SOME REMARKS ON THE FENESTRATION OPERATION AND WHAT LEADS TO THE RESULTS

J. VENKER

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Page

INTRODUCTION.

The operative treatment of otosclerosis is a subject in which I was keenly interested already before the outbreak of the second world war, but unfortunately during the war I was unable to follow developments in this field owing to the absence of any contact with the outside world.

In April 1946 I managed to get to London, where I was enabled to practise the technique of otosclerosis operation under the guidance of MONKHOUSE and MACKENZIE in the FERENS Institute of the Middlesex Hospital.

The results achieved in Great Britain and the U.S.A. encouraged me to practise this operation also in the Netherlands. After having performed a number of these operations I was struck by several vasomotor symptoms playing a part in otosclerosis, which symptoms are described in this thesis.

That the cause of otosclerosis lies in vascular defects was already suspected in 1908 by SOHIER BRYANT. After him, O. MAYER, SIEBENMANN, WITTMAACK, DELIE, KATZ, RAINISCH, SPARER and especially GRAY pointed out the importance of this.

According to GRAY otosclerosis is due to a permanent defect in the vasomotor reflex system of the whole organ of hearing.

In this thesis the operation will be described and the vasomotor symptoms I observed will be dealt with, thus contributing, as I hope, towards the aetiology of otosclerosis.

SYNOPSIS OF LITERATURE.

Since A. VALSALVA first described in 1704 the stapes ankylosis as the cause of otosclerosis, a large number of treatments have been applied both locally and generally, but until recently none of these has really achieved any result of importance. Surgical operations were the most successful, although even then in the majority of cases the improvement in hearing was of very short duration.

Already in 1876 KESSEL tried to improve hearing in cases of otosclerosis by removing the malleus and the incus, but without any appreciable results. Yet, these first attempts were the beginning of the surgical operations for otosclerosis applied so frequently and in various ways in later years.

After KESSEL, in 1885 GELLÉ carried out tenotomy of the musculus stapedius, and LUCAE resected the malleus in a large number of cases and also in some the incus. In 1888 BOUCHERON separated the stapes and incus and tried to loosen the stapes by moving it to and fro. MIOT and MAURE likewise attempted to improve hearing in this way.

BLAKE and JACK resected the stapes and had some success at the beginning.

In 1897 PASSOW made an opening in the promontory. FLODERUS did the same but covered this fistula with a THIERSCH graft; he also contemplated applying a fistula in the horizontal canal but decided not to do so because he thought it would then be too far removed from the cochlea.

In 1910, in his manual, URBANTSCHITSCH recommended resection of the malleus and if necessary also the incus. He says that such an operation will lead to improved hearing also in the other ear.

Gradually these surgical methods disappeared because no permanent success of any value was ever achieved. Other means were sought and eventually one arrived in the region of the semicircular canals.

In 1910 BÁRÁNY suggested that a fistula should be applied in the posterior semicircular canal without opening the drum cavity.

JENKINS, in 1913, was of the opinion that otosclerosis was accompanied by elevated intralabyrinthine pressure and applied a fistula

in the horizontal semicircular canal. In one case he covered the fistula with a THIERSCH graft and in another case with a flap of the auditory canal; in both cases there was only a temporary improvement in hearing, of short duration.

BÁRÁNY and JENKINS were followed by HOLMGREN, who has done much meritable work in the field of surgical operations for otosclerosis and in the course of years has been untiring in his endeavour to improve the technique in various ways.

In 1917 HOLMGREN applied a fistula in the posterior semicircular canal, which he reached by elevating the dura and exposing the eminentia arcuata; the fistula was then covered with the dura. In 1920 he suggested using for his operation the Zeiss-Gullstrand glasses or the Zeiss binocular microscope. He also applied a fistula to the promontory, but with only short-lived success.

In 1922 HOLMGREN applied a fistula in the horizontal canal and covered it with a mucoperiostal flap of the canal itself; the improvement of hearing lasted only a few weeks.

