alone, return a verdict of *felo-de-se*. Both coroners and juries usually attribute suicide to insanity, and out of 4,846 inquests on suicides in the year 1928 the verdict of *felo-de-se* was only returned in 88.⁹ But although the memory of the deceased may be so relieved from the stigma of a crime his relations may be burdened with an unnecessary dread of insanity. If the alternative verdict to *felo-de-se* was "suicide the result of mental illness" the modern outlook and phraseology of the Mental Treatment Act, 1930, would be represented.

An analysis of 1,000 consecutive cases of attempted suicide examined by me some years ago showed that only about one-fifth were certifiable as insane, and the experience gained by the examination of more than 2,000 additional cases confirms this view. But many patients suffer from temporary mental disorder at the time of the attempt, and mere trivialities often cause determined attempts at suicide.

An attempt to commit suicide by a person of sane mind, being an attempt to commit a felony, is in law an indictable misdemeanour, and the offender may be committed for trial at the Assizes or Quarter Sessions, and is punishable under the provisions of the Hard Labour Act, 1882 by imprisonment with hard labour. The case can be dealt with also in a court of summary jurisdiction with the consent of the accused.¹⁰ The suicide must be of sound mind and have attained the years of discretion (1 Hale P. C. 411; 3 Inst: 54), therefore, it is no crime for a person to take his life before reaching that age. The mere fact of drunkenness is no excuse for the crime of attempted suicide, but it is a material fact for the jury to consider before coming to a conclusion whether the accused really intended to destroy his life (*R v Doody*).

The number of cases of suicide and attempted suicide reported to the police are affected probably by many factors. No doubt the increased sobriety of the community during recent years, the receipt of unemployment benefit, trade depression, and the modern policy not to take proceedings in cases of attempted suicide unless detention for a time is in the interest of the person, or for other sufficient reason, operate in one or other direction. And although the number of persons coming before the courts on this charge in 1928 was considerably more than double the average number in the years immediately preceding the war, the number of persons now sent to prison for this offence is about halved. Many of these recognize that they need care and supervision, but would be unwilling to remain for a sufficient period in a mental hospital as voluntary patients under the Mental Treatment Act, 1930, and are ineligible for detention as temporary patients under Section 5 of the Act. And as they cannot be certified, a short term of imprisonment under medical care is inevitable sometimes: it is usually accepted with equanimity and often with gratitude.

The mental condition of an accused person charged with attempted suicide is frequently a matter for special consideration in a criminal trial. In the summary courts an insane person is usually dealt with by transfer to a Public Assistance Institution or private mental hospital. If the Grand Jury at Courts of Assize or Quarter Sessions ignore the bill of indictment the accused is forthwith released from the custody of the court whatever his mental condition may be. If, however, he is found to be insane on arraignment or guilty but insane he is sentenced to be detained during His Majesty's Pleasure. A plea of guilty is sometimes made in order to avoid this, and if the court is satisfied with the medical evidence the prisoner may be considered a proper person to be detained in a mental hospital. A recent decision of the Court of Criminal Appeal is of interest in this connexion. The appellant after pleading guilty to an offence at Liverpool Assizes in

May 1929 entered into a recognizance to be of good behaviour for two years, and to remain in a mental institution until he was discharged. The breach of recognizance with which he was subsequently charged, and for which he was sentenced to ten months imprisonment, consisted in his escape from the institution. At the time when he entered into the recognizance he was a certified lunatic, and the Court of Criminal Appeal held that the certificate raised a presumption of insanity which prevented the recognizance from being binding on the appellant, and that there had been no evidence to displace that presumption. The Court allowed the appeal and quashed the sentence (*R v Green-Emmott*).¹¹

The association of murder with suicide is important. The Criminal Statistics for England and Wales in the years 1927 and 1928 show that one murderer out of four commits suicide—66 out of 234. The figures of attempted suicide are very similar, for in a series of 182 persons convicted of murder, whose mental condition I examined, 41 attempted suicide in connexion with the crime. The suicidal act is due sometimes to the certainty of conviction and to the desire to end an intolerable situation. It is also the result of insanity, and Dr. H. P. Foulerton informs me that of 527 admissions to Broadmoor during the last 20 years following charges of murder, 149 attempted suicide at the time of the crime.

