THESIS

for the Degree of Doctor of Medicine
of Edinburgh University.

SIMPLE BASAL MENINGITIS IN CHILDREN

by

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DEFINITION:

Simple Basal Meningitis defines itself as regards its position, though it might be more correct to describe it, as is sometimes done, as Posterior Basic Meningitis, for it is invariably met with at the posterior part of the bases of the brain, at the junction of the cerebellum with the medulla, and in the interpeduncular space. It is named "simple" in contra distinction to the tuberculous variety also affecting the base of the brain.

It is due to a definite organism to be later described.

I have used the phrase "invariably met with at the posterior part of the base of the brain" intentionally as in every case that I have seen, this region has been affected. It may be the only region affected, but frequently the inflammation has spread down the cord, and occasionally up to the vertex. Changes occur in the purulent lymph at the base of the brain just as they do in a purulent collection in any other part of the body. If the case is not very acute, then adhesions form round it and one gets a Posterior Basic Meningitis. Before adhesions have had time to form, however, even in cases which are not acute, the exudation passes in/
in the direction in which there is least resistance, namely, down the cord, and we then get the condition most frequently seen in the post mortem room - a cerebro-spinal meningitis. In very acute cases, the inflammation spreads, against resistance, up to the vertex. Such cases are almost invariably fatal, because the inflammation is so acute. It seems to me simplest and best to describe the disease as above, and not to subdivide it as Lees and Barlow \(^{(1)}\) do into:

(a) A Post Basic Class and,
(b) A Vertical Class.

Such a subdivision is, I think, only confusing and leads to many qualifications in the description. This is doubtless due to the fact that in their classification of the vertical class, Lees and Barlow have included not only meningitis due to Still's diplococcus, i.e., Post Basic Meningitis, but also other varieties of Simple Meningitis, such as pneumococcal. Thus we get the following qualifications in their description of the Vertical class:

"In the Vertical Class, the inflammation is often most marked about the anterior part of the brain and the region of the cerebro-spinal foramen may/
may escape." Again, in the description of the Vertical case:- "The ventricles are frequently normal; in some cases they share slightly in the inflammatory extension and may contain a little purulent lymph, but as a rule they are not distended in this form." Again, "Inflammatory conditions of the viscera and serous membranes often coexist; especially pneumonia, empyema and pericarditis." It will be evident from such statements that Less and Barlow have included in their list of cases not only these due to Still's organism, but also cases due to the pneumococcus, and it may be other pyogenic organisms, for I shall endeavour to show later on that Posterior Basic Meningitis has no special tendency to follow Pneumonia or Pleurisy, or in fact, any other catarrh. Pneumococcal Meningitis affects the vertex and need not affect the base, hence in such cases, the ventricles escape, thus leading to some of the above statements of Lees and Barlow. In cases of Posterior Basic Meningitis which have spread to the vertex, the ventricles are liable to be affected just in the same way that they are in the localised simple basal; as such cases, however, die soon, the effect is much less marked than in the localised variety./
variety. I think, therefore, it would be much simpler to classify meningitis from the pathological or bacteriological aspect rather than the anatomical, and to speak of tuberculous, pneumococcal suppurative meningitis, and meningitis due to Still's organism.

In this thesis, I have included only cases which have been proved either by bacteriological examination to be due to the diplococcus of Still, or where from a clinical evidence, one may conclude that such was the cause. Pneumococcal and meningitis secondary to suppurating conditions, I have not included. The cases to be tabulated later have all been culled from the records of the Royal Hospital for Sick Children, Edinburgh, and comprise those that were admitted to that hospital during the years 1900, 1901 and 1902.

HISTORICAL NOTE:

This disease did not attract much attention till Gee and Barlow (2) in 1878 drew the attention of the medical profession to it. It is only within recent years, however, that the condition has been seriously enquired into. Carr (3) in 1897 published a paper on it. Still later in 1899, Lees and Barlow published their article which one may/
may describe as the standard one; while at almost the same period, Still described the bacteriology of the condition. The fact that they base their paper on as many as a hundred and ten cases, gathered since 1877, goes to show that the disease is by no means rare, though one must bear in mind as mentioned above, that some of these cases do not come under the category of the present paper. In the Sick Children's Hospital, Edinburgh, thirty cases of the disease were admitted during the years 1900, 1901, 1902.

With the increased knowledge of the disease that we now possess, doubtless many cases which were hitherto classified as tuberculous, would now be recognised as simple. The reported "cures" in cases of tuberculous meningitis, in all probability are cases of simple basal meningitis.

The relation of simple basal meningitis to epidemic cerebro-spinal meningitis is still a matter of dispute. The two diseases have much in common and I will draw attention later on to one or two signs amongst the present cases leading one to think that the one disease is only a modified form, or sporadic case, of the other. Bacteriology also bears this out.
AETIOLOGY:

The fons et origo of the disease is an organism, and to Still, must the credit of the discovery be given. In 1887, Weichselbaum discovered the organism of epidemic cerebro-spinal meningitis, and to it, he gave the name of "Diplococcus intracellularis meningitidis", so called from the fact that the diplococci are found within the cells. In 1897 Still discovered the organism of the present disease. It has a close resemblance to Weichselbaum's organism. In his own words (4) "The diplococcus of Posterior Basic Meningitis shows only slight differences from diplococcus intracellularis of epidemic cerebro-spinal meningitis. It differs widely from the pneumococcus and is easily distinguished by culture and inoculation. The diplococci are very small, average length being 1.2 m. to 1.5 m. The opposed surfaces of the two cocci are more or less flattened and separated by a narrow clear space. Shows marked tendency, especially in culture, to grouping in pairs, side by side, so that an appearance like Tetracoccus is produced. It is aerobic and is easily cultivated at a temperature of about 37°C. Its vitality is greater than the pneumococcus or diplococcus intracellularis,"
intracellularis, while its virulence on inoculation is less. The diplococci are found both free in the cells of the exudation and in the cerebro-spinal fluid during the acute stage of the disease, but they disappear usually after a few weeks, as the inflammation subsides. Occasionally, mixed infection may occur - but rarely. Channel of infection not ascertained. No evidence that through the ear. Has pathogenic influence on guinea-pigs, rabbits and mice, if injected intraperitoneally, but not subcutaneously." The above statement has been abundantly verified by different observers and in a considerable number of the present cases, this organism has been found, as will be seen on perusing the notes of the cases. We have here then a ready means of diagnosis. By performing a lumbar puncture, the organism is readily seen by staining with any of the simple stains. It is readily cultivated on blood-agar and has the appearance of "thick white paint". It grows readily in broth, unlike intracellularis, but the most important difference is the much greater vitality of Still's organism. According to Still, "Weichselbaum lays diplococcus stress upon the remarkably short vitality of intracellularis as one of its chief characteristics, and states/
states that in no case did it live beyond the sixth day; whereas, the diplococcus of posterior basic meningitis shows in almost all cases, a maximum vitality of twenty-four to thirty-four days on ordinary media (agar-agar and glycerine-agar) and on blood-agar lives considerably longer (fifty-three days in one case)." Still has also found the diplococcus in pure growth in the exudation about the joints, which is occasionally found complicating the disease. One can confirm Still's statement as to the organisms not being found in cases which have lasted for a considerable time. In such cases, however, there is not much difficulty in diagnosing the condition from the tuberculosis (which is always the chief difficulty), as the tuberculous variety never lives so long.

SOURCE OF ORGANISM:

The question arises, what is the source of the organism? The fact that the trouble always starts at the same place, suggests a local source and not a general one. There is no evidence, and as far as I know, no one has discovered, that the organism is in the blood. In one case where pure cultures were got from a lumbar puncture, Dr Stuart M'Donald withdrew some blood from a radial/
radial vein, but the culture was sterile.

Drs Lees and Barlow suggest that infection occurs through the naso-pharynx, thence through the eustachian tubes to middle ear and brain. From the position of the internal auditory meatus to the effusion at the base - the latter being often just over the former; this seems a likely theory, but there is very little proof that it is so.

