On epilepsy in its relation to ear-disease : a dissertation for the degree of M.D. / by J.A. Ormerod.

With the Author's Compliments

ON

EPILEPSY

IN ITS

RELATION TO EAR-DISEASE.

A DISSERTATION FOR THE DEGREE OF M.D.

BY

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[*REPRINTED FROM "BRAIN," April*, 1883.]

LONDON:
PRINTED BY WILLIAM CLOWES AND SONS, Limited,
STAMFORD STREET AND CHARING CROSS.
1883.

PREFACE.

THE following paper was read last year as a graduation thesis before the Regius Professor of Medecine in the University of Oxford, and is published with a few slight alterations in the April number of 'BRAIN,' 1883. In addition to Dr. Acland, I wish to thank Dr. Andrew, Dr. Hughlings-Jackson, and Mr. Cumberbatch for their kindness in looking over the manuscript. I have also to thank Mr. Cumberbatch and Dr. Urban Pritchard for much practical instruction at their aural cliniques. The importance of the relations between affections of the eye and of the central nervous system is universally admitted; and few will doubt, I imagine, that affections of the organ of hearing have similar bearings. This is a large subject; but in the present paper I have attempted to discuss their connection with epilepsy only.

April 1883.

ON EPILEPSY, IN ITS RELATION TO EAR-DISEASE.

BY J. A. ORMEROD, M.D.

It needs no very extended observation to show that these two phenomena frequently co-exist. In such a case there are several modes of relation possible: either they are simply coincident, or both are the effects of some common cause, or the one is the cause of the other, the epilepsy of the ear-symptoms, or the ear-disease of the epilepsy. It will be my object to discuss these various relations, together with some subjects of interest that suggest themselves in connection with them.

The term ear-disease I shall use with considerable latitude, wishing to consider on the one hand the production of aural symptoms only in the course of a simple epilepsy, and on the other the possible production of epilepsy by a definite aural disease, viz. purulent otitis.

Instead of attempting a regular definition of the term epilepsy, it will be sufficient perhaps to make a provisional grouping of chronic convulsive disorders, and indicate that kind to which I refer. We may distinguish as follows: First, the "hysteroid" fit. The patient is usually a female or a boy. The coma is not so deep but that the patient can be, to a certain extent, roused from it; nor so sudden in its onset but that she avoids hurting herself as she falls. The convulsions are not limited to mere spasms, but co-ordinated movements occur, such as biting, striking at the bystanders, resistance to restraint. Secondly, the epileptiform fit. In this, coma is either absent altogether or comes on late in the attack; the convulsions are apt to spread slowly from some given point, and may be limited to one side; not unfrequently there is double optic neuritis, and post mortem, some localised

structural disease is found. Both these types of fit I have endeavoured in the present paper to exclude. The third kind, which I shall provisionally call epilepsy proper, and to which I limit myself, may be roughly characterised as follows. There may indeed be "warnings," but the coma itself comes on suddenly and early in the fit, and is profound. The patient falls anywhere, often injuring himself. The tongue is often bitten. The convulsions, as a rule, spread rapidly, and are usually bilateral. They may, however, be very limited or very slight. Minor attacks may occur, consisting of the "warning" only, or of transitory coma without perceptible convulsion (petit mal). If paralysis occur, it is incomplete, transient, and immediately consequent upon a fit. There is no optic neuritis, and no definite anatomical lesion can be predicted.

It cannot be denied that in some, perhaps in many cases, the co-existence of epilepsy and ear-disease is merely accidental. The greater the absolute frequency of the two diseases, the higher does the probability of such a relation become, and it is difficult to exclude unless some causal connection can be shown to exist.

Or again, the two diseases may be connected, not by mere accident, nor in the sense that one causes the other, but that they both have a common parentage. The effects of scarlet fever are an example of this. Scarlet fever, by extension of the throat-affection to the tympanum, produces, as is well known, many and severe cases of middle-ear disease. It may also, as Dr. Gowers[1] has suggested, produce epilepsy; an effect due, he thinks, to some peculiar action of the poison on the nervous system. It is tempting where both ear-disease and epilepsy are left after the fever, to regard the ear-disease as the link between the epilepsy and the fever; but this is not always so, for in only one out of Gowers's nineteen cases of epilepsy after scarlet fever did ear-disease appear. It may therefore be fairly said that in some cases epilepsy and ear-disease may result from scarlet fever directly and independently of each other. We may ask whether other acute specific diseases, in which the throat is severely affected, as for

[1] 'On Epilepsy and Chronic Convulsive Diseases,' p. 28.

instance diphtheria, may not sometimes produce the same effect? Acquired syphilis appears to produce deafness in exceptional cases, and sometimes epileptic fits, either of the Jacksonian type, or else, according to Fournier, indistinguishable from those of ordinary epilepsy. In congenital syphilis, on the other hand, deafness is well known, and it seems probable that some cases of epilepsy at an early age may be ascribed to this cause. The two following cases are offered in illustration of this:

A. B., æt. 12, was brought to me at Queen Square Hospital in March 1882. He had incisors of the type described by Hutchinson; the right cornea was dim, the left sclerotic injected in the neighbourhood of the cornea. He had been subject to twitchings of the limbs for eighteen months, and, during the last six weeks, to fits, with loss of consciousness. A sister, about 15 years old, had also characteristic incisors, and had had fits from birth. The mother had had six miscarriages. The mother's brother had also recently developed fits. This last fact makes it probable that the syphilitic taint was not the only agency at work: but in the next case there was no such history of epilepsy in the previous generation.

A. F., æt. 9, had had fits for four years; at first they had been "like faints," accompanied by sleepiness and vomiting, and preceded by loss of appetite: during her attendance they changed to sudden falls, followed by general convulsions. She had the characteristic incisors; there was disseminated choroiditis in both eyes, and she had one or two attacks of keratitis while attending. There was in this case no deafness. The mother had had four miscarriages. A sister, 4 years old, suffers from vomiting, is rather wanting in intelligence, and is supposed to have had a fit.