In some insane persons the suicidal intention precedes the homicidal; thus the altruistic melancholiac having decided to kill himself first kills his wife to save her from disgrace, suffering, or the necessity to fight the world alone. But the homicidal intention precedes the suicidal in many senile, alcoholic, and delusional murderers and may result from vindictiveness, revenge or hate based on insane beliefs and false premises.

Bogus suicides appear to be uncommon in free life but are sometimes staged in an attempt to defraud an insurance company or for another purpose. Feigned attempts at suicide are made in prison for ulterior gain also; they constitute nearly 70 per cent. of the attempts and are of medical importance as they sometimes miscarry and end fatally.

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VERTIGO IN SUPPURATIVE CONDITIONS OF THE MIDDLE EAR*

BY

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Vertigo is defined as "a disordered condition in which the person affected has a sense of whirling either of external objects or of himself, and tends to lose equilibrium." (O.E.D.) It may be due to a variety of causes, labyrinthine and non-labyrinthine, but labyrinthine vertigo is by far the more common.

It is generally believed that normally both labyrinths are in a state of equal tonic activity and send continuous

* Read in opening a discussion in the Section of Oto-Rhino-Laryngology at the Annual Meeting of the British Medical Association, Eastbourne, 1931.

impulses to the higher centres. If both labyrinths are stimulated equally, however great the stimulus may be, there is no vertigo; the slightest difference in stimulation between the two labyrinths immediately produces vertigo. Labyrinthine vertigo is the result of unequal activity of the two labyrinths. The most common cause of such inequality is some affection of the ear, which may be non-suppurative or suppurative. Between these two kinds of vertigo there is a distinction literally vital. Aural vertigo of non-suppurative origin may make a patient's life a burden to him; aural vertigo in the presence of suppuration may mean that his life is in danger.

I propose to confine myself to vertigo associated with aural suppuration. In these conditions we are concerned with the perilymphatic space. The only barrier between perilymph and endolymph is the flimsy wall of the membranous labyrinth; therefore, if the perilymphatic space is invaded, we are justified in assuming that the endolymph space will also be invaded. The perilymphatic space communicates with the cerebro-spinal spaces by the piraachnoid sheath of the eighth nerve and by the aqueduct of the cochlea; the endolymphatic space is not in communication with the cerebro-spinal space, as the saccus endolymphaticus lies between the layers of the dura. Thus any infection of the perilymphatic space puts the patient's life in immediate peril.

The perilymphatic space is separated from the middle ear and its accessory cells by two layers, the dense bone of the labyrinth wall and the lining endosteum. At the fenestrae the bony layer is absent, and the only barrier is the endosteum, reinforced at the fenestra ovalis by the footpiece of the stapes. It may be laid down as a rough clinical rule that, so long as the barrier is adequate against invasion the signs will be due to irritation of the affected labyrinth; once the barrier is passed and the perilymph space invaded there will be a brief period of intense irritation followed by destruction, and all signs will then be due to the unopposed activity of the healthy labyrinth.

When vertigo occurs in the presence of suppuration it is probably due to the suppuration, but we must not overlook the possibility of some independent cause—for instance, a patient with a suppurative otitis media on one side may suffer from vertigo which has been produced by blockage of the eustachian tube on the side which has been regarded as normal. On the other hand vertigo must never be attributed to "neurasthenia" or "bilious attacks." Vertigo can certainly cause neurasthenia; under modern traffic conditions a sufferer from vertigo is in danger of his life whenever he crosses a street, and certainly the neurasthenia can aggravate the vertigo, but it is very doubtful if it can ever cause it. Vertigo is often associated with severe nausea and vomiting, but the most intense gastric disturbance cannot cause vertigo. "Gastric vertigo" is a term that should never be used. We still see cases of acute suppurative labyrinthitis which have been diagnosed as "bilious attacks."

In vertigo due to aural suppuration past or present we can distinguish three principal groups of cases:

1. Cases where the labyrinth wall is intact.
2. Cases where the labyrinth wall has yielded to infection to a greater or less degree—the labyrinthitis.
3. Cases where infection has reached the posterior fossa—cerebellar abscess—retro-petrous abscess.

WHERE THE LABYRINTH WALL IS INTACT

These cases are characterized by increased irritability of the affected labyrinth. This may be due to: (a) Changes of pressure at the fenestrae; (b) abnormal sensibility caused by exposure of the inner tympanic wall.

