CEREBROSPINAL FEVER:

WITH AN ILLUSTRATIVE SERIES OF FORTY CASES.

being a

Thesis for the Degree of M.D.,
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by

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CEREBROSPINAL FEVER

With an Illustrative Series of Forty Cases.

Only in 1803 was the term Meningitis first introduced by Herpin\(^1\); since when attention has been drawn to the interesting varieties grouped under that heading, and of these, cerebrospinal fever certainly takes a very prominent place of interest, from the point of view both of the clinician and public health authority.

Nomenclature.

The earliest name recorded is, "Fièvre cérébrale maligne non-contagieuse", so called by Vieusseux\(^2\) in 1805.

Spotted fever - characteristically given it by the Americans - and purpuric fever of Irish christening - so called on account of the petechial rash occasionally seen, - are a little unfortunate, as they lead most people to hunt for, and expect to find the rash in every case, but this has over and over again been shown to be somewhat uncommon, and to vary in different epidemics considerably.

The Genickstarre, Genick Krampf, and Nackenstarre of the Germans is somewhat nearer the mark as indicating one of the most common symptoms, but
still it is only a symptom and inexpressive of the disease.

Black death, and Purpura Maligna were given it on account of the marked post mortem lividity that is so often seen.

Typhus cerebralis is quite excusable as many cases, in the early stage, are not very unlike typhus. Illustrating that fact I may add that I had cases of Cerebrospinal Fever admitted as Typhus and vice versa, Typhus cases admitted as Cerebrospinal Fever.

Sinking Typhus is another American name.

Idiopathic Suppurative Meningitis is not sufficiently expressive as the term idiopathic is hardly correct.

On account of its relation to Posterior Basic Meningitis I suggest that Epidemic Posterior Basic Meningitis exactly fits it as it is really nothing more than Posterior Basic in epidemic form.

Better still, and without raising a controversial point, why not Meningococcal Cerebrospinal Meningitis? This at once accurately describes the disease without any chance of confusing it with any other variety of Meningitis and includes both sporadic and epidemic forms. For the sake of brevity simply "Meningococcal Meningitis" would be quite sufficient.
3.

Cerebrospinal Meningitis or Cerebrospinal Fever have, however, become to be almost the universally recognised terms and custom has to be respected. The latter has the merit of associating the disease at once, both in the lay and professional mind, with the infectious diseases.

Geographical and Historical.

Geneva has been generally recognised as possessing the unenviable distinction of being the earliest known starting point of the disease, where it was first noticed and described by Vieusseux(2) in 1805. The curious thing is that since then this town has remained comparatively unaffected.

In the following year it was described by Danielson and Mann(3) as appearing at Medfield, Mass. From that time onwards it has periodically been noted both in Europe and America.

These periods which have marked its exacerbations have been sketched out by Hirsch(4) into the following four groups:-

I. 1805 - 1830
II. 1837 - 1850
III. 1854 - 1875
IV. 1876 - the time of writing

which was 1884.
The first period comprised isolated epidemics in Europe, whereas in the United States it was much more general.

In Europe, it was limited to Geneva and district, Grenoble, Paris, Metz, Albenga in the province of Genoa, Vesoul, Destin in Westphalia and in Britain for the first time when it appeared in Sunderland in 1830.

In the States, starting in Mass., it spread both North to Canada and South to the Southern States.

The second period began in the South of France in 1837, in Bayonne, when it was limited to the garrison and remained so for three years. Meanwhile it spread along the South coast, when all classes were equally affected. It occurred at Bordeaux, and La Rochelle and was confined to the garrison. In 1839 Versailles was attacked, first among soldiers who had been transferred there from affected garrisons in the South. It attained epidemic form, but was limited to the soldiery.

In this period garrisons were most frequently affected and in many instances the spread was aided by the movements of soldiers from the infected
districts. The towns attacked included Metz, Brest, Cherbourg, Orleans and Paris.

Algiers was infected by troops from France.

Southern Italy was extensively attacked and Sicily to a less degree.

It appeared in Denmark in the forties.

The rest of Europe was only affected by small isolated epidemics.

Ireland was attacked for the first time, Dublin, Bray and Belfast being affected in 1845 and 1846.

Liverpool also had some cases, which was not surprising considering its close relation to the Irish Ports.

The United States were again widely affected, but not to such an extent as previously.

In the third period there was a most widespread diffusion all over Europe, near Asia, Africa, United States and South America. Appearing first at Gottenburg it spread for the next ten years over Sweden up to 63° N., but since then that country has not been revisited by it. Norway fortunately remained almost exempt. The rest of Europe, excepting the South and West was generally affected.

It appeared in London for the first time in 1865, reappeared in Dublin in 1866-67, and there was a small epidemic in a village near Lincoln.
The United States were again widely affected, the troops suffering most. It was supposed to have been carried by the latter, and by them two hospitals and an orphanage were infected.

In New York in 1867-68 it was confined to one block of buildings.

The fourth period differed from the others in being less severe, consisting of a number of minor epidemics, Poland, France, Silesia, Sicily, and Greece all having a few small outbreaks. In Britain, it occurred at Birmingham in 1876, and Galston, near Glasgow, in 1884. In 1885-88 there were many cases in an outbreak in Dublin.

Latterly the outbreaks in Belfast and Glasgow have been attracting much attention owing to the large number of cases in both cities. The cases under consideration were observed in the Fever Hospital of Leith, which town experienced its first epidemic in the spring of 1907.

This last period may be considered to continue to the present time, though Osler suggests that a fifth period began about 1901. There has, however, hardly been a long enough break between the epidemics to enable one to limit the fourth period.

Looking at the distribution as a whole it will be seen that few countries have escaped. Cases
have been reported from India, Ceylon and Australia. There are, however, certain limits within which the occurrence of the disease has been most marked. They are as follows:

To the North - Sweden to 63° N.
" " South - The North Coast of Africa.
" " West - Minnesota and Iowa.
" " East - Asia Minor, Syria and Persia.

The United States from the outset has undoubted-ly suffered more than any other country. Of the European countries, France has been most affected, with Germany next, South Italy and Sweden being more fortunate. Britain has escaped until quite recently, except for minor outbreaks of little importance; Scotland has been particularly exempt.

It has been rare in Russia and almost unknown in Belgium, North and Central Italy, Switzerland, Norway, Faroe Islands and Iceland.

Taken generally the more mountainous the country the freer has it been of the scourge. This seems to be the only feature of the disease that presents any constancy in distribution. Take the States as an example; I find no cases reported from the Western, that is the mountainous, half. Similarly Norway practically escaped when Sweden was so exten-sively attacked.
General Considerations.

Five outstanding peculiarities which were recently pointed out by Osler\(^5\) are worth considering as they sum up the disease in a very distinct manner.

1. The disease appears in cycles.

2. It occurs in epidemics in distantly separated areas in which it prevails severely, yet does not spread widely; it is never pandemic.

3. It shows a maximum intensity in densely populated cities and scattered mining towns.

4. The high rate of mortality - ranking second to Plague in the infections.

5. It is the most virulent and at the same time the most mild of the infectious diseases.

This gives at once a clear insight into the general characteristics of the disease.

The occurrence in cycles we have already considered in the Geographical resumé.

The way in which the epidemics are scattered in a most disjointed manner, has been noticed by many. In some instances, areas hundreds of miles apart, with no apparent connection, will be attacked at about the same time; or again towns will be affected, whereas the country communicating between the two will escape. Or there is the very extreme instance in New York, the disease being strictly
limited to one block. The exceptions to this restricted nature of cerebrospinal fever are those instances when it was spread by the movements of affected troops, especially in France.

A pandemic extension such as occurred with influenza is fortunately unknown. It is in small localised regions scattered far apart that it generally occurs.

The height of the rate of mortality is looked at in comparison to the rates of other infectious diseases, of which Plague certainly seems to have the unenviable distinction of ranking first. Payne\(^7\) says, "In India the general case mortality has been 70 to 85 per cent among natives". In Hong Kong, he goes on to say, it has reached as high as 96 per cent.

For cerebrospinal, I think 75 per cent or possibly lower is about the usual. Netter\(^8\) gives 30-75\%, Hirsch 20-75\%, Flexner\(^10\) 73\%, and Councilman, Mallory and Wright\(^9\) 68.5\% as the mortality figures. These are undoubtedly high, but compared with the mortality of other forms of meningitis they are not so depressing; it is in fact the only form of acute meningitis where we can hope for a recovery - as Osler expresses it, "It is the silver lining of the meningitic cloud". 
In my own series there were eleven recoveries out of forty cases, but deducting case 21, which died two weeks after discharge from Hospital, the case mortality was exactly 75%.

One very noticeable feature in this series was the number of chronic cases in the first half. Taking an acute case as that which terminated in a week, there were only two in the first half of the series, but eight in the second. The reason of this may be explained by the fact that the general practitioners were much quicker at diagnosing the disease as the epidemic advanced so that the more acute ones were able to be sent in at once. In the early part of the epidemic I know that in one week alone there were ten cases which died in the town, before we were able to get them into Hospital.

The variation in virulence is one of the most peculiar features of the disease. All grades are to be seen, from the fulminating case terminating in death in a few hours, to the long protracted chronic cases whether ending in recovery or death, and further those most interesting transient cases of mildest symptoms which may pass off in less than 24 hours. A case of this nature came under my observation recently, where the onset was sudden, with stiffness of the neck and severe pain on the
top of the head and in the neck. The head could not be moved forward without great pain. The patient felt sick and vomited once, but after a rather bad night was almost all right the following day, and by the next morning had no remaining discomfort.

In the present series case 29 is a fairly good example of the fulminating type. The actual duration was barely 22 hours from the time of feeling the headache, and only 12 hours from the actual onset of the acute symptoms. Case 21, on the other hand, illustrates the transient type. Cases 7 and 11 are perhaps the best examples of the chronic types, the one ending fatally and the other recovering, although at one stage they were both equally severely ill.

This introduces the question of classification, and the three types above mentioned illustrate my idea of a useful and simple one:

1. Transient
2. Malignant or Fulminating
3. Chronic.

A more extended classification is doubtless possible, but does not appear to me to be of much practical use. Tourdes (11), for instance, gives no less than eleven varieties classed under two
major headings, as follows:

I. Cerebrospinal Forms:
   1. Foudroyant
   2. Convulsive comatose
   3. Inflammatory
   4. Typhoidal
   5. Painful
   6. Hectic
   7. Paralytic

II. Cerebral Forms:
   1. Cephalgic
   2. Delirious cephalgic
   3. Delirious
   4. Comatose

A purely spinal cases did not come under his notice.

Classifying the present series under my three headings, the percentage of each variety works out as follows:

- Transient: 2 Cases = 5%
- Malignant: 19 " = 47.5%
- Chronic: 19 " = 47.5%

The abortive or transient I have described in its mildest type, but it may be seen in more severe grades. The malignant cases include all those where the onset is severe and gets a thorough hold of the patient at once, and is almost invariably fatal, within a few hours to twenty-one days. The chronic include all the cured, excepting transient cases, as the convalescence is always protracted.
A further peculiarity to be noted is the great variability in the duration of epidemics, in some places lasting a matter of weeks, but in others extending over a period of years.

The cases do not gradually increase in numbers and then slowly subside, rather there will be a run of cases for a short time, then an abatements, followed by another run, and so on with alternate periods of remissions and exacerbations.

The definition of the disease as given by Grimshaw and Still\(^ \text{6} \) is that it is, "An acute febrile disease characterised by sudden invasion with extreme nervous shock, vomiting, excessive pain referred to the back of the neck and spine, spadmodic contraction of the muscles, hyperaesthesia and frequently cutaneous eruptions." This gives a concise idea of the symptoms, though a general description is not easy owing to the great variations seen.

One may, however, take it that usually the onset is sudden, with headache and vomiting, a sore throat is occasionally present and may even simulate the onset of scarlet fever. A stiffness of the neck almost invariably becomes apparent after a short interval. Backache soon follows and with the increasing headache, onset of head retraction and loss of consciousness, the disease has got its
hold of the unfortunate victim.

The picture at this stage is indeed distressing; restlessness is frequently extreme, pain makes the patient shriek out constantly, and unconsciousness is a luxury.

The patient is found lying on the side almost invariably, due probably to the greater ease obtained in that position on account of the retraction of the head. The legs are drawn up, due undoubtedly in some instances, but not always, to abdominal pain.

The face has an expression of pain, is usually flushed in the early stage, and is somewhat cyanosed.

There may early be present, but only in a small percentage of cases, the petechial rash, or a crop of herpes - usually labial, but I have also seen it on the shoulders and chest.

There undoubtedly is a smell characteristic of the disease as is also found in diphtheria, typhoid and other of the infections. In some cases it is hardly perceptible, whereas in others it is most marked and invades the whole atmosphere of a well ventilated ward. A description of a smell is always somewhat difficult. I can only describe this as being exactly the same as that which pervades a blanket or a thick bath towel that has been washed with a lavish supply of black or soft soap which
has not been properly wrinced out. I therefore have
alluded to it as the "soapy smell".

The temperature may range anywhere between 97° and
104°, but whether high or low the patient feels cold
and is with difficulty kept warm.

The pulse and respiration vary independantly, both of the
temperature and of one another. The pulse is usually rapid and full at the outset, but becomes feeble and irregular later. The respirations are frequently increased.

Such is the general aspect during the first few days. Later the headache becomes more severe and the vomiting may continue; the retraction of the head and rigidity of the neck increase.

The patient is unwilling to take food and in some cases there is obvious difficulty in the act of swallowing, due I think to the marked degree of head retraction and not to any paralysis. Constipation of a most aggravated type appears. There may be a slight cough due to some bronchitis, and this may be followed by a bronchopneumonia which proves fatal. There is often general hyperaesthesia slight or marked. The reflexes are quite irregular and even Kering's sign, which one would expect to be constant, is not so.
Much attention has been drawn to the presence or absence of the abdominal and epigastric reflexes. Fowler suggests that their early absence signifies primary infection of the cord, and that their return indicates a favourable prognosis. Certainly they are frequently both absent in the early stage, but the questions relative to their absence and return we will discuss later.

The urine may contain a trace or more of albumin and occasionally some reducing substance.

A leucocytosis is invariably present.

This rough general description must suffice, as being all that is possible to give of a disease which presents such variety.

If death does not supervene during the first or second week, a tedious course may be looked for. At the end of the first three weeks the temperature has usually subsided and the patient has become extremely emaciated, he then either drags on to his end, happily hastened by the advent of some complication, or else attains a long convalescence with ultimate recovery.
Etiology.

The active causal agent is undoubtedly the Diplococcus Intracellularis Meningitidis, which organism will be considered more fully under Bacteriology. In the meantime there are other factors to be considered.

Season.

Statistics show that winter and spring are its most favourable months. This is well illustrated in the following table, taken from Netter, being the deaths in Sweden between 1855-1884.

<table>
<thead>
<tr>
<th>Month</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jan.</td>
<td>1</td>
</tr>
<tr>
<td>Feb.</td>
<td>10</td>
</tr>
<tr>
<td>Mar.</td>
<td>23</td>
</tr>
<tr>
<td>Apr.</td>
<td>29</td>
</tr>
<tr>
<td>May</td>
<td>21</td>
</tr>
<tr>
<td>June</td>
<td>14</td>
</tr>
<tr>
<td>July</td>
<td>7</td>
</tr>
<tr>
<td>Aug.</td>
<td>0</td>
</tr>
<tr>
<td>Sept.</td>
<td>3</td>
</tr>
<tr>
<td>Oct.</td>
<td>0</td>
</tr>
<tr>
<td>Nov.</td>
<td>0</td>
</tr>
<tr>
<td>Dec.</td>
<td>0</td>
</tr>
</tbody>
</table>

Why should this be? Is it because of the prevalence of cold and wet in these seasons? Such is a very natural conclusion, but does not seem to be proved. For instance, epidemics have begun in summer as in Bordeaux in 1839, Toulouse in 1842, and
Dublin in 1850. Again, they have increased with the onset of warmer weather, as at Sicily in 1843, New York 1872 and Southern Italy in 1874. Further it is unknown in the arctic regions. On the other hand in Upper Franconia in 1865, the places most exposed to East and North East winds were most affected, and when intense cold came at the end of March, the epidemic was greatly increased. At New Orleans also in 1847 one regiment, which was in good quarters and well clad, was exempt, whereas another brought up from the warmer climate of the South, unsuitably clad and in cold damp barracks, was attacked.

Wetness of the Soil.

The last statement would appear to point to the fact of wetness of the soil predisposing to attack. This, however, does not hold good in all cases, as for instance when in 1861, of the army of the Potomac, only one regiment was attacked, and this occupied a site that was particularly dry and wholesome.

Age.

Most writers lay stress on the fact that children are most affected. This is strongly brought out by the following figures given by Netter as representing the deaths in Mass. between 1887 and 1895.
The ages of the present series were as follows:

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5 years</td>
<td>9</td>
</tr>
<tr>
<td>5-10 years</td>
<td>16</td>
</tr>
<tr>
<td>10-15</td>
<td>4</td>
</tr>
<tr>
<td>15-25</td>
<td>5</td>
</tr>
<tr>
<td>25-35</td>
<td>2</td>
</tr>
<tr>
<td>35-45</td>
<td>3</td>
</tr>
<tr>
<td>Over 45</td>
<td>1</td>
</tr>
</tbody>
</table>

This represents 72.5% as occurring under 15 years, 62.5% under 10 years and 50% between 5 and 15 years. There was only one case under 12 months. The percentage of the number of infants under 12 months appears to vary widely in different epidemics. In Councilman, Mallory and Wright's 111 cases, only one was included. In Billings(13) figures for 365 cases
no less than 60 under the one year age were noted, and higher still are the figures of Netter given above in which over 30% occur.

Sex.

Appears to have but slight influence. In my series 23 were males and 17 females. Of Billings 365 cases 56% were males and in Glasgow in 1906 Wright(14) states that 121 were males and 84 females. If anything, then the males are a little more frequently affected.

Race.

All are equally subject to attack. In America the disease has occurred equally among the alien, negro and American. This is also noticed in Algeria with the Arab and European.

Occupation.

From the outset the soldiery have been very prone to attack. Why this should be is difficult to explain. It may be that frequently over-fatigued their resistances are consequently lowered, so rendering them susceptible to infection; probably, however, the overcrowding in barracks which used to be excessive, had more to do with it. Under the present day conditions these big barrack epidemics are not seen, and the disease is distributed more evenly.
Injury to the Head.

In only 6 cases (see Table I.) could I find a history of such injury. How much importance to attach to it varies greatly with the parent's imagination and the length of time between the alleged injury and the onset. In some instances it is, however, too closely associated to be disregarded.

Fatigue.

Physical fatigue, by lowering the general resistance, is an important factor in all the infections, and none the less in this one. Mental fatigue plays a similar role and seems to be of particular significance in cerebrospinal fever.

General Development.

The general opinion seems to be that robust healthy persons are the most susceptible, and this is fairly well illustrated in the present series, taking into consideration the class affected. In Billings cases 91% were in good health at the onset.

Overcrowding.

This has been frequently noted as favouring the spread. Hirsch draws attention to the fact and states that in Metz in 1839 an abatement in the number of cases immediately followed a diminution of overcrowding. He suggests that this may have
something to do with the prevalence of the disease in winter, as at that time overcrowding, and deficient ventilation, which is its usual companion, are naturally exaggerated.

Without actual figures of cubic feet capacity it is difficult to give an idea of whether cases come from an overcrowded house or not. In estimating the present series, I have allowed that one room accommodating two persons is a fair basis to take. This of course is very rough but works out pretty well for practical purposes. On this basis 25 of my cases or 62.5\% came from an overcrowded house.

Class of Case.

This is illustrated well by Billings figures; 299 of 365 cases = 82\%, lived in tenements, that is to say the labouring class. In Leith the majority were poorer labourers, though the better class artisan did not escape. This can be shown by reference to the number of rooms occupied by the different cases (See Table I.).

<table>
<thead>
<tr>
<th>Cases occupied</th>
<th>1 roomed House</th>
<th>2 roomed House</th>
<th>3 roomed House</th>
<th>4 roomed House</th>
<th>5 roomed House</th>
<th>6 roomed House</th>
<th>7 roomed House</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>24</td>
<td>2</td>
<td></td>
<td></td>
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<td>5</td>
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<td>1</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

That is to say that over 60\% occupied 2 roomed houses, which fairly represents the class of case affected.
This seems from the beginning to have been the rule, though when an epidemic extends widely, no class is exempt.

Abuse of Alcohol.

This has been suggested as a predisposing cause. In a few odd cases it may possibly have had some influence, but I do not think it has much to do with it, the usual age period is too young for alcohol to have any influence. Only one of my cases was a heavy drinker.

Condition of the Throat.

Westenhoeffer(14) says that predisposition seems to depend largely on the condition of the tonsils - the larger they are the greater the risk of infection, and further that in every cases of acute infection, inflammatory conditions of the fauces, and especially the tonsils, were present.

In my cases when first examined, that is after the onset, 22 or 55% showed inflammatory changes, and a sore throat was often complained of. I think it of more importance, however, to know the condition of the throat previous to the onset.

Epidemiology.

In the first place is it infectious? In the epidemic form it is now generally recognised to be
The actual degree of infectivity apparently varies, at times being very great, at others, as at the end of an outbreak, being quite insignificant.

That it is contagious is apparently doubted by many; the general opinion, however, is in favour of it being so.

From the first, Vieusseux named it "non-contagieuse". Holt(15) definitely states that it is not contagious. Osborne(16) says that it is only through carelessness that contagion takes place.

In support of the non-contagious theory is the fact that there have been no school epidemics, and that cases can be nursed in the general wards of an Infirmary.

The absence of school epidemics may be explained in two ways. First, that it may not be contagious in the initial stage. Secondly, that the onset is usually so definite that the child either never goes to school ill, or is at once sent home and has no time to infect the other children.

Nurses and medical attendants have been attacked while attending to cases in Hospitals. Leichtenstern(17) gives instances of a sister and three nurses contracting the disease while in the cerebro-spinal wards.
A most striking and conclusive example of contagion is furnished by Dr Hare. His assistant attended a friend and sat up with him for 40 hours, till he died. In less than 24 hours after, he himself was attacked and succumbed within 24 hours. Dr Hare, who attended him, suffered two days later from a severe headache and stiffness of the neck, with slight temperature, but luckily nothing more serious resulted.

Instances of two or more cases in one house may be brought forward. They might be infected, of course, from a common source, and not from one another, but the theory of contagion is possible. Sewall's reports an instance of six cases, and Osler one of five in one family.

In the present series there were four instances of two cases in one house.

I have no doubt as to the contagiousness of the disease, but recognize that it is possessed in a limited degree.

As well as being directly contagious the disease is also infectious through a third party or intermediary. There are numerous instances of this but none more conclusive than Jehle's in Silesia. He noted, what had already been commented on, that cases which occurred in rapid succession in point of
time were often separated by distances that excluded direct infection. He then found that the children who were infected belonged to fathers all working in one particular mine; that the children of fathers in neighbouring mines escaped. He concluded that the mine was the source of infection and the fathers the means of dissemination. His idea was that in the infected mine the meningococci flourished in most suitable conditions for their growth, namely, warmth, darkness and moisture. They would easily be inhaled and get into the nasal passages and throats of the miners, who, going home, would spit on the floor and so scatter the organisms, and the children while grubbing about would become infected. In this way two or three cases might occur in one house yet would not be due to contagion but to infection from a common source.

Following up this idea Fraser and Comrie(20) tried to discover a common source in the Leith cases. This was not an easy undertaking, but in one instance they found a ship in which the fathers of five families that had contracted the disease were working. On exposing petri plates in the ship meningococci were obtained. The fathers had presumably infected their families, though they themselves had escaped. Four of these cases come into my series.
They also found the meningococcus in ten of the noses of sixty-nine contacts. These contacts consisted of fifteen fathers and fifty-four women and children, and it is significant to note that of these ten positive findings, five were in the fathers. I should add that the existence of the meningococcus was not accepted until the most recent and exhaustive tests had been applied.

Jochman(21) demonstrated the presence of meningococci in seventeen out of twenty-four healthy people's nasopharyngeal tract.

Briefly then the disease in epidemic form is infectious, contagious, and transmitted by a third party who does not necessarily contract the disease.

Facts illustrating these points occurred in my series in the following cases.

Cases II., VIII., XI., XII. were four of the five children that belonged to the parents working on the infected ship above mentioned. Case II. is worthy of special note in that he slept with the father; the rest of the children, who all escaped, slept with the mother. Cases II., VIII. and XI. had a double connection in also coming from the same school.

Case III. became ill four days after her mother took cerebrospinal fever.
Cases IV. and IX. worked together in the same Roperie. Case IX. got ill nine days after case IV.

Case X.'s mother helped to nurse a neighbour's child who died of cerebrospinal fever five days before the onset of Case X.'s illness.

Case XII. lived on the same flat as Case X. and visited him frequently before his removal to Hospital.

Case XV. came from the same lodging house as Case XIV., getting ill five days after the removal of Case XIV.

Case XVI. was at school with a child that had recently died of cerebrospinal fever.

Case XX. came from a tenement in which there had been a death from cerebrospinal fever ten days previously.

Cases XIX. and XXV., XXVI. and XXIX., XXVII. and XXXII. came from the same houses, separated by eleven, six, and three days respectively.

Case XXXVII.'s father had been burying a brother at Antwerp who died of cerebrospinal fever. He had returned nine days previous to the onset. There was also another case on the same stair.
What is the method of Infection?

This important question has not been finally satisfactorily answered.

At present there are two alternative theories:

1. The alimentary tract offers the portal by means of food infection.

2. The organism is air borne and inhalation is responsible.

The first is strongly upheld by Fowler\(^{(12)}\). He says there is not clinical or pathological evidence to prove that there is any course of infection from the nasal passages, but "there seem to be certain facts tending to show that in cerebrospinal meningitis the cord lesion is primary and that the diplococcus invades the nervous system through the spinal meninges".

He then goes on to say, "In most cases the P.M. findings show that the cord lesion is of older standing than that of the brain. The constant, early and complete abolition of the abdominal reflex seems to point to the implication of the lower dorsal cord." He further supports the theory by comparing pneumococcal meningitis, which is primarily cerebral with meningococcal. In the former, in one case where lumbar puncture fluid was clear the abdominal reflex was present, but in two others when the fluid
was turbid and the cord therefore implicated, the abdominal reflex was absent.

Now I hold that there is both pathological and clinical evidence to prove the existence of a course of infection from the nasopharynx.

In the first place in Flexner's (10) experimental work he found "that if the dura mater beginning at the olfactory bulbs, surrounding the olfactory nerves and extending through the cribriform plate into the nose, be carefully removed with the adjacent portion of the ethmoid bone and olfactory mucous membrane, fixed in Linker's fluid, sectioned longitudinally, stained in haematoxylin or methylene blue and eosin, and examined microscopically the passage of leucocytes from the brain cavity into this membrane and about the olfactory nerves towards the ethmoid can be traced". If then there is a passage in this direction there must be one in the reverse way from olfactory mucous membrane to dura mater. Further, he states: "The abundant lymphatics of the mucous membrane are in communication with the lymphatic spaces which enclose the branches of the olfactory nerves, and these again communicate with the subdural and subarachnoid spaces of the cranium, .... ."

In further support of the presence of a direct
communication I should like to quote the following extract from Osborne's paper. "I will only refer to some recent investigations under the auspices of the German Government. Twenty nine autopsies on patients dead of this disease were carefully made, twenty-two were children under 10 years of age and seven were adults. This report shows that the infection started in the pharyngeal tonsil, entered the cranium through the sphenoid bone travelling along the vessels which run from the pharynx into the sella turcica through the sphenoid foramina. The part of the brain first affected was the hypothysis or pituitary body. There was no evidence in these 29 autopsies of the infection passing through the cribriform plate of the ethmoid and the infection seemed to travel by lymph channels rather than by blood channels."

Direct infection from the nasopharynx or tonsils is therefore possible, it is also by far the most probable. The only other way might be by lymphatics into the general circulation and thence to the brain, but this is a very round about route, and also normal serum has a natural bactericidal action for meningococci. Certainly they have been found in the blood but only when the patient has been fully infected, and probably with resistance much
lowered. For the food infection theory this delicate organism is expected to run the gauntlet of the gastric juices and then to migrate by some means to the spinal column. One cannot conceive this as being possible unless a pure culture were swallowed instead of the few meningococci that might conceivably have become attached to the food. In the inhalation route on the other hand, the few organisms inhaled have a most suitable ground in the warm, dark and moist nasopharynx to settle and increase in numbers, before their absorption or migration.

For clinical support we have the associated sore throat and presence of catarrhal changes so frequently seen early in the disease.

A very definite example of meningeal infection by inhalation is mentioned by Councilman (22). A pathologist while working with meningococci suddenly acquired a severe rhinitis with congestion of the mucous membrane and profuse muco-purulent discharge: at the same time he had severe headache and retraction of the neck. Examination of the nasal discharge demonstrated the presence of meningococci.

As to the statement of the cord lesion being of older standing, this does not seem to be the case. Holt in the micro-pathological description of cerebro-
spinal having described the condition in the brain says: "Changes in the cord similar to those just described may be found, but these are less frequent and as a rule much less severe than those in the brain." This would lead one to suppose that the brain lesion therefore was of older standing. Moreover I have never found a report of a purely spinal case, whereas purely cerebral ones undoubtedly are not uncommon. Case 29 is an instance of it.

The loss of the abdominal reflex upon which so much stress is laid by Fowler is not constant. Its absence, though, may be explained by the profundity of the toxaemia present. Of my 40 cases the reflex was absent in 19, present in 13, and not noted in 8. Of the 19, the toxaemia was marked in 18, but of the 13 in which the reflex was present only one was profoundly toxic. In two cases, Nos. XXIX. and XXXII., on admission when little was noticeably amiss, the reflexes were present, but as the toxaemia supervened they disappeared.

The early finding of the meningococcus in the cerebrospinal fluid seems to me to point more to the rapidity with which the spinal meninges are implicated by direct extension from the cerebral infection, than to spinal origin.

Then supposing the food of a family was infected, one would expect to see two or three of the
children attacked at the same time, but whenever two cases do come from the same house, there is an interval between them. Again, cases as young as 5 days old (Billings) and 3 days (Allchin\textsuperscript{23}) have been reported, these surely could not be food infection.

The frequent inflammatory condition of the intestines is the strongest point in favour of food infection and not easy of explanation. I think it, as well as the petechial haemorrhages seen in the mesentery, is due to the general toxaemia. That this toxaemia is a real result and product of meningococcus has been demonstrated by Ohlmacher\textsuperscript{24} experimentally in horses.

In aiding the spread of infection, fomites play an important part. The organism clings to clothes, etc. for long periods, and when these are shaken it is mingled with the dust and inhaled. Charteris\textsuperscript{25} draws attention to the fact that in Glasgow where second cases had occurred, the carelessness in dealing with fomites was strikingly manifest. Netter gives an account of a striking instance, reported by Kohlmann, of infection being carried by fomites. Case A. died of cerebrospinal fever and clothes were borrowed from houses B. and C. to go to the funeral
in. After a delay of some days they were returned. Both houses B. and C. were attacked and the first to take it in each house was the owner of the borrowed clothes.

My opinion, then, is most strongly in favour of the infection being air borne, and that by inhalation the organism is carried to the seat of infection - the posterior nasopharynx. The communication from this site is by lymphatics direct to the cerebral meninges. In aiding the spread, fomites, overcrowding, and deficient ventilation are the most important factors.
How is infection introduced into a district?

It must be admitted that there are sporadic cases of cerebrospinal fever practically always present in any district, in the form of Posterior Basic Meningitis. Some writers there are who still consider the two separate diseases, but the general opinion is now in favour of them being varieties of the same disease, on a parallel with sporadic and epidemic forms of pneumonia.

When an epidemic occurs, therefore, it is not a question of the introduction of a new disease, but the increase in virulence of the organism, and with this, the increase of infectiousness and contagiousness is coincident. What we want to know is, what are the influences to which this change is due?

