many small subpleural haemorrhages. The other organs were normal. Microscopically the appearances were characteristic of encephalitis lethargica. The sections of the mid-brain, pons, and medulla showed the following features: great congestion of all the vessels with infiltration of the perivascular spaces, which were also dilated. This perivascular cuffing was most marked in the sections from the pons and medulla. Haemorrhages were seen in various parts, a very large one being present near the lamina terminalis. The ganglion cells in the brain stem showed all degrees of degeneration—some of it pigmentary—and loss of nuclear staining.

COMMENTARY

Three cases of encephalitis lethargica, proved at post-mortem, are described. In all of them there was a prodromal period lasting from one to two weeks, consisting of malaise, headache, lethargy, and constipation.

The first case pursued the characteristic course of a sub-acute disease, with its maximal incidence on the mid-brain, spreading thence upwards to the hypothalamic region and basal ganglia, and downwards to the pons and medulla oblongata.

Cases II and III were similar to one another in type. Both were remarkable for the fulminating course of the disease once the prodromal period was passed, the acute stage lasting only four days or so. Both showed a complete mental obliteration, and both terminated similarly with a hyperpyrexia.

The macroscopic post-mortem findings were typical of encephalitis lethargica, the main points being the pink discoloration of the grey matter, the vascular congestion, and the presence of multiple minute haemorrhages, giving the brain a flea-bitten appearance. This was especially noticeable in the region of the basal ganglia and in the brain stem. The exceptional severity of the disease in Case III was attested by the presence of haemorrhages beneath the endocardium, pericardium, and pleura—a finding to be expected where an acute toxic process has been at work.

Microscopically the characteristic cuff of lymphocytic infiltration in the perivascular spaces was present in two of the cases. At the same time there was degeneration, often pigmentary in nature, of the nerve cells. The sections also showed a certain amount of spreading apart of the neuroglial elements due to oedema. Although the sections of Case II did not show the lymphocytic cuffing of the small vessels, neuronc degeneration was a marked feature, and the similarity of the clinical course of the disease to that of Case III, taken in conjunction with the macroscopic appearance of the brain, leaves little room for doubt as to the true nature of the condition.

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PAIN REFERRED TO THE EAR *

BY

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Pain felt in an otherwise apparently normal ear presents a diagnostic problem often of some interest. I say "apparently normal" since at times the membrane shows no appreciable change, even when mastoid suppuration or yet more serious trouble is present.⁵ But when the earache is really due to disease elsewhere, if on examining the ear we find some lesion possibly sufficient to account for the pain it is only too easy to walk into the trap—particularly where the pain is severe or the patient manifestly ill and we feel compelled to adopt active measures without delay. The pain may be due to trouble near but not in the ear, such as arthritis of the temporo-mandibular joint. Earache is not infrequently the first symptom of mumps, and may occur before any swelling is noticed. Even a mixed tumour of the parotid may have earache as the only symptom.

HERPES OTICUS

Herpes oticus provides another possible catch. The pain is intense, sometimes with great tenderness of the meatus and mastoid, and may precede the eruption by several days—among ten cases I found intervals of six, four, and three days. There is some fever; in one case the temperature was 102° F. Deafness, nausea, and slight vertigo are common, and the membrane may be a trifle pink. Points of diagnostic importance are a constant boring ache—as contrasted with the fluctuating, throbbing pain of otitis—and extreme tenderness of the skin to very light touch. Even when a vesicle simulates a perforation, careful examination will reveal the difference, the discharge being slight and sanious; sometimes a history of exposure to varicella is obtained. No doubt the majority of cases of herpes oticus are due to involvement of the geniculate ganglion,² though in my experience only one-third have facial paresis, and I have only once

seen vesicles on the tongue. Twice I have seen an eruption on the soft palate, with dysphagia, which is perhaps evidence of glosso-pharyngeal affection. Herpes oticus with paralysis of the vocal cord has been recorded.³ The two following cases illustrate vagus herpetic earache, and the third case vagus referred earache without herpes.

CASE I.—*Earache from Herpes Oticus, with Pneumonia*

W. H., aged 14, had severe pain in the left ear for three days, following a cold. He had become much worse, with giddiness, vomiting, and a temperature of 104.4° F. and pulse of 120. There was slight sanious oral discharge and extreme tenderness of the mastoid tip. I was sent for late at night to perform an operation. When I saw the boy in the anaesthetic room I was struck by the "pneumonia" look—flushed cheeks, hot dry skin, bright eye, etc. The ear showed what looked like a perforation just in front of the umbo, but on careful inspection I thought it was a ruptured vesicle. All that could be found in the chest were a few crepitations in the right base, but thirty-six hours later (not before) there were typical signs of lobar pneumonia.

