it sounds, involves, however, a number of suppositions in regard to the cerebro-spinal fluid which are very far from being proved; and there are, moreover, several very important arguments which tell directly against it. Firstly, there can be no doubt that in a large number—probably the great majority

—of cases of hydrocephalus, Hilton's statement to the contrary notwithstanding, the membranes at the base of the brain are perfectly normal; there is no need to cite individual cases to prove this.

Secondly, effusion into the ventricles occurs in cases of tubercular meningitis, in which, as shown very definitely by the investigations of Mr. Morton, of Bristol ('*Brit. Med. Journ.*,' 1893, vol. i, p. 741), there is certainly no closure of the cerebro-spinal aperture, and Dr. Wynter published four cases of tubercular meningitis, treated in the Middlesex Hospital, in which the ventricles were actually drained by opening the spinal dura mater in the lumbar region ('*Lancet*,' 1891, vol. i, p. 981). I have notes also of several cases of simple meningitis attended with considerable ventricular effusion, but without any thickening of the pia mater over the posterior part of the base of the brain.

Thirdly, it seems probable that even in posterior basic meningitis the opening is not closed in by any means all the cases. Thus, in Case 4 it seemed to be represented by a large circular aperture, surrounded by thickened membrane, though it is not possible to say with certainty that this was not accidentally produced in the removal of the brain; in Case 5, at the operation, fluid poured out immediately the dura mater was carefully opened, and at the autopsy there was no evidence of thickening of the membrane between the medulla and cerebellum; in Case 8 there was a small but distinct opening in the thickened membrane through which fluid passed freely; in Case 9 there was certainly no closure of the usual orifice, nor was there probably in Case 11; it was patent also in one of Dr. Barlow's cases.

Hydrocephalus sometimes supervenes upon epidemic cerebro-spinal meningitis, and has been attributed to blocking of the foramen of Majendie; but Dr. Merkel, of Nuremberg, has recorded a series of such cases in which a large quantity of fluid was also found beneath the spinal arachnoid around the cauda equina; this would, of

course, be obviously inexplicable on any mechanical theory. At most, therefore, obliteration of the foramen of Majendie can only be an explanation of the accumulation of fluid in the ventricles in some of the cases ; and if we are dealing with a definite disease there ought surely to be one and only one definite cause for so important and constant a condition as the hydrocephalus.

Is the effusion then inflammatory ? Two objections at once suggest themselves ; firstly, neither the choroid plexuses nor the ependyma showed any naked-eye appearance of inflammatory change, although in three cases a few flakes of lymph were found in the effusion. Of course it may be argued that inflammatory changes in the ependyma may only be recognisable by the microscope. Secondly, the fluid has certainly not the characters of an inflammatory exudation ; in the cases (5 and 6) in which it was obtained from the ventricles by tapping during life, its specific gravity varied between 1004 and 1006, and it contained only the merest trace of albumen ; in two other cases (9 and 11), when obtained after death, it showed a very small quantity only of albumen, due probably to a little blood with which it unavoidably got mixed. In one instance (3) the fluid obtained at the autopsy thirty hours after death was highly albuminous, but I do not think much importance can be attached to this, for it is probable that the composition of the fluid rapidly alters when it remains in contact with the dead and softening brain tissue.

To escape the difficulty presented by the chemical composition of the fluid it has been suggested that an inflammatory effusion into the cerebral ventricles must have different characters from one elsewhere. Thus in Fagge and Pye-Smith's ' Medicine ' (3rd edition, vol. i, p. 665), it says that " in one and the same case the various serous membranes may pour out fluids of very different specific gravity, and it is not improbable that the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when the process

is inflammatory ;” or, as it is put somewhat differently in Ziemssen’s ‘Cyclopædia,’ “some nutritive disturbances, as yet unknown, are undergone by the delicate vessels of the pia, in consequence of which a rapid transudation of fluid becomes possible.”

It is necessarily difficult to disprove such a theory directly, but it suggests rather an ingenious attempt, disguised in scientific phraseology, to suit the facts to the theory, rather than the theory to the facts ; at any rate there seems no doubt that under suitable conditions of sufficiently acute meningitis the fluid poured out does assume the characters of a normal inflammatory exudation. Dr. Halliburton (‘Chemical Physiology and Pathology,’ p. 356) says that “in cases of tubercular meningitis the specific gravity of hydrocephalic fluid rises and the solid constituents also increase, especially the proteids ; and in such cases the fluid resembles the exudations which occur in inflammations elsewhere.” Certainly in many cases of tubercular meningitis, the post-mortem appearances hardly suggest that the inflammatory process is so much more acute than in the simple basic form, as to lead to such a marked difference in the chemical composition of the resulting exudation.

