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BENIGN FORMS OF OTOGENIC MENINGITIS

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THE object of this paper is to lay before the Section of Otology of the Ninetieth Annual Meeting of the British Medical Association my experience of the benign forms of otogenic meningitis, based upon the observation of 210 patients with meningitis of all forms, caused directly or indirectly by acute or chronic middle-ear suppuration. All have been treated, and nearly all of them operated on, in my department of the Copenhagen Commune Hospital during the last seventeen years, and a post-mortem examination was made upon 133 of the patients who died.

Clinical Material.—The 210 cases were divided according to their age and the number of recoveries in the following way:—

TABLE I.—210 Cases of Otogenic Meningitis.

Under 1 year . . .	6, of whom	1 recovered, i.e. 16.6 per cent.
1 to 4 years . . .	12, "	1 " " 8.3 "
5 " 14 " . . .	61, "	26 " " 42.6 "
15 " 19 " . . .	23, "	8 " " 35.0 "
20 " 29 " . . .	34, "	10 " " 30.0 "
30 " 39 " . . .	27, "	8 " " 30.0 "
40 " 49 " . . .	15, "	3 " " 20.0 "
50 years and over . . .	<u>32,</u> "	<u>2</u> " " <u>6.2</u> "
All ages . . .	<u>210,</u> "	<u>59</u> " " <u>28.0</u> "

It will be seen from Table I. that benign forms of otogenic meningitis appear most frequently in children between five and fourteen years of age, and that the frequency is not only absolute

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but also relative. It will also be observed that children of this age are most frequently attacked by meningitis. This is due to the circumstance that middle-ear suppuration appears most frequently in this decade of life.*

Further, the percentage of recoveries amongst children under five years of age is comparatively small, the absolute frequency of meningitis being, on the other hand, much less than might be expected from the frequency of middle-ear suppuration during this age.*

Finally, it will be seen that the percentage of recoveries decreases in adults with increasing age, being particularly low after fifty years, while the number of patients attacked is much smaller than would be expected.*

The youngest patient treated was ten months old, the oldest seventy-nine years. The youngest patient who recovered was ten months, the oldest fifty-one years.

The benign forms of otogenic meningitis appear principally amongst older children and young individuals under twenty years.

If the present material is divided into groups according to whether meningitis was uncomplicated, or complicated with other intracranial affections, the number of recoveries will be seen from Table II.

TABLE II.—210 *Cases of Meningitis, Solitary and Associated with other Intracranial Complications.*

			Total.	Recoveries.	Percentage.
Meningitis (uncomplicated)	.	.	115	38	33.0
„ + Sinusphlebitis .	.	.	42	16	38.1
„ + Brain Abscess †	.	.	21	4	19.0
„ + Subdural Abscess	.	.	2	0	0.0
„ + „	+	{ Brain Abscess }	2	0	0.0
„ + Sinusphlebitis	+	{ Brain Abscess }	15	0	0.0
„ + „	+	{ Subdural Abscess }	6	0	0.0
„ + „	+	{ Brain Abscess† }	7	1	14.3
			<u>210</u>	<u>59</u>	<u>28.0</u>

Table II. shows that the greatest number of benign cases occur when meningitis is combined with phlebitis of the sigmoid sinus.

* According to a statistical examination in the Oto-laryngological Department of the Commune Hospital. † Including encephalitis.

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The uncomplicated cases of meningitis exhibit a somewhat smaller percentage of recoveries. They were, on an average, operated upon on the third day after the first symptoms of meningitis had appeared. Curiously enough, the patients who died underwent operation, on an average, one day earlier. This is explained by the fact that in cases of this kind the meningeal symptoms develop more rapidly and with greater intensity, causing the patients to be brought earlier to the hospital. It will be seen, therefore, that a statistical proof of the unquestionable value of early operation cannot be given.

In connection with the deductions drawn from Tables I. and II., it may be mentioned that chronic suppuration of the middle-ear was more frequently the cause of benign than of malignant meningitis.

