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Diagnostic Problems in Otogenous Intracranial Complications

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IN the *Transactions* of a foreign society last year there was a report of two cases of intracranial complications with sudden death.

The first was the case of a man who had been seriously ill for some time with an intracranial inflammatory condition, and had been operated on repeatedly. A radical operation had been performed on the left ear, with resection of the petrous portion of the temporal bone and evacuation of a cerebellar abscess. One day when the patient was otherwise feeling fairly well, he complained of headache, sat up in bed, and produced a little bloody expectoration. Suddenly he became cyanotic, his pulse became small and rapid, and he died within three minutes. The case was diagnosed as embolism of the pulmonary artery. Permission for autopsy was restricted to the cranium, and it was found that, in addition to various changes in the neighbourhood of the abscess which had been opened, there was a marked swelling of the brain and a pronounced dilatation of the lateral ventricles, which were filled with cerebrospinal fluid.

The second case was that of a man who had entered the hospital with vague intracranial symptoms and presented the contrast syndrome which will subsequently be discussed. On the day he was to be operated on he was unexpectedly found dead in his bed. The autopsy revealed a cerebellar abscess.

I will not deny that in the first case the diagnosis " embolism " may have been correct, but I doubt it. It is a surprising thing, however, that neither of these two cases gave rise to a discussion of the diagnosis which is most probable and, in the second case, in my opinion, unquestionable—namely, respiratory paralysis resulting from compression of the medulla oblongata. The clinical picture of this condition is briefly outlined as follows: Suddenly, or after a spell of Cheyne-Stokes' respiration, the patient stops breathing, becomes cyanotic and, at the same time, unconscious. If active treatment is not instituted at once he dies within a few

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minutes, and the autopsy shows in typical cases that the tonsils of the cerebellum are pressed down into the foramen magnum, that the lateral ventricles are dilated, and that the brain tissue is œdematous.

I do not think it is necessary in this country to recall the clinical picture of this condition, because it has been studied many years ago by British authors (e.g. Hilton, Fagge, Horsley, Macewen). It seems strange that numerous textbooks all over the world entirely fail to mention this complication, and there are many papers in the literature which illustrate plainly that it is not generally known—whether as to diagnosis, prognosis, or treatment.

In 1919 I had occasion to treat a case of this kind. This patient had sudden respiratory paralysis and I began artificial respiration at once; then while my assistant kept up artificial respiration with the left arm of the patient, I performed a craniotomy on the right side and evacuated a large cerebral abscess; after this, the respiration was restored and kept going till the patient died the next morning.

I collected in a small monograph,[1] all that had been published in the literature on this subject—a total of 47 cases. The diagnosis had not been made at all in many of these cases, and an attempt to open the abscess had been made in only 21 of the 47. The diagnostic errors which were committed, and are probably still being made, were: (1) *Suffocation*, the cyanosis being often interpreted as due to a laryngeal spasm. This applies in particular to those cases in which the respiration ceases under narcosis. In such cases the surgeon is led to perform tracheotomy. On account of this erroneous diagnosis, tracheotomy was performed in no less than seven of the 47 cases. (2) *Death agony:* I am inclined to think that the most frequent erroneous diagnosis in such cases is death agony. If faced by a patient who suddenly becomes unconscious and cyanotic, most physicians will think the condition is coma—the beginning of the end, and naturally will not operate. It does not occur to the doctor that he is dealing with a respiratory paralysis which can be cured. And I wish to emphasize that not only may such a respiratory paralysis be cured in the sense that the respiration is restored, but the patient may also recover completely.

In 1919 such successful treatment had been accomplished in only three cases—those of Waterhouse, Denker, and Hasslauer respectively—and it is characteristic that Hasslauer, who succeeded in pulling one of these patients through this critical condition did not mention this fact at all in his report of the case. A few more cases only have been reported since then, undoubtedly owing to the fact that the condition is still generally unrecognized.

It should be strongly emphasized that: (1) Sudden cyanosis in an otogenous brain lesion means respiratory paralysis, most

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frequently due to a cerebellar abscess. (2) The treatment consists in immediate institution of artificial respiration and evacuation of the abscess. (3) Sudden death in bed in cases of otogenous cerebral lesions is practically always due to respiratory paralysis, most often owing to a cerebellar abscess.

I shall now turn to another symptom which is practically never mentioned in the textbooks, namely bradycardia—the slow pulse-rate in labyrinthitis to which I called attention in 1923.[2] If a patient with severe otitis presents this symptom, it is generally taken as a sign of increased intracranial pressure. Several years ago I noticed, however, that a good many patients with otitis would show this symptom without any other sign of an intracranial lesion. But all these patients were suffering from labyrinthitis. Further investigation gave the surprising result that a slow pulse is a very common symptom in acute labyrinthitis in the so-called serous stage.

On searching the literature it was found that bradycardia in labyrinthine affections had been observed before, but the surprising thing was that this phenomenon had been the subject of merely a few short remarks in discussion—by Brieger,[3] Biehl[4] and Bárány[5] —and then forgotten.

Labyrinthine bradycardia was then practically unknown to the clinician. It is, I think, a little disgraceful to our branch of medicine that eminent otologists could overlook or forget that such an elementary symptom is quite common in one of the most frequent and dangerous diseases in otology.

On May 2nd, 1923, I read a paper dealing with the subject of labyrinthine bradycardia before the Danish Oto-Laryngological Society, and seven months later there appeared, quite independently, a report on this subject from the Clinic in Vienna—Neumann[6] in a lecture to the Otological Society of Austria on December 17th, having arrived at the same conclusions. Subsequent experience has confirmed the fact that we are dealing here with a quite ordinary, even frequent, symptom of acute labyrinthitis. In Denmark, in a report from the Otological Department of the City Hospital in Copenhagen, 1926, Lund has confirmed my findings. The figures from the records in the Rigshospital from 1924 to 1926 show 11 cases of slow pulse in 35 patients, namely :—

Pulse-rate.		No. of cases.
60	in	3
56	,,	4
52	,,	3
50	,,	1
		11 out of 35 patients (= 31%).

