SPHENOIDAL EMPYEMA AND EPIDEMIC CEREBROSPINAL FEVER.¹

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There are four varieties of the clinical course taken by cerebrospinal fever:

Firstly, there is the case which, commencing as an acute illness, may then go on to recovery or may result in death.

There is, secondly, the type of case that, after having shown considerable improvement from the acute stage, suffers from a recrudescence, both in general symptoms and in the condition of the cerebrospinal fluid. This usually occurs from the fifth to twelfth day, and may subsequently lead to recovery or death.

Thirdly, there is the type of case that, after convalescence from the acute stage, still retains a tendency to suffer from malaise, headaches and vomiting, which gradually become more pronounced. The patient becomes extremely emaciated and death occurs from hydrocephalus.

Fourthly, there is the case that, having entirely recovered from the preliminary attack, months later has a relapse which may prove fatal.

Thirty-four post-mortems were made in the Netley district between 1915 and 1919, on cases of cerebrospinal fever, dying as a result of one of these four types of the disease. These are all grouped in the ensuing table under three of these types:

(A) Those dying during the acute illness—twenty-one cases.

(B) Those dying during a recrudescence—six cases.

(C) Those dying from hydrocephalus during convalescence—seven cases.

One case only (22 Mn.) died as a result of a relapse occurring months after the preliminary attack, but as he suffered from an apparently typical attack of the disease with a recrudescence he is grouped under heading B for convenience.

Of these thirty-four deaths, only eleven occurred within the first week of illness. In this observation we differ from Rolleston [1] who states that "more than half the fatal cases occur during the first week of the disease." A possible explanation of this difference is that at Netley we were fortunately able to insist that every case in which the least suspicion of cerebrospinal fever was entertained should be immediately admitted to the isolation hospital. On admission lumbar puncture was performed as a routine

¹ Read at the Pathological Section of the Royal Society of Medicine, on November 4, 1919.
within a few minutes of arrival, antimeningococcic serum being administered there and then to cases showing turbid cerebrospinal fluid. A bacteriological diagnosis was only waited for when there was middle ear disease or a head wound. This very early administration of serum, often within an hour or two only of the suspicion of cerebrospinal fever being present, probably saved many cases who would otherwise have died during the first week.