I think the organism is normally present with us more often than is supposed, in the same way that the pneumococcus and tubercle bacillus are. I think McDonald's (26) suggestion is very probably correct. His idea is that the organism commonly exists in an attenuated form, possibly in the nasal secretions, which may increase in virulence giving rise to Posterior Basic Meningitis, or still more increasing in virulence giving rise to Cerebrospinal Fever.
Incubation Period.

Netter gives between 3 to 11 days. In the few instances in my series where any idea could be got, 5 to 10 days seemed to suit the conditions.

Immunity.

A few cases are on record of second attacks. Councilman, Mallory and Wright have found five such cases, but they appear to be rare.

Relation of Cerebrospinal Fever to Pneumonia.

I have already alluded to the similarity of these two diseases. It is to be noted in the following:

The degree of infection and contagion.

The existence of sporadic and epidemic cases in each.

They not infrequently complicate one another, and meningitis is also complicated by other inflammations caused by the pneumococcus.

Their onset is similar: sudden, attacking healthy individuals, and both set up constitutional disturbances, the severity of which is out of proportion to the local lesions.

Both show a high leucocytosis with the onset, which increases with the severity of the attack, and diminishes with improvement.

Both frequently have an associated herpes.

The relations of the two organisms will be considered in the bacteriological section.
Bacteriology.

The organism responsible for the disease I have already mentioned as being the Diplococcus Introcellularis: it is also called the Micrococcus Meningitidis Cerebrospinalis - more conveniently termed Meningococcus.

This diplococcus was first described by Weichselbaum in 1887 when he found it in 6 out of 8 cases; in the other two it is of interest to note that the organism was the pneumococcus.

In shape and size it is very like the gonococcus. It occurs in pairs, of two hemispheres, with the flat surfaces opposing, but may also be found singly or in tetrads, and is said to sometimes form chains, though these I have never seen, and Gordon\(^27\) definitely states that it never does so.

It is actively mobile: I have never seen any flagellae nor heard of their having been noted.

Occasionally I have observed a distinct capsule. It stains readily with ordinary stains but does not retain gram. In this connection it is interesting to note a report by McDonald\(^26\) on a case in the Children's Hospital, Edinburgh. He found gram negative was the rule, but on careful search in the original films from lumbar puncture noted some gram positive. Cultures at an early stage showed gram
positive diplococci resembling pneumococci; subcultures, however, of these colonies showed typical flattened diplococci. In another of his cases the organism was the typical gram negative diplococcus from lumbar puncture and tissues, but in culture became gram positive. A slide showed diplococci in which one half was gram positive and the other negative, and further, chains were seen of gram positive diplococci with an occasional gram negative in them. This variability in staining with gram was also noted by Councilman, Mallory and Wright, and may be explained by the fact that the diplococcus may be in a state of commencing dissolution.

The meningococcus occurs in the cerebrospinal fluid, and purulent material occurring on the surface of the brain and cord. They have also been found in other sites. Simon found them in the blood. Elser found them in the blood of 10 cases, 7 of which died. He therefore regards it as a bad prognostic. He also found them in the nose in 6 out of 22 cases, in the middle ear in one case P.M., but failed to discover them in the urine, the herpetic vesicles, or fluid from a swollen knee joint. Robinson obtained them in the pus from conjunctivitis in one case, but did not find them in the lungs of cases complicated by pneumonia.
Warfield and Walker\(^{31}\) obtained them from a case of endocarditis: in the blood during life and from the vegetations at autopsy.

Formaldehyde 1 in 22,500 rapidly destroys the meningococcus (Weichselbaum). A temperature of 55\(^{\circ}\) C. for 30 minutes kills it (Flexner). Charteris says it cannot sustain drying at 37\(^{\circ}\) C. for 24 hours, but at ordinary temperature of the room Wilson\(^{32}\) says it can exist for 127 days.

The serum from patients has agglutinating properties in from 1 in 10 to 1 in 400 dilutions.

The organism is found to be difficult to grow, and to keep it active has to be subcultured every two days.

The media upon which it grows well are: blood agar, ascitic agar, or Loeffler’s serum gelatin, mixed with three parts of sheep’s serum and one part of bouillon, to which glucose has been added (Netter) or better still, Gordon’s "nasgar", i.e. nutrose ascitic agar.

For the preparation of nasgar I will quote direct from Gordon.

\[\begin{array}{ll}
A. & \text{Ascitic Fluid} \\
& 15 \, \text{c.c.m.} \\
\text{Distilled Water} & 35 \, \text{c.c.m.} \\
\text{Nutrose} & 1 \, \text{gram.} \\
\end{array}\]

Put in a flask and bring to the boil, constantly shaking till the fluid boils.
Filter.
B. Ordinary peptone agar.

Mix one part of A. with two parts of B. Steam for 30 minutes, filter and put in Tubes."

It grows in milk with no clotting or reaction, and feebly on potato.

It fails to grow in anaerobic conditions.

The optimum temperature is 37° C.; it will not grow above 42° C. or below 25° C.

For a full description of the growth characteristics I shall draw extensively on Robinson's report.

"Plain agar-agar - after 24 hours at 37° C. there is a rather delicate grey sharply defined, raised pearly translucent, thin edged growth which often has a slight milky appearance in the thicker parts. The growth often takes place only along part of the streak of innoculation and small gray, round, discrete colonies, 2-5 mm. in diameter may develop along the streak. No difference is to be made out between the appearance of the 24 hours and 48 hours old culture, as a rule, but after 4-6 days, drying can be noticed. The edges of the growth then usually take on a rather characteristic wavy laminated appearance. A moderately delicate growth takes place in a stab culture. The addition of glucose to the agar increases the luxuriance of the growth.
No gas is produced in glucose agar stab cultures. The growth extends over the surface of the glucose agar slightly. On human blood agar the growth is more certain, and more luxuriant than on plain agar. Here separate colonies are apt to form along the streak of inoculation. On Loeffler's blood serum there is seen a rather abundant gray moist raised growth. Bouillon is slightly clouded and a small sediment which increases in size with several days incubation is seen. No pellicle formation is noted. Litmus milk is not altered, although cover slip preparations of the cultures show growth to have taken place. No growth takes place as a rule in gelatin at room temperature."

The manner in which the meningococcus acts has been shown by Flexner to be by a toxin liberated from the diplococci, probably through its disintegration and not to a secreted extracellular poison. This view he supports by proving that cultures killed by heat, and autolysed fluids of cultures are fatal to guinea-pigs; and also exudates obtained from the peritoneal cavity of guinea-pigs in which many diplococci have been dissolved also proved to be toxic for mice.
Pathogenesis.

For this branch I have relied entirely on Flexner's work.

He found that there was a great degree of variance in the virulence of different strains of the meningococcus.

Fatal results rapidly followed the inoculation of guinea pigs intraperitoneally with small doses - .5 to 2.0 c.c. - of the meningococcus in suspension. In a few cases the guinea-pigs temporarily recovered to die after some weeks, death being preceded by marked emaciation. With the exception of this symptom, there was no constant pathological condition in these protracted cases. The peritoneum was clear and no diplococci were found there or elsewhere in the body.

In the more rapid cases fluid exudates were present in the peritoneal cavity, and more or less pus and fibrin covered the anterior surface of the liver, and the surfaces of the rolled up omentum.

In monkeys he conducted some very successful experiments.

Intramuscular injections of virulent cultures produced no result or only trifling effects.

Inoculations by spinal puncture with variable doses killed the animals in from 14 to 43 hours, after producing symptoms similar to cerebrospinal fever as seen in man.
In the first monkey which died about 18 hours after the spinal puncture, the surface of the brain was greatly congested and punctiform haemorrhages existed in the superficial portion of the cortex. The lumbar and thoracic portions of the cord were covered posteriorly with an opaque white exudate. The diplococcus was recovered in cultures from the spinal cord, brain, and blood of the heart. Cover-glass preparations of the exudate from the brain and cord showed the organism within the polymorphs.

Another monkey died in 12 hours, and showed extensive meningeal infection and acute encephalitis.

A third survived 48 hours, and illustrated a point noted by Flexner in all that survived the second day, that the quantity of exudate may not be very considerable and the diplococci, if still present, may fail to grow on a suitable culture medium. In this particular case, the convex surface of the brain showed great injection of the pial vessels, and the pia-arachnoid contained a gelatinous fluid exudate. Purulent exudate was visible surrounding the infundibulum only. The fluid in the ventricles was slightly increased, no visible exudate covered the cord, and the cultures remained sterile.

In the animal dying in 14 to 15 hours, the chief interest lay in the fact that the cerebral lesions
were much more marked and extensive than the spinal, the thoracic region of the cord being relatively free.

An important point to be noted is that Flexner states it is usual to find in monkeys succumbing to the experimental infection, evidences of inflammation of the nasal mucosa,

Finally, not all the monkeys inoculated died, and some apparently recovered to die suddenly at a later date of some cause unexplained P.M.

The point now for the Bacteriologist to determine is what relation the meningococcus has to the pneumococcus.

I have touched on the similarity between pneumonia and cerebrospinal meningitis, and this is supported by the many points of resemblance between the two organisms, which are:-

Both occur as diplococci.

Both are encapsuled.

Both are said to occur occasionally in chains.

Both are difficult to cultivate.

Both cause a suppurative cerebrospinal meningitis, and in some instances have been both found together in cases of this disease.
In eight cases of cerebrospinal fever complicated by pneumonia, meningococci were found in the lungs.

The points of difference are:

- The staining retention of gram.
- The difference of shape.
- The motility of the meningococcus.
- The frequency of the intracellular position of the meningococcus.

That they are very similar is beyond question. Netter suggests that the meningococcus is a degenerated form of the pneumococcus.

Maher suggests that it is a phase in the life cycle of an organism which at times is larger and rod shaped, and at others small and of the shape of the pneumococcus.

On this point I cannot form an opinion, but certainly if they are not varieties of one organism, or the same organism in different stages of a life cycle, they are very near relatives, if one may use the expression, in relation to bacteria.
Symptoms.

Prodromal symptoms do occasionally occur, but not commonly. Case 37 is a good illustration. Seven days before the actual onset she felt cold, shivered, and complained of headache, pain in the back, and she vomited. For the next week she was quite herself again, playing about with the other children, but on the seventh day the headache returned and in a few hours she was unconscious. Other instances of prodromal symptoms two or three days before onset may be seen in cases 5, 20, 24 and 31.

The onset is a noteworthy feature, usually being strikingly sudden. Foster(34) reports the case of a man who was in perfect health, and had had no preliminary headaches, and who, while walking along the street, was seen to stumble and fall, a convolution following and this was the onset of cerebrospinal fever. James(36) notes a case where some children were out in the country for a holiday; one of them was found unconscious by his playmates, and he died that night of cerebrospinal fever.

These are marked instances but not rare: fully a quarter of my cases illustrate this great suddenness of onset, noticeably cases 14, 26, 30 and 34, and in 72.5% the patients were thoroughly "down" with the disease under 24 hours from the outset of
the first symptom. In a few cases two or three
days elapse before the symptoms are so pronounced;
in one exceptional case, No. 24, the initial symptoms
extended over a period of from 10 to 15 days.

It is the abruptness of the initiating symptom
which is so striking. The two most commonly seen
first are headache and vomiting. The former is not
of the usual type, but very severe and sometimes
excruciating, and most difficult to relieve. It
accounts, I think, for the frequent screaming in
children that is so trying. It was present in 32
of my cases, unascertained in 5, and only definitely
absent in 3.

Vomiting is also very common. It was present
in 27, unascertained in 4, and absent in 9. It is
spontaneous without previous nauseaion.

Convulsions occasionally initiate the disease
or accompany the early symptoms.

Shivering, though not a definite rigor, was
present in nearly half of my cases.

Following the headache, pain in the neck occurs,
and this may later continue down into the back.

Rigidity of the neck, or as Wall(37) more
descriptively terms it, "resistance to forward
flexion of the head", early appears, and may be fre-
quently seen in its exaggerated form as head retrac-
tion.
Two other symptoms rapidly appear, namely marked restlessness and unconsciousness.

This neck rigidity I lay great stress on, owing to it being almost constant. It was only absent in 2 of my cases, i.e. Nos. 4 and 26. It is due, I think, to a tonic spasm of the muscles, and the head retraction is the natural result of the increase of contraction. The rigidity always appears first, and often a day before the retraction, which was only present in 55% as against 95% for the rigidity. It is the latter, therefore, that I look upon as being pathognomonic, and not the retraction, as Ormerod\(^{(38)}\) suggests. The Germans have recorded their recognition of the frequency of it by their name "Genik-starre" for the disease.

The retraction may be simply a slight deflection from the straight, or may increase to the extreme degree, when the occiput touches the interscapular region. Only 3 of my cases showed this so markedly, i.e. Nos. 9, 35 and 40.

The temperature is characterised by its variability. (The charts will be found attached to the cases.) Osborne says that this is dependent upon whether the nervous system is in a condition of excitement or shock. I have instances where on admission the temperature was as low as 97.6° F. both in
adult and child - Cases 7 and 31 -; on the other hand cases 4 and 15 were admitted with a temperature of 102.6° F.

In acute cases the temperature frequently rises rapidly before death, as seen in cases 28, 29 and 31. In case 29 it rose as high as 106° F. Foster notes a case rising to 109.2° F. In chronic cases the temperature remains irregularly high for a period of about three weeks, when it descends by lysis: it does not always ascend again before death, so that a subsidence of the temperature must not necessarily be regarded as a hopeful prognostic. In fact from this point of view it is of very little value.

Travers Smith (39) drew attention to the occurrence of an inverse type of temperature, which might be useful in diagnosing a difficult case from typhoid. It does occasionally occur, but did not do so in my cases for more than a day or two.

The pulse and respirations are as irregular as the temperature, and vary quite independently of it, and of one another; in the majority of cases the rapidity of both is increased.

The decubitus in over half my cases was lateral with the legs drawn up, and knees and elbows fully flexed. This lateral position is due to the presence of head retraction. Some cases would only lie
on one side. Case 2 was a noticeable example of
this. His cheek became rubbed, chafed and then
ulcerated on the right side, but he would not lie
on the left, and always turned back on to the right
when moved, though the face on that side must have
been very sore to lie on.

The purpuric rash from which the name "spotted
fever" originated I only saw present in 11 cases or
27.5%. I am able to show a photograph (No.I.) of
a good example. The rash on the chest does not
show in it on account of the movements of respira-
tion having blurred the spots. When limited in
extent the chest and upper half of the abdomen are
the chief sites, but in pronounced cases it appears
broadcast over all parts of the body and extremities.
Case 29 was the best example I had, and in it the
rash increased after death. The spots vary in size
from 1 to 5 or 6 mm. in diameter, have irregular but
sharply defined edges, and are of a purplish colour,
not disappearing on pressure.

Wright describes a very marked example of the
rash which is interesting. "Two haemorrhagic spots
with irregular edges of about the size of a lentil
and maroon in colour were seen on the inside of the
right thigh. Spots appeared within the next hour
on the feet, legs, and hands, and an hour later on
the abdomen, back and neck; during the hour follow-
ing several showed on the upper and lower eyelids." In the rapidity of the spread this resembles case 29, but I think both these must be looked upon as very exceptional cases.

Herpes I obtained in 12 cases or 30% and reproduce a photograph of the usual type (No.2) which is labial, but in case 1 was also present in both axillae.

Other eruptions consisted of Erythematous, Urticarial rashes, and Bullous eruptions. In a few cases on the extensor surfaces of the elbows and knees, and strictly limited to the region of the joint, a pin-pointed red purpuric rash appeared.

The tache cerebrale I found very inconstant, being only present in 19 cases.

The restlessness is a very common symptom, frequently is marked and sometimes extreme. I have seen a patient, case 9, lying on her left side, toss herself right over to the right and on to the floor. It is done in a moment, but they seem unconscious of the effort required to do it. Netter has exactly described this state by saying: "The patient is the subject to extraordinary convulsive movements, as if he were trying to throw off a frightful weight." In this stage they are indifferent to the bruising
which results from hitting their arms and legs against anything that comes in the way. All cases of course are not so severe as this, many simply tossing about mildly in bed.

The facial expression is of pain, even though unconsciousness be present. The face is frequently flushed. This is a cyanotic flush, bluish pink in colour and was present in quite half my cases. Ormerod says the face is usually pale - this is so in the later stage of chronic cases, but very unusual in early acute ones.

The smell I have already fully described.

Unconsciousness varies from a state of stupor, from which they may easily be roused, which is most common, to the extreme state of coma; or there may be a muttering delirium, the patient chattering quietly and incessantly as in case 36. In this case it was always of a pleasant nature, and he amused himself greatly by it.

Emaciation is most noticeable in any case that is protracted; I show two examples of this by photograph (Nos.3 and 4) and the one shows some head retraction as well.

In the nervous system there is little loss of muscular power early, except that owing to the stiffness in the neck and pain on attempting to rise the
patient cannot sit up. In the later stage, general weakness accompanies the emaciation.

Localised paralyses have been recorded. Mandouli describes a left facial paralysis which he thinks was due to thickening and adherence of the membranes. The case recovered and the paralysis disappeared. Lesynsky reports four cases of facial paralysis, but all ended fatally. Other paralyses such as hemiplegias and monoplegias, have been reported, but none appeared in any of my cases except paralysis of the ocular muscles, which occurred four times.

Hyperaesthesia is frequent and was present in about half of my cases. The localised hyperaesthesia over the spine mentioned by Osler I only found once.

Kering's sign was present in only 24 cases, and more often in children than in adults. It seems to be much more common than this as a rule; Travers Smith obtained it in his 36 cases without exception. I am inclined to agree with Ormerod that it is due to the pain caused by the tension which is put upon the inflamed sacral nerve roots.

The tendon reflexes are quite irregular, they may be markedly exaggerated, normal, or absent. I obtained a definite jaw-jerk seven times and in case
20 a well marked jaw clonus. The abdominal reflex I have already commented on.

Babinski was present seven times, but only in children.

Aphasia occurred in two cases, both of which were fatal.

The pupils were as irregular as the reflexes, sometimes being widely dilated, at other times quite small. In chronic cases they usually became dilated and remained so.

I made no ophthalmoscopic examinations, but it appears that optic neuritis is very seldom present. Travers Smith had no instance of it in his 56 cases and Koplik(44) says it is very uncommon. It does occasionally occur however.

Deafness I noted three times, one in a case that recovered, but the deafness remained. In all it was bilateral and absolute. Hypersensitiveness to noises so often mentioned, I was never able to elicit.

In the alimentary system. - The tongue in half my cases was coated with a brownish fur, the surface was dry and sometimes cracked, the edges often raw looking, and the papillae were sometimes prominent.

The appetite varied considerably. In a few cases it was good throughout, but never in a very acute one. The majority lost it during the acute
stage, to regain it as the more severe symptoms subsided. Some of the chronic cases had no desire for food, and even objected to it for weeks. Case 11 was an amusing extreme of this. He not only refused all food for about 10 days, but was so obstinate about it that after being nasal fed, he would induce vomiting by putting his fingers down his throat. Later on, however, he was never satisfied with the amount given him.

In the acute stage they are all very thirsty.

The vomiting, which was so frequent, in many cases was accompanied by a good appetite. I have seen a patient vomit severely and immediately call out for more food.

Seven cases were unable to swallow. At first they regurgitated part of the food through the nose, but later nothing could be swallowed at all. This difficulty is due, I consider, to the presence of marked head retraction. In these cases nasal feeding or gavage had to be resorted to.

Case 13 had a definite haematemesis.

Constipation was the rule and often of a very obstinate nature.

Loss of control of the sphincters was present in the acute cases, and in the chronics, when they became very emaciated and weak.
Haemorrhage from the rectum occurred in case 24 and was severe.

Tenderness of the abdomen on palpation was present in nine cases.

The liver dullness was occasionally diminished in extent.

The anterior border of the spleen was palpated only once.

In the respiratory system. - I have already referred to the presence of congestion of the throat. In one case there was a definite pseudo-membranous yellowish exudate on the tonsils. These were only enlarged in two cases.

In three cases a thick purulent discharge from the nose occurred.

Pulmonary trouble rarely was present at the outset; but in some cases undoubtedly hastened a fatal termination. Bronchitic and Bronchopneumonic signs were observed in 16 cases.

Cheyne-Stokes respiration was only twice present.

The pulse at the outset was invariably full, regular, and strong; towards a fatal termination it became irregular and feeble.

In one case the heart was dilated. In three others there was noted the presence of a murmur, but
these were presumably associated conditions and not due to the disease.

The examination of the urine. - The reaction was always acid. The specific gravity varied from 1.028 to 1.042. Albumen was present in 14 cases, i.e. 35%. In 10 of these it was only as a trace; in 3 the quantity varied from .2 to 11.4 grams per oz. Sugar was present in 3 cases. A deposit of urates was common. The Diazol-reaction was invariably negative and was of great use in diagnosing from typhus. The estimation of urea was not consistently carried out, but in the few cases where it was done, the quantity was under the normal.
The Blood.

I was able to make an examination of the blood in 32 cases, though unfortunately in only 23 did this include a differential leucocyte count.

The counts were made as soon as possible after admission, though some were delayed as long as 48 hours. They were taken before food to avoid digestion leucocytosis.

A Thoma-Zeiss Haemocytometer was used for the estimation of the red and white corpuscles, and Gowers Haemoglobinometer for the estimation of the haemoglobin, which was always made in daylight. For the differential count 250 leucocytes were counted.

The results are shown in Table II.

Leucocytosis, which has been the chief point in the blood noted by all previous writers, is very evident, ranging from 13,125 to 62,500 per c.mm., that is excluding the third count of case 5, which was taken two weeks after admission, when the acute symptoms were subsiding. The average count is 24,700, and 56% were over 20,000 per c.mm.

A fairly high leucocytosis is, I consider, the most favourable to find. The average count of cases that died under one week was 18,545, whereas on the contrary two cases with counts as high as 62,500 were fatal.
The low count designates a feeble resistance, the high count a profound invasion. Probably the best count is one of about 25,000, which I look upon as signifying a moderate invasion with a good resistance.

Cabot (45) says that with improvement the leucocytosis subsides. Cases 5 and 6 support this view but I have not sufficient examples to prove it.

The next noticeable feature is the high count obtained of the red corpuscles and corresponding high percentage of haemoglobin.

Taking 5,000,000 per c.mm. for a male and 4,500,000 per c.mm. for a female to be the normal, no less than 21 of my cases exceeded it. This fact was also noted by Curl (46) in his study of the blood in meningitis in children (not the cerebrospinal fever type).

It will be seen in cases 5 and 6, that the red count and percentage of haemoglobin subside with improvement.

The colour index is high, being in the majority of cases over 1.

No nucleated red corpuscles, no poikilocytosis, and no alteration in the size of the red cells were notices in any case.
Looking at the differential leucocyte counts, one is at once struck by their variability. Taking the following figures which I quote from Cabot as representing the normal:

<table>
<thead>
<tr>
<th>Cells</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Polymorphs</td>
<td>62 - 70%</td>
</tr>
<tr>
<td>Small Lymphocytes</td>
<td>20 - 20%</td>
</tr>
<tr>
<td>Large do.</td>
<td>4 - 8%</td>
</tr>
<tr>
<td>Eosinophiles</td>
<td>1/2 - 4%</td>
</tr>
<tr>
<td>Mast Cells</td>
<td>4 - 8%</td>
</tr>
</tbody>
</table>

My table shows the polymorphs to be somewhat in excess, generally at the expense of the lymphocytes which only figure between the normal given in five instances.

Another noticeable feature is the absence of eosinophiles in a great number of the cases. Cabot says they disappear in the early stages to reappear later.

In all instances where the glycogenic or iodine reaction was tried a negative result was obtained.

Briefly summarised, then, the results of the blood examination in the early stage presented the following conditions:

1. A high red count.
2. A pronounced leucocytosis.
3. High percentage of haemoglobin.
4. High colour index.
5. Polymorphs in excess.
6. Diminution of small lymphocytes.
7. Absence of eosinophiles.
8. Negative glycogenic reaction.
Lumbar Puncture.

This I did on 37 cases of which 35 were successful, the other two being "dry taps".

The method of procedure I shall describe in detail. The following are required:

1. Needle and stilette.
2. Pressure gauge.
3. Test Tubes.
4. Syringe that will fit the needle.
5. Thoma-Zeiss pippette for counting the cells. The one used ordinarily for white corpuscles is the best.
6. The diluting fluid for 5, e.g. a 0.3% solution of acetic acid coloured with methyl green.
7. Glass capsules of Eudrenine 0.5 c.c. size.
10. Swabs and small quantity of cotton wool.
11. Stimulants in case of collapse.

The needle selected should be a 3 inch, fine bore, platino-iridium one with a short sharp point. These details are important. Needles are sometimes supplied too short; it is most exasperating both to oneself and the patient to discover this when it is in the latter. The metal should undoubtedly be
flexible. I have seen the rigid ones used as a routine and have used them myself, but there is no comparison in the feeling of safety with the flexible needle, when the patient suddenly arches back his back. A broken needle in this site would be most awkward.

Then too, why use a large bore? It is supposed that the fluid when very turbid will not flow through a fine one. This is a mistake. It does not flow so fast certainly, but can always be got through a fine bore and the patient is far less disturbed. The outside measurement of my needles is only one millimetre. The detail of the point seems very trivial; the instrument maker delights in a long tapering point, which though beautifully sharp turns directly it touches bone and is thereafter useless, whereas a short sharp stumpy point remains sharp and unbent for a long period in spite of occasional contact with bone.

The pressure gauge that I used was of a simple if somewhat cumbersome type. It consisted of a scale 85 c.m. long graduated in centimetres. Attached to this was a fine glass tube ending at the bottom in a right angled bend. The capillary error was then measured and found to be 1.5 c.ms. The glass tube was therefore fixed to the scale so that the lower end commenced 1.5 c.ms. below the beginning of the graduations. This permanently allowed for the capillary
error so that the reading was taken direct. The gauge was attached to the needle by a short rubber tube in which was inserted a three-way glass tube, and a short rubber tubing was fixed on the third arm of this. A clip was fixed on each arm of the rubber tubing. The whole presented the appearance seen in the diagram. In using it the needle is inserted with the rubber tubing attached to it, in order to avoid any unnecessary discomfort to the patient by attaching it with the needle in situ. Clip A. is then closed and attached to its arm of the three way glass piece. The level of the lowest part of the glass tube D. is then brought to the level of the needle. With Clip C. shut, Clip A. and B. are opened and the fluid runs up the tube till it finds its level. The pressure may then be read off directly in c.m.s. Clip A. then is closed and Clip C. opened, so allowing
the fluid in the glass tube - a very small amount - to be collected. Clip B. is then closed. The scale may then either be detached or kept on, to take a second pressure reading at the end of the operation. Clip A. is opened and the fluid collected.

I give this detailed description of the apparatus because it does not seem to be much used in this country, though for years has been in use in the Johns Hopkins Hospital. It is of course open to error on account of the escape of fluid into the glass tubing, but this is such a small quantity that it may be disregarded.

The former manner of judging pressure according to the flow of fluid from the needle, is most inaccurate. Case 31, for instance, recorded a pressure of 56 c.m., yet the fluid only ran in very slow drops, and in case 34, only 2 c.c. could be collected after patient waiting, the fluid coming in the most dilatory drops, yet the pressure was 77 c.m. Conversely in case 25 the fluid spurted out as if under very high tension: the pressure was only 27 c.m.

We are now ready to follow through the operation. The patient should be placed on his side, left side preferred, with the knees drawn well up and head bent well forward so as to arch the spine as far forward as possible in order to give the greatest amount of space between the vertebrae.
Should a local anaesthetic be used? It is not necessary, but strongly to be recommended. Since I started using Eudrenine, I would never do it without: .5 c.c. is almost painlessly injected and in five minutes time the anaesthesia is perfect, allowing the needle to be deliberately and accurately inserted, with a minimum of disturbance to the patient. It is also far more comfortable for the operator; there is no crying or sudden jerking on the part of the patient. The pressure too is much more accurate on account of the absence of crying.

All the needles, syringes, etc. must be perfectly aseptic and the skin sterilised as much as possible. If bacteriological work is to be done an anti-septic compress will be necessary to get rid of the staphylococcus albus, and the test tubes must be sterilised.

The needle is inserted in the third lumbar space. The level of this is obtained by drawing a line joining the crests of the illia; this line passes through the required space. A very good guide is to draw the line actually with a skin pencil to within an inch or two of the spine on either side. One can seldom go wrong when this is done.

Having found the space, a point about three-eighths to half an inch from the middle line, on the upper side is then selected, a trifle below the mid
point of the interspinous space and the anaesthetic injected. I never go in through the middle line, though this is often recommended as being the best. How it can be so I cannot see, as the thick and tough supraspinous ligament has to be gone through, and there seems to be bleeding more often by that route.

The lumbar puncture needle now follows through the puncture made in giving the anaesthetic. It is directed forwards, inwards and a trifle upwards. When about 1½ inches from the surface there is sometimes an obstruction and a false impression that the point is right in; by tilting a slight amount either up or down one can feel the way in past this, and the point enters the spinal meninges with an unmistakable feeling as if the resistance had been suddenly removed. If now no fluid escapes, insert the stilette. Frequently this clears any slight obstruction. If still no fluid escapes attach the syringes prepared (No.4) and gently draw out the piston. A small clot of pus is often drawn through the needle in this way, and permits an uninterrupted flow. I think this simple manoeuvre should diminish many of the "dry taps" recorded. If, however, in spite of these precautions no fluid is obtained, the explanation is probably that the theca has just been missed: withdraw a little and point more towards the middle line.
If still unsuccessful withdraw altogether, clear the needle and repuncture.

The pressure gauge may now be attached in the manner I have described. The level will be seen to fluctuate with the respirations and pulse; if it does not, the needle is probably partially blocked. The reading should be taken as the mean of the highest and lowest level. The fluid is next collected in the test tubes which are ready. While this is being done the fluid should be drawn up into the Thoma Zeiss tube for the cell count. This is usually done after the operation, the fluid being taken from the test tube. I do not consider this so good, as the cells begin to settle directly. I consider that more accuracy is assured by using the fluid that is flowing from the needle. The amount of fluid drawn into the pippette must be varied according to the cloudiness of the fluid, more of a clear looking fluid being taken than of a very turbid one, naturally. The count can be made in the ordinary way and the dilutions calculated.

How much fluid can be collected? This depends entirely on the case. The only rule to go by is to stop directly headache is complained of. For ordinary diagnostic purposes 10 c.c. is ample. Very large quantities have been withdrawn with no bad effect, but unless for therapeutic reasons it is unadvisable, as by so doing the patient is exposed unnecessarily to a
slight risk.

That there is a risk is undoubted. Case 6 illustrates this. A moderately large quantity of fluid was collected (42 c.c.) but after 25 c.c. had been obtained the patient complained of severe headache which I ignored. He was apparently quite all right at the end of the operation excepting for the pain in the head. Within half an hour I was called back to the ward to find him dead. I have found 13 other cases of a fatal result after lumbar puncture (Gumprecht and Ossipow) but the danger seems to be greatest in cases of cerebral tumour. To guard against accident the patient should always be in the horizontal position, fluid should never be aspirated, and the operation should be stopped immediately the headache is complained of.

The fluid when obtained should be centrifuged, and the clear fluid examined chemically for albumin and reducing substance. Films should be made of the deposit, for the examination of the cells and presence of meningococci.

Before proceeding it would perhaps be as well to give an idea of the normal characters of cerebrospinal fluid.

It is perfectly clear and watery with specific gravity of 1003 to 1004 (Kopetzky). It is
faintly alkaline and contains albumin in quantity less than \( \frac{1}{2} \) gram per litre (Rous\(^{50}\)). Warrington\(^{51}\) says a trace of reducing substance - not sugar - is present, and Emery\(^{52}\) is most emphatic about this point as a diagnostic aid; he says it is always diminished or absent in meningitis. Foster\(^{53}\) says the substance is Pyrochatechin.