There still remains a third explanation of the ventricular effusion, viz. that it is a simple dropsical exudation. Dr. Bastian, in a paper published by him in 1867, “On the Pathology of Tubercular Meningitis,” describes a case of that disease, with considerable effusion into the ventricles, associated with thrombosis of the veins of Galen ; the velum interpositum was much thickened, and the superficial veins of the ventricular surface were all distended. He attributes the effusion to venous hyperæmia, due to the obstruction in the veins of Galen. After my attention had been directed to this observation I examined the veins of Galen carefully in every case of posterior basic meningitis, but found thrombosis in one only (8), and in that it was evidently quite recent ; neither have I seen it in any case of tubercular meningitis, nor

have other observers (Dr. Gee and Mr. Morton) who have looked for it. Although, however, no cause for a dropsical effusion was found within the veins, yet in several instances I noted marked thickening of the fold of pia mater in the great transverse fissure, sufficient, probably, to cause very considerable obstruction to the flow of blood through the thin-walled veins there, and in Case 9, in which this fold of pia mater was not much thickened, the effusion was much smaller than in most of the other cases. Moreover, even before my attention had been called to Dr. Bastian's suggestion, I had been struck in more than one case by the marked fulness of the veins on the walls of the lateral ventricles, and subsequently I found this to be present in every case examined, so that in seven out of the ten necropsies, fulness, and generally very marked fulness, of these veins was noted, whilst in the remaining three the same condition may have been present, but there is no record, for at that time not being aware of its significance I was not on the look-out for it. The first time I was impressed by this venous fulness was in Case 5, which died a few hours after being trephined, from rapid escape of fluid, and I attributed it to the sudden fall of pressure within the ventricles, consequent on the withdrawal of fluid; but this explanation would not, of course, hold good for the other cases, and I can only attribute it to some obstruction to the return of blood, most probably in the veins of Galen, and an obstruction sufficient to produce such venous engorgement would seem to be adequate to lead to the effusion, and would also account for the œdematous condition of the brain substance found in nearly all the cases.

Nevertheless, although I am inclined to regard the hydrocephalus as mainly dropsical, it is quite possible that in some, at any rate, of the cases an inflammatory process may be contributory, and very probably the combination of the two, in even slight degrees, would be very efficacious in causing rapid effusion, in the same way as hepatic

cirrhosis is brought about very quickly by a combination of even slight alcoholic excess with congestion resulting from mitral disease.

I must not digress into any attempt to explain the excess of fluid often present in ordinary tubercular meningitis, but doubtless the same causes are operative. In some instances the fluid may be, as Dr. Halliburton's investigations indicate, inflammatory in origin; whether it can be so always seems doubtful. Thus the fluid drawn off by Dr. Wynter, in the cases already referred to, had a specific gravity of 1004 to 1006, and was almost non-albuminous. Several times, also, I have found very distinct thickening of the velum interpositum and of the pia mater in the great transverse fissure, with more or less dilatation of the veins on the ventricular walls; so that it seems probable that at least in part, and in some cases, the effusion may be dropsical in origin, or, in other words, that, as in the simple basic meningitis, both causes are contributory, though inflammation may play a more important part in the tubercular than in the non-tubercular disease.

This explanation of the hydrocephalus, as due partly to inflammation and partly to venous obstruction, has the advantage of accounting for its presence in all the different inflammatory conditions of the membranes with which it is associated, and in which there is no blocking of the cerebro-spinal aperture.

Meanwhile, the characters of the effusion in both acute and chronic hydrocephalus call for careful investigation at every opportunity.

Ætiology.—The disease seems to be almost confined to infants under one year old, and, as is to be expected at this period of life, to be about equally common in the two sexes, whereas tubercular meningitis is less common in infants than in older children, and more frequent in boys, perhaps from their greater liability to injuries and blows on the head.

It is noteworthy that in nine out of my eleven cases

the infants were still being fed from the breast only at the date of onset of the disease, one had been weaned at ten months, not many weeks before admission, and there is no note as to the remaining child ; as would therefore be expected, in none was there any marked rachitic condition.

As to the actual cause of the disease, the question of injury is the first to demand consideration. In the first case convulsions and vomiting were distinctly said to have begun the day after a blow on the head, and Dr. Barlow obtained a similar history in some of his cases. What may be the exact connection between the two it is difficult to say : that an injury is *the* cause in all cases is inconceivable ; that it may act as an efficient exciting cause, when other conditions are favorable, is possible. The position seems to be similar in regard to tubercular meningitis ; obviously no injury could produce that disease in the absence of the tubercle bacillus, but possibly if the bacilli be circulating in the blood a blow on the head may determine their incidence upon the membranes of the brain, just as an injury to a joint may be the starting-point of tubercular arthritis ; *e. g.* in a boy aged 3 years, in whom tubercular meningitis was fatal about six weeks after a severe blow on the head from a stone, at the autopsy I found miliary tubercles in the cranial cavity only, together with an old caseous focus in the right lung. In gouty conditions a similar relationship to injuries is frequently observed.