In 1924 BÁRÁNY performed an operation in two stages. In the first stage he carried out antrotomy, applied the fistula and filled up the cavity with fat. In the second stage the fat was removed and the fistula closed with gutta percha. It was a moderately successful operation.

NEXT to HOLMGREN, one of the pioneers in otosclerotic surgery was SOURDILLE, and, except for some modifications, it is his method that is mainly applied up to the present day.

SOURDILLE began operating in 1924, at first in three stages and at times in four or five stages. Later on he changed his technique and operated in two stages. In the first stage he opened the antrum, removed the bony posterior wall of the auditory canal, resected the head of the malleus and covered the horizontal canal with a skin flap of the auditory canal. In the second operation about two months later, after complete epithelization of the cavity he carefully loosened the skin periostic flap on the horizontal semicircular canal, chiselled the canal open and then covered the fistula again with the loosened flap. In cases where the fistula closed up he opened it again in a third, fourth or, if necessary, fifth operation. SOURDILLE thought in this way to exhaust the regenerative power of the bone. In more recent operations he also resected the incus.

SOURDILLE calls his operation "tympano labyrinthopexy", because he considers it of importance that the fistula should be kept in contact with the tympanic membrane by means of the plastic flap.

MOULONGUET is of opinion that the SOURDILLE method is too difficult and suggests that the fistula be covered with a skin graft from the patient's upper eyelid.

In 1935 HOLMGREN tried covering the fistula with subcutaneous fat or gold-leaf, but the results attained with this method were not permanent either. Then by another method he filled the operational cavity with paraffin wax, closed the mastoid primarily and after some time reopened it; the cavity was then covered with a thin membrane from which a graft was taken to cover the fistula subsequently made. This method, too, proved not to be a permanent success.

Having regard to the fact that fistulas formed in the semicircular canals in cases of cholesteatoma tend to keep open so long, HOLMGREN thought that the thin membrane covered with stratified squamous epithelium might play an important role. He therefore performed first a conservative radical operation and covered the horizontal semicircular canal with a skin flap from the auditory canal, and in a second operation he folded the flap back, opened the canal and covered the fistula with the membrane.

HOLMGREN has experimented a great deal with monkeys, treating the applied fistulas in various ways (fat, gold-leaf, transplanted epithelium, radium). After some time the mastoids were examined by NAGER and later also in cooperation with ENGSTRÖM. They found that the bone regeneration in the fistula always proceeded from the periostal bone covering. Stratified squamous epithelial covering and irradiation with radium gave protracted regeneration. The application of a high-speed burr likewise retarded bone regeneration. Bone dust considerably promoted bone regeneration. They also found that large fistulas caused the canal to close up entirely, but that the endolymphatic system remained intact. In this connection they draw attention to the danger of applying a too broad fistula in the vicinity of the ampulla and thereby causing damage to the sensory epithelium through the closing up process.

FOWLER found experimentally that cubic epithelium checked

regeneration, whereas infection in the wound cavity had a stimulating effect.

After HOLMGREN and SOURDILLE, LEMPERT had success with his operation in one stage. His method is really an improved technique on that of SOURDILLE. LEMPERT's idea is to cover the fistula with SHRAPNELL's thin membrane. Most surgeons following his method of operating, however, are of opinion that this membrane is too far removed from the semicircular canal to cover the fistula with it. At first LEMPERT left the incus intact and resected only the head of the malleus, but later on he started removing the incus too. He applies the fistula with a high-speed dental finishing burr, being of the opinion that the bone dust counteracts regeneration of the bone on account of it clogging up the Haversian canals. He first used a gold burr because he thought the particles of gold would likewise penetrate the bone and check regeneration. CANFIELD found experimentally that bone regeneration was retarded after drilling but not after chiselling. The GUGGENHEIMS found bone regeneration when a burr was used but not when electrocoagulation was simultaneously applied. LEMPERT also removes the endosteum from the fistula opening, because bone regeneration may take place from there; FOWLER discovered the same possibility experimentally. CAMPBELL, on the other hand, leaves the endosteum, because his idea is that the trauma excites the endosteum and thus causes the fistula to close.