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If we collate the figures obtained by the different authors, we find the following :—

Borries	1923	13 cases.	4 slow pulse.
Neumann	1923	18 ,,	12 ,, ,,
Lund	1926	14 ,,	5 ,, ,,
Borries	1926	35 ,,	11 ,, ,,
	Total	80 cases.	32 slow pulse (= 40%).

Another peculiar fact is that a slow pulse was encountered not only in labyrinthitis but also in vascularized labyrinthine fistulae, in particular the periodically alternating nystagmus described by me on November 15th, 1919.

It would take us too far here to enter into the theory about this slow pulse in labyrinthitis. I may remind you of the well-known oculocardiac reflex, in which digital compression of the bulb of the eye is followed by a distinct decrease of the pulse-rate ; and I may add, that in many cases of infectious lesions of the eyeball (iridocyclitis, etc.), I have also been able to demonstrate a decrease of the pulse-rate. It will suffice here to emphasize that a *slow pulse in patients with otitis is often a sign of labyrinthitis or labyrinthine fistula.*

With regard to nystagmus: As you know, after mastoid operation we examine a patient for post-operative nystagmus in order to see whether a labyrinthine reaction has appeared or whether a pre-existing nystagmus has changed. There is one condition we have to take into consideration, namely narcosis nystagmus. There is at the beginning of narcosis a marked nystagmus in the direction of the movement of the eyeball. This nystagmus is absolutely straight and equal to either side, but it disappears when the patient looks straight ahead. It vanishes in full narcosis, but it reappears when the patient awakes. I have mentioned this form of nystagmus because I have examined a number of patients who were operated on, under ether anæsthesia, for non-otological lesions (mostly for abdominal conditions) and in whom I noticed that such a narcosis nystagmus of an apparently pathological degree often persisted long after the patient had recovered from the narcosis. It was not infrequently present twenty-four hours after the anæsthesia had ceased, in a few cases even forty-eight hours afterwards.[7] This condition should be taken into consideration on examination for nystagmus in newly-operated patients, as one might otherwise think that the present nystagmus was produced by a labyrinthine or intracranial lesion.

In the diagnosis of intracranial complications, more rare diseases may occasionally give rise to mistakes. Tonndorf[8] has reported the case of a man who had one day some trouble in opening his left eye and therefore on *June 1st* he went to the ophthalmological

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clinic, where a facial spasm was noted. The patient was then referred to the neurological clinic, and there too a facial spasm was diagnosed, but as the patient was also suffering from chronic purulent otitis media on the same side, he was transferred to the otological clinic, where a radical operation was performed on *June 3rd*. On *June 5th*, two days after the operation, he had trouble in opening his mouth, and as this persisted, he walked, on *June 9th*, all the way (1·5 Km.) back to the neurological clinic. Here the correct diagnosis—*tetanus*—was now made for the first time. It was first thought that this infection was a result of the ear operation, but it was found that the infection had taken place through a slight injury to the left eye that had been overlooked—a tiny lesion incurred on *May 28th* by getting a little particle of emery in the eye.

This case history, with the patient walking for a whole week from one clinic to another before the diagnosis was made and going through an unnecessary operation, is very instructive. The literature contains several other reports of cases in which tetanus with facial paralysis has been mistaken for otogenous intracranial complications. I have myself seen a case of tetanus in a patient who was admitted for otogenous meningitis. This case is described in a paper in the *Revue d'oto-neuro-ophtalmologique*,[9] where I give a survey of all the cases of otogenous tetanus reported up to that time. The chief point is, then, that if we are faced by a patient with facial spasm or facial paralysis of uncertain origin, we must not forget to think of tetanus. And we must keep in mind that tetanus may originate from a middle-ear suppuration without any previous trauma.

I will now turn to the differential diagnosis between abscess of the brain and meningitis, or rather to one point of this question where I think I have contributed something to the diagnosis.

In 1900 Rupprecht[10] reported a case which brings the question before us. This was the case of a man with typical signs of diffuse otogenous leptomeningitis: rigidity of the neck, opisthotonus, drowsiness, herpes, and turbid spinal fluid. It was only natural to make the diagnosis of otogenous meningitis. It was therefore rather astonishing when the autopsy revealed an uncomplicated abscess of the brain and no meningitis.

It is not surprising that a case like Rupprecht's attracted such wide attention and that various investigators at once began to look for an explanation of the remarkable fact that an uncomplicated walled-off abscess of the brain gave a marked turbidity of the spinal fluid, although there were no visible signs of meningitis.

Nor is it to be wondered at that the most obvious explanation was looked into at once—namely that there must still be a condition of meningitis, not a visible meningitis, but a microscopic

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inflammation of the meninges. This assumption turned out to be correct.

After this finding it was thought at first that the question was now settled: Turbid spinal fluid indicates meningitis, and if the inflammation of the meninges is not visible to the naked eye, it can be demonstrated under the microscope. But actually the problem had not really been solved at all, or, to put it more precisely it had not been realized that this was not merely one problem but two problems, and that the problem which had been solved was not the main point at all but quite a secondary matter.

What had been ascertained under the microscope was merely this—that turbid spinal fluid may be due not only to a visible meningitis, but also to an invisible inflammation of the meninges. But clinically this did not mean any advance whatever. For what good is it to us in a case like Rupprecht's to know that there is a meningitis present, as long as this meningitis is an entirely microscopic process, " minimal meningitis ", of no essential importance, when we fail to realize the main thing, the presence of an abscess?