The pressure has been estimated at widely different figures from 40 to 60 mm. by Dana and Hastings\(^{54}\) to 70 to 300 mm. by Rous.

The normal number of cells appears to be about 0 to 2 per c.mm. Larnelle\(^{55}\) says over 5 per c.mm. is pathological, but Rous thinks this is too low: he found 6.5 per c.mm. in a case of pelvic abscess in which P.M. no meningeal changes were found. He therefore suggests 7 per c.mm. as the normal limit.

Such then is the normal fluid. The results obtained from the present cases are shown in Tables III. and IV.

The amount of fluid obtained varied from 2 to 42 c.c.

In character it was turbid, or very turbid in the majority of cases, often looking like thin watery pus. In 23 instances was it like this, in 7 only slightly turbid, nearly clear in 2, that is having the appearance of dust in a ray of sunlight, clear in one, and
bloodstained in one. In a case that is recovering the turbidity gradually disappears.

The pressure I unfortunately only recorded in centimetres in the latter half of the cases, the earlier ones being judged by the manner of flow from the needle. In the latter 16 instances where it was noted, however, it varied from 8 to 77 c.ms. and averaged 48 c.ms. excluding the second record of case 32. This shows considerable increase of pressure, which is due I consider to inflammatory hypersecretion of the cerebrospinal fluid.

From the cell count I had hoped to obtain some useful information, but the results were disappointing. In Flexner's experiments the greater the number of leucocytes exuded into the peritoneal cavity, the greater was the resistance; that is after inoculation into the peritoneal cavity. I thought, therefore, that a high count would indicate a high resistance and favourable prognosis. This is not the case. No prognostication can be obtained from the count. Taken alone and also in conjunction with the leucocyte blood count, I could discover nothing but discrepancies.

Case 25 with L.P. count of 143,750 recovered, yet case 34 with count of 97,344 died in 3 days, and case 12 with only 9,370 recovered. Similarly case 23 with L.P. count of 35,750 and blood count of 18,750
survived, whereas case 26 with corresponding counts of 34,320 and 21,840 died in four days.

Speaking generally a high L.P. cell count and a high leucocyctic blood count are co-existent. Whether they appear in large or small numbers will depend on the individual resistance and the virulence of the infection.

The actual figures show an enormous increase from the normal, ranging from 780 to 143,750 per c.mm., the average being 36,945 per c.mm. (omitting second counts) for the 22 cases in which the estimation was made.

The differential count which was made from stained films of the deposit from the test tube, show the polymorphonuclear cells to be greatly in excess. Kopetzky says that as the case progresses the polymorphs decrease, and are replaced by lymphocytes which gradually disappear with recovery.

In estimating the albumen I used an ordinary Esbach tube as used for albumen in urines. This method is coarse but gives comparative results and is simple. Rous uses a tube similar to Esbach but with a small bore. He suggests that a solution of phosphotungstic acid in hydrochloric acid would be a better reagent than Esbach's. The results show that it is always greatly in excess of normal, but it varies considerably in individual cases.
In every case there was an entire absence of any reducing substance. This quite agrees with Emery's statement above referred to. As it was consistently absent I have not included it in the table.

Meningococci were present in every case except Nos. 33 and 30. They were gram negative in all instances but one - case 8. They were usually both intra- and extra-cellular. They are said to disappear early in the disease, but I have found them present on the 32nd day and Fowler(56) says they have been found as late as the 90th day. Converseley Elser reports negative findings in three fatal cases, 24, 14 and 9 hours before death.

I show two photographs as seen in a film made from Lumbar Puncture. They are not always so numerous as represented, sometimes requiring a careful search.

The results on the whole were disappointing. The facts established were: the increase of pressure, the great turbidity of the fluid, the high cell count, the high proportion of polymorphs, the increase in the amount of albumen and absence of reducing substance, and presence of meningococci.

Primarily the value of the puncture was diagnostic and I shall again refer to this.
Clinically the severity of the attack was indicated by the percentage of polymorphs, but this can be judged equally well without a lumbar puncture.

No fact determined by the puncture was of any prognostic significance.

Fowler says that very turbid fluid swarming with organisms is unfavourable. Case 17 and 25 were marked examples of this state of the fluid, but both recovered. In case 33 the fluid on the other hand was very slightly turbid, and no meningococci were seen, yet he died.

Flexner concludes that by following the changes in the cerebrospinal fluid by repeated punctures, a favourable prognosis was indicated by disappearance of the meningococci, early immigration of leucocytes and active phagocytosis, and dissolution of the diplococci both within and without the leucocytes.

As a therapeutical measure I shall consider lumbar puncture with the treatment.
Complications.

These are numerous and may be serious.

In the nervous system the most frequent is hydrocephalus in some degree. Definite dilatation of the ventricles was seen P.M. in four out of fifteen cases, but besides these there were instances where symptoms of it being present were observed clinically.

The idea generally held is that it is due to closure of the foramen of Majendie. This cannot be correct because were it so we could not relieve it by lumbar puncture. I do not say that the foramen may not be occasionally occluded for it has been found P.M. to be so, but it is conceivable that in these cases the occlusion has occurred subsequently to the hydrocephalus.

I consider that the real cause of the hydrocephalus in these cases is due to inflammatory hypersecretion of the cerebrospinal fluid from the pia, which is forced up into the ventricles.

Unless very pronounced it is often difficult to diagnose it early, whereas it is important to do so. There is a better chance of a good recovery if relieved early, and moreover it is one thing in cerebrospinal fever that can definitely be relieved.

Koplik\(^{(57)}\) lays great stress on the importance of McEwen's sign, i.e. a tympanitic note on percussion
of the skull. "This," he says, "I consider one of the most useful guides in the subacute cases of meningitis in which there is threatened or well established hydrocephalus." For a description of the skull percussion I give McEwen's own account of it after 10 years experience. "The percussion note is obtained by the cranial walls vibrating when struck, the note being modified by the consistency and volume of the contents and their relative position to the bone. The sound elicited depends first on the susceptibility of the skull to vibrate, and secondly on the effect which the intracranial contents exert on the vibrations. When struck, a thin cranium vibrates more easily than a thick one. A skull may be so thick that it vibrates little if at all, to ordinary digital percussion. In the cranium of an infant, whose bones are only united by a membrane and when they lie somewhat loosely on the brain, the percussion note elicited is so slight, dull, and flat as scarcely to be perceptible. Should, however, the contents of such a cranium increase sufficiently to produce tension of the whole parieties - bones and intervening membranes - then the note becomes clear, particularly as the density in such a case is always low - the brain containing a considerable quantity of serous fluid. In the child whose fontanelles remain open,
especially in delayed closure from over distension, a clear drum like note is elicited on percussion. A comparatively thin skull may emit almost the same kind of note as a thicker one, when the contents of the cranium are intimately applied to the whole of its surface. Where the ordinary density, consistence and relation of healthy brains to the cranial bones exist, a dull note is produced on percussion, but where the contents are more fluid the bones will vibrate more readily, and a clearer percussion note will be obtained.

The note is best elicited near the pterion or a little behind that point, but when distinct may be produced all over the parietal and frontal regions."

The patient should sit up so as not to have the head touching the pillows. The head should be shaved and the ear of the investigator should be in contact with the head. Ordinary digital percussion is sufficient.

In hydrocephalus then, the percussion note should be high pitched or tympanitic.

I must confess that in no case was I ever able to elicit a definite high pitched note that I was certain of. The variation in the note seemed to me so fine, even in a definite case of hydrocephalus, that one imagined that it had been elicited when one wanted it. This of course is useless to be of
diagnostic value. Presumably my failure was owing to the non observance of some detail, but except for shaving the head I was unaware of any omission.

The clinical evidence of hydrocephalus in these cases consisted of headache, vomiting which was independent of food and without nausea, increasing stupor, irregular pulse, tremors and emaciation.

Of other complications in the nervous system, those affecting the ear are the most serious. Colles\(^59\) goes so far as to say that one fifth to one sixth of the cases of acquired deafness is due to cerebrospinal fever and that most deaf mutes owe their defect to an attack of the disease in infancy.

Four of my cases became completely deaf, Nos. 7, 13, 23 and 25; the latter two recovered but the deafness remained.

Acute otitis media is not very uncommon, but rapidly subsides without impairment of hearing.

The most common eye complication that I had was conjunctivitis which occurred seven times, and in one case was very severe. Strabismus was present in four cases. Of others that do occur may be mentioned Choroiditis, Keratitis and perforation of the anterior chamber (Lesynsky), Trichoroiditis with separation of the retina, and neuritis.

Next to the nervous system the respiratory is the most important, pneumonia and bronchopneumonia
being the most serious and often fatal complications; besides these, pleurisy, congestion and oedema occurred in my cases.

Of other complications I had few examples. Case II. developed Tabes Mesenterica during convalescence; Case III. produced a good specimen of Ascaris Lumbri-coides, though this of course is only an associated condition. Arthritis occurred in Case VI. in the right elbow.

Post Mortem Examinations.

Of the 29 cases that died in Hospital, I obtained a post mortem examination in 15.

In the more chronic cases emaciation was very pronounced.

Ievers and Elder(60) drew attention to the increase of the rash P.M. This was only noted in one case, No.XXXIX.

The meninges were definitely congested in 11 cases; there was no note in the other four. The congestion varied with the case, being most marked in the acute ones.

The convolutions were flattened in 7 cases. This was only slight in three, but well marked in the four others, which also showed dilatation of the ventricles.

In Case XXXV. accompanying the hydrocephalus there was present bilaterally at the posterior corner of
the ventricles, an area of purulent looking breaking down brain substance, pale pinkish-brown in colour of the size of a walnut.

The presence of the purulent exudate at the base of the brain was the most constant feature. Limited as a rule by the optic chiasma in front, it extended on to the pons and medulla posteriorly. In acute cases this was only seen as a thin film, but in others of longer duration it presented a yellowish, or pale greenish yellow, thick gelatinous appearance. It was only absent in case 29, i.e. the most acute case I had, but showed on the vertex. This is interesting as illustrating a purely cerebral case, as not only the cord but the base of the brain was clear. A thin film of purulent material usually occurs covering the whole surface, occupying chiefly the fissures, - the sylvian fissure particularly.

Why should the purulent exudate so constantly be present in large amount in a specially defined area at the base? The reason is an anatomical one. The pia and arachnoid membranes are closely approximated over the vertex, excepting when the pia dips down into a fissure, but at the base there is much more space between them and hence there is room for the accumulation of the exudate.

The cord shows acute congestion. A film of
purulent material is found, most frequently on the posterior surface, though sometimes on the anterior, or less commonly still, on both. In only three cases was this film absent, i.e. in case 29 mentioned above, in case 9 which had been treated by Lysol injected intraspinally, and case 33. It is curious that the exudate is not found on the upper parts of the cord, though the congestion is always general.

Next to the brain the condition of the lungs was of most interest, and it is noteworthy that every case had some pathological pulmonary condition. Four cases showed broncho-pneumonia, bronchitis was present in 12, marked acute congestion and oedema of both lungs in one, pleuritic adhesions in 10, two of which, however, were not recent. One case (No.16) showed a marked example of acute miliary tuberculosis not only in the lungs but also in the spleen and kidneys. Gangrene of the lung was present in case 10.

The heart showed little of interest except occasionally a few petechial hemorrhages, and in two instances evidences of myocarditis.

In cases 26, 30 and 35 the thymus was noticeably large. This was present in 6 of Elser’s 130 and all ran an acute course, as did my three.

In the alimentary system there was never noted any peritonitis. The mesenteric glands were enlarged
and congested in 11 cases. Inflammatory conditions of the intestines were common; they occurred towards the lower end of the ilium and upper part of the large intestine. This was noted twelve times. The solitary glands were very prominent and peyer's patches were often congested. In one case, No.5, an ulcer was present in the small intestine near the ilio-caecal valve.

The liver generally presented some cloudy swelling; in one instance cirrhosis was present.

The spleen was never enlarged; acute congestion was present in five cases, and acute miliary tubercle in one.

The kidneys also showed acute miliary tubercle in one case, pyaemic infarcts in one case and cloudy swelling in five others.

The suprarenals were without exception unaffected.

Prognosis.

There is no doubt, the prognosis is very serious. A mortality of 75% speaks for itself. Nevertheless, individually, no case at the outset should be despaired of. The actual severity of the symptoms counts for nothing. Only two cases out of my eleven cases
were mild ones; the other nine were clinically very severe.

The one fact observed in my cases that was of prognostic value was, the onset and its character. A sudden invasion was invariably followed by a fatal result. By this I do not mean the sudden onset of the first symptom, but this may be strikingly sudden yet the patient may for the next 24 hours not be fully "down" with the disease. In all my cures the onset was noticeably less severe than the others.

Apart from this I could discover no constant feature to be of any value in forming a correct prognosis. Some of my worst cases recovered, whereas others apparently recovering died, and the case least ill of all, with the shortest attack, died. This was case 21, which brings out a point not generally recognised, that some cases which apparently recover die suddenly at a later date. Flexner had examples of this with his monkeys and the cause of death was unexplained P.M.

The duration is most variable; acute cases may live only a few hours, whereas chronic ones may linger on for 8 to 10 months.
Diagnosis.

The diagnosis of cerebrospinal fever during an epidemic with a well marked case, and with the aid of lumbar puncture, is not particularly difficult. These conditions, however, are not always obtained, and to arrive at a correct diagnosis is frequently far from easy.

This is well shown in Billing's figures. Out of 2180 cases diagnosed as cerebrospinal fever, 357 comprising 38 varieties of illness proved to be of mistaken diagnosis.

In the early stage particularly, it often is exceedingly difficult to be positive.

Lumbar puncture is the most useful aid to diagnosis that we possess. It is simple, and when properly done is a perfectly harmless procedure, and the examination of the fluid for diagnostic purposes simply, is rapidly performed, and one which any practitioner may easily do himself, but even if he does not do so, a bacteriological and cytological report may be quickly obtained, which will at once establish the diagnosis of the disease, if present. By it one may first discover whether meningitis exists at all. This may be determined by the quantity of cells present, the increase of albumen and absence of reducing substance. If meningitis is found to be
present, then the nature of it may be discovered by the cytological or bacteriological examination. Excess of lymphocytes signifies tubercular meningitis, excess of polymorphonuclear variety, cerebrospinal fever or pneumococcal meningitis. The determination of the organism present, if found, further clears up the exact nature of the disease.

Kering's sign is of very much less value, in fact I doubt if much reliance can be placed on it at all. It is often absent, and further may be present in other forms of meningitis or when no meningitis exists at all. Menier reports that it may be found in Typhoid, Pleurisy with effusion, Hysteria and Vertebral rheumatism. It is also obtained in Sciatica and many chronic nervous diseases.

As I have mentioned previously, the neck rigidity should always be felt for - it is most characteristic.

It is of course in the initial stages when most difficulty occurs. The onset may simulate almost any of the infectious diseases, but of these Typhus bears most resemblance. I had three cases sent in as typhus that were cerebrospinal, and two sent in as the latter which turned out to be typhus. The first cases seen in London in 1865 were pronounced by Murchison to be closely allied to typhus. Though there are many points of resemblance, the onset in
typhus is not so severely abrupt, the temperature is very characteristic with its rapid rise, maintenance and subsidence by crisis, the face is much more congested and suffused, leucocytosis is slight or absent, and the urine gives a beautifully positive diazo reaction.

Scarlet fever may be mistaken for cerebrospinal with its sudden onset of vomiting and headache, especially with a slight sore throat, but the illusion should not be long lasting.

Influenza, commencing suddenly, may perfectly mimic the disease the first few days.

Tubercular meningitis is more insidious in its onset, causes less headache, and the cerebrospinal fluid is cytologically characteristic.

When seen late, a chronic case will often be regarded as one of typhoid. Several times I had Widal examinations to do for practitioners, who on the receipt of a negative result performed a lumbar puncture and forwarded characteristic meningococcal cerebrospinal fluid.

Of other diseases which came under my notice for differential diagnosis, were pneumonia, tonsillitis, uraemia, and gastro-enteritis.

A case of cerebrospinal fever may easily be mistaken for a pneumonia, as they not only have points
of similarity but an actual patch of consolidation is so often present after the first few days.

Uraemia may also easily be diagnosed as in cerebrospinal it is no uncommon thing to find albumin in considerable quantities, and if coma is also present as it often is, one is apt to jump to conclusions and miss the real nature of the disease.

As to Posterior Basic Meningitis, this I consider to be the sporadic form of cerebrospinal fever. A careful study of the account of posterior basic meningitis as given by Barlow and Lees\(^{(63)}\) reveals no noticeable differences, excepting minor details, the chief of which is that of age. In the sporadic or mildest form only children under 2 years, or the most susceptible and of least resistance, are attacked, whereas during an epidemic, when the virulence is increased, what is more natural than to find the age period broadened. The symptoms, pathological changes, and the cerebrospinal fluid in the two diseases are all similar, and the diplococcus seems to differ only in its viability. Still now holds that they are the same organism. Clinically too, the two diseases are indistinguishable.

Koplik\(^{(64)}\) distinguishes the two. During the epidemic of 1904 he saw 30 cases of supposed cerebrospinal fever of which 8 were typical posterior basic;
but he qualifies this by stating that both varieties may be due to the same cause, that under 2 years the disease takes on the posterior basic type, whereas if over 2 years old then the patient develops the cerebrospinal type. This seems to me to be equivalent to saying that the two are the same disease, with slight variations according to age.

Treatment.

Prophylaxis. The public health authorities during an epidemic should take the necessary steps to add this to the list of notifiable diseases, and render all possible aid to the visiting physician in any instance where cerebrospinal fever is suspected.

The system in New York is perhaps the most perfect. The Health Department supply outfits which may be obtained at certain drug stores throughout the city, for forwarding specimens of cerebrospinal fluid to them. These are examined and the result telephoned to the physician. If the latter does not feel able to do the puncture himself, he may call in from the Health Department a special medical expert to do it for him, or otherwise assist in diagnosing the case. This having been arrived at, it is at once notified.
When a case occurs it is always advisable to have it isolated either in hospital, or if circumstances permit at home. In the latter case two good nurses will be required, for the work entailed is exceptionally heavy. The poorer classes are quite unable to properly look after the case at home, and if it is at all an acute one, they are generally quite glad to have it removed to the isolation Hospital.

All contacts should be removed to a reception house for at least one night so that the house may be thoroughly sprayed and every stitch in it removed for proper disinfection. While at the reception house the contacts can have their clothing disinfected and their throats and noses syringed with some antiseptic, a weak solution of formalin being probably the best. Strong pungent solutions such as chlorine water, which was used in Leith last winter, are not accepted with a pleasant grace, though it is particularly efficient.

Whether treated at home or in hospital the nurses should be as careful in keeping their hands frequently cleansed, as in typhoid, and should gargle the throat with some antiseptic before going off duty. Soiled linen should be disinfected. Children who have been in contact with the case are better kept at home for a week or ten days. Visitors should be restricted,
and both they and the visiting physician should wear special cover coats kept for the purpose, before entering a ward.

Considering the high mortality these stringent precautions are well worth observing during an epidemic. With sporadic cases during a quiescent period they may be relaxed owing to the infectiousness being so much less.

**General.** The room or ward should be large, well ventilated but warm, and quiet.

The patients, unless where the bath treatment is employed, should be sponged over at least twice daily with hot water. This is of the greatest comfort to them and keeps down the smell that they emit.

Hot bottles will be required in plenty to keep the patient warm.

In the later stages, the greatest care is required to prevent bed sores, and often the nurse's skill will be severely tested.

A daily evacuation of the bowels is desirable if possible, but is often not easy to obtain when the obstinate constipation so common in this disease is present.

Nourishment may be given by small quantities of fluids in the acute stage, but later the greatest
care must be exercised to combat the rapid emaciation without overtaxing the digestion. Forced feeding either by nasal or stomach tube must be resorted to when food is refused, or unable to be swallowed.

Water may be given freely and is constantly asked for.

Alcohol is avoided by some on the ground that it is a brain stimulant, but I found that in small quantities it was often beneficial.

An ice cap, or ice applied to the nape of the neck, is strongly recommended. Osborne says:—

"The ice cap to the head and special ice bag are, I believe, very necessary, and especially an ice bag to the back of the neck is of positive value. I keep up these ice applications more or less constantly...."

If the temperature is subnormal he uses dry hot applications. I discarded ice entirely in favour of hot applications, as the latter gave such infinitely better results. In patients sufficiently conscious to tell the difference, the hot application was always preferred as giving more relief. It is required at the nape of the neck.

Cupping dry and wet have both been recommended as useful. Theoretically they should be, but I found the former of no value. Wet cupping I did not try.

During convalescence tonics, massage and gentle exercising are required.
Hot baths. This simple measure is undoubtedly the most effective. I used them at a temperature of 107° - 110° F. The patients were immersed for 10-15 minutes. The manner of action is, I think, to relieve the congested membranes by withdrawing the blood to the superficial capillaries. Why chronic cases should obtain so much benefit from the baths I do not understand, it should be of more use to acute ones. Such was not my experience. While acute cases obtained much temporary relief, so much so that they would cry out for them to be repeated, the chronics permanently and visibly improved, and it was this class of case that most forcibly brought out the usefulness of this method of treatment. Cases 11 and 7 were the most marked instances where improvement began with the administration of the hot bath.

The acute cases obtained relief from their pain, and obtained a good quiet sleep after the bath, in fact they often dropped off to sleep in it. The chronics, however, were encouraged to kick and splash about and so obtained splendid gentle exercise. The headaches were relieved and became less constant, their appetites improved, they became less peevish, and their muscularity and nutrition much improved. They always looked forward with pleasure to the bath and had a good refreshing sleep after it.
I consider that there is no single remedy we possess of such value as the regular and frequent use of the hot bath. Three immersions in the 24 hours I consider sufficient as a rule, though if possible in some cases four hourly ones would be preferable.

Unfortunately I met with such strenuous opposition in introducing this treatment, that in only a few cases was it systematically carried out, and even then imperfectly: in others it was done as occasion permitted, so that I can give no figures that could be at all accurate to illustrate its benefits.

Rogansky\(^{(65)}\), however, shows conclusively the excellent results to be obtained. He used the bath treatment in the female ward, treating 51 cases. Of these 34 recovered. In the male ward on the other hand, where he did not use it, only 10 recovered out of 50 cases.

**Drugs.** Those that have been tried are very numerous, but those that are of use are singularly few. First and foremost above all others must be mentioned morphia. This must be given in liberal doses; this can be done fearlessly, as even children appear to acquire great tolerance of the drug.

Quinine I also found of value in the early acute stage, though it is said to be contra-indicated as being a cerebral stimulant.
Ergot I tried, but with no beneficial results. This may be due to the manner of its administration. I gave it per mouth, whereas it appears that to obtain the best action it must be given intramuscularly or deep subcutaneously, at least so says Osborne who greatly praises it. Of it he says: "Physiologically it contracts the blood vessels and is of course indicated in cardiac weakness or with soft pulse and dilated arteries, but it also has a decided sedative action on the central nervous system, as it seems to contract and relieve congestion in the cerebral and spinal vessels. A patient who cannot sleep even with large doses of morphine will generally be found to sleep well after a dose of ergot has been added."

Stimulants will be required often and continuously. I gave strophanthus and sal volatile with the occasional use of a small amount of alcohol. Strychnine should be avoided.

In the convalescent stage, besides tonics, potassium iodide is of undoubted value and should be given as a routine.

Other drugs have been used, some of which I mention, but have no personal experience of their efficacy. Seibert recommends sodium salicylate given in large doses rectally. Five cases were treated and all showed a marked improvement. The
first case was given grs.XV. every hour for 10 doses, the subsequent cases had grs.XV. every 6 hours until 150 grains had been given. In a tubercular case it was also tried but no improvement was noted. The results certainly merit a further trial.

Intravenous injections of 4 c.c. twice a day of mercuric chloride 2 parts, sodium chloride 9 parts in distilled water up to 1000 parts, is another remedy well spoken of. Mercuric chloride has also been recommended given in hypodermic injections along the spinal column. Saline injections given rectally or subcutaneously were found beneficial by Ievers and Elder. They gave a soothing effect to the nervous system and stimulated the heart.

Lumbar Puncture. It was as a therapeutical measure that Quincke originally introduced this operation in 1891. It was done in a child of two years to relieve a recent hydrocephalic effusion. Recovery was complete after three punctures. The indication in this case was obviously increase of intracranial pressure. This is the one and only indication for a lumbar puncture to be of any use therapeutically, but when done for this reason the benefit obtained is assured. The headache ceases, as does also the vomiting, stupor or coma is lessened and the patient markedly improves at any rate temporarily.
Apart from relieving pressure it is questionable if the operation is of any therapeutical value. I never saw any improvement after it had been done in an ordinary case, but I cannot but think that the removal of fluid so purulent as it often is, containing probably thousands of meningococci must be beneficial. The remaining fluid must become diluted by the secretion of fresh fluid which may also be accompanied by more active phagocytic leucocytes. The toxicity will thus be reduced, and destruction of the remaining meningococci aided.

**Spinal medication.** Chipault\(^{67}\) was the first to replace the cerebrospinal fluid by a medicated one or a serum.

Since then the fluids used have been many, as for instance lysol, perchloride of mercury, iodoform, potassium iodide, anti-meningococcal, -pneumococcal, and -diphtheritic serums.

So far the results have been inconsistent, but the method is worth persevering with, as it is the only way one can attack the organism directly.

I must admit that my own experience with lysol injections was decidedly discouraging, as 6 out of 7 cases in which I tried it died. I varied the strength of the solutions from 50\% to 1\%, and as normal salt solution is also injurious to the meningococci...
coccus, I made the dilutions in it. Saeger (68) who introduced the lysol method used 9-10 c.c. of a 1% solution and obtained good results. Franca (69) also used a 1% solution, giving it in quantities varying with the age of the patient - for children 3-9 c.c. and for adults 12-18 c.c. He first withdrew 25-50 c.c. of the cerebrospinal fluid and if this was very purulent the canal was flushed with saline solution. Of 47 cases treated by simple puncture 30 or 64% died, but of 58 cases treated by the lysol injection only 17 or 29% died. He considers that the course of the disease is shortened, and relapses are prevented.

My bad results I put down to the fact that the cases were so severe, and were bad subjects to begin on, and too the number of cases was too few to draw conclusions from. I think it is well worth a trial, but advise the weaker solutions in preference to the strong ones. Lysol seems to have been used most frequently and to be the only antiseptic followed by good results.

Serums.

Antidiphtheritic Serum. The use of this was suggested by Wolf, of Hartford, Conn., to whom it occurred that there might be some antagonism between the two organisms because he noticed that there was a decrease of diphtheria coincident with the increase of
cerebrospinal fever. He then found that pure cultures of meningococci were killed by antidiphtheritic serum. He therefore treated four cases of cerebrospinal fever by anti-diphtheritic serum and all recovered. The success hoped for by this method has, however, not been realised. The difference in the seasonal incidence quite sufficiently explains why the cases of diphtheria decreased with the outbreak of cerebrospinal fever. The meningococci are so difficult to keep alive that it is not to be wondered at that they did not grow after being treated with anti-diphtheritic serum. Further, contradicting the idea that the two diseases are antagonistic, is the report by Simpson (70) of a case of cerebrospinal fever being complicated by severe laryngeal diphtheria. Peabody (71) treated 22 cases and Waitzfelder (72) treated 17 by antidiphtheritic serum and both considered that no influence was exerted either for good or the reverse.

Antipneumococcal Serum. This seems more likely to be of use than the above owing to the close resemblance of pneumonia to cerebrospinal fever, and of the pneumococcus and meningococcus to one another. I only used it once - Case 8 - owing to the presence of some gram positive diploccoci in the cerebrospinal fluid. No good result followed its use. Fevers and Elder, however, noted improvement after its injection
saying that the patient became quieter and went to sleep; the temperature rapidly fell, followed by a rise sometime later, but the patient seemed better and the pulse stronger.

**Antimeningococcal Serum.** The production of a really satisfactory serum of this nature should not be long delayed, and to this we must look as being the most rational and the best method of combating this disease.

Jochman(73) produced a serum which he claims to have distinct therapeutic properties: with it he was able to confer immunity to mice against six times the ordinary lethal dose of the cocci. For humans doses of 20 c.c. are required either intraspinally or subcutaneously. Of 17 cases treated by it only 5 or 29.4% died. Merck of Darmstadt claims to put on the market a serum produced according to Jochman. I unfortunately have not had an opportunity of trying it.

Ruppel's serum I tried in three cases. Case 27 was first tried as being a very acute case - he died the same night. Case 36 was a typical chronic. No difference was noted after the injection one way or the other. Case 37 was an example of a moderately acute case. She appeared to improve a little after the injection and ultimately recovered. Three cases
are too few to draw any conclusions from, however poor the results may be.

Burroughs Welcome & Co.'s serum I tried once, but the patient became so much worse after it that I never used it again, though at the Belvedere Hospital, Glasgow, they considered it the best they had used.

Until a satisfactory and well tried serum has been introduced, the best means of treating this disease I consider to be:-

Hot applications to the nape of the neck.
Hot baths.
Morphia.
Lumbar puncture whenever pressure is indicated.
Injections of 1½ lysol intraspinally.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age in years</th>
<th>Sex</th>
<th>Rooms in house</th>
<th>Adults</th>
<th>Children</th>
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<th>Occupation of patient</th>
<th>Occupation of patient's father</th>
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<th>Result</th>
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<td>2</td>
<td>2</td>
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M. = Male.  
F. = Female.
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<th>Result</th>
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<td>18</td>
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<td>1</td>
<td>-</td>
<td>Slater</td>
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<td>-</td>
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<td>?</td>
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<td>F.</td>
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<td>3</td>
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<td>-</td>
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**EXAMINATION OF THE BLOOD.**

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* Basophiles .5%
TABLE III.
LUMBAR PUNCTURE.

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<th>Meningococci</th>
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<td>+ I. few</td>
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<td>+ E. swarms</td>
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<td>Men + all I.</td>
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<td>Men -</td>
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<td>+ I and E.</td>
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TABLE III. Cont.

LUMBAR PUNCTURE.