As to the influence of the different forms of bacteria, my material shows that pneumococcal infection plays the principal part in malignant cases, while streptococci are predominant in benign cases. Coli infection is especially malignant.

Finally, experience shows that cases of otogenic meningitis are brought to the hospital at some periods in such unusual numbers, that it gives the impression that epidemic influences are at work, and further, that otogenic meningitis at some periods is more benign than at others.

The benign forms of otogenic meningitis do not, however, appear solely amongst patients who recover. In a series of cases of meningitis complicated with other intracranial lesions, the meningitis disappears before death after having presented distinct objective symptoms.

Cases of this description are very interesting and have been discussed in literature, the negative findings at the post-mortem examinations having given rise to the opinion that phlebitis of the sigmoid sinus, cerebral and subdural abscess can produce meningitic changes in the cerebro-spinal fluid, as obtained by lumbar puncture, without any meningitis being present.

Before going into the details of this important matter, we must answer the question: What is meningitis?

Pathological Definition.—From a pathological aspect, meningitis is generally defined as a diffuse inflammation of the leptomeninges, with the formation of a purulent exudate in the subarachnoid spaces. That this definition does not hold good in all cases, will be seen from the following observations.

Amongst the 133 post-mortem examinations mentioned

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above, 4 did not reveal any macroscopic sign of meningitis, although the patients had exhibited distinct evidence of uncomplicated meningeal inflammation, the lumbar puncture having also given very turbid cerebro-spinal fluid. In 3 of those cases, a microscopic examination was performed, which showed distinct signs of diffuse acute leptomeningitis. These patients died within the first few days after the onset of the symptoms.

There are, further, 3 cases in which the clinical diagnosis of acute diffuse leptomeningitis was made, and where the post-mortem examination showed a "local meningitis." When, however, the leptomeninges outside of the "local meningitis" area were examined microscopically, distinct signs of inflammation were found.

Finally, there are a few instances of fatal intracranial lesions, where the cerebro-spinal fluid obtained by lumbar puncture showed meningitic changes, but where the macroscopic appearance of the leptomeninges was normal at the post-mortem, while the microscopic examination revealed the existence of leptomeningitis.

Reference to the cases discussed in literature shows that in none were the leptomeninges examined microscopically. They cannot, therefore, be used to prove that intracranial lesions, other than meningitis, can change the cerebro-spinal fluid in the same way as meningitis.

It will be seen, then, that in some cases absence of purulent exudate in the subarachnoid spaces, and of other macroscopic inflammatory signs, does not contradict the correctness of the clinical diagnosis of meningitis; also, that the results of lumbar puncture are sometimes safer than macroscopic examination of the leptomeninges.

Indeed, examination of the cerebro-spinal fluid *in vivo* is a very reliable and most important method of examination in aural surgery, and should never be omitted where there is the slightest suspicion of an intracranial lesion; it provides a means of detecting microscopically the most important inflammatory products of acute leptomeningitis from its very commencement, when macroscopic changes may yet be absent.

Clinical Definition.—As lumbar puncture must be considered of such great importance, I may perhaps be permitted to state briefly the experience I have had with about 2000 lumbar punctures.

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While the cerebro-spinal fluid obtained by lumbar puncture in cases of fatal meningitis does not, as a rule, become turbid before twelve to thirty-six hours have elapsed from the onset of the disease, pleocytosis may appear as early as three hours from the onset, and it is exceedingly rare that a case of otogenic meningitis is examined so early that an increase in the cells is not present. The only exception I have seen is Case I.

CASE I., No. 409/1918. — Female, aged 24. Admitted 23rd December—died 26th December 1918.