Here we are at the central point. The important point is not the pathologico-anatomical question: " May an invisible meningitis be demonstrated microscopically *post mortem*? " More important is the clinical question: " Is it practicable *in vivo* to demonstrate that this meningitis is not an uncomplicated meningitis proper, but a meningitis secondary to an abscess? " In other words: " Is it possible to demonstrate the abscess? If to-morrow we meet with a case like Rupprecht's, will we be able to diagnose the abscess? "

This question was long unsettled, but the answer is, I think, given by the rule (symptom complex, or syndrome) which I was the first to describe at a meeting of the Danish Otolaryngological Society, on April 4th, 1917, and have further elaborated in several subsequent papers.[11]

My starting point was the view that the meningitis induced by an abscess of the brain—or by a subdural abscess—is in some instances not identical with the ordinary meningitis induced directly by mastoiditis, but presents a quite particular clinical type—so special in its features that it is possible clinically to differentiate during life between this type and other forms of meningitis. Its special character comes out particularly in two properties, namely, its strikingly benign course and its relative independence of other conditions.

As a matter of fact, it is possible, even in fatal cases in which the patient dies from the brain abscess, to see this type of meningitis take a quite benign course which does not correspond to the course of a fatal meningitis but to the course of a meningitis proceeding

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to recovery. This is in keeping with the *post mortem* findings where the meningitic process may be so inconspicuous that it cannot even be seen with the naked eye, as in Rupprecht's case—a minimal meningitis, as I have called it. But in order to ascertain this benign character it is not enough to make a single lumbar puncture ; the changes developing in the spinal fluid are studied comparatively in two or more punctures. I set up the term " benign spinal fluid picture "—in analogy with the " benign picture " of a disease—as evident from the findings that the fluid keeps sterile through several punctures, or the turbidity subsides, or the microbes disappear, even when the patient dies from an unopened brain abscess.

I then set up the rule that such a " benign spinal fluid picture " coinciding with a malignant picture of the other clinical symptoms, suggests a secondary meningitis, most often secondary to a cerebral, cerebellar, or subdural abscess. In doing this I diagnose the abscess simply by ascertaining the benign character of the meningitis, not the *simple* benignancy but what I would call the *paradoxical benignancy*. You must not think that to demonstrate the benign character of a meningitis is something rather vague. We are not dealing here with a simple difference in the degree of the spinal symptoms.

The simple benignancy is found : (1) In all cases of meningitis in the initial stage ; (2) in all light cases of uncomplicated meningitis. The *paradoxical* benignancy, however, is characteristic of a secondary meningitis, induced by an abscess.

But even though we characterize this benignancy as surprising and paradoxical, we do not wish to imply by this that it is quite inexplicable or mysterious. From a bacteriological point of view it is not inexplicable that when an infection, instead of extending directly from the middle ear into the subarachnoid space, has to pass first through a brain abscess, it becomes less virulent than the infection which extends directly from the middle ear to the subarachnoid space.

The abscess has an attenuating effect on the infection. For the sake of illustration this may be expressed in popular terms as follows: As microbes and toxins pass through the abscess on the way from the mastoid cells to the meninges, they have to go through a sort of passport station where the most offensive individuals are retained, while the others have to give up their arms.

So the feature on which the diagnosis hinges is not the benignancy in itself but the paradoxical benignancy, in other words, the contrast between the benign course of the spinal fluid symptoms and the malignant course of the other clinical symptoms—that is, the contrast syndrome or " le syndrome de discordance " as it has been called by a French author.

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This is not merely a theory; it is a rule that can be used in practical clinical medicine.

Only six months after my first paper on this subject in 1917, E. Urbantschitsch[12] reported to the Austrian Otological Society some similar experiences, and several other authors have later confirmed the correctness of this observation.

It is more convincing to see case records in which the syndrome was found. For example we may take the case described by Berggren.[13] The patient was a boy, aged 8 years, with a bilateral acute suppurative otitis media, severe pain, beginning drowsiness, positive Kernig's sign, and slight rigidity of the neck. The first lumbar puncture showed turbid fluid, containing 200 cells per c.mm. A bilateral mastoid operation was performed. The next day he was feeling better and the second lumbar puncture showed 100 cells per c.mm.

The improvement kept on until the fifteenth day after the operation, when the condition of the patient became aggravated. He was now somewhat stuporous and vomited twice. The third lumbar puncture gave a perfectly clear fluid with only two lymphocytes per c.mm. Neurological examination showed no abnormality except a little occipital rigidity.

In the afternoon of that day respiratory paralysis set in suddenly, but the author had enough presence of mind to institute artificial respiration at once and operate on the patient. The operation revealed a cerebellar abscess. The patient recovered.

Turning again to Rupprecht's case, the first lumbar puncture showed a marked turbidity of the spinal fluid with numerous polynuclear leucocytes, but no bacteria. Five days later the second lumbar puncture gave a slightly opalescent sterile spinal fluid, although the patient died the next day.

Even though lumbar puncture was performed only twice in this case, it is quite plain that the aforementioned contrast between the benign spinal fluid picture and the malignant course of the other symptoms was present here: the spinal fluid cleared up and remained sterile, in spite of the fatal outcome of the case. An uncomplicated meningitis that kills the patient shows no such course of the spinal fluid findings.

Otogenous encephalitis.—Naturally the classification of otogenous intracranial complications is not quite the same in the different textbooks published in the various countries, but the classification given by Koerner[14] in his famous monograph of 1925 may be taken as a general type.

I. External pachymeningitis and extradural abscess.
II. Interdural (interlamellar) pachymeningitis and abscess.
III. Internal pachymeningitis and subdural abscess.