Out of twenty-eight cases, I find that there is a history of otorrhoea, previous or present, in only three cases. This cannot be considered a large number, when one remembers the frequency with which one sees this condition in children, especially of the Hospital class. On going through the records of 28 cases of tuberculous meningitis, also in the Sick Children's Hospital, I find that there is a previous history of otorrhoea also in their cases. I do not see, therefore, that one has any reason to infer that the otorrhoea in the simple basal cases had anything to do with the condition.

Notwithstanding this, one cannot get away from the fact, that anatomically the middle ear presents the most natural part through which infection might occur. Lees and Barlow point out that/
that it has been shown that: "The eustachian tube is shorter, wider, and more horizontal in the child than in the adult: and that the petro-squamosal suture, which passes through the roof of the tympanic cavity, is still incompletely ossified, bringing the membranes of the brain and its sub-arachnoid space into close relations with the lining membrane of the middle ear."

The fact that otorrhoea is absent does not put the above theory out of count; but it is still sub judice.

PREDISPOSING CAUSES:

Season: Everyone seems to be agreed that the disease is most common during the winter and spring months. Such is my experience also. It is to be noted that epidemic cerebro-spinal meningitis is more common during these months, due, it is said, to the greater herding together during the cold season. It will be seen from the table below that three-fourths of the cases occur during the first six months of the year.
During 1900, 13 cases were admitted. " 1901, 8 " " " 
" 1902, 9 " " " 
So that there is no sign here of increase of the disease nor of any epidemic.

Sex:

Of my thirty cases, seventeen were males and thirteen females. The disease then appears to be about equally common amongst the sexes, with the majority to the males.

Age:

The disease is one of infancy. The oldest child in my series was three years and ten months, and the youngest six weeks. It occurs in older children. Still and Lees and Barlow mention one eleven years of age. The majority are under one year and this is a very important point in the differential/
ferential diagnosis of the condition.

I find that the average age (at incidence) is thirteen and a half months, while 66.6% of the thirty cases occur under one year.

As a comparison, I have taken all the cases (sixty-nine) of tubercular meningitis admitted into the Sick Children's Hospital during the three years 1900-1902 and I find their average age to be twenty-nine months. They occur most commonly and almost equally during the second and third year respectively - 42%, the next commonest being during the fourth year. I append the table below as regards the ages at onset in tubercular meningitis in the sixty-nine cases.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
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<tbody>
<tr>
<td>First</td>
<td>6</td>
</tr>
<tr>
<td>Second</td>
<td>14</td>
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<tr>
<td>Third</td>
<td>15</td>
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<td>Fourth</td>
<td>9</td>
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<td>Sixth</td>
<td>4</td>
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<td>Seventh</td>
<td>8</td>
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<td>Eighth</td>
<td>6</td>
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<td>Ninth</td>
<td>2</td>
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<tr>
<td>Tenth</td>
<td>0</td>
</tr>
<tr>
<td>Eleventh</td>
<td>2</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>69</strong></td>
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</table>

During first year................. 6 cases.
(Cases are only admitted up to twelve years of age.)
The difference between the two is striking and speaks for itself.

**INFLUENCE OF PREVIOUS ILLNESSES AND ACCIDENTS:**

**Injury:** In only two cases was there any history of a previous injury. One of these was most indefinite and I do not think that either can in any way be connected with the subsequent trouble. Lees and Barlow mention injury as a possible cause, but from a perusal of the present cases, one would be inclined to say that it was only incidental.

As regards antecedent diseases, it is noticeable that the great majority of the cases were fed on the breast and were quite healthy up to the onset of the meningitis. Ordinary infants' troubles have occurred in a few, but one might almost say less frequently than in ordinary healthy babies. There is a history of exanthemata in only three cases, with a doubtful fourth, and beyond one case of bronchitis, and three cases of diarrhoea, there is absolutely no history of previous illness. This is, I think, somewhat striking, and contrasts markedly with the tuberculous variety. Dr Stuart M'Donald has pointed out to me that he has never seen tuberculosis in connection with infants suffering/
ing from simple basal meningitis, and certainly there has been none noticed in the present cases.

In no case is there a definite history of congenital syphilis. At one time this was looked upon as a possible cause, but it is evident that it is not so.

From the acuteness of the onset, in apparently previously healthy children — and in some cases the onset is accompanied with fever — one might expect that the disease would be infectious, but there has been little evidence to shew this. Such an idea is based on a possible relationship to the epidemic cerebro-spinal variety. An interesting fact in favour of its being contagious is, that amongst the thirty cases, two of them were twins, the one being affected one month after the other. There is no further evidence of contagion however. One has never seen the cases of simple basal meningitis affect any of the other cases in the ward.

As regards heredity, there was nothing in the family history of the twins to suggest any constitutional taint.

MORBID ANATOMY:

As a result of the lepto-meningitis, and if the/
the patient lives long enough, purulent lymph is deposited beneath the arachnoid. The most common situation is at the reflection of the arachnoid from the cerebellum to the medulla posteriorly, and also anteriorly, extending forwards as far as the optra chiasma. In every case I have seen in the post-mortem room, the whole, or part of these regions has been affected. As stated before, almost invariably the cord becomes secondarily affected, while in the very acute cases the exudation may extend along the Sylvian fissure and longitudinal fissure on to the vertex. As such cases terminate early, the exudation at the base is then more scanty, but is always present. In all cases the inflammation may spread up into the third and lateral ventricles, which may contain flakes of lymph and be filled with a misty fluid.

As time goes on, this exudation - just as in the pleura and peritoneum, - becomes organised and fibrous. As a result of this, the openings from the different ventricles, and especially the foramen of Majendie and the foramina of Luschka become constricted; the fluid in the ventricles becomes retained, and hydrocephalus develops. At first, during the acute stage, the retained fluid is misty and/
and may contain flakes of lymph, but during the chronic stages, it may be perfectly clear, is of low specific gravity, and contains little albumen, thus negativing any idea that it may be inflammatory in nature. It can easily be understood, that in the cases terminating fatally and quickly, as in cases that have spread to the vertex, hydrocephalus is rarely present, as the patient dies before any organisation has taken place.

This is, I think, the explanation why, as Lees and Barlow, say, "in the vertical class the ventricles are frequently normal; in some cases they share slightly in the inflammatory extension, and may contain a little purulent lymph: but, as a rule, they are not distended in this form of meningitis." I have already pointed out that Lees and Barlow include amongst the vertical class, meningitis due to pneumococci, and in such cases, certainly the ventricles may be normal, as the base of the brain may not be affected. In meningitis due to Still's organism however, the base of the brain is always attacked, even in the vertical variety, and in most cases the ventricles.

Round the cause of the subsequent hydrocephalus there has raged, and still rages, a considerable/
able controversy. These are the main theories:

1. That closure of the foramen of Majendie and the foramina of Luschka occurs as a result of the surrounding meningitis. The fourth ventricle, and also the third and lateral ventricles are thus shut off from the general subarachnoid cavity, and as a result of this, the cerebro-spinal fluid accumulates, leading to hydrocephalus.

2. That there is pressure on the Veins of Galen, leading to congestion of vessels, with consequent exudation of serum into the ventricles.

3. That the exudation is comparable with pleural and peritoneal effusions, the arachnoid being a secreting membrane, and there being some disparity between the secretion and absorption.

The second theory has been, for the most part, discredited. Dr Halliburton, and Drs Lees and Barlow have shown that the fluid in hydrocephalus, and the normal cerebro-spinal fluid differ considerably in composition from any transudation from the blood. Dr Halliburton therefore considers it to be a secretion.

In all the post-mortems, that I have seen, in which hydrocephalus was present, there were adhesions binding the cerebellum to the medulla.
Only a few days ago, I saw a post-mortem in a case of hydrocephalus following post basic meningitis, where, even after a part of the cord had been removed - the brain being not yet opened - a certain amount of tension could still be felt through the dilated anterior fontanelle. I do not see how one can get over the fact that there was obstruction here.

On the other hand, one has tapped several cases of hydrocephalus through the third lumbar interspace with the result that the tension of the anterior fontanelle was immediately lessened. In one case, tapping was performed twice, with, on each occasion, a lessening of tension in the anterior fontanelle, the tension reappearing gradually after each tapping.

These two cases are certainly conflicting. I think the probable explanation is that there is an obstruction, but not an occlusion of the passages leading from the lateral ventricles. An analogy here is found in hydronephrosis following an incomplete, or an intermittently complete obstruction of the urinary passages.

