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[*Reprinted from the* PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE, *May, 1930, Vol. XXIII* (*Section of Otology, pp. 45—52*).]

London

JOHN BALE, SONS & DANIELSSON, LTD.

83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W. 1.

1930

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In taking stock of my own experience I find that I have personal records of forty-eight cases in which brain abscess was either suspected or overlooked. In twenty-three of these the presence of an intra-cerebral abscess was subsequently verified by operation or necropsy. Seven never came to operation; in some others operation was performed too late. Thus, in one case, that of a frontal abscess secondary to frontal sinusitis, I arrived at the bedside of a patient only to find that she had stopped breathing about one minute before. A hasty examination while artificial respiration was being performed showed severe papillœdema, slight right facial weakness, and X-ray evidence of increased density of left frontal sinus. The left frontal lobe was hastily tapped and 2 oz. of pus were withdrawn. The patient improved temporarily, but died eight hours later from pyocephalus and leptomeningitis.

I cite this example that you may have less cause to disbelieve me when I say that, in my own practice at least, the bad results are almost entirely due to delays or mistakes in diagnosis, and it is to the difficulty of diagnosis that I would particularly like to direct attention. I had originally hoped to say something about treatment, but in the course of preparation of this paper I found that there was so much to say about diagnosis that it seemed best to limit myself on the present occasion to this aspect of the subject.

There is one aspect of treatment, however, that I am prompted to bring up by listening to Professor Neumann's interesting paper. Professor Neumann has made a careful comparison between the recent results in treatment of otogenic brain abscess and the results obtained by Macewen over thirty years ago, and he has sought an explanation for the fact that the results of otologists of the present day are very much worse than those of Macewen. This fact is one which has puzzled me also, and I have read through Macewen's book several times in the hope of finding some adequate explanation. Probably there are a number of causes for this divergence, but there is one possible cause that has not, so far as I am aware, received adequate consideration. It is, I believe, the usual practice among otologists of the present day to use the mallet and gouge or chisel in clearing out the mastoid process. As far as I have been able to ascertain from his book, Macewen never used a mallet and gouge, but always opened the mastoid process with hand burrs. I would like to learn from Professor Neumann and the Members of this Section whether the more modern method, involving as it does, intermittent percussion of the skull, may not produce in the underlying brain a disturbance that tends to promote the spread of the infective process. The success of any method of treatment depends in the first instance on the ability of the brain to localize the infection. No form of operation avails if an abscess spreads rapidly and ruptures into the ventricle within a few days of its inception. But this is, fortunately, a rare event. The brain is richly endowed with connective tissue cells, in addition to its own supporting glia, and these cells are rapidly mobilized when any focus of infection occurs. That they are often successful in limiting the spread of infection is shown by histological examination of the wall of an abscess, and by the not infrequent observation of cases in which an abscess is found to be completely surrounded by a dense fibrous capsule. The barrier

against the spread of infection from a recently-formed abscess into the ventricles is always rather precarious, and transmission of any force through the brain—such as might conceivably occur from the use of a mallet and gouge on the skull overlying a temporo-sphenoidal abscess—might diminish the chances of successful limitation of the abscess by natural methods. The importance of avoiding anything but the slightest injury to the abscess wall and the overlying brain is well recognized as the basis of modern methods of treating brain abscess.

In twelve of my twenty-three verified cases—just over half—the abscess had followed infection through the nose or ear. This represents fairly accurately the relative frequency of these varieties of brain abscess. For the opportunity of seeing most of these cases, I have to thank my otological colleagues at the London Hospital.

I propose first to make some general remarks about the diagnosis of brain abscess and then to consider particularly the diagnosis of the temporo-sphenoidal abscess.

Errors in the diagnosis of brain abscess.—How does it come about that so many cases are never given the benefit of surgical exploration—as, for example, seven out of twenty-three cases in my own series, and a considerable percentage of the two hundred and more cases that have come to necropsy at the London Hospital since Professor Turnbull first began keeping accurate records in the year 1908? You will, perhaps, be thinking that this is not very much the concern of the otologist, but many of the unexplored cases in the London Hospital series proved to be cases of abscess following infections of the ear and nose.