Acute suppuration of left middle-ear for ten days. During the last two days infiltration of the mastoid region took place. No labyrinthine symptoms except considerable reduction of hearing. Simple mastoid operation performed at once. Temperature the same evening, 99.0°. In the middle of the following night severe giddiness and repeated vomiting; later on, shivering. On 24th December the temperature was 104°; nystagmus to the sound side; hearing completely lost; slight rigidity of neck. Lumbar puncture revealed perfectly clear fluid with $\frac{3}{3}$ cells per cubic millimetre in Fuchs-Rosenthal's counting camera. Globulin = 0; Albumin > 10. Labyrinthectomy performed at once. Cerebro-spinal fluid on 26th December opalescent, containing numerous polynuclear cells and pneumococci. Death early the following night from diffuse purulent basal meningitis.

The Cerebro-spinal Fluid in Meningitis.

When the cerebro-spinal fluid obtained by lumbar puncture is turbid, when there is an abundance of polynuclear cells, and when bacteria are present, then of course all agree that we have before us an undoubted case of meningitis, indeed often a malignant form.

It must, however, be pointed out that in 70 per cent. of my cases leading to death from uncomplicated meningitis, the cerebro-spinal fluid obtained by lumbar puncture was sterile, although in all these cases bacteria were found post mortem when searched for. The conclusion must, therefore, be that the presence of bacteria is not an absolutely necessary finding in true meningitis. On the other hand, bacteria were found in about 10 per cent. of the cases which recovered, thus demonstrating that they are not an absolute *signum malum*.

As to the type of cells found in the cerebro-spinal fluid, they might be—broadly speaking—divided into two forms: polynuclear and mononuclear. In the vast majority of fatal

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cases of uncomplicated meningitis, the cells are, as is well known, exclusively or principally polynuclear. In a few fatal cases, however, mononuclear cells were found principally or solely, all these cases having a protracted subacute course much resembling tuberculous meningitis. Otherwise, the mononuclear form belongs to the benign cases. There are, nevertheless, as will be shown further on, many exceptions.

Finally, the pressure of the cerebro-spinal fluid is generally very high in malignant cases, 1000 mm. being the highest pressure measured in our cases. In benign cases it is sometimes not much beyond 180 mm., which I consider a normal pressure; frequently it is moderately increased. Exceptionally it is very high, the highest pressure measured in my clinical material being 950 mm. Increased pressure of the cerebro-spinal fluid is not of much importance in prognosis, and in benign cases it sometimes increases while the pleocytosis decreases (see Case II.).

As to the globulin- and sugar-tests I do not place much stress upon these modes of examination of the cerebro-spinal fluid in cases of otogenic meningitis. Their importance is much inferior to those mentioned above.

If I were to make a table—to which there are, however, many exceptions—showing the difference between malignant and benign meningitic cerebro-spinal fluid, it would be as follows:—

Meningitic Cerebro-spinal Fluid.

	Malignant.	Benign.
Appearance . . .	Turbid	Clear or opalescent.
Type of Pleocytosis . .	Polynuclear	Mononuclear.
Bacteria	Present	Absent.
Pressure	Very high	Moderately high.

Using lumbar puncture as a diagnostic measure, I should venture to define otogenic meningitis, from a clinical point of view, as an acute febrile brain disease, caused directly or indirectly by an acute or chronic middle-ear suppuration, exhibiting “diffuse” brain-symptoms and a cerebro-spinal fluid with pleocytosis, whether there are bacteria or not, and whether the type of the pleocytosis is polynuclear or mononuclear.

As to the number of cells which constitute a pleocytosis, opinions differ: Rehm, and others with him, considered 10 per c.mm., *i.e.*, 30 in Fuchs-Rosenthal’s counting camera, as the limit.

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Later on, Holzman lowered the figure to 5, considering 3 to 5 as being on the boundary line between normal and abnormal.