As stated above, from the composition of the fluid found in a case of hydrocephalus, Drs Lees and/
and Barlow conclude that it is a secretion, and arguing from this, they consider the choroid plexuses as analogous to the renal glomeruli, and suggest that the fluid is filtered off by the epithelium covering the plexus.

Now it is known that in complete and permanent blockage to the ureter, hydronephrosis does not occur, but suppression, and it is quite legitimate to apply the same analogy to the ventricles, if their theory be correct. I therefore submit, arguing on their theory, that the hydrocephalus is due to an incomplete obstruction of the passages leading from the lateral ventricles to the general subarachnoid space.

Another reason for my thinking that the obstruction is incomplete, is that in many cases, the tension within the skull, in cases of increasing hydrocephalus, and as felt through the anterior fontanelle, varies greatly from day to day and even from hour to hour - at one time it may be high, at another, low. The symptoms in such a case vary with the tension, becoming worse when the tension is high and vice versa.

As a result of the hydrocephalus, the cerebral convolutions are flattened, and the cerebral cortex thinned.
SYMPTOMS:
1. General Description:

A child, generally under one year old, almost invariably under two years, who has had good previous health, is suddenly attacked with persistent vomiting. The child may be feverish. The vomiting continues, with little relation to food. Associated with this is constipation, or less frequently, diarrhoea. A day or two after the commencement, the head of the child is noticed to be "hanging back". The child is dull and listless, but is irritable if touched.

Such may be taken as perhaps the most frequent history. In many cases, there is a history of "convulsions". This term, however, is used very loosely by the lay public, and it is doubtful if such a history can be accepted in many cases. Except in the very acute variety spreading to the vertex, general convulsions are very rare. It may however, form an early symptom, but more commonly, it consists of some general rigidity with rolling of the eyes.

In a few cases the disease starts insidiously with drowsiness and head retraction, the disease advancing steadily up to a certain point.
In whatever way the disease started, most of the cases on admission show fairly similar symptoms and signs. The following may occur in a well-marked typical case:

A. GENERAL APPEARANCE:

Child looks very ill. Is very emaciated. Skin is harsh, dry and wrinkled. There is no flushing or eruption of the skin. The child lies quite still, - like a log - apparently unconscious, and days may pass without the child making any sound, or taking any notice of anything. The head may be somewhat globular in shape, if hydrocephalus is commencing. Anterior fontanelle is dilated, bulging and tense. Head greatly retracted, and some opisthotonos may be present.

The muscles of the back, but especially of the neck, are rigid. Any attempt to straighten the neck seems to cause pain, the child moaning. There is no hydrocephalic cry.

Temperature on admission was about 100° F., is now normal or subnormal, with perhaps an occasional rise to 100° F.

B. SYSTEMS:
B. SYSTEMS:-
1. Nervous System:

Cranium, Fontanelle and spine already seen. Drowsiness and irritability already described. Very rarely is there any delirium or excitement.

Sensory Functions:
Eyes: The child appears to be blind, (this is always difficult to elicit in such young children.) There is no photophobia, but the eyes appear staring, owing to the retraction of the upper lid, the sclerotic thus showing above the cornea.

Pupils are equal and moderately dilated. They may be contracted however. They react sluggishly to light. Nystagmus is occasionally seen, but not frequently. Very rarely is there a squint or ptosis. On ophthalmoscopic examination no neuritis found.

Ears and Nose normal. Very occasionally otorrhoea is present, but this possibly accidental.

Motor Functions:
Muscles: There is no paralysis.

The posterior cervical muscles are rigidly contracted and perhaps also the muscles of the back and even of the limbs. There are no clonic spasms.

Reflexes/
Reflexes:

a. Superficial frequently normal. In some cases extension of the large toe (Babinski's sign) is present.

b. Tendon rather exaggerated on both sides.

Vasomotor and Trophic Functions:

There is great wasting. Very rarely is there any flushing of the skin, it being dry and frequently wrinkled.

2. Circulatory System:

Heart normal.
Pulse in the majority is normal or increased in rate. In quite a number, even in young infants, it is slowed as I shall show later. It is almost invariably regular, unlike tuberculosis, but in a few cases of the series irregularity was noticed.

3. Respiratory System:

Breathing is quiet and regular. In a few cases it is irregular. It may be a little quickened. There is no sighing. Cheyne-Stokes respiration very rarely occurs.

Lungs generally normal. It should be noted, however, that several cases at the post-mortem have been found to have slight broncho-pneumonia which had not been noticed during life. Such is probably only/
only terminal and may have been the ultimate cause of death.

4. Digestive System:

Vomiting is frequent and continuous. In some cases, it has continued from the commencement of the trouble right on to the death of the patient, many weeks later. In a few cases, vomiting may be absent, except at the start. The vomiting has no relation to food.

In tuberculous meningitis, the vomiting, which is usual during the earlier part of the disease, is generally absent towards the termination unlike the simple variety. The bowels are more commonly confined than loose; they are seldom regular. The abdomen has some tendency to become cup-shaped and in case No. 11, this was most marked, but this sign is never so evident as in tuberculous meningitis. This seems to be the general experience.

5. Integumentary System:

No rash to be seen.

6. Genito-Urinary and Haemopoietic Systems:

Normal.
7. Locomotor System:

Kernig's sign I have found present in all the cases I have seen, i.e., on flexing the thigh on the abdomen and the leg on the thigh, it is impossible to completely extend the knee. Swellings in and around the joints are described by Lees and Barlow and seem to have been seen by them quite frequently. Still has also grown a pure culture of his diplococcus from the periarthritic effusion. No joint affections have been noticed in my series.

FURTHER COURSE OF EVENTS:

In the majority of the cases, the child goes steadily down the hill; the head retraction and opithotonos may steadily increase, with, however, occasional intermissions, during which the child seems better in every way. The temperature, which may have been subnormal throughout, frequently rises just before death.

Clonic spasms or General Convulsions are quite exceptional in the chronic variety. There are all varieties, from the above well-marked case, to a very slight one. In all, the outstanding sign is stiffness of the neck and retraction of the head. This sign is never absent during the whole or part of the disease.
In the very acute class spreading to the vertex, the symptoms are somewhat different. In them, clonic spasms are more noticeable and the trouble may commence with a general convulsion. The illness only dates from a day or two before admission, and it terminates fatally usually within a week.

**DURATION OF CASES:**

As regards the duration of life of fatal cases, one of my series lived a hundred and eight days, whilst the shortest was five days. I find the average duration of life of the fatal cases to be fifty days. This forms again a marked contrast to the duration of life in tuberculous meningitis - most of which die within three weeks. It is quite impossible to give the duration of the cases which recover, as it is very difficult to say when such cases are really cured - in the majority the "cure" is only partial.

Before going on to any detailed description of some of the above symptoms, I wish now to tabulate briefly the cases on which I found this paper. Many of the cases I have had under personal observation, while others I have taken from the hospital records. In some, the notes are very scrappy and unfortunately, this applies to the post-mortem accounts. Owing to the post-mortem book having gone amissing/
amissing, I have to depend for the most part on the notes in the case books themselves.

I have tabulated the cases in the order of their admission to hospital.
CASE 1: Maggie Monroe, aet. 1 year, admitted March 31st, 1900; dismissed improved, 19th May, 1900.

Complaint: Vomiting and inflammation in head of 14 weeks' duration.

Previous Illnesses: Breast-fed. No previous illnesses.

Present Illness: Started fourteen weeks ago with vomiting. This has continued ever since. Very costive. Has taken "fits", with squinting of eyes. Head retracted fro twelve weeks. Getting very thin, very irritable.


Treatment: Pot: Iodid gr. 1½ T.I.D.

Pot: Bromid: gr. iv. T.I.D.

Progress/
29.

Progress: Dismissed 19th May, 1900 improved. Rigidity less. Temperature was subnormal throughout illness.

Child brought up for inspection on June 10th, 1900 - "much improved, and can now see."

CASE 2: Robert Inglis, aet. 1 year 10 months; admitted April 5th, 1900; discharged, cured, May 12th, 1900.

Complaint: Vomiting, wasting, restlessness of four weeks' duration.

Previous Illnesses: Breast fed. Measles at 10 months.