The reason for all these mistakes is that the symptomatology of brain abscess is not, as yet, clearly defined. The localizing signs of abscesses in various parts of the brain have been well established by such admirable papers as that delivered before this Section by Dr. C. P. Symonds a few years ago.[1] A temporo-sphenoidal abscess is rarely missed if it is examined for in the ways laid down by Dr. Symonds, but it is not so easy to consider the possibility of temporo-sphenoidal abscess in every case of mastoiditis, and to make the requisite full neurological examination in every case before mastoidectomy is performed. It is even more difficult to become alive to the possibility of brain abscess in a patient who has lapsed into a state of stupor after a relatively short and insignificant period of shivering, vomiting and headache. Such a case is often sent to hospital with a diagnosis of encephalitis or of cerebral thrombosis, and is admitted to the medical wards. It is clear to me from the variety of cases which I am asked to see, that my medical colleagues at the London Hospital are now fully alive to the varied symptomatology of brain abscess. But it is not many years since these cases often lay in the medical wards until they finally came to the necropsy room, where the tradition grew up among the pathologists that cases coming to them with the clinical diagnosis of cerebral thrombosis usually turned out to be cases of brain abscess!

Some mistakes in diagnosis may be assigned to neglect of ophthalmoscopy and perimetry, but it is well to emphasize the fact that many brain abscesses produce little or no papillœdema. A more frequent source of error lies in the common misconception that a brain abscess, like abscesses in other parts of the body, must produce a rise of temperature. It cannot be too strongly stressed that the temperature in a case of brain abscess that has not been operated on is usually subnormal.

In some cases the patients die because, although it may be realized that there is a brain abscess, the condition is not considered so urgent as it actually is. These, I believe, are cases in which an abscess has formed during some previous illness. A patient may have a mastoidectomy performed and the post-operative period is not entirely smooth: there is some headache and vomiting, and the temperature is not even, but shows falls to 96° and rises to 100°. However, the symptoms clear up within two or three weeks and the patient is discharged from hospital apparently

1 "Some points in the Diagnosis and Localization of Cerebral Abscess," by C. P. Symonds, M.D., *Proceedings*, 1927, xx, 1139 (Sect. Otol., 41).

well. He is walking about months later when suddenly he has violent headache, goes into coma, and dies within a few days. At necropsy an abscess is found which is surrounded by a firm capsule, that has obviously taken many weeks or even months to form. In this type of case it is probable that the abscess has formed during the disturbed convalescence from operation. The patient's resistance at that time has been good, and the abscess (call it purulent encephalitis if you wish) has become well localized and even partially absorbed ; in this condition he is able to go about his work, apparently in good health. When convalescence after mastoidectomy is disturbed in this way, the patient should be kept under close observation for a long period, because these partially subsiding abscesses are as dangerous as volcanoes, and they erupt rapidly and without warning. It is not only after mastoidectomy that such an abscess may form, but also after mild attacks of mastoiditis and frontal sinusitis that go untreated.

The localizing signs of temporo-sphenoidal abscess.—I have practically nothing to add to Dr. Symonds' paper on this subject, but I would urge the importance of repeated daily perimetry in these cases. There is no investigation so valuable in the diagnosis of temporo-sphenoidal abscess. The finding of a homonymous quadrantic defect not only clinches the diagnosis of abscess, but it also gives valuable information as to the situation of the lesion. The usual type of defect is in the superior quadrants, but a homonymous inferior quadrantic defect may also be produced by a cerebral abscess secondary to mastoiditis. The discovery of this type of defect indicates clearly that it is useless to attempt to drain the abscess through the mastoidectomy wound. The exploration in such cases must be made at a higher level.

By choosing a period of temporary improvement I have been able to take on the Bjerrum screen, the fields of patients who were acutely ill with brain abscess. A portable perimeter would perhaps be better, but, whatever the method employed, it is important not to rely on confrontation tests if there is the slightest chance of obtaining more accurate records. The condition of the fields in a case of abscess of a cerebral hemisphere after mastoiditis, not only indicates the level at which the abscess should be sought, but it is also a valuable guide as to when to operate. Extension of a quadrantic hemianopia, into a complete hemianopia calls for immediate operation. If the defect is not expanding, it is safe to wait, if waiting is indicated.