According to LEMPERT the extremities of the fistula should be rounded and not pointed, because this, too, may cause the fistula to close quickly. He has thought of several ways of keeping the fistula open, even by inserting a metal obturator, but he very soon dropped that idea. Then in 1945 he placed first an ivory and later on a cartilaginous stopple in the fistula, not only to stop it closing up but also to prevent post-operative serous labyrinthitis, for he believes that this labyrinthitis is due to an infection arising from the plastic graft, and closing of the fistula with this stopple should check the infection. LEMPERT is of opinion that in course of time a fibrous connection is formed between the bone of the fistula and the cartilage, so that the stopple remains movable in the fistula. Meanwhile he has departed from this method and in view of the labyrinthitis just referred to he now leaves the endosteum intact. LEMPERT operates entirely through the auditory canal, for this, he says, has the advan-

tages of better observation during the operation, less risk of post-operative infection and better cosmetic result, whilst the plastic flap could not be obtained endaurally. Many are operating in this way, whereas others, among whom CAMPBELL, KOPETZKY, GOODYEAR, SIMSON HALL, KEND and, until recently, also HOLMGREN operated behind the ear.

LEMPERT used to apply the fistula in the horizontal semicircular canal but now he, and also SHAMBAUGH and others, apply it closer to the ampulla, because there the fistula can be made wider (8 mm—1.5 mm). KOPETZKY, on the other hand, believes the best results are obtained with a small fistula (1—3 mm).

SHAMBAUGH holds that closure can be prevented by removing the periosteal layer deep down to the enchondral bone over a large area around the fistula, because regeneration proceeds mainly from the periosteal layer. Another advantage, according to him, lies in the continuous, thus not intermittent, irrigation with physiological salt solution, removing all bone splinters and bone dust. As opposed to HOLMGREN, he believes that regeneration may come also from bone dust. SHAMBAUGH, too, applies the fistula in the region of the ampulla, but more to the front and below it, as close as possible to the tympanic membrane. Further, he removes the endosteum from the fistula but warns against damage beyond the edge of the fistula, thus in the canal, because this causes excitation and may lead to bone regeneration.

LINDSAY applied fistulas in the semicircular canals of monkeys. When the fistula closed up the bone regeneration took place mainly from the periosteal layer and only to a small extent from the endosteal layer. The fistulas were covered in different ways but the best results were obtained with the thin skin of the auditory canal. To keep the fistula open it is important that the plastic flap should grow together with the endolymphatic canal. Bone splinters and endosteum must be carefully removed from the fistula opening.

The GUGGENHEIMS found that bone regeneration was considerably checked when the fistula was covered with a piece of copper. Through catalytic oxidation this metal binds vitamin C, which is essential for regeneration.

KEND puts his patients on a special diet for two weeks before and four weeks after the operation. His idea is that bone regeneration

comes mainly from the periosteal layer. Experiments with rats showed that when rachitis is induced changes take place only in the periosteal layer of the labyrinth. In the case of rachitis the periosteum functions badly or ceases to function at all. The diet on which KEND puts his patients corresponds approximately to what the rats were fed with when rachitis was experimentally developed. It contains no vitamin D, plenty of calcium, little phosphorus and some bovine ovarium extract.

CAWTHORNE has recently pointed out that it is of importance to apply the fistula on an elevation, for a better propagation of the sound stimuli to the fistula. He applies it at the ampulla, making it long and narrow above the horizontal canal and wider above the ampulla. Contrary to CAWTHORNE, KEND applies a saddle-shaped fistula in the canal.

According to LEMPERT damage to the endolymphatic system usually reduces the power of hearing. LOWY, when applying fistulas in the semicircular canals of pigeons, did not find that slight damage to the endolymphatic system affected hearing, but resection of a part of the endolymphatic canal resulted in considerable loss of hearing.

