displaced downwards. On aspiration a pint and a half of sero-purulent fluid was removed from the chest; this showed on microscopical examination pneumococci in pure culture. As this fluid rapidly accumulated again it was found necessary, under local anaesthesia, to remove it more thoroughly than by aspiration. Through the opening formed by the aspirating needle a probe was passed, and then Sir St. Clair Thomson's needle tonsil forceps were introduced, which, when opened, allowed a sero-purulent fluid to pass away very freely. The opening was enlarged by the little finger, and lastly two drainage tubes were passed in. These drained the chest satisfactorily. The child was carried out into the fresh air the following day and went on to complete recovery.

What I wish to point out is the marked effect for good of the serum injections. The fluid in the pleura accumulated after the pneumonia appeared to be subdued, but notwithstanding the undoubted presence of soluble poisonous substances in the blood the antipneumococcal serum was effective. Lastly, it was not necessary to verify the type of pneumococci according to the method of Dachez and Amery, and use large quantities of this specific and modified serum intravenously. The serum was effective although used subcutaneously. If Cole's contention were correct, serum treatment would be quite useless in the stage of grey hepatization in pneumonia. My experience in many cases is quite the opposite.

REFERENCE.
1 *Journ. Exper. Med.*, 1918.

ABLATION OF THE LABYRINTH IN A CASE WITH MÉNIÈRE'S SYMPTOMS.

BY

COURTENAY YORKE, M.D., L'POOL, M.B., B.S. LOND.,
F.R.C.S. ENG.,

HONORARY LARYNGOLOGIST TO THE DIVERPOOL HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST; HONORARY ALBIST AND LARYNGOLOGIST TO THE VICTORIA CENTRAL HOSPITAL, LISCARD, AND TO THE BIRKENHEAD BOROUGH HOSPITAL, ETC.

IN 1861 Ménière published a case of sudden deafness, with severe dizziness and vomiting. The patient died, and at the autopsy a reddish plastic exudate was found filling the semicircular canals and extending into the vestibule.

This was the first case of the kind in which a *post-mortem* examination had been made and a relationship established between an acute disease of the labyrinth and a group of symptoms similar to those caused by an injury of the semicircular canals in animals.

Ever since Ménière's publication it has been customary, in strict nomenclature, to designate by the name of "Ménière's disease" a sudden haemorrhage (?) into the labyrinth, causing the violent onset—sometimes apoplectic form in character—of deafness, tinnitus, vertigo, nausea, and vomiting. It is now well known, however, that a combination of symptoms similar to the above may result from other causes than haemorrhage. Vascular disturbances of the labyrinth—hyperaemia or anaemia—which occur in many local and general conditions, cause temporary disturbance of hearing with transient tinnitus, vertigo, nausea, and vomiting. Again, in infective processes in the inner ear, these symptoms occur in a severe form, while in chronic middle-ear catarrh, otosclerosis and syphilitic disease of the labyrinth, Ménière attacks are not uncommon.

It must be remembered, also, that the exact pathology of Ménière's symptom-complex is often very uncertain, which is but to be expected when one considers the rarity of the opportunity for a detailed examination of the inner ear in a recent case and the difficulty of the technique involved.

Having regard to these considerations it would thus, both on clinical and pathological grounds, be more scientific and less confusing to discontinue the term "Ménière's disease," but to retain Ménière's name for the symptom-complex, which has its origin in the internal ear, and which is evoked by various states of the organ—namely, by injury, hyperaemia or anaemia, haemorrhage, serous or inflammatory effusion, and by degenerative changes and new growths.

It is of course obvious that, before a definite diagnosis of Ménière's symptom-complex can be made, the cochlear and vestibular functions must be carefully investigated on

both sides, and all other possible causes of similar symptoms eliminated. In cerebellar and other diseases of the nervous system, vertigo, nausea, and vomiting occur, and these are also common in many general disorders, particularly those of renal, cardiac, and gastric origin.

The case to be described was a man, W. E., 51 years old, who has been engaged for many years as a house steward, in which position he has had a quiet, leisurely life. He has always been very temperate in his habits, has never smoked or taken alcohol, and has never had syphilis or any serious disease.

The first symptoms commenced in May, 1913, with an attack of vertigo and sickness. Six weeks later he had a second similar attack, which left him with nausea and giddiness for three days. The attacks now recurred regularly every month or so, coming on suddenly and lasting for several minutes to several hours. The typical attack was ushered in with a "feeling of sea-sickness," which gave sufficient warning to enable him to lie down on the ground. This prodromal stage lasted only a minute or two, and then the attack set in acutely, with intense vertigo, nausea and vomiting, and subjective sensations of the ground rising and objects rotating. During the progress of the attack, which in some cases lasted six hours, he felt quite helpless and was obliged to lie motionless, with the eyes closed, as the slightest movement, or any attempt to look at objects, greatly aggravated the symptoms. Following the attack a state of prostration ensued, and a sick, dizzy feeling continued, often lasting for several days. In August, 1914, whilst blowing the nose, he became suddenly deaf in the left ear; with the onset of loud tinnitus. He states that, previous to this, he had observed nothing abnormal with the hearing. The deafness on the left side soon became almost complete and the Ménière attacks now steadily increased in frequency and severity.