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IV. Purulent leptomeningitis.
V. Serous meningitis and serous meningo-encephalitis.
VI. Sinus-phlebitis and sinus-thrombosis and otogenous pyæmia.
VII. Brain abscess.

In this classification it will be noticed that, while inflammations of the dura occupy no less than three chapters, and hence are divided into three separate diseases, the non-purulent encephalitis is not even registered at all as a disease.

This view—that the non-purulent inflammation of the brain tissue is not to be considered a special disease, but merely a preliminary stage of the abscess—is the conception generally adopted in international otology.

In 1921[15] I first advanced the view that otogenous non-purulent encephalitis ought to be set up as a disease in itself. This means that besides the cases in which encephalitis is a preliminary stage, or an abortive stage of the abscess, there are also cases in which there is a non-purulent hæmorrhagic encephalitis without tendency to suppuration—analogous to hæmorrhagic encephalitis in whooping-cough, scarlet fever, etc.

This non-purulent encephalitis, I think, ought to be recognized as an independent disease.

My reasons for this are as follows: If, as is usually assumed—otogenous encephalitis were always a primary stage of the abscess, the abscess would be demonstrable, either on operation or on *post mortem* examination. Of course this does not apply to the quite slight and undeveloped abortive cases, but in all the fully-developed cases the abscess must be demonstrable either on operation or on autopsy. But, as a matter of fact, we meet with cases that were not abortive but fully developed, with pronounced cerebral symptoms, general and local—even fatal—without any abscess.

There will, for instance, be cases with aphasia and hemiplegia; the presence of an abscess is excluded by the negative brain puncture and diffuse leptomeningitis is excluded by the normal spinal fluid. The patient recovers. What other diagnosis is possible in such a case but non-suppurative encephalitis?

Or take another case: A patient dies with cerebral symptoms, and the autopsy shows a non-suppurative encephalitis but no abscess and no leptomeningitis. In such a case we have to say the patient died of his encephalitis; we cannot say he died of an abortive stage of a brain abscess. A disease that is serious enough to kill the patient is a disease and not an abortive stage of another disease. We die of a *disease*, not of an abortive condition.

Transitional cases.—Now, I should not be surprised if the objection was raised that there are transitional cases between

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non-suppurative encephalitis and brain abscess and, hence, that the encephalitis is not to be considered a disease but merely a preliminary stage of the brain abscess. I by no means wish to deny that there are transitional cases between this type of encephalitis and the brain abscess, but I wish to emphasize that, on the whole, the existence of transitional stages between two diseases in no way contradicts the assumption that the two diseases are independent ones. Transitional cases are found in any branch of clinical medicine. As you know, there are transitions between serous and suppurative otitis, between pneumonia and abscess of the lung, between an ordinary phlegmon and an abscess in the connective tissue. Yet nobody will deny that each of these diseases is an independent nosological entity.

Simple (?) or hæmorrhagic (?) encephalitis.—The question now arises as to when we are to attribute these mild encephalitic symptoms to a so-called serous or simple encephalitis, and when to an hæmorrhagic encephalitis.

I am not able to give a final answer to this question. It can be difficult enough clinically to differentiate between hæmorrhagic encephalitis and abscess of the brain, but—with our present knowledge—to differentiate between hæmorrhagic encephalitis and simple encephalitis is almost impossible.

It is quite justifiable to assume that the severe cases are chiefly due to hæmorrhagic encephalitis—we know that from findings at the operating table. But it is not possible now with certainty to answer the important question whether a simple non-hæmorrhagic encephalitis may also give focal symptoms. Yet I think we have to reckon with this possibility. At present we can merely say: (1) that probably simple and hæmorrhagic encephalitis may both give focal symptoms, and (2) that probably very pronounced focal symptoms are suggestive of hæmorrhagic encephalitis rather than of simple encephalitis.

Symptoms.—Time will not permit me to go into details of this question, so I will have to refer you to my previous papers on encephalitis. Here I shall merely emphasize two points:—

(1) In a good many cases the symptoms are very slight, only a little pronounced, and not very typical. In many cases, no doubt, there are practically no symptoms at all—or, what amounts to the same, in other words—in many cases the diagnosis is not made at all. For instance, we now and then encounter cases with indefinite or vague cerebral symptoms and optic neuritis. The spinal fluid is normal, and the patient recovers. In my opinion most of these patients have encephalitis.

(2) As to the severe and more pronounced cases, it may be said in general that the symptoms are on the whole the same as the

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symptoms of brain abscess. I have suggested the rule that otogenous encephalitis is characterized by the syndrome, *symptoms of abscess without an abscess.*

We find, for instance, hemiplegia or aphasia. The spinal fluid is normal. A brain puncture is performed but no pus is discovered and the patient recovers. Or the patient dies, and the autopsy shows a non-suppurative encephalitis, but no abscess. In other words, it is not so much the special symptoms as the course of the case that is decisive. The diagnosis is made on the absence of the supposed abscess.

It is interesting to see that the literature contains a number of case histories in which this syndrome—symptoms of abscess without an abscess—was present, and in which the diagnosis was not made by the surgeons, the cases being characterized as quite obscure and quite mysterious. Yet in these cases the diagnosis is quite clear if only we reckon with this nosological entity.

In other cases, however, the diagnosis was made, as, for instance, in some very interesting cases reported by Symonds,[16] who still considers the encephalitis to be a preliminary stage of the brain abscess, and by Key-Åberg,[17] who subscribes to my view of encephalitis as a disease *per se.*

It is true that a so-called serous encephalitis is mentioned in the textbooks, but it is there combined with serous meningitis, forming the so-called serous meningo-encephalitis, and in practice this condition is taken into consideration very little.

It is also true that hæmorrhagic encephalitis is mentioned in the textbooks, but always as the primary stage of an abscess. A pronounced severe hæmorrhagic encephalitis, however, giving distinct symptoms which dominate the whole clinical picture of the case and may even kill the patient—that is something which is practically never reckoned with.