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</tr>
<tr>
<td>32'</td>
<td>11.5 c.c.</td>
<td>28 c.m.</td>
<td>Slightly turbid</td>
<td>3.5</td>
<td>+I. many</td>
</tr>
<tr>
<td>33</td>
<td>13 c.c.</td>
<td>20 c.m.</td>
<td>Very slightly turbid</td>
<td>.75</td>
<td>None found</td>
</tr>
<tr>
<td>34</td>
<td>2 c.c.</td>
<td>Very slow</td>
<td>Very turbid</td>
<td>-</td>
<td>+I. and E.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>drops 77 c.m.</td>
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</tr>
<tr>
<td>35</td>
<td>16 c.c.</td>
<td>Rapid flow</td>
<td>Turbid</td>
<td>2.25</td>
<td>+I. and E.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>59 c.m.</td>
<td></td>
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</tr>
<tr>
<td>36</td>
<td>15 c.c.</td>
<td>33 c.m.</td>
<td>Very turbid</td>
<td>2.00</td>
<td>+I. &amp; E. few</td>
</tr>
<tr>
<td>37</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>38</td>
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<td>Flowed rapidly</td>
<td>5.00</td>
<td>+I. &amp; E. few</td>
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<td>Rapid drops</td>
<td>Turbid</td>
<td>3.5</td>
<td>+I. very few. None E.</td>
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<td>58 c.m.</td>
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<tr>
<td>40</td>
<td>3.5 c.c.</td>
<td>23 c.m.</td>
<td>Turbid &amp; blood stained</td>
<td>-</td>
<td>+I. and E.</td>
</tr>
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</table>

I. = Intracellular.  E. = Extracellular.
### Table IV.

**Lumbar Puncture.**

<table>
<thead>
<tr>
<th>Case</th>
<th>Cell count per c.mm.</th>
<th>Poly.%</th>
<th>Lymph</th>
<th>Large Monon.</th>
<th>Transitions</th>
<th>Degenerate forms</th>
<th>Notes</th>
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<tbody>
<tr>
<td>1.</td>
<td>-</td>
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<tr>
<td>3.</td>
<td>-</td>
<td>88%</td>
<td>12%</td>
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</tr>
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<td>5.</td>
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<td>-</td>
<td>-</td>
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</tr>
<tr>
<td>6.</td>
<td>-</td>
<td>Large number,</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>6'</td>
<td>2500</td>
<td>&quot; &quot;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>Followed by sudden death.</td>
</tr>
<tr>
<td>7.</td>
<td>-</td>
<td>94%</td>
<td>6%</td>
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<td>0</td>
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<td>-</td>
</tr>
<tr>
<td>8.</td>
<td>-</td>
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<td>3%</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>-</td>
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<td>8'</td>
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<td>3%</td>
<td>2%</td>
<td>2%</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td>9.</td>
<td>-</td>
<td>98%</td>
<td>2%</td>
<td>0</td>
<td>0</td>
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<td>-</td>
</tr>
<tr>
<td>10.</td>
<td>-</td>
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<td>0</td>
<td>0</td>
<td>20%</td>
</tr>
<tr>
<td>10'</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>All cells degenerated.</td>
</tr>
<tr>
<td>11.</td>
<td>31,250</td>
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</tr>
<tr>
<td>12.</td>
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<td>2%</td>
<td>7%</td>
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</tr>
<tr>
<td>12'</td>
<td>3,750</td>
<td>79%</td>
<td>21%</td>
<td>0</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>13.</td>
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<td>86%</td>
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<tr>
<td>14.</td>
<td>-</td>
<td>Many</td>
<td>Few</td>
<td>-</td>
<td>-</td>
<td>-</td>
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</tr>
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<td>0</td>
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<tr>
<td>16.</td>
<td>12,500</td>
<td>92%</td>
<td>8%</td>
<td>0</td>
<td>0</td>
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</tr>
<tr>
<td>17.</td>
<td>56,250</td>
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<td>5%</td>
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<tr>
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<td>41%</td>
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### TABLE IV. Cont.

**LUMBAR PUNCTURE.**

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<thead>
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<th>Cell count per c.mm.</th>
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<th>Lymph.</th>
<th>Large Monon.</th>
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<tr>
<td>19.</td>
<td>43,750</td>
<td>89%</td>
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</tr>
<tr>
<td>20.</td>
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<tr>
<td>21.</td>
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<td>13%</td>
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<td>24.</td>
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<td>29.</td>
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<tr>
<td>33.</td>
<td>780</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
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</tr>
<tr>
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<td>-</td>
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iv.


CASES.
CASE I.

D.O., aged 10 years. Schoolboy.

History.

Two weeks previously had been complaining of backache and pains in head and chest, and his neck had been painful.

A week before admission he vomited, and the next day complained of a sore throat. A scarlatinaform rash was supposed to have been seen as well.

On the strength of the above symptoms he was sent to Pilton Hospital, diagnosed as Scarlet Fever, and was admitted to the Scarlet Wards.

On admission:

Patient does not look very ill.

He is well nourished and well developed.

He lies on his right side with the legs drawn up and cannot straighten them fully without difficulty; when fully extended there is some slight lordosis.

There is a fresh crop of herpes on lower lip and in both axillae.

No rash.

Pulse 96. Respiration 82. Temperature 101.8°.

Nervous System - Patient shows some general muscular weakness.

Sensation is unimpaired.
Reflexes - Knee jerks increased on both sides, but more on right than left.
Patella clonus absent on both sides.
Ankle clonus present right and left; right most.
Plantar - lively: shows Babinski.
Kernig's sign is well marked.
Eyes - Pupils both dilated but react to light and accommodation. No paralysis of eye muscles.

Alimentary - Tongue is red with large papillae such as is seen in scarlet fever.
Spleen, liver, and stomach, normal.
No retraction of abdomen.

Circulatory - Nothing of note.

Respiratory - Throat slightly congested.

Urine - 1020, acid. Slight trace of albumin.

Lumbar puncture. Three attempts were made but in each case though the needle was properly in, there was a dry tap.

Progress.

For first week patient made none whatsoever, remaining absolutely stationary. He complained of thirst frequently. The bowels were constipated.

January 14th. Patient developed scarlet fever from having been admitted to that Ward, but made an uninterrupted recovery from it with no complications.
In three weeks the temperature had come down to normal, but after that time it would occasionally run up to 100 or 102°. These rises could always be foretold. The patient would become restless, cry out with headache and occasionally vomited, but at the same time his spirits would revive and he would sing songs in intervals of crying out with pain in his head. The temperature would begin to rise about 3 to 4 hours after the onset of these attacks, and the P. and R. were increased simultaneously. These attacks became more frequent towards end of February and more severe. He became very restless indeed and frequently required morphia to quieten him and enable him to get a night's rest. Progressive emaciation steadily increased till he became a mere bag of bones, as seen in the photograph No.3. Incontinence of urine and faeces followed, and with difficulty a whole skin was kept on him: he required a water bed.

In May he developed difficulty in swallowing, with regurgitation and had to be nasal fed. The head retraction became extremely marked and the legs and arms developed marked contractures.

Died May 22nd.

Post Mortem, May 23rd.

Emaciation extreme.
Arms and legs remained permanently flexed and permitted of no extension.

**Brain.** Convolutions were markedly flattened. In a few of the sulci were small deposits of firmly adherent pale yellowish material. Base of brain was covered with a thin layer of pale tough material.

On section, a very marked condition of hydrocephalus was present.

**Cord.** Posterior surface was covered with a thin layer of adherent tough material similar to that on base of brain.

Middle ears and ethmoid cells unaffected.

**Chest.** Right Lung firmly adherent to chest wall and upper surface of the diaphragm.

Bronchial glands enlarged.

No bronchitis.

**Left Lung.** Hard, enlarged, caseated Bronchial glands. Lung tissue healthy.

**Heart.** Nothing pathological to note.

**Abdomen.** No peritonitis.

Mesenteric glands very much enlarged and mesenteric vessels engorged.

The whole of the lower part of the small intestine is much congested. No ulceration: no involvement of peyer's patches. Solitary glands are prominent but not markedly so.

**Liver.** Small, hard and firm.
Spleen is also small and firm.

Kidneys - nothing of note.
CASE II.

D.J., aged 6½ years.

History.

He had been ill for 2 days; the onset was sudden with severe headache, followed shortly by vomiting. He then complained of his legs being sore. The same evening he was breathing very heavily and was delirious.

The child was one of five, all the rest were healthy. The house was clean, and there were three rooms for two adults and five children. The boy slept with the father; the other four with the mother.

On admission:

Patient well developed and fairly well nourished. Lies on his right side with legs drawn up, and he will not lie on left side: he always gets back to the right side when he has been turned.

Both cheeks have a cyanosed purplish appearance. He is very restless, very irritable and bad tempered: he is semi-conscious.

He objects to being touched or handled and whenever moved complains of pain in the neck.

Skin:— About the neck and upper part of the chest there are a few scattered dark brown subcutaneous haemorrhagic spots, in size about 1-2 millimetres
in diameter. There is no herpes.

Pulse 124; regular but weak. The respirations are 32 per minute. Temperature 101°.

Head retraction is marked, and neck is held absolutely rigid.

Apart from the fact that Kering's sign is well marked and that the motor power is fully retained, little else can be accurately determined in the nervous system owing to the extreme irritability and semi-conscious condition of patient.

Pupils are equal and neither dilated nor contracted. The left eye shows a small subconjunctival hemorrhage.

The tongue is covered with a thick white fur with red edges and papillae slightly enlarged, resembling somewhat a tongue of scarlet fever. Fauces, tonsils and soft palate are also red and congested.

The abdomen is neither distended or retracted. Alimentary organs are normal. Resents palpation of abdomen, but one cannot say that there is any abdominal tenderness.

There are no abnormal physical signs in the chest. The heart is not enlarged and the sounds are closed in all areas.

The urine - Acid:1018: shows trace of albumin.
The Blood - R.B.C. 5,550,000
W.B.C. 16,000
Hb. 110%

No differential count made.

Lumbar Puncture.

Quantity withdrawn - 5 drams.
Fluid ran in rapid drops, pressure slightly increased. No estimation.
Fluid was thick and turbid, whitish colour.
Rapid deposit of yellowish white sediment similar to pus.
Clear fluid after centrifugalisation showed distinct presence of albumin.
Fehling reducing substance absent.
Film of deposit stained both by Jenner and Meth.
blue showed many polymorphonuclear cells and the presence of the meningococcus—Gram negative.

After the L.P. patient was relieved for a short time, the restlessness ceased and he became easier—this lasted about an hour.

Progress.

February 8th - morning. Child very restless and noisy.
Morphia gr. 1/8th given 12.30 p.m., and he obtained some sleep afterwards.
Pulse was weak early in the night, but improved towards morning.
Takes feeds fairly well.
Passes urine in bed. Bowels not moved.

February 8th - evening. Restlessness continues all day. Has been delirious and trying to get out of bed.

Lumbar puncture repeated at 5.30 p.m. Slept quietly afterwards till 8 p.m.

February 9th - morning. Some difficulty in swallowing and some slight regurgitation.

Morphia gr. 1/8th repeated to give him some rest.

February 9th - evening. More sensible. Talks coherently but is nasal, as if there was some paralysis of soft palate.

Continues to lie on right side. The skin at the angle of the jaw on right side of the face is breaking down.

February 10th. Patient much worse.

Head retraction is extreme.

Face shows presence of a few haemorrhagic spots similar to those on the neck. The right cheek has broken down and the sore at the angle of the jaw is enlarging rapidly.

Left elbow shows subcutaneous purpuric rash.

The slightest pressure on the skin gives rise to redness which lasts a considerable time.
Pulse very variable in regularity and fullness. Difficulty in swallowing is again present with some regurgitation.

Tips of the fingers are blistered.

February 11th. Difficulty in swallowing and regurgitation more marked.

Has been very restless and has not slept all night.

Is quite conscious and talks rationally and answers sensibly.

Right leg shows occasional twitching; also twitching in both hands.

Sores on face increased. Right side is now covered with sores of broken down skin.

Left cheek also broken down.

Profuse pusy discharge from nose. Round nose on both alae nasi is ulceration spreading rapidly outwards.

Is very cyanosed.

Head retraction very pronounced and is slight opisthotonos.

Pulse still very irregular.

After midday feed about 1 oz. of greenish pus flowed out of the mouth and both nostrils.

Frequently now he has a tetanic spasm of whole body and limbs.
Have had to feed by nasal tube owing to swallowing being impossible.

February 12th. Face became covered with sores.
Sensibility retained.
Twitching more frequent.
Died at 11.30 a.m. No Post Mortem obtained.
Record of Temperature every 6 hours.

<table>
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<tr>
<th>Date</th>
<th>Time</th>
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<th>Respiration</th>
<th>Temperature</th>
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</thead>
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<td>06</td>
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<td>20</td>
<td>106.5</td>
</tr>
<tr>
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<td>12</td>
<td>70</td>
<td>25</td>
<td>104.5</td>
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<tr>
<td>6/14</td>
<td>18</td>
<td>65</td>
<td>20</td>
<td>84.5</td>
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</tbody>
</table>

Note: The temperature chart shows a steady decrease over the course of 6 days, indicating improvement.
History.

Patient got ill on March 2nd. The onset was apparently gradual with recurring headaches, shivering and a sore throat, but the details are not forthcoming as a neighbour only has brought her up.

The mother got cerebrospinal meningitis on Feb. 26th, i.e. four days before the child became ill. She was treated at home, but died on March 13th.

Patient's home is an excellent one. There are two adults and two children in four large well kept, well lit and ventilated rooms.

On admission.

Child looks pale, but is quite bright and cheerful.


Lies on right side curled up with knees drawn right up and seems to feel the cold much when bed-clothes are withdrawn, but body is warm.

No rash. No herpes. No smell.

Tache cerebrale well marked.

No retraction, but the neck is held quite rigid. Can lift up the body by the head without any flexion of the neck.

Understands what is being said and answers properly. Quite conscious.

Nervous System. Motor and sensory functions unimpaired.

Reflexes. Superficial - Epigastric, marked; abdominal, marked; plantar, marked flexion.

Tendon - Jaw jerk present; patella clonus present; knee jerk very active; ankle jerk and clonus slightly present; Kernig's sign present.

Spinal senses unimpaired.

Pupils - No strabismus: no nystagmus.

Respiratory. Impaired note at the right apex anteriorly and base posteriorly, with harsh breathing. Vocal resonance slightly increased and there are a few rhonchi present.

Circulatory. Nil.

Urine. Acid, urates. No abnormal constituents.

Alimentary. Tongue covered with white fur.

Throat congested.

Liver dullness very small. Nothing else of note.

March 24th. Lumbar Puncture.


Film from centrifuged deposit shows numerous polymorphonuclear cells, and one or two suspicious looking objects like meningococci.
124.

Blood count -  R.B.C.   5,910,000  
              W.B.C.   53,100  
              Hb.    110%  

Differential count - Polymorphs.  83%  
                           Lymphocytes  17%  
                           No others.  

L.P. Dif. count - Polymorphs.  88%  
                           Lymphocytes  12%  

Patient made an uninterrupted recovery and was discharged April 19th.

Seen in August - Was well grown, well fed and in splendid health except for a slight cough.
Daily Record of Temperature

<table>
<thead>
<tr>
<th>Date: 1st March</th>
<th>Subject: Mark</th>
<th>Age: 32 yrs</th>
<th>Disease: Cerebro-Spinal Meningitis</th>
<th>Result: Death</th>
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<tbody>
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<td>10</td>
<td>105°</td>
<td>42°</td>
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Temperature recorded every 4 hours.

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Temperature recorded every 4 hours.

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<tbody>
<tr>
<td>Time</td>
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<tr>
<td>Temperature</td>
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</tbody>
</table>

Temperature recorded every 4 hours.
CASE IV.

M.S., aged 45 years.

History.

On Friday 15th patient complained of a headache but went as usual to her work. She did not feel well all day and the headache increased. At midnight on 15th she vomited. At 4 a.m. on 16th she crawled out of bed and got underneath it and was violently restless, and knocked herself so much that she had to be restrained. She appeared to wish to speak but was unable to do so.

She then got violent and tried to strike her friends; later coma supervened and she became quite rigid.

Saturday 16th she was removed to Leith Hospital where Dr Ievers lumbar punctured her and obtained some turbid fluid with polymorphs in excess and meningococci present.

Sunday 17th she was transferred to Pilton Hosp.

Previous illness - She appears to have been a fairly healthy woman, but a drinker.

She works in a ropery with No.9.

She lives in a two roomed house in a very poor district. The house is occupied by three adults and is in a very dirty condition. Her husband was a dock labourer.
On admission:

Patient is quite comatose and cannot be roused by any means.

Development is poor: nutrition medium. Expression is vacant. Eyes are kept closed as a rule, but when she does open them the right eyelid is constantly winking.

Restlessness is very marked, arms and legs being thrown about constantly and she continually throws herself from side to side.

The legs are usually drawn up.

There is absolutely no retraction of the head: no neck rigidity.

Skin - No rash, and no herpes. The elbows and forearms and the head are bruised.

Patient is cold and with difficulty kept warm.

There is a soapy smell present.


Nervous System.

Motor power is quite unimpaired and she resists everything with greatest vigour.

Sensation - Cannot test: she felt the L.P. needle, but to a less degree than usual.

Reflexes also unobtainable owing to resistance offered.

Speech - Can phonate, but does not speak or answer questions.
Pupils are equal with medium dilatation; they react to light.

Lumbar Puncture.

Pressure is low: fluid only flowed in very slow drops.
Quantity - only 5 c.c.
Fluid is turbid: contains albumin = 3.5 grams per Litre: no sugar.
Film shows polymorphonuclear cells in excess, differential count being

| Polymorphs | 93% |
| Lymphocytes | 2% |
| Transitionals | 5% |

Meningococci - a very few intracellular, but swarms of extracellular present.

Alimentary.

Mouth is in a foul state; the tongue is covered with a brown fur and the mouth gets dry and sticky with thick dirty mucus.
Stomach - nothing of note.
Liver - superficial dullness is small in extent.
Spleen - no enlargement.

Respiratory.

Throat cannot be seen.
Chest - A few crepitations to be heard over base of right lung.
Circulatory System.

Apex is on L.M.L. in 6th space.
Sounds closed in all areas.
Pulse is rapid and weak.

Urinary.
Urine has to be drawn off.
Specific gravity 1035. Alkaline.
Albumin - a trace. No other abnormal constituents. Thick deposit of triple phosphates.

Blood.

<table>
<thead>
<tr>
<th>Count</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.B.C.</td>
<td>5,570,000</td>
</tr>
<tr>
<td>W.B.C.</td>
<td>15,600</td>
</tr>
<tr>
<td>Hb.</td>
<td>118%</td>
</tr>
</tbody>
</table>

Differential count - Polymorphs. 88%
Lymphocytes 11%
La. Mononucl. 1%

February 18th. Patient refused to take all food. Has had to be nasal fed.
Restlessness still marked. Been getting out of bed.

February 20th. Patient is sensible to-day and takes her feeds properly.
The Headache still present and back of neck is tender.
Complains of soreness all over her body.
Still has incontinence of urine and faeces.
Towards evening became delirious again.

February 28th. Wh.B.C. = 13,750.
March 2nd. Patient has been improving slightly being quieter except at nights when she gets more restless and requires bromides and sometimes morphia to give her any rest.

To-day she has got much worse and seems to be sinking: lies helpless and has assumed a typhoid state.

Pulse is feeble and irregular.

Bowels have been very constipated.

Chest. Right side has impaired note at apex; breath sounds are harsh with prolonged expiration and some rales.

March 3rd. Lumbar puncture performed. About 2 c.c. turbid fluid collected; flowed very slowly. the Film shows same characters as at first.

Lysol m.xx. were injected through the L.P. needle after the fluid had been collected.

March 5th. Patient in extremis; pulseless, respirations rapid but regular. No Cheyne Stokes respiration.

Emaciation very marked.

Post Mortem 38 hours after death.

Rigor Mortis general.

P.M. rigidity marked. Nutrition poor.

Superficial abrasions on body. No haemorrhagic rash.
Chest. Left pleural sac - no adhesions; recent pleurisy lower lobe. Right pleural sac - No fluid, no adhesions.


Right Lung - Marked pleurisy at the anterior part of upper lobe. On section, upper lobe markedly consolidated and on anterior surface a patch of beginning gangrene with fetid odor. Middle lobe emphysematous, dry, no distinct consolidation. Lower lobe; patches of collapse, congested, oedematous, capillary bronchitis and early bronchopneumonia.


Abdomen. No peritonitis. Small mesenteric and retro peritoneal glands slightly enlarged, not so markedly as in No.9
Small intestine - Distinct hyperaemia and catarrh.

Large intestine - No special pathological change; slight hyperaemia in the upper parts.

Liver. Smooth, cloudy, fatty, especially at the periphery of the lobules.

Spleen. Soft, small. On section dark acute congestion. The malphigian bodies slightly prominent; slight thickening of vessels.


No evidence of tubercle.
CASE V.

Mrs D., aged 41 years.

History.

All the history obtainable on admission was that the onset was sudden with sickness, headache, backache and vomiting, followed by pain in the neck. Herpes appeared two days later.

This does not quite tally with patient's description of the onset on her regaining full consciousness a month later. Her account of onset is as follows:

On 10th she felt light in the head and "faintish".

On 12th she felt wretched - "heavy and headachy"; the next day, 13th, took an"awful shaking": the whole body shook and the teeth chattered; she got into bed and it shook. At this time she saw everything yellow.

On 14th she got up for a few minutes and felt her head sore; the neck was stiff and sore when touched.

From that day on she doesn't remember clearly, but remembered being lumbar punctured and said it gave her great relief.

Patient looked after a day nursery where the poorer class left their children when going to work. Her own rooms were clean and roomy, five adults being in four rooms.
The children left were in all grades of dirt.

On admission.

Patient lies helpless and log-like.

She is quite sensible and answers questions perfectly rationally, but cannot remember recent events.

Lies on her back with the knees drawn up.

There is no head retraction, but the neck is absolutely stiff.

Skin - no rash. There is a large crop of herpes covering the lower lip and chin.

Temperature 103.2°. Pulse 108. Respiration 28.

Face flushed but not cyanosed. Has the characteristic smell well marked.

Nervous System.

Excepting for the stiffness of the neck the motor power is unaffected.

Sensation is unimpaired.

Reflexes - Marked jaw jerk: biceps and triceps jerk present: supinator jerk present; knee jerk increased; patella clonus absent: ankle clonus slight; no ankle jerk.

Epigastric and abdominal reflexes present.

Plantar reflex variable.

Kernig’s sign absent.

Alimentary.

Tongue is covered with dirty brown fur.
Takes food fairly well.
Nothing of note about abdomen.
Spleen - no enlargement.
Stomach - normal.
Liver - superficial dullness extends from the 6th rib to the costal margin.

Circulatory.
Pulse varies both in regularity and force. Is rather flickering.
Cardiac sounds closed in all areas.

Respiratory.
Note resonant throughout, and breath sounds vesicular.

Urine.
Acid: 1022. No abnormal constituents.

Lumbar Puncture.
No fluid obtained.

Blood count - R.B.C. 5,200,000
W.B.C. 25,000
Hb. 115%

February 19th. Patient is rambling continuously and has become restless, attempting to get out of bed. Complains of pain in the back of the neck being severe. Incontinence of both bladder and bowels.

February 25th. Abdomen is becoming distended and is tender. No vomiting. Flatus is passed
freely but bowels are constipated. Note of percussion is tympanitic all over abdomen.

General condition is much worse: is picking at the bed clothes and has low muttering delirium. Has now retention of urine.

Urine.

Alkaline, 1017. No abnormal constituents.
Deposit of phosphates.

February 28th. Wh.B.C. 31,000.

March 3rd. Patient seems slightly improved.

Catheterisation discontinued.

She complains of the cold constantly and has to be given extra blankets though the ward is not chilly.

Slight external strabismus of right eye, and right pupil seems slightly higher than the left.

March 7th. Patient now can move the head in any direction without discomfort, but she says she feels sore all over and as if there was a very tight cap on her head.

March 21st. Patient has made a slow but steady improvement.

Blood count - R.B.C. 4,500,000
W.B.C. 8,125
Hb. 98%

March 25th. Patient was allowed up for 1 hour.

April 21st. Patient has steadily improved.

She can now walk the length of the ward. There are
occasional headaches, frontal, and occasional vomiting but not to upset her at all.

Sometimes there is some incontinence of urine: never of feces.

Nystagmus still present, but sight is good. There is slight impairment of hearing, chiefly on the right side.

April 29th. Patient while up to-day went along to the bathroom by herself and was later found to have fainted there. She was put back to bed and felt none the worse in the evening.

April 30th. Patient not so well; did not let her get up. Felt return of headaches and was sick twice.

May 2nd. Patient still getting worse. Losing interest in her surroundings and is greatly discouraged at not improving.

May 8th. Has some difficulty in speaking, talking very thickly as if mouth was full of mucus which she cannot clear.

May 10th. Incontinence of urine and feces returned.

May 19th. Patient getting very much weaker and is rapidly sinking. Voice very hoarse.

June 2nd. There is a heavy urinous odour about her. She is absolutely helpless, lies on her back and cannot move her position herself.
Voice is reduced to a whisper.
There is low muttering delirium.

**June 4th.** Pulse has got much feeble. Breathing increased in shallowness.
Died in the afternoon.

**Post Mortem.**
Emaciation marked. Foul heavy odour.

**Brain.** Membranes very much congested. Adherent to brain substance, slightly at vertex but firmly so over all base.

Brain firm in consistence. No marking flattening of convolutions. The surface has slight evidences of a thin greyish film in the sulci.

Base has this film more marked.

**Section - Nothing of note.**

**Cord.** Posterior surface covered with grey tough film.

**Chest.** Lungs - No adhesions. Bronchial glands normal. Evidence of bronchitis in larger bronchi, and oedema at the base of the right lung.

**Heart - Nil.**

**Abdomen.** No peritonitis.

Mesenteric glands markedly enlarged and congested. Small and large intestines show slight congestion: in lower end of ilium is a small round ulcer
with indurated edges curled over on themselves into floor or ulcer.

Liver - Soft: shows fatty change.
Spleen - Soft: substance almost pasty.
Kidneys - Nil.
**Clinical Chart**

**Name:** J. C. Coonag

**Age:** 27 yrs

**Diagnoses:** Cerebral Spinal Hemorrhage

<table>
<thead>
<tr>
<th>Day</th>
<th>Pulse</th>
<th>Respiration</th>
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<tbody>
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<td>17</td>
<td>106°</td>
<td>20</td>
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<tr>
<td>18</td>
<td>102°</td>
<td>20</td>
</tr>
<tr>
<td>19</td>
<td>101°</td>
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<td>100°</td>
<td>20</td>
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<tr>
<td>21</td>
<td>99°</td>
<td>20</td>
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</table>

**Result**

- Death

**Records of Temperature—every 1 hour**

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<tr>
<th>Date</th>
<th>Time</th>
<th>Temperature (°F)</th>
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</thead>
<tbody>
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<td>Feb. 17</td>
<td>106°</td>
<td>106°</td>
</tr>
<tr>
<td>Feb. 18</td>
<td>102°</td>
<td>102°</td>
</tr>
<tr>
<td>Feb. 19</td>
<td>101°</td>
<td>101°</td>
</tr>
<tr>
<td>Feb. 20</td>
<td>100°</td>
<td>100°</td>
</tr>
<tr>
<td>Feb. 21</td>
<td>99°</td>
<td>99°</td>
</tr>
</tbody>
</table>

**Result**

- Death
CASE VI.

J.G., aged 27 years.

History.

On Saturday 16th complained of headache: on Sunday the headache was severe and he vomited 2-3 times and was markedly ill.

No further details of the history were obtainable. He was removed on Sunday 18th to Leith General Hospital where he was lumbar punctured; 25 c.c. of turbid fluid was drawn off under high pressure, and contained polymorphonuclear leucocytes in abundance and a few intracellular diplococci. He was then transferred on 19th to Pilton Hospital.

While in Leith General, squinting developed in the right eye.

Patient was a miner, working both in Musselburgh and Fife, and recently also at Leith Docks.

He lived with five other adults in a two roomed house which was fairly clean.

On admission:

Patient appears very ill.

He lies quite quiet but can turn himself about.

He is in a semi-conscious dazed condition, having to be shaken and shouted at before he will answer
a question, which he does rationally.

Skin. Slight herpes under the chin: on left elbow are two vesicles.

Patient has to be kept warm with plenty hot bottles. He has a very strong characteristic odour. It permeates the whole ward.

There is some slight degree of head retraction, but the neck is held absolutely rigid, and is tender to pressure over the vertebrae. Muscles on either side are very tender.

Temperature 100°. Pulse 70. Respiration 32.

Nervous System.

Motor power and sensation unimpaired.

Reflexes - Well marked jaw jerk; biceps jerk exaggerated; supinator well marked; patella clonus absent. Knee jerks well marked; plantar reflex present and active. Epigastric and abdominal reflexes present.

Kernig's sign is very marked.

Eyes. There is internal strabismus of the right eye; double vision occasionally. Pupils equal and small; do not react to accommodation or light.

Alimentary.

Tongue is covered with a thick brown fur.

Abdomen is neither distended nor contracted.

Liver, spleen, stomach, normal.

Bowels very constipated.
Respiratory.

Throat is congested.

Chest shows no abnormal physical signs.

Circulatory.

Heart is not enlarged and sounds are closed in all areas.

Pulse varies both in rate and regularity.

Urine.

Urine all passed involuntarily. Specimen collected shows specific gravity 1018; acid, with a trace of albumin.

Blood count -

<table>
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<th>Component</th>
<th>Value</th>
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<tr>
<td>W.B.C.</td>
<td>21,000</td>
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<tr>
<td>Hb.</td>
<td>115%</td>
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Differential count -

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<th>Value</th>
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<td>Polymorphs</td>
<td>72%</td>
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<tr>
<td>Lymphocytes</td>
<td>23%</td>
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<tr>
<td>Transitional</td>
<td>2%</td>
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<tr>
<td>Large Mono-nuclear</td>
<td>3%</td>
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</tbody>
</table>

Lumbar puncture.

Twenty-three c.c. turbid fluid was collected; pressure was not very high, the fluid running in slow drops.

Film showed nearly all polymorphonuclear leucocytes with a few meningococci - all intracellular.

Albumin present; 9 grams per litre.

Fehling reducing substance - none.

Patient seemed to be much easier after lumbar puncture.
February 20th. Was restless and noisy over night and got no sleep till 4 a.m. when ½ gr. morphia was given.

February 21st. Much worse. Lies absolutely log like. Breathing has become stertorous.

There is slight conjunctivitis in both eyes, and the right eye he cannot close at all. The left pupil is dilated, and there is some ptosis of upper lid - of left eye.

Herpes is spreading over the whole of lower lip and there is a fresh crop on the edge of the nostrils.

February 22nd. Coma increasing. Cannot rouse him at all. Pulse feeble and irregular.

February 25th. Much better: answers questions sensibly again.

Pupils are equal but he still has difficulty in closing the right eye.

Passing urine and feces involuntarily.

February 26th. Right elbow has become painful and stiff: gives much pain when moved, but there is no redness, or swelling.

February 28th. Very much improved in every way. The elbow is much less painful and he can move it.

From now on till March 7th patient made very good progress indeed. He slept well, took his food well, was quite sensible and in every way was much improved,
and he was allowed to be proped up in bed.

He was weakened considerably and had got very thin.

March 5th. Blood count - R.B.C. 4,900,000
W.B.C. 18,750
Hb. 105%

March 6th. Complained of headache.
March 7th. Temperature up to 102.6°. Pulse and respiration simultaneously were increased. Complained of severe headache. Pain and stiffness in the neck has returned but no sickness.

Left pupil dilated and there is ptosis of the upper lid.

March 8th. Temperature has come down and patient feels quite well again. Pulse is slow and irregular.

March 10th. Temperature again raised with fresh onset of headache, throbbing frontal variety. Occasionally he has shivering attacks.

March 11th. Patient slightly better to-day. Lumbar punctured at 6.30 p.m.: 42 c.c. of fluid were collected.

Estimation of leucocytes by Thoma Zeiss' haemocytometer = 2500 per c.m.

Fluid slightly turbid. Film shows polymorphs preponderating.

Patient complained of severe headache towards end of the operation.

After L.F. 25 c.c. of normal saline and lysol 2% were injected through the L.P. needle.
The fluid was kept in two parts and albumin estimated in each.

1st Part gave 4½ grams per litre.
2nd " " 2½ " "

There were no meningococci found.

After L.P. patient was very quiet and at 7.15 he was noticed to change colour. He turned suddenly white and was pulseless and died immediately.

No Post Mortem was obtained.
CASE VII.

Mat. A., aged 5 years.

History.

On 18th patient complained of severe headache chiefly at the back of the head. On 19th the headache was worse and there was pain in the neck and down the back: the neck was noticed to be a little stiff.

There was some epistaxis yesterday.

A rash was noticed to have come out yesterday on the chest.

Patient lived in a fairly clean house with a moderate amount of room, there being three rooms occupied by two adults and four children. The father worked as a salesman about the docks.

On admission:

Nutrition good. Development fair.

Patient is quite sensible and conscious.

He lies very quiet, moaning frequently.

The head is kept perfectly still, as any movement of it causes pain. There is no retraction at all, and no board like stiffness of the neck, but it is held steady on account of the pain.
Skin - over the upper part of the chest there is a scattered purpuric eruption. The spots are few in number and widely scattered. A few are to be seen on the legs and arms.

The cheeks have both a cyanotic flush.

Tache is well marked.

No characteristic smell.

No coldness.

Eyes are bright and intelligent, and pupils equal, medium, and react to accommodation and light.


Nervous System.

Motor power and sensation are unimpaired.

Special senses are unaffected.

Reflexes - Jaw jerk present, but no other tendon reflex obtained.

