fluid and nerve endings. On the peripheral aspect of the canals is the middle ear, which communicates with the canals via the oval and round windows.

Just as we can get changes in the walls of the blood vessels of the eyes, so we believe we can get similar arteriosclerotic changes with or without haemorrhages in the blood vessels of the labyrinth, which would account for the vertigo that occurs in elderly people on stooping or turning their heads. These blood vessels may undergo spasm with loss of hearing, then relaxation followed by tinnitus, giddiness, and return of hearing, and it is a question whether such cases could not be cured by stripping the sympathetic nerves of the internal carotid artery, or by removing the superior sympathetic ganglion. Russell Brain considers that spasm of these arteries also accounts for the vertigo that occurs in sufferers from migraine.

The fluid in the canal can produce vertigo when it is moved, as when one performs the rotation or caloric tests, or is on a ship. It is said that to avoid sea-sickness it is best to lie flat on the back. This fluid may also be affected by toxins; thus in acute otitis media there may be vertigo and nystagmus to the affected ear due to absorption of toxin stimulating the nerve endings, or, if the infection is more severe, destruction of the nerve endings and consequently a nystagmus to the opposite side. In the case of the nystagmus to the same side, suction applied to the Eustachian tube or a paracentesis of the tympanic membrane will allow the toxic fluid to escape, or in severer cases a mastoid operation will be necessary. When there is nystagmus to the opposite side a serous or suppurative labyrinthitis must be diagnosed. As long as any hearing is present we diagnose serous labyrinthitis and do not operate, but when all hearing has gone and the hot caloric test is incapable of affecting the nystagmus we are forced to diagnose the suppurative condition. If the suppurative labyrinthitis is secondary to an otitis media I do not operate unless the signs of labyrinthitis increase instead of decrease, or unless meningitis appears, but if it follows an operation such as the removal of a polypus or a mastoid, I then perform a labyrinthectomy at once.

Besides reaching the semicircular canals directly from the middle ear as just described, toxins may reach the canals via the blood stream, as in the case of those from the intestine or from alcohol, tobacco, or a septic focus; thus one patient drove his car from side to side of the street, but when his tonsils were removed his vertigo disappeared.

Displacement of the Stapes

Another cause of vertigo is displacement of the stapes, which may be due to an oversecretion of fluid in the semicircular canals. This is considered by Mygind and Dederding to be the commonest cause of the kind of aural vertigo which is usually termed Ménière's disease. This water-logging of the labyrinth occurs in those who have faulty water metabolism; such patients are usually easily recognized, as they show a loss of low notes and absence of or decreased bone conduction, deafness which varies from day to day, an increase in weight, and a thickening of the subcutaneous tissues. However, I would not diagnose a patient as water-logged without giving 1,000 c.cm. of fluid, and seeing how much fluid the patient had passed, and how much he or she had increased in weight after an interval of four hours. The increase in weight equals the weight of the retained fluid, and thus, by weighing the patient before the fluid is taken and at the end of four hours, the amount of fluid retained is estimated. Such patients are cured by dehydration with salyrgan, a no-salt diet, and extreme limitation of fluid.

An attack of aural vertigo in which the patient has nystagmus and vomiting may be arrested by an injection of bulbo-capnine. This drug, orally administered, is also useful in some cases to ward off attacks.

Another cause of displacement of the stapes is blockage of the Eustachian tube, with consequent absorption of air in the middle ear. This occurs in inflammatory states of the Eustachian tube or in swelling of the mucous membrane due to water-logging. Such patients are cured by reducing the swelling in the Eustachian tube and by catheterization. To reduce the swelling in the Eustachian tube a nasal operation may be necessary.