Ear disease must, of course, be thought of as a possible cause, but the evidence that it is so seems to me of the slightest. In seven of my cases both middle ears were healthy ; in each of the other three inodorous pus or semi-purulent mucus was found on one side, but no other mischief whatever, and we must remember that it is exceedingly common in infants to find thick gelatinous mucus or mucopus in the middle ears ; the readiness with which this, under the influence of a slight catarrh, may become purulent, probably explaining the frequency of otorrhœa in young

children. Of course, otitis media by spreading along lymphatic or venous channels may set up inflammation of the cerebral membranes, just as it may a cerebral abscess, without any disease of intervening parts ; but in such cases the resultant meningitis is probably always of a very acute character, and I have made autopsies upon several cases of acute purulent meningitis which have most likely been produced in this way.

Dr. Gee, in the ' St. Bartholomew's Hospital Reports ' for 1872 (vol. viii), described cases of purulent meningitis and otitis interna (unattended by any disease of the bone), and showed that the symptoms of the otitis may precede those of meningitis, may show themselves in its course, or be latent and undiscoverable throughout ; but, be it noted, these were all cases of purulent meningitis. Moreover, as will be explained directly, I do not consider any cause to be satisfactory, which will at most explain only a minority of the cases.

Congenital syphilis has, of course, been invoked as a cause of the disease, but there is, I think, an ever present danger of endeavouring, without sufficient reason, to make it responsible for this and for many other obscure conditions for which no other explanation is readily available. Any one sufficiently energetic in pushing inquiries can, in a large proportion of infants seen in hospital practice, elicit some facts in the history or in the child's condition which are capable of being attributed to syphilis, and when once some evidence of it has been obtained in a majority of the cases, it is very easy to argue that similar evidence would be forthcoming for the remainder, if only all the facts were available. Such a line of argument, though sufficiently tempting, is obviously open to many fallacies ; that syphilis may produce a localised meningitis is admitted, but there seems nothing to prove or even to indicate that it leads to the particular form of meningitis now under consideration. There was no strong evidence of syphilis in any of my cases, in most there was none at all. The mere occurrence from time to time of basic

meningitis in obviously syphilitic infants is not of much importance, since hereditary syphilis is certainly sufficiently common to render such a coincidence not infrequent; in fact, if the coincidence did not sometimes occur it might fairly be argued that the two diseases were antagonistic. That it can be the cause in some cases and not in others seems most unlikely, for I have endeavoured to show that we are dealing with a disease as definite as tubercular meningitis itself, a disease with well-defined and easily recognisable symptoms, running a pretty definite course, and presenting fairly constant pathological appearances; in fact, in all these respects, the variations met with are hardly if at all greater than those seen in different cases of tubercular meningitis.

It is, no doubt, possible for diverse causes to produce the same result, but I can hardly regard it as conceivable that this definite clinical and pathological condition can in some cases be due to syphilis, in some to traumatism, in some to middle ear disease, in some to other causes; for no one probably will contend that any one of these possible causes will explain all the cases. Therefore, instead of always thinking that, because syphilis is or may be a cause of so many different morbid conditions, we have done our duty by industriously striving in this or in other obscure diseases of infancy to ferret out a history or even the remotest possible suspicion of syphilitic taint, let us rather try and find some one cause which will serve as a sufficient explanation of all the cases.

If, then, we are led to think of posterior basic meningitis as a definite disease, with a definite—perhaps specific—cause, we are naturally also led to consider whether it has any relationship with that distinctly specific disease, acute cerebro-spinal meningitis. The latter occurs in epidemics which are certainly rare in this country, but it is in accordance with the analogy of many other diseases,—and the behaviour of cholera in Europe during the last few years may especially be referred to,—that sporadic cases, milder, more chronic, modified in character, and not liable to spread,

may from time to time occur; and most writers describe such cases.

The resemblance in the pathological anatomy and symptoms of the two conditions, allowing for differences in severity, acuteness, and duration, is somewhat marked. The epidemic meningitis is certainly common in young children; in fact, according to Dr. Oscar Medin, of Stockholm (quoted by Dr. Eustace Smith), infants under twelve months old are specially liable to it. Dr. Lewis Smith, of New York (Keating's 'Encyclopædia of the Diseases of Children,' vol. i, p. 514), states that "a larger proportion of fatal cases are in the first year of life than in any other year;" and Dr. Gowers says that "children possess a special liability, and in some epidemics have been almost exclusively affected."

The most marked lesion is purulent lymph, which may be present anywhere over the surface of the brain, but most abundantly and constantly at the base, especially at the posterior part over the medulla; it is found also along the posterior part of the spinal cord. There is effusion into the cerebral ventricles.