CASE II.—*Earache from Herpes Oticus, with Pneumonia*

J. E. T., a man aged 31, had symptoms of fever, deafness, and right earache, worse on coughing, following influenza. He had typical pneumonia of the right upper lobe, and a herpetic eruption on the right tympanic membrane.

CASE III.—*Earache due to Pneumonia*

A boy of 10 came to the casualty department of the Royal Infirmary complaining of bad earache of a few hours' duration, following a "chill." There was no tenderness, and the ear appeared quite normal; but the boy was obviously ill, and examination of the chest showed consolidation of the left lower lobe.

OTHER CAUSES

Neuritis is a not uncommon cause of earache. Exposure to cold is the usual aetiological factor, and there is sometimes facial paralysis in addition. I have seen a number of cases in taxi and lorry drivers, generally on the right side—probably because that ear is more exposed to the wind than the other. The trouble is usually transient, but some cases have proved very persistent. The pain of neuralgia is seldom confined to the ear; indeed, it tends as a rule to be felt round rather than in it. True geniculate tic douloureux seems to be very rare; the pain is paroxysmal, confined to the ear, severe and lancinating in character.¹

* Opening paper to discussion on referred pain, at a meeting of the Sections of Otolaryngology of the Royal Society of Medicine, held at Portsmouth, June, 1932.

When no local explanation of the pain is found, attention is, of course, directed to the mouth, pharynx, larynx, and nose. Dental disease is, in my experience, much the most common cause of "referred" earache. Any tooth may be responsible, but nearly always it is a molar. Even when the teeth appear quite sound, a meticulous examination by a dental surgeon may bring to light unsuspected caries, or a skiagram may show an impacted wisdom tooth. I have been puzzled by a case of earache in a child of 3. Twice, after a cold, there was intense earache all night. The membrane was normal, warm glycerin drops did not relieve, adenoids were absent, and the teeth appeared to be healthy. The dental surgeon, however, found an area of caries, and removal of the offending tooth banished the trouble.

Such conditions as a foreign body in the pharynx, a quinsy, laryngeal tuberculosis, or a nasopharyngeal fibroma may produce earache; but it is not likely to be the first or the most prominent symptom. It may be, however, in the case of a tonsil calculus or a crypt infection. Ulceration, due to Vincent's spirillum concealed behind the faucial pillar, may occasion difficulty. Cancer in any part of the nasopharynx, pharynx, larynx, or tongue frequently gives rise to pain in the ear, especially when the tonsil or the pyriform fossa is involved. Such pain, worse at night but not related to swallowing, may precede other symptoms by several weeks. Abernethy observed: "Wool in the ear and a lump in the neck mean cancer." The point is important, since many of these patients have very evil teeth, and only too often, even where manifestly malignant glands are present, time is wasted in awaiting the results of dental treatment. When the primary growth is in the nasopharynx or the auditory tube, otitis media is an early and often misleading complication.

AFFECTIONS OF THE SINUSES

Nasal sinusitis may provide a most elaborate trap for the surgeon—especially sphenoidal sinusitis, which (particularly in the young) is easily overlooked. It arises often as a complication of influenza: the pain is severe, and accompanied by fever and other evidence of suppuration; the severity of the symptoms may actually be due to the *absence* of discharge from the affected cell. Sometimes the ear itself is infected. Difficulty with regard to the other sinuses arises only rarely. In my experience the pain of chronic sphenoidal sinusitis is typically felt somewhat behind the mastoid; but in acute sinusitis the pain is actually in the ear, as pointed out by Mr. Tilley in 1905.⁴ A vertico-submental skiagram is of great value in diagnosis.

CASE IV.—*Earache due to Antral Suppuration*

D. G., aged 10, complained of left earache during four days, following a cold. The pain was throbbing in character, and worse at night. The mastoid was slightly tender. Temperature 100.6° F., pulse 90. Ears and nose showed nothing abnormal at all; there were no adenoids, but the tonsils were enlarged. Four days later the temperature was still high; earache persisted; there was vomiting and giddiness—no nystagmus; hearing normal; nose normal. The attack subsided, and he went home. A week later muco-pus was seen in the left middle meatus. Exactly a month after the first complaint I explored the left antrum. It was full of thick pus, and was opened; the right antrum contained clear mucus; there was a complete recovery, with no further attacks.