Both these authors, and many others, seem to base their experience especially on chronic diseases of the leptomeninges. Looking into the present clinical material, I consider even Holzman's figures too high. I should say that the existence of between 1 and 2 cells might be considered normal, adding, that in the vast majority of cerebro-spinal fluids there are no cells at all, if there is no intracranial lesion, and neither syphilis nor any other infectious disease present. When there are between 2 and 3 cells, I consider the case in question as suspicious. If there are 3 or more cells, and if there are "diffuse" brain symptoms present, I consider the diagnosis of meningitis as certain. Cells numbering up to 150 to 200 per cubic millimetre do not, as a rule, alter the clear appearance of the cerebro-spinal fluid, while an excess of this number makes it first opalescent, and, if the meningitis progresses, distinctly turbid. As soon as the fluid is not *perfectly* clear, I consider the pleocytosis high; 15 to 150 to 200 cells I should call moderate, and 3 to 15 slight pleocytosis.

As will be seen, great stress must be put upon the cell count when the cerebro-spinal fluid is clear. It should be added that this procedure ought to be done at once after the fluid is withdrawn by lumbar puncture.

If lumbar puncture is performed at intervals, we are also able to follow more accurately the progress or the decline of the meningitis, taking into consideration the characters of the cerebro-spinal fluid mentioned in the table above.

Lumbar Puncture not Infallible. — The importance of lumbar puncture as a diagnostic means is somewhat reduced by the circumstance that it shares one quality with all other clinical methods of examination: it is not infallible. It does not always act with the exactness of a water-gauge measuring the rise and the fall of the meningeal inflammation; the biological processes are too varied and complicated. Now and then, we see cases where there is a distinct contradiction between the benign character of the cerebro-spinal fluid and the grave state of the patient.

This happens, first of all, in the cases of complicated meningitis mentioned before, where the associated intracranial complication runs its fatal course while the meningeal changes decrease or disappear. In such cases it

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is really not the lumbar puncture which has given a wrong answer, it is our interpretation which is wrong.

It is quite another matter when in fatal cases of uncomplicated meningitis, the cerebro-spinal fluid assumes a less malignant or even a benign character *sub finem vite*. I have seen two of the latter cases, and have each time found at the post-mortem examination considerable plastic and thick exudate on the base of the brain. In these, it is easy to understand that the free communication of the cerebral fluid with the spinal fluid has been interrupted, and that the lumbar puncture principally indicates the state of the leptomeninges in the spinal canal.

This might also be caused by the presence of a brain-abscess, as will be demonstrated further on.

Now and then, one sees fatal cases of meningitis, uncomplicated or not, where one lumbar puncture shows a less malignant type of cerebro-spinal fluid than the puncture preceding it. In cases which recover, occasionally it happens that lumbar puncture shows the fluid of a less benign character than does the preceding puncture without aggravation of the symptoms.

Benign Uncomplicated Meningitis.

(a) *Turbid Cerebro - spinal Fluid* ("Benign Purulent Meningitis").—The cases of uncomplicated meningitis ending in recovery afford the most ample opportunity of studying benign meningitis in its purest forms. They were, as will be seen from Table II., 38 in number, nearly half of them being children five to fourteen years of age. A little over 50 per cent. of the cases were caused by streptococcal otitis. This group may be divided into two: one in which the lumbar puncture gave turbid fluid, and the other in which it was perfectly clear.

The first group consists of 23 cases of which 3 were post-operative.

In all the 23 cases, with one exception, polynuclear cells were present in the cerebro-spinal fluid; they were predominant in 16 cases, while in 4 there was an equal number of polynuclear and mononuclear, and in 2 an excess of mononuclear cells. Bacteria were present four times (once in the exceptional case, where only mononuclear cells were found); in these cases pneumococci appeared twice, streptococci once; in one the forms could not be determined. The pressure varied from 130 to 800 mm.

The disease often started with shivering or sensation of

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cold, more frequently with vomiting, the latter symptom being only absent in two cases, while headache appeared in half of the patients. The temperature was never normal, but was in a few cases, only slightly elevated, while in about one-third of the cases, it was about 104° and beyond. The pulse was generally quick, as would be expected; five times there was a slow pulse. Drowsiness was often present, and twice the slow cerebration had developed into deep stupor. Rigidity of the neck was occasionally absent, as it is not infrequently also in fatal cases even up to death. The same was the case with Kernig's sign, while Babinski's sign was only present five times. Optic neuritis was observed in seven patients. In one case, where an abscess was suspected on account of the presence of deep stupor and slow pulse, the dura was opened, the brain explored, and a piece removed for microscopical examination; this revealed a considerable acute lepto-meningitis (but no encephalitis as expected) with fibrinous exudate on the surface.