Abdominal and epigastric reflexes are present.

Plantar reflex - active Babinski.

Kernig Sign - very faintly marked.

Alimentary System.

Tongue covered with dirty brownish white fur.

Abdomen - nothing of note.

Liver - Dullness extends from the 6th rib to half an inch below costal margin.

Spleen - no enlargement.

Stomach - normal.
Circulatory and Respiratory

systems presented nothing abnormal.

Urine.

Acid. 1017. Albumin - a trace. No other abnormal constituents. Deposit of urates.

Blood.

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<tbody>
<tr>
<td>R.B.C.</td>
<td>5,580,000</td>
</tr>
<tr>
<td>W.B.C.</td>
<td>20,280</td>
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<td>Hb.</td>
<td>115%</td>
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Differential count -

<p>| | |</p>
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<tbody>
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<td>Polymorphs</td>
<td>75%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>18.5%</td>
</tr>
<tr>
<td>Large Mononuclear</td>
<td>5.0%</td>
</tr>
<tr>
<td>Eosinophiles</td>
<td>1.5%</td>
</tr>
</tbody>
</table>

Lumbar Puncture.

20 c.c. were collected. Fluid turbid; rapid deposit of purulent matter.

Film showed Polymorphonuclear cells 94%

Lymphocytes 6%

Meningococci, both Intra- and Extra-cellular were present in small numbers.

Albumin - 9 grams per litre.

Fehling reducing substance - absent.

February 24th. Lumbar punctured. Two c.c. allowed to flow but no more taken off. Lysol m. v. pure in m. xv. sterile water was then injected through the lumbar puncture needle. The fluid was slightly clearer than in first L.P.
The film shows a few polymorphs and leucocytes, but the majority of cells are broken down. A few intracellular meningococci were seen.

**February 25th.** Temperature rose after lumbar puncture to 100.2°.

He, however, seems very much better to-day. Complains of no pain when touched and can move his head about quite easily and seems generally much brighter.

**February 28th.** The urine has to be drawn off. Lysol m. v. in sterilized water m. xv. again injected per L.P. needle.

**March 4th.** Wh.B.C. 15,600.

Patient has become quite deaf.

He does not appear so well. Constantly flushes up for no reason and as quickly pales. Catheterization continued.

Urine - faintly acid, 1020. No abnormal constituents. Patient is rapidly losing flesh.

**March 6th.** Right forefinger has developed a superficial whitlow.

**March 8th.** Patient has now incontinence of foeces; the stools are loose, greenish, with some undigested curds.

The catheterization has not been needed to-day. Put on peptonised milk and rectal irrigations daily.
March 10th. Emaciation is getting very marked, and it is becoming difficult to keep the skin from breaking.

The urine is now freely passed, there being incontinence both of foeces and urine. There appears to be pain in abdomen. He lies with the knees drawn up and resents being touched at all. Cannot localise any spot where tenderness is greatest. There is internal strabismus of both eyes.

March 18th. Patient taking feeds better during the past week, but is making no satisfactory progress. On lower half of abdomen several small pustules have appeared.

Squinting continues to be very marked.

Character of the motions has improved considerably.

The pulse is feeble and has been very irregular.

March 24th. Emaciation becoming extreme.

Has been vomiting once or twice a day for no reason and no special time.

Occasionally hands become very tremulous.

He has taken to screaming out at times - piercing scream.

April 3rd. Patient more emaciated.

Takes food badly; has had to be nasal fed.

Over abdomen is a thick crop of purpura.
April 8th. Refusing all food: spits it out.
Fed nasally and by nutrients.

April 11th. For the first time for some weeks the legs are fully straightened.
Kering's sign still present and reflexes active.
Pupils widely dilated.
Some nystagmus present.

April 13th. Patient appears brighter and face a little fatter. Takes notice of things.
Purpura of abdomen decidedly less.
Very tremulous, hands and arms particularly; head also. Objects very much to bed clothes being taken off.

April 15th. Strabismus much less.

April 16th. Temperature up. General condition much worse. Strabismus returned. Face pale and more pinched. No sickness.

April 18th. Left ear discharging.

April 21st. Put on to hot baths yesterday.
Seems very much brighter; takes notice again.

No squinting.
In the bath he managed to straighten legs and move them about.
Emaciation still marked.
Incontinence of both bladder and rectum still present.
No cough.
April 28th. Left ear stopped discharging.
Can move head about quite easily.

Patient died on 1st May. No Post Mortem.
### Daily Records of Temperature

<table>
<thead>
<tr>
<th>Name: Annie McDonald</th>
<th>Age: 6 yrs</th>
<th>Diagnosis: Cerebral Spinal Meningitis</th>
<th>Result: Death</th>
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</table>

#### Chart Details
- **Date**
- **Day of Discharge**
- **Temperature (°F)**
- **Pulse**
- **Respirations**
- **Urine**
- **Bowels**

#### Graphical Representation
- Temperature fluctuations over time.
- Pulse, respirations, and other vital signs are noted.

---

### Record of Temp. every 4 hours

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<th>4th</th>
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<td>105°</td>
<td>104°</td>
<td>103°</td>
<td>102°</td>
<td>101°</td>
<td>100°</td>
<td>99°</td>
<td>98°</td>
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</table>

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### Pulse, Respiration, Bowels

- **Pulse**: Variations indicated.
- **Respirations**: Variations indicated.
- **Bowels**: Variations indicated.

---

### Clinical Details

- **JVP**
- **H. Silverlake**
- **Blood Pressure**

---

*References and Notes*:
- Copyrighted material indicated.
CASE VIII.

Annie McD., aged 6 years.

History.

On 18th patient came home in the evening and felt cold and was noticed to be shivering a great deal. She was put to bed but woke up in the night and vomited.

On 19th she got up and went about as usual, but was out of sorts and did not join in playing with the others, and was off her food. On 20th she was the same.

On 21st she woke up with a severe headache, which was frontal, and again vomited. She was quite felled and unable to get up.

She complained of abdominal pain.

She got rapidly worse and a doctor was called in who diagnosed Cerebrospinal Fever.

A purpuric rash was reported to have been seen on 21st.

The next day she was admitted to L.P.H. Hospital.

Patient lived in a fairly clean house of two rooms occupied by two adults and five children, all of whom slept in one bed. The father was an iron driller. He was in contact with father of Case 2, working on board a German steamer.

The stair was inclined to be dirty.
On admission:

Nutrition is poor: development fair; is in a very dirty state.

Patient is unconscious, but seems to understand what is said when roused but all the rest of the time rambles.

Restlessness is marked, continually tossing about.
Lies on right side chiefly, and with legs drawn up.

There is very marked head retraction and the neck rigidity is absolute. There is marked tenderness at the back of the neck and the muscles are very tense.

The face has a suffused appearance, with a purplish red flush on each cheek.

The eyes are clear and uncongested.

The skin shows a faint mottling as of old flea bites, but which could possibly be a faded purpuric rash.

There is no herpes.

Tache cérébrale is well marked.

Has the soapy smell.

Hands and feet are cold.

Temperature 98.2°. Pulse 112. Respiration 28.

Nervous System.

Motor power is unaffected.
Sensation - to touch is unimpaired, but there is marked general hyperaesthesia.
Kernig's sign well marked.
Eyes - Pupils equal, and moderate dilation.
Reflexes - Abdominal and epigastric absent.
Plantar present. Other reflexes, cannot determine.

Circulatory.
Pulse is full and tension moderate. Sounds closed in all areas.

Respiratory.
The tonsils are large and congested, and the posterior pharyngeal wall is congested.
The chest shows nothing abnormal.

Alimentary.
Tongue is covered with a white fur.
The abdomen presents nothing abnormal. The liver, stomach and spleen are of normal size.

Lumbar Puncture, February 22nd.
Fluid turbid - of greenish colour. Fifteen c.c. were collected.
There was rapid sedimentation of pus.
Film showed diplococci both intra- and extra-cellar; not very numerous. Some were distinctly gram positive.

Polymorphs. 97%
Lymphocytes 3%

Albumin - 5 grams per litre.
Urine.

Specific gravity 1018. Acid.

Albumin - a trace. Deposit of urates.

Blood count.

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<td>R.B.C.</td>
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<td>W.B.C.</td>
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<tr>
<td>Hb</td>
<td>118%</td>
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</table>

Differential count - Polymorphs. 81%
Lymphocytes 13%
Large Mononuclears 4.5%
Transitional 1.0%

February 24th. On account of the presence of gram + diplococci, antipneumococcal serum was given subcutaneously in the abdomen.

February 25th. Abdomen is tender, but due probably to the serum. No distension or retraction. Bowels have been moving properly, but the motion this morning has some blood stained mucus in it.

Lumbar puncture (ii.)

19 c.c. collected.

Needle frequently blocked with clots of pus, which were withdrawn by fitting a syringe to the needle and sucking the pus through.

Fluid - turbid with clots of pus.

Film - Very few meningococci: all intracellular.

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<table>
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<tbody>
<tr>
<td>Polymorphs</td>
<td>93%</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>3%</td>
</tr>
<tr>
<td>Large Mon.</td>
<td>2%</td>
</tr>
<tr>
<td>Disintegrated</td>
<td>2%</td>
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</tbody>
</table>
Albumin - 1 gram per litre.

After lumbar puncture 15 minims of pure lysol diluted to 45 minims in normal saline were injected into spinal canal.

Urine

Neutral. 1022. Albumin absent.

Skin. An erethematous rash appeared on arms and dorsal surfaces of the hands.

Has been very restless and noisy, crying out frequently, and once got out of bed.

February 27th. Since L.P. of 25th patient has seemed to be improving; she has been quieter, slept better and taken food better. The pain in the neck has subsided and hyperaesthesia has disappeared.

February 28th. Lumbar puncture (iii.). 2 c.c. collected with difficulty.

Fluid is turbid with small clots of pus in it.

Film - Polymorphs. are all disintegrated; there are very few intracellular meningococci.

Lumbar puncture was followed by a further injection of lysol m. xx. being given, intraspinally.

White B. C. 62,500.

Urine has to be drawn off by catheter.

March 2nd. Patient has not improved in the last four days. Has taken her food badly, and been Occasionally sick and is frequently retching.
The abdomen is distended and slightly tender.

The bowels are inclined to be loose and the motions are green.

The pulse has become weak and she frequently gets cyanosed.

The respirations vary very much: inclined to be Cheyne Stokes. The respirations vary from 16 up to 30 per minute.

The urine now is passed involuntarily frequently.

March 4th. Patient has slight cough and at right base there are some fine crepitations.

The skin appears to be itchy; both sides of chest and abdomen are severely scratched on both sides.

The skin has become very tender, the least pressure leaving a red mark; the elbows and knees especially have become almost sore.

March 6th. Crepitations still present at right base. No dullness.

Pulse has become very weak and irregular.

Has been difficult to keep warm.

Over chest and abdomen a purpuric rash, widely scattered, has appeared.

Vomiting occasionally.

Bowels constipated.

March 7th. Patient is failing.
With difficulty can only get her to take a very little nourishment.

Vomits occasionally without effort.

Bowels very constipated.

Right side of chest - The breathing is harsh and accompanied by rales.

Left side of chest - Moist crepitations at base.

Pulse very weak and irregular.

Both arms and hands are very tremulous.

Hyperaesthesia has returned - general.

The right pupil is dilated. The left eye shows slight conjunctivitis and ptosis.

Head retraction is marked.

March 8th. Died, 8.15 a.m.

Post Mortem.

The post mortem rigidity general; the post mortem lividity slight.

Thorax. There is no fluid in either pleural sacs, but the left one shows slight adhesions above.

The right lung. The bronchial glands not enlarged - copious mucopurulent exudate. The lower lobe shows bronchitis in medium and larger bronchi - no distinct pneumonia. The upper lobe is paler and drier. Very distinct emphysema in lung. The middle lobe is pale and dry.
The left lung. Larger bronchi similar in lower lobe: also well marked emphysema. Section similar, but not so congested as right lung. The upper lobe shows same characters but not so marked.

The heart. No fluid in pericardium; no pericarditis. No petechial but some hyperaemic areas on surface, though no evidence of pyaemia. Mixed stringy thrombus in right and left ventricles. The colour is pinkish and not so markedly toxic in appearance. Some mottling from fatty change and cloudy swelling. Stringy thrombus up into pulmonary artery. Aortic valves normal.

The abdomen. The Liver. Surface smooth; no evidence of pyaemia; bile is normal.

The spleen. Not enlarged; consistence firm; colour - pinkish red; malpighian bodies not very prominent.

Suprarenal Glands. Left suprarenal gland shows nothing to note.

The left kidney. Capsule strips readily: stellate veins engorged. Towards lower ends are small pale infarcts, septic, studded with pyaemic foci.

The kidney otherwise shows cloudy swelling, with probably some fatty change.
The right kidney shows similar changes - cloudy swelling, but no infarcts.

The intestines. In upper part of small intestinal mucosa hyperaemia is distinct, but passes off as you go lower down. Swelling of lymphoid tissue is more than in James Aitken's case, No.10.

The mesenteric glands. Distinctly enlarged: no hyperaemia about them at all.

The Brain. Marked hyperaemia of convolutions with some flattening.

Extensive yellow gelatine-purulent lymph over base of brain, especially from optic chiasma back to pons. Also much lymph in posterior arachnoid cistern.

Elsewhere in brain very little lymph: slight amount along Sylvian fissures; over sides of vertex only turbid fluid in meshes of pia arachnoid.
CASE IX.

I.P., aged 16 years.

History.

Patient was at work on 25th. She went home in the evening and complained of a headache and sore throat, and went to bed. During the night she became very restless and by morning was quite delirious. She vomited three times over night, and complained of backache which was severe.

During the day she became much worse, but no head retraction was noticed.

Her three brothers were up all night keeping her in bed owing to the great restlessness.

She was admitted on 27th.

Patient lived with two other adults in a two roomed, badly kept, dirty house, in a poor quarter of the town.

She worked in a Ropery with Case 4; apparently did not get proper food and allowed herself to get into a very dirty and neglected state.

On admission:

- Development and nutrition very good.
- General condition filthy and verminous.
- Is absolutely unconscious, cannot be roused and resembles an animal more than a human. She is very
restless throwing herself about continually. Her arms and legs are always on the move, the movements being purposeless, and regardless of pain, for she will whack her arms against the bedposts quite oblivious to bruising. She has to be kept in bed by a restraining sheet and other devices, but in spite of these once threw herself right out on to the floor. She lies almost always on the left side with legs drawn up.

The right arm is constantly twitching.

There is no head retraction and no stiffness of the muscles of the neck.

The skin - no rash and no herpes.

The skin feels cold, especially the extremities; the cheeks have a bluish cyanosed appearance - very marked.

There is no tach. She has a marked soapy smell.

Eyes - Pupils are equal, moderate dilatation, and react to light.

Pulse 120° Temperature 102.2. Respiration 24.

Nervous System.

Motor power is quite unimpaired.

Sensation - Undetermined. There is no hyperesthesia apparent.

Special senses - nothing notable.
Reflexes undetermined.
Kernig's sign very slightly marked.

**Alimentary.**
Tongue covered with dirty brown fur and the mouth generally in a foul condition.
Patient refuses food, takes it in her mouth but immediately spits it out again.
Abdomen normal.
Liver, stomach and spleen normal.
Constipated.

**Respiratory.**
Throat - marked congestion.
Lungs - nothing of note.

**Circulatory.**
Pulse full and strong but rapid.
Over all the precordia a systolic murmur is to be heard, most marked in the tricuspid area: it is rough and high pitched.

**Urinary.**
Urine passed involuntarily.
Catheter specimen. It is brownish yellow and cloudy, with an incipid alkaline odour.
Reaction acid. Specific gravity 1035.
Albumin present - 4.8 grains per oz. No sugar.
Deposit of urates.
Lumbar Puncture.

8 c.c. fluid collected. Pressure high (unestimated) at first spurting out but very soon only came in slow drops.

Fluid turbid - yellowish green colour.

Rapid sedimentation of pus.

Film showed Polymorphs. 98% Lymphocytes 2%

Meningococci present - very few, and all were intracellular. They had to be carefully hunted for.

Chemical examination - Albumin 6 grams per litre.

After lumbar puncture m. xx. of lysol diluted to \( \frac{1}{3} \) were injected intraspinally.

Blood.

<table>
<thead>
<tr>
<th>R.B.C.</th>
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<tr>
<td>Hb.</td>
<td>120 +</td>
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</table>

No differential count made.

February 28th. Since admission the head has become markedly retracted and the neck become rigid.

March 1st. Patient is more sensible and is much quieter. She answers fairly rationally when spoken to but cannot concentrate her attention. She is more eager for her feeds but appears to have some difficulty in swallowing. The head is still markedly retracted and that seems to have something to do with this difficulty.
March 3rd. The difficulty in swallowing has increased. The food is regurgitated through the nose. She wants the food and asks for it. The head retraction is more marked. Patient has difficulty in speaking; can only whisper to-day. She complains of feeling short of breath.

When left alone she still mutters nonsense, but is quite sensible when spoken to. Is shivering frequently: cannot keep her warm in spite of blankets and hot bottles.

Was more restless over night again, but not severely so.

Urine is still passed involuntarily, but the bowels are very constipated.

Patient has diplopia: no strabismus.

March 5th. Swallowing has become impossible, whole feed now being regurgitated. Have had to resort to gavage.

Patient cannot speak at all now, owing to lack of phonation. Could whisper yesterday nasally. Head retraction is extreme, with some opisthotonos.

The urine has had to be drawn off by catheter. Acid reaction, specific gravity 1030. Albumin 10 grs. per oz.

Has had bad colour all day and is still very cold. Pulse seems to be failing.
Has been coughing slightly over night.
Few rales both bases, but nothing else of note.
Kernig's sign is now well marked.
Patient died at midnight March 5th.

Post Mortem, 36 hours after death.

Nutrition of body poor. Rigor mortis general.
P.M. rigidity marked. Well marked mottling on left arm.

Chest. Left pleural cavity shows subacute and chronic adhesions. No free fluid. Right pleural cavity practically obliterated.


On section: Upper lobe pale, congested and oedematous. No distinct pneumonic consolidation. Lower lobe also congested and minute areas of broncho pneumonia present. There are also haemorrhages in lower lobe.

Right Lung. Chronic pleurisy. Slightly enlarged pigmented caseous glands at the root. Bronchi as other side.

On section: Upper lobe congested and oedematous, capillary bronchitis. Lower lobe, acute broncho-pneumonia (early). Anterior part of the upper and
lower lobe shows more confluent conditions of broncho-
pneumonia.

Heart. Pericardial sac contains $\frac{1}{2}$ oz. clear fluid. No pericarditis.

Right side slightly distended. Myocardium pale in colour with dark green looking areas. Stringy antemortem thrombus in right ventricle. No pulmonary or tricuspid endocarditis. Mixed thrombus in right auricle. No mitral or aortic endocarditis. Cloudy swelling and fatty change, possibly acute interstitial myocarditis and dilatation.

Abdomen. No peritonitis. Some superficial congestion of the loops of the small intestine.

Mesenteric glands show recent enlargement and are hyperaemic.


Liver. The gall bladder contains "dark greenish black" bile. Surface smooth.

On section: Fatty change at the periphery of the lobules and cloudy swelling.

Spleen. Not enlarged; dark in colour and soft. Malphigian bodies unduly prominent.

Acute congestion is present.
Right kidney somewhat soft and flabby. Capsule strips readily: no chronic disease.

On section: Cloudy swelling; fatty change.

Left kidney - same as right.

Brain. At the base small amount of thick gelatinous lymph, most marked over upper part of pons but forward also to the optic chiasma. Convolutions slightly flattened. Vessels unduly full at sides and vertex.

Slight excess of turbid fluid in the meshes of the pia arachnoid, but no fluid at vertex.

Fluid from the spinal canal pretty clear.

No evidence of spread of disease from region of the cribriform plate or ethmoidal cells.

Cord. Some turbid fluid escaped on cutting the dura. No exudate upon the cord, but some distinct adhesions.
CASE X.

J.A., aged 4 years.

History.

On 28th February the parents thought he had got a chill. He was cold and shivering; later became very feverish. Was put to bed and slept well that night.

Next day (March 1st) he vomited and complained of his head. Had three convulsions and has remained unconscious since the third one.

Was admitted on 2nd inst. there was no head retraction, no stiffness of the neck, restlessness or any marked sign of cerebrospinal meningitis.

Only previous illness was pneumonia at Xmas.

Patient lived in a clean two-roomed house, with its two parents, being the only child. The father was a respectable dock labourer.

The mother had helped five days before the onset with nursing a baby who had died suddenly with symptoms which pointed strongly to cerebrospinal meningitis.

On admission:

Development and nutrition fairly good.
General condition - filthy dirty.

Is quite unconscious.
Lies on his right side with knees drawn up and will not be moved off it; if turned on to left side he will be back again on the right in a few minutes. Is intensely restless, arms and legs being thrown about incessantly. Is constantly crying out and frequently is shrieking at top of his voice.

Head retraction is most marked. the occiput touching the back, and the neck is quite rigid.

Skin - No rash: no herpes.
The cheeks have the purplish pink flush. The skin is cold - extremities most.
There is no tache.
There is no smell.
Temperature 98°. Pulse 120. Respiration 48.

Nervous System.
Motor power unimpaired, but cannot sit up.
Reflexes - None obtained.
Kernig's sign absent.
Sensation - Impossible to test.

Circulatory.
Pulse is not very strong. Easily compressible.
Sounds are closed in all areas.

Respiratory.
Breathing is rapid, but resonance is good throughout and breath sounds are normal with no accompaniments.
Throat is congested.

Alimentary.

Tongue is covered with brown fur and mouth full of sticky brown mucus.

Is taking his feeds well.

No discomfort in abdomen, but is constipated.

Area of liver dullness from 7th rib to costal margin.

Stomach normal.

No enlargement of spleen.

Urine.

Acid. Specific gravity 1032.

Albumin present - 5.6 grains per oz. Deposit of urates.

Blood.

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<tr>
<td>Hb.</td>
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Differential leucocyte count:

- Polymorphs 87%
- Lymphocytes 12%
- Transitional 1%

Lumbar Puncture.

Fluid ran out under high pressure: 17 c.c. collected.

Fluid turbid - rapid sedimentation of pus.

Reaction faintly alkaline.

Albumin - 4 grams per litre.
Film - Polymorphs 65%
Lymphocytes 6%
Degen. 29%

Meningococci present in large numbers, both extra- and intra-cellular.

Lysol m.x. diluted to 3T were injected intra-spinally.

March 4th. Patient still very restless and crying out frequently.
Pulse has become irregular and weaker.
Abdomen is distended, hard, and a little tender.
The bladder is being emptied. Is very constipated.
Hands and legs have both been twitching.
Is a little conscious at times.

March 5th. Slight conjunctivitis in right eye.
Twitching of arms has become almost continuous.

March 6th. Both eyes - conjunctivitis.
Quite unconscious again, and is much worse.
Appears to be some difficulty in swallowing, but there is no regurgitation.

March 7th. Patient coughing a good deal.
Chest - Right side; impaired note throughout.
Auscultation - Breath sounds on right side faint and are accompanied by coarse rales.
Left side - Breath sounds are harsh: no accompaniments.
Lumbar puncture.

Only a small clot of pus was obtained. Film shows only some disintegrated leucocytes. No meningococci are to be found.

Patient died shortly after midnight.

Post Mortem.

External Examination:

The post mortem rigidity was general, the post mortem lividity marked, and the body emaciated.

Internal Examination:

Thorax:

The pleural sacs show no adhesions, nor do they contain any fluid.

The bronchial glands are not enlarged, but only slightly congested with some muco-pus in the larger ones.

The right lung - The bronchi are slightly congested. The lower lobe is very distinctly collapsed. The upper lobe shows some lobular collapse posteriorly: few petechiae.

On section: The lower lobe is collapsed and shows signs of early bronchopneumonia. The upper lobe is congested and oedematous and shows early capillary bronchitis. The middle lobe shows early bronchopneumonia towards its root.
The left lung - The upper lobe shows patches of emphysema and lobular collapse.

On section: It is congested and oedematous: no distinct pneumonia. The lower lobe shows few petechial on its outer aspect.

On section: It is congested and oedematous. Mucopus in the smaller bronchi. Some areas of slightly more solid consistence that suggest early bronchopneumonia are present.

The heart - There is no pericarditis; the pericardial sac contains 3/4 of fluid. The left ventricle is in a state of rigor and shows cloudy swelling. There is a stringy antemortem thrombus in the left ventricle. The right ventricle contains a stringy antemortem thrombus. The pulmonary cusps show nothing abnormal. The aortic cusps show no endocarditis. The mitral cusps show antemortem thrombus entangled at margin, but no endocarditis.

The Abdomen:

The Liver on surface shows patches - probably anaemic. On section shows cloudy swelling with distinct fatty change.

The Gall Bladder contains dark orange, almost black, bile.

The Spleen is slightly enlarged and flabby; shows post mortem discoloration. The malpighian
bodies are not very prominent. On section shows acute congestion.

Suprarenal Glands show no haemorrhages.

The Right Kidney shows cloudy swelling and fatty change.

The Left Kidney shows practically the same changes.

The Intestines. - The small intestines are slightly granular. No special hypertrophy of lymphoid tissue: only slightly more prominent in the lower part.

The upper part of large intestines shows some distinct hyperaemia: small haemorrhages below mucosa. No ulcers.

The Mesenteric Glands show signs of recent enlargement, \( \frac{1}{2} \) in size; many are hyperaemic; old caseous and tubercular glands.

The Spinal Cord

shows diffuse lepto-meningitis with thick yellow gelatinous lymph in subdural space.

The Brain

shows marked purulent effusion with gelatinous lymph over pons, medulla, and in front of optic chiasma. Purulent lymph in posterior arachnoid cistern of greenish tint.
Small patches of lymph under inferior aspect of frontal and temporal lobes.

Leptomeningitis extending up over both sides of the brain: exudate most marked in sulci on both sides, especially along sylvian fissures and in some parts the convolutions are covered.

On vertex the exudate most marked over anterior part, and as on sides, the purulent lymph is not only in the sulci but also covers the convolutions. The surface not only hyperaemic, but the sulci are distinctly full.
CASE XI.

G.F., aged 7\(\frac{1}{2}\) years.

History.

On 8th patient came back from school and did not feel well; that night he was shivering badly and was thought to have caught a cold.

The next day he felt all right again and went out as usual.

On 10th he could not sleep at night and was very restless: complained of his head being sore.

On 11th he vomited three times and did not feel at all well, and the headache persisted.

On 12th his legs began to feel sore.

On Wednesday, 13th, his headache was worse and as he was making no improvement he was taken next day to the Sick Children's Hospital, Edinburgh, where he was admitted.

There was no head retraction, but the restlessness which began on the night of the 10th steadily increased. Since 11th, he frequently vomited.

There was no constipation.

Previous Illnesses: Measles, German Measles, Chicken Pox: was cut for an abscess on leg 18 months ago. He has had a cough for some weeks past.

Patient was a bright boy and quick at his work.
and eager to get on. He was at the same school as Case II. and Case VIII.

The father is a brassfinisher, working in same firm as the fathers of Cases II. and VIII. and going to the docks for his work.

The house is one of four apartments, for two adults and three children. The whole five sleep in one room and the house is not very clean.

On admission.

Development good: nutrition fair.

Is quite conscious and intelligent: answers questions at once and quite rationally.

No restlessness.

Head retraction is slight: neck absolutely rigid.

No tenderness at neck.

Skin - No rash: no herpes.

No flushing of cheeks.

Tache is well marked.

No coldness.

No smell.


Nervous System.

Motor power unimpaired.

Sensory. Slight general hyperaesthesia.

Reflexes - Abdominal and epigastric active.

Plantar - Babinski present.
No jaw jerk. No tendon reflex obtained except the right knee jerk.

Kernig's Sign slightly present.

Sight, hearing, taste and smell - unaffected.

Alimentary.

Tongue covered with dirty brown fur.

Nothing of note in abdomen.

Is constipated, but previously was not so.

Respiratory.

Throat congested and tonsils are covered with a yellowish thin film.

Has a slight cough.

Nothing of note on examination of the chest.

Circulatory.

Pulse is fairly strong.

Cardiac sounds closed all areas.

Urine.

Is passed frequently in small quantities. Acid 1028. No abnormal constituents.

Blood.

\[
\begin{align*}
\text{R.B.C.} & \quad 5,160,000 \\
\text{W.B.C.} & \quad 38,750 \\
\text{Hb.} & \quad 120\%
\end{align*}
\]

Differential leucocyte count:

- Polymorphs: 95.5%
- Lymphocytes: 2.1%
- Large Mononuclear: .5%
- Transitional: 1.5%
Lumbar Puncture.

Fluid flowed in rapid drops: 26 c.c. collected. Fluid - slightly turbid. No deposit of pus, but formation of a coagulum.

Leucocytes estimated = 31,250 per c.mm.

Film -
Polymorphs 83%
Lymphocytes 3%
La. Mononucl. 14%

Only two meningococci - both intracellular - were found after searching three films.

Albumin - 1 gram per litre.

Lysol m.xx. diluted to 3⅓ injected intraspinal-

March 17th. B.W. & Co.'s automeningococcal serum 25 c.cm. injected subcutaneously.

March 18th. Patient is getting restless, throwing his arms about.

Abdomen is hard and tense: legs drawn up.

Is not so well. Complains of headache being severe, the pain being on the top of the head.

Faint raised and pointed papular rash seen about seat of injection of serum.

March 19th. Rash general over whole abdomen - bright, very bright. On legs and arms - faint arid papules more discrete.

On evening of 19th face was well out in very bright scarlet confluent papules.
March 20th. Patient much worse. Has become very weak.

Has incontinence of urine and faeces.

Rash more marked. Abdomen covered with bright red eremathematos rash resembling a blotchy scarlet. Face only flushed.

Legs and arms have many more papules, and more pronounced.

Abdominal and epigastric reflexes still well marked.

March 29th. The rash continued up till 27th, passing through various stages, disappearing to re-appear in a different form. Papules gave place to urticarial blotches, and then later a diffuse eremathema appeared over whole of back and chest.

April 2nd. Patient has kept much the same. Is getting very much emaciated.

Headaches persist.

He occasionally complains of pains in the legs and muscles of the calf and thigh. Both legs are very tender.

The rigidity of the neck persists, but there has been no tenderness.

Abdominal and epigastric reflexes persist active.

Kernig's sign is well marked now.

Knee jerks are both active.

April 22nd. Patient has remained in a stationary condition except for emaciation, which is
steadily getting worse.

He has been vomiting occasionally and refuses to take food so that he has to be nasal fed. He is very obstinate about the feeds if given ordinarily and will put his fingers down his throat to make himself sick.

Chest is still clear.

Has been put on hot bath treatment.

May 6th. Since baths begun he has improved immensely. He likes them and kicks about in them. In this way he gets exercise and is beginning to be able to move better and is not so helpless. His appetite is returning.

The rigidity of the neck is disappearing.

May 20th. Patient very much improved. He is taking an interest in everything that is going on and his intelligence is returning.

Has begun to put on flesh.

Memory for recent events is defective, but is good for events previous to illness.

Legs are still tender and Kering's sign still present.

June 5th. Patient up and trying to walk. Is a good deal of spasticity and walks on tip toes.

July 23rd. During past month patient has continued with steady improvement.
The abdomen has, however, become enlarged and though there is very little to go on I think there is early tabes mesenterica. His general health, however, admits his discharge to-day.

Patient seen one month after leaving Hospital. Has been steadily improving. Has been attending Leith General Hospital on account of the distended abdomen. Early tuberculous trouble is suspected at the Hospital.

No headaches or sickness: perfectly strong and able to run about. There are no signs of any emaciation and except for the abdomen is in excellent health.

Sight and hearing are good.
M.A., aged 16 years.

History.

On 15th, while at work, patient suddenly got giddy and had a severe headache, which was frontal. She had to leave work and go home. When she got home she vomited.

She had been in perfect health up to then: had had no sore throat, no cold, or any other cause to make her run down.