At times no cause can be discovered to account for the presence of aural vertigo, and in those cases the best treatment is Mollison's—that is, destruction of the nerve endings by injecting alcohol into an opening made in one of the semicircular canals. This I have done on three occasions. Before adopting this treatment I used to destroy the cochlea and semicircular canals by a labyrinthectomy. The injection of alcohol, however, is easier, and exposes the patient to less trauma. Brain specialists such as Dandy, Cairns, and Dott cure this form of vertigo by dividing the seventh nerve, and lately Dandy has been successful in dividing the vestibular branch alone, and so retaining the remnant of hearing that is often present in these patients.

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TONSILLECTOMY WITH LOCAL ANAESTHESIA

A REVIEW BASED ON 500 CASES

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For some reason unknown to me local tonsillectomy has never received very favourable consideration in England; yet the mere fact that this is almost the only method employed in many of the large Continental clinics should be sufficient evidence that it is definitely worthy of greater consideration than it has hitherto received. Having just completed a series of five hundred cases in patients whose ages ranged from 16 to 74, I feel that a short description of the technique may not be out of place, since many advantages are claimed over the more universal dissection under general anaesthesia. The advantages of using a local anaesthetic are that the operation is considerably more speedy without there being any sacrifice of efficiency; that there is a minimum amount of bleeding (often there is literally none at all); that there is no vomiting; and that there is a cleaner dissection. The patient suffers no pain and extremely little discomfort.

Technique

The nurse sprays the patient's throat several times with 10 per cent. cocaine and adrenaline, and then the injection, which is the most important part of the operation, is commenced. It should be stressed that the quantity of novocain solution injected is even more important than its strength. About 30 to 40 c.cm. of a 1/2 per cent. solution is recommended.

With a long needle, the point of which is curved almost to a right angle, the novocain is injected from a 20 c.cm. syringe. Five injections, each of approximately 3 c.cm., are made into each tonsil, the anterior portion of the capsule receiving three and the posterior portion two. The needle is inserted just under the mucous membrane, which is immediately thrown out into a bleb as soon as the novocain reaches the tissues. It should be inserted first near the uvula, with the point turned outwards and downwards, a few millimetres from the free border of the pillar; then midway between the upper and lower pole, with the point directly downwards; and finally into the lower pole near the base of the tongue, with the point directly downwards. The remaining two injections are made behind the posterior pillar in the upper and lower poles respectively. Here, however, the needle point should enter the tissues horizontally, and the shaft of the needle be drawn well over into the opposite angle of the mouth. After the injection the pillars are, of course, very swollen and oedematous, and the patient should be left for ten minutes for absorption to take place. This wait is important, for during this time the capsule becomes so well infiltrated that the actual dissection of both tonsils may be completed with ease in less than three minutes.

The patient is now led into the theatre and placed in an ordinary chair. The surgeon sits facing him, and for convenience the patient is made to place a hand on each of his (the surgeon's) knees. A nurse standing behind the patient's chair holds the head in the required position and wipes away from the mouth any blood-stained saliva, after occasional spitting. No gag is necessary; nor, after the vulsellum has first gripped the tonsil, is a tongue depressor required. Beyond a sharp dissector, to incise the pillars, and a blunt dissector with a serrated edge, to free the tonsil from its now plainly marked capsule, no other instrument is required, except possibly a snare for the lingual prolongation. After the first tonsil has been removed it is a good plan to pack a small, loose piece of gauze into the fossa before proceeding.

If there are two or more patients for tonsillectomy it is a great saving of time to complete all the injections first, since the best effect from the novocain may be obtained if left for a minimum of ten minutes and a maximum of forty minutes. In this way two or three cases may be completed in considerably less than half the time taken by general anaesthesia. As there is little or no bleeding, an assistant is quite unnecessary. A powerful headlight is, of course, an essential.

Conclusion

Invariably the patient volunteers the statement that the operation has not been as painful as a tooth extraction, while all will admit, on being questioned, that "it didn't hurt a bit." It would seem from the patient's point of view that the apprehension is the worst part of the whole operation. This may be considerably allayed by a preliminary injection of morphine, and also during the operation itself by what the Viennese call "vocal anaesthesia." There is no doubt that a patter of conversation, however irrelevant, helps to quiet the patient. After the operation has been completed the patient walks back to his bed, and is able to return home within three or four days.