These cases were all operated on except one, a child aged two, where operation was refused. The lumbar puncture yielded in this case a very turbid fluid containing an equal number of polynuclear and mononuclear cells, a slight optic neuritis being also present. This very rare and remarkable case shows that there may be a tendency to spontaneous recovery in some forms of otogenic meningitis.

There can, I think, be no doubt about the character of this type of benign meningitis, namely, that it is a genuine acute diffuse purulent meningitis. One can, of course, say that it is the initial stage of this disease, arrested in nearly all cases by an operation, but in several of them the objective signs and the grave state of the patient seemed to indicate that the initial stage had passed.

It is, therefore, not infrequently difficult to distinguish this form of benign meningitis from the malignant forms until twenty-four to thirty-six hours have elapsed after the operation has been performed; the subjective symptoms and the objective signs of meningitis generally clear up directly or soon after the operation, while a rapid progress of these is characteristic of fatal cases whether operated on or not. Fatal cases are often also recognised by the presence of septic symptoms, especially very high fever and albuminuria, the latter symptom being rare in benign cases. In fatal cases irregularity of the pulse also appears early.

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(b) *With Clear Cerebro-spinal Fluid* ("Collateral Meningitis").—The fifteen patients belonging to this group were still younger, the youngest being ten months, the oldest fifty years. Three cases were post-operative.

The cerebro-spinal fluid contained cells varying in number from 9 in Fuchs-Rosenthal's camera (where the figure must be divided by 3, *i.e.*, 3 per cubic millimetre) to 598. Five times mononuclear cells only were present, five times an excess of these, and three times an excess of polynuclear elements. In no case were bacteria found. The pressure was normal only twice; generally it was 300 to 500 mm., but in one case it was increased to 950 mm. (Case II.).

The temperature was normal only once; in the majority of cases it was slightly raised, and in five it was about 104° . The pulse was frequently quickened, as might be expected; twice it was noticed to be slow. Shivering or chilly sensations were reported five times. Vomiting was a constant initial symptom, and often appeared later in the course of the disease, even when the patient otherwise did well. Headache was less frequent in this group, and was not particularly prominent in cases with high pressure of the cerebro-spinal fluid. Drowsiness, which I consider a symptom of great diagnostic value, but which is often overlooked when slight (as it often is in these cases), was very frequent; in only one case was stupor present. Rigidity of neck only appeared eleven times, being sometimes so slight that this symptom was not discovered unless the head of the patient was bent very much forward. Kernig's sign only appeared five times, while Babinski's sign, sometimes only on the opposite side, was present six times. Five of the patients had optic neuritis; paresis of the abducent nerve was found twice. The recovery often took a long time, the pleocytosis disappearing very slowly, and some of the cases had a remarkably protracted subacute course, as seen from Case II.

CASE II., No. 11/1922.—Female, aged 17. Admitted 1st November 1921. Discharged 7th January 1922.

Acute inflammation of left middle-ear for four days. Ten hours before admission, shivering, repeated vomiting and giddiness. Temperature 101.5° ; pulse, 108. Drowsiness and Kernig's sign present. C.S.F. clear, pressure 400, cells $\frac{79}{3}$, exclusively mononuclear. Simple mastoid operation performed at once. 2nd November, Temperature 100.4° ; pulse, 92. Headache and photophobia present. 3rd November, Increased drowsiness. Temperature, 100.4° ; pulse,