We turn now to the question, How far can epilepsy be the cause of aural affections? It is not of course asserted that structural diseases of the ear, such as perforations of the membrane, disease of bone, inflammation or chronic thickening of the tympanum, can be so caused; but that certain subjective symptoms commonly met with in aural practice, which must ultimately, like all subjective symptoms, be referred to the

nervous system, may occur in the course of epilepsy uncomplicated, so far as can be ascertained, by disease of the ear. Such symptoms are pain in the ear, noises in the ear, deafness; and on the other hand, vertigo, rotatory movements or other disturbances of equilibrium, vomiting.

Some epileptics complain of pain in the ear, apart from any aural disease, but I imagine that this can usually be recognised as part of the general headache to which they are so prone, analogous perhaps to the pain of megrim when localised in the eye. Let us, however, note the fact that the pain of actual tympanic inflammation is relieved, like the epileptic headache, by bromide of potassium.

Noises in the ear are due to various causes. Apart from those that are really not subjective, but correspond to an actual physical phenomenon, e.g. the bruits of anæmia and of aneurism, noises may be caused by wax in the meatus, catarrh or chronic thickening of the tympanum, &c.

Irritation of the auditory nerve appears to be the chief modus operandi of these causes; in fact Erb [1] maintains that tinnitus in some cases is due solely to hyperæsthesia of the auditory nerve, and that such cases may be distinguished from others by the curative effect of the galvanic current properly applied. A subjective sound in connection with epilepsy usually occurs as an aura. Since such an auditory aura is held to be rare,[2] I give the following instances:—A woman, æt. 27, epileptic for five years, said that, as the fit came on, she had sometimes a ringing noise in the ear, not always in the same ear apparently. She then fell unconscious; she had bruised herself and bitten her tongue.—A man, æt. 32, had as warning "an indescribable sensation" starting from his big toes, and usually noises in his ears.—Another man had a hissing noise in his ear before the fits.—A fourth had occasionally a "ringing;" a fifth, "a drumming feeling" in the ear, independently of the fits. In all these cases the ears were normal. Where the ears are diseased, noises of course may be caused by the ear-disease only; yet sometimes even then they

[1] 'Elektrotherapie,' p. 231.

[2] Hughlings-Jackson, 'British Medical Journal,' 1877, vol. i. pp. 386, 703; and 'Lancet,' March 11, 1876.

seem to occur in close connection with the fits. Thus a woman with slight and variable impairment of hearing, and subject to a noise in the left ear, which was aggravated by stooping, said that her first two fits "were preceded by noises like rushing water in that ear." Again, a man whose ears were partially filled with wax, said that as an aura he used to have a humming noise in his ears, accompanied by strange sights before his eyes. Possibly such facts may point to a connection between the peripheral and the central disease. From cases of auditory aura we may at least infer that, if as is generally held, the phenomena of the epileptic fit are due to "discharges" in the cells of the cerebral cortex; the sensation of sound usually caused by some disturbance, natural or morbid, of the auditory nerve or its peripheral expansion can be caused also by processes originating in the cortex. Experimental evidence of an auditory centre in the cortex has been offered by Ferrier and others. Pathological illustration of the connection between the action of the auditory nerve and that of the cerebral centres may be sought in the cases where the mere tinnitus of aural disease becomes transformed into aural illusions. Thus a woman, æt. 61, had been epileptic for thirteen years, and deaf for a longer period. Lately, however, her deafness had got much worse, she had noises in the head, and attacks of vague vertigo. The condition of the ears (the membranes were depressed and thickened) seemed enough to account for the aural symptoms, though her inability to hear the tuning-fork through the skull made it probable that the labyrinth or the nerve had also suffered. The tinnitus increased steadily, till she began to hear not only noises, but words, names of streets, songs, &c. She was said to be irritable, but seemed perfectly sane, though subsequently illusions of smell and vision developed.

Roosa[1] quotes the opinions of Schwartze, that "subjective aural sensations, which are caused by demonstrable affections of the ear, may in predisposed persons, especially when there is any hereditary tendency to mental disease, become the direct cause of aural hallucinations, that may accelerate the outbreak of a disease of the brain."

[1] 'Treatise on Diseases of the Ear,' p. 265.

The opposite affection, deafness, may also occur in the course of epilepsy. With regard to infantile convulsions, Itard affirms this in very positive terms. Speaking of what he calls "paralysis of the acoustic nerve following convulsions,"[1] he says, "this cause of deafness is very rare in adult life, and very frequent in early life. When hearing is lost in the first three or four years of life, it is nearly always a consequence of convulsions. A great number of deaf mutes owe their infirmity to this cause, which, destroying the hearing at a very early age, finally entails the loss of speech. It is worth remarking that it is commonly the less violent and less prolonged convulsions which produce this effect. Numbers of infants who had become deaf about the time of dentition, and for whom I had been consulted, had for the most part ceased to hear suddenly, directly after a convulsive attack." Itard himself does not illustrate these remarks, but six cases of the kind are given by Knapp.[2] Arguing from the sudden outset and the complete character of the deafness, Knapp suggests that a serous or hæmorrhagic effusion into the labyrinth causes, on the one hand the convulsions by reflex irritation of the medulla, on the other, the deafness by destruction of the labyrinth. I cannot myself call to mind more than one case of this kind out of a large number of epileptics who have had convulsions in early life; and Knapp himself remarks that "most writers on otology say little or nothing on the subject." But there appears to be a different kind of epileptic deafness, viz. a deafness which may be slight, unilateral, occurring after the fit, or intensified then, and sometimes connected with an auditory aura. A young woman, æt. 19, had had fits in infancy, chorea when 8 years old, and fits again during the last two years. There was no warning; she had occasionally bruised her face and bitten her tongue. After the fits she said a pain went through to the left ear, and she felt rather deaf. The tympanic membranes were normal. A watch (the distance for hearing which was 4 feet or more) she could hear through 4 feet with the right ear, through 40 inches with the left. The tuning-fork applied to

[1] 'Traité des maladies de l'oreille.' Tom. ii. p. 315.