The onset of the disease is described as sudden, in children it is said often with convulsions; severe and often persistent vomiting, with extreme retraction of the head, are always marked symptoms, and opisthotonos is sometimes present. Twitchings of the limbs are very common, and general convulsions may occur during the course of the illness. Squint is frequently, and nystagmus sometimes seen. Optic neuritis was only found six times in forty cases. The pulse is usually quickened, and not often irregular or infrequent. The knee-jerks vary, sometimes being increased and sometimes absent. The temperature is very uncertain, but rarely high. There is great and rapid emaciation, and death—preceded by coma—is the usual termination in from two or three days to three or four weeks. The attacks vary much in severity, and abortive ones are described. In the epidemics a rash is often present, but is said to be absent in most sporadic

cases. Finally, as if to complete the resemblance to posterior basic meningitis, in cases of recovery chronic hydrocephalus has supervened, and post-mortem the cerebro-spinal aperture has been found to be closed.

On the other hand, it may be objected that nearly all forms of meningitis are commoner in children than in later life, and that a mere resemblance of symptoms in cases of intra-cranial disease is of no great importance, and may even be very misleading, in the absence of other indications of identity between what may be different diseases; for we might, in fact, draw as close a parallel between the simple meningitis I am describing and the tubercular form, as between the former and cerebro-spinal meningitis. Also it may be said that no such affection of the spinal cord has been made out as to justify us in regarding it as closely allied to the cerebro-spinal disease; although if the base of the brain be the part most affected in the ordinary cases of the epidemic variety, it may well be the only part attacked in milder sporadic ones.

On the whole, I think that clinical observation and even morbid anatomy will not carry us much further, especially in this country, where the epidemic form of meningitis is so rare, and that we must turn next to bacteriological investigation to help in determining the true pathology of the disease. That it is associated with a definite micro-organism is, I think, very probable, but on this point I am unfortunately unable to give any information. The bacteriology of the epidemic disease has not yet been satisfactorily worked out; but, so far as has been ascertained, the organism which is associated with it seems to be identical with, or closely related to that which is responsible for croupous pneumonia, and the pneumococcus is found also in most cases of acute purulent meningitis which have no obvious cause (Dr. Ormerod's article on "Epidemic Cerebro-spinal Meningitis" in Dr. Clifford Allbutt's 'System of Medicine,' vol. i, p. 673). It is, of course, possible that the pneumococcus, whilst giving rise to sporadic cases of meningitis, may, under

suitable conditions, cause an epidemic variety, just as it occasionally seems to give rise to an epidemic and infectious pneumonia; but we still know far too little of the part it plays in morbid processes generally, to be able to hazard more than the most vague hypotheses on these matters. Certainly in the four or five cases I have seen of meningitis in children associated with, and probably secondary to pneumonia, neither the symptoms nor the post-mortem appearances were in the least suggestive of the posterior basal form; the meningitis was more or less general, the lymph abundant, and, as a rule, the base of the brain, particularly the posterior part, was less affected than the vertex. Mr. D'Arcy Power ('The Clinical Journal,' vol. viii, No. 4) has noted that more children suffering from meningitis have been admitted to the Victoria Hospital since the epidemic prevalence of influenza during the last few years; but in the cases I have recorded no connection with this disease could be traced, nor did they occur specially during the actual outbreaks of the influenza.

Before we can determine that any micro-organism is the true cause of the posterior basic disease, much work will have to be done, not only in connection with this, but also with other obscure forms of meningitis, in order to clear up their mutual relationships; and even should a definite result be obtained, we should still be only on the threshold of the problem as to why such a limited area of the cerebral membranes is attacked, and why infants, usually previously healthy, are almost exclusively affected.

Some further help in regard to the relationships of the disease may be gained by the investigation of non-typical cases, especially by endeavouring to trace connecting links with acute purulent meningitis. I have examined a considerable number of examples of the latter in infants, but without definite results; the disease in most is general, but the base of the brain is sometimes not involved at all or less so than the vertex, and the children in whom it

occurs are either marasmic or already suffering from some definite disease, whereas in the simple basic form the previous good health and nutrition of the patients is rather striking.

The following case may possibly have been an acute variety of the posterior basic disease. A child aged 5 months was brought to the Hospital in a moribund state; the only history obtained was that he had been ill for three weeks with convulsions and vomiting, and had had head retraction throughout. At the autopsy the body was much emaciated. At the base of the brain was a dense mass of yellow lymph, most abundant between the medulla and cerebellum; it extended along the velum interpositum into the lateral ventricles and over the choroid plexuses. There was also a thin patch on the tip of the left temporo-sphenoidal lobe. The convolutions were flattened, and the lateral ventricles contained 2 or 3 ounces of turbid fluid, with many flakes of lymph. The veins on the ventricular walls were markedly dilated. The spinal cord and the other internal organs were healthy, and there was no sign of tubercle anywhere.