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100. C.S.F. $\frac{10}{3}$ cells, pressure 300. 4th November, Temperature, 99.7° ; pulse, 72. Double vision from paresis of right abducent nerve. No Kernig's sign. 5th November, Temperature, 99.5° ; pulse, 52. Babinski's sign and rigidity of neck present. C.S.F. cells $\frac{14}{3}$, pressure 650. 7th November, Temperature 100.5° ; pulse, 68. Slight optic neuritis, 9th November, Temperature 99.9° ; pulse, 60. Rigidity of neck and Babinski's sign disappeared, instead of which brain-nystagmus was present. C.S.F. $\frac{11}{3}$ cells; pressure 700. From 9-15th November all brain-symptoms disappeared, except the right-sided abducent paresis and the optic neuritis which had increased. Temperature normal; pulse, 56-64. 16th November, Headache and vomiting. C.F.S. $\frac{2}{3}$, pressure 950. From 17th-21st November, Headache and vomiting ceased. Paresis of right abducent nerve less and present also on the left side; increasing optic neuritis. Temperature normal; pulse 50-84, irregular. 22nd November, Temperature normal; pulse, 88, regular. C.S.F. $\frac{2}{3}$, pressure 650. 23rd November to 7th January 1922. The patient felt on the whole well, but now and then nausea and headache appeared. Temperature became normal, pulse varying from 80-92. Optic neuritis remained stationary, when it had reached the height of four diopters on the right side and three on the left side, and showed only slight decrease when the patient was examined on 6th May—six months after it had first appeared. When examined on this day there still remained slight paresis of the left abducent nerve and the pressure of the cerebro-spinal fluid was 600. The cells which had disappeared entirely on the 19th of December were present only in the number of $\frac{2}{3}$. The patient felt well but had a slight headache now and then.

The question naturally arises: Is it possible that such a small number of cells in the cerebro-spinal fluid as $\frac{9}{3}$ in Fuchs-Rosenthal's camera, *i.e.*, 3 per cubic millimetre, indicates the presence of meningitis? This must, I think, be answered in the affirmative, if we consider the following example:—

CASE III., No. 512/1921.—Male, aged 10. Admitted 7th November 1921. Discharged 7th December 1921.

Chronic middle-ear suppuration on the left side. Initial shivering and vomiting two days before admission. Temperature 100.5° , pulse 88. Slight drowsiness, otherwise no objective signs of meningitis present except optic neuritis and paresis of the right abducent nerve. C.S.F. perfectly clear, pressure 600, $\frac{6}{3}$ mononuclear cells (=2) per cubic millimeter. Radical operation performed at once, by which a considerable cholesteatomatous osteitis was found with fistula on the external semicircular canal. Two days later C.S.F. contained $\frac{9}{3}$ (=3) cells per cubic millimetre, some polynuclear forms also being present;

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pressure 750. On the 10th November there was vomiting, and Kernig's sign was present. 11th November, C.S.F., pressure 600 mm., $\frac{9}{8}$ exclusively mononuclear cells. 19th November, C.S.F. $\frac{5}{3}$ exclusively mononuclear cells. Slow recovery, Kernig's sign still present on the 26th November. On 4th January 1922 optic neuritis still present.

Moreover, there is a small series of cases of fatal meningitis which started in exactly the same way as Case III. In cases, especially of acute suppurative labyrinthitis with clear cerebro-spinal fluid and very slight pleocytosis, our experience in the hospital has been, that if labyrinthectomy is performed on the very first warning of the slight pleocytosis, the patients are frequently saved, while disregard to this warning in a great majority of cases leads to the death of the patient from meningitis. In no other class of case is it so important to watch the cerebro-spinal fluid, if necessary, by repeated lumbar puncture every twelve to forty-eight hours.

The question whether cases with clear cerebro-spinal fluid and slight pleocytosis should be classified as meningitis or not is, as will be seen from the above, not an academic one but one of eminent practical importance.

Whether the meningitis in such cases is diffuse or of a local nature, one cannot of course decide with certainty, but I presume it is generally of a more diffuse character. It is often difficult to distinguish—even at a post-mortem examination—between a “local” and a “diffuse” meningitis, and most fatal cases of meningitis start surely as “local” inflammation. I think, therefore, that it would be better to distinguish between a “circumscribed” meningitis, meaning thereby a process where there is soldering of the membranes in the circumference, and a “diffusing” meningitis, whether localised to small or large areas, without these adhesions being present and therefore with tendency to progress.