Present Illness: Vomiting for last four weeks. Very restless and screaming at night.


Temperature 97.4°F. Pulse 120, feeble and varying in rate. Respirations 32, are quiet and regular. Heart and lungs normal.

Progress: April 11th, vomiting frequently. Slight head retraction.
April 26th: Twitching of left side of face, also of left arm and leg.

Got chloral and chloroform, which stopped the fits.
May 12th: Dismissed cured. Temperature subnormal throughout. Pulse at times fell to 99 (normal at this age = 105.)

CASE 3: Barbara Hanson, aet. 3½ years, admitted 23rd April, 1900; died 23rd April, 1900.

Complaint: Screaming, vomiting, pain in back of head - duration two months.

Previous Illnesses: Breast fed: had measles, whooping cough and chicken-pox.

Present Illness: On January 5th, fell on back of head. Following this was drowsy and screamed for three days. At end of February, vomiting for a time - no relation to food, and was very costive. On April 13th, vomiting started again, also great pain in back of head, screaming.
CASE 4: Mabel Carswell, act. 2 years 7 months, admitted June 19th, 1900; died, July 25th, 1900.

Complaint: Vomiting and retraction of head (for seven weeks). Blindness (for two or three days - now gone.)

Previous Illnesses: None. Breast-fed.

Family History: Mother two miscarriages (first two pregnancies.)

Present Illness: Started suddenly seven weeks ago with vomiting and at the same time head was retracted. Both symptoms have continued up to admission. Became blind two weeks ago, but recovered sight after a few days.


Treatment: Pot: Iodid: and Pot: Bromid: ice cap to head.

Progress: June 4th: Slight twitchings of facial muscles round eye. Some nystagmus.

July 2nd/
State on Admission: Child dazed looking. On sitting up cannot hold up head. Head appears large and is distinctly retracted. Eyes staring - sclerotic visible above cornea. No squint. No nystagmus. No otorrhoea. Cries frequently. Pulse 80 (normal is 100) and very irregular at times. Temperature 97.2° F. Respirations 32, quiet and regular. Abdomen not retracted. Heart and lungs normal.

Progress: At 10.15 p.m., whole body rigid and extended. No squint. Pulse fairly good. Respiration very feeble and at long intervals - not Cheyne-Stokes. Respiration failed suddenly - the heart continuing to beat for some time after respiration ceased.

Sectio: Marked flattening of convolutions of both sides. Extremely well marked chronic basal meningitis, with delicate adhesions between pia and dura. Marked matting of membranes in region of fourth ventricle with closure of foramen of Majendi. No tubercle. Great dilatation of lateral ventricles, of foramen of Monro, of third ventricle, of iter, and of fourth ventricle. All cavities filled with a clear, watery fluid. All other organs quite healthy, except some emphysoema of lungs.
July 2nd: Child not so well. Lies very quietly. Temperature, which has been subnormal since admission, is now 99° F.


CASE 5: May Glossy, aet. seven months, admitted 18th June, 1900; discharged 25th August, 1900 - cured.

Complaint: Retraction of head for three weeks.

Previous Illnesses: None. Breast fed.

Present Illness: Head been retracted for three weeks. Day before admission vomited frequently and squinted. Crying out at times.


Treatment/

Mercury ointment locally to head, also ice-cap.

Progress: June 28th: Fontanelle tense. Still sick.
June 30th: Lumbar puncture performed. Fontanelle less tense following this.

Temperature rose to 100°F during first two days after admission and following that, was subnormal throughout. Pulse at times fell to 90 (normal = 105.)

Sept: 6th: Returned, complaining that "can't hold head up well." Head is retracted and child does not appear to see. Dismissed improved.

Sept. 24th: Readmitted again with increased head retraction and vomiting. Occasional squinting and feverishness.

Oct. 2nd: Dismissed improved, but the head retraction still present.

CASE 6: James Budge, aet. 6 months, admitted 22nd June, 1900; died 27th June, 1900.

Complaint/
Complaint: Squinting - for 2 weeks.

Previous Illnesses: Had otorrhoea when two months old. Bronchitis. Fed on breast and bottle.

Present Illness: Had Bronchitis 4 weeks ago. Never been well since.

Eyes been staring for some weeks. Squinting occasionally during last fortnight. Head retraction and neck rigid for one week.


Head is well retracted; fontanelle is tense. Eyes fixed and staring. Pupils medium size, but the left is slightly larger than the right. They react to light. No squint, no otorrhoea. Sick occasionally. Bowels regular. Abdomen not retracted. Pulse 144, is regular. Temperature 98.4°F. Respiration normal and quiet. No cry. Heart and lungs normal.

Treatment: Ung: Iodoform (5%) Calomel gr. i at bedtime.

CASE 7: James Carnegie, aet. 1 year 9 months, admitted June 29th, 1900; died July 23rd, 1900.

Complaint: Vomiting with constipation - 2½ weeks.

Previous Illnesses: None. Breast fed.

Present Illness: Commenced 2½ weeks ago with a fit of vomiting.

Bowels very active. Mouth occasionally twitches. Head retraction. Stares greatly, very irritable.


Progress: July 3rd: Head retraction increased. Tremors and twitching of lips.

July 23rd/
July 23rd: Death. Temperature was subnormal till July 22nd, and on July 23rd it reached 100° F.

Sectio: Marked hydrocephalus of skull. Exudation of lymph, sharply limited over cerebellum. Foramen of Majendi obstructed. All ventricles dilated, the 3rd notably raising up the optic chiasma, and appearing in front of it as a globular translucent swelling.

CASE 8: George Drummond, aet. 9 months, admitted July 9th, 1900; died July 31st, 1900.

Complaint: Vomiting, squinting, retraction of head - fourteen days' duration.

Previous Illnesses: None. Fed on breast and bottle.

Present Illness: Quite healthy till fourteen days ago; became very fretful and feverish. Head was "full". Next day vomiting, head retraction and squinting noticed.

Abdomen not retracted. Pulse 140 and regular, tension rather high. Respiration, lungs and heart normal.

**Treatment:** Pot: Bromid:

**Progress:** July 29th: Head retraction increasing. Sickness frequent. Temperature, which had been normal, is rising.

July 30th: General convulsions. Lumbar puncture performed, the fluid coming out with a jet.

July 31st: Death. Sickness continued throughout.

**CASE 9:** Alex. Murray, aet. 3 years, admitted July 21st, 1900; died July 25th, 1900.

**Complaint:** Twitching - one day's duration.

**Family History:** Father epileptic. One brother died of tuberculours meningitis.

**Previous illnesses:** None. Fed on breast.

**Present Illness:** Running about quite well till yesterday at mid-day "fell down in convulsions." Became comatose. Frequently sick. No squinting.

**State on Examination:** Nutrition very good. No eruptions. No tâche cerebrale. Generally lies quietly, at times very restless. Expression dull, but/

Treatment: Pot: Bromid.

Progress: July 24th: Temperature steadily rising, reached 105° F. to-day. Ice-cap applied. From a Lumbar puncture, 3 vi turbid fluid got. Temperature fell after this.
July 25th: Death.

Sectio: Lungs congested - no consolidation. Brain shewed marked purulent arachnoiditis in interpedun-
cular space, along fissures, and over whole frontal region. No injury, no ear disease, no nasal dis-
ease visible.

CASE 10: Alice Fagan, aet. 6 months, admitted Aug-
ust 9th, 1900; died August 16th, 1900.

Complaint: Hanging back of head - since two or three days.

Family history/
Family History: Two brothers died of "fits".

Previous Illnesses: Snuffles(?) when a few weeks old. Fed on breast and bottle.

Present Illness: Head been hanging back and eyes staring for three days. Very fretful.


Treatment: Hydrag: Oleat: locally to head. Pot: Iodid. and Sod: Bromid. internally. Also Calomel gr. \( \frac{1}{6} \) two hourly.

Progress: April 12th: Spasmodic movements of arms and legs. Head retraction, squint and nystagmus are marked. On lumbar puncture \( \frac{3}{11} \) fluid withdrawn.


Aug. 15th: Puncture through right angle of anterior fontanelle and \( \frac{3}{iv} \) of clear fluid withdrawn. Head retraction/
extraction extreme, with squinting.