Only when evident facts compel the otologist to adopt a new point of view, does he realize that the old nosological entities do not suffice and that there is a missing link which has to be taken into account in practical clinical work also; this missing link is the non-suppurative encephalitis.

To realize the existence of this disease is important not only for diagnosis but also for treatment. Say, for instance, we have a patient with hemiplegia and aphasia; we perform brain puncture, and no pus is discovered. If now we do not know there exists a non-purulent encephalitis that may cause these symptoms, we shall think that the patient's only chance depends on our finding an abscess, and we shall perhaps feel it our duty to perform deeper punctures and repeated punctures—perhaps with serious harm to the patient.

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And our view of the prognosis will change too. Practical experiences have taught us that even apparently hopeless cases of encephalitis—with hemiplegia, aphasia, etc.—may go on to complete recovery.

All my publications on this question since 1921 point emphatically to a definite aim, namely the establishment of the otogenous non-purulent encephalitis as a clinical entity *per se*. Every textbook of otology should devote a special chapter to this disease.

Discussion.

Mr. Eric Watson-Williams said it was very interesting to find in Dr. Borries' paper so much which gave rise to thought, and so much new material. It was, to him, a new idea that labyrinthitis might be signalized by bradycardia. On looking up his own series of cases he had found a record of one patient with a pulse rate of 60, but no other symptom.

On four occasions he had been asked to " see a patient with brain abscess ", and had found the patient lying happily in bed, looking fairly well but with a pulse rate of about 50. A month ago he had had a patient whose pulse rate was only 48, and little was wrong with him apart from that. He had had influenza a week before being seen, and he had some purulent nasal discharge—it was a sinus case. This was an example of what he, Mr. Watson-Williams, called a " lazy " heart, occurring in a young adult male, whose pulse-rate, lying in bed, might be remarkably slow ; the bradycardia disappeared when he sat up.

He had been interested in one or two cases of intracranial suppuration in which there was extreme pulsus paradoxus. One was in a patient presented at the meeting held at Bristol in June, 1935, convalescent after a brain abscess. The pulse-rate had varied between 46 in inspiration and 72 in expiration. He had had another patient with the same phenomenon who had a cerebellar abscess.

Dr. Borries had mentioned post-operative nystagmus. One of the " catches " was a congenital nystagmus noticed for the first time after operation. Until one had made up one's mind that it was congenital it was somewhat disturbing. But such a patient would not have any vertigo.

An important point in Dr. Borries' paper was that of the distinction between brain abscess and meningitis. That was a problem which might arise at any moment, and particularly in the small hours of the morning, when laboratory resources were not available. There was often no available history, as the patient was brought in comatose, and one had to rely considerably on clinical signs and the results of lumbar puncture. But when a cerebellar abscess was suspected he (the speaker) always performed lumbar puncture with trepidation, and he postponed that step until the patient was on the table. One could find out as much by looking at the cerebrospinal fluid at that point as by performing lumbar puncture while the patient was in bed and transporting him to the table later.

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The differential diagnosis could be extremely troublesome. Following the teaching of Dr. Greenfield, he had been in the habit of laying emphasis on the question of the chloride content of the spinal fluid. He agreed that where the cerebrospinal fluid showed a diminution in chloride below 0·7 per cent. there was certainly meningitis, yet he had seen several cases of meningitis in which there had not been, in the early stages, a reduction in the cerebrospinal fluid chloride. And if there was a suppurative meningitis, it was in the early stage that some good could be done. By the time the patient presented the textbook clinical picture of meningitis, the opportunity for successful intervention had, in three cases out of four, been lost. It was important to keep in mind that the cerebrospinal fluid might improve while the patient himself was not improving. Nine months ago he had seen a woman who had been brought in comatose with meningitis due to chronic mastoiditis ; she wriggled a little when the mastoid was pressed, but gave no other evidence of hearing or feeling. He performed a radical mastoid operation and nothing more ; lumbar puncture was performed on the table and the spinal fluid was just opalescent. The report the next day was that though the fluid was turbid there was a normal chloride content, and a polymorphonuclear leucocyte count of less than 50 per cent. Those symptoms were more suggestive of abscess of the brain than of suppurative meningitis. The patient improved greatly after the radical operation ; he (the speaker) thought she was stupid, but her husband thought she was normal. She was watched for four days ; the symptoms of meningitis cleared up, but the pulse gradually became rather slow, and on the fourth day she suddenly became unconscious. He was glad he had had that early cerebrospinal fluid report, as she had shown no localizing signs, but he thought the report suggested the presence of a temporo-sphenoidal lobe abscess and such an abscess was opened by operation. The patient made a complete recovery. The mental condition nine months later had shown no change, and that seemed to confirm the husband's view that what he (the speaker) had considered a stupid mentality was normal to her.

Mr. E. D. D. Davis said that Dr. Borries' paper had presented a new aspect of the subject. The Section had had many discussions on intracranial complications, but he did not remember one in which so many new points had been raised.

He had seen one or two cases of cerebellar abscess which had left the hospital, and subsequently a report of sudden death had been received, yet there were no signs of abscess.

There was also the interesting point as to the course of a brain abscess. One liked to wait for a brain abscess to localize and for the signs to become definite before operating, and if one was able to wait it was valuable to do so.

He had been surprised to learn that the cell-count of the cerebrospinal fluid, instead of increasing, showed improvement in fatal cases. In the past one had relied on the fact that the fluid was steadily improving as a supposed indication that localization was occurring and that it was not too dangerous to delay. In the cases quoted by Dr. Borries, though the fluid had improved, the patients had died.