On account of the uncertainty mentioned, I have for several years designated the benign forms of otogenic meningitis with clear cerebro-spinal fluid by the name of “Collateral Meningitis.” I consider this name better than the many names used, such as *meningitis serosa*, *m. localis*, *m. sympathetica*, “meningeal irritation,” as one avoids using a name which involves a distinct interpretation of the character or of the extension of the disease. Further, “collateral meningitis” expresses the dependence of the process upon a primary focus and also its

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tendency, like other collateral inflammations, to recede when the primary disease either is cured by operation or—if it is a phlebitis of the sigmoid sinus or a brain-abscess—passes into a subacute state.

In cases where there are diffuse brain-symptoms and where the cerebro-spinal fluid either does not contain any cells or only one or two, I think one might use the name “meningismus.”

The term “local meningitis” ought to be restricted to those cases where there is slight pleocytosis and unilateral headache without any diffuse brain symptoms or fever, *i.e.*, a “circumscribed meningitis.” Such patients ought to be operated on early, for sometimes a diffuse meningitis suddenly sets in. On the other hand, there is also risk connected with the operation, because this sometimes acts like an injury, causing post-operative fatal meningitis. We are, therefore, in such cases—as in most other surgical cases—in a position in which there is risk both in operating or in not operating; in each individual case one must decide in which direction the risk is smallest.

Benign Complicated Meningitis.

(a) *With Phlebitis of the Sigmoid Sinus.*—This group embraces 16 cases of recovery, of which 8 exhibited clear and 8 turbid cerebro-spinal fluid. The pleocytosis was never under $\frac{2}{3}$ = 7 cells per cubic millimetre; on the other hand, the fluid never had such a turbid appearance as in the majority of patients with uncomplicated benign meningitis. The predominant form of cells was polynuclear. Bacteria were only found once, which is rather strange, as I have seen a small series of cases of uncomplicated phlebitis of the sigmoid sinus with a cerebro-spinal fluid containing a very large quantity of bacteria but no cells and with no signs of meningitis.

The diffuse brain symptoms of meningitis were often covered by the symptoms of the phlebitis. Optic neuritis only appeared twice.

In one case (with slightly turbid cerebro-spinal fluid) microscopic examination of a piece of brain tissue excised during operation revealed the existence of considerable inflammation of the cerebellar leptomeninges.

In all cases, the inflammation of the sigmoid sinus and of the leptomeninges seemed to have started simultaneously.

In several cases, where there was a combination of fatal

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sinus phlebitis and meningitis the latter disease showed a benign character, being either very slight or disappearing entirely before death. Terminal meningitis is, on the whole, not a frequent cause of death in phlebitis of the sigmoid sinus.

One single case deserves mentioning, inasmuch as the patient died one year later from uncomplicated meningitis originating from the other ear. The diffuse brain symptoms were almost exactly alike on each occasion.

In two cases in which collateral meningitis complicated fatal sinus phlebitis, the post-mortem examination showed no macroscopic signs of leptomeningitis, while the microscope revealed the existence of this complication.

(*b*) *With Brain Abscess*.—In these cases the meningitis is generally secondary, and its symptoms are usually covered by the symptoms of the brain-abscess, but now and then they assume a more independent character.