Another question which naturally arises, is whether the disease is really limited to infants, for with our present knowledge it is difficult to see why it should be, although eventually we may not find this age incidence to be any more remarkable than the limitation of the severe forms of summer diarrhœa and vomiting to young children. Dr. Newton Pitt records a case in a girl, aged 3 years ('Med. Times and Gazette,' 1885, vol. ii, p. 827). Personally I have no experience of the occurrence of the disease after infancy; the nearest approach to it which I have met with was in a boy, aged, however, only 2 years, admitted to hospital August 20th, 1891, with a history of vomiting, headache, and stupor, developing gradually after a fall on the head five weeks before. In hospital there was squint, irregular pulse, and a little head retraction; not much sickness after admission. Death, preceded by convulsions and hyperpyrexia, occurred on

September 3rd. At the autopsy there was a good deal of lymph over the posterior part of the base of the brain; the lateral ventricles were dilated, and contained several ounces of turbid fluid. No evidence of tubercle was found anywhere, although the resemblance to tubercular meningitis was in many respects very close, and the nature of the disease remains obscure.

Dr. Bastian kindly allows me to mention the following case, which was under his care in the National Hospital, Queen Square. Patient, a girl aged 10 years, was admitted April 2nd, 1894. Her illness had begun suddenly three weeks before with headache, followed in a few hours by a convulsive seizure; afterwards there was headache, some head retraction, delirium at times, but no vomiting. In hospital there was considerable head retraction; no paralysis; a good deal of pain in the back; some general hyperæsthesia; no optic neuritis; no loss of consciousness; pulse about 120; temperature 100° to 103.4° . On April 6th the skull was trephined; a large quantity of cerebro-spinal fluid escaped, and patient died on the table. At the autopsy the pia arachnoid at the base was found much thickened; the vessels on the pia were deeply injected on each side; the lateral ventricles were not much dilated; there was considerable excess of sub-arachnoid fluid in the spinal canal, with great engorgement of vessels on the surface of the cord. Although the membranes at the base of the brain were specially affected, I think that both clinically and pathologically the case suggests a subacute general cerebro-spinal meningitis rather than the special posterior basic variety. As to whether there may be any relationship between the two, we are—as I have already said—at present quite ignorant.

Diagnosis.—Until some experience of these cases has been obtained there can be no doubt that during life it is very easy to regard them as examples of tubercular meningitis. A young child is seen for the first time lying in an unconscious state, its head retracted, breathing

irregularly, and swallowing with difficulty ; the history is of a short illness, very probably ushered in by convulsions and attended by severe and persistent vomiting ; examination of the chest and abdomen reveals nothing definite. What more natural than that it should be looked upon as an ordinary case of tubercular meningitis in a late stage ? In my own cases what first aroused suspicion was the persistence of the condition just described, for when a child suffering from tubercular meningitis has reached the stage of stupor and rapid pulse, its remaining term of life is commonly to be reckoned by hours or at most by a few days ; but these simple cases last on day after day and even week after week, with but little variation, except that the head retraction becomes if possible more extreme and the coma more profound. Consequently when at last death did occur the pathological changes were looked for with special interest, and the suspicions which had arisen during life as to the unusual nature of the case, were confirmed by finding that the lesions were not those of the tuberculous disease. After an experience of a few such cases, one naturally began to look out for them during life and to endeavour to diagnose them as soon as possible, and thus the first important step was made towards an early diagnosis of the disease, viz. the recognition of the possibility of its occurrence, for naturally we rarely diagnose diseases which we are not expecting to meet with. Thus prepared, we may, I believe, make a fairly confident diagnosis of most cases of simple posterior basic meningitis some time before the fatal termination.

True it is that the symptomatology of tubercular meningitis is so varied and irregular that it may at first seem impossible to say that any symptoms of acute cerebral disease in a child may not be due to it, nevertheless I think experience shows us that the cases of posterior basic meningitis have a fairly characteristic clinical course, difficult to indicate in definite terms, but yet sufficient to say in dealing with a case, that the symptoms are not those, or have not the characteristic grouping of those

met with in any case, however aberrant, of tubercular meningitis.

The age of the patient does not afford much help in diagnosis; tubercular meningitis is certainly commoner after the first year of life, but a small number of cases do occur earlier, and all we can say is that whilst in children much over one year the simple variety of basic meningitis is excessively rare, during the first year of life either form may occur with perhaps about equal frequency.¹

Perhaps the most characteristic of all the symptoms of the simple basic meningitis is the head retraction, for though often present in the tubercular disease, it is, I should say, rarely so marked and never so persistent throughout the whole course of the illness as in the cases now under consideration. It may, of course, arise from other causes than meningitis; Dr. Money ('Treatment of Disease in Children,' p. 456) mentions cerebellar tumour, rheumatic affection of the muscles of the back of the neck, ear disease, tender cervical glands, abdominal disturbance, and other peripheral irritations; in several of the cases recorded by Dr. Gee it was probably due only to the muscular weakness associated with rickets, or to gastrointestinal disturbance, but in such, as in some of the other conditions mentioned by Dr. Money, the other signs of cerebral disease,—convulsions, coma, &c.—would of course be absent, nor is the retraction likely to be extreme or persistent. Once, however, I saw very marked head retraction lasting for a fortnight before death in a boy, aged seven years, suffering from typhoid fever; nothing was found post mortem to account for it, and one can only suppose it was reflex and due to abdominal disturbance. In another case, a boy aged nine months, an out-patient, suffering from bronchitis, broncho-pneumonia, diarrhœa and vomiting, there was well-marked head retraction for five or six weeks, the child had every appearance of