There is sometimes, however, a grave difficulty about examining for field defects and the other signs that Dr. Symonds has described, and that is when the patient is so drowsy that he does not coöperate sufficiently. It is the important ocular tests, especially tests of ocular movements and of the visual fields, that cannot be made. Much may be discovered in these cases by patient observation at frequent intervals, but, in some cases, and particularly in children, the diagnosis and localization of the abscess cannot be made with certainty by clinical methods. X-ray examination of the skull is particularly valuable in this type of case. Abscesses of the brain very often contain gas. How often, when the brain needle enters the abscess cavity, do we not first observe a little puff of offensive gas before the pus wells out ? This gas will show in the skiagram and it indicates exactly the position of the abscess.

Differential diagnosis.—In this series of cases confusion in diagnosis has arisen between brain abscess and the following conditions : Encephalitis, cerebral thrombosis, cerebral aneurysm, tuberculous meningitis, purulent pachymeningitis, purulent leptomeningitis, arachnoiditis (serous meningitis), and intracranial tumour. I do not propose to go into the clinical differentiation of these groups. Indeed, this is often impossible in the present state of our knowledge, especially when the intracranial disturbance affects an individual who incidentally suffers from a chronic running ear. In many cases a diagnosis is only reached by doing an exploratory puncture of the brain. The following case may be cited as an example :—

Case I.—Meningo-encephalitis simulating brain abscess. Otitis media and chronic

mastoiditis. Mastoidectomy. Exploratory puncture of the brain, with findings that excluded brain abscess. Recovery.

History.—A boy, aged 8, was admitted to the London Hospital in a semi-conscious condition on December 26, 1928. Six days before he had come home in the evening complaining of feeling tired. On the following afternoon he had complained of frontal headache, and had vomited several times without warning. The next day he gradually became unconscious.

Past History.—There were several points that were likely to be of importance in the diagnosis. He had had bronchitis and pneumonia at the age of five months, and from that time onwards he had had much chest trouble, and for four years before admission had attended a tuberculosis dispensary. At the age of two he had had discharge from both ears, and this had continued for twelve months. A week before the onset of the present illness the left ear had begun to discharge again, and then the right ear had begun to discharge. Those who saw him at the time of his admission were thus prepared to consider a diagnosis of tuberculous meningitis or of brain abscess.

Examination.—The boy was semi-conscious and had some head retraction. Temperature 100·6°. Pulse-rate 112. Internal squint of the left eye. Slight right facial weakness. Bilateral extensor plantar responses. On clinical examination there was no evidence of any disease of the lungs. Examination of the right ear showed no abnormality. There was profuse offensive discharge from the left ear, and the left drum was perforated anteriorly and elsewhere reddened. Lumbar puncture showed clear fluid under raised pressure. The fluid contained twenty white cells per cm., 0·035% protein, no tubercle bacilli.

Progress.—After admission the patient became more unconscious and gradually developed a right hemiparesis. The pulse-rate became slower.

First Operation.—December 28, 1928. An incomplete left mastoidectomy was performed. In the region of the antrum there was a polypoid mass of soft, red tissue, and this contained a little mucopurulent fluid, which was subsequently shown to be sterile. No pus was found. The dura of the middle fossa was exposed over an area about one-half inch in diameter. It did not bulge unduly. Histological examination of the fragments of bone removed at operation showed fibrosis of the marrow.

Post-operative Notes.— On the following day there was slight papillœdema and the right hemiparesis was rather more pronounced. The boy continued in a semi-conscious condition, and the pulse-rate slowed to 44, the respiration rate to 16.

Second Operation.—January 2, 1929. Through a separate incision above and in front of the mastoidectomy incision, the left temporal lobe was tapped for abscess, but none was found. When the subarachnoid space was opened a free escape of cerebrospinal fluid occurred. This observation was considered to be strongly against the presence of an abscess. In addition the small fragment of brain tissue that was removed in the brain needle after tapping of the temporal lobe looked to the naked eye normal—quite unlike the soft, glistening yellowish white tissue that is recovered when the needle has passed near an abscess. It was felt as a result of the operation that abscess of the left temporal lobe could be excluded.