CAWTHORNE treated 52 cases of Ménière's disease, by opening the horizontal canal and resecting the endolymph canal. Only in two cases did the hearing become worse, in 11 cases it was improved and in all the other cases it remained the same as before the operation.

Attention should also be drawn to POPPER's technique. He makes an incision in front of the ear and reaches the front side of the bony auditory canal, which he removes together with the covering auditory canal skin. He draws the jaw joint forward and approaches the tympanic membrane perpendicularly. He makes a skin tympanic membrane flap from the posterior auditory canal wall, resects the bony posterior wall only in the posterior upper corner and thus reaches the horizontal canal. The advantages of this method are: short duration of the operation, minimum operative trauma, good observation and quick postoperative healing.

Before ending this review I would point out two other methods of performing the operation based on entirely different principles.

In the first place, ROLLIN operates by a method based on WITTMAACK's theory of otosclerosis. His opinion is that otosclerosis is due to congestion or a return flow of the blood in the veins of the

organ of hearing. He therefore applies a ligature to the veins running through the roof of the tegmen tympany to the vena petrosa superficialis.

Finally HUGHSON, who holds the view that the membrane of the round window has a damping effect upon the sound waves, tries to minimize this effect by plastic grafting of tissue in the recess of the round window. He operates not only for otosclerosis but also in cases of perception deafness.

OWN OPERATIONAL CASES.

1. *Instrumentarium.*

A good and practical instrumentarium is of great importance for the operation.

The instruments I use are the following:

1. A diathermic apparatus.
2. A good forehead lamp.
3. Magnifying glasses, power 3 ×.
4. A suction-flushing installation. For this I had a small apparatus made to my own design (fig. 2) which maintains a constant suction and can flush as required. It consists of a sort of open hand grip into which two lengths of rubber tubing are fitted, the upper one for suction and the lower one for flushing, the latter when not required being closed by a spring clamp, which is opened by a light pressure with the forefinger. The hand grip is 11 × 2.25 cm. The suction tube is 15 cm long and 3.5 cm in diameter, bent at the end. Underneath this is the flushing tube 2.5 mm in diameter, slightly shorter than the suction tube, so that it does not extend right up to the end of the suction tube. The suction tube is connected to an electric suction-installation. The flushing liquid is a physiological salt solution, heated to a temperature of 37° C. To keep the solution up to this temperature I use a flask of 2 litres capacity hung up in a large cylinder filled with a thick layer of cotton wool; a large thermos flask also answers the purpose. The advantages of such an apparatus are that it gives a beautifully clear operative field, there is little risk of damaging the plastic flap and flushing and sucking can be done with one hand, leaving the other hand free for burring the fistula (fig. 4).
5. A drilling stand with straight grip as used in dentistry, with various sizes of burrs and a No. 5 polishing burr, highly important when opening the fistula; the finer the burr, the finer the bone dust and the less chance of particles of bone being left behind to cause regeneration of the bone.
6. A set of instruments as used in the radical operation on the mastoid.

7. Special forceps for severing the head of the malleus (fig. 3). I have had an instrument made larger than that originally recommended by LEMPERT. These forceps are strong and comfortably held in the hand; they have never yet been found to be too thick in a narrow cavity because the fairly long points completely neutralise the rather heavy part where the blades are pivoted together.

8. As raspatory for skeletonising the osseous posterior wall of the auditory canal I use an instrument employed by dentists (fig. 3).

9. For resecting the osseous posterior wall I first used bone forceps with a thin and a thick leg, but I now more usually employ my

10. "swallow tail" (fig. 3), a hollow chisel, 5 mm broad, made to my own design, which is notched in the middle, the only place where it is sharp. The advantage of such a chisel is that it does not slip through when the posterior wall of the auditory canal is resected and the blunt corners do not damage the plastic flap. For the same purpose I sometimes use

11. a 4 mm ordinary flat chisel with the corners rounded off, likewise to avoid damage to the flap (fig. 3).