Aug. 16th: Temperature rose to 102.4° F. just before death.

CASE 11: Jeannie Roberts, aet. 16 months, admitted October 25th, 1900; died November 21st, 1900.

Complaint: Vomiting and wasting - 7 weeks' duration.

Previous Illnesses: None. Fed on breast.

Present Illness: 7 weeks ago became listless and drowsy. Vomiting ensued two weeks later, and has continued ever since. Is costive. Two weeks ago mother noticed neck stiff. No fits, gradually losing flesh.

State on Admission: Great emaciation. Skin harsh, dry, and wrinkled. Child likes like a log, apparently unconscious, but cries if handled. Head greatly retracted and whole body rigid. Left knee flexed; right leg straight. Both wrists acutely flexed. No clonic spasms. No opisthotonos. Fontanelle depressed. Maximum circumf. of head = 17"; circumf. from ear to ear (parietal) = 12"

Child blind. Fundi normal. Slight vertical nystagmus/
tagmus. No squint. Great retraction of upper lids.


Treatment: Pot: Iodid. internally and Mercury locally.

Progress: Oct. 29th: Left hand = main-en-griffe position - flexed at wrist, dorsi-flexed at metacarpo-phalangeal joints, and flexed at inter-phalangeal joints, except those of the first digit, which are extended. Arms are over-pronated and elbows extended.

Nov. 9th: Head retraction and opisthotonos so marked that head and shoulders are at right angles to the axis of the rest of the body. Slight clonic movements of arms, legs rigidly extended, and toes pointed. Getting Chloral and Bromide.

Nov. 10th: Pulse regular = 140. Breathing quiet, irregular and quick. It quickens and slows at times.

Nov. 21st: Rigidity continued till death to-day.

Temperature was subnormal till Nov. 5th, but has been swinging since then, and latterly reached 104° F./
104° F. Great purulent discharge from thrush of mouth during last week. Pulse at times was as slow as 84 (normal = 105.)

Sectio: Broncho-Pneumonia left lung lower lobe. "Brain" shewed posterior basal meningitis with usual conception. Flattening of convolutions from distention of lateral ventricles.

CASE 12: John Malloy, aet. 17 months, admitted December 15th, 1900; died, January 11th, 1901.

Complaint: Vomiting and head retraction - twenty days' duration.

Previous Illnesses: None. Fed on breast.

Present Illness: Commenced with feverish attack, vomiting, and head retraction. Became drowsy; during the last fortnight, some twitching of the lips and strabismus. Bowels costive.

Treatment: Pot: Bromid.

Progress: Dec. 19th: Sent home as measles in ward - improved.
Dec. 27th: re-admitted. Internal squint of left eye. Child unconscious; otherwise, as before.
Dec. 29th: Vomiting frequently.
Jan. 11th: Death. Temperature was normal till Jan. 1st, after that very swinging, and rose to 106°F before death.

CASE 13: Robert Sinclair, aet. 8 months, admitted January 4th, 1901; discharged January 18th, 1901, cured.

Complaint: Vomiting and head retraction, - 10 days' duration.
Previous Illnesses: None. Bottle fed.

Present Illness: Commenced suddenly ten days ago with vomiting, which has continued since. Two days later, head noticed retracted. Later on, some squinting with rolling of eyes. Bowels regular.

State on Examination: Well nourished, skin rough. No eruptions: tâche cerebrale slight. Head rickety and very slightly retracted. Anterior fontanelle/

Treatment: Pot: Iodid. internally and mercury locally.

Jan. 18th: Made uninterrupted recovery. Temperature fell to normal on second day and remained so. Sept. 17th, 1901: Came up to-day - "Thriving splendidly".

CASE 14: Thomas Malone, aet., 14 months, admitted January 5th, 1901; died January 20th, 1901.

Complaint: Stiff neck for one week.

Family History: Mother had "many miscarriages".

Previous Illnesses: None. Breast fed.

Present Illness: Cough six weeks ago - never gone away. Neck became rigid a week ago. No vomiting or fits. Bowels regular. Eyes were "working" day before admission.

State on Examination: Nutrition fair. Apparently unconscious/


Progress: Jan. 20th: Got steadily worse, with no outstanding features and died to-day.

Sectio: Large collection of greenish pus under arachnoid at base, extending from under surface of cerebellum forwards over pons and down medulla, over interpeduncular space and chiasma to posterior part of orbital surface of temporal lobes, and for a distance up Sylvian fissures. The collection over cerebellum was so great as to form a hollow in it. In the same region, just behind foramen magnum to either side posteriorly, the arachnoid was adherent to dura, over an area the size of a sixpence. No tubercle. Other organs healthy.
CASE 15: Hugh Mackintosh, aet. 4 months, admitted February 9th, 1901, died March 12th, 1901.

Complaint: Diarrhoea and full fontanelle, six weeks' duration.

Previous Illnesses: None. Fed on breast.

Present Illness: Diarrhoea commenced along with retraction of head, six weeks ago. Continued since. Vomits frequently. Head bulged.


Feb. 11th: Lumbar puncture. Pathological report:

"Distinct opacity. Microscopically shows a large number of leucocytes, mostly polymorphous, but also many mononuclear, large and small. No organisms found."

March 12th: Child gradually weakened and died today.

Temperature subnormal for the most part.

Sectio: "Fairly chronic simple basic meningitis extending well forward past commissure and backwards to cerebellum. Punctures were made before post mortem done, and the result gave no growth on culture. Great dilatation of both ventricles. Mesenteric glands enlarged and congested. Death probably due to the diarrhoea complication.

CASE 16: Robert Nicol, aet. 4 months, admitted February 18th, 1901; died April 1st, 1901.

Complaint: Vomiting - three weeks' duration.

Previous Illnesses: None. Bottle fed.


Progress: February 24th: Some head retraction and diarrhoea.

March 7th: Unconscious. Temperature subnormal. Lumbar puncture gave a "pure culture of meningococcus."

March 10th: Head noticeably enlarging, circumference = 16½"; lambdoidal sutures open.

March 18th: Circumference of head 16½"; transverse circumference = 12½".

March 27th: Circumference of head 17": Transverse circumference = 13½".

April 1st: Circumference of head 17½": Transverse circumference = 13¼".

Patient died to-day. Patient been like a log for last month. No spasms. Pulse occasionally was as low as 80 per minute (normal 115).

Sectio: Fairly acute basal meningitis, there being still some yellowish-green lymph. All the ventricles are greatly distended with clear fluid, and cerebral wall thin. A few adhesions in cord. Bronchopneumonia in both lungs.
CASE 17: Lizzie Wright, aet. 2 years, admitted March 4th 1901, discharged April 13 1901 cured.

Complaint. Stiffness of neck - 16 days duration.

Previous Illness. None. Breast fed.


Treatment. Pot: Iodid and mercury by the mouth. March 7th, Lumbar puncture showed meningococcus. March 2nd, Diarrhoea and temperature swinging. April 3rd, Constipation and temperature normal.

Frequently sick.

April 13th, left to-day "cured". Head retraction gone.

September 16th, Came up to-day. Has occasional headache and suffers from sleeplessness.
CASE 18: Robert Craigie, aet. 3 years 10 months, admitted November 23rd, 1901, discharged May 17th 1901, cured.

Complaint: Head retraction. Vomiting. Raving - three days duration.

Previous Illnesses: Whooping cough when 14 days old. Breast-fed.

Present Illness: Commenced suddenly three days ago with raving and vomiting. Head became retracted and spasms of limbs and rolling of eyes occurred. Very costive.

State on Examination: Listless, lies huddled up and appears to be unconscious. Head markedly retracted. Some opisthotonos. No blindness. Pupils equal and moderate in size. No squint.


Treatment: Ung. Iodoform (10%) on head.

Progress/
Progress: March 25th duller. On lumbar puncture—only a few drops of misty fluid got. This contained "pure culture of meningococcus in large amount."

April 10th. Child doing well till to-day some rigidity of right leg. Temperature up.

April 24th. Greatly improved.

May 17th. Discharged "cured."

October 14th: 1902. Child returned to-day. Has been quite deaf since illness. Otherwise quite well and intelligent. Can only say a few words.

CASE 19: Ellen Mitchell, aet. 19 months, admitted March 25th, 1901, died March 27th, 1901.