¹ During the time that my eleven cases of the non-tubercular disease occurred at the Victoria Hospital I find records of nine autopsies on children under one year suffering from ordinary tubercular meningitis.

tubercular disease, but got quite well, the head retraction passing off as the lungs cleared. This symptom seems also to occur occasionally in meningitis not affecting the base of the brain, for in a child aged eight months with acute purulent meningitis it was extreme for ten days, though at the autopsy there was no lymph over the pons, medulla, or cerebellum, and no distension of the ventricles. On the other hand, it was altogether absent in Case 11 of my series, but this was an anomalous one and probably impossible of diagnosis during life. In all of Dr. Barlow's cases it was well marked.

A slow and irregular pulse is well known to be a characteristic symptom of the intermediate stage of tubercular meningitis, whereas in the simple basic form, so far as my experience extends, the pulse is rapid throughout, irregularity is rare, and when present is very slight.

Some less marked differences may be referred to. Vomiting usually ceases in the later stages of tubercular meningitis, as the symptoms of increasing intra-cranial pressure become more marked; but in the simple form it often persists to the last. The bowels are usually costive in tubercular meningitis, at any rate in the early stages; in the non-tubercular disease I have never observed constipation, though Dr. Barlow notes its presence in two of his cases. The temperature is generally somewhat, though not often much raised in the tubercular disease, whilst in the simple form it is often quite normal for long periods; in both conditions, however, there is frequently a rapid ante-mortem rise.

Lastly, the duration of the symptoms confirms the suspicions to which their character has already probably given rise, for whereas if tubercular the case ends fatally in two or the very most three weeks from the onset of definite symptoms, the simple form—as judged from Dr. Barlow's and my own cases—seems to last at least four or five weeks, commonly about six, and often considerably longer.

It is not necessary to discuss here the diagnosis of the disease from non-cerebral diseases which may for a time

simulate it, *e. g.* gastro-intestinal disturbance, typhoid fever, cerebral symptoms due to reflex irritation or pyrexia, &c. The points of distinction are necessarily much the same as those between tubercular meningitis and the conditions just mentioned, and are sufficiently dealt with in the ordinary works on medicine and on diseases of children.

The method of lumbar puncture of the sub-arachnoid space, which has lately been introduced as a means of diagnosing meningitis from other conditions, would not be reliable in the posterior basic disease, for in those cases in which the foramen of Majendie is closed, and the communication between the cerebral ventricles and the sub-arachnoid space of the cord consequently cut off, no indication could be obtained by the puncture of the amount of the intra-cranial pressure, or of the character of the intra-cranial fluid.

Prognosis.—Although the disease is undoubtedly most serious, yet there is good reason for thinking that a small proportion of the cases may and do recover, at least partially, for in some hydrocephalus will be left. It seems to me that in Case 5 recovery had certainly occurred so far as the meningitis was concerned: the child had presented all the characters of well-marked basic meningitis, and definite remains of it were found post-mortem; but all symptoms, notably the head retraction, vomiting, and difficulty in swallowing, had disappeared; the child was gaining weight, becoming more lively and observant, and was in fact suffering simply from chronic hydrocephalus. There had, I believe, been thickening of the pia mater over the posterior part of the base of the brain, which had very largely disappeared, but would probably have been still recognisable there but for the disturbance of the parts in the operation, and the almost diffuent condition of the brain at the post-mortem.

I have notes of another case in which there had apparently been posterior basic meningitis which had not proved fatal. Edith S—, aged $3\frac{1}{2}$ years, was admitted to the Hospital on April 30th, 1889. Not much history could

be obtained, but she had been born with a spina bifida, had never attempted to stand or bend the knees, and was backward in talking and in cleanliness. The spina bifida was healed, but the child had rigidly extended legs with talipes equinus. For this tenotomy was performed on May 13th; broncho-pneumonia developed shortly after, and the child died on June 2nd. At the autopsy the pia mater between the medulla and the cerebellum was found converted into a firm fibrous membrane, and the cerebro-spinal aperture closed; there was no lymph, the change being evidently of very old standing. The lateral ventricles and the aqueduct of Sylvius were much dilated, and contained several ounces of clear fluid; velum interpositum and choroid plexuses rather congested. No sign of tubercle in brain, though the thoracic and mesenteric glands were caseous. Right middle ear healthy, left contained a little muco-pus. The anterior fontanelle was widely open, and the frontal suture unclosed. The case was evidently one of hydrocephalus associated with chronic changes in the membranes at the base of the brain.