12. Some needle-like straight and curved instruments and a very small curved and sharp curette for removing endosteum residue in the fistula opening.

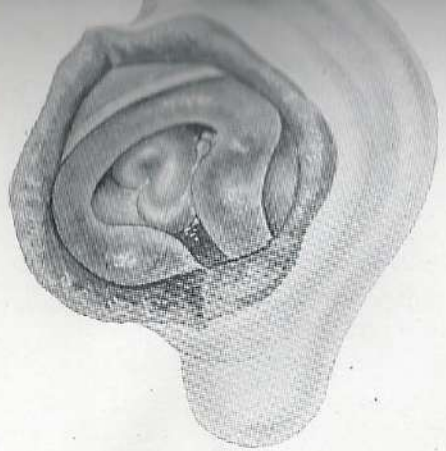
13. Paraffin wax with a melting point of 40° C., in which gauze 1 cm wide is saturated for covering the plastic flap. It is advisable to use a heated pincette and scissors when cutting the saturated gauze.

14. A piece of sterilised sponge for maintaining an elastic pressure on the flap in the wound cavity during the first few days following the operation.

With this instrumentarium and particularly by using the suction-flushing apparatus described above, several hands are saved and the operation can easily be performed without an assistant surgeon, the only help required being a capable theatre nurse. The operation takes, on an average, two hours.

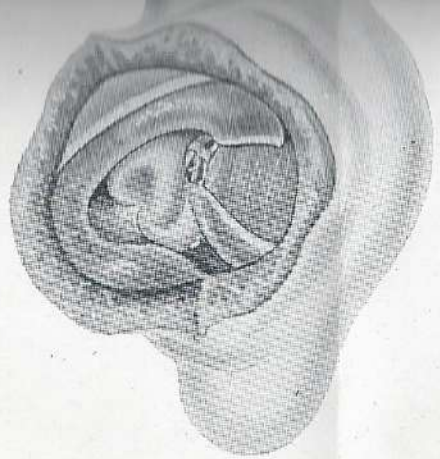
2. *Indications for operation.*

As regards what constitutes an indication for operation the literature is rather confusing and nobody has really given a sharply defined guide. The difficulty lies in the secondary nervous degene-



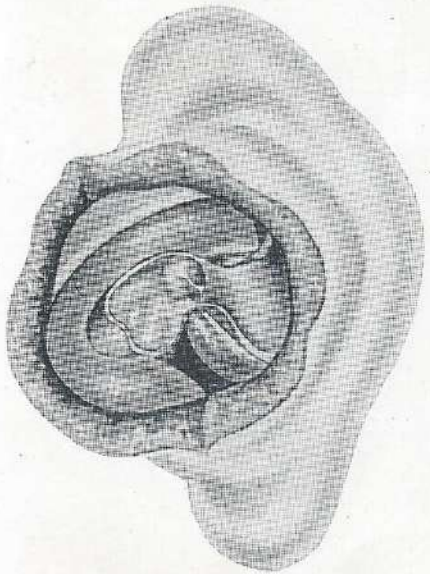
A

Antrotomy.



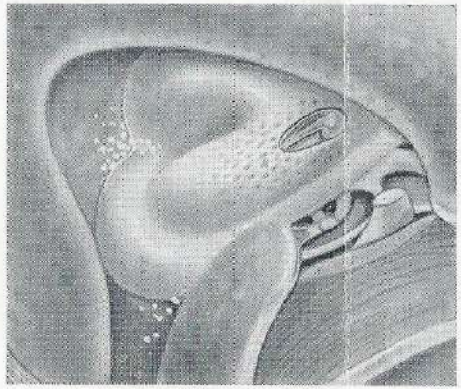
B

The bony meatal wall resected, the incus removed and the head of the malleus amputated.



C

Skin flap laid over the fistula.



D

Fistula applied, endosteum removed and enchondreal bone exposed around the fistula.

These drawings have partly been taken from publications of LEMPERT and SHAMBAUGH.

