REFRESHER COURSE FOR GENERAL PRACTITIONERS

THE ACUTE EAR

BY

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The cardinal symptoms of ear disease are earache, deafness, vertigo, and tinnitus. Any of these symptoms, if of acute onset or if severe, might be called an "acute ear." This article will deal only with acutely painful affections of the external and middle ear. The pain is, in the majority of cases, inflammatory, but in a small minority no local lesion is to be found in the meatus or tympanic membrane.

To decide whether the site of pain lies in the external or middle ear it is essential to examine the ear carefully. The electric auriscope affords the easiest view, especially if the patient is a child or is in bed. The skin of the meatus is normally pale pink, and the presence of the speculum should cause no discomfort. The tympanic membrane can be seen at a distance of about one inch (2.5 cm.) from the concha. Since the ceruminous glands and hair follicles occupy only the outer two-thirds of the meatus, infections—e.g., boils—occur only in this area.

Acute Infections of the External Ear

The Pinna.—The pinna is subject to the same infections as other parts of the skin and does not come within the compass of this article.

The External Auditory Meatus.—Furunculosis, or boils in the glands or hair follicles of the outer two-thirds of the meatus, is an extremely painful condition. At an early stage the boil may be too small to produce any swelling and can be diagnosed only by the presence of an area of red skin and the discovery of a tender spot by a probe. Later, an obvious tender swelling or swellings simplify the diagnosis.

The lymphatics of the meatus drain to glands (1) in front of the tragus, (2) behind on the surface of the mastoid process, and (3) below the tip of the process. Tenderness in any of these positions is an aid to diagnosis. Lymphangitis may occur as a complication. The swelling to which it gives rise is over the mastoid process and is often sufficient to push the pinna forward and downwards. It pits on pressure and may raise a suspicion of acute mastoiditis with subperiosteal abscess. The type of tenderness differentiates between the swellings—that of lymphangitis is superficial, and if pressure is maintained the bone can be reached without causing any increase in pain; in the case of mastoiditis, on the other hand, the harder the pressure the worse the pain.

No loss of hearing occurs in furunculosis unless the boils are extensive enough to block the meatus; even then a

narrow speculum insinuated between the boils will restore hearing to normal.

Treatment of Furunculosis

The meatus, including that part deep to the boil, must be kept clean and dry. Every otologist has favourite drugs for this purpose, either in the form of drops or for application on gauze wicks. Well-tried prescriptions are:

Glycerin of phenol and glycerin, equal parts.

Saturated solution of magnesium sulphate, with or without glycerin.

Ichthyol in glycerin (10%).

Gentian violet in spirit (2%).

Silver nitrate (10%).

Aluminium acetate (8%).

Penicillin drops should not be used.

Incision should only be employed when a furuncle shows pus at its apex, though rupture of the boil may be hastened by the application of a minute drop of pure carbolic acid. The application of warmth is comforting, and an electrically heated pad is satisfactory for the purpose.

Although a course of sulphonamide may effect a dramatic cure, the fact that so many different drugs have been advocated is an index of the unsatisfactory results of treatment of this most painful affection.

Acute Infections of the Middle Ear

Any acute infection of the upper respiratory tract involves the nasopharynx and may spread to the Eustachian tube and so to the middle ear.

Anatomy.—The middle-ear tract extends from the nasopharyngeal end of the Eustachian tube, through the tympanum or middle ear, and thence through the aditus to the mastoid antrum to the air cells in the mastoid process. The tract may be conveniently divided into three parts: (1) The Eustachian tube, (2) the middle ear or tympanic cavity, and (3) the mastoid antrum and mastoid cells (see Figure). Inflammation may be mainly confined to part 1, or to parts 1 and 2, causing acute otitis media. Inflammation can spread from here through the aditus to the

1. Opening of tube into tympanum. 2. Aditus—opening from attic into the antrum. 3. Plate of bone forming the roof of attic in front and of the antrum behind; it is also the floor of the middle cranial fossa. 4. Dura mater.

mastoid antrum. It is probable that some inflammation develops in the antrum in most cases of acute otitis media, and in suppurative cases the mastoid cells become involved to produce acute mastoiditis. In the case of severe haemolytic streptococcal or pneumococcal infections the whole tract may be rapidly involved.