Complaint: Vomiting. Costive. Four days duration.

Previous Illnesses: Measles last summer. Also chicken pox. Some diarrhoea occasionally. Breast fed.

Present Illness: Commenced suddenly four days ago with vomiting. Hardly conscious since then. Bowels costive. Crying out frequently. Rolling of hands, feet and head day before admission.

State on Examination: Child unconscious, if disturbed cries a little. Slight cyanosis. Nutrition poor/

Treatment: Stimulants.


Sectio: No tubercle. "Yellowish green lymph over lower surface of cerebellum and superiorly in middle lobe; also over tip of inferior temporal lobe and up towards vertex. Sub-arachnoid of spinal cord also inflected in its length.

CASE 20: Robert Mackenzie, aet. 22 months, admitted 7th August, 1901, discharged 11th August, 1901 - in statu quo.

Complaint/
Complaint: Swelling of head since six months old.

Previous Illnesses: Measles eight months ago.
Breast fed.

Present Illness: Had "fits" when a fortnight old. Quite well again till six months ago became dull, feverish; vomited and "buried head in pillow." Head been gradually enlarging and cannot lift up head. Is listless. Bowels rather loose.


Discharged August 11th, 1901.

Re-admitted February 19th, 1902. Circumference of head now $20\frac{7}{8}$". Cannot speak. Very dull and drowsy. Cannot feed himself. No rigidity. Taken home by parents on February 27th - in statu quo.

CASE 21: James Pitcher (vide photographs) aet. ten months, admitted March 6th, 1902. Died April 25th, 1902.

Complaint/
Complaint: Tossing hands, arms and legs during fully two weeks.

Previous Illnesses: None. Breast fed.

Present Illness: Began fully a fortnight ago by child throwing about its arms and head. Could not see at this time. Head retraction and drowsiness followed. On March 1st mouth twitched. Vomits occasionally and has diarrhoea.


Treatment: Pot: Iodid: internally and mercury locally.

Pot: Bromid when required.

Progress: March 14th, fluid from lumbar puncture "showed meningococcus," Some general twitchings over body. Plantar reflexes show extension of big toes.

March 22nd, General convulsion. Head retraction much more marked. Limbs rigid, especially left side/
At noon Mr. Stiles drilled a small hole through cranium and withdrew $\frac{3}{iv}$. of clear straw-coloured fluid from lateral ventricles. Child much collapsed after operation, and absolutely rigid - like a rod - with head retracted.

April 4th: Going downhill - some difficulty in swallowing. Lateral ventricles again explored. Diarrhoea bad: also vomiting.

April 20th: Temperature rising. Cheyne-Stokes' breathing.

April 24th: Purpuric rash over left side of thorax low down.

April 25th: Death. No sectio.

CASE 22: Alice Laing, aet. eight weeks, admitted April 10th, 1902, discharged June 3rd, 1902, in statu quo.

Complaint: Convulsions - duration 14 days.

Previous Illnesses: None. Breast fed.

Present Illness: Been taking frequent general convulsions for a fortnight. Some squinting. Head been retracted for some period.

State on Examination: Child drowsy. Marked head retraction/

Treatment: Pot: Bromid.

Progress: April 14th: three general convulsions today.
        April 18th: Convulsions continue frequently.
        May 5th: Fluid from lumbar puncture "contains meningococcus."
        May 15th: Right lateral ventricle tapped.
        June 3: Discharged in status quo. Ventricles were repeatedly tapped, with temporary relief.

CASE 23: Dorothy Railton, aet. eight months, admitted April 12th, 1902. Died April 21st, 1902.


Previous Illnesses: None. Breast fed.

Present Illness: Started four weeks ago with general convulsions: had about a dozen in twenty four hours. Soon after this, a rash "like chicken pox" appeared. Since then child been getting more and more drowsy. Very costive. Took severe convulsions/
sions on day of admission. Does not see (?).

State on Examination: Very ill appearance. Very apathetic, but peevish if disturbed. Lies on side with head much retracted and back rigidly arched. When laid on back, the neck, at summit of arch, is about four inches above the bed. Limbs not rigid. Reflexes normal. Pupils moderate and equal. React to light. No nystagmus or squint. No otorrhoea. Breathing quiet and regular. Pulse 144 and regular. Temperature 98° F. Lungs - dullness at bases with medium crepitations and vesicular breathing.

Treatment: Pot: Iodid and mercury locally.

Progress: April 20th: Steadily getting worse. Clonic spasms in head and arms at intervals. Lumbar puncture been performed several times and sometimes no fluid got.
April 21st: Death, the temperature rising from no-to 103.2° F. at death.

tricles, some softening of the wall. Some flakes of lymph in the choroidal plexuses, which are contracted. Fluid in ventricles slightly turbid. Spinal cord - flakes of yellowish lymph all down and well-marked in the region of cauda equina.

CASE 24: Morris Freedman, aet. 4½ months, admitted May 16th, 1902. Died May 24th, 1902.

Complaint: Fits - two weeks duration.

Previous Illnesses: None. Breast fed.

Present Illness: A fortnight ago became feverish at night and next morning a rash came out all over the body. During the afternoon and evening following this case had fits. Crying out constantly. Bowels active. Head retraction from the beginning.


Progress: May 24th: Rigidity and tension in fontanelle gradually increased till death to-day. Pulse generally quick, at times only 112 per minute (normal = 118).
CASE 25: Archie Anderson, aet. nine months, admitted 24th May, 1902, discharged 29th May, 1902. Worse.


Previous Illnesses: German measles and erysipelas at five months. Fed on bottle.

Present Illness: Dwindled since birth. Worse since erysipelas. Working mouth the last few weeks.

State on Examination: Head shows marked hydrocephalus, and is retracted. Arms flexed and legs rigidly extended. Great retraction of upper lids. No neuritis. Pulse 120 and regular. Temperature 97° F.

Treatment: Hydrarg: cum cret: night and morning.

Progress: May 27th. Sudden rise of temperature to 104° (was sub-normal before). Septic looking rash developed on body, but disappeared in two hours. Child fretful.

May 29th. Discharged. Temperature normal.

CASE 26: Catherine Fitzpatrick, aet. nine months, admitted 22nd August, 1902. Died 29th September, 1902.

Complaint/
Complaint: Vomiting and twitching of hands - duration two weeks.

Family History: Twin of case 27. Eight other brothers and sisters.

Previous Illnesses: None. Bottle-fed.

Present Illness: A fortnight ago began vomiting and shaking of hand. Feverish at the start. Head retraction been present for one week. Vomiting now stopped.


Treatment: Pot: Iodid and Bismuth mixture.

Progress: 25th March. Lumbar puncture. No meningococci found.
14th April: Vomiting frequently.
18th April: Arms and legs twitching. Chloral given.
19th April: Head more retracted and hands clenched.
29th April: Death. Considerable diarrhoea week before death.

Sectio/
Sectio: Ventricles and spinal cord distended with large quantity of cerebro-spinal fluid. Pia mater at base opaque and thickened. Other organs healthy.


Complaint: Cough and vomiting for six days.

Family History: Twin of Case 26.

Previous Illnesses: Otorrhoea (left) since four months old. Fed on bottle.

Present Illness: Commenced suddenly six days ago with vomiting and feverishness, but has had a cough for months.

State on Examination: Well nourished. No head retraction or eye signs. Some crepitation in lungs. Temperature 102° F.

Treatment: Liq: Ammon: Acetat: M xx T.I.D.

Progress: October 10th: Slight head retraction and opisthotonos.

October 11th: Head retraction increasing. Fluid from/
from lumbar puncture "contains meningococci." Vomiting occasionally.

October 29th: Child steadily improved since above and discharged to-day as cured.

CASE 28: Isabella Moffat, aet. nine months, admitted 22nd November, 1902. Died 24th December, 1902.

Complaint: Losing flesh during last three months.

Previous Illnesses: Vomiting and diarrhoea occasionally. On breast till five months old.

Present Illness: Child became restless and sleepless three months ago and has dwindled ever since. The mother says child's head is getting larger and is 'held back.'


Temperature 98° F. Heart and lungs normal.

Treatment:/
Treatment: Stimulants.