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The subject of encephalitis was interesting. He had seen cases in which the signs and symptoms of brain abscess were not very definite and he had intended to wait, but his hand had been forced, and so he had exposed the dura mater and found its appearance normal—no thickening, etc., being apparent. The area of the brain was explored thoroughly by a needle, but with no result, and the patient unexpectedly recovered. A certain diagnosis of such a case was difficult. He assumed that Dr. Borries carried out lumbar puncture on every possible occasion. He, Mr. Davis, regarded it as a most valuable procedure, and he had never known any harm arise from it if only a small quantity of fluid was removed. Sixteen years ago a bogey was raised by Dr. Weed and Dr. Wegeworth of the Rockefeller Institute, who found that if lumbar puncture was carried out when septicæmia was present, meningitis occurred. He (the speaker) had since been on the look-out for such a case, but had never seen one. A blood-culture could be made first, and if septicæmia was present one would then hesitate to carry out lumbar puncture. He would like to hear more from Dr. Borries about the diagnosis of encephalitis by lumbar puncture and the examination of spinal fluid, and whether in most of his cases the patients recovered by simple rest. He understood that some recovered though the brain was not explored.

The question of the early diagnosis of meningitis interested him very much; it was, however, a big subject. He was relying more on the detection of those conditions likely to lead to meningitis. For example, he regarded a case of scarlet fever, or one of measles, with severe osteomyelitis as dangerous. Patients of ages up to 20 years were rather more susceptible to meningitis than older people were. Another type of case in which meningitis might be expected was that in which the patient was desperately ill with an acute streptococcal mastoiditis.

How could one detect meningitis early ? One of the first symptoms was severe headache, with a slight mental change. A child would show a disinclination to play with his toys as usual, or might develop a changed mental state. Stiffness of the neck was a late sign of manifest meningitis. Such indications demanded immediate lumbar puncture, which would lead to the earlier detection of meningitis.

Mr. RITCHIE RODGER said that the subject of sudden deaths from cerebellar abscess was of great interest. His own attitude towards the question of early operation had been for many years influenced by two incidents during the Great War when he was in charge of the aural cases in the Royal Naval Hospital in Hull. In the first two cases of cerebellar abscess he met with the patients had died suddenly before operation. He had seen each patient late at night, arrived at his diagnosis, and arranged to operate first thing the next morning. Both had died in the night. His diagnosis of the cause of death was pressure on the respiratory centre, as Dr. Borries had stated. It would be remembered that a discussion on the subject of brain abscess was opened by the late Sir Percy Sargent a few years ago, and at that date a swing of the pendulum towards late operation had taken place. Macewen's results were quoted at that discussion, and there seemed

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to be agreement that the excellent results of Macewen were attributable to the fact that, in most cases, he operated late. The extreme position on the subject was expressed a few weeks ago by Mr. Jefferson, of Manchester. In the course of a lecture in Hull Mr. Jefferson had given a description of a case in which he had excised a brain abscess, instead of draining it. He (the speaker) had asked Mr. Jefferson whether he thought it was possible to do so in any large proportion of cases, and the reply was that he could not answer the question, but he thought it would be a good rule in cases of brain abscess when the diagnosis had been definitely made, to say, "We shall operate in three weeks if the patient is still living."

Mr. SYDNEY SCOTT said that he greatly appreciated Dr. Borries' paper. He had met examples of cases similar to those which Dr. Borries had described. Listening to Dr. Borries his mind recalled a case of "Acute Internal Hydrocephalus, secondary to Streptococcal Infection of the Labyrinth," which he had reported in the *Archives of Otology*, 1908, xxxvii., No. 2.

Dr. BORRIES (in reply) said he would suggest that Mr. Sydney Scott's case was one of localized serous meningitis; this might have given the same indication as cerebellar abscess or cerebellar tumour. Distension of the posterior fossa had occurred, and there had been compression of the medulla into the foramen magnum. The lateral ventricles had been distended and contained fluid, and there might have been œdema of the brain, which, however, was very rare.

As to the cause of sudden death: There occurred obstruction followed by distension, then more obstruction and more distension, and death ended the series. In cerebellar abscess respiratory paralysis might occur after lumbar puncture, especially if a considerable quantity of spinal fluid was removed, as had been the custom in earlier days.

If the examination of the pulse in cases of labyrinthitis were made every hour, a slowing would more often be detected; the pulse-rate varied at different times of the day in the same patient. The serous stage of labyrinthitis was a very short one, lasting perhaps no longer than half a day, and in the purulent stage of labyrinthitis one might not find any slowing of the pulse. It was desirable to examine this question further. The quickening of the pulse in these cases when the patient sat up in bed might be due to intracranial conditions.

He had cured two of his cases of encephalitis. One, an extremely serious case, was that of a young man with hemiplegia and aphasia. He was now a practising physician in Denmark. The other patient had almost the same symptoms; she was a woman aged between 35 and 40, and had made a perfect recovery. Ten years later she had gone to hospital because of another condition, and had died. At autopsy a cavity in the brain was found, filled with clear fluid but without pus. This might have been the result of an encephalitic process, which had become resolved.

With regard to the early diagnosis of otogenous lepto-meningitis, he did not know any better guide to this than a cell-count made immediately after the puncture.

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The Prescribing of Hearing Aids

By TERENCE CAWTHORNE (London)

IT would be difficult to imagine any branch of otology that has received less attention from otologists in the past than the subject of hearing aids. In consequence our incurably deaf patients have been forced to turn elsewhere for advice on the choice of an aid. Either as a result of our recommendation, or in response to an attractively worded advertisement, the deafened patient seeks the help of the retailer of hearing aids. At his best this retailer

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is a man who relies for his customers upon the goodwill of the medical profession, and he genuinely tries to remedy the hearing defect with the most suitable aid at a reasonable cost. At his worst the retailer attracts customers by extravagantly worded advertisements, the cost of which is reflected in the price of his instruments. Also he may be willing to sacrifice efficient performance to appearance.