The infecting organism is usually a streptococcus, but may be a staphylococcus or pneumococcus. Some specific fevers, notably measles, scarlet fever, and diphtheria, as well as influenza, may lead to acute otitis media. Paranasal sinusitis may supply the infection, and fresh-water bathing and the presence of adenoids are sometimes contributory causes.

Symptoms and Signs of Acute Otitis Media

Local Symptoms.—Earache is severe and of a stabbing character, with intermissions. Deafness is always present, but varies in degree.

General Symptoms.—Toxaemia, loss of appetite, malaise, rise in temperature to as much as 104° F. (40° C.) in children or 100° F. (37.8° C.) in adults.

Signs.—As the tympanic membrane is the only part of the middle ear that can be examined, recognition of the changes occurring in it are important. Normally the membrane has a pearly colour, is shiny, and slightly concave. In the early stages of inflammation a sheaf of tiny vessels is seen running immediately behind the malleus; small vessels directed towards the centre of the membrane also become apparent at the periphery. The posterior part soon becomes red. The next stage is bulging of the membrane, at first in the posterior part but later affecting the whole structure. Finally, if there is no surgical interference, rupture takes place and the blood and serum are discharged. Should the infection be mild, resolution may occur in the earlier stages and the discharge last for a day or two only. The rapidity with which these changes take place is to a great extent an indication of the severity of the condition.

Drug Treatment

It is a truism to state that chemotherapy and antibiotics have changed the picture of acute infections, and therefore of acute otitis media, but it is equally correct to point out that best results can only be achieved by early and adequate dosage. It must also be realized that these drugs do not replace surgery, but aid recovery and go far to prevent complications.

The choice of drugs lies between the sulphonamides and penicillin. It has been suggested that the former should take first place and penicillin be reserved for cases that fail to respond. Whether sulphadiazine, sulphamezathine, or any other alternative is chosen, 2 g. (four tablets) are given as a first dose, and thereafter 6 to 8 g. in equal doses in the following 24 hours. In the case of children half these doses are to be used, or dosage given on the scale of 0.15 g. per kilo body weight. Treatment is continued for four or five days.

Penicillin.—The preparation of choice is procaine penicillin (an oily preparation with aluminium stearate), since this has the advantage of maintaining a therapeutic blood level in 80–90% of patients for 24 hours. One ml. of this preparation contains 300,000 units of penicillin. Injection is intramuscular and is given daily for as long as necessary. Should a watery solution of penicillin be used, 500,000 units should be given twice a day for at least four days. This treatment must be instituted as soon as the diagnosis has been made by observation of the red appearance of the membrane.

Surgical Treatment

Should the membrane continue to appear red after 48 hours, should pain persist, or should the membrane bulge, the operation of myringotomy must be performed. If the membrane is bulging when the patient is first seen myringotomy should precede the administration of drugs. After spontaneous rupture and discharge, penicillin should be

administered. If discharge from the ear continues for two weeks in spite of chemotherapy or penicillin the mastoid cells must be opened.

In the pre-sulphonamide days the diagnosis of acute mastoiditis, and therefore the indications for operation, rested on a sound basis of symptoms and signs—pain; continued, often profuse, purulent discharge; swinging temperature up to 99.6° F. (37.6° C.); toxaemia, deafness, and tenderness over the mastoid process; and in late cases swelling and redness of skin. If all these features were present the diagnosis was obvious. There were cases, however, in which some, or indeed most, of these signs were absent. Indication for operation then rested on deafness and the persistence of one other symptom or sign. This rule is still more important to-day, because sulphonamides and penicillin, though cutting short the acute otitis, have the drawback of masking the symptoms. Pain and fever cease and the patient feels well, but the disease may progress. In consequence some otologists forbid drug treatment, but this drastic step is unjustified if the rules concerning the indications for operation are strictly observed. The absence of typical symptoms and signs following drug treatment has been named "masked mastoid."