Progress: December 1st. Vomiting frequently. Rigidity as before. Fluid on several occasions been withdrawn by lumbar puncture with temporary benefit. December 24th. $\frac{3}{4}$ fluid withdrawn by lumbar puncture. Child died suddenly during the night.

CASE 29: Lizzie Collins, aet. eleven months, admitted February 7th 1900. Died February 11th 1900.

CASE 30: Francis Murray, aet. five months, admitted 5th May, 1900. Discharged 17th June, 1900—cured.

(Notes of last two cases amissing).

Special Features and Symptoms.

I wish now to take up in more detail some of the symptoms of this affection. Such details have been formed, for the most part, from a comparison of the symptoms presented in the above cases. They form a useful comparison also with the statistics of Lees and Barlow.

Mode of Onset of the Disease.

In eleven cases the primary symptom was vomiting.
" seven " " " " convulsions.
" five " " " " head retraction.
" three " " " " screaming or raving.
" two " " " " drowsiness.

In/
In quite a number of cases, the primary symptom was associated with feverishness, e.g., in case No. 24. "A fortnight ago became feverish at night. Next morning rash appeared all over the body and during the afternoon and evening had "fits". Crying out constantly." This history is most interesting and suggests at once a comparison with the epidemic variety.

The above statistics are very similar in results to those of Lees and Barlow. Doubtless the head retraction is present from the start in most cases, but as it commences very gradually, it has been masked by the more evident vomiting, etc. It was present in almost every case on admission when frequently one found it had not been observed by the parent.

As regards convulsions, this is frequently described in the histories, but is rarely seen after admittance and never to anything like the extent it is present in the tuberculous. In almost all, the onset was sudden, and in a previously healthy child.

RELATIVE FREQUENCY OF SIMPLE AND TUBERCULAR BASAL MENINGITIS:

That this is not a rare trouble is proved by Lees'
Photographs by Dr W. J. Stuart.
Lees' and Barlow's cases and also by the fact that thirty cases were admitted to the Sick Children's Hospital during the three years 1900-1902. As a point of interest, I have found the number of cases of tuberculous meningitis admitted to the same hospital during the same years, to be sixty-nine, so that the simple variety occurs nearly half as often as the tuberculous. This is a much larger proportion of the simple than most people would have imagined.

HEAD RETRACTION AND OPISTHOTONOS:

The head retraction may vary from a slight stiffness of the posterior cervical muscles to a most extreme degree, as witness the photographs of Case No. 21.

In severe cases the retraction of the head is associated with opisthotonos and this may be so great that the occiput almost touches the sacrum. This is a tonic spasm — never clonic — and is thus distinguished from tetanus. Though tonic, it varies in degree from time to time, the tension in the anterior fontanelle varying synchronously. The rigidity is great, so much so, that the child can be raised up like a board by means of the hand on the/
the occiput. In case No. 11, the opisthotonos was very marked.

In many cases also - I have notes of ten - the limbs are rigid. Most commonly, the lower limbs are rigidly extended with the toes pointed, while the elbows and fingers are flexed with the thumbs upper and lower inverted. In a few cases, both limbs are flexed. In case No. 11, an interesting contraction was seen at one stage of the disease. The arms were over-pronated, the wrists flexed, the metacarpo-phalangeal joints extended, and the phalangeal joints flexed except the first digit, which was extended, thus resembling the main-en-griffe seen in ulnar paralysis. The legs were rigidly extended. Later on, the hands took the closed fist position.

It seems reasonable to infer that the head retraction, opisthotonos, and general rigidity are due to irritation of the spinal nerves, as it has been shown that the spine is so frequently secondarily affected. The inflammation starts at the base of the brain, and thus from the irritation of the upper cervical nerves we get the head retraction which always appears before the other rigidities. The inflammation spreading down the cord, later on opisthotonos and general rigidity ensue. In every/
every case in which I have seen the spinal cord examined, it was affected. Sometimes in very chronic cases, the affection consisted of only a few adhesions, which might have been overlooked unless searched for. Lees and Barlow, however, mention one case where opisthotonos occurred and where the spinal cord was not affected. They point out, that Ferrier has shown that stimulation of the middle lobe of the cerebellum, and of the corpora quadrigemina, by a weak faradic current leads to head retraction and some spasticity of the limbs, and it may be that the inflammation affects these areas in such a way. In support of their theory, they also draw attention to the occasional occurrence of nystagmus, this being a recognised sign in affections of the cerebellum.

In most cases, as already stated, convulsions do not occur, (i.e., apart from the histories) unless the inflammation has spread to the vertex, when it occurs from irritation of the motor centres.

In none of the present series has any paralysis of the limbs been observed. Lees and Barlow mention the occasional occurrence of facial palsy, and in one case a hemiplegia.

"Champing"/
"Champing" movements of lower jaw, lips, and tongue described by Lees and Barlow as not uncommon, I have rarely seen. Grinding of the teeth has been present in one or two cases, but never with the frequency seen in tuberculous meningitis.

EYE AFFECTIONS: In the manner which simple posterior basal meningitis affects the eyes, we have a marked contrast to the tuberculous. It gives us an important link in distinguishing the two conditions.

To simplify matters, I have gone through all the cases and find the following:

Sight: Blindness occurs in five out of twelve cases noted. This is always a difficult symptom to elicit in such young children and it is possible that the average should be higher.

Curiously, although blindness is common, optic neuritis is rare, and in none of the eleven cases examined (by an oculist) was it definitely found. In two cases, it was doubtful. Why blindness should occur with no evidence of neuritis, I am unable to explain. It cannot be due to pressure, as fortunately, in all the cases which got better, the sight returned, even although hydrocephalus remained. It is possible due therefore to some/
some toxine evolved during the acute stage.

The reaction to light in most cases was sluggish.

Pupils: Are equal in sixteen out of eighteen noted.

Out of 18 cases:

- In 7 the pupils were dilated.
- " 5 " " " contracted
- " 5 " " " of medium size
- " 1 " " varied from time to time.

The above does not quite tally with what Lees and Barlow have found, namely, that contraction was the usual.

Photophobia: Was absent in all cases.

Nystagmus: Was noted in only two out of sixteen cases.

Squint was present in their cases out of nineteen noted - though quite frequently mentioned in the histories. In no case was ptosis present.

Retraction of the upper eye-lid was noticed in ten cases out of twenty-eight.

The above statistics speak for themselves and I will draw attention, when I consider the differential diagnosis, to the marked differences found in tuberculous meningitis.
Ears: Deafness occurs occasionally, as in case No. 18.

This patient was still quite deaf five months after his discharge from the hospital. He could only speak a few words.

Unfortunately, it seems to be the general experience that the deafness is frequently permanent. Occurring as it does in such young children, it necessarily leads to deaf-mutism.

Pulse: According to Lees and Barlow, the pulse is either normal or increased in rate. They say that in infants of not more than twelve months, they have "not found a slow pulse." The reason for this is given, that "the inhibitory control of the heart by the vagus is, in these young children (below twelve months), imperfectly developed."

The above has not been my experience. Undoubtedly, in the majority the pulse is either normal or increased in rate, but in quite a large majority, (I have notes of seven such) the rate is slowed. A notable one is that of case No. 16., (Robert Nicol, aged four months) where, during the last fortnight, the pulse ranged from eighty to ninety-six per minute (the normal at that age is about/
about one hundred and twenty.)

Again, in case No. 11, (Jeannie Roberts, aged sixteen months) the pulse for a considerable time ranged from eighty-four to one hundred per minute (the normal at that age is about one hundred and five.

Irregularity has only occasionally been met with (four cases), in marked contrast to the well known irregularity of the pulse, which occurs in the earlier stages of tuberculous meningitis. The constricted artery, as got in the latter disease, I have not seen in the simple.

Respirations: The breathing is, as a rule, regular, quiet and often quickened. Occasionally, sighing is met with, and sometimes the breathing is irregular, but this is rare. In one case (No. 11), the breathing was most irregular - quick one minute and slow the next. I have seen one case only of Cheyne-Stokes breathing.

In several instances, the patients have died suddenly, through failure of respiration, the pulse continuing to beat, and Lees and Barlow mention, that in some "cures", the patients have died suddenly. Can this be due to a sudden blockage of a hitherto/
hitherto patent foramen of Majendi, leading to pressure on the respiratory centre?