Changes of Pressure

Ankylosis of the ossicles. Dundas-Grant and others have described cases in which vertigo was caused by malleo-incudal ankylosis. The probable explanation here is that the stapes is driven in and thus the normal give-and-take between the fenestrae is altered. This alters the intralabyrinthine pressure and so disturbs the balance between the two labyrinths. Similar phenomena are observed in unilateral eustachian obstruction, as has been described by Sydney Scott.

Fluid or granulations in the middle ear may alter the play of the fenestrae in the same way as an ossicular ankylosis. In these cases the proper treatment is to provide drainage or to remove the ossicles by one or other of the mastoid operations.

Abnormal mobility of the stapes can cause vertigo by irregular alterations of pressure in the affected labyrinth. Such cases are not uncommon in congenital syphilis, but they are fortunately rare in suppurative or post-suppurative conditions. If the stapes is loosened during active suppuration, invasion of the labyrinth usually follows. If the stapes is loose when suppuration is extinct (for example, after a radical mastoid operation) it is possible that we might be driven to destroy the labyrinth. This will be discussed later.

Abnormal Sensibility due to Exposure of the Inner Tympanic Wall

This condition is sometimes found with a large dry perforation of the membrane, and more often after a radical mastoid operation. Vertigo is usually caused by sudden changes of temperature, especially a cold wind. The unprotected inner tympanic wall is cooled and the victim endures an unregulated "cold caloric test." One patient of mine with a large radical cavity, suddenly fell down with a violent attack of vertigo when walking along an exposed sea-front on a cold and windy night. As a rule the condition can be remedied by closing the meatus with a plug of greased cotton wool. This condition may be a danger if the patient swims or dives; the sudden rush of cold water may cause a "caloric response" which completely incapacitates the victim.

If the patient is seen during an attack considerable relief is often given by painting the promontory with 5% cocaine and packing the meatus with gauze or wool soaked in warm paraffin. Accumulations of cerumen or debris in the cavity are often responsible for the attacks. An important point in these cases is that the patients are often abnormally sensitive to sudden falls of blood pressure. Ephedrine is useful in meeting this emergency. Of other drugs chloretone and luminal are sometimes useful, and Dundas-Grant has obtained good results with small doses of quinine, which is believed to diminish the labyrinthine activity.

As a rule in these cases of labyrinthine hyper-sensitivity after a radical operation the condition gradually passes off and compensation between the two sides is reached. Occasionally this does not take place, and the patient's misery makes us consider the advisability of destroying the labyrinth. This may be necessary as a last resort, but only as a last resort. However severe the vertigo may be it is not dangerous to life, and opening an uninfected labyrinth in an ear that has been infected, even though the infection appears extinct, is accompanied by a certain degree of risk, slight it is true, but present. Probably the simplest and safest method is that suggested by Mollison, opening the external canal at the torus, and injecting alcohol into the vestibule through the opening.

If, in spite of an adequate radical operation, granulations persist on the inner tympanic wall and vertigo continues, the probability is that there is a labyrinthine infection.

Lastly we must remember that there is no reason why a patient with a radical mastoid cavity should not suffer from Ménière attacks entirely independent of his suppurative condition. On the law of averages it is unlikely, but the possibility must not be overlooked.

WHERE THE LABYRINTH WALL HAS YIELDED TO THE INFECTIVE PROCESS

This group comprises all cases of labyrinthitis of suppurative origin. In these cases we are faced by an entirely different situation. As has already been stated, once the labyrinth has been invaded by a septic infection the free communication between the perilymph and the cerebrospinal fluid implies the risk of spread to the subarachnoid space and consequent meningitis. Labyrinthitis of septic origin, therefore, is a surgical problem, and the suitable surgical solution is determined by the extent to which infection has advanced. Thus in any septic labyrinthitis vertigo and other labyrinthine symptoms are important only as indications of the degree of invasion of the affected labyrinth.

The whole sensory area of the labyrinth is so small that we are entitled to believe that invasion of the labyrinth cavity must quickly cause destruction of the labyrinth function. From this it follows that so long as the labyrinth preserves any trace of function it is unlikely that the cavity is infected, and until that infection is complete the risk to the patient's life is very small.