### A.—Acute Cases.

<table>
<thead>
<tr>
<th>Name</th>
<th>Number of days from onset to death</th>
<th>C.S.F. type</th>
<th>Nasal type</th>
<th>Grib. ethmoid</th>
<th>Sphenoidal sinus</th>
<th>Sphenoidal bone</th>
<th>Pituitary fossa</th>
<th>Middle ear</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Em-pyema</td>
<td>Meningo-cocci</td>
<td>Em-pyema</td>
<td>Meningo-cocci</td>
</tr>
<tr>
<td>(1)</td>
<td>Sh.</td>
<td>1</td>
<td>N.O.</td>
<td>Nil</td>
<td>Serous effusion</td>
<td>Yes</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(2)</td>
<td>Wh.</td>
<td>1</td>
<td>II</td>
<td>N.O.</td>
<td>Not found</td>
<td>Yes</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(3)</td>
<td>Cl.</td>
<td>2</td>
<td>II</td>
<td>N.O.</td>
<td>II</td>
<td>Nil</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(5)</td>
<td>Ha.</td>
<td>3</td>
<td>Pos.</td>
<td>Pos.</td>
<td>Not found</td>
<td>II</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(6)</td>
<td>Mo.</td>
<td>3</td>
<td>Pos.</td>
<td>Pos.</td>
<td>Not found</td>
<td>II</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(7)</td>
<td>Wa.</td>
<td>3</td>
<td>II</td>
<td>N.O.</td>
<td>Serous effusion</td>
<td>Yes</td>
<td>Meningo-cocci</td>
<td>Nil</td>
</tr>
<tr>
<td>(8)</td>
<td>Es.</td>
<td>3</td>
<td>I</td>
<td>I</td>
<td>Meningo-cocci</td>
<td>Yes</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(9)</td>
<td>Va.</td>
<td>4</td>
<td>I</td>
<td>N.O.</td>
<td>Meningo-cocci</td>
<td>Nil</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(10)</td>
<td>Hr.</td>
<td>5</td>
<td>Pos.</td>
<td>N.O.</td>
<td>Not found</td>
<td>Not found</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(11)</td>
<td>Ch.</td>
<td>7</td>
<td></td>
<td>N.O.</td>
<td>Not found</td>
<td>Not found</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(12)</td>
<td>An.</td>
<td>9</td>
<td>I</td>
<td>I</td>
<td>Not found</td>
<td>Yes</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(13)</td>
<td>Ho.</td>
<td>10</td>
<td>Pos.</td>
<td>Pos.</td>
<td>Not found</td>
<td>Yes</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(14)</td>
<td>Po.</td>
<td>11</td>
<td>I</td>
<td>N.O.</td>
<td>Not found</td>
<td>Nil</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(15)</td>
<td>Cr.</td>
<td>16</td>
<td>II</td>
<td>II</td>
<td>Meningo-cocci</td>
<td>Yes</td>
<td>II</td>
<td>Nil</td>
</tr>
<tr>
<td>(16)</td>
<td>He.</td>
<td>19</td>
<td>Pos.</td>
<td>N.O.</td>
<td>Meningo-cocci</td>
<td>Meningo-cocci</td>
<td>Meningo-cocci</td>
<td>Nil</td>
</tr>
<tr>
<td>(17)</td>
<td>Su.</td>
<td>27</td>
<td>III</td>
<td>N.O.</td>
<td>Nil</td>
<td>Not found</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(19)</td>
<td>Ti.</td>
<td>40</td>
<td>Pos.</td>
<td>N.O.</td>
<td>Not found</td>
<td>Not found</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(20)</td>
<td>Th.</td>
<td>47</td>
<td>Pos.</td>
<td>Pos.</td>
<td>Not found</td>
<td>Not found</td>
<td>Not found</td>
<td>Nil</td>
</tr>
<tr>
<td>(21)</td>
<td>Wt.</td>
<td>68</td>
<td>Pos.</td>
<td>N.O.</td>
<td>Meningo-cocci</td>
<td>Yes.</td>
<td>Not found</td>
<td>Nil</td>
</tr>
</tbody>
</table>

N.O. = culture not obtained.

Roman numerals denote Gordon's four types of meningococcus as proved by serological tests.

Pos. = meningococcus grown, but serological tests not performed.

Meningococci = organisms having the morphological and staining characteristics of the meningococcus seen, but not grown.
**B.—Cases with known Recrudescences.**

<table>
<thead>
<tr>
<th>Name</th>
<th>Number of days from onset to—</th>
<th>C.S.F. type</th>
<th>Nasal type</th>
<th>Sphenoidal sinus</th>
<th>Pituitary fossa</th>
<th>Middle ear</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Recrudescence</td>
<td>Death</td>
<td>Crib. ethmoid</td>
<td>Empyema</td>
<td>Meningococci</td>
<td>Empyema</td>
</tr>
<tr>
<td>(22) Mn.</td>
<td>8</td>
<td>8</td>
<td>II</td>
<td>II</td>
<td>Nil</td>
<td>Yes</td>
</tr>
<tr>
<td>(23) Ws.</td>
<td>5</td>
<td>9</td>
<td>I</td>
<td>I</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>(24) Pu.</td>
<td>9</td>
<td>14</td>
<td>I</td>
<td>I</td>
<td>Nil</td>
<td>I</td>
</tr>
<tr>
<td>(25) Co.</td>
<td>12</td>
<td>16</td>
<td>III</td>
<td>I or III</td>
<td>Nil</td>
<td>—</td>
</tr>
<tr>
<td>(26) Bo.</td>
<td>42</td>
<td>48</td>
<td>II</td>
<td>N.O.</td>
<td>Normal</td>
<td>—</td>
</tr>
</tbody>
</table>