Continuance of any one symptom—e.g., pain, discharge, toxaemia, or even deafness alone—calls for operation. X-ray examination of the mastoids aids diagnosis, for the affected cells appear clouded when compared with those on the opposite side. Should operation not be performed in a case of masked mastoid because the patient appears to have recovered, the sudden occurrence of facial paralysis may reveal that the disease is progressing and requires immediate operation. Results are good, and the paralysis clears up in about three weeks.

It has been stated that intracranial complications of otitis are more common since drug treatment became almost universal. If this is so, it may be the result of reluctance to operate on the mastoid, or delay in doing so, in cases of apparent recovery. Sulphonamides have been accused of causing deafness, but some of the cases at least were due to unresolved middle-ear infection and would have been avoided by operation on the mastoid to drain the middle ear efficiently.

Acute Otitis in Infants

The problem of acute otitis in infants presents special difficulties, for the patients are unable to indicate the site of pain, and diagnosis is largely a matter of exclusion. The infant is ill and has a high temperature; he cries, refuses food, and may have diarrhoea and vomiting. Should the physician fail to find signs of abdominal or chest disease, or any local lesion, the ears must be considered as a probable cause of the illness. Even when changes in the tympanic membrane are minimal, myringotomy may release a small quantity of fluid or pus. If otorrhoea persists and the condition of the infant does not improve in two or three days the necessity of opening the mastoid arises. Infants tolerate injections of procaine penicillin, and by this means the prognosis is improved. Investigations have shown that injections of 300,000 units can be given to infants aged 1 to 5 days without causing disturbance or pain. This dose should therefore be administered to infants or young children found to be suffering from acute otitis media. The injection is made once a day. It is to be expected that this treatment will reduce the incidence of mastoid infection, but no figures are yet available.

In cases in which the infecting organism proves to be penicillin-resistant, streptomycin offers an alternative treatment.

Trauma

Rarely, an "acute ear" is the result of trauma. Accidental puncture of the membrane occurs from thorns, needles, hair slides, and hot or corrosive fluids entering the external meatus, and rupture of the membrane is a common result of near explosions.

Treatment must be directed to prevent infection. Syringing is to be avoided. Any debris should be wiped out with wool moistened with spirit and the meatus lightly plugged with sterile wool. Prophylactic injections of procaine penicillin may be given. Should infection and suppuration follow, the same rules apply as in the case of primary acute middle-ear infection.

A group of cases due to flying is also to be considered. Many fliers notice unpleasant sensations in the ears even when descending gradually, and if descent has been rapid some complain of severe pain. The ears usually become normal in an hour or so, but when pain and deafness persist inflation of the Eustachian tube or tubes generally relieves the patient. Politization, or self-inflation, is often successful. If not, the Eustachian catheter will be needed on a few occasions. Examination shows retraction of the membrane, which is often reddened. The condition is one of barotrauma.

FUNCTIONAL DIFFERENTIATION IN MAMMARY TUMOURS

The fifth Kettle Memorial Lecture was delivered at the Royal College of Surgeons on October 7, Mr. Lionel Norbury presiding, by Dr. BEATRICE PULLINGER, whose subject was the significance of functional differentiation in mammary tumours.

Dr. Pullinger said that she was one of the few of Kettle's students who, inspired by his courage and his love of pathology, and many other great qualities in him, persisted in their devotion to pathology in spite of his grim warning not to do so. His teaching and work were never limited to the descriptive field, but he had regretted that circumstances prevented him devoting his whole time to experimental pathology.

It was therefore fitting to recall Kettle's teaching with an account of what might be learned from an experimental study of functional differentiation in the mammary gland and its tumours. In the course of some experimental work the need arose to vary functional differentiation at will, and this had led to a quantitative study of such differentiation around the time of puberty. A few, not very successful, attempts had been made to measure the quantitative response of the mammary gland to hormones, mice being the test animals. She had been able, with the help of a colleague, to prove on a statistical basis that there was a quantitative relationship between dosage and response, but the precise relationship was variable, revealing a change in sensitivity the source of which had not been determined.