Unfortunately, in the past, this latter policy has paid, owing to the aversion of the hard-of-hearing to advertise their disability by wearing a hearing aid. Most of us have deplored this fact and have hoped to see the day when a hearing aid attracted no more attention than a pair of spectacles. This can be only if and when the results of accurate prescribing and dispensing are as successful for hearing as they are for seeing, which at the moment is not the case.

The correction of a hearing defect presents so many complex problems that their complete solution is not possible in the present state of our knowledge. The invention of the thermionic valve has, however, so increased the range of the measurement and amplification of sound that already there has been a great increase in the efficiency of hearing aids, so that we may look forward to the day when we can expect as good results from prescribing hearing aids as we now do from prescribing eye-glasses.

The prescribing of a hearing aid calls not only for an accurate estimation of the hearing loss over the whole audible range, but also for a knowledge of the optimum amplification desirable for the different frequencies. This should always be the concern of the otologist. Armed with this knowledge and knowing the main use for which the aid is required, he can say which type of aid is likely to be most practicable. An idea of the performance of the various aids is essential but unfortunately it cannot, in many cases, be more than an idea, as there are not at the moment any standards of performance for our guidance and the patient's safeguard.

The actual dispensing of an aid is, of course, a matter for the technician-retailer who, with advantage, may also be the manufacturer. His business is to supply, primarily on loan, an apparatus whose performance corresponds as nearly as possible to the prescription of the otologist. The patient should again be tested with the aid to see that the prescription has been accurately dispensed. The importance of this step does not always appear to be realized either by the patient or the otologist, and more often than not it is omitted.

In brief, the greatest service that the otologist can offer his incurably deafened patient is to guide him in the choice of a hearing aid.

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In order to be able to do this with any degree of accuracy and success, the following factors must be considered :—

(1) The nature and extent of the hearing defect.
(2) The type and performance of the available hearing aids.
(3) The personal feelings and pocket of the patient.
(4) The main use for which the aid is required.

Before going into these factors in further detail, I will discuss briefly the measurement of sound intensity.

In order that there should be a definite understanding between the prescriber and the dispenser of hearing aids, it is essential that some clearly understood unit of hearing loss should be employed. The most desirable unit of sound intensity has been the cause of much dissension in the acoustical world, the final result of which is not yet apparent. At the present moment the most favoured unit is the decibel, and it has the merit of being generally used by most workers in acoustics.

This decibel represents the smallest increase in sound-intensity that can be appreciated by the human ear under normal conditions. It is a logarithmic unit, that is to say, 10 decibels correspond to a tenfold increase in sound intensity, 20 decibels to a hundredfold increase, 30 decibels to a thousandfold increase, and so on. The maximum range between the threshold of sound and the threshold of feeling lies in the region of 2,000 cycles per second and extends for 140 decibels. The level for conversational speech in average surroundings is 55 decibels. This figure depends upon the surrounding noise, because with normal hearing the voice is unconsciously kept as far as possible from 10 to 25 decibels above the level of the background noise. The ear is most sensitive to sounds in the middle range of frequencies between 500 and 2,000 cycles per second, and fortunately it is within these limits that most speech sounds occur.

To return to the factors concerned with the prescribing of hearing aids :—

(1) *The nature and extent of the hearing defect.*—An accurate estimation of this is essential. The nature of the hearing defect is the first consideration, because all forms of conduction-deafness, however severe, will derive benefit from an aid. The use of an aid in perception-deafness however is limited by its degree. In cases where the total loss is not more than 50 per cent., an aid is likely to help, but in more advanced perception cases the benefit is very limited. The explanation of this is that in conduction-deafness, any amplification is absorbed by the defective conducting apparatus and sounds of normal intensity reach the cochlea. In

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perception-deafness there is no barrier to the passage of amplified sounds to the cochlea and, in consequence, those parts of the cochlea which are functioning may be over-stimulated and the result will be confusion and possibly discomfort.

Having determined the nature of the deafness, the next step is to estimate its extent. This can be done by the use of calibrated tuning-forks or the audiometer. The latter may be used for testing pure tones, or for speech. The advantage of the audiometer over the tuning-fork is that its output can be raised at will, or kept constant, and that it is capable of accurate measurement. Despite the possible objection to the use of headphones with an audiometer, I have no hesitation in saying that it is far quicker and far more accurate than the tuning-fork for measuring capacity for pure tones.

As the main reason for wanting a hearing aid is usually an inability to hear normal speech, ordinary methods of testing the speech-hearing capacity are not of much use. At the same time it is of the greatest importance for prescribing purposes to find out what degree of amplification is required to enable a patient to hear normal speech. It is not sufficient to know the hearing loss in decibels for the various pure tones, because it has been found that the provision of an aid which more or less exactly corrects the pure tone defects does not necessarily give the best results for speech. Therefore, in addition to calibrated tuning-forks, or a pure tone audiometer, some form of calibrated speech-amplifier is desirable. This can conveniently be made by the incorporation of a speech circuit in an audiometer which enables the output of the voice to be amplified to a measurable extent, irrespective of the input. In other words such a device is nothing more or less than a hearing aid, with the added advantage that its performance can be measured in decibels, or whatever other unit is chosen. I am sure that it is only by using such an apparatus, possibly with the incorporation of various filters to give selective amplification, that the best results from prescribing aids are to be obtained.

The most satisfactory way of assessing the performance of any amplified speech circuit is to employ monosyllabic words, each of which has been chosen to accentuate a different vowel or consonant sound. As there are less than forty different speech sounds in English, the list of words to be used need not be very long. This method of speech testing has been perfected by telephone engineers and is of value to the otologist, not only in his preliminary tests, but also in assessing the performance of any aid.