VARIETIES OF LABYRINTHITIS

The usual classification of the different forms of labyrinthitis is admittedly faulty, especially in the inaccuracy of the terms employed, but it usefully emphasises important clinical distinctions. Three principal varieties are recognised: (1) Circumscribed labyrinthitis; (2) serous labyrinthitis; (3) diffuse suppurative labyrinthitis, which may be either (a) "latent," or (b) "manifest."

It is possible that an infection of the lumen of a canal might be localised by adhesions, but in practice circumscribed labyrinthitis usually means an incomplete breach of continuity of the labyrinth wall without invasion of the lumen. The bony wall may be eroded until the endosteum is exposed, but as soon as the endosteum is penetrated invasion of the cavity follows, and the characteristic features of the condition are lost.

This probably corresponds to the "sympathetic" effusions into serous cavities. It may be regarded as an inflammatory reaction to a septic process outside the labyrinth. The labyrinth wall has remained as an adequate barrier against the invasion of organisms but has been traversed by the toxic products of infection. Such entrance might be made through the fenestrae or through endosteum exposed by erosion of the bony wall.

Diffuse suppurative labyrinthitis is the result of microbic invasion of the cavity.

In acute suppurations of the middle ear the invasion is usually through one of the fenestrae, either by destruction of the membrana secundaria of the fenestra rotunda, or by loosening of the footpiece of the stapes. It is stated in the textbooks that infection through the fenestra rotunda is the more common; in the few cases of invasion through a fenestra which I have seen this has not been the case, the invasion has been through the fenestra ovalis. Occasionally in acute suppurations, especially in those due to the pneumococcus mucosus capsulatus, infection has entered through an erosion on the torus of the external canal. In chronic suppurations invasion usually takes place through an erosion made by the growth of cholesteatoma. Such erosions are more common on the external

canal, but are occasionally found on the promontory. The course of the disease differs according to the site of invasion. If infection enters through the promontory or the fenestrae the main cavity of the vestibule is attacked at once, and destruction is much more rapid than when the infection enters by the narrow lumen of the external canal. For this reason suppurative labyrinthitis is more dangerous in the acute middle ear suppurations than in the chronic. This accounts for the fact that in the acute suppurations meningitis is more common than intracranial abscess.

The distinction between "manifest" and "latent" labyrinthitis is entirely clinical. While the signs and symptoms of recent destruction exist the labyrinth is "manifest," when the affected labyrinth is paralysed and the labyrinth of the other side has taken over all the vestibular functions of equilibration, that is to say, when compensation is complete, the labyrinthitis is "latent." Some cases of so-called "latent labyrinthitis" would be better described as "residua of obsolete labyrinthitis," for the whole cavity of vestibule, cochlea and canals is filled with fibrous tissue. Other cases are really labyrinthine necrosis; the greater part of the labyrinth wall lies as a sequestrum in an abscess cavity in the petrous. In other cases the labyrinthine cavity remains, filled with pus and granulations, an ever-present danger to the adjacent meninges.

We have stated a preliminary axiom that, so long as the infection has not reached the perilymphatic space, the signs and symptoms will be due to irritation of the affected labyrinth; when the perilymphatic space is invaded there will be a brief period of intense irritation followed by destruction. As the destruction proceeds signs are more and more due to the unopposed activity of the healthy labyrinth. When the destruction is complete the surviving labyrinth assumes double duties and we have the stage of compensation of "latent labyrinthitis."

Of the signs that enable us to diagnose between the different forms of labyrinthitis, excepting always the "latent," vertigo is the most prominent. This must not be taken as implying that our decision can be based on the vertigo alone; it is a subjective symptom and the evidence that it affords must always be rigorously checked by the labyrinth tests and examination of the nystagmus and of the alterations in hearing.

Of the labyrinth signs the slow, or labyrinthine component of the nystagmus, the pointing error, and falling are away from the more active labyrinth. The quick, non-labyrinthine component of the nystagmus and the direction of the sensation of falling or movement during vertigo are towards the more active labyrinth. This may be stated as a general rule, but the sensation of the direction of vertigo is unreliable. During the stage of destruction the sensation is so severe that the patient often describes the sensation as one of being "thrown about" without any particular direction of movement. This is probably due to the combination of irritation and paralysis at this period.