[2] 'Archives of Ophthalmology and Otology,' vol. ii., No. 1, p. 260.

the middle line of the cranium was heard best in the right ear (she was quite positive of this). On one occasion she had had a curious attack; without losing consciousness she turned round and round from left to right, and fell against a table.[1] Another woman, æt. 35, had had fits since her first pregnancy, fourteen years ago, and fainting fits before that. In the fits she bit her tongue and lips, afterwards she felt "lost and silly." Since the fits, she has been quite deaf with the left ear, having been slightly deaf with it for some years previously. Inasmuch as she stated that she had always giddiness and a hissing noise in the head, it seemed probable that she had aural disease, but I could find no evidence of it; the tympanic membranes were normal, and the tuning-fork, whether in the air or upon the cranium, was only heard with the right ear. Moreover, when the fits were stopped by treatment, the deafness certainly improved; she could then hear the watch ½ an inch from the left ear. Two other cases, of which, however, I have no satisfactory aural examination, seem somewhat similar. The one, a single woman, æt. 30, said that after each fit she was partially deaf chiefly with the left ear; she felt a "sort of numbness" of the ear. The other, a man, æt. 21, gave the following account—"it" (i.e. the sensation constituting the aura) seemed to rise up from the toes and leg of the right side into the right ear; he became deaf with that ear at the time, and remained so for two or three days after the fit.

I put forward these cases with much reserve, because it is difficult for any but a skilled aurist to exclude with certainty disease of the middle ear.[2] But analogy seems to show that there may be such a post-epileptic temporary deafness dependent upon a morbid condition of the cortex cerebri. Epileptic patients will sometimes complain of attacks of blindness, while the ophthalmoscope shows the optic nerve to be healthy. Then there are cases of post-epileptic paralysis

[1] This vertiginous attack and the slight internal ear-deafness suggest the possibility of Menière's disease; there was, however, no tinnitus, and the deafness which the patient connected with the epilepsy, was not progressive.

[2] Since writing this, I find a case given by Knapp, 'Archives of Otology,' vol. xi., No. 3, p. 232. A man, æt. 37, had become permanently deaf after epileptic fits; the middle ear and tympanic membranes were normal.

of the limbs, incomplete and transient (in cases of pure epilepsy), affecting the limbs which have been most convulsed, and dependent (as has been shown in cases of gross lesion) upon disease of a corresponding cortical area. Moreover, there are post-mortem records of cortical lesions where there had been deafness during life.[1] But unfortunately we cannot do much, clinically, towards localising the various forms of nervous deafness. Not only are the anatomy and physiology of the various apparatus of hearing less well known than those of vision, but our means of clinical investigation are much more limited in regard to the ear. Perhaps the symptom insisted upon by Knapp,[2] viz. deafness for certain groups of tones, may suffice for diagnosis of disease of the cochlea, or perhaps electrical investigation may afford some clue to the condition of the auditory nerve;[3] but these methods, to say the least, necessitate the intelligent co-operation of the patient, and cannot make up for the lack of an instrument like the ophthalmoscope for the direct inspection of the nerve and its expansion. Even the simple diagnosis by the tuning-fork of nervous deafness (i.e. deafness due to lesion of the internal ear and parts beyond) from middle-ear deafness, rests upon information derived from the patient.

In close connection with the organs of hearing, both by anatomical and pathological relation, stand the organs of equilibrium. If not justified in speaking of a special sense of equilibrium, we are at least conscious of perturbation of that sense, the feeling expressed as vertigo. Vertigo is defined by Brunner[4] as "an impairment of the sense of equilibrium," and described by Ramskill[5] as "the sensation of moving, or the appearance of moving objects, without real movement." It is worth while considering some of the conditions under which this sensation arises. The peripheral organs from which we derive a knowledge of the position of our bodies in space, appear to be of two kinds. First, organs which seem to have a purely sensory function, and which enable us to appreciate

[1] E.g. cases quoted in 'Brain,' October, 1882.
[2] *Op. cit.* p. 235.
[3] Erb, *op. cit.* p. 226.
[4] 'Archives of Ophthalmology and Otology,' vol. ii. No. 1, p. 293.
[5] 'Reynolds's System of Medicine.' Article on Vertigo.

the effects even of passive movements apart from the other senses. These are mainly the semicircular canals. Perhaps certain mobile abdominal viscera may contribute to the same end. Secondly, a combination of the muscular sense with the special senses of sight and touch. Retinal impressions per se give us little information as to the position of objects. To judge of their distance from us, and of their position above, below, or to either side of us, we must appeal to the muscles which accommodate the eye and move the eyeballs. A similar combination of touch with muscular effort aids us in the absence of the sense of sight. Thus a patient with locomotor ataxy, whose muscular sense is impaired, finds it difficult to stand with his eyes shut; but the difficulty is very much increased if the soles of his feet have become anæsthetic. Whether the sense of hearing can enter into any such function appears extremely doubtful; the other senses certainly do not.