Subsequent Course.—Two days after this operation the boy began to recover consciousness, and from that time onward his condition steadily improved. The right hemiparesis cleared up almost completely within a week, though traces of it could still be detected for a further three weeks. The incision for puncture of the brain healed by first intention, notwithstanding its proximity to an open mastoidectomy wound. The boy recovered completely and was discharged from hospital on February 12, 1929. He was last seen on January 9, 1930, when it was found that his complete recovery had been maintained and that he was entirely free from symptoms of any sort.

Comment.—No more definite diagnosis than one of meningo-encephalitis can be made in this case. The patient would probably have recovered without either of these operations, but, in view of the difficulty of excluding brain abscess on clinical grounds, it would be hazardous at the present day not to explore such a case when explorations of the type described above can be shown to be free from danger. There would be risk of purulent leptomeningitis if such intracranial explorations were made through an open mastoidectomy wound, but no such risk attends a small exploratory incision through an adjacent region, provided the field of operation is carefully prepared. I use gutta-percha tissue for sealing off the mastoidectomy wound from the field of exploration. It is laid over the mastoid wound and it sticks

firmly to the shaved scalp when a hot moist swab is pressed against it for a second or two. The brain is then explored through a small perforation, or a series of perforations, above the level of the zygomatic process. This method should be used in all cases in which, after mastoidectomy, a temporo-sphenoidal abscess is suspected, but is not clinically certain. Should an abscess be encountered in such a position that it cannot be satisfactorily drained through the same perforation, then it is easy, either at once or, preferably, at a subsequent stage, to drain the abscess through the mastoidectomy incision or through an appropriately placed burr hole. By this method the risk of producing meningitis is avoided, and one is not over-reluctant to explore the brain in a doubtful case.

The evidence of a negative character that can be obtained by this method of exploratory puncture is not limited to failure to encounter pus within the brain. There are several other signs which strongly suggest that there is no cerebral abscess.

(1) The finding of free pus in the subdural space, after a tiny incision has been made in the dura, practically excludes the presence of an intracerebral abscess. It indicates rather that the condition is one of purulent pachymeningitis, of which more will be said later.

(2) An abundant flow of cerebrospinal fluid from the subarachnoid space, as in the case of meningo-encephalitis just described, is also strongly against the presence of an abscess in the underlying brain. It is a common observation, in cases both of abscess and of tumour of the brain, that the surface of the brain over the lesion is swollen and that its subarachnoid space is almost empty of cerebrospinal fluid.

(3) If during exploration for a temporo-sphenoidal abscess the temporal horn of the lateral ventricle is entered by the brain needle and more than 1 c.c. of cerebrospinal fluid is obtained from it, then it is almost certain that there is no abscess in the temporal lobe. Examination of post-mortem specimens shows that the temporal horn of the lateral ventricle is almost completely obliterated when there is an abscess in the corresponding lobe of the brain.

(4) Additional evidence may sometimes be obtained by examining the small fragment of brain tissue that is found in the needle after puncture of the brain. It is sometimes possible to distinguish by naked-eye, fragments of brain tissue that are œdematous from those that are normal. The œdematous brain tissue is very soft, yellowish-white and unduly glistening, in contrast to the moderately soft, pure white, slightly glistening appearance of normal white matter. If the tissue obtained from the brain needle is obviously normal to the naked eye it is unlikely that there is an abscess near the site of puncture, for an abscess of the brain is always surrounded by a very wide area of œdema.

Exploratory puncture of the brain for abscess is thus not merely a matter of finding pus, or of failing to find it. There are numerous observations to be made, and from them one can get a fairly accurate idea, even at the stage when the needle has been passed only once into the substance of the brain, whether subsequent explorations are likely to reveal an abscess. A small abscess may be difficult to find, even though the operator is fully armed with an exact clinical diagnosis and with an accurate knowledge of the topography of the brain. But skill and care in use of the brain needle will usually lead him to the abscess, though it may be necessary for him to complete his exploratory punctures at a second operation. As a rule, it is advisable not to do too much at one session. When the abscess is entered, it is important to observe whether there is any resistance to the passage of the needle, for on the thickness and firmness of the abscess wall depends to a considerable extent the variety of surgical treatment that should be undertaken.