The general health consequently became impaired. He lost weight and was reduced to a depressed and neurasthenic condition, which entirely unfitted him for any kind of work and made life almost unbearable. In February, 1917, he had two attacks of unconsciousness, which lasted, on the first occasion one hour and on the second occasion two hours.

I first saw the patient on March 7th, 1917, when I made the following record of his hearing on the two sides:

Left Ear.—Almost completely deaf to air conduction, only just hearing the loud voice close up to the ear. Bone conduction markedly reduced (20 seconds). Rinne negative. Weber to the right. Drum normal.

Right Ear.—Slight deafness to air conduction. Bone conduction slightly reduced (5 seconds). Rinne positive. Drum normal.

On testing the vestibular functions by means of the caloric test, it was found that both labyrinths gave normal reactions, the left, however, being somewhat more sensitive than the right. The nose and throat were healthy, and there were no signs of catarrhal changes in the tympana. There was no spontaneous nystagmus and no Romberg's sign.

A review of the case showed it to be one of bilateral labyrinth disease, very considerably more advanced; however, on the left side than the right. The Ménière paroxysms were, no doubt, occasioned by slight vascular or mechanical stimuli acting on a vestibular end-organ rendered morbidly sensitive by chronic inflammatory or degenerative changes.

When considering the expediency of an operation to destroy the labyrinth the important question arose as to which ear was the real source of the Ménière attacks. It was of course impossible, apart from operation, to determine this with certainty, but it was felt that there was sufficient presumptive evidence in favour of the left side to warrant one assuming that it was the side which gave origin to the Ménière attacks. Every other consideration favoured operation, namely:

1. The hearing on the left side was, for all practical purposes, lost, and therefore could not be made any worse by operation.
2. The vestibular function on the left side was still active.
3. There was no infective disease in the ear and therefore no special risk of sepsis in operating.
4. The symptoms were steadily becoming worse and the patient's life was becoming intolerable.

On April 13th, 1917, the operation was performed, with the assistance of my partner, Dr. J. E. McDougall, and the labyrinth on the left side completely destroyed.

Chloroform anaesthesia was used throughout to minimize bone haemorrhage, which would have added greatly to the difficulty of the operation. The pinna was turned forward and the antrum opened. The bone cavity was made with a broad bone superficially, so as to allow easy access to the deeper parts. The posterior meatal wall was partly removed, but not wholly, so that a slender bridge of bone remained as an outer wall to

the aditus. As soon as the antrum was opened a minute plug of gauze was inserted into the aditus to prevent blood or minute fragments of bone entering the tympanum during the subsequent stages of the operation. By proceeding in this way the tympanic cavity was not disturbed, and thus a considerable amount of trauma was avoided, and the risks of sepsis reduced. The prominence of the external semicircular canal was now distinctly seen in the depths of the wound and an opening made into it with a very narrow gouge. This is the most critical step of the operation and should be done slowly, with good

Drawing Illustrating the Operation: A, Arrow passing through aditus; B, Opening in external semicircular canal; C, Facial nerve; D, Bridge forming outer wall of aditus; E, Foramen ovale; F, Foramen rotundum.

illumination and a dry field. The facial nerve lies immediately below and in front (see drawing), so that the gouge must be very carefully applied in the first stroke of the mallet, as then the prominence of the canal is easily discernible and the landmarks undisturbed. The lumen of the canal is very small—about one-twenty-fifth of an inch—and lies very close to the surface. In order to make sure of striking it only very small scales of bone should be removed at a time and a strict adhesion to the original starting point observed.

When the canal was entered the opening was enlarged forwards and the conjoined ampullae of the external and superior canals opened up. A sufficient access to the vestibule was thus obtained to allow the introduction of the end of a piece of fine steel wire with which the membranous labyrinth was "pithed."

This method of destroying the labyrinth is, so far as I know, original, and was adopted as the one involving the simplest operative technique and the minimum of trauma. It presented less danger to the facial nerve than the usual superior vestibulotomy, and yet at the same time offered a very certain way of destroying the labyrinthine functions, for, even assuming that minute parts of the membranous labyrinth escaped direct destruction by the end of the wire, it is not to be supposed that such highly specialized sense organs could long survive the replacement of their normal endolymph medium by organizing blood clot.

The operation was concluded by stitching the pinna in position, and a small drainage tube was inserted into the lower end of the wound. The tube was removed in twenty-four hours, and healing occurred by first intention.

Subsequently to the operation the patient exhibited the typical clinical picture which results from traumatic destruction of the labyrinth. Vertigo was severe at first, and was accompanied by subjective sensations of rotation, photophobia, and defective accommodation. Nystagmus was marked to the right side, with a tendency to fall to the left side. He was able to sit up in bed in a week, and in fourteen days could walk about the room with support. The hearing on the left side was entirely absent and the tinnitus was greatly reduced. There was no reaction to the caloric test on the operated side, prolonged syringing with cold water producing no response. As time went on the vertigo and nystagmus steadily diminished, and the sense of balance became restored. Within three months of the operation he was able to do light work, but was obliged to avoid anything causing head congestion, such as stooping or lifting, which produced vertigo.