More important than the direction of sensation are the periodicity of the vertigo, either continuous or intermittent, and its severity.

In circumscribed labyrinthitis as the perilymph space is not invaded the signs are those of irritation; the affected labyrinth is often hypersensitive to the caloric test, hearing is affected only by the casual middle ear lesion, and vertigo is intermittent and rarely severe. The attacks of vertigo are sudden and transitory, often brought on by sudden movements of the head and accompanied by a brief nystagmus, usually directed to the affected side. If the lesion progresses until there is an erosion of the bony wall with exposure of the endosteum

of the canal the "fistula sign" can usually be elicited. Under this term we may include all conditions in which sudden changes of pressure in the external auditory meatus—as, for example, pressure on the tragus or suction by a Siegle's speculum—produce vertigo and nystagmus. The reaction is produced by the transmission of the pressure to the perilymph and so to the endolymph. Movement is started in the endolymph and the endolymph wave stimulates the sensory endings of the cristae or maculae as the case may be. It is a clinical application of Ewald's experiment.

Two points in connection with the fistula sign deserve special mention:

1. The presence of the "fistula sign" is positive proof of the activity of the affected labyrinth. It follows that the term is a misnomer, for if there was a true fistula the lumen of the canal would be invaded and destruction would quickly follow. If the "fistula sign" can be obtained it is safe to say that the endosteum is intact.

2. It is seldom possible to diagnose the site of the erosion from the direction of the nystagmus and vertigo produced by the "fistula test." We may hazard a guess but it is no more, and is usually wrong.

Serous labyrinthitis is characterized by the prolongation of the irritative stage and the incompleteness of the paralytic stage. The preponderance of irritation or paresis depends on the degree of permeation of the labyrinth wall. It may be said that in all cases of serous labyrinthitis there is a well marked irritative stage. This is emphasized by Baldenweck, who points out that the more usual nystagmus of a serous labyrinthitis is towards the affected side, that is to say a nystagmus of irritation. Vertigo is always present during the earlier stages of serous labyrinthitis, and at this period a fistula sign is sometimes found; this is to be expected as serous labyrinthitis is often a sequel to the circumscribed form. But the vertigo, though troublesome, is rarely severe, often it is only noticed during movement, and the fistula sign when present is never as dramatic as it is in true circumscribed labyrinthitis. The most likely reason is that although the labyrinth in the early stage of a serous labyrinthitis is unduly irritated it is not really even normally irritable. The irritation is due to the increased stimulus of the intralabyrinthine effusion, which overcomes the commencing paresis. This explains the paradox that a labyrinth showing signs of irritation often shows also a diminished sensitivity to the caloric tests. It will be remembered that an analogous reaction is sometimes found in cases of the so-called "Ménière syndrome." When the irritative stage of a serous labyrinthitis passes off the subjective symptoms often disappear. The labyrinthine paresis may advance so gradually that the sound labyrinth never exerts itself violently. Compensation is acquired gradually and without discomfort. The labyrinthine functions of the affected side, cochlear and vestibular, are diminished but never lost, and traces of hearing and vestibular activity survive.

Diffuse suppurative labyrinthitis presents a very different picture. The irritation is short but violent, and the swiftness of destruction allows no compensation before the full activity of the healthy labyrinth comes into play. Thus the vertigo is of the utmost intensity, continuous and prostrating, usually accompanied by nausea and vomiting. The slightest movement of the head accentuates the victim's distress. A patient once told me that it surpassed the most violent miseries of seasickness. Anyone who has seen a case of diffuse suppurative labyrinthitis in the stage of invasion can understand the cryptic statement that in the Wide-Awake Club of which Mr. Jorrocks was recorder "no member was drunk if he could lie without holding." The vertigo may be so violent that the subject feels as if he were being "thrown out of his bed."

Vertigo gradually passes off when destruction is complete, but until full compensation is reached transitory attacks may be brought on by sudden movement. It is a clinical observation, which is not easy to explain, that compensation occurs much more rapidly if the affected labyrinth is opened and drained, even though prior to operation no trace of function can be elicited by the caloric tests. With the vertigo appear the other labyrinth signs. The period of irritation may be so brief that the typical nystagmus to the affected side with falling and pointing to the sound side are lost in a violent nystagmus in both directions and total incapacity to stand or even sit upright. This stage is quickly succeeded by the signs of complete destruction, total loss of labyrinthine function, cochlear and vestibular, on the affected side, with labyrinthine signs from the sound labyrinth.