The Temperature in complicated cases, as a general rule, is normal or subnormal. This is much more marked than in tuberculous meningitis. Just before death, there is frequently a rise, often high, but in some of the worst cases, it may be subnormal throughout. To a certain extent, the temperature may be taken as a guide in the prognosis. In the acute and fatal, the temperature is generally raised; in the chronic and localised, it is more frequently normal or subnormal.

State of the Bowels: In the majority of cases, the bowels are confined. In a large minority, diarrhoea occurs, and in some, the two conditions alternate. In thirteen of the cases, I find a definite history of constipation, and in eight, diarrhoea. In four, the bowels were regular.

Looking to the frequency of diarrhoea in children, and to the fact that all patients were taking medicine by the mouth (always apt to produce diarrhoea in infants), I think one may fairly conclude that constipation is the rule.

Skin:
Skin: In four cases (Nos. 21 and 24, and 25) there is either a history of a rash, or one ensued after admission. In case No. 21, a purpuric rash developed just before death. I have already pointed out that this fact, along with others, tends to show that this disease has some affinity to the epidemic variety. In the latter, purpuric rashes are described.

DIAGNOSIS:

There is little difficulty in determining that the case is one affecting the meninges of the brain. The head retraction, spasticity, vomiting and constipation are in themselves sufficient. Then, in addition, the eye changes are distinctive. The diagnosis can usually be rendered definite by performing a lumbar puncture, and examining the fluid for Still's diplococcus. It must be remembered, however, that in chronic cases, in the later stages, the organism may be absent. The method of performing this little operation is as follows:-

The patient is brought to the edge of the bed and is laid on his side, with the back bent, so as to open up the interspaces. The space generally selected/
selected is between the second and third, or between the third and fourth lumbar vertebrae, a little to one side of the spinous processes. The parts having been well cleansed with antiseptics and then with sterile water, an ordinary exploring syringe is thrust in. The fluid runs out of its own accord, if the part be kept dependant. Sometimes it comes out in drops, at other times, with a squirt. It is received in a sterilised test tube. The operation seems to cause little pain, and in no case have I seen any harm result.

The outstanding feature of the disease is the head retraction and in no other disease does a tonic retraction of the head occur to such an extent. It has to be distinguished from other forms of meningitis either simple, or tubercular.

In other forms of simple meningitis - whether pneumococcal or suppurative - a primary cause is present; the disease is much more acute, and clonic spasm is more evident than tonic.

On tapping the spine, the respective organism may be found.

I have already pointed out the similarity between this disease and epidemic Cerebro-spinal Meningitis. Both occur most commonly during the winter and/
and spring. In both, the base of the brain and spinal cord may be affected.

In the epidemic form, a rash is common, while rashes have been met with in the simple variety. The organisms present many similarities. The simple form, however, never occurs epidemically.

It is in the differential diagnosis from the tuberculous variety that most difficulty is met with. In well marked cases, the diagnosis is easy; in slight cases with slight head retraction, it is more difficult.

I append the following differences:

<table>
<thead>
<tr>
<th>Simple Basal Meningitis</th>
<th>Tuberculous Meningitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Age</strong></td>
<td><strong>Most common below 1 yr., average age 13½ months.</strong></td>
</tr>
<tr>
<td><strong>2. Duration of illness</strong></td>
<td><strong>May last months, with recovery.</strong></td>
</tr>
<tr>
<td><strong>3. Primary Focus</strong></td>
<td><strong>Absent</strong></td>
</tr>
<tr>
<td><strong>4. Prodromata</strong></td>
<td><strong>None</strong></td>
</tr>
<tr>
<td><strong>5. Retraction of head</strong></td>
<td><strong>Always present; may be marked, with general rigidity.</strong></td>
</tr>
<tr>
<td><strong>6. Clonic spasms and paralysis</strong></td>
<td><strong>Rare</strong></td>
</tr>
<tr>
<td><strong>7.</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Simple Basal Meningitis</td>
</tr>
<tr>
<td>---</td>
<td>-------------------------</td>
</tr>
<tr>
<td>7. Temperature</td>
<td>Usually normal or subnormal</td>
</tr>
<tr>
<td>8. Pulse</td>
<td>Usually fast, may be slow. Is regular.</td>
</tr>
<tr>
<td>9. Respiration</td>
<td>Quiet and regular</td>
</tr>
<tr>
<td>10. Hydrocephalic cry</td>
<td>Rare</td>
</tr>
<tr>
<td>11. Eyes</td>
<td>Blindness frequent. Optic neuritis and squint rare. Meibomian secretion scanty.</td>
</tr>
<tr>
<td>12. Vomiting</td>
<td>May continue to the end</td>
</tr>
<tr>
<td>13. Abdominal retraction</td>
<td>Occasional and usually slight.</td>
</tr>
<tr>
<td>15. On lumbar Puncture</td>
<td>Still's diplococci found.</td>
</tr>
</tbody>
</table>

In Middle Ear Disease head retraction may occur, and Lees and Barlow have pointed this out. Permitting free exit to the pus, clears up the diagnosis.
In Marasmic children head retraction is occasionally seen. Apart from this, there is very little similarity between the two conditions. In Marasmic children, the fontanelle is depressed: in meningitis, it is generally bulging and tense.

One has seen a marked case of tetany, associated with gastro-intestinal catarrh, which simulated this condition.

Catarrh, however, is rare in simple basal meningitis, while the tetany position of the hands is excessively rare.

Cerebellar tumour with head retraction may occur. In such a case, optic neuritis may be present and it does not occur in such young children.

The prognosis is grave, but, fortunately, one can hold out hopes of recovery. In the acute cases, spreading to the vertex, recovery is seldom, but in the more localised, it is quite frequent. Of the thirty cases, nineteen or 63.3% resulted in death. The average duration of life in the fatal cases was fifty days. Of the other cases:

One was discharged "worse."
Two were discharged "in statu quo."
One was discharged "improved."
Seven were discharged "cured."
Of these "cures", however, one was deaf, while two of them returned at a later date with a slight recrudescence of the trouble; so that only four can be definitely stated to have been cured, and even they are doubtful. One of these cases was "thriving splendidly" eight months after its discharge from hospital.

In many of the so-called "cures", hydrocephalus is present. Lees and Barlow point out that the prognosis in a "cured" case should always be guarded, as sudden death has occurred in a patient apparently healthy.

**TREATMENT:**

No specific for the disease has as yet been discovered, and for the most part, the treatment is symptomatic.

The bowels must be kept regular by laxatives, or sedatives, as the case may be, while vomiting is occasionally alleviated by means of Bismuth or Hydrocyanic acid. Nerve sedatives, as the different bromides and chloral, are most useful when the retraction is intense, or when convulsions or clonic spasms are present.

As in other exudations, Iodide of Potash internally/
ternally and Mercury locally are frequently used for this trouble, and in most of these cases, one or both of these drugs has been tried, but I cannot satisfy myself that they have been of much benefit. In one or two cases, Iodoform ointment (10%) was applied to the head, but not in a sufficient number of cases to estimate its value.

Cold or ice to the head is not very feasible in these young children, but it may be tried. Surgical intervention in some cases does good. In cases where the anterior fontanelle is very bulging, and the spasticity is great, the withdrawal of fluid from the spine, or even from the lateral ventricles undoubtedly relieves the condition. The improvement is in most cases temporary, however. In the treatment of the resulting hydrocephalus, the fourth ventricle has been drained, but the results have not been satisfactory.

Within more recent years, the lateral ventricles have been drained into the subdural space by means of a loop of horse hair, one end of which was pushed into the lateral ventricle, and the other left in the subdural space, this constituting a permanent drain. This was first performed by Mr Watson Cheyne. The theory on which the operation is based is, that whenever the pressure of the cerebro-spinal fluid rises above that of the veins, absorption into the latter occurs. It is too soon to pronounce any judgment/
judgment on this procedure, but in one case, the result was very satisfactory.
REFERENCES.

1. Lees and Barlow in Clifford Allbutt's System of Medicine, Vol. VII.

2. St. Bartholomew's Hospital Reports - Gee and Barlow on "Cervical opisthotonos of Infants."


5. Halliburton - Journal of Pathology, Vol. X.