On 16th she did not get up and the headache was more severe and pain was now felt in the back of the head but there was no more sickness. She spat up some blood on this day. There was slight delirium.

On 17th she had pain in the back and the headache had become much more severe.

Both on 17th and 18th there was again some streaks of blood in the sputum.

The headache has become intolerable, worse at occiput and neck, but is also present in frontal region as well.

Has not been able to take any food; the bowels have only moved once since Friday.

Previous Illnesses. She frequently gets headaches. Six weeks ago had some indigestion due, she
was told, to bloodlessness.

The patient works in a good Biscuit Factory where working conditions seem to be quite satisfactory. At home she lives in an overcrowded house, there being four adults and five children in two rooms. These, however, are kept clean.

Her father - an engineer - has been in contact at work with fathers of Cases II., VIII. and XI., the common source of infection appearing to be the ship on which they were working.

Case X. was from the same flat in the same tenement and when he was at home, before removal, she was frequently in to see him.

On admission:


Is perfectly conscious and rational and gave full account of herself.

Lies on left side, as quiet as possible, any movement causing her headache to be more severe. There is therefore no restlessness.

Head retraction is well marked and the neck is absolutely rigid; the muscles of the neck are held tense and are tender.

The skin - No rash: no herpes: no flush on cheeks.

She is quite warm - extremities as well as body.
Tachy is well marked.
There is no smell.

Nervous System.
Motor power weak, but is present generally except cannot sit up or stand.
Sensation to touch - pain, heat and cold unimpaired: no hyperaesthesia.
Special senses unimpaired.
Pupils equal and moderately contracted.
Reflexes - Epigastric absent: abdominal and plantar well marked: no tendon reflexes elicited except knee jerks which are moderate.

Respiratory.
Throat congested and covered with a yellowish exudation.
Nothing of note in chest.

Circulatory.
Pulse full and strong.
Apex in L.M.L. 5th space.
Sounds closed in all areas.

Alimentary.
Tongue very red, dry and cracked, and the papil-

lae are prominent.
Abdomen - Nothing noteworthy.
Stomach, Liver and Spleen normal.
Urine.
Acid. 1022. No abnormal constituents.

Blood.

R.B.C. 6,550,000
W.B.C. 16,250
Hb. 110%

Differential Leucocyte count:

- Polymorphs. 82%
- Lymphocytes 10.5%
- Large Mononucl. 4.0%
- Eosinophiles 1.5%
- Transitional 2.6%

Lumbar Puncture.

Three c.c. collected. Fluid flowed in very slow drops under no tension.

Estimation of Wh.B.C. = 9,370 per c.mm.

- Polymorphs. 81%
- Lymphocytes 10%
- La. Mononucl. 2%
- Degenerates 7%

Only two typical meningococci were seen, both extracellular. One tetrad made up of two diplococci apparently placed side by side also seen.

March 20th. Headache still severe; crying out frequently with it.

There is a faint C.S. smell now, and slight cyanotic colour on both cheeks.

Very slight herpes on the upper lip.

Abdominal reflex faint.
March 21st. Abdominal reflex cannot be elicited. There is slight internal strabismus of right eye and diplopia for distant objects.

Complains of pains in both legs.

March 25th. Patient has some pain in both ears and she is getting deaf. Nothing of note in examination of ears.

March 26th. Deafness is increasing, but the pain is getting less.

The throat is sore; appears congested with much yellowish mucus looking material on tonsils. Cannot swallow properly: there is no pain, but she cannot get the food down.

March 29th. Patient complains of pain in the back and in the calf of both legs, and they are both tender to the touch.

Tongue is very sore: has several pusy looking sores on it.

Abdomen is tender. There is slight diarrhoea; motions green, with blood stained mucus.

Left eye - pronounced blepharitis.

April 3rd. Slight conjunctivitis in left eye. The headaches have persisted but are not so severe; the rigidity of the neck is distinctly less.

Abdominal reflex is present again now.

The diarrhoea has stopped and motions are normal.
April 10th. Patient is decidedly much better and is steadily improving in spite of occasional vomiting and recurrence of headache.

Lumbar puncture. 15 c.c. collected. Fluid ran under slight pressure. It was clear and there was no sedimentation of pus.

White B.C. = 3,750 per c.mm.
Polymorphs. 79%
Lymphocytes 21%

Albumin 5.5 grams per litre.

April 11th. Patient is not so well: headache has been more severe and she has been sick twice. Complains of pain in her back.

April 22nd. Patient has been worse ever since the second lumbar puncture was done. Is peevish and takes food badly. She is getting emaciated and headaches are almost as constant as during the first two weeks.

April 25th. Put on hot bath treatment. She likes the baths and feels much better after them and they relieve her headache.

May 2nd. Patient has improved steadily since hot baths begun.

May 12th. Able to get up to-day for first time.

May 28th. Dismissed cured. She is a little weak, but otherwise healthy.
Seen two months after discharge.

She is looking perfectly well, and is well nourished. Is at work as a housemaid and walked over to see me about $3\frac{1}{2}$ miles.

Has had no return of headaches or sickness since discharge, or of vomiting.

She eats well but is still constipated. She gets a little out of breath at times on stair climbing. She is very nervous; the least thing startles her, which was not so previous to illness.

Sight and hearing good.
CASE XIII.

L.W., aged 14 years.

History.

On 18th patient complained of headache and pain down the back, and felt sore all over and her legs were stiff. She shivered most of the evening and vomited. She went to bed, but had a bad night, the headache getting worse. The next day she was much worse and vomited again; the headache was becoming unbearable.

On the 20th she was admitted to Leith Hospital, where cerebrospinal fever was diagnosed, and so she was sent on to Leith Public Health Hospital on 21st.

When admitted to Leith Hospital she could hear quite well, but when we got her (next day) she was stone deaf.

The father is a trawl-engineer. The house is one of seven rooms, in which five adults and five children live. It is kept in a moderately clean condition.

No connection with any previous case or common source can be traced.

Previous illnesses - None.

On admission:

Patient well developed, but only moderately nourished.
Lies on right side, with mouth and eyes half open, and prefers to keep as quiet as possible. She is quite conscious and apparently intelligent but as she is stone deaf one cannot get much information out of her. There is no restlessness. Head is markedly retracted, the neck rigid and tender.

Skin - No rash. There is slight herpes on chin. Cyanotic flush on the cheeks.

She does not complain of cold, but always is so. Tache is present but sluggish in appearing. There is slight C.S. smell.


Nervous System.

Motor power unimpaired.

Sensation - Marked hyperaesthesia.

Reflexes - Superficial, abdominal and epigastric very active. Plantar - active with flexion.

Tendon - No jaw jerk; knee jerks increased; ankle clonus slight. No other present.

Pupils react to light. They are equal and moderate. No nystagmus or strabismus.

Circulatory.

Pulse full and strong.

Sounds closed all areas.
Respiratory.

Throat congested and on left side is yellowish thick exudation, spreading on to the uvula.

Nose is in a puffy mess and herpes on alae nasi outside and in.

The chest - Nothing of note.

Alimentary.

Tongue covered with whitish fur.

Abdomen presents nothing of note.

Stomach, liver and spleen are normal.

Urine.

Acid. 1018. Deposit of urates. No abnormal constituents.

Blood.

\[
\begin{align*}
R.B.C. & \quad 5,950,000 \\
W.B.C. & \quad 37,500 \\
Hb. & \quad 98%
\end{align*}
\]

Lumbar Puncture.

Thirty-three c.c. collected: ran out under moderately high pressure.

Fluid turbid - not thickly purulent, like the previous ones: only slight sedimentation of pus which was present only in the first half of the fluid, but there was no coagulum: in the second half there was a coagulum but no sedimentation.

\[\text{Wh.B.C.} = 9300 \text{ per c.mm.}\]
Albumin in both halves of the fluid was same, i.e. 1 gram per litre. No reducing substance.

Film shows -

Polymorphs. 88%
Lymphocytes 5%
Large Mononucl. 7%

Only very few meningococci were found.

March 28th. Patient making no progress. Headache is almost constant and at times very severe. She has no appetite and only takes her feeds after a great deal of persuasion. She vomits frequently.

Constipation is very troublesome.

The urine has to be drawn off by catheter for the last three days.

March 30th. Has a slight cough and is spitting a little - white frothy mucus. Complains of pain in the chest. Nothing of note on examination.

April 2nd. Passing her urine quite well again.

April 11th. Lumbar Puncture - 13 c.c. obtained. Fluid faintly turbid.

Wh.B.C. 3750 per c.mm.
Polymorphs. 81%
Lymphocytes 19%

Albumin - 3 grams per litre.

April 18th. Still vomiting frequently and can only with difficulty be persuaded to take any food at all. She is getting very markedly emaciated.

April 25th. Has had to be put on nasal feeds. Put on hot bath treatment.
April 30th. No improvement since bath treatment begun. Is steadily getting worse. Colour gets bad at times - becomes cyanosed.

The headaches are becoming more severe again, and the vomiting continues in spite of all remedies that have been tried.

Emaciation is now extreme.

The patient is becoming demoralised and filthy in her habits.

May 11th. Baths have been stopped as there is no improvement with them and they disturb the patient too much.

To-day she vomited a small quantity of bright red blood.

She has been hiccupping frequently during the last day or two.

May 12th. Vomited about 12 oz. of thick coffee-coloured fluid. Pulse is failing and patient very pale.

May 15th. Vomited some green fluid with some blood clots in it.

May 18th. Patient died, after gradual cessation of heart's action. Pulse was unable to be felt at 2 a.m. and heart sounds weaker as morning drew on.

No Post Mortem obtained.
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Clinical Course:

- J.A.P.
- Silver-lode.
- Blood and urine.

Result: Death.
CASE XIV.

E.C., aged 5 years. Admitted 26th March.

History.

Was a history of a fall on 24th, when head was struck. Yesterday afternoon (25th) while patient was playing he was sick, and he then complained of his head being sore. He went into the house and vomited again, and shortly complained of headache being severe.

In the evening the parents noticed he was unconscious and that his head was held back. This morning a doctor was sent for who sent him at once to Pilton.

Patient is living in a common lodging house of low order and of doubtful cleanliness.

The father is a vagrant.

On admission:


Is quite unconscious.

Restlessness is very marked, patient tossing about incessantly and once rolled himself out of bed.

Skin - There is a faint purpuric eruption over the chest and arms. There is no herpes.

Both cheeks are a purplish cyanosed colour.
He is cold and resents the clothes being removed at once.

The right arm is much colder than the left, and the pulse in the right arm is feebler than that in the left.

Head retraction is very marked, but the head can be moved slightly, the characteristic board like rigidity being absent.

Tache cerebrale well marked.

There is no characteristic smell.


Nervous System.

No reflexes were obtained owing to the impossibility of eliciting them in patient's restless condition.

Similarly sensation was impossible to determine, but patient appeared to be generally hyperaesthetic as he resented being touched.

Kernig's sign was present.

Pupils equal and contracted.

Lumbar Puncture.

Only 4 c.c. fluid obtained - turbid, containing polymorphonuclear leucocytes, a few lymphocytes and very few intracellular meningococci. No extracellular.
Circulatory.

Sounds closed all areas.
Pulse full and regular. 140 per min.

Respiratory.

No cough. Respiration 40.

Small area of impaired resonance at left apex in front and under right axilla. In these areas the breath sounds are bronchial and high pitched. No accompaniments.

Urinary.

No urine obtained.

Blood.

Wh.B.C. 18,000 per c.m.

Patient died same night.

No Post Mortem obtained.
March

Date

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Time

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Pulse, Respiration, Tone, etc.
CASE XV.

M.A.K., aged 3 years. Admitted March 31, 1907.

History.

Patient to-day complained of severe headache and vomiting: was quite well yesterday. No more history than that is obtainable.

Patient came from same lodging house as No.XIV. The room was overcrowded, there being three adults and two children in one room, which was not clean. The parents are dissolute and drunken. They were rag pickers.

On admission:

Development and nutrition both good.

In unconscious. Lies on left side with eyes and mouth half open, looking very ill, with a greyish cyanosed colour and purplish flush on the cheeks.

Restlessness is marked, tossing from side to side and waving her arms about.

Has the characteristic "soapy" smell.

Skin - Child is filthy and flea bitten all over, but no purpuric rash or herpes.

There is no head retraction, but the neck is quite stiff and painful when any attempt is made to move it.

She is very cold: hands and feet are blue with cold.
Tache cerebrale is well marked.
Temperature 102.6°. Pulse 148. Respiration 44.

**Nervous System.**
Impossible to determine any facts except the presence of a well marked Kering's sign. Patient is too restless to allow any reflexes being elicited.

**Alimentary.**
Tongue is dirty brown.
Abdomen shows nothing of note, and organs normal.

**Circulatory.**
Heart sounds closed in all areas.
Pulse full bounding and regular.

**Respiratory.**
Left side posteriorly there is harsh breathing with fine rales. Resonance unimpaired.

**Urine.**
No urine obtained.

**Lumbar Puncture.**
Seven c.c. collected. Turbid fluid, flowing in moderately rapid drops.

Film shows - Polymorphs 97%
Lymphocytes 3%

Meningococci - a few extracellular.
Albumin - 6 grammes per litre.

**April 1st.** Patient has vomited three times over night - green fluid.
201.

Was very restless. Morphia, gr. 1/8th, given, after which she got a little rest.

April 2nd. Restlessness continued unabated.

Child got more cyanosed and colder.

Vomiting continued and increased.

Pulse gradually failed, and child died 8 a.m. to-day.

Post Mortem. April 3rd. 4.30 p.m.

Development good. Rigor mortis general. Post mortem lividity marked on dependent parts of the body.

Thorax.

Old adhesions general in left pleural cavity.

Left Lung: Bronchial glands not enlarged.

Bronchi - slight bronchitis. On section - no consolidation: some congestion.

Right Lung: Caseous T.B. bronchial glands.

Bronchi - as in left. Pleura - No adhesions, occasional haemorrhagic points. In lower part of upper lobe, a nodule the size of a marble was seen projecting above the surface: consistence firm. On section upper lobe crepitant and shows acute congestion: lower part of upper lobe nodule is seen of a mottled grey appearance, firm in consistence, and extension from the pleura to the root of the lung. The direct infection can be traced from tuberculous lymphatic
glands into the nodule.

**Heart.** Thickened epicardium; petechial haemorrhages. In subserous tissue are minute harmorrhages. A.M. thrombus right auricle; tricuspid orifice is dilated; pulmonary competent. Myocardium of the left ventricle is pale, somewhat mottled, soft and friable: shows distinct cloudy swelling or possibly diffuse fatty change.

**Abdomen.** Liver - Intense cloudy swelling. Spleen - It is enlarged, shows a nodule size of a small pea: chronic tuberculosis.


**Stomach and intestines.** Healthy. Mesenteric gland distinctly swollen.

**Pancreas and pelvic contents - normal.**

**Head.** Dura mater showed increased vascularity: more than usually adherent to adjacent vertebral canal.

Subdural space shows slight increase of fluid, which is turbid.

In subarachnoid space, over the posterior aspect of the cord mainly, purulent greenish grey gelatinous exudate is present and reaches from lower part of cervical to lower part of lumbar enlargement. This
obscures structure of the cord which beyond exudation shows marked injection.

Underneath the arachnoid a purulent exudate, greenish grey colour, is seen. Following outer and surrounding vessels, the veins are much engorged. This is present over whole of the vertex, most abundant on left side sylvian fissure.

Base: Anterior and posterior subarachnoid cysterns are much distended with a thin watery, slightly brownish coloured purulent fluid. Below this the characteristic thick tenacious exudate is seen which extends over cerebellum, pons and medulla.

Ethmoidal and sphenoid - All sinuses contained muco purulent contents.
CASE XVI.

A.C., aged 9 years.

History.

For six weeks before the onset the patient had not been well.

The onset was sudden, on March 19th, when she complained of her head and pain in the neck. She got very cold and shivered a good deal on 19th. She vomited that night.

On 22nd she complained of her back being sore. She retained consciousness perfectly until March 25th, when she began to wander a little.

She was making no improvement, and vomiting and headache continued, so that yesterday a doctor was called in and diagnosed cerebrospinal fever and sent her on to us to-day.

Previous Illnesses - Diphtheria three years ago.

The patient was in same class at school as a case who died at home of cerebrospinal meningitis. The father worked in a miller's sack store. The house was roomy and clean, there being four rooms for three adults and three children.

On admission:

Development good, but nutrition is poor.

Patient lies in a semiconscious state, and answers
to questions if roused, as to name and headache, etc.

There is no restlessness: she is very listless and appears to be very weak. Lies on left side with legs drawn up.

There is no head retraction, but there is absolute rigidity of the neck, which is painful is any attempt is made to move it. If left alone though, there does not appear to be much pain or headache.

Skin - No rash: no herpes: the nose is sore, but not as if there had been any herpes on it. The skin generally is very dry and is covered with a branny desquamation.

Cheeks have the cyanotic flush faintly.

There is very slight cerebrospinal smell.

Is no tache.

Hands, feet, and face very cold.


Nervous System.

Motor power is generally impaired through general weakness.

Sensory - General hyperaesthesia.

Reflexes - Epigastric and abdominal both absent.

Plantar - Babinski. Knee jerks are both present and are the only tendon reflexes elicited.

Kernig's sign present.
Alimentary.
  Tongue is clean.
  Abdomen - Complains of general tenderness when it is palpated.
  Nothing else of note.
  Has been constipated.

Respiratory.
  Throat is clean.
  Nothing of note on examination of chest.

Circulatory.
  Pulse rapid and feeble.
  Cardiac sounds closed in all areas.

Urine.
  Acid. 1028. No abnormal constituents.

Blood.
  R.B.C. 4,900,000 per c.mm.
  W.B.C. 34,625 " "
  Hb. 110%

  Differential leucocyte count:
  Polymorphs. 70%
  Lymphocytes 25.5%
  Large Mononu. 3.0%
  Transitional 1.0%
  Basophile .5%

Lumbar Puncture.
  Fifteen c.c. obtained. Fluid ran out under high pressure.
  W.B.C. 12,500 per c.mm.
  Polymorphs. 92%
  Lymphocytes 8%
The fluid was practically clear in comparison with the previous cases, there being just the "dust-in-the-sunlight" appearance when looking through the test tube.

Meningococci were present in very small numbers, intracellular only.

Albumin - 1 gramme per litre.

April 4th. Patient is not improving. Head retraction is now present and is increasing.

Patient has become very restless, is tossing about and crying out continually. The headache is now very severe.

Consciousness is entirely lost.

Pulse is getting very feeble.

Cannot get her to take any feeds at all.

April 5th. Patient much worse.

Has to be nasal fed entirely as she will not or cannot swallow anything by mouth.

The arms are peculiar. They are held fully extended at the elbow joint with the hands fully flexed and fingers and thumb flexed. The whole arm is twitching regularly about ten to the minute.

April 6th. Death.
Post Mortem, 3 p.m., April 7th.

Port Mortem lividity marked on dependent parts. Rigor mortis general and marked. Development fair: nutrition poor.

Thorax.


On section: Marked advanced miliary tuberculosis.

Right Lung. Same as left, only no pleural adhesions.

Heart. Pericardium contains some clear fluid. Organ healthy.

Abdominal cavity.

No peritonitis. Mesenteric glands show slight enlargement.

Small intestine and upper part of large intestine show acute congestion.

Liver. Surface is pale.

Section - Cloudy swelling and fatty change.

Spleen. Surface covered with miliary tubercles.

Section - Pale, with malpighian bodies very prominent.

Kidneys. Right Kidney - surface covered with miliary tubercles. Section shows tubercles scattered
throughout: some slight congestion.

Left kidney - ditto.

Cord.

On cutting dura some turbid fluid escaped. Dura was unduly firmly attached to bony canal. Posterior surface of cord is healthy. Anterior surface - vessels slightly engorged and whole anterior surface is covered with thin, tough, greenish yellow exudate.

Brain. Convolutions flattened. Vessels engorged: on right side in sylvian fissure is a fine yellow exudation. On left side, whole of the upper and posterior part, the sulci contained a fine yellow exudation.

Base - Covering the medulla, pons and forward to the optic chiasma is tough pale yellowish exudate.
Temperature (Centigrade)
CASE XVII.

C.C., aged 7 years.

History.

Yesterday, April 6th, about 3 a.m. patient woke up and cried out for a drink. He remained in a restless state all the rest of the night. At 8 o'clock instead of getting up for school, of which he was very fond, he was disinclined to move. At midday he complained of a headache and vomited, and soon after complained of a pain down his back and he felt "sore all over".

His throat felt sore and he was disinclined for food. In the evening the headache was more severe and he again vomited, was shivering, and the rash was then first seen. The doctor was called in and sent him on to us on 7th, i.e. the next day.

There was a history of a recent blow on the head.

Previous Illnesses. Measles four weeks ago, followed by pneumonia, which he had just recovered from: had only just gone back to school four days ago.

The father was a carter and worked in the docks. The house was clean; there were two rooms for two adults and two children.

On Admission.

Patient's development is poor. Nutrition very
poor. Patient looks very ill. Lies on right side with head markedly retracted; legs drawn up.

Face is flushed on both cheeks; eyes kept shut; mouth half open; constantly grinds the teeth.

Consciousness retained.

Back of neck is tender. Absolute rigidity of neck.

Is very restless, constantly shifting about, and crying out as if in pain.

Complains of cold when bed clothes are removed.

Skin - Dry, and poorly nourished. About chest and abdomen are small haemorrhagic spots about 1/16th to 1/8th of an inch diameter: do not disappear on pressure.

No tache.

No smell.

No general tenderness.

Temperature 98.4°. Pulse 130. Respiration 36.

Nervous System.

Patient seems weak. Sensation appears unaffected.

Reflexes - Abdominal and epigastric not present.

Plantar present - flexion.

Tendon - No jaw jerk; knee jerks both exaggerated; ankle jerk present; no ankle clonus obtained.

None others obtained.

Kernig's sign well marked.
Alimentary.

Tongue bright red, covered with white fur.
Breath is foul. Takes milk very well.
Stomach, liver and spleen normal.
There is some abdominal tenderness.

Respiratory.

Throat slightly congested but clean. Nothing of note in chest.

Circulatory.

Pulse rapid, strong and regular. Cardiac sounds closed in all areas.

Urine.

Acid. Specific gravity 1025. Albumin - a trace. Urea - 11 grains per oz.

Lumbar Puncture.

Seven c.c. obtained. Fluid under moderate pressure, turbid.

Wh. B. C. 56,250 per c.mm.
Polymorphs. 95%
Lymphocytes 5%

Film shows many meningococci extracellular and a few intracellular.

Albumin 7 grammes per litre.

Blood.

R.B.C. 5,220,000 per c.mm.
W.B.C. 34,375 " "
Hb. 115%
Differential leucocyte count:

Polymorphs. 83%
Large Mononucl. 8%
Lymphocytes 11%

April 10th. Patient still very restless and noisy. There is slight cough, and on examination there is slight impairment at right base and rhonchi on both sides at bases.

On forehead are many, and on legs are a few, raised pale red spots about 1/6" diameter, which do not disappear on pressure.

He complains of his legs being very sore.

There is a crop of herpes on the upper lip.

April 12th. Lip is becoming very sore and is swelling. Patient is becoming emaciated.

Last night he vomited for first time since admission.

April 15th. Put on hot baths, twice daily, 20 minutes at 110° - 108°.

The lips are very swollen, sore and bleeding.

There has been some general hyperaesthesia latterly.

Right base - Still impaired resonance; breath sounds distant and are a few rales.

There is a constant short cough.

April 17th. Emaciation is getting worse, but the patient seems to be making satisfactory progress.
He is brighter, quieter and is beginning to take an interest in the ward. He sleeps better and takes his food better, and enjoys his baths very much. After each bath he has a good sleep.

Abdominal and epigastric reflexes have returned. Kering’s sign still present.

April 22nd. Right base is now clear and cough is improving. There is much less headache – it is only occasional.

May 6th. Patient made uninterrupted progress since baths begun. He can now bend his head right forward on to his chest. Headaches have gone, sleeps well, eats well and is beginning to put on some flesh.

May 8th. Got up in chair for first time.

May 28th. Patient dismissed.

Patient when seen six weeks later was in perfect health. He was as bright as ever, plump; no recurrence of headaches or sickness; appetite good; no constipation. Sight, hearing and memory as good as before.

Has made a perfect recovery.
CASE XVIII.

R.N., aged 11½ months.

History.

Three weeks ago patient had a fit; she twitched all over. Head got drawn back, and patient squinted.

Previous to this five days, patient had severe diarrhoea and vomiting.

Since the fit she has been ill and apparently in a half conscious condition, and the head has been retracted all this time. Child has been very tender.

The day after the fit patient was taken to the Sick Children's Hospital, Edinburgh, when cerebrospinal fever was diagnosed, but the prognosis given frightened the mother and she took the child home. Now as it has survived so long she though she would give it a chance.

No previous illness.

The father is a slater. The house is roomy, but dirty; has three rooms for the two parents and the patient.

It is not a breast fed baby.

On admission:

Patient is poorly developed and badly nourished. Is obviously very ill; colour, pale greyish.
There is no restlessness: lies quite unconscious on left side with the knees drawn up.

Cries out occasionally and feebly.

Head is markedly retracted and neck is quite rigid: any attempt to straighten the head causes pain. There is no tenderness of neck on pressure.

Skin - No rash; no herpes. No cyanotic flush.

Extremities cold.

No cerebrospinal smell.

Temperature 100.2°. Pulse 150. Respiration 44.

Nervous System.

Motor power generally weak.

Sensory - General hyperaesthesia.

Reflexes - Abdominal and epigastric absent.

No tendon reflexes obtained.

Kernig's sign absent.

Respiratory.

Throat clean. Chest clear.

Circulatory.

Pulse very feeble. Sounds closed in all areas.

Alimentary.

Patient is frequently vomiting. Tongue is clean.

Abdomen is retracted and tender.

Stomach and spleen normal.

Liver dullness reaches from 7th interspace to costal margin.
Urine.

Acid. Specific gravity 1028. Albumin - a trace.

Blood.

R.B.C. 6,500,000 per c.mm.
W.B.C. 13,125 " "
Hb. 115%

No differential count made.

Lumbar Puncture.

Twelve c.c. obtained. Fluid under slight pressure.

W.B.C. 1560
Polymorphs. 59%
Lymphocytes 41%

Fluid very slightly turbid - of lemon yellow colour.

Only very few meningococci seen.

Albumin - 18 grammes per litre.

April 10th. Patient is difficult to keep warm.
There is a thick pusy discharge from right nostril.

April 13th. Condition much worse.

Cannot get her to take any feeds; nasal feeds have been resorted to.

Vomits frequently. Bowels are inclined to be loose: green slimy motions.

Pulse is very feeble. Colour at times gets bad, becomes cyanotic.

Head retraction is very marked, the occiput touching the back.
April 14th. Patient in extremis. Has Cheyne-Stokes respiration.

Died 8.55 p.m.

No Post Mortem obtained.
A.P., aged 4 years.

History.

On April 14th patient was not well and shivered; was put to bed. Vomited that night.

April 15th. Vomited again in the morning, but went out to play as usual. In the evening he was drowsy, complained of his head, and there was some retraction noted.

April 16th. His headache was worse and he could not lift his head up. There was no feverishness, no vomiting and no restlessness.

April 17th. Being no better, he was taken to Leith Hospital where the diagnosis was made and he was sent on to us.

There is history of his having fallen on his head two weeks ago.

No previous illness.

The father is a dock labourer.

The house is of only two rooms, with two adults and four children in it, and is not very clean, with doubtful sanitation.

On admission:

Patient well developed and fairly well nourished. Looks felled. Lies on left side, moaning feebly.
with eyes drooping and mouth slightly open. There is no restlessness: is cold, especially extremities.

Slight cyanosis about face: finger tips and toes are blue.

Skin - Is branny desquamation about sides of neck. Both feet show desquamation on heels.

Over chest and abdomen are widely and irregularly scattered yellowish brown spots, which do not disappear on pressure.

The skin marks very easily on abdomen and chest, but tache cerebrale is sluggish on forehead.

There is no smell.

Patient resents being touched: appears to be general tenderness. He does not answer when spoken to; cannot determine whether due to deafness or not. Generally appears to be under the influence of morphia. Pupils are contracted and do not react to light.

There are four marks of hypodermic needles in arms. There is evidence of lumbar puncture.


Nervous System.

Motor - generally impaired from weakness.

Sensory - is general hyperaesthesia.

Reflexes - Abdominal and epigastric absent. Cannot determine tendon reflexes.
Kernig's sign present. 
Eyes - Pupils equal, contracted; do not react to light.

**Alimentary.**
Tongue covered with brown thick fur, and mouth full of sticky mucus.
Abdomen - Nothing of note.
No enlargement of spleen.
Stomach and liver normal.
Patient is constipated.

**Respiratory.**
Throat congested. There is slight impairment of resonance at left base, and the breath sounds are distant, but there are no accompaniments.

**Circulatory.**
Pulse weak and irregular. Cardiac sounds closed.

**Urine.**
Acid. Specific gravity 1025. Albumin - 0.2 grains per oz.

**Blood.**

\[
\begin{align*}
\text{R.B.C.} & \quad 4,400,000 \text{ per c. mm.} \\
\text{W.B.C.} & \quad 32,000 \quad " \quad " \\
\text{Hb.} & \quad 102\% \\
\end{align*}
\]

Differential leucocyte count:-
Polymorphs. 86%
Lymphocytes 8.5%
Large Mononucl. 5.5%
Lumbar Puncture.

Twelve c.c. obtained. Fluid ran out in rapid drops; very turbid, with rapid sedimentation of pus.

Wh. B. C. 43,750 per c.mm.
Polymorphs. 89%
Lymphocytes 11%

Meningococci in large numbers, both intra- and extracellular. An estimation of the meningococci was attempted by counting 100 cells: 99 meningococci counted, therefore .99 per cell.

Albumin - 2 grammes per litre.

April 19th. Patient is very restless and crying out all day. Lumbar puncture gave no relief, but temperature ran up immediately after it.

Put on hot baths, which seemed to give great relief.

Has incontinence of both urine and foeces.

April 22nd. Patient still very restless. Is vomiting frequently.

General hyperaesthesia very marked.

Sight and hearing are unaffected.

The abdomen is distended.

April 23rd. There is some purulent discharge from the left eye. Patient is quieter, not screaming so much, and is not so restless.

Is taking his feeds very badly.
April 25th. Patient has to be nasal fed. Under chin is a bright red erythematous rash, which has well defined edge. Disappears on pressure.

April 29th. Rash spread to arms and legs.

May 2nd. Rash has disappeared.

There is thick discharge of pus from left nostril. Patient very much weaker and very emaciated.

May 5th. Patient died to-day. Got steadily weaker: vomiting continued: could only digest very smallest quantities of food, which was given pepto-nised.

In this case the hot baths seemed to do very little good but temporarily relieve headache. Patient slept well afterwards for an hour or two.

Post Mortem.

Rigor mortis general. Post mortem lividity marked in dependent parts.

Chest.

Left Lung. No pleuritic adhesions: bronchial glands enlarged and hard. Large bronchi show acute bronchitis.

On section, lower lobe cuts very solid and shows extensive bronchopneumonia, which is tuberculous.

Right Lung. No adhesions: bronchial glands enlarged. Large bronchi show acute bronchitis.
On section, upper and middle lobe show bronchitis. Lower is firm, with numerous small solid nodules of bronchopneumonia.

The heart shows nothing pathological.

Abdomen.

There is no peritonitis.

The mesenteric glands are enlarged and are tuberculous.

The liver - pale, fatty degeneration: shows numerous miliary tubercles.

Spleen slightly enlarged, firm. Malpighian bodies unduly prominent. No tubercles.

Kidneys. Both show some cloudy swelling.

Intestines. No undue prominence of peyer’s patches. No areas of congestion.

Brain. Meninges are markedly injected. Convolutions are flattened. The sulci contain a thin film of yellowish exudate.