When the report of the special senses and of the muscular sense are inharmonious, vertigo is the result. This is well seen in the case of ocular vertigo.[1] A man with paralysis, say of the right external rectus, is giddy when, the sound eye being closed, he looks to the right. The motor effort required to fix objects in that direction is greater than, under normal circumstances, is necessary: hence, so far as this is concerned, he places objects to the right of what they should be; as will be seen, if he tries to put his finger quickly on any small object. But inasmuch as his other senses contradict this neuro-muscular report, vertigo is the result. In this case the motor effort is in excess of the effect produced. The reverse relation may hold. In the case of involuntary muscular spasm, e.g. the rotatory movement which precedes many epileptic fits, the motor effort (or at least the volitional part of it) is insignificant as compared with the result; hence the vertigo. In view of the important part played by the muscles with reference to equilibrium, it has been said that the physical basis of vertigo is an incipient motor process.[2] The sensation of vertigo is the

[1] Hughlings-Jackson, 'British Medical Journal,' 1877, vol. i. p. 605.

[2] The phrase applies not so much to the actual muscular contraction, as to the nervous current which causes it.

representation of such a process in consciousness. In the vertigo produced by disorderly action of the semicircular canals, as in the experiment of whirling a person round with his eyes shut, it is not so clear that the muscular sense plays any part, though it is true that in severe paroxysms of aural vertigo compensatory muscular movements do take place.

With respect to the co-ordinating mechanisms by which equilibrium is actually maintained, it is to be remarked that in the lower animals at least it can continue acting after the removal of the cerebral hemispheres, and therefore in a reflex fashion, and independently of consciousness. This is shown by Goltz's balancing experiment. (Are there not indications of the same fact in the hysteroid fit, where, notwithstanding the apparent, and I think I may say real, loss of consciousness, the patient so adjusts herself as not to hurt herself in falling?) Nevertheless, we should imagine that the superadded state of consciousness which we call vertigo, must correspond to changes in some area of the cerebral cortex, if physical changes in that part accompany consciousness upon the psychical side. And considering the numerous and important character of the organs concerned in the maintenance of equilibrium, it is likely that this area would be large. Assuming, therefore, that epileptic vertigo consists in morbid action within such area, as epileptic spasm in the motor area, sensory auræ in the sensory area, &c., we should expect vertigo to be a frequent phenomenon in epilepsy. That it is so is a well-known fact, which I need scarcely stop to illustrate. But it is not always easy to arrive at the fact in any given case. The patient usually complains of giddiness. But that expression, as has been remarked by Dr. Hughlings-Jackson, ought not to be taken as equivalent to vertigo. It may mean a number of vague sensations, such as faintness, and the like; and many patients cannot analyse their feelings further, unless we put the leading question, whether things seem to move, when, of course, the affirmative answer is less valuable than if it were volunteered. Nevertheless, there are many who will voluntarily describe a sensation of vertigo, and many more, I suspect, who have such feelings, but cannot express them.

However, the point which in the present place I wish to

insist upon is, that vertigo, though corresponding to action of the cerebrum, may be occasioned by disease in very different parts, either in the cerebrum itself, as in the case of epileptic vertigo, or in a peripheral organ, the commonest being the semicircular canals. This latter kind is aural vertigo. The semicircular canals may be actually diseased, as we suppose them to be in Menière's disease, or affected secondarily and as to their function only, as may happen in middle-ear disease. The explanation usually given of this latter fact is that the middle-ear disease causes increased pressure in the labyrinth,[1] and possibly also increased excitability of the auditory nerve. Such vertigo may be distressing and alarming to the patient, but a knowledge of its peripheral origin may enable us to assure him that it is unimportant, at least as regards life and mental functions. A well-known aural text-book says:[2] "When vertigo occurs in aural disease, it is a consequence of increased pressure upon the labyrinth through the fenestra ovalis. It is by no means a serious symptom when the cause is to be found in the middle ear, for it is usually relieved by a mechanical treatment through the Eustachian catheter."

Clinically associated with vertigo is the symptom of vomiting. Every one who has been sea-sick will admit its connection with perturbation of equilibrium. Vomiting often concludes a fit of convulsions (especially in children); or concludes, on the other hand, an attack of aural vertigo. In the latter case the explanation usually offered is that the irritation overflows, so to speak, from the auditory into the adjacent pneumogastric nucleus. Indeed, irritation of the pneumogastric appears to be the proximate cause of vomiting generally (Ferrier). There is no doubt that vertigo may produce vomiting, but can gastric derangement produce vertigo? Such a "gastric vertigo" has been forcibly described by Trousseau, but recent writers appear either to doubt its existence, or at most to admit that gastric derangement may occasion vertigo in the presence of aural disease. Theoretically, if we suppose that irritation can spread from the auditory to the pneumogastric centres, then it does not appear why it

[1] Knapp, *op. cit.* p. 214; Brenner, *op. cit.*

[2] Roosa, p. 264.

should not also spread the other way. It may be remarked that Trousseau, though he does not mention the state of the ears in his patients with gastric vertigo, was well aware of the existence of aural vertigo, which he describes in the same chapter.[1]

We may ask whether diarrhœa ever takes the place of vomiting in aural vertigo? It appears to do so in some nervous affections. I have known a successful struggle against sea-sickness to be followed by diarrhœa the next day. A man, recently under my observation for locomotor ataxy,[2] who suffered from the well-known "crises gastriques," had also unaccountable attacks of diarrhœa; the same thing sometimes occurs in epileptics, while, reversely, bromide of potassium occasionally constipates.

I append brief notes of a case in which the symptoms of vertigo, vomiting and diarrhœa seemed distinctly to take the place of epileptic fits, the ears being quite healthy:—

William H., æt. 15, became an out-patient at Queen Square Hospital, in June 1880. He had had fits for twelve months, undoubtedly epileptic. There was no warning, he had bitten his tongue, in the first fit he had fallen and cut his face. He was also subject to "startings" of the hands and feet, without loss of consciousness; these "starts" had preceded the outset of the fits by some three months. Under bromide of potassium and arsenic the fits were stopped.

Feb. 1881.—Nausea and abdominal pains, on account of which the arsenic was dropped.