I think that possibly the most significant indication of the value of the audiometer is that most retailers of hearing aids not only make full use of it but in some cases even offer to supply the doctor with an audiogram.

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(2) *The type of hearing aid.*—Naturally, any prescription will be governed by the performance of the hearing aid, and a knowledge of the advantages and drawbacks of the different types of aid is important. They may be conveniently divided into non-electrical and electrical.

The non-electrical consist of horns, auricles, and speaking tubes. They serve as collectors of sound waves and so bring the speaker nearer to the listener and exclude extraneous sounds. Some of the horns also act as resonators. Their power of amplification is limited (somewhere about 12 to 15 decibels), and their chief use is for severe cases of perceptive deafness where much amplification would only bring the amplified sound near the threshold of feeling and so cause confusion.

The electrical aids. These consist essentially of an amplifying circuit connected to a microphone, and a receiver. The amplifying circuit may either be a small dry cell or it may be a series of wireless valves fed by a large dry cell and an accumulator. For the sake of brevity these will be called " battery-operated " and " valve-operated " respectively.

The battery-operated circuit is capable of a maximum of about 60 decibels of magnification, whilst the amplification of the valve-operated circuit is only limited by the number of valves and size of the set. In the usual portable variety where there may be as many as three valves the amplification may reach 100 decibels.

The microphone is the most important part of the set. The carbon-granule type of microphone is the most commonly used because it is the only one that transmits sounds sufficiently intense to admit of the circuit being operated by a small battery. Unfortunately this is the least efficient type of microphone and an added objection is its tendency to develop internal noises. By altering the quality and arrangement of the carbon granules it can be made more sensitive to the higher frequencies, and this is of importance when prescribing such an aid where high tone amplification improves the performance.

Much more efficient microphones are the crystal type and the condenser microphone. Because of the weakness of the intensity of their response, they can only be used with a valve circuit. Their performance is so much better than that of the carbon-granule microphone that there is no doubt that in practically all cases in which an electrical aid is required the valve-operated set will give by far the best results.

The receiver may operate either by air- or bone-conduction. The most efficient one is undoubtedly a high quality telephone receiver and its only drawback is in its appearance. Small receivers chosen for their inconspicuousness sacrifice much of their efficiency for the sake of appearance. The bone-conduction receiver

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has a limited use for those cases in which the amplified sound by air-conduction would still be 30 per cent. below normal. The sphere of usefulness of this type of receiver is much less than would appear at first sight, because hearing by bone-conduction is 30 per cent. less efficient than by air-conduction, and is by no means free from distortion. Each receiver has a characteristic response which amplifies certain frequencies more than others, so that by choosing an appropriate receiver, different kinds of frequencies may be accentuated.

To summarize : By altering the microphone and receiver, much can be done to suit an electrical aid to a prescription, and it is possible that with the valve-operated sets selected amplification for any kind of frequencies may be obtained, although up to the present it has been found that the best results are obtained either with uniform amplification or with increased amplification of the higher frequencies. However, there is no doubt that improvements in hearing aids will be on the lines of selective amplification.

(3) *The personal feelings and pocket of the patient.*—This is by far the most difficult factor in prescribing a hearing aid. In the first place every patient, not unnaturally, wants his set to be as inconspicuous as possible. That is, one presumes, why battery sets and bone-conduction, or tiny air-conduction, receivers are so popular. Although these sets are by no means always the best for the patient, he often chooses them for their appearance and size. Another factor is the upkeep. The battery set requires a new battery fairly frequently. The valve set not only needs a new battery every few months, but also the accumulator requires weekly attention. I understand, however, that for actual cost of upkeep, valve sets compare favourably with battery sets. In the matter of upkeep, the non-electrical aid has the advantage in that all that is needed is to see that the ear-piece is kept free from wax.

The question of price, unfortunately, is an important one for many patients. Roughly speaking, at a reputable retailer's such as one of those recommended by the National Institute for the Deaf, the non-electrical aids vary in price from £1 to £5, the battery-operated sets from £5 to £20, and the valve-operated sets from £12 to £25. Recently a 2-valve aid has been described and its price should be about £6. It is obvious that however desirable they may be, most valve-operated sets are beyond the means of many, and it is to be hoped that the increased use of hearing aids will materially help in reducing their price.

(4) *The conditions under which the hearing aid is to be used.*—This important factor is frequently neglected. For individual speech, a speaking-tube or trumpet is frequently sufficient, although many people find it most disconcerting to have to speak down a tube. An electrical aid obviates this and is consequently preferred

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by most. For general conversation, for a theatre, lecture, or church, one of the valve aids gives by far the best results. Sometimes the patient needs the aid to listen to one particular person, and it is then important to take into consideration the pitch of that person's voice when prescribing the aid.

I think that we can look forward to a future of great possibilities, when the increased efficiency of hearing aids will result in their more general use, and the present high prices will be brought down to suit the pockets of poorer patients. It may even be that a hearing-aid department will be an essential part of every well-equipped otological clinic.

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ABSTRACTS

EAR

Labyrinth Balancing Reactions in Men and Animals following Rapid Tilting round a Longitudinal Axis. A. DE KLEYN and C. VERSTEEGH. (*Acta Oto-laryngologica*, xxiv., 1.)

The experimental and clinical researches on this subject, which the authors have carried out for some years, have been published previously only in Dutch.

Guinea-pigs and rabbits were used for the animal experiments. The board or mattress was so arranged that the animal or person (resting on hands and knees) under investigation could be tilted round, either a longitudinal or a bitemporal axis. In order to obtain pure labyrinth reactions the tilting must be rapid.

The following results were obtained: (1) the reactions that normally follow tilting round a longitudinal axis no longer occur

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