The difficulties in the way of proving the possibility of complete recovery are obvious, and must necessarily be almost insuperable, the more so because such cases, if they do occur, will naturally be of a milder type than the fatal ones, and will consequently present less definite and less recognisable features. I have, however, seen a few infants who got quite well after an illness the symptoms of which most closely resembled those due to posterior basic meningitis; the following is a good illustration. Edith G—, aged 9 months, admitted May 7th, 1895. First child; no previous illness except bronchitis; no evidence of syphilis or injury; had had otorrhœa at times. Fed on breast only. Illness began rather suddenly about three weeks before, with frequent vomiting and persistent and increasing head retraction; the bowels had been regular; the child had taken the breast badly, and had wasted. On admission patient swallowed badly and took very little notice, crying only when disturbed; had marked

though not extreme head retraction, with some opisthotonos; fontanelle distinctly bulged; no convulsions, rigidity, squint, or nystagmus. Pupils equal and of moderate size; pulse rapid and regular; breathing regular; knee-jerks and plantar reflexes brisk; very slight rachitic changes in bones. The child remained in this condition, with little alteration, for some weeks; the head retraction was constant, but varied in degree; no convulsions, but slight rigidity at times. For the first five weeks vomiting occurred usually several times a day; bowels rather loose; pulse rapid (150 to 160) and regular; no optic neuritis; temperature rose occasionally, at first as high as 102.6° , but remained normal after the first week. The only drugs given were iodide and bromide of potassium.

Improvement slowly occurred, the head retraction and bulging of the fontanelle diminished, the vomiting gradually ceased, the child began to look brighter and became more natural in manner, and was discharged on July 17th. On July 23rd she was doing well, took notice, and was not sick; there was still slight head retraction. So far, at any rate, as symptoms can guide us, this case presented a fairly typical picture of posterior basic meningitis.

Two further questions arise; the first is as to whether some of the supposed cases of tubercular meningitis which recover are not really examples of this simple posterior basic disease. There is evidence, not necessary to adduce here, which seems to show that tubercular meningitis is perhaps not invariably fatal; but without entering upon this still disputable question, the cases of recovery must necessarily be excessively rare, especially when we consider that the disease is, as a rule, merely a part of a general tuberculosis. Simple posterior basic meningitis is a condition not as yet generally described or recognised, and, as has been shown, very easy to be confused with the tubercular form by those unfamiliar with it, and we may therefore, I think, fairly assume that, at any rate in infants, some at least of the credited recoveries from the latter were really examples of the somewhat less fatal simple form. That

such a child should afterwards fall a victim to tubercular disease, and even to tubercular meningitis, is certainly not impossible, and does not in the least prove, as has sometimes been assumed, that the first cerebral attack, old evidences of which may be found post-mortem, was necessarily of a tubercular nature.

The second question is, to what extent is this form of meningitis a cause of ordinary chronic hydrocephalus? One such case has just been mentioned, that of hydrocephalus and spina bifida, and Case 5, which was operated on, may fairly be considered another. Dr. Baxter recorded one in the 'Transactions' of the Pathological Society, vol. xxxiii; a child previously healthy had an attack of convulsions when six weeks old; at three months the head began to enlarge, and great hydrocephalus developed; death occurred at two and a half years. At the autopsy five pints of fluid were found in the brain, the cerebro-spinal aperture was obliterated, and there was also some recent meningitis over the pons and within the circle of Willis; no tubercle. Hilton, as already quoted, attributed most cases of internal hydrocephalus to closure of the cerebro-spinal aperture, and details several examples. The number of such cases must, however, I think, be few, for posterior basic meningitis is far from common, and of the cases which do occur probably most are soon fatal. Dr. Baxter himself said that though he had been looking for this condition in hydrocephalus, the one he described was the first he had found after examining about six cases. Moreover, non-congenital hydrocephalus begins, as a rule, with a gradual enlargement of the head, without any very marked disturbance of the general health—very different to the severe symptoms which accompany basic meningitis. Nevertheless, the possibility of the occurrence should not be overlooked, and in old-standing cases too much reliance must not be placed upon the absence of extensive pathological changes at the autopsy. Speaking of epidemic meningitis, Dr. Ormerod (*loc. cit.*, p. 668) says, "It would appear that in cases which survive the acute stages of the

disease, the meningeal exudation gradually degenerates and is absorbed, but the pia arachnoid becomes thickened and shrunk, and the *ependyma ventriculorum* hypertrophies. The ventricular effusion, probably in consequence of these cicatricial changes, either remains unabsorbed or increases in amount, albeit now less purulent and more passive in character." These words doubtless apply equally to those cases of posterior basic meningitis which are followed by chronic hydrocephalus, the more marked signs of inflammation subsiding, and only slight chronic changes being left ; these may very easily be missed at an autopsy, for in advanced cases of hydrocephalus a satisfactory examination of the membranes at the base of the brain is almost impossible. Certainly Case 5, already referred to, suggests that a basic meningitis may occur, nearly all pathological evidence of which may, like the symptoms, disappear ; and yet that it may start a ventricular effusion which may increase and become permanent.