April.—"Swimmings in the head."

May.—Giddiness, a feeling as if he must fall, obliging him to sit down; nausea also.

June.—Has been vomiting. Alternate diarrhœa and constipation. . . . Later in June, definite vertigo, "the room seems to turn from right to left." The hearing, according to his statements and as measured by the watch, was normal.

Hydrobromic acid relieved all his symptoms (except slight giddiness) during the rest of the year.

January 1882.—Severe attacks of vertigo, retching and

[1] Syd. Society Translation, vol. iii., Lect. 67.

[2] *Cf.* Buzzard, 'Clinical Lectures on Diseases of Nervous System,' p. 199.

vomiting. . . . Hearing (watch and tuning-fork) normal. Tympanic membranes normal. (Same results later on.)

During the first half of the year the symptoms abated again, but he had some "faints."

July.—Has had a bad attack of giddiness, vomiting and diarrhœa: the diarrhœa persists, and appears to be made worse by the hydrobromic acid. It was changed for bromide of potassium.

August.—One fit. Diarrhœa comes and goes. Occasional giddy attacks; no vomiting.

October.—After the attacks of giddiness, now becomes extremely sleepy: once slept soundly for three hours. Their connection with the epilepsy is thus made all the more manifest.

So far we have considered certain symptoms common in aural disease (meaning by that term chiefly middle-ear disease) and have endeavoured to show that they may occur to a greater or less extent in simple epilepsy. But they are the symptoms which characterise a complaint which may be fairly said to stand midway between these two, i.e. the affection known as Menière's disease. It will be asked, since these symptoms occur in middle-ear disease, what are the clinical characteristics of the so-called Menière's disease? A consideration of Menière's original cases (Note A), shows, I think, that in most of them, at least, the paroxysms of vertigo and vomiting are more sudden, more violent, more definitely paroxysmal than in chronic middle-ear disease, and that the "vital symptoms," as they have been called by Dr. Hughlings-Jackson,[1] viz. perspiration, pallor, faintness carried even to coma, are more marked. Secondly, the deafness is of the kind known as internal ear-deafness, i.e. inspection of the ears yields negative results, and sounds conducted through the cranial bones, as well as aerial vibrations, are heard imperfectly or not at all with the affected ear.

But since the vertigo of middle-ear disease may be paroxysmal and severe,[2] and since there may be cases of Menière's disease which in the early stages exhibit little if any impair-

[1] "On Nervous Symptoms with Ear-disease." 'British Medical Journal,' March 1877.

[2] Knapp, *op. cit.* pp. 218, 241; Brunner, *op. cit.* p. 319; Trousseau, *op. cit.* p. 552.

ment of hearing, further distinctions are necessary, and these seem to lie (as Dr. Pritchard suggests to me) in the course of the symptoms. In Menière's disease, pari passu with the repetition of the paroxysms, the tinnitus and the deafness increase, till at length the patient is left free from the vertigo and other symptoms, but hopelessly, perhaps absolutely deaf.

The differentiation from cerebral disease was made by Menière himself, and based partly on the occurrence of tinnitus and deafness (Note A), partly on the post-mortem evidence of his last case. With respect to this case it has been remarked that, though valuable for purposes of localisation, it does not give us much information as to the pathological process in the typical disease. In the typical disease there is a series of paroxysms, perhaps persistent impairment of equilibrium, but ultimate destruction neither of life nor of any function except hearing. The case in question consisted of a single paroxysm with rapidly fatal result.

To recur to the connection with cerebral disease. Seeing that the morbid anatomy of many grave nervous diseases is yet to seek, we cannot from the negative results of a post-mortem infer that the nerve-trunks or nerve-centres have not been essentially affected during life. Indeed, the "vital symptoms," faintness, sometimes sudden coma, &c. (in one of Menière's cases there were even convulsions) — make it certain that the higher centres are engaged, if only secondarily. The paroxysmal recurrence of the symptoms points in the same direction. It is possible that a close inquiry into the family and personal history of the patients affected with this disease may disclose some inherent weakness of their nervous system. Dr. Gowers[1] suggests "that if we locate the centre for equilibrium in the cerebellum, we may perhaps regard Menière's disease as a cerebellar epilepsy reflex in character, determined by an irritation of the semicircular canals, either alone or in conjunction with impressions from other nerves with which the centre is in connection."

The question whether ear-disease can contribute to the production of epilepsy is evidently one of some practical

[1] "On Auditory Nerve Vertigo." 'British Medical Journal,' March, 1877.

importance. Although most writers on epilepsy do not allude to it, there seems to be a certain à priori probability of such a relation. "Excentric" causes of epilepsy are generally admitted to exist; and it is not unnatural to place ear-disease among them, considering on the one hand the nervous phenomena (such as we have already discussed) which may be produced by the irritation, even of non-suppurative ear-disease, and on the other the fact that such gross diseases as cerebral abscess and meningitis may result from a neglected otorrhœa. Limiting the discussion to the subject of suppurative ear-disease, I shall quote what Dr. Hughlings-Jackson says on this subject:[1] "Epilepsy, or epileptiform seizures, occasionally occur with ear-disease. Admitting a relation, the accepted explanation would be, that the ear-disease provokes the seizures by reflected irritation. The author suggested another hypothesis, the possibility that the aural disease led to disease in Hitzig's and Ferrier's region; for in some cases, the aural disease was associated with epileptiform convulsions, starting in the hand, face, or foot of the opposite side. In one such case, the author had found a large tubercular or scrofulous tumour of the hemisphere on the side of the ear-disease, and on the side opposite the convulsions. He thought it possible that the disease is sometimes local softening from venous thrombosis, the convulsions depending on instability of grey matter around the part softened. He had no direct evidence of this, but he thought he had some indirect evidence of it. He related[2] a case in which a man who had phosphorous necrosis of the jaw had, during an acute illness, frequent epileptiform convulsions affecting the left side of his face, both sides of the thorax, and slightly the left arm. In the intervals of the attacks there was paralysis of the face almost as complete as in ordinary Bell's paralysis. At the necropsy there was, although not ear-disease, yet an equivalent state of things, there being pus in the right lateral sinus, and from this a vein with creamy contents was traced to the part most diseased of the right side of the brain; about the anterior end of the Sylvian fissure

[1] "On Nervous Symptoms with Ear-disease." 'British Medical Journal,' March 24, 1877.