**C.—Convalescent Cases dying from Hydrocephalus.**

<table>
<thead>
<tr>
<th>Name</th>
<th>Number of days</th>
<th>C.S.F. type</th>
<th>Nasal type</th>
<th>Pituitary fossa</th>
<th>Middle ear</th>
</tr>
</thead>
<tbody>
<tr>
<td>(28) Sp.</td>
<td>87</td>
<td>Pos.</td>
<td>II</td>
<td>Nil</td>
<td>Yes</td>
</tr>
<tr>
<td>(29) Ck.</td>
<td>90</td>
<td>Pos.</td>
<td>Pos.</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(30) Fl.</td>
<td>93</td>
<td>II</td>
<td>II</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(31) Tr.</td>
<td>109</td>
<td>Pos.</td>
<td>N.O.</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(32) Mu.</td>
<td>133</td>
<td>Pos.</td>
<td>I</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(33) Co.</td>
<td>147</td>
<td>Pos.</td>
<td>II</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>(34) Br.</td>
<td>190</td>
<td>I</td>
<td>I</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

N.O. = Culture not obtained.

Roman numerals denote Gordon's four types of meningococcus as proved by serological tests.
Pos. = Meningococcus grown, but serological test not performed.
Meningococci = organisms having the morphological and staining characteristics of the meningococcus seen, but not grown.

In a previous paper [2] it was shown that of thirty cases in which it was possible to obtain cultures from both the nose and the cerebrospinal fluid, in thirty-five the types of the meningococci in the two sites were identical, as proved by both agglutination and saturation tests. The irregularity shown by the remaining case has been discussed.

In no case was any inflammatory process discoverable about the cribiform plate of the ethmoid by naked-eye examination.

The contents of the pituitary fossa showed inflammation in every case. The middle ear was seen to contain pus in one case, and there was congestion of the bone, but no pus, in another case.

There was an empyema of the sphenoidal sinus in:

(a) Acute case deaths—twenty out of twenty-one cases.
(b) Recrudescence deaths—five out of six cases.
(c) Hydrocephalus deaths—seven out of seven cases.

An empyema of the sphenoidal sinus is taken to mean a cavity filled with pus, mucous-pus containing fresh and autolysed pus cells or glairy fluid showing pus cells, and the mucous membrane of the sinus showing congestion. Thus in thirty-four post-mortems, an empyema of the sphenoidal

...
sinus was present in thirty-two cases. In the pus from the sphenoidal sinus:

Meningococci were proved serologically in eight cases.
Meningococci were cultured but were not tested serologically in two cases.
Meningococci were seen but not grown in ten cases.
No meningococci were seen or grown in twelve cases.

Inflammation of the sphenoid bone over the empyematous sphenoidal sinus was not found at all in recrudescence and hydrocephalus deaths, but was found in seven cases of deaths from the acute illness. In these seven cases the meningococcus was only found three times, and the type of the meningococcus was only able to be proved once, but was in this case the same as that found in the nose and cerebrospinal fluid.

The association of sphenoidal sinus inflammation with cerebrospinal fever was first noticed by Westenhoeffer [3], who only found it to occur in one-third of his twenty-nine necropsies. Other workers have also reported cases showing empyema of the sphenoidal sinus, but have generally considered that this condition is not the cause of the disease, either because they have been unable to find an empyema in all cases, or because they have been unable to trace the meningococcus in the walls of the sphenoidal sinus or in the body of the sphenoid bone. Very great improvements have recently been made by Mervyn Gordon in the culture medium for the propagation of the meningococcus, so that a meningococcus can now, under suitable circumstances, be made to grow as rapidly and luxuriantly as a Bacillus coli. These improvements were not available for most of the workers in this connexion. In spite of these advantages the meningococcus was not demonstrated in the sphenoidal empyema in twelve of these cases. This was possibly due to the length of time that elapsed between death and the necropsy in many cases, and also to the chilling of the plates during transport from a distant camp to the laboratory in very cold weather. The majority of deaths occurred during cold weather, and meningococci are difficult to cultivate in primary culture unless precautions against chilling are taken.

Thus in 34 deaths from cerebrospinal fever, empyema of the sphenoidal sinus was found 32 times, meningococci were found 20 times in the sinus and 3 times in the sphenoid bone. In no other diseases were empyemata of the sphenoid sinus observed, except during the epidemic of “Spanish influenza,” when empyemata of the sphenoidal sinus were frequently found.

The relationship between an empyema of the sphenoidal sinus and cerebrospinal fever may now be considered. The primary site of a meningococcus infection in the human body is undoubtedly the nasopharynx. The meningococci has been demonstrated in this position not only early in the acute attack, but also in the incubation period [4], in carriers who never develop meningeal symptoms, in meningococcal pneumonia, etc.