The cerebro-spinal fluid obtained by lumbar puncture is generally of the benign character described above, and only once contained bacteria (streptococci). This is often the case also in patients who die with brain abscess, even if death is caused by terminal meningitis. The feature, however, which is most characteristic of brain abscess associated with meningitis—and this is nearly always the case, as pointed out nearly thirty years ago by Sir William Macewen, to whom brain-surgery is so much indebted—is that the cerebro-spinal fluid changes very much from one lumbar puncture to another. It becomes now turbid, now clear; sometimes the pleocytosis is of a mononuclear, sometimes of a polynuclear type during the course of the disease in the same patient. These facts are, I presume, due to two circumstances. The first is that the encephalitis causing brain abscess does not develop continually, but in small spurts, during which the secondary meningitic process is more active, and therefore produces more polynuclear cells. The other circumstance is that a brain abscess—as can be seen by many post-mortem examinations—often causes an augmentation of the volume of the brain which presses down the medulla oblongata into the upper part of the spinal canal, thus impairing the free communication between the cerebral and spinal subarachnoid spaces. The lumbar puncture represents then, as mentioned before, the state of the spinal rather than of the cerebral leptomeninges.

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Finally, it must be stated that a piece of a convolution of the brain was removed in a patient who recovered from acute encephalitis, in whom the cerebro-spinal fluid was very turbid, containing an excess of polynuclear cells and streptococci; the microscopical examination only revealed the existence of encephalitis. Although in two other patients who died from meningitis (the diagnosis being confirmed at the post-mortem examination) a similar microscopical examination *in vivo* also gave no evidence of meningeal inflammation, I think still that the above case might raise the doubt whether a brain abscess or a suppurative encephalitis may not give the cerebro-spinal fluid a "meningitic" character when it is situated close to the surface of the brain or to the ventricles. *Qui vivra verra!*

Operative Measures.

I have, in the beginning of this paper, already touched upon some more remote causes which favour the arrest of the progress of meningitis. But they are, of course, of secondary importance. A very important factor is, as I am sure we are all fully convinced, the *early* elimination of the primary focus, whether this is in the middle ear alone, in the petrous bone, or in the labyrinth.

But in many cases even an operation performed as early as possible is not able to avert the fatal course of the disease. My experience has led me to believe that there are—from a clinical point of view—really two forms of meningitis. In the one, and by far the more frequent, the inflammatory process in the leptomeninges spreads like the explosion of a barrel of gunpowder ignited by a spark, and any operative measure is useless, the disease showing a very rapid progress, which is a prominent clinical feature of great diagnostic value. In a minority of cases—say one-third—the spark, *i.e.*, the inflammatory products from the primary focus, is not able to cause the explosion before the fire has been nourished for some time from the focus. If, in such cases, the latter is eliminated, the further feeding by inflammatory products ceases, and the nascent inflammation of the meninges recedes.

This idea has, in any case, been my principal guide during the last decade in the operative treatment of otogenic meningitis. For seven years previously I tried, in vain, all sorts of operations supplementary to the elimination of the primary focus such as

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opening of the subarachnoid space and draining the cavity. I consider them not only as useless but even as injurious, and my impression is that my results are better now than before. My experience has made me believe that if one does not stop the development of a meningitis by elimination of the primary focus, nothing can stop it.

My view is especially corroborated by examining the results of the operative treatment of labyrinthine meningitis. Although this form of meningitis is particularly serious, I have seen one recovery after another without any further operation than mastoid operation and labyrinthectomy, performing this operation when the function of the labyrinth is extinguished or nearly extinguished, as soon as there are about 9 cells or more in Fuchs-Rosenthal's camera, *i.e.*, 3 per cubic millimetre.

As, however, craniotomy involves no danger when the dura mater is not opened, I regularly perform this operation in order to examine minutely the state of the dura mater. If the membrane shows no signs of a deeper going disease, I go no further when there is no suspicion of subdural or cerebral abscess.

While formerly I always performed the radical mastoid operation in acute cases in order to be able to examine also the labyrinth minutely, I now only perform the simple mastoid operation in acute cases, as we possess means of testing accurately the function of the labyrinth, fistula of the labyrinth being exceedingly rare in acute suppuration.

Repeated lumbar punctures I consider not only very useful as a means of following the decrease or increase of the meningeal process, but I believe they have a beneficial influence, especially when the pressure of the cerebro-spinal fluid is very high or the patient is suffering from severe headache or nausea.