[2] This case is recorded: 'Medical Times and Gazette,' January 6, 1872. In the first line of the report of the autopsy, *for* "left" *read* "right."

there was purulent softening and red softening. As the lesions were more wide-spread, and as the case was acute, the evidence was only very indirect towards the interpretation of chronic cases, but was, the author thought, worth consideration in that regard."

While awaiting further post-mortem proof of this hypothesis, we may perhaps look for a certain amount of collateral evidence from cases of cerebral abscess resulting from ear-disease. We have here gross disease of the brain, connected in all probability with the ear-disease by disease of the veins; and if, as Dr. Jackson puts it elsewhere,[1] minute changes in venous tracts may set up the cerebral changes which result in epilepsy, we might fairly expect that these minute changes would sometimes precede the greater; in other words, that epilepsy would form a point in the clinical history of patients dying with cerebral abscess in the course of an otorrhœa. Looking over the cases of cerebral abscess given by Gull and Sutton,[2] Ogle,[3] Jackson and Hutchinson,[4] I find two cases in which there was a fit before the acute symptoms began. In the first[5] there was "a convulsive fit apparently epileptic in nature" *six weeks* before admission to the hospital; the second fit occurred a week after the first, leaving permanent symptoms such as ptosis amentia, and the like. In the second case[6] the patient had had a fit *six months* before admission, leaving only pain in the forehead and a certain amount of mental affection. The second fit occurred two days after admission, and was followed by acute symptoms. It may be, however, that in these cases the first fit was caused by the presence of the abscess, as it is difficult to fix a date for its first formation, and in any case one fit does not make an epilepsy. But a third case, recorded by Jackson and Hutchinson,[7] is more to the point:—A young man had had discharge from the ears since 3 years of age, after scarlet fever. About three years before his

[1] 'Gulstonian Lectures,' 1869.
[2] Reynolds's 'System of Medicine.'
[3] 'British and Foreign Medico-Chirurg. Review,' No. 70.
[4] 'Medical Times and Gazette,' 1861.
[5] No. 99, Ogle; No. 61, Gull and Sutton.
[6] No. 104, Ogle; No. 65, Gull and Sutton.
[7] 'Medical Times,' 1861, p. 196.

death he fell into the Thames and was much frightened — the discharge had previously stopped. Epileptic fits followed, and continued for two years. Then pain in the right ear, offensive discharge, and death from abscess of the cerebellum. This is a clear case of chronic epilepsy occurring in a patient who finally died of abscess in the brain. Was the epilepsy, like the abscess, connected with the ear-disease?

Kopp and Schwartze uphold the view that epilepsy, when caused by ear-disease, is due to reflected irritation. They give two cases, the first of which is certainly striking:[1] A man, age 21, whose mother had had otorrhœa and convulsive attacks of uncertain nature, had himself ear-disease from 3 years of age, after scarlet fever. Discharge from the left ear, and attacks of earache continued on and off till the age of 15, then stopped for two years. At the age of 17, after exercise at a gymnasium, there came on violent pain in the ear, with tenderness of the mastoid process. At the same time epileptic fits began, of the following character: sense of oppression for some hours, then cold sweating; then pain in the head, gradually localising itself behind the affected ear, giddiness, spasm of the left side of the face (?), loss of consciousness (usually), and on recovery, vertigo, semi-paralysis, coldness of the extremities. These attacks were at first benefited by treatment, but became in four years' time very frequent (daily or oftener) and prolonged. On admission, there was paresis in the district of the left facial, tenderness and redness of the left mastoid process, fetid pus in that meatus, a perforation of the membrane with granulations projecting through it, and the Eustachian tube was blocked. Schwartze trephined the mastoid, and by degrees removed the pus, and opened up the Eustachian tube. A violent epileptic fit occurred just when the first incision was being made, but after this they stopped, and did not recur. From the prompt effect of the operation, Schwartze argues that the fits were reflex in character, and not due to secondary cerebral disease. In favour of this view, he urges also the cases in which epilepsy is said to have been caused by foreign bodies in the external meatus, and to have ceased with their removal. This theory, it will be noted, opens

[1] 'Archiv für Ohrenheilkunde,' Bd. V. p. 282.

up a wider field than is covered by suppurative ear-disease only; for symptoms indicative of nervous irritation, e.g. tinnitus, vertigo, &c., occur perhaps with even greater frequency in non-suppurative middle-ear disease—fibrosis, sclerosis as it may be termed—than in purulent catarrh.

I shall now quote some statistics which I have collected on the subject. They are not yet, I fear, sufficiently numerous, but I shall hope to remedy this in the future. I examined the hearing power of 100 epileptics at Queen Square Hospital, and endeavoured to ascertain the nature of the aural disease, when any was indicated by defect of hearing. (Note B.) There was a total of 17 cases of suppurative disease, past or present. Another series of 100 epileptics, whom I merely questioned as to otorrhœa, gave 14 as the number who had or had had it; this made 31 in the 200, or 15·5 per cent. This proportion might seem large enough to justify us in concluding that the two facts were connected, but to do so would be to leave out of sight the very great frequency of ear-disease among hospital out-patients. For on starting a counter-series in an out-patient room not devoted to nervous diseases (Victoria Park Chest Hospital), I found that out of 200 patients 29 had had otorrhœa, making 14·5 per cent. In this counter-series I found only one case of chronic epilepsy (and in that case there was probably aural disease); a second had been discharged from the army for "faints," possibly epileptic, this patient had otorrhœa; a third who had also had otorrhœa, had had two fits under somewhat peculiar circumstances, which I shall mention presently.