The infection is propagated from person to person, as is a common cold,
from nose to nose. The frequency of the carrier condition and the relative infrequency of the disease is noteworthy. It is probable that the whole population of England has been a carrier at some time or other during the last five years, and yet the total number of cases is relatively small.

At the time when contacts to cases of cerebrospinal fever were examined for the carrier condition, it was always noticed that the people under examination were suffering from coughs and “colds.” Many of the contacts gave pure cultures of the meningococcus in the nasopharynx. This suggested that the “colds” were in some cases due to the meningococcus. Also several carriers, when first isolated, suffered from a profuse, watery nasal discharge, from which only the meningococcus could be isolated. Chronic carriers nearly always had a gluey nasal discharge, which was apparent on examination of a nasal swab. It thus appears that the meningococcus can give rise to nasal catarrh. It seems probable that this is the natural disease produced by this organism, and it is only when some other factor comes into play that meningitis or septicemia results.

This other factor does not seem always to be a rise in virulence of the meningococcus, for of the camp epidemics about Southampton and Winchester during the war it was only in one epidemic that all the cases of meningitis were due to one serological type of meningococcus. Usually two or three serological types of meningococcus were recognized in the cases of one epidemic, the carriers found in the camp corresponding with the serological types of meningococcus in the cases. Thus in the Winchester epidemic of the spring of 1917 there were 8 cases, 3 due to Gordon’s Type I coccus, 2 due to Gordon’s Type II, 2 due to Type IV and 1 in which the type was not proved. The carriers found were all infected with one of these three types of meningococcus, no Type III carriers were found. On another occasion two cases occurred in one ward in a hospital within four days of one another. If serological tests had not been performed, it would have been felt certain that either one case infected the other, or that both were infected from the same source. This was not so, as the first case was due to Gordon’s Type II, and the second due to Gordon’s Type I or III coccus.

Some epidemics in other parts may have been produced by a particularly virulent type of meningococcus of one serological type, but it is evident that this is by no means a necessary condition for the outbreak of an epidemic.

The frequency of the appearance of a sphenoidal sinus empyema suggests that this might be the determining factor in the onset of the meningitic form of the disease at any rate. The empyema may be the result of an inflammatory reaction to the meningococcus, on the part of the mucous membrane lining the ostia. The fact that the meningococcus was several times isolated in pure culture from the sinus suggests that, for the inflammatory closure of the ostia, a mixed infection is not necessary, although streptococci and staphylococci were also found on occasions to be present. Thus a sphenoidal empyema might be produced by a meningococcal infection of the nasal mucosa of an individual, whose resistance to the
meningococcus was such that the infection resulted in a vigorous inflammatory reaction. Anatomical or pathological peculiarities in this region might also favour the production of an empyema.

In cases dying from cerebrospinal fever, an empyema of the sphenoidal sinus was common, in only two cases in this series (17 Sn. and 26 Bo.), out of thirty-four, was this condition not found. Both these cases died at a considerable number of days from the onset, twenty-seven and forty-eight respectively. Forty-seven completely recovered cases on the other hand were examined by rhinological experts, Dr. Peters and Dr. Bryant, who found that no sphenoidal empyemata were present. Chronic carriers [5] were also carefully examined bacteriologically, and no meningococcic infection of the sinus was found. Thus in dying cases an empyema was frequently found; in completely recovered cases no empyemata were discovered. A series of five cases showing symptoms of hydrocephalus were operated on for drainage of the sphenoidal sinus [6]. Of these cases all showed the presence of a sphenoidal empyema. The meningococcus was only cultivated and proved serologically in one of these cases from the sphenoidal pus obtained at the operation, a typical Gram-negative diplococcus was seen only but not grown in another case. But in two other cases, subsequent to the operation, the meningococcus reappeared in the throat in pure culture, after it had entirely disappeared for some weeks previously. Subsequent to the operations all cases appeared temporarily worse, three made complete recoveries, two died with typical hydrocephalus post mortem. Of the recovered cases one went through a typical relapse after the operation, with reappearance of the meningococcus and pus in the theca, subsequently completely recovering.