When compensation is reached nystagmus, falling and pointing cease, the caloric test is negative but the rotation tests are normal. This normality of rotation tests with a dead labyrinth (Ruttin's reaction) must be due to some change in the healthy labyrinth which alters the normal relations of ampulli-petal and ampulli-fugal currents in the canals and makes the stimulus equal in either direction.

SOME GENERAL PRINCIPLES

Details of the operative technique are not germane to the present discussion, but we may consider some general principles.

In the first place, in the suppurative labyrinthitis the aim of surgery is not to alleviate the vertigo, but to ensure against meningitis. It will now be generally admitted that, in the first enthusiasm for labyrinthine surgery far too many labyrinth operations were done, and sometimes ill-judged operations precipitated the very disaster which the surgeon hoped to prevent. On the other hand many of us believe that the reaction has now gone too far; the belief that no labyrinth operation should be done until meningeal signs appear is responsible for dangerous delays. No meningeal signs can be found until the meninges are irritated, and in the most dangerous infections massive invasion precedes irritation.

On this basis it is possible to formulate some general rules.

1. So long as the labyrinth preserves any function it is unlikely that infection of the cavity has taken place.
2. When the labyrinth is destroyed in the presence of a middle ear infection, it is legitimate to assume that the labyrinth is itself infected.
3. When the cavity of the labyrinth is infected, and not till then, the meninges are in danger.

On these rules our treatment will be as follows:—

1. In circumscribed labyrinthitis the labyrinth cavity is unaffected. A mastoid operation, either partial or complete, must be done so as to expose the diseased area of the labyrinth wall. The diseased area must not be interfered with in any way. No labyrinth operation must be done. The only exception is in the rare cases where the erosion does not heal, although the rest of the mastoid cavity does, and incapacitating vertigo persists. In this condition it may be necessary to destroy the labyrinth in order to relieve the patient. The best way of doing this is by injection of alcohol into the labyrinth through the fistula. The operation, although simple, is not devoid of risk, and must only be undertaken as a last resort.

2. In serous labyrinthitis the defences are obviously adequate. Unless signs of a suppurative invasion appear no operation of any sort should be done until all the signs of the attack have passed off. Then a radical mastoid should be performed.

3. In diffuse suppurative labyrinthitis the labyrinth should be opened and drained as soon as destruction is complete, as shown by the caloric test. As vertigo is absent in latent labyrinthitis perhaps this should not be included in our discussion. It may, however, be laid down as a general rule that in latent labyrinthitis if any operation at all is done it should include drainage of the labyrinth unless it is already obliterated by fibrous tissue.

WHERE INFECTION HAS REACHED THE POSTERIOR FOSSA

In this group are: (i) cerebellar abscess; (ii) retro-petrous collections—(a) extradural retro-petrous abscess, (b) localized meningeal collections, either serous or purulent.

Cerebellar Abscess

The signs of cerebellar abscess are classified as (a) signs of increased intracranial pressure, and (b) localizing signs due to disturbance of special cerebellar function.

We are concerned here only with the signs due to disturbance of special function, and particularly with one sign—vertigo. Vertigo is the most common special sign of cerebellar abscess, but a few cases have been described in which no vertigo was present in the early stage. The difficulty in distinguishing between cerebellar and labyrinthine vertigo may be great, and it is increased by the fact that between forty and fifty per cent. of all cases of cerebellar abscess are caused by extension of a labyrinthine suppuration. Some generalizations are permissible.

1. In labyrinthine lesions vertigo is more noticeable than motor anomalies of equilibrium. In cerebellar lesions the opposite is true.

2. In cerebellar lesions the irritative signs are more noticeable than the compensatory; in acute labyrinthine lesions the irritative stage is very short.

3. In acute labyrinthitis the signs such as vertigo follow an orderly course, progressing rapidly until destruction is complete, remaining for a while at a maximum, then gradually diminishing. In cerebellar abscess vertigo and other local signs increase as the disease advances, but unevenly; there are remissions and exacerbations.