From these two series, then, no particular conclusion can be drawn, unless it be that otorrhœa is extremely common among hospital patients, and epilepsy very much less common. But a third set of inquiries gave more definite results. I inquired as to the history of "fits" from 100 patients with suppurative disease of the middle ear. These were patients under Mr. Cumberbatch at St. Bartholomew's, or Dr. Urban Pritchard at the Royal Ear Hospital, so that the diagnosis of the ear-disease rests on the best authority. Neglecting such things as "faints," hysteria, and the like, I found that seven out of the 100 had had bonâ fide epileptic fits. This is a

very large proportion; for Niemeyer[1] gives the absolute frequency of epilepsy as six cases in every thousand persons; and Russel-Reynolds[2] maintains that this estimate is far too high. If then further inquiry should verify the figures which I have obtained, it would appear that epilepsy is more than usually frequent among patients with suppurative disease of the middle ear.

To turn to the consideration of particular cases. It is difficult from most out-patients to get precise accounts of their complaints, but I give the following facts as being possibly of some significance. One of my epileptic patients certainly improved when the condition of the ears was treated; he had been previously on bromide of potassium without very marked improvement. The parents of another (a boy with profuse and fetid discharge) said that the fits were always worse when the discharge was bad. A man, under treatment by Dr. Pritchard for polypus auris, had previously had fits, which he said occurred whenever the discharge stopped (when it became pent up?). A young woman had had epilepsy for eighteen months, and discharge from the left ear during the same time; during the three weeks preceding her first attendance both the epilepsy and the otorrhœa had been much worse than before. Her fits were preceded by rotatory movements. These accounts seem to point to some connection between the epilepsy and the ear-disease. As to the kind of connection, I cannot say much; but I have had three cases in which there was otorrhœa on one side only, with convulsions limited to, or principally affecting the other side. These might be urged in favour of Dr. Jackson's view, for we should expect reflex irritation to give rise to movements on the same side as the irritation; whereas cerebral disturbance from thrombosis of veins in connection with the affected ear would be manifested in the limbs of the other side.

Upon the other (reflex) theory we might justly expect that the fits would occur in close connection with some of the nervous phenomena, e.g. pain, tinnitus, vertigo, which are apt to occur in ear-disease. But here comes the difficulty, that

[1] 'Text-book of Practical Medicine,' *s. v.* "Epilepsy."

[2] System of Medicine, *s. v.* "Epilepsy."

many of these very phenomena are met with (as I have endeavoured to show) in epilepsy uncomplicated with ear-disease. Hence, we might feel unable to say whether the vertigo (for instance) was aural, or epileptic, or both. Perhaps we may be sometimes able to make out a transition from the one to the other: as a possible illustration I submit this case.

A girl, æt. 18, attending at Victoria Park Hospital for some trivial chest affection, had had earache and discharge from the right ear from babyhood. She had fits when teething, and off and on since childhood so-called "bilious attacks," consisting of pain in the head and vomiting, and beginning usually with vertigo, the patient feeling as if things were turning round and round, and as if she herself were going to pitch on to her head. Such attacks might well be due to the ear-disease. But once, while jumping with other children (being then 9 years old and in perfect health), a violent retching came on, resembling (as her mother, who witnessed it, told me) one of the "bilious attacks;" but, instead of that, she became unconscious, convulsions set in and lasted the whole day. The next day she was quite well. A similar attack came on three months later, this time while she was swinging.

The suggestion is, that in this case the equilibrium apparatus was enfeebled by the ear-disease, as may be inferred from the habitual attacks of vertigo; and that, under circumstances particularly liable to upset the equilibrium (jumping, swinging), the ordinary attack of vertigo was replaced by an attack of convulsions. It may be noted that in the case quoted from Schwartze the first fit occurred under similar circumstances, viz. after exercise at a gymnasium.

To conclude, I think that further inquiries are necessary as to the production of epilepsy by ear-disease. It is important to determine the existence of this, as indeed of any other cause of the disease. For the best means we have of checking the fits, viz. bromide of potassium, often fails altogether to prevent their recurrence when the drug is discontinued. This we might hope to do if we could act on some causal indication. It is also important, assuming the existence of such a cause, to know its modus operandi. For if it act by nervous irritation, then removal of the source of irritation, i.e. treatment of the ear-

disease, must be our chief object; if by the indirect production of cerebral disease, then the epilepsy has to be treated per se, while treatment of the ear is required as a prophylactic, either against extension of the epilepsy in patients already epileptic, or against the production of it in those predisposed to become so. But in any case we must treat the ear-disease.

NOTE A.

The leading symptoms in Menière's original cases (which will be found in the 'Gazette Médicale de Paris,' 1861, and in Knapp's article in the 'Archives of Otology and Ophthalmology,' vol. ii. No. 1, p. 229) are as follows:—

Case I.—Tinnitus, 20 years, gradual impairment of hearing. Then violent attack of sneezing, followed by staggering and inability to walk in straight line. Next day, walking more difficult, involuntary motions to left, vomiting. Forty-two days later, vertigo, vomiting, convulsions of face, contortions of left side of body, followed by incomplete and transient paralysis. Hearing became worse than before.

Case II.—Tinnitus following intermittent fever. Afterwards, attacks of vertigo and vomiting: patient once fell down in them. These lasted a year; in two years complete deafness. Examination of ears negative.