Purulent pachymeningitis.—Two of the conditions that I have mentioned as simulating brain abscess deserve more consideration. The first of these is purulent pachymeningitis. This condition may sometimes follow mastoiditis, and it is characterized by the pressure of free pus in the subdural space. Its importance in connection with the present subject is that it may produce symptoms and focal signs identical with those of temporo-sphenoidal abscess—aphasia, hemiparesis, hemianopia, headache, papillœdema, bradycardia, and so forth. The fact that the temperature is

high, instead of being low as in abscess, is scarcely a distinction on which differential diagnosis can be based, and the only certain way in which the diagnosis can be made is by careful observation at operation. Even at operation the condition may be overlooked, for it is the uppermost part of the subdural space that is opened, whereas the pus, being free, tends to sink to the lowest level of the space. Whenever purulent pachymeningitis is suspected, the head should be tilted after a small opening has been made in the dura, so that any pus that is present in the subdural space may gravitate towards the opening. If pus is found at this stage of the operation it is most unlikely that there is also an intra-cerebral abscess. This condition is not common. Mr. Patterson and I have had two examples in recent years, one associated with a large cholesteatoma, and the other after acute mastoiditis and osteomyelitis of the skull.

Arachnoiditis (serous meningitis).—The second condition that I wish to describe more fully is that known as arachnoiditis, or serous meningitis. This condition simulates cerebellar abscess rather than temporo-sphenoidal abscess. It comes on after acute mastoiditis and chronic mastoiditis, especially when the labyrinth has been infected. The main clinical feature is papillœdema, often of extreme degree, and the headache and other general symptoms may be so slight that the condition is not recognized until sight is seriously impaired. The following is an example, and I think it is so important that I will describe it in detail:

Case II.—Left mastoiditis. Two mastoid operations. Lateral sinus thrombosis. Arachnoiditis. Blindness.

J. K., a boy, aged 5 years, first came under my notice on October 29, 1929. His illness had begun in January, 1929, when a sore throat had developed, followed two weeks later by left-sided earache. On January 31, 1929, an incomplete mastoidectomy was performed on the left side. The lateral sinus and a large area of dura were exposed, but were not opened. During convalescence there was slight fever and occasional vomiting, but the boy was discharged from hospital five weeks after the operation, quite free from symptoms.

He remained well until his tonsils were removed on June 27, 1929. Within three days the left ear began to ache and to discharge, and vomiting also occurred. On June 30, 1929, lumbar puncture revealed clear fluid under pressure, and subsequent analysis of the fluid showed no white cells, protein 0·02%, sterile cultures. Immediately after the lumbar puncture, the left mastoid was reopened, revealing much pus and granulation tissue over the dura and the lateral sinus. A needle was passed into the left temporal lobe in four directions, but no pus was found. The lateral sinus was then opened and was found to contain no clot. The wound was packed tightly with gauze. At the first dressing two days later there was sharp hæmorrhage, which was stopped by further tight packing of the wound.

The post-operative course was this time rather stormy: the child was very drowsy for several days, and suffered from severe headaches and frequent vomiting. However, he was sufficiently recovered to leave hospital on August 1, 1929, one month after operation. Headache recurred during the first two weeks in August, but then disappeared. The first indication of any failure of vision occurred on August 21, when the mother noticed that the boy tripped over curb-stones. In September he began to fall over objects and to feel his way about as he walked. Ophthalmological examination was carried out on October 14, apparently for the first time. The boy could not count fingers. The discs showed severe papillœdema and optic atrophy, and there was exudate around each macular region. Two teeth were removed on October 15.

Condition on examination, October 29, 1929: Left eye completely blind; right eye perception of light only. No reaction of left pupil to direct light, moderate reaction of right pupil. Severe secondary optic atrophy, with 2·5 D. swelling on right side, but no swelling on left side. Exudate around each macula. No other physical signs except generalized hypotonia and diminished tendon-jerks, and slight enlargement of the head. The boy was cheerful and, apart from his blindness, free from symptoms. Lumbar puncture: pressure 230 mm. In unilateral Queckenstedt tests, the usual rise of pressure occurred when the right jugular vein was compressed, but there was no rise of pressure when the left jugular vein was compressed. In this case a thrombosis of the left lateral sinus had thus evidently developed since the

lateral sinus was opened at the second operation on the left mastoid process. The cerebrospinal fluid was clear. It contained one cell per c.mm.; 0·02% protein. Cultures sterile.