In this series of cases, which comprises every tonsillectomy that has recently come under my care—that is, unselected—I have had only one case where I was embarrassed by bleeding, and that finally required a general anaesthetic. There were three cases in the series that bled after getting back to bed, requiring the use of the clamp, but these were due to an incomplete dissection. No vessel has had to be tied. From the injection of novocain I have seen no harmful effects.

Clinical Memoranda

NON-TROPICAL SOLITARY ABSCESS OF LIVER

The subject of solitary abscess in the liver is usually associated in the mind with a tropical disease. It is of interest, therefore, to review a case where no possibility of a tropical origin could be admitted. Cabot,¹ discussing liver abscess, states that as a rule the diagnosis is made on the aetiology rather than on physical signs. Where there is no obvious cause such as amoebic dysentery or appendicitis the diagnosis becomes increasingly difficult. However, as Horder² has pointed out, such cases are often due to *Staphylococcus aureus* and are characterized by a latent interval between the original staphylococcal infection and the development of a liver abscess.

CASE RECORD

A. L., 20 years of age and single, had been in good health and following her occupation as a weaver until July, 1933. She then began to complain of slight intermittent pain in the right side of her chest which was at first regarded as pleurodynia. However, the pain became constant, more severe, and localized to the region of the eighth rib interspace in the right mid-axillary line. To digress for a moment, it is to be noted that later the liver was found adhering to the parietes at the place where she located the pain. Slowly her health deteriorated, anorexia, loss of weight, and night sweats being added to her original symptoms. After five weeks she took to bed, and one week later (September 7th) she was admitted to the Blackburn Royal Infirmary.

On admission, she appeared thin and anaemic. Her temperature was raised and there was obvious toxæmia. Her heart and lungs were healthy, and the chest moved normally on respiration. There was, however, a suggestion of bulging of the lower part of the right side of the chest, which must have been due to enlargement of the liver. The lower margin of the liver could not be palpated as there was considerable muscular rigidity in the right hypochondrium. The abdomen was thin and the spleen was not palpable. A search for tenderness was not very satisfactory as the patient could not distinguish between pain and tenderness. The kidneys were not palpably enlarged nor was tenderness elicited during palpation. A provisional diagnosis of perinephritic abscess was made, leading to a thorough investigation of her kidneys. An x-ray photograph of the urinary tract was negative. Her urine on examination revealed no abnormalities. Normal pyelograms were obtained from both sides, so it was decided to look to the liver as the probable cause of her illness. The chest was examined with the x-ray screen, but the diaphragm was seen to move normally with respiration and did not present any abnormal bulging or fixation.

On September 8th her blood was examined. The red blood cells were estimated at 4,400,000 per c.mm., haemoglobin 65 per cent. The white blood cells showed a leucocytosis of 20,000 per c.mm. with 80 per cent. of neutrophils in the differential count.

Whilst these investigations were in progress her temperature rose every evening to 102° F. and fell in the morning to 99°, and she was obviously losing ground every day. Liver abscess was suspected, and on September 10th the liver was explored with a Barker's lumbar puncture needle under a general anaesthetic. Introduced in the mid-axillary line and passed upwards and backwards, as is usually recommended, no pus was found, but, on directing the needle downwards and inwards towards the abdomen, thick pus was easily obtained. The abdomen was then opened by an incision just below the right costal margin. The liver was not enlarged downwards, but omentum and bowel were firmly adherent along its lower margin. Working up on the right side between the diaphragm and the lateral surface of the liver, an area of adhesions was encountered about three inches from the lower margin of the liver. On breaking this down the finger slipped easily into a large unilocular abscess cavity in the right lobe of the liver.

¹ Cabot: *Physical Diagnosis*, eighth edition, p. 380.

² Horder: *Medical Notes*, p. 73.