Case III.—Sudden fall, pallor, perspiration, vomiting, momentary loss of consciousness, then vertigo. Loud tinnitus, deafness, dating from the attack. Several less violent attacks, leaving patient with tinnitus, increasing deafness and some constant loss of equilibrium. Examination of ears negative.

Case IV.—Sudden fall, loss of consciousness, pallor, perspiration: persistent vomiting and vertigo. Final results: uncertainty of gait, loud tinnitus, increasing deafness. Hearing had been good before.

Case V.—Frequent and sudden attacks of giddiness, nausea, vomiting: resulting in impairment of hearing, tinnitus, staggering gait. Examination of ears negative.

Case VI.—Frequent attacks of vertigo, sudden falling, loss of consciousness. Tinnitus and deafness set in and increased gradually. Attacks subsided in a year, leaving complete deafness only.

Case VII.—"Coup de sang" in left ear, with faintness, giddiness, noises. Next night, sudden, violent and persistent vomiting, vertigo as if in a storm at sea. Deafness of left ear. Final results: uncertain gait, tendency to vertigo, deafness of left ear. Examination and treatment of ears negative.

Case VIII.—Slight vertigo, dimness of vision, nausea, and sometimes falling, with transient loss of consciousness. Twenty such attacks in a year; then nothing but deafness. Examination of ears negative.

Case IX.—Dimness of vision, wavering of objects before eyes, then vertigo. Vertigo recurred on sitting up during two months. Consequences; tinnitus, progressive deafness, loss of equilibrium. Examination of ears negative.

Case X.—A "cold" caught during menstruation. Sudden deafness, persistent vertigo and vomiting. Death on fifth day. Nervous centres healthy: reddish plastic exudation in semicircular canals, traces of it in vestibule, cochlea healthy.

Menière remarks as regards the differentiation from cerebral disease:—

"Les vertiges, les étourdissements survenant tout à coup et donnant lieu à un état syncopal, à des nausées et à des vomissements, n'appartiennent pas exclusivement à une lésion de l'oreille interne: il peut se faire que ces désordres symptomatiques dépendent de certaines affections cérébrales, d'un état congestif des méninges, de quelque lésion du cervelet et de ses dépendances; mais nous croyons que, quand les symptômes s'accompagnent de bourdonnements des oreilles, de bruits continus, variables, et surtout quand on voit bientôt survenir une diminution notable de l'ouïe, alors le mal a son siège dans le labyrinthe, et plus particulièrement dans les canaux demi-circulaires."

And of the general characters of the disease he gives the following summary:—

"1. Un appareil auditif, jusque-là parfaitement sain, peut devenir tout à coup le siège de troubles fonctionnels consistant en bruits de nature variable, continus ou intermittents, et ces bruits s'accompagnent bientôt d'une diminution plus ou moins grande de l'audition.

"2. Ces troubles fonctionnels, ayant leur siège dans l'appareil auditif interne, peuvent donner lieu à des accidents réputés cérébraux, tels que vertiges, étourdissements, marche incertaine, tournoiement et chute, et, de plus, ils sont accompagnés de nausées, de vomissements et d'un état syncopal.

"3. Les accidents qui ont la forme intermittente ne tardent pas à être suivis de surdité de plus en plus grave, et souvent l'ouïe est subitement et complétement abolie.

"4. Tout porte à croire que la lésion matérielle qui est cause de ces troubles fonctionnels réside dans les canaux demi-circulaires."

NOTE B.

Analysis of 100 uniselected cases of epilepsy, with respect to the aural symptoms presented by them: to show the frequency with which such symptoms occurred in those whose ears were sound or diseased:—

Fifty-four cases were normal as to hearing power; and of these:—

Noise in the ear, as aura	occurred in	4 cases	= 8 per cent. (or just under.)	
Do. independently of the fit . . .	„	2	„	4 per cent.
Pain in the ear	„	3	„	6 „
Vertigo, definite in character . .	„	11	„	22 „
Do. vague (staggering, &c.) . . .	„	7	„	14 „
Vomiting or nausea	„	12	„	24 „
Well-marked rotation	„	2	„	4 „

(The vomiting and vertigo were associated in 5 cases, in one of which also the rotatory movements were present.)

In 21 cases there was slight deafness; the cause of which appeared to be:—

In 6 cases, wax.
„ 3 „ slight thickening or depression of the membrane.
„ 6 „ doubtful.
„ 1 „ central mischief, post-epileptic deafness.
„ 5 „ previous catarrh, with history of discharge.

Out of these 21:—

Noise in the ear, as aura. . . .	occurred in	2 cases	= 10 per cent.	
Do. of uncertain nature	„	1	„	5 „
Pain in the ear, after the fits . .	„	1	„	5 „
Do. in connection with discharge or of doubtful nature	„	2	„	10 „
Vertigo, definite in character . .	„	7	„	35 „
Do. vague	„	4	„	20 „
Vomiting	„	1	„	5 „
Rotatory movement (with vertigo).	„	1	„	5 „
Do. (with vomiting)	„	1	„	5 „
Deafness, increased by fits . . .	„	2	„	10 „

In 25 cases there was well-marked deafness; the cause of which appeared to be:—

In 12 cases, suppurative middle-ear disease (present or past).
„ 6 „ chronic thickening or depression of membranes.
„ 4 „ wax.[1]
„ 3 „ of doubtful nature.

Out of these 25:—

Noise in the ear, as aura	occurred in	1 case	= 4 per cent.
Do. independently of the fits. . .	„	2 „	8 „
[Pain in the ear occurred in connection with suppuration.]			
Vertigo, definite in character. . .	„	6 „	24 „
Do. vague	„	6 „	24 „
Vomiting	„	3 „	12 „
Rotatory movements	„	1 „	4

[1] One of these cases subsequently developed paralytic symptoms, sufficient to take it out of the category of simple epilepsy.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.