At the time of writing it is nearly a year and a half since the operation. The patient states that during the whole of this time he has never once vomited, nor had any return whatsoever of the old attacks of dizziness. His balance has so far improved that he can cycle with ease, while the tinnitus in the operated ear is much subdued. It is felt that sufficient time has now elapsed since the operation to justify the view that the patient is permanently cured of the Ménière attacks and that the operation has been entirely successful.

A CASE OF ACUTE YELLOW ATROPHY.

BY

A. C. ROXBURGH, M.B., B.Ch. CANTAB.,
TEMPORARY SURGEON R.N.

IN view of the rarity of this disease in men, apart from those cases in which it follows the intravenous injection of arsenical compounds or trinitrotoluene poisoning, the following case, for permission to publish which I am indebted to Fleet Surgeon P. H. M. Star, R.N., seems worth putting on record:

C. H. S., aged 20, a stoker from a destroyer, was admitted to the Royal Naval Hospital, South Queensferry, on July 26th, 1918. There was no history of syphilis or other serious disease since he joined the service. In the latter part of May, 1918, he began to have pain in the left hypochondrium, which lasted until he came under observation. On June 27th he reported sick, complaining of abdominal pain, of passing very little urine, and of having passed blood in the urine on June 23rd. The urine had a specific gravity of 1030, but contained no albumin and no blood. On June 29th he returned to duty.

On July 2nd he reported sick again and was found to have influenza (which was then prevalent) and was noticed to be jaundiced. The influenza soon subsided but the jaundice remained. On July 9th he had pain in the right hypochondrium with dullness for two inches from the right costal margin towards the umbilicus, and tenderness on pressure over the region of the gall bladder. He vomited on this day, and on July 12th. He had been on full diet, but was now put on a milk diet with sodium bicarbonate, and given a mixture containing sodium salicylate and sodium bicarbonate.

On July 18th the jaundice appeared to be fading and he was allowed up, but on July 20th he was not so well, vomiting again and being more depressed. The sodium salicylate was discontinued and he was given grey powder and an acid mixture containing tincture of nuxvomica.

On July 25th he had an epileptiform seizure and was semi-conscious for some time afterwards. He became drowsy and was sometimes delirious. The jaundice was deepening, he had incontinence of urine, and the bowels were not open. The abdomen was flaccid and not tender.

When admitted to hospital, in the afternoon of July 26th, his temperature was 98.2°, pulse 92, and respirations 17. He was deeply jaundiced, semi-conscious, and though he could be roused he could not be made to speak. The first sound of the heart was short and sharp, but otherwise nothing abnormal was found in heart or lungs. The liver dullness in the nipple line extended only from the sixth to the seventh rib. The abdomen was otherwise natural. There were a few small petechial haemorrhages on the arms.

The urine was acid; sp. gr. 1026. It contained a trace of albumin, a large amount of bile pigment, but no blood or sugar. The deposit contained many cellular and granular tube casts and some sheaves of needle-like crystals which resembled those of tyrosin. On evaporation unmistakable leucine cones were seen. At 8 p.m. he was still unconscious and could not be roused. The pupils were dilated, equal, and inactive; there was an external squint. He lay on his back making chewing and biting movements with his jaws and uttering at intervals a cry like the "meningeal cry" of children. He resisted attempts to feed him. At 8.30 p.m. lumbar puncture was performed. The fluid was under increased pressure and a test tube full was removed before the normal rate of flow was established. The fluid was clear and straw-coloured; no cells could be found in it. He was much quieter for an hour after the lumbar puncture, breathing regularly and quietly and appearing to be asleep. During the night, however, at intervals, he made gasping motions with the jaws and uttered the same peculiar cry. The pulse-rate varied from 96 to 136, but the volume and tension were quite good.

At noon on July 27th, when the nasal tube was passed, he vomited a few ounces of fluid, nearly black, and containing some red blood. He had been given *mistura alba* at 11 a.m., and at 1 p.m. the bowels were opened for the first time since admission. The stool was small, formed and brownish-grey. There was no smell suggestive of phosphorus poisoning. At 5 p.m. he was still unconscious, sweating profusely, and a good deal of mucus could be heard in the air passages. The temperature and pulse and respiration rates began to rise about this time. At 8 p.m., when the nasal tube was passed, he vomited 2 oz. of brown treacly fluid with a few streaks of blood. Temperature and pulse and respiration rates continued to rise during the night, reaching 103°, 136, and 44 respectively at 5 a.m. on July 28th.

He died at 6.30 a.m. on July 28th, after an illness lasting about nine weeks in all, and about twenty-four days from the onset of jaundice. Treatment had consisted chiefly of rectal salines and nasal feeding.

He passed only about eighteen ounces of urine during the forty hours he was in the hospital. He never vomited except when the nasal tube was passed. It is regretted that a Wassermann reaction was not done nor a blood culture made.

Post-mortem Examination.

The skin was jaundiced and showed hypostatic post-mortem staining.

Both lungs were congested and oedematous. The right lung