Comment.—As far as it is possible to judge from the available evidence, this appears to be a case of the so-called arachnoiditis or serous meningitis. The pathology of "arachnoiditis" is at present quite obscure. In the only case of the condition in which I have been able to make a histological examination, the arachnoidea showed no evidence of inflammation, and I think the term "arachnoiditis" is probably an incorrect one. All that we know about the pathology of the condition is that there is hydrocephalus, but whether this is due to increased formation of cerebrospinal fluid, or to decreased absorption of cerebrospinal fluid, or to a combination of both these factors, is at present unknown. The condition follows middle-ear and mastoid infection, but it may also occur without any demonstrable focus of infection, either in the ears or elsewhere.

This is the first case of arachnoiditis that I have seen in which thrombosis of the lateral sinus has been demonstrated. Such an occurrence would tend to interfere with the absorption of cerebrospinal fluid, and thus to aggravate the rise of intracranial pressure. This may account for the extreme degree of destruction of the optic discs by papillœdema and atrophy. The case is typical in the absence of severe headaches, except for a short period at the beginning of the illness. This absence of headache is not to be explained solely by the fact that in this instance the patient was a child whose head could expand in response to increased intracranial pressure, for absence of severe headache is a noticeable feature also in adults.

The condition tends to recover spontaneously. I have seen two cases—both in children—in which perfect recovery occurred without any treatment at all. In one of these the papillœdema reached 4 diopters before it began to subside; in the other the swelling was never higher than 2 diopters. In both cases vision eventually became normal. In other cases, however, although all the symptoms clear up, they do not do so until vision is seriously affected. The little boy whose case I have just described is now permanently blind, though perfectly well in other ways. From my experiences with this condition I believe that a cerebellar decompression, performed before the destruction of the optic discs has become far advanced, is a sure method of preserving vision. It should be carried out in every case in which the papillœdema becomes at all severe, or in which there is any sign of lowering of visual acuity. In one case of this condition in which the patient was practically blind, vision returned in a most dramatic manner after a cerebellar decompression had been done. The operation, however, cannot be expected to effect a return of vision after blindness has been present for some weeks. Nevertheless, I would have been prepared to operate in the case described, in the faint hope of getting some recovery of sight in the right eye, if it had not been that my colleagues expressed a desire to treat the boy by repeated lumbar punctures. This course of treatment was carried out. That no return of vision occurred in a case so advanced as this cannot be counted against the method employed, but the intracranial pressure, as measured by the spinal manometer, rose instead of falling, and this suggests that treatment by repeated lumbar punctures is not a satisfactory method in severe cases.

The pressing need in this type of case is early diagnosis and, as the neurological signs are scanty, this depends on the employment of ophthalmoscopy in all cases of ear disease that show any signs of intracranial disturbance. The case described shows how it is possible for the condition to be overlooked through neglect of ophthalmoscopy. Even if the headaches and vomiting are severe in the early stages, they are always very much better at the time when the patient is going blind. Treatment, therefore, should be entirely governed by the state of the eyes.

This condition of "arachnoiditis" comprises a part of that mixed group of cases known as "pseudo-brain abscess." Mild cases of purulent pachymeningitis are probably also included in the same category.

Conclusion.—Finally, diagnosis must be carried on during treatment. Operation should often be carried out in stages. There is a right and wrong time to operate in each case, and this can only be decided after accurate observation of the clinical condition. Additional symptoms often arise in the first few days after drainage of the abscess, due to œdema, to a slight degree of meningitis, or to faulty drainage of the abscess. Is the tube to be moved, or is it wiser to leave it undisturbed? These and many other problems depend for their solution on careful day-to-day examinations. In my experience there is no disease in which repeated clinical investigations are so important as in abscess of the brain.