Treatment.—As we must recognise the possibility, small though it be, of the inflammation subsiding and recovery taking place, we may justly, I think, employ some such vigorous line of counter-irritation as Hænoch recommends for meningitis ; not, perhaps, in such young children, leeches or blisters, but at least mercurial ointment to the throat and neck, an ice-bag to the head, and calomel internally. Most of the cases described were treated with iodide and bromide of potassium, or with grey powder or calomel, without, however, any obvious effect, except that of causing diarrhœa. Should there be signs of commencing hydrocephalus, the tense bulging fontanelle almost invites puncture of the lateral ventricles with a small trocar and cannula ; and whilst this seems a harmless procedure, if carried out with proper precautions, it must surely be beneficial, should the meningitis be recovered from, by preventing injurious and permanently harmful pressure being exercised on the brain. Tapping does not, of course, prevent the supervention of chronic hydrocephalus, still there seems no reason why in some cases

the ventricular effusion should not be transitory, and in such, timely withdrawal of fluid during the acute stage may make all the difference between recovery with a useful or with a permanently damaged brain. Care must obviously be exercised, especially in earlier punctures, to remove only a small quantity of fluid at once, but my limited experience tends to show that in the soft compressible skulls of young children several ounces may be withdrawn without any harm resulting. Some writers say that with each successive tapping the fluid becomes more inflammatory in character, but in the two cases (5 and 6) recorded above, in which it was several times repeated, there was no such change.

Finally, the question of operative interference has to be considered; this may be resorted to either in the acute stage, as soon as marked pressure symptoms develop, as in Case 8; or later on, if, after the acute symptoms have subsided, the hydrocephalus is evidently increasing, in spite of the drawing off of fluid by trocar and cannula, as in Case 5. If the accumulation of fluid in the ventricles be due to closure of the cerebro-spinal aperture, it seems a reasonable thing to try and reopen it; whilst if it be dropsical in origin and produced by pressure on the veins of Galen, or even if it be due to chronic changes in the ependyma, continuous drainage from the sub-cerebellar arachnoid space may allow time for the inflammatory changes, either in the velum interpositum or in the lining membrane of the ventricles, to subside, and so permit of ultimate recovery. In either stage, acute or chronic, the most immediate danger in operating seems to arise from the difficulty in preventing too rapid an escape of fluid. The child, aged 10 years, under Dr. Bastian's care, to whom reference has already been made, was trephined over the right occipital region, between three and four weeks after the commencement of her illness, but died on the table from rapid escape of fluid; the same cause led to a rapidly fatal result in my Case 5, trephined long after all acute symptoms had quite subsided. Mr. Lowson, of

Hull, also records a case of hydrocephalus in which drainage of the fourth ventricle proved fatal in a similar way in a few hours ('Brit. Med. Journ.,' 1893, vol. i, p. 1322).

A few cases, however, have been recorded, which show that this is not an insuperable difficulty. In 1893 Mr. Parkin, of Hull, published the case of a child, aged 11 months, suffering from chronic hydrocephalus, in which he trephined below the superior curved line of the occiput, and inserted a horse-hair drain below the cerebellum into the subarachnoid space; fluid escaped slowly for fifteen days, and the drain was removed after eighteen days. The child was discharged greatly improved, and three months afterwards there was no indication of recurrence ('Lancet,' 1893, vol. ii, p. 1244). Mr. Parkin has since published a second successful case of trephining in a child of 3½ years, and also one in an infant aged 5 months, in which death occurred from hyperpyrexia on the ninth day from too rapid withdrawal of fluid ('Lancet,' 1895, vol. ii, p. 1166).

Dr. Glynn and Mr. Thomas, of Liverpool ('Lancet,' 1895, vol. ii, p. 1106), have also recorded a case of hydrocephalus in a man aged 18 years. The skull was trephined through the cerebellar fossa and the fourth ventricle opened up, after breaking through a soft partition, which they took to be the thickened posterior medullary velum bounding the fourth ventricle; a quantity of fluid escaped, and the wound was closed without any drainage into the cranial cavity. The patient recovered. All the above were chronic cases, but Dr. Ord and Mr. Waterhouse ('Lancet,' 1894, vol. i, p. 597) drained the subarachnoid space for eighteen days in what appeared to be a case of tuberculous meningitis in a child aged 5 years, with perfect success; so that the presence of recent inflammation constitutes no bar to the success of the operation.

True it is that probably none of the successful cases here mentioned were examples of simple posterior basic meningitis; but the conditions and indications for opera-

tion must be the same, and the chances of success at least as good in this as in the other varieties of meningitis, or as in cases of hydrocephalus due to other causes. The further discussion of this method of treatment I must, however, leave to the surgeons, although both from theoretical considerations and from practical experience I am inclined to think that operative interference offers at present but little prospect of success, and the argument sometimes advanced that because a case will almost certainly prove fatal if left alone, it should therefore be operated upon—however remote the prospect of success—would, if frequently acted upon, and with almost uniformly fatal results, tend to bring surgical practice into disrepute.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. ix, p. 114.)