The mammary gland developed in response to comparatively few stimuli. Of the known stimuli oestrogen was surpassed only by its combination with progesterone. It was thought by some investigators that a product of the anterior pituitary also stimulated the growth of the mammary gland. The lecturer went on to mention certain types of pathological stimulus, and said that there was one which had so far eluded all research, starting and perpetuating the growth of adeno-carcinomas and squamous carcinomas. All traces of the Bittner agent were absent.

She then put forward a classification of different types of mammary tumours in mice according to type, agent, and morphological characteristics. It was known that heritable mammary tumours contained the Bittner agent, a virus-like entity, on which they were dependent for their growth and transmission. Viruses might be graded as fibroblastic, breaking up and destroying the cells; as passengers, causing neither harm

nor benefit to the host cells; and as formative agents, combining with the cells to cause cell proliferation and their own reproduction. There was also the possibility that both oestrogens and virus excited an intracellular link to effect these changes, though that hypothesis would be difficult to prove.

Certain non-virus-containing tumours gave no clues to the nature of the stimulus to cell division, and yet were morphologically indistinguishable from those which contained virus. Either a positive stimulus or the removal of an inhibition must be looked for; she herself favoured a positive stimulus, and the responses to specific hormones were themselves pointers to the presence of such a stimulus. The elucidation of abnormal stimuli was a matter of great difficulty on account of the lack of knowledge of normal stimuli, but, assuming that nature was economical in her means, the specific hormones provided clues for experimental trial.

Preparations and Appliances

HEPARIN ADMINISTRATION BY POLYTHENE TUBING

Dr. W. A. L. MACFADYEN, Royal Infirmary, Edinburgh, writes: It has been found possible to abolish the inconvenience, both to patient and medical staff, of repeated venepuncture in the administration of heparin by the use of indwelling intravenous polythene tubing and a special attachment. The apparatus required is shown in Fig. 1. The attachment (D) consists of a Record-fitting mount on to which is screwed a small hollow screw cap containing a replaceable rubber diaphragm (E). This has been specially made for the purpose by Archibald Young and Sons, Edinburgh. The tubing (A) is a length of the polythene cannula, size 1 (bore 0.5 mm.) of Allen and Hanburys. It has been found that this tubing can usually be threaded through a needle of bore B.W.G. 14 (B), but occasionally, owing to slight variations in the diameter of the tubing, a B.W.G. 12 needle may be required. Some 10-12 in. (25-30 cm.) of the tubing, having been found to slip easily through the introducing needle, is sterilized by boiling, with the precautions advocated by the makers. The attachment (D and E), having been sterilized and assembled, is filled with heparin (it requires only a fraction of 1 ml.) and attached to a sterile hypodermic needle (C) of such diameter that it is held firmly when inserted into the tubing. A needle of bore 15 S.W.G. is usually satisfactory. The tubing is filled with heparin and the initial dose of heparin injected into the chosen vein by means of a syringe and the chosen sterile introducing needle. The syringe is removed, the polythene tubing threaded through for 4-5 in. (10-12 cm.) into the vein, the introducing needle withdrawn from the vein and quickly slipped off the tubing, and the hypodermic needle with the special attachment quickly inserted into the free end of the polythene tubing. The point of entry of the tubing is sealed with collodion and covered with a sterile dressing, and the whole apparatus strapped down. It is helpful to tie a loose knot in the exposed portion of the tubing to prevent it slipping further into the vein. Succeeding heparin injections, are then given by the nursing staff by means of a syringe and a fine intradermal needle, the latter being inserted through the rubber diaphragm. The nurse should be instructed that the syringe should contain no air and that she should not draw back the plunger after inserting the needle, since it is

FIG. 1.—Apparatus for intravenous administration of heparin.