Base of brain from optic chiasma right on to the cord is covered with a thick layer of greenish yellow gelatinous pus.

The under surface of the cerebellum is covered with a thin film of pus.

The ventricles are slightly dilated.

The cord. On cutting through the membranes a large quantity of turbid fluid escaped. On posterior
surface of cord particularly, and slightly on anterior surface, is a layer of pus.
CASE XX.

M.F., aged 15 years: Biscuit worker.

History.

Two days ago (on 24th) patient while at work had a very severe headache. She, however, kept on with her work. When she went home she felt worse and vomited, and the headache became very much worse. The next morning she tried to get up to go to her work but could not. She felt her neck very sore, and the headache persisted. A doctor was called in, who did a lumbar puncture, and was able to send her in to us on the diagnosis of cerebrospinal meningitis.

Patient lives in an overcrowded house. There are two very dirty rooms occupied by six adults and three children.

The father is a carter.

A child recently died at home on the same stair of cerebrospinal meningitis.

On admission:

Patient well developed and well nourished, and does not appear very ill.

Lies on her back rather log like, and looks a little dazed.

Speaks when spoken to and answers questions moderately accurately, but is easily confused.
No smell. No rash. No herpes. No coldness. No retraction of head, but is absolutely rigidity of neck; can lift up body by head, neck remaining rigid. Tache well marked. No tenderness of neck. Pulse 118. Respiration 26. Temperature 98°.

Nervous System.
Marked general hyperaesthesia. Reflexes - Superficial, Abdominal and Epigastric present. Plantar - flexion marked.

Tendon - Jaw, almost a clonus; biceps marked; supinator marked; patella clonus marked; knee jerks much exaggerated; ankle clonus very marked; ankle jerk marked.

Eyes - Sight good. Pupils equal and react to accommodation and light; slight conjunctivitis. Hearing very good.

Circulatory.
Cardiac sounds closed all areas.

Alimentary.
Tongue clean. Nothing of note.

Respiratory.
Percussion note higher on right than left side in front. No alteration in breath sounds.

May 10th. Slight tremors in hands. Pulse quick and full.
Incontinence of urine and foeces.

May 30th. Nystagmus present and marked.
Tenderness less marked.

June 1st. Purpuric eruption faint, over abdomen.

Vomiting frequently.
CASE XXI.

A.F., aged 12 years: Schoolboy.

History.

On 23rd patient had been playing football, came in tired and complained of a headache.

On 24th the headache was still present, but not so severe. It was worse on the top of his head. He vomited in the morning. There was no pain or stiffness in the neck, and he went about as usual.

On 25th he was much the same, but found he could not bend his head forward on to his chest; there was no pain if he held it up.

On 26th, overnight the pain in his head was very severe, so much so that he screamed out with it. He then went himself, walking, a distance of about 1 1/2 miles to Leith Hospital, where cerebrospinal fever was diagnosed.

As I was rung up about the case I went down to the Hospital and saw him in the waiting room. He was a perfectly healthy looking boy, well nourished and developed, with at first sight very little the matter with him.

He complained of headache, held his neck absolutely rigid, and could not bend it forward as it gave him sharp pain in the neck.
Further there was a distinct purpuric rash on the chest, which patient had not noticed before that morning.

Kernig's sign was well marked.

Abdominal reflex present.

A lumbar puncture had already been done at Leith Hospital, the film of which Dr Levers kindly allowed me to see. There were a few polymorphonuclear cells with only three meningococci to be seen on searching the whole slide. There were no lymphocytes.

The fluid had spurted out of the needle under high pressure.

Patient was transferred at once to Pilton.

Seen next morning there was nothing to be found the matter with him. The headache had gone, the stiffness of the neck had disappeared, and there was no Kernig's sign.

The rash was fading.

He was kept in bed for four days, then let up. On the sixth day after admission he was discharged, i.e., on May 2nd.

On enquiring later, I obtained the following information:

On May 18th he had been told to go a message - an occupation which he objected to always. On this occasion he flew into a temper, which lasted about
20 minutes, when he suddenly put his hands to his head and cried out as if hurt, then changed colour and had a convulsion; and died half-an-hour later.

He had been in perfect health from the time of leaving hospital till the 18th.
CASE XXII.

C.R., aged 3\(\frac{1}{2}\) years.

History.

Yesterday, 26th, patient suddenly got ill, complained of headache and was sick. At night he was shivering and it was noticed that he held his neck very rigid.

He was very restless all night, and being no better in the morning a doctor was called in, cerebrospinal fever diagnosed, and he was sent straight out to Leith Public Health Hospital.

The father is an ice cream vendor and lives in a filthy dirty two-roomed house, occupied by two adults and three children.

On admission:

Patient is badly developed, badly nourished, thoroughly rickety child.

Is very anaemic, soft and flabby.

Is perfectly conscious.

Lies on his left side and is constantly crying out or whining, and frequently screaming out at the top of his voice. He is very restless.

Head retraction is very slight, but rigidity of neck is very marked, and the neck is tender to the touch.
Skin - There is small purpuric spotted rash over the upper half of abdomen and lower half of chest.
No herpes.
Tache well marked.
There is no smell.
Does not appear to be cold.
Temperature 100.6°. Pulse 144. Respiration 28.

Nervous System.
Motor power unimpaired.
Sensory - general hyperaesthesia.
Reflexes - Cannot test them, owing to patient's resistance.
Kernig's sign absent.
Special senses unaffected.

Alimentary.
Tongue covered with white fur.
Takes food very unwillingly.
Abdomen is distended.
Spleen - anterior border can be just palpated.
Liver - lower border projects one inch below costal margin.
Stomach normal.
Patient is constipated.

Respiratory.
Throat is congested.
Nose is sore on the inside of the alae nasi, with
appearance of herpes.

Chest: Left base - impaired resonance with harsh breath sounds.

Right apex in front also impaired resonance, but no alteration of breath sounds.

Circulatory.

Nothing of note.

Urine.

Acid. Specific gravity 1022. Deposit of urates.

Albumin - a trace.

Blood.

R.B.C. 4,600,000 per c.mm.
W.B.C. 14,635 " "
Hb. 96%

Differential count:

Polymorphs. 84%
Lymphocytes 10%
Large Mononucl. 6%

Lumbar Puncture.

Seventeen c.c. obtained. Pressure = 31 c.m.

Fluid turbid, with rapid sedimentation of pus.

W.B.C. 40,000 per c.mm.
Polymorphs. 92%
Lymphocytes 3%
Large Mononucl. 5%

Albumin - 2 grammes per litre.

Film showed many meningococci both intra- and extra-cellular.
April 20th. Patient much the same. Is still very restless and cries a good deal. Resents any handling at all; hyperaesthesia seems acute. Abdominal and epigastric reflexes absent. Kernig's sign slight to-day. Rash has entirely faded. The head has gradually been more retracted. Until now the occiput touches the back. Put on hot baths.

May 6th. Head retraction is not evident but neck is still rigid. Is taking his food better and is less tender.

May 15th. Passed a good specimen of ascaris lumbricoides. Patient is making very good recovery.

June 12th. Progress uninterrupted and patient discharged to-day.

Seen six weeks later.
Patient is in excellent health and has grown. Is quite strong, not nervous, no headaches, no vomiting: eats all he can get; no constipation, and in every way has made a perfect recovery.
CASE XXIII.

J.D., aged 2½ years.

History.

On 24th patient was playing about as usual, but in the evening he complained of his head being sore. He was put to bed, was feverish and shivering, and he vomited over night.

On 25th he was still complaining of his head, but was not ill enough to alarm the parents. He was kept in the house. That night he was very restless and kept tossing about in bed.

On 26th he was much worse, and a doctor was called in. Headache was now very acute; patient very restless, and head was held stiff.

On 27th head retraction was first noted. He was vomiting frequently. He resented being touched.

Patient continued getting worse and was sent to Leith Public Health Hospital.

Patient's father was a labourer in Newcastle. The mother and three children lived in one room in Leith. The room was kept clean and the children well kept, but the room is badly lighted and ventilated.

On admission.

Patient is a moderately developed, poorly nourish-
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ed child. He is looking very ill. Is quite unconscious and very restless, tossing about from side to side, hitting his arms and head against the bed. The face has marks of bruising where he has evidently hit himself.

He screams out now and then at the top of his voice in a piercing manner.

Head is moderately retracted, but the neck is absolutely rigid and tender to the touch.

Skin - Over chest and back are a few scattered purpuric spots. No herpes.

Tache is well marked.

No smell.

No flush on cheeks.

Extremities are cold.


Nervous System.

Motor power unimpaired.

Sensory - No hyperaesthesia except at back of the neck.

Reflexes - Abdominal present; epigastric absent; knee reflexes are exaggerated. No others obtained.

Kernig's sign absent.

Alimentary.

Tongue clean.

Abdomen - normal.
Stomach, liver and spleen normal.

Respiratory.

Throat clean.

Anteriorly - Right apex has impaired resonance: the breath sounds are harsh with increased vocal resonance, but no accompaniments.

Circulatory.

Pulse feeble and irregular. Cardiac sounds closed in all areas.

Urine.

Acid. Specific gravity 1018. No abnormal constituents.

Blood.

R.B.C. 4,850,000 per c.mm.
W.B.C. 18,750 " "
Hb. 105%

Differential leucocyte count:

Polymorphs. 88%
Lymphocytes 7%
Large Mononuclears 5%

Lumbar Puncture.

Only 3 c.c. obtained: very slow drops.

Fluid turbid.

Wh.B.C. 35,750 per c.mm.
Polymorphs. 87%
Lymphocytes 13%

Meningococci, both extra- and intra- cellular, in large numbers, present.
This patient went steadily down hill until about the beginning of June.

The headaches were persistent, vomiting was frequent, and he became emaciated to an extreme.

The abdominal reflex was soon lost.

The head retraction became extreme.

There was the usual difficulty of swallowing with the marked head retraction and objection to food.

From the beginning of June there was a slow but steady improvement, but I think there was some permanent hydrocephalus.

On inquiry in October I found that he had steadily improved and was discharged on October 8th. The mother said he was almost well again at the end of the month.
CASE XXIV.

M. McL., aged 9 years.

History.

On April 1st patient fell off a chair and hit her head. From that date she has not been well. Until the 7th she was very constipated, then a doctor was called in and gave her some powders. She seemed to get worse and complained of her head aching. About the 15th her neck began to feel sore and the headache got worse. She never was sick. As she continued to get worse another doctor was called in and he diagnosed cerebrospinal fever.

Previous illness. When three years old she had a fit and has had a squint ever since.

Measles when 7.

The father is a dock labourer. The house is dirty and overcrowded. There are four adults and one child in two rooms, which are badly ventilated and lighted.

A baby died two weeks ago of pneumonia (?)..

On admission.

Patient well developed, but poorly nourished.

She is quite unconscious. She lies on the left side, knees drawn up, quite still, but frequently cries out at the top of her voice.
Head is moderately retracted; neck rigidity is absolute, and back of neck is tender.

Skin - There is no purpuric rash. Is evidence of recent herpes on the upper lip; is purplish pink flush on cheeks.

Tache poorly marked.

She is very cold to the touch all over the body, and especially the extremities.

General tenderness is very marked.


Nervous System.

Motor power unaffected.

Sensory - general hyperaesthesia.

Reflexes - Jaw jerk present; both knee jerks present. Abdominal, epigastric and plantar all active. No others elicited.

Hearing is good.

Eyes - Internal strabismus of right eye; nystagmus both eyes.

Alimentary.

Tongue is clean.

Takes food very unwillingly; spits most of it out all over the place.

Abdomen is tender, and is slightly retracted.

Spleen - No enlargement.

Liver - Only small area of impaired resonance.
No absolute liver dullness is to be made out.

Bowels fairly regular.

Respiratory.

No congestion of throat.

Nothing abnormal in chest.

Circulatory.

Pulse is rapid and feeble. Cardiac sounds closed all areas.

Urine.

Acid. Specific Gravity 1025. No abnormal constituents.

Blood.

No count.

Lumbar Puncture.

Four c.c. obtained. Fluid turbid.

Wh. B.C. 14,750 per c.mm.
Polymorphs 87%
Lymphocytes 9%
Large Mononucl. 4%

Meningococci - few present both intra- and extracellular.

May 1st. This morning patient had a severe haemorrhage from rectum. Blood dark reddish brown. Patient collapsed, cold, with rapid feeble pulse.

May 8th. Patient has steadily improved. Has regained consciousness, but still cries out occasionally.
Complains of headache still.
Takes her feeds properly and more willingly.
There has been no more haemorrhage, but the stools were tarry until the 5th.
Pulse has been irregular occasionally, but has steadily improved.
Abdominal tenderness is still present.
From this point she steadily improved without a hitch until she was discharged on July 23rd.

Seen four weeks later she was running about enjoying herself and was fat and perfectly well. The mother says that previously she used to be nervous but since the illness has not been so. There have been no recurrences of the headaches; the bowels have been perfectly right, and in every way the recovery has been complete.
CASE XXV.

J.P., aged 2 years.

History.

On April 29th patient vomited everything he took and was shivering a good deal; the parents simply put him to bed and did not call in a doctor. He, however, made no improvement and on May 1st (yesterday) he was very much worse. The neck became stiff and he could not stand any handling, as he was so tender: a rash was also seen on this day.

He was then seen by a doctor who sent him on to us.

This is a brother of Case XIX. He had undergone the town treatment of nasal syringing, as also the rest of the household, at the time of disinfecting house after removal of Case XIX.

On Admission:

Development and nutrition are good.

Patient is quite unconscious.

He cannot be said to lie in any position for he is never in one for two minutes at a time, the restlessness being most severe. He is wriggling about and tossing from side to side, screaming at the top of his voice almost continuously excepting an occasional pause for breath. In fact if allowed to go
on he would apparently wear himself right out. The whole cot has to be padded.

There is no head retraction but there is absolute rigidity of the neck.

Though a rash was reported present yesterday, of a purpuric variety, there is no sign of one to-day. There is no herpes.

The left eye is always shut, and there is some purulent ophthalmia.

There is very marked tenderness all over the body, and I think this is one cause of the extreme restlessness, as it may be too tender to lie on one spot for any time.

Temperature 101.2°. Pulse 144. Respiration 36.

It is impossible to examine him properly on account of the restlessness.

Lumbar Puncture.

Pressure 27 c.m. Quantity 15 c.c. Fluid very turbid, and spurted out. Rapid sedimentation of pus.

Wh.B.C. 143,750 per c.mm.
Polymorphs 88%
Lymphocytes 2%
Large Mononucl. 3%
Transitional 7%

Albumin 5.5 grammes per litre.

Meningococci, both intra- and extra-cellular in plenty.
May 3rd. Patient is still exhibiting most extreme restlessness. He has got quite hoarse from screaming and has hardly had any sleep. The only time he is quiet at all is when he is picked up and carried about by a nurse, when he immediately goes off to sleep. Unfortunately, not having a nurse to spare for this, he has to be kept as quiet as possible by other means.

As far as is possible there is nothing of note in the circulatory or respiratory systems.

Urine.

Acid. Specific gravity 1022. Deposit of urates. No abnormal constituents.

No Diazco reaction.

May 5th. The restlessness and screaming are still almost incessant, except at night, which he sleeps through now without any trouble.

The left eye is still shut, and the internal rectus seems paralysed.

He is still very tender.

Is quite conscious now and asking for "pieces".

May 10th. Patient has persistently kept up the screaming of the first few days during the day. The restlessness has completely subsided, and he seems perfectly well except for the screaming, but as he will only stop this by being nursed he has had to be
discharged owing to the great discomfort of the other patients. His left eye is still closed and left internal rectus still paralysed, otherwise he seems quite well.

August 15th. Patient seen three months later. He is strong and healthy and in excellent spirits. He has a slight cough, but nothing to be made out on examination of chest.

Takes his food well. There is no vomiting; is rather constipated. There are occasional headaches and he is very nervous. He is, however, absolutely deaf in both ears. The paralysis of the left internal rectus has gone and though for a month after leaving Hospital the left eye remained closed, it is gradually improving and he can open it to a considerable extent, but not perfectly.
CASE XXVI.

A.G., aged 5½ years.

History.

She went to bed on Friday night (3rd) with a severe frontal headache, and a slight cough and pain in right side. At 11.30 p.m. she was talking rubbish and was quite unconscious. She felt cold and was shivering.

Next morning early she vomited, and since then has frequently vomited again.

She was a little more conscious in the morning, answering sensibly when spoken to and complained of her legs being sore, but when left alone goes on rambling.

Previous Illness. Measles, and she has frequently had fits.

The father was a maltman.

The house was of two rooms, accommodating three adults and two children, but was dirty, as also were the inmates.

On admission:

Patient well developed and nourished. Is most markedly restless, being continually tossing about and at one time actually got up on to the top of her head.
Frequently screaming out and moaning. Is quite unconscious.

There is no head retraction and no rigidity of the neck.

Skin - There are a few petechial spots on abdomen and chest. No herpes.

Tache is well marked.

She is bruised all over the trunk and limbs from knocking herself about evidently.

There is no smell.

Patient is cold.

Temperature 101.2°. Respiration 36. Pulse 100.

Circulatory.

Pulse is full and rapid: it varies considerably, however, in regularity.

Cardiac sounds closed all areas.

Respiratory.

No congestion of throat. There is impairment of resonance all over right side posteriorly, and breath sounds are harsh. No accompaniments.

Alimentary.

Tongue has whitish brown fur covering it.

Patient takes food unwillingly.

Nothing of note on examination of abdomen.

Spleen not enlarged. Liver - no enlargement.

Stomach normal. Bowels constipated.
Nervous System.

Motor power unimpaired.

Sensory - Marked general hyperaesthesia. Screams out at once on slightest touch anywhere.

Reflexes - Knee jerks much increased; ankle jerk present. Plantar - Babinski.

Abdominal and epigastric reflexes absent.

Urine.

Acid. Specific gravity 1035. Contains trace of albumin. Sugar present - quantity unestimated.

Blood.

| R.B.C. | 4,850,000 per c.mm. |
| W.B.C. | 21,840 " |
| Hb.    | 105% |

Lumbar Puncture.

Quantity 15 c.c. Pressure 46 c.m. Fluid turbid, with rapid deposit of pus.

| Wh.B.C. | 34,320 per c.mm. |
| Polymorphs. | 94% |
| Large Mononuclears | 6% |
| Lymphocytes | 0% |

Film shows very few meningococci, both intra- and extra-cellular.

Albumin - 5 grammes per litre.

Patient died on 6th.
Post Mortem.

Brain. Vessels of meninges engorged, convolutions flattened, vessels congested. Whole surface of brain, especially in sulci, is covered with thin yellowish exudate which is thicker over a small area in centre of vertex than elsewhere.

On base this film is also seen with a small mass of pus about the size of a threepenny bit just over the optic chiasma. On upper surface of cerebellum is also a small collection of pus.

Cord. Posterior subarachnoid space contains a thin layer of greenish yellow pus.

Heart. Is large, but nothing abnormal.

Right Lung. Recent pleurisy with adhesion on the surface of lower lobe and on diaphragmatic surface. Bronchial glands are enlarged and firm. Large bronchi show acute bronchitis. On section, nothing of note.

Left Lung. Bronchial glands and bronchitis same as right. No adhesions.

Abdomen. No peritonitis. Peritoneal glands show enlargement and are congested. Small intestines show patches of congestion towards lower part, and solitary glands prominent. Large intestines - nothing of note.

Stomach contains large quantity of dark greenish black fluid.

Kidneys - Nil.
Spleen - Malphigian bodies very prominent.
Thyroid and thymus was markedly enlarged.
Middle ears and ethmoid clear.
CASE XXVII.

J.C., aged 40 years. Canvasser.

History.

Patient has been drinking heavily lately. Two weeks ago he got rather badly mauld in a fight. On Saturday 4th was again drinking, and on Sunday 5th he got suddenly ill. He complained of headache and vomited.


There was no rash: no head retraction.

Admitted Wednesday 8th.

He knocked about the country canvassing and in Leith lived in a lodging house. It was dirtily kept. There were three rooms for six adults.

On admission:

Patient is a very well developed, well nourished, big, strong man.

He is semiconscious.

He is very restless, continually shifting from side to side.

He knows his name and answers questions, but is in a dazed condition. He is continually groaning and complains of his head.

The head is slightly retracted; the neck is held absolutely rigid.
Skin - No rash: no herpes. Both elbows are red and bruised.

No tache: no general tenderness.

The typical smell is very well marked.


Nervous System.

Motor and sensory - unimpaired.

Reflexes - Only knee jerk elicited of the tendon reflexes. Abdominal and epigastric absent. Plantar present with flexion.

Kernig's sign present and well marked.

Eyes - Pupils small, react to light. Internal strabismus of right eye.

Alimentary.

Tongue dry and dirty brown. Breath is very foul.

Stomach is normal.

Spleen - no enlargement.

Liver - Lower border extends 3/4" below costal margin.

He is constipated.

No abdominal tenderness.

Respiratory.

Throat is congested.

Nothing of note on examination of chest.

Circulatory.

Pulse is rapid, full and regular. Cardiac sounds all closed.
Urine.


Blood.

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<td>W.B.C.</td>
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<td>Hb.</td>
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Differential leucocyte count:

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<tr>
<td>Lymphocytes</td>
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<tr>
<td>Large Mononuclears</td>
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Lumbar Puncture.

Quantity 10 c.c. Pressure 19 c.m. Fluid very turbid. Rapid deposit of pus.

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<tr>
<td>Lymphocytes</td>
<td>1%</td>
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<tr>
<td>Large Mononuclears</td>
<td>1%</td>
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Albumin - 10 grammes per litre.

Film - Only a few meningococci, both intra- and extra-cellular.

May 10th. Patient is quite unconscious.

Still very slight head retraction.

Cannot swallow food properly; appears not to be able to swallow.

7 p.m. Is much worse. Still very restless.

On back is a faint purpuric rash - only a few spots and they do not appear elsewhere on the body.
Appears to be cold and is shivering frequently, though he feels quite warm to the touch.

The chest is quite clear.

Face is cyanosed.

10 p.m. Face is quite blue. Pulse remains full but more rapid.

Is breathing with great difficulty.

Legs and arms are twitching.

10.30 p.m. Colour slightly improved.

Breathing becoming slower and more laboured. This increased till breathing stopped, but pulse kept on beating regularly with full volume for three minutes, then gradually weakened till imperceptible. The face then became almost black.

No Post Mortem obtained.
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<th>Pulse Rate</th>
<th>Resp Rate</th>
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**Patient:** William Smith  
**Age:** 16 years  
**Disease:** Meningitis  
**Result:** Death
CASE XXVIII.

W.E., aged 16 years.

History.

On 7th - two days ago - patient suddenly got a severe pain in his head, both frontal and occipital, and he vomited and was shivering.

On 8th he was much worse and complained of severe backache; he became a little delirious towards evening and entirely lost his hearing.

Admitted on 9th, having been diagnosed as Typhus. Patient works at wood chopping, and is of a low type. His home is dirty and overcrowded. There are two rooms occupied by four adults and two children.

On admission:

Patient is a very well developed and well nourished boy for his years.

He is quite unconscious. He lies on his left side, with the legs drawn up; is shouting out at the top of his voice, and is very restless.

Head retraction is moderate; neck rigidity absolute; but no tenderness of neck.

Skin - Slight herpes on the alae nasi and upper lip. On the abdomen there is a faint scattered purpuric rash.
Face is of a dusky brownish bluish colour, and has a cyanosed appearance, but the lips are of good colour.

There is no coldness.

Tache is present, but not well marked.

There is typical soapy smell.

Temperature 99.4°. Pulse 130. Respiration 34.

Nervous System.

Motor power extremely good.

Sensory - No hyperaesthesia.

Reflexes - No tendon reflexes elicited. Abdominal, Epigastric and Plantar all well marked.

Special senses. Become completely deaf since onset.

Eyes - Pupils equal, both small: no reaction to accommodation or light: no squinting.

Kernig's sign is well marked.

Alimentary.

Lips good colour: tongue dry and brown.

He is constantly wanting water, and takes it and his feeds greedily.

Abdomen - No retraction or distension. Complains of much tenderness on palpation.

Liver - 6th rib to costal margin.

Spleen - No enlargement.

Stomach - No enlargement.
Respiratory.

Throat congested. No enlargement of tonsils. Nose is sore, with herpes both inside and out on both nostrils.

Slight impaired note anterior and posterior right apex, with breath sounds slightly high pitched and prolonged expiration. No accompaniments. Vocal resonance - no alteration.

Cardiac.

Pulse full and strong: heart - no enlargement: sounds closed all areas.

Urine.


Blood count, 10th May.

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<td>W.B.C.</td>
<td>16,875</td>
<td>per c.mm.</td>
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<tr>
<td>R.B.C.</td>
<td>5,960,000</td>
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<td>Hb.</td>
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Differential leucocyte count:-

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<tbody>
<tr>
<td>Polymorphs.</td>
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<tr>
<td>Lymphocytes</td>
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<tr>
<td>Large Mononucl.</td>
<td>7%</td>
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<td>No others</td>
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Lumbar puncture.

Pressure 48 c.m. Quantity 19 c.c. Fluid spurted out under high tension. Fluid very turbid.

Wh.B.C. 49,500 per c.mm.
Albumin - 6 grammes per litre.
Film - Polymorphs, in abundance; meningococci, intra- and extra-cellular.

Polymorphs. 97%
Large Mononucl. 2%
Lymphocytes 1%

May 10th. Patient got rapidly worse and died day after admission.

Post Mortem 11/5/07.
Rash noted on 10th almost entirely faded.
Post mortem lividity marked on back: rigor mortis general.

Chest.
Left Lung - Firm adhesions posterior part of upper lobe.
Section - Bronchitis of larger bronchi; upper and lower lobe dark, firm, congested.

Right Lung - Marked adhesions upper and lower lobe; very firm. No bronchial glands enlarged.
Section - Bronchitis larger bronchi. Lung as in left.

Heart - Nothing of note.

Abdomen.
No peritonitis. Mesenteric glands very much
enlarged; pale yellow. Lower part of small intestine shows marked congestion.

Liver. Early fatty change.

Spleen. No enlargement: is very friable and soft, any pressure being sufficient to convert it into a sort of paste. Malpighian bodies very much unduly prominent.

Kidneys healthy.

Brain.

Meninges - Vessels very much engorged. Surface, marked congestion of vessels; no flattening of convolutions. Whole surface, vertex and sides particularly, covered with fairly thick layer of yellowish pus. Base has thin film covering it, and over optic chiasma is a small clot. Upper surface of cerebellum has thick layer of pus on it.

Cord. Posterior surface only has a thin layer of pus on it. Large quantity of very turbid fluid escaped on cutting cord.

Middle ears clear.

Ethmoid cells clear.
CASE XXIX.

J.G., aged 50 years.

History.

This is the father of Case XXVI. After the child had been removed the whole house was disinfected and the contacts had their noses and throats syringed. This man, though, was not feeling well at the time. Nothing however was perceptibly the matter with him till this morning. He had been very melancholic the night before, and about 3 a.m. to-day was wanting to get up to go to his work and was irrational.

About 8 a.m. he began to hiccough and continued this for an hour. He complained of headache and vomited, and was shivering.

When being removed to Hospital he was perfectly rational and apparently well.

He has had fracture of the base of the skull three years ago.

He is a maltman. House conditions same as Case XXVI.

On admission:

Patient seen at 4 p.m. soon after admission, was apparently in good health. He complained of nothing and felt quite well. Talked perfectly rationally.

There is no retraction; no stiffness of the neck.
No rash: nothing in fact to arouse any suspicion of illness except the Temperature of 102.4°.

The pulse is 136. Respiration 40.

On examination.

Excepting for presence of slight Kering's sign, there is nothing abnormal in any of the systems. Both abdominal and epigastric reflexes are present.

At 8 p.m. - The patient is quite unconscious and very ill. There are now a few purpuric spots on the abdomen and chest.

Kernig's sign is more marked, and abdominal and epigastric reflexes absent.

At 10 p.m. - Temperature is now 105.2°. The rash is well out all over the trunk chiefly, but to some extent on the arms and slightly on the legs.

Lumbar Puncture.

This was done very imperfectly to obtain a film. The fluid was slightly turbid and showed a large number of polymorphs with many meningococci both intra- and extra-cellular.

Blood.

Leucocytosis of 28,500 per c.mm.

At 5 a.m. - The rash almost entirely covered the whole skin.

Death - 6 a.m. on 11th.
Post Mortem.

Chest. The right lung was firmly adherent to both parietal and diaphragmatic surfaces.
Both lungs showed very marked cedema.

Heart. Showed a few petechial hemorrhages over the surface of the left ventricle.

Abdomen.
No peritonitis. The intestines are mottled with recent petechial haemorrhages.
The liver shows early fatty change.
The spleen is normal.
The kidneys are normal.

The Brain.
The whole surface is covered with a thin film of yellowish exudate, excepting the basal surface, which is clear.

There is marked congestion of all the superficial vessels.
The cord. The vessels are much engorged but there is no exudate on either anterior or posterior surfaces.
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<thead>
<tr>
<th>Time</th>
<th>Temperature (Fahrenheit)</th>
<th>Pulse Rate</th>
<th>Deep Breathing</th>
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**Clinical Chart**

**Result:** Death
CASE XXX.

K.R., aged 7½ years.

History.

Patient went as usual to school this morning, but when she was there complained of headache and vomited. She was therefore sent home.

At 12 noon she had a convulsion and has been unconscious since.

She was sent to us in the afternoon.

The house is overcrowded, there being nine adults and six children in five rooms, which are badly ventilated and dirty.

The father is a dock labourer.

On admission:

Patient is well developed and fairly well nourished. She is quite unconscious and extremely restless, and is continually screaming out. She lies on her side with legs drawn right up in a crouched up position. The hands are peculiar: the thumb is fully flexed across the palm and the fingers at the distal interphalangeal joint are flexed.

There is no head retraction, but neck is held rigid.

Skin - No rash; no herpes. Cheeks and lips are cyanosed.
No tache.

Faint cerebrospinal smell.

She is inclined to be cold and is very sensitive to cold.

Temperature 101°. Pulse 96. Respiration 32.

Nervous System.

Motor - Unimpaired.

Sensory - General hyperaesthesia.

Reflex. No tendon reflex elicited. No abdominal or epigastric present.

Kernig's sign present.

Eyes suffused. Pupils dilated and equal. No strabismus.

Alimentary.

Lips cyanosed. Tongue dry and covered with brown fur.

Refuses to take any food.

Abdomen - No retraction or distension. No special tenderness.

No enlargement of liver or spleen.

Stomach is normal.

She is constipated.

Respiratory.

No congestion of throat.

There is no alteration in the resonance anteriorly, but there is bronchial breathing with coarse rales.
Posteriorly, there is slight impairment of resonance on the left side, with breath sounds and accompaniments same as in front.

Circulatory.

Pulse is full, regular - 108 per minute. Cardiac sounds closed in all areas.

Urinary.

Acid. Specific gravity 1032. No albumin or sugar. Diazo negative.

Urea - 6\(\frac{1}{2}\) grains per oz.

Blood.

No count.

Lumbar Puncture.

Pressure 8 c.m. Quantity 2 c.c. Fluid, which was very turbid, only flowed in very slow drops, but the patient was in extremis when it was done.

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<thead>
<tr>
<th>Wh.B.C.</th>
<th>58,750 per c.mm.</th>
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<tr>
<td>Polymorphs.</td>
<td>93%</td>
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<td>Lymphocytes</td>
<td>4%</td>
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<td>Large Mononucl.</td>
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</table>

No meningococci found.

May 11th. At midnight patient became quite quiet. She got very cold; the left arm and both legs became rigid; the hands were both clenched. This lasted for 7 minutes.

Died 8.30 a.m.
Post Mortem.

Abdomen. No peritonitis.

Mesenteric glands enlarged and congested.

About two feet from lower end of small intestine is area of 16" where intestine shows acute congestion.

Solitary glands are very prominent.

Spleen. Slight prominence of malphigian bodies.