CASE V.—*Acute Sphenoidal Sinusitis simulating Mastoiditis*

T. G., aged 15, was brought to me in January, 1930, as a case of acute left mastoiditis. Early in 1926 he had had pneumonia with empyema, since when the left ear had discharged. Six weeks after an operation for tonsils and adenoids in December, 1929, he complained of pain behind the left ear, with tenderness. The temperature was 101° F. He was considered in the out-patient department to be

suffering from mastoiditis. I was operating at the time, and regret that I did not myself examine him. There was no post-aural oedema, and a Schwartze operation revealed perfectly normal bone. The cells contained air and a trace only of moisture, the antrum was empty and healthy, and only a trace of pus came back through the aditus when it was cleared. It seemed impossible that this would account for his symptoms. I therefore passed a cannula into the left sphenoidal sinus, and drew up several cubic centimetres of thick pus. The right sinus was healthy; both antra were infected, the pus being streptococcal. Chips from the mastoid were sterile. The left sphenoidal sinus was opened. He made a good recovery.

CASE VI.—*Sphenoidal Sinusitis, Meningitis, Septicaemia : Death*

E. E., a boy aged 9, previously healthy, had fourteen days' fever and malaise, seven days' headache and right earache; vomiting, collapse. On admission, obvious meningitis. Lumbar puncture—fluid turbid, streptococcal. Two c.cm. of thick pus aspirated from right sphenoidal sinus. Death occurred two days later. Necropsy revealed purulent meningitis; the right sphenoidal sinus was full of pus, and there were vegetations on the mitral valve.

CASE VII.—*Recurrent Sphenoidal Sinusitis simulating Mastoiditis*

R. A., aged 4, complained of "attacks" during which he suffered intense headache, with fever, and "lost control of himself; he would beat his head on the floor with the pain." In September, 1930, he had measles, and came to me a month later with right suppurative otitis media (first attack). The nose was running; he was drowsy and irritable, and was vomiting—temperature 102.8° F. There was no nystagmus or rigidity of neck, nor was there any additional evidence of mastoiditis. I advised against operation. The aural discharge ceased and the hearing became normal, but pain in the ear persisted. He had another exactly similar attack in December. In January I explored the antra, a skiagram having shown the right one to be opaque. Both were healthy and sterile. A week after this he had a similar attack, with earache but no other sign of otitis. During the year he had several more attacks, in one of which there was right frontal headache and nasal discharge. In January, 1932, I again explored the sinuses. The antra were quite healthy, as was the left sphenoidal sinus. The right sphenoidal sinus was full of thick streptococcal pus, and was opened. He has since been free of his trouble.

This child had precocious development of the frontal sinuses, but on account of his age I fear I neglected the possibility of sphenoidal sinusitis when I first saw him.

OTHER POSSIBILITIES

Earache may be the first symptom, and the only "localizing sign," of meningitis of any type—tuberculous, meningococcal, pneumococcal, or septic. In the latter particularly it is intense and persistent, and is not relieved by rupture or incision of the membrane. Any of these varieties of meningitis may occur either with a normal membrane or with otorrhoea.⁶ Pain referred to the ear from some other intracranial lesion may lead one astray.

CASE VIII.—*Earache due to Aneurysm of the Circle of Willis*

R. B., aged 40, had had very bad pain in the left mastoid area for ten months. For six months the vision of the left eye had been getting misty, and now the left visual field was reduced to a 10-degree circle. The ear was quite normal. A skiagram showed that the right sphenoidal sinus was less clear than the left. Exploration of the sinuses was negative, except that from the right sphenoidal a little sterile muco-pus was drawn, from the left sphenoidal streptococcal muco-pus, and from the left posterior ethmoidal cell pus which grew pneumo-bacilli; the two latter were opened. Six days after the operation he developed signs of meningitis, to my horror; on the ninth day his temperature came down, but he suddenly collapsed, with extreme cyanosis of the left side of the head and neck, and died immediately. At necropsy

we found a leaking aneurysm of the left side of the circle of Willis, which had burst.

In another case in my department, under Mr. Angell James, mastoiditis was simulated and an operation performed; the lesion was an aneurysm of the basilar artery.

Oöphoritis appears occasionally to produce earache. Sometimes we are compelled to regard the earache as functional in origin, as in the case described below.

CASE IX.—*Neurotic "Earache"*

R. N., a female aged 24, was admitted as a case of mastoiditis, with severe left earache, intense tenderness to lightest touch on mastoid and behind, giddiness, and falling. No nystagmus or nausea. Temperature 98.6° F., pulse 76; hearing normal, tympanic membrane normal. She seemed quite quiet and intelligent, and anxious to have an operation—"anything to get rid of the pain." Careful examination revealed no physical signs. I made the diagnosis of quasi-hysterical neurosis, which rather startled her own doctor. When told that no operation was necessary she lost all pain. Later I found out that she had just lost her job. Three years before she had suffered from hysteria, her betrothed having married her best friend just before the date fixed for her own wedding.