From this it would seem that if early and vigorous serum treatment be given, and if the sphenoidal empyema disappear, recovery will probably occur; if, however, the empyema persist the case will, if it recover from the acute illness, either relapse or go on to hydrocephalus.

In order to see if draining the sphenoidal sinus during the acute stage of the illness would accelerate recovery three cases of cerebrospinal fever were operated upon; a sphenoidal empyema was found in each case, but each case died. This discouraged further operative procedure during the acute stage.

The next point under consideration is, how do the meningococci get from the nasal mucosa to the meninges? There are many possible routes:

1. The arachnoid prolongations round the olfactory nerves.
2. The perineural lymphatics.
3. Direct inflammatory extension.
4. The pituitary.
5. The systematic lymphatics.
6. The blood-stream.
7. The middle ear.
(1) The Arachnoid Prolongations round the Olfactory Nerves.

This route is favoured by Netter and Debré and Flexner. Netter and Debré show that dyes, Indian ink and meningococci can get from the cerebrospinal fluid into the nose by this route, but they do not show that the reverse can take place. Their experiments show that the natural direction of the current is from the cerebrospinal fluid to the lymphatics; it is thus difficult to see how the meningococci could progress against the stream, to gain access to the meninges, except by inflammatory extension. In none of this series of post-mortem examinations was there any naked-eye appearance of inflammation about the cribriform plate of the ethmoid.

(2) The Perineural Lymphatics.

In papers by Orr and Rows [7], Teale and Embleton [8], it had been shown that not only dyes and toxins, if injected into a nerve will travel up the perineural lymphatics to the cord, but also particulate material, such as washed spores. These spores can be traced up the perineural lymphatics into the substance of the cord itself. In this way it might be possible for the meningococci to travel up the perineural lymphatics into the brain substance. Cerebrospinal fever does not, however, appear to start as an encephalitis, so although this route would be a satisfactory one for polioencephalitis, it does not seem suitable for cerebrospinal meningitis, especially as it has been shown that neither toxins, dyes nor particulate material, when passing up a perineural lymphatic, get into the cerebrospinal fluid.

(3) Inflammatory Extension.

It has been suggested that meningococci could gain access to the cerebrospinal fluid by a process of inflammatory extension through the bone separating the nose and accessory sinuses from the meninges. In this series of necropsies, there is evidence of direct spread from the sphenoid sinus to the sphenoid bone, and so to the overlying meninges. In seven cases there was inflammation, and the meningococcus was found three times in the bone. In the other cases no evidence, as viewed by the naked eye, of inflammatory extension could be discovered.

(4) The Pituitary Route.

An infection from Luschka’s pharyngeal tonsil along a possibly patent connexion between this and the pituitary gland has been suggested. Luschka’s tonsil is one of the sites in the nose most richly infected with the meningococcus. In this series of necropsies the pituitary body always showed infection, but it is obvious that this would certainly occur anyhow from its intimate relationship with the cerebrospinal fluid.

(5) The Systemic Lymphatics.

The ordinary flow of lymph from the nasal passages is away from the brain, and in connexions between the subarachnoid space and the lymphatic
system the current is away from the meninges. If bacteria are injected into the arachnoid space they rapidly appear in the lymphatics of the head and neck. If however a process occurs such as an empyema of the sphenoidal sinus, the ordinary direction of lymph flow might be reversed, and bacteria be thus forced from the lymphatics of this neighbourhood towards the meninges, without showing any sign of inflammatory extension. In the case of a sphenoidal empyema large numbers of meningococci would get into the lymphatics, and by this means would also get into the blood-stream. It would only be necessary for a sphenoidal empyema to exist for a very short time to produce these effects, possibly only for half an hour. If the sphenoidal empyema discharged at the end of a short time into the nose, and if the meningococci that had reached the meninges were destroyed, a recovery would ensue. If, however, the sphenoidal sinus empyema persisted, and kept on pouring fresh infection into the meninges, the case would probably die.