Kidneys. Healthy; right one very much smaller than left.

Liver - Early fatty change.

Chest - Thymus very large, extends to third interspace.

Lungs. No adhesions. Both lungs - acute bronchitis of large bronchi and slight enlargement of bronchial glands.

Left lung shows slight congestion on section.

Right lung appears healthy.


Brain. Meninges markedly congested. Surface of brain, convolutions slightly flattened. Vessels very much engorged. The whole surface, more especially the sulci, is covered with a thin yellow exudate. Base is clear except for small film over anterior cystum. Upper surface of cerebellum has a small layer of pus.
Cord. Anterior surface only has thin layer of pus.
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**Clinical Chart**

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CASE XXXI.

A.P., aged 20 years.

History.

Patient was not feeling well all earlier part of the week.

On Wednesday. 8th, she came back at dinner time from work feeling wretched, but she went back after dinner. On leaving work at 5 she could not stoop to get her boots on, but had to get someone else to do it for her and she could hardly get home. She felt queer about the head and eyes and was shivering one moment but was quite hot the next.

Thursday 9th - She was sick over night; complained of a sore throat and of being thirsty.

After midday the headache got worse and she vomited again. The headache was frontal and she felt as if it were swollen.

She went to bed and fell off to sleep. At 9 p.m. she was found to be unconscious and had a convulsion.

Previous Illnesses. None.

The father is a school board officer and has been visiting houses infected with cerebrospinal. The house is irreproachable.

On admission:

Patient is well developed and fairly well nourished.
She is quite unconscious. She hears sounds and turns towards you when you speak, but makes no attempt to answer.

She is intensely restless, is throwing herself about almost continuously and persists in throwing off the bed clothes and taking all her clothing off.

There is no head retraction, but the neck is rigid.

Skin - No rash: no herpes.
No tache.
Is cyanosed, the lips are dark purple and cheeks have the cyanotic flush.
Has the black soapy smell.
The extremities are cold.
No tenderness.

Nervous System.
Motor power unaffected.
Sensory - No hyperaesthesia.
Reflexes - No jaw jerk: knee jerks both present to normal extent: ankle clonus marked on left side: ankle jerk present on left side. No other tendon reflexes elicited.
Abdominal and epigastric reflexes absent.
Plantar present, with Babinski.
Kernig's sign very well marked.
Eyes - Pupils moderately dilated, equal and react to light. No strabismus or nystagmus.

Respiratory.

Throat red and congested. No enlargement of tonsils.

Nothing of note on examination of chest.

Circulatory.

Pulse varies considerably. Sometimes it is full and regular, while at others it is quite irregular and later on will be quite weak.

There is a slight indistinctness of the mitral systolic.

Alimentary.

Tongue dry, brown and cracked. Has difficulty in swallowing.

Stomach is normal.

Liver and spleen show no enlargement.

No tenderness of abdomen.

Patient is constipated.

Urine.


Blood.

R.B.C. 5,930,000
W.B.C. 15,000
Hb. 120%
Differential leucocyte count:

Polymorphs. 87%
Lymphocytes 6%
Large Mononucl. 4%
Transitional 3%

Glycogenic negative.

Lumbar Puncture.

Quantity 17.6 c.c.  Pressure 56 c.m.  Fluid escaped in slow drops and was almost thick.

W.B.C.  81,250 per c.mm.
Polymorphs. 96%
Lymphocytes 4%

Film showed very few meningococci indeed, and only intracellular ones.

Albumin - 5½ grammes per litre.

May 12th. Patient very much worse. Cyanosis has increased. Head retraction has appeared and become pronounced. Does not seem to be able to swallow at all.

Restlessness has passed off and patient seems a little more conscious.

Died at 1.45 a.m. of 13th.
CASE XXXI.

H.E., aged 6 years.

History.

Yesterday, May 11th, patient complained of severe headache and vomited. He was put to bed. On Sunday, 12th (to-day) he was much worse; he was shivering and his neck was sore. A doctor was then sent for, who sent him on to us at once.

This is the brother of Case XXVIII. that was admitted three days ago. The house has been disinfected and the inmates had their noses syringed, but patient always took care not to be found when the syringing was done.

On admission.

Patient seems to have very little the matter with him except the temperature of 100.8°.

He is well developed and well nourished.

Perfectly conscious: no restlessness: no head retraction and no rigidity of neck: no rash: no herpes. He has a slight headache.

Kernig's sign is present to small degree.

Abdominal reflex present.

He takes his food well and seems quite contented.

So far he exactly resembles Case XXI.
May 13th. Patient has become very much worse. He was very restless over night, and got out of bed twice: was crying out frequently, complaining of his headache. Has been vomiting. The headache is frontal.

There is no retraction, but the neck is now rigid. There is no rash, but slight herpes on the upper lip and inside the left nostril.

Is no tache and no smell. No coldness. He is still quite conscious and can tell all about the onset of the illness, but every now and again he starts up as if dazed and does not know where he is.

He lies on his back with the knees drawn up.

There is a scar at the base of right lung, the result of an operation for empyema following pneumonia three years ago.


Nervous System.

Motor and sensory unaffected.

Reflexes - Knee jerks present and normal.

Kernig's sign well marked to-day.

Abdominal and epigastric reflexes absent.

Plantar - no response. Skin too thick to feel.

Eyes - Pupils contracted: react to light.

Hearing is unaffected.
Alimentary.

Tongue moist and covered with brown fur.
No tenderness of abdomen.
No enlargement of spleen or liver.
Stomach normal.
Patient is constipated.

Circulatory.

Face is flushed. There is no cyanosis.
Pulse full and strong. Cardiac sounds all closed.

Respiratory.

Has a sore throat and is husky. Congestion of throat and tonsils moderately large.
Impaired resonance and breath sounds are distant at right base. No accompaniments.
Left side - Nothing of note.

Urine.

Acid. Specific gravity 1042. Deposit of urates.
No albumin: no sugar. Diazoe negative.

Blood.

R.B.C. 4,630,000 per c.mm.
W.B.C. 30,375 " "
Hb. 108%

Lumbar Puncture.

Pressure 45 c.m. Quantity 9.5 c.c. Fluid was slightly turbid.
Wh.B.C. 10,000 per c.mm.
Polymorphs. 79%
Lymphocytes 3%
Degenerates 18%

Albumin 0.5 grammes per litre.
Film showed no meningococci.

May 15th. Patient is still getting worse; is more unconscious and more restless.
The head is more retracted.
The herpes is spreading and the upper lip is becoming very sore by it.
He doses a great deal.
Pulse inclined to be irregular.

June 11th. For a week after last note patient steadily improved, but after that he steadily got worse. The chief complaint being a steady increase of headache and backache with increasing occurrence of vomiting. Emaciation is getting very marked. Considering this due to increasing intracranial pressure lumbar puncture was again resorted to. The headache at the time was very much increased but afterwards gradually subsided and he then had a very much better day than he has had for the past fortnight.

The result of the lumbar puncture was as follows:
Pressure 28 c.m. Quantity 11.5 c.c. Fluid was slightly turbid.

Wh.B.C. 9,900 per c.mm.
Polymorphs. 96%
Lymphocytes 2%
Transitional 2%
Albumin 3.5 grammes per litre.

Film showed many intracellular diplococci; none extracellular.

From this date patient made an uninterrupted recovery till date of discharge, July 23rd.

When seen a month after discharge he was in splendid health, though still a little thin and weak.
CASE XXXIII.

L.M., aged 8 years.

History.

On 6th patient complained of severe headache which got much worse on 7th, when he vomited and complained of a backache.

His throat was also sore.

He felt sore all over and did not like being touched.

The father works in the docks.

The house is overcrowded, there being four adults and five children in two rooms. They were, however, clean and fairly well ventilated.

On admission:

Patient is very ill.

He is a badly nourished, fairly well developed child. He is semiconscious.

He is lying very quiet, no restlessness at all: is very pale.

Head retraction is moderate, but rigidity of neck is absolute. No special tenderness at back of neck.

Skin - There is no rash, and no herpes.

Slight cyanotic flush on cheeks.

Tache well marked.
No smell: no coldness.

Nervous System.
Motor power is weak.
Sensory - There is general hyperaesthesia.
Reflexes - No tendon reflexes obtained.

Abdominal and epigastric absent. Plantar - present with extension.
Kernig's sign is well marked.
Eyes - Pupils dilated, equal and react to light.
Hearing unimpaired.

Alimentary.
Tongue covered with brownish white fur. The breath is foul and has an odour of salol.
The abdomen is retracted and the superficial veins on the right side are dilated.
No enlargement of spleen or liver.
Stomach is normal.

Respiratory.
No soreness of the nose. The throat is red and congested. There is impaired resonance at the left base. All over left side, both anteriorly and posteriorly, is very loud harsh breathing - no accompaniments.

Cardiac.
Pulse full and regular. Over apex is slight roughening of the first sound.
Urine.

Acid. Specific gravity 1.034. Deposit of urates. No albumin or sugar. Diazo is negative.

Blood.

R.B.C. 5,900,000
W.B.C. 14,375
Hb. 110%

Differential leucocyte count:

Polymorphs. 68%
Lymphocytes 21%
Large Mononucl. 6%
Transitional 3%
Eosinophiles 2%

Lumbar Puncture.

Quantity 13 c.c. Pressure 20 c.m. Fluid very faintly turbid.

Wh.B.C. 780 per c.mm.

The film shows no meningococci.

A proper differentiation of the leucocytes was not possible owing to the fact that quite over half were disintegrated. Those that were still to be distinguished gave a count of

Polymorphs. 36%
Lymphocytes 64%

Albumin - .75 grammes per litre.

May 14th. From 2 p.m. for 1½ hours, the patient was in a convulsion.

The left side of the mouth was noticed to twitch and this continued: the left eye then began to blink.
This was followed by clonic contractions in left arm, left side of abdomen, and then the left leg. They remained limited to these areas for some time and then the right side of the abdomen was affected, but no other part on the right side.

Patient gradually failed till death on June 12th.

Post Mortem, June 14th.

Nutrition - emaciation extreme. Post mortem rigidity passed off.

Chest.

Heart. Pericardium has about 2 drams clear fluid in it. Right side of heart not distended. Nothing else of note.


Abdomen.

No peritonitis. Mesenteric glands not enlarged to any extent. Blood vessels of mesentery are unduly engorged with blood.

Intestines show irregular patches of inflammation, more marked towards lower end of ilium.
Peyer's patches are hyperaemic and raised. Solitary glands very prominent.

Large intestine shows slight patches of inflammation, not to same extent as in small intestine.

There were marked post mortem intussusceptions.

Liver dark, firm. Nothing of note.

Kidneys - nil.

Suprarenals - nil.

Spleen very small.

Cord.

Shows nothing abnormal.

Brain. Membranes were adherent. At base of brain there was a mass of pus size of halfpenny, just posterior to optic chiasm. Thin film of pus on vertex limited. Brain substance very soft.

Marked hydrocephalus.
CASE XXXIV.

Q.L., aged 7 years.

History.

Yesterday (15th) patient got a knock on the back of his head. The same night he had severe headache. He vomited towards midnight. By 2 a.m. he was unconscious and delirious. Father is a dock labourer and the whole family are dirty and drink. There are four adults and three children in two rooms.

On admission:

Development and nutrition are poor.

Is quite unconscious.

Restlessness is extreme.

He lies on the right side, constantly moaning, and the legs are drawn up.

The head is held quite straight; no retraction, but the neck is rigid.

There is no rash: no herpes: no coldness: no smell.

Temperature 98°. Pulse 120. Respiration 28.

Nervous System.

Motor - general weakness.

Sensory - no hyperaesthesia.

Eyes - Pupils contracted, equal: react to light.

Alimentary.
Tongue is dry, with brown fur. Abdomen is not tender. No enlargement of spleen or liver. Stomach normal. Has had slight diarrhoea.

Respiratory.
Nothing of note.

Circulatory.
Pulse is full and regular. Cardiac sounds closed.

Urine.

Blood.
No count.

Lumbar Puncture.
Quantity 2 c.c. Pressure 77 c.m. Fluid flowed in very slow drops: was very turbid.
Wh. B.C. 97,344 per c.mm.
Polymorphs. 91%
Lymphocytes 7%
Large Mononucl. 2%

Film showed meningococci, both intra- and extracellular. Intracellular = .01 per cell.

Patient, after increasing coma and gradual subsidence of restlessness, died on 18th.

No Post Mortem.
CASE XXXV.

E.G., aged 5 years.

History.

Patient had a fall and struck his head on Tuesday, 21st. On Wednesday, 22nd, patient vomited and complained of a bad headache, and was shivering. One hour afterwards he had a fit, epileptiform in character.

He was very restless all that night.

Next day he seemed worse and complained of his back aching.

He has had an abscess at the back of his neck, two years ago, which was cut twice.

The father is a seaman and came back from Antwerp nine days before onset of illness.

There are six adults and three children occupying three rooms, which are clean and well ventilated.

On admission, 24th.

Development fair: nutrition good.

Patient obviously very ill: he lies on his right side, crying out frequently. He is very restless.

Is quite unconscious.

Retraction of head is slight, but neck rigidity is absolute. No tenderness on pressure of neck.
Skin - No rash: no herpes.
Tache is well marked.
Face is pale; cheeks have the cyanotic flush.
The extremities are very cold.

Nervous System.
Motor power good.
Sensory - No hyperaesthesia.
Reflexes - Jaw marked. Abdominal and epigastric absent. The others cannot be tested.

Alimentary.
Lips - colour good.
Tongue dry, parched and raw red: a little furry at the back.
Stomach normal.
Liver and Spleen - no enlargement.
No retraction or distension of abdomen.

Respiratory.
Nose has appearance as if it has been bleeding recently.
Throat red, congested. No enlargement of tonsils.
Nothing of note on percussion, or auscultation.
Circulatory.

Pulse feeble, regular, and fairly strong.
Cardiac sounds closed in all areas.

Urine.

Acid. No deposit. 1028. No albumin.
Albumose present. No sugar or other abnormal constituents.

Blood count.

<table>
<thead>
<tr>
<th>R.B.C.</th>
<th>4,950,000 per c.mm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.B.C.</td>
<td>62,500 &quot; &quot;</td>
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<tr>
<td>Hb.</td>
<td>102%</td>
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Differential count:-

<table>
<thead>
<tr>
<th>Polymorphs</th>
<th>73.5%</th>
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<tr>
<td>Lymphocytes</td>
<td>15%</td>
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<tr>
<td>Large Mononucl.</td>
<td>7.5%</td>
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<tr>
<td>Transitionals</td>
<td>2.0%</td>
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<tr>
<td>Eosinophiles</td>
<td>2.0%</td>
</tr>
</tbody>
</table>

Lumbar Puncture.

Quantity 16 c.c. Pressure 59 c.m. Fluid turbid, flowed out rapidly.

Albumin - 2.25 grammes per litre.

Differential Count. Leucocytes are very much larger than in blood.

<table>
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<tr>
<th>Polymorphs</th>
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<tr>
<td>Large Mononucl.</td>
<td>1%</td>
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<tr>
<td>Lymphocytes</td>
<td>6%</td>
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<tr>
<td>Degenerates</td>
<td>4%</td>
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</table>

Meningococci, both intra- and extra-cellular, present in about equal numbers and in moderate amount.
May 29th. Patient very tremulous and more fretful. Takes food well. Colour improving, but varies frequently.

Head retraction is now extreme.

May 30th. Thick yellow discharge from nose last night.

On examination of chest - Left apex impaired note with some fine rales.

Post. Left side - impaired note throughout, and dull at base. Breath sounds harsh all over, and tubular at base. Rales throughout.

Right side - Fine crepitations over right base.

June 1st. Dullness less on left side.

Tubular breathing replaced by harsh bronchial. Rales coarse and loose all over left side.

Slight conjunctivitis left eye.

Died June 2nd.

Post Mortem.

Chest.

Right Lung. Slightly adherent at the posterior edge; bronchial glands enlarged. Slight evidence of bronchitis in the large bronchi. Upper and middle lobe show no pathological change. Lower lobe shows slight congestion.
Left Lung. No adhesions: bronchial glands enlarged: bronchitis of large bronchi. Upper lobe shows no pathological change. Lower lobe shows capillary bronchitis, but not patches of consolidation.

Heart. Appears healthy.

Thymus. Slightly enlarged.

Abdomen.

No peritonitis: no free fluid.

Mesenteric glands enlarged and congested.

Small intestine - The lower two thirds show patches of marked congestion, and on the inner surface are small areas of congestion, which in some places have produced haemorrhage.

Peyer's patches are slightly prominent.

Large intestine shows nothing abnormal.

Spleen is small, but healthy.

Kidneys present nothing abnormal.

Liver is firm, heavy, with a mottled surface.

On section it shows well marked cirrhosis.

Brain.

Membranes all markedly congested. No flattening of the convolutions.

There is a layer of fine, filmy, purulent material, covering the surface of the brain, which is thicker in the sulci.
On the base of the brain is thick layer of greenish yellow gelatinous purulent matter, reaching from the chiasma onto the surface of the medulla.

The ventricles are dilated. At the posterior corner of the ventricle on each side there is an area of disintegrated brain substance - purulent in appearance - the size of a walnut. It is soft, friable and pale pinkish brown colour.

Cord. Posterior surface covered by a thick gelatinous layer of greenish yellow pus. Anterior surface has a thin film of same covering it.
CASE XXXVI.

H.G., aged 36 years, Byreman.

History.

On Tuesday, 23rd, he felt dizzy and weak and could not wheel his barrow. His head ached and he was sick. He went home and slept all right that night. On Wednesday, he tried to get up, but had to go to bed again as he felt so dizzy and his headache was worse - it was frontal. He felt a pain in his neck when he tried to move his head.

Wednesday he could not sleep, he was so restless and feverish. Further than this he cannot remember.

He was sent to Leith General Hospital on 26th and sent on to us from there.

He works as a byreman.

His house is fairly clean. There are three adults and three children occupying two rooms.

On admission:

Patient is well developed and well nourished - a big, strong, healthy man.

Is semiconscious, answering rationally when spoken to but when left alone us continually rambling and muttering a lot of disconnected nonsense.

His memory is clear to Wednesday.

He is in an exceedingly cheerful and happy frame of mind.
He lies quite quietly, but his hands are never still a moment. He is continually pulling his bedclothes about and throwing out his pillow, etc., but very apologetic and cheerful when told what he is doing.

He lies on his back quite comfortably though. There is a slight degree of head retraction. The neck is quite rigid, but not tender to the touch.

Skin - No rash: there is good crop of herpes on right side of the lower lip. No cyanotic flush.

No smell.

Complains frequently of cold.

Tache well marked.

Temperature 100°. Pulse 88. Respiration 20.

Nervous System.

Motor and Sensory unaffected.

Reflex - Jaw absent; arms and leg, absent; plantar present, flexion; epigastric present; abdominal absent.

Kernig's sign well marked.

Eyes - Pupils small, equal, and react to accommodation and light.

Alimentary.

Breath very foul: tongue foul, coated with brown fur.

Constipated. No abdominal tenderness.
Liver - Dullness extends from upper border of 5th rib to costal margin.
Stomach not enlarged.
Spleen not enlarged.

Respiratory.
No congestion of throat.
Resonance good throughout.
No alteration in breath sounds.

Circulatory.
Sounds closed all areas.

May 2nd. Neck rigidity is absolute. Tenderness at back of neck on pressure. Head retraction slightly increased.
Pupils slightly dilated.
Getting very thin.

Blood Count.

<table>
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<tr>
<th>R.B.C.</th>
<th>4,950,000 per c.mm.</th>
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</thead>
<tbody>
<tr>
<td>W.B.C.</td>
<td>19,375 &quot;</td>
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<tr>
<td>Hb.</td>
<td>120%</td>
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</tbody>
</table>

Differential count:-

| Polymorphs. | 85.5% |
| Lymphocytes | 8.5%  |
| Transitionals | 3.5% |
| Large Mononuclears | 2.0% |
| Eosinophiles | .5%  |

Urine.

Lumbar Puncture.

Fifteen c.c. withdrawn. Pressure 33 c.m.
Albumin - 2 grammes per litre.
Wh.B.C. 5,625.

Fluid very turbid. Cells so degenerated that cannot do differential count. A few meningococci present both intra- and extra-cellular.

May 13th. Abdominal reflex returned.
Patient getting very emaciated.
Neck still rigid.

May 14th. Patient continually sleeping, both night and day; sleeps over food; does not seem to be able to keep awake.

May 31st. Slight conjunctivitis left eye.

Patient continued to get progressively weaker and emaciated. Died on July 6th.
CASE XXXVII.

E.W., aged 8 years.

History.

On April 23rd patient felt cold and was shivering. She later complained of a headache and pain in the back and vomited, but until May 1st she was quite bright and cheerful and was running about as usual. On May 1st she had a stiff neck, and that evening when she went to bed she again complained of her head as aching and said her throat was sore. Later that night she was quite delirious and shouting out with the pain of her head and back. She got very much worse, but no doctor was called in until yesterday.

She was admitted May 8th.

Family history is good. One brother died of pneumonia.

The father is a dock labourer and worked on the Hamburg steamers. An uncle died in Hamburg of cerebrospinal recently, and the father went over to see him.

A playmate of patient died earlier in the year of cerebrospinal. A case of cerebrospinal had previously occurred with fatal result in the same stair.

The house was moderately roomy, there being two adults and two children in two fairly clean rooms.
On admission:

Patient is a well developed, very poorly nourished girl.

She is quite conscious, but obviously very ill.

She lies quietly on her back, constantly whining, but there is no restlessness. The legs are drawn up. There is very little head retraction, but the neck rigidity is very marked.

Skin - No rash: no herpes: but there are marks of recent herpes on both upper and lower lip.

Has the cyanotic flush very marked.

Tache poorly marked.

There is no smell.

She resents being handled in any way, not from hyperaesthesia, but from objection to being disturbed.

Temperature 100.4°. Pulse 104. Respiration 28.

Nervous System.

Motor and sensory unaffected.

Reflexes - Abdominal and epigastric both absent.

Plantar active, with flexion. Knee jerks are both present, but not accentuated. Ankle clonus on both sides is most marked.

Pupils equal, contracted: react to accommodation and light.

Hearing is unaffected.
Respiratory.

Throat is congested. Nothing of note on examination of the chest.

Circulatory.

Pulse is regular, but of poor volume. Cardiac sounds are closed in all areas.

Alimentary.

Tongue covered with slight brown fur. Takes food well and complains of hunger.

Liver dullness - 6th rib to costal margin.

Spleen - No enlargement.

There is slight tenderness in right iliac fossa.

Urine.

Acid. Specific gravity 1035. Deposit of urates. No abnormal constituents. Diaz o reaction negative. Urea - 6.5 grains per oz.

Blood.

R.B.C. 4,600,000 per c.mm.

W.B.C. 22,460 " "

Hb. 90%

Differential leucocyte count:-

Polymorphs. 70%

Lymphocytes 28%

Eosinophiles 2%

May 9th. Patient been restless and crying out. Head and body kept very rigid, but legs and arms are moved about.

No incontinence.
Complained of abdominal pain.


May 13th. Patient quite conscious. Resents any handling, however slight. Feels cold very much. Abdominal tenderness very marked. Abdominal and epigastric reflexes still absent. Legs are drawn up and any attempt to straighten them is resented very much.

Incontinence of urine present.

May 20th. Child has been progressively emaciating. The general hyperaesthesia is getting very pronounced.

Abdominal and epigastric reflexes absent. Flushes very quickly at times. Incontinence continues.

May 30th. Patient been screaming out all afternoon and imagining all sorts of vagaries, wandering. Out of bed over night of 30th to meet some one.

June 11th. Since May 28th temperature remained between 97 and 98, and patient made uninterrupted recovery.
CASE XXXVIII.

P.R., aged 20 years, engineer.

History.

A month ago patient had all his teeth extracted, since when he has had frequent headaches. On Saturday the 7th he thought he caught a chill. On Monday 9th he complained of a headache and wanted to lie down. He vomited and then went to bed. All through the night he was retching, felt feverish, and his head ached severely.

His neck became sore and stiff on 10th.

By afternoon of 10th he was unconscious, and restlessness set in.

Admitted on 11th.

Patient is an engineer, working in the docks.

His family history is good.

His home conditions are fair. Six adults live in three rooms, which are clean and well ventilated.

On admission:

Patient well developed and well nourished. Lies on left side and is quite unconscious.

Restlessness is very marked - tossing about and pulling off bed clothes, etc.

No head retraction.

Neck does not feel rigid, but cannot get head to
come forward; he can move head from side to side slightly, and further back than it is, but not forward.

Skin - Over chest on right side is a pinkish yellow rash, which is not raised, and which does not disappear on pressure. Some spots are discrete, whilst others run together forming a blotchy appearance. On back, both sides, there is a similar eruption, limited to upper half of back.

There is no herpes. No coldness.

No tenderness of neck.

There is slight cerebrospinal smell.

**Nervous System.**

Motor power is very good.

Sensory - cannot test.

Special senses - Pupils small and equal. React to light.

Reflexes - Jaw absent; arm absent; knee jerks slight. Others absent. Plantar present. Abdominal and epigastric absent.

Kernig's sign well marked.

**Respiratory.**

Nose unaffected. Throat is red and contains much thick, sticky mucus.

Chest - Nothing of note.

**Alimentary.**

Tongue dry, dirty brown.
Lips dry, cracked, and covered with dry mucus.

Breath foul, with acid smell in it.

Liver - No enlargement.

Spleen - Anterior border comes to anterior axial line.

Circulatory.

Apex $\frac{1}{2}$" outside L.M.L.

Sounds closed all areas.

Pulse full, strong and regular.

Urinary.

Acid. Specific gravity 1032. Urea 7 grs. per oz. Albumin - a trace: no sugar: no deposit. Diazo negative.

Lumbar Puncture, 11th June.

Pressure 26½". Quantity 26 c.c.; much more could have been collected without discomfort to patient. Fluid flowed rapidly and was very turbid.

W.B.C. 45,000 per c.mm.

Albumin - 5 grammes per litre.

Meningococci - a few intra and extra cellular present.

<table>
<thead>
<tr>
<th>Polymorphs</th>
<th>92%</th>
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<tbody>
<tr>
<td>Lymphocytes</td>
<td>5%</td>
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<tr>
<td>Large MonoNucl.</td>
<td>1%</td>
</tr>
<tr>
<td>Transitionals</td>
<td>2%</td>
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</tbody>
</table>

Blood Count. 12th June.

<table>
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<th>R.B.C.</th>
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<tbody>
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<td>W.B.C.</td>
<td>15,250</td>
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<tr>
<td>Hb.</td>
<td>120% +</td>
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Differential Count:

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<th>Cell Type</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Polymorphs</td>
<td>84%</td>
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<tr>
<td>Lymphocytes</td>
<td>13.5%</td>
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<tr>
<td>Transitionals</td>
<td>2.5%</td>
</tr>
<tr>
<td>No others</td>
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</table>

June 12th. Patient been restless all night, but is a little quieter this morning and decidedly more sensible only has to be shaken and roused before getting anything out of him.

Early herpes appearing on chin.

June 13th. Herpes well out. Restlessness less marked. Still rather hazy about where he is and has to be roused thoroughly before can get him to answer.

June 30th. Patient gradually got weaker, with progressive emaciation, and died to-day.
CASE XXXIX.

W.C., aged 4 years.

History.

On Monday 9th patient was sick in the forenoon and said his hands were sore. In the afternoon he got much worse, and was sick frequently: he was very thirsty, but each drink he was given he vomited at once. That night he was very restless and screaming out, and became unconscious. His head became retracted over night and neck was stiff.

Patient has had measles followed by whooping cough, which he was just convalescing from.

The father is a biscuit baker and family history is good. There were two adults and four children in two moderately clean rooms.

On admission:

Patient is badly nourished and poorly developed. He is quite unconscious: is very restless and resents anything being done for him.

The head retraction is extreme. No special tenderness of neck.

Skin - No herpes: no rash. No cyanotic flush.

No smell: no coldness.

Temperature 99°. Pulse 120. Respiration 32.
<table>
<thead>
<tr>
<th>Date</th>
<th>Day of Dis.</th>
<th>Time</th>
<th>Temperature (Fahrenheit)</th>
<th>Pulse</th>
<th>Resp</th>
<th>Bowels</th>
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</table>
Nervous System.

Motor power good.
Marked general hyperaesthesia.
Special Sense. Eyes - pupils dilated, equal.
Reflexes - None present.
Kernig's sign present.

Alimentary.

Tongue moist; has slight white fur.
Liver and spleen - no enlargement.

Respiratory.

Has short, loose cough.
Harsh breathing on left side anteriorly.
Throat slightly red.

Circulatory.

Pulse regular, strong and full.
Sounds closed all areas.

Lumbar Puncture 11th June.

Pressure 58 c.m. Quantity 16 c.c. Fluid turid, flowed in rapid drops.

W.B.C. 21,000 per c.mm.
Polymorphs. 92%
Lymphocytes 5%
Large Mononucl. 3%
Meningococci I. + but a few.

Albumin - 3.5 grammes per litre.

Blood - June 12th.

R.B.C. 4,600,000
W.B.C. 24,375
Hb. 11.2%
Differential count:

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<tbody>
<tr>
<td>Polymorphs</td>
<td>76%</td>
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<td>Lymphocytes</td>
<td>18%</td>
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<tr>
<td>Transitionals</td>
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<tr>
<td>Large Mononucl.</td>
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</table>

June 13th. Head retraction more marked. Still shouting out a good deal.

June 16th. Patient more sensible: answers when spoken to; knew his people. Head less retracted.

Patient lingered on till death August 3rd.
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**Diagnosis:** Cerebro Spinal Meningitis

**Result:** Death
A.W., aged 12 months.

History.

On the 8th patient got ill suddenly. He vomited and soon after apparently had pain in his head and the mother noticed that night that the neck was stiff.

On 9th patient was much worse, and the head was then noticed to be retracted. He was so much worse that she took him to the Sick Children's Hospital, from which he was sent on to us.

His father is a dock labourer and the house is dirty and overcrowded. There are two rooms for three adults and five children.

On admission:

Patient is a very badly nourished, badly developed child: a typical rickety child. He is apparently unconscious, and lies quite quiet, there being no restlessness.

The head is retracted to an extreme point, and the neck is rigidly stiff and tender to the touch.

The skin is dry and loose, hanging in folds.

No rash: no herpes.

No smell.

No tache.

He is very cold and it is difficult to keep him warm.
Nervous System.

Reflexes - knee jerks marked, especially on left side. Others cannot test.

Kernig's sign absent.

Eyes - Pupils equal, moderate in size; react to light.

Abdominal and epigastric reflexes absent.

Plantar - Babinski.

Circulatory.

Nil.

Respiratory.

Right side - apex in front, note impaired.

Breath sounds high pitched, tubular, with coarse rales.

Vocal resonance increased.

Base, posterior - Note not impaired, but harsh breathing with rales and increased vocal resonance.

Alimentary.

Tongue is red, raw and dry, with enlarged papillae; tongue dirty, white furred, and sticky mucus in throat.

Liver and spleen - no enlargement.

Lumbar Puncture.

Pressure 23 c.m. Quantity 3½ c.c. Fluid stained with blood.

W.B.C. unreliable owing to blood.
Film shows only blood cells and few lymphocytes, such as would normally be in amount of blood.

Albumin - Too small quantity for estimation.

**Blood.**

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<tr>
<td>R.B.C.</td>
<td>6,250,000</td>
<td>per c.mm.</td>
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<td>W.B.C.</td>
<td>27,575</td>
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<td>Hb.</td>
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Differential count:

- Polymorphs: 77.5%
- Lymphocytes: 19.5%
- Transitionals: 2.0%
- Large Mononucl: 1.0%

**June 13th.** Head more retracted.

Eyes, nose and ears discharging.

Hands clenched.

**June 14th.** Cheyne Stokes.

**June 15th.** Cheyne Stokes more marked. Eyes swimming in pus. Pulse very feeble.

Abdomen is distended and skin shows some purpura.

**June 16th.** Died.