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THE LATENT MASTOID AS A CAUSE OF CHRONIC AURAL SEPSIS

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In the vast majority of cases of acute otitis media the path of infection is via the Eustachian tube. Owing to the early closure of the latter, the inflammatory exudate is under pressure. It must be rare indeed for the mastoid antrum to escape infection.

The acute mastoiditis which occasionally supervenes may present the classical signs of mastoid involvement. Often, however, these are conspicuous by their absence. The only definite physical sign may be the persistence of the meatal discharge. Reluctantly we operate, and we find a frankly purulent mastoid. Many such cases must be missed. What becomes of them? The answer is given by the chronic discharging ears that come to the radical mastoid operation. Whatever may be the condition determining operation in these latter cases, however completely mastoid signs and symptoms may be absent, they almost all agree in presenting at the operation unequivocal indications of old-standing mastoid infection.

When we consider the occasional, almost fortuitous, discovery of the more or less latent mastoid infection in the recent case, and the almost constant indications of prolonged latency of the mastoid infection in the old-standing case, is not the conclusion inevitable that these latter represent the mastoid extension of the original acute infection which, having run throughout a latent course, has been missed? If this is so, if these latent mastoid infections remain after the subsidence of the acute otitis media, then our failure to diagnose them is responsible for the great bulk of chronic aural sepsis, with its progressive deafness and potential dangers.

How far the mastoid bone with its diploic tissue or air cells is involved will depend on the virulence of the infection, and on the resisting power of the patient. It will, however, depend also on the rapidity with which drainage is established. Rupture or paracentesis of the drum is at the best imperfect drainage for the antrum. With partial obstruction of the aditus it becomes still less efficient. With actual involvement of the mastoid bone this drainage can do little more than serve to maintain the latency of the mastoid infection.

The discharge of chronic aural sepsis may show prolonged periods of complete remission. A scarred drum alone remains to tell the tale of previous active sepsis. As there is no reasonable ground for denying this complete latency in the case of the mastoid infection, while admitting it, as we must, in the case of the tympanic cavity, we are driven to the conclusion that even the absence of a discharge is no certain ground for assuming a non-infected mastoid. Indeed, we have only to postulate the early and complete closure of the aditus to envisage the possibility of a chronic but entirely latent mastoid infection, coexisting side by side with a normal middle ear, normal drum, and normal hearing. The two following cases suggest that this actually occurs:

CASE I

A boy of about 12 was sent in with pain, tenderness, and marked swelling over the right mastoid, with a corresponding displacement of the pinna. The drum and external meatus were, however, absolutely normal. So also was the hearing. There was nothing on the scalp, and no concurrent disease to suggest a blood-borne infection. Operation disclosed a mastoid filled with pus.

CASE II

A woman of about 40 came to me with pain in the right ear of recent onset. There was no previous history of ear trouble. She had a red and bulging, but otherwise normal, drum, which I incised. Some fourteen days later there were signs of mastoid involvement. At the operation the mastoid presented every appearance of an infection of many years' standing.

Neither of these cases definitely proves that the mastoid infection originated in an acute otitis media, which, as far as the middle ear was concerned, cleared up completely. Blood-borne infections of the mastoid are, however, so rare and infections via the Eustachian tube so common that this explanation is at least a probability.

These cases must be rare, and would, in any case, be difficult to diagnose till physical signs arose. Cases in which a discharge persists after the acute stage has subsided are very much more common. They represent nearly all cases of chronic suppurative otitis media. In how many of these is the persistence of the discharge due to the presence of residual mastoid sepsis—sepsis latent save for the discharge? My experience suggests that it is in a very large proportion of them. The following cases lend support to that view:

CASE III

A girl of 12, with a copious discharge from both ears, dating from an attack of measles eighteen months previously, developed a swinging temperature. As nothing could be found to account for this, I was called in because of the aural discharge. By that time the temperature had been swinging for eleven days. A bilateral cortical operation showed both mastoids full of pus and the left lateral sinus exposed in the abscess. She recovered.

CASE IV

Another child, of about the same age, came to the outpatient department with a history of a discharging ear and some very doubtful signs of mastoid involvement. She reappeared a week later, desperately ill, with marked adenitis round the right jugular and a swinging temperature. Immediate operation on the mastoid and thrombosed lateral sinus failed to save her, and she died of meningitis.