Many workers have considered that the infection of the meninges occurs by way of the blood-stream, the meningococci gaining access to the blood from the nasopharynx. This suggestion has been based, partly upon the early appearance of the meningococci in the blood, and partly on those cases of meningococcal infection where no meningitis occurs. The blood-stream, however, would receive meningococci very early in the infection of the nasal mucosa, at any rate as soon as an empyema of the sphenoidal sinus occurred. The meningococci would reach the blood-stream from this region via the deep cervical lymphatic trunks, which join the thoracic duct just before it enters the venous system in the neck. Also, as soon as the meningococci gained access to the cerebrospinal fluid, they would appear [9] in the blood-stream, both direct, via the Paccionian bodies, and also by way of the lymphatics, through their junctions with the sub-arachnoid space. The blood then would, from the very earliest stages of the disease, receive repeated and heavy showers of meningococci from the sphenoid region and the cerebrospinal fluid. Thus it can be said that the early finding of the meningococcus in the blood-stream is no indication that the meninges are primarily infected by this route.

In the recent outbreaks of “Spanish influenza” empyema of the sphenoidal sinus was a common finding in the fatal cases, but meningitis was rarely found. It is not necessary then that an empyema should be followed by meningitis. It is thus possible that the meningococci can gain access to the blood-stream from a sphenoidal empyema and so be carried round to the meninges and set up inflammation.

(7) The Middle Ear.

Inflammation of this region was only found in two necropsies in this series. It is unlikely that this is the portal of entry of the meningococcus to the meninges.
Thus it would appear that it is improbable that the meningococci gain access to the meninges through the arachnoid prolongations round the olfactory nerves or by way of the perineural lymphatics. It is probable that the meningococci pass by way of the lymphatics from the sphenoidal sinus direct to the meninges, with or without naked-eye signs of inflammation. It is possible also that they can be conveyed to the meninges by the blood-stream.

The relationship between a persistent sphenoidal empyema and hydrocephalus may now be considered.

In the seven deaths from this condition an empyema was found in each case. Five cases were operated on and pus was found in the sphenoidal sinus each time, two of these cases died and are grouped in with the post-mortem cases. There were thus a total of ten cases of hydrocephalus, and in each case a sphenoidal empyema was found. The cerebrospinal fluid in the ventricles of each of the cases which died contained pus cells. The cerebrospinal fluid taken from the lumbar region was usually quite clear. This suggests that there is a close relationship between empyema of the sphenoidal sinus and the hydrocephalus that develops after an attack of cerebrospinal fever. The hydrocephalus develops as a result of a chronic infection about the foramina of Luschka and Majendie. This chronic infection also appears to exist in the ventricles. The exact path by which the meningococci in the sphenoidal sinus empyema reach the interior of the ventricles is not clear, unless it be by the blood-stream. But there would appear to be some relationship between the two positions inasmuch as the only three cases who recovered from the hydrocephalus condition did so after an operation for drainage of the sphenoidal sinus. It might be argued that these three cases who recovered were not examples of hydrocephalus at all. Against this it is urged that all five cases operated on were advanced cases with typical symptoms, the two that died showed typical necropsies, and all other cases showing these symptoms died with typical necropsies.

The course of events in a case after infection of the nasal mucous membrane with the meningococcus might be as follows:

1. A simple catarrh followed either by recovery or a chronic infection.
2. A vigorous local reaction in the nasal mucosa followed by sphenoidal empyema. This may produce a general blood infection with or without infection of the meninges.

If the sphenoidal empyema disappears, the body, with or without the aid of antiserum, may be able to deal with the meningococci that have gained access to it or death may result. If the sphenoidal empyema persists and very heavy discharges of meningococci are poured into the body death will probably result. If, however, the empyema remains quiescent improvement may occur, but at any time a recrudescence or a relapse may supervene. A quiescent empyema may also keep up a smouldering infection which will lead to hydrocephalus.
Operation on the sphenoidal empyema always produced an increase in general symptoms. During the acute stage of the disease this increase in symptoms appeared to accelerate at any rate a fatal termination. In hydrocephalus cases recovery did occur in three cases, though one case went through a severe relapse.

**Summary.**

1. In thirty-four necropsies on persons dying from cerebrospinal fever empyema of the sphenoidal sinus was found thirty-two times.
2. In ten cases of hydrocephalus following cerebrospinal fever an empyema of the sphenoidal sinus was found in every case.
3. In forty-seven completely recovered cases of cerebrospinal fever no sphenoidal empyemata were present.

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**References.**