In cases of cerebellar abscess with an intact labyrinth there are some recognized peculiarities of the vertigo. The sense of movement is in the direction of the nystagmus, that is, of the rapid component, but the actual falling is often in the same direction; it is usually towards the affected side. The vertigo and nystagmus, unlike labyrinthine vertigo and nystagmus, are not usually altered by changing the position of the head.

When labyrinthine function is lost and vertigo and nystagmus persist or increase, especially if any signs of intracranial hypertension are present, a cerebellar abscess should be suspected. The direction of nystagmus and of the sensation of movement are unreliable guides.

Retro-petrous Collections

(a) Extradural retro-petrous abscess. This is a rare condition, and is usually due to caries of the petrous associated with suppurative labyrinthitis. The vertigo is usually that of labyrinthine destruction, and the diagnosis is made more on the signs of meningeal irritation.

(b) Localized meningeal collections are characterized by signs of cerebellar irritation, intense vertigo associated with headache and sometimes mastoid tenderness.

Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

EXFOLIATIVE DERMATITIS: ? STREPTOCOCCAL SEPTICAEMIA

The following case is of interest because of the unusual nature of the terminal symptoms.

The patient, a man aged 56, was a gifted musician and rather highly strung, though otherwise very healthy. About the middle of May, 1930, he began to be troubled by irritation of the skin, which in June began to peel. Between July 8th and July 20th he was treated as an in-patient at a provincial hospital. His condition at that time was considered to be a seborrhoeic eczema, and affected the neck and ears, the arms and legs. He improved, and took his own discharge. The improvement was not maintained, and on October 17th he was admitted to another hospital. On this occasion he was found to have a generalized affection of the skin, which came off in sheets. The toe-nails were likewise affected. The glands in the axillae and groin were enlarged, hard, and painful. Examination of the blood and urine did not then reveal any abnormality, and he improved for a while, until transferred to this hospital on November 9th on account of cardiac complications.

When admitted he was thin, weak, and ill. The skin was taut, greyish, and shiny, with scattered exfoliating patches. No petechiae were noted. The right knee-joint was swollen and tender; the left ankle-joint was similarly affected, but to a less degree. A few enlarged glands were found in the axillae, together with what appeared to be recently healed furuncles. The teeth were in extremely bad condition. The heart appeared to be slightly enlarged, and systolic murmurs were heard at the apex and base. No thrill was detected. The liver was enlarged to three fingerbreadths below the costal margin, and the abdomen contained free fluid. Albuminuria was present. Temperature 99.8° F., pulse 80, respirations 20. The next day the temperature rose to 104.4°, and slight delirium was noted. The Wassermann and Widal tests were negative. The right knee-joint was aspirated, and

a thin yellow pus was found, direct smears and cultures of which showed long-chained streptococci. Although the general condition was rapidly getting worse a distinct improvement in the skin had followed the use of zinc and boric ointment. By the fourth day exfoliation had almost ceased, but it was increasingly evident that this was of little moment. Fever persisted, and delirium and weakness became more marked. The knee-joint was repeatedly aspirated. The legs became oedematous, and the patient finally died in coma seven days after admission.

At necropsy a number of minute petechiae were seen in the skin. They were partly hidden beneath the few remaining patches of exfoliation. The pleural cavities contained about a pint of clear fluid. A thin purulent fluid was found in the pericardium. The heart was rather pale, and long, soft vegetations were found on the aortic and mitral valves. A gland near the aortic arch contained pus. There was a scarred white nodule near the apex of the right lung. The liver, spleen, and kidneys were enlarged and congested. There was considerable free fluid in the peritoneum. The synovial membrane of the right knee-joint was injected and roughened. No other abnormalities were noted. Sections of the kidneys showed an acute haemorrhagic glomerulo-tubular nephritis. A few Gram-positive cocci in pairs were observed. Sections of the liver showed old cirrhotic changes, with marked hyaline degeneration.

It is interesting to speculate on the course of events in this case. The relationship between the long-continued exfoliation and the apparently terminal embolic phenomena is uncertain. What part was played by the grossly septic teeth is doubtful; but it is possible that the endocardium was directly infected from this source, and not from the primary disease of the skin. The latter might have been traceable to the oral sepsis.

Cases of exfoliative dermatitis complicated by endocarditis and pericarditis, arthralgia, etc., have been reported by various authors, and some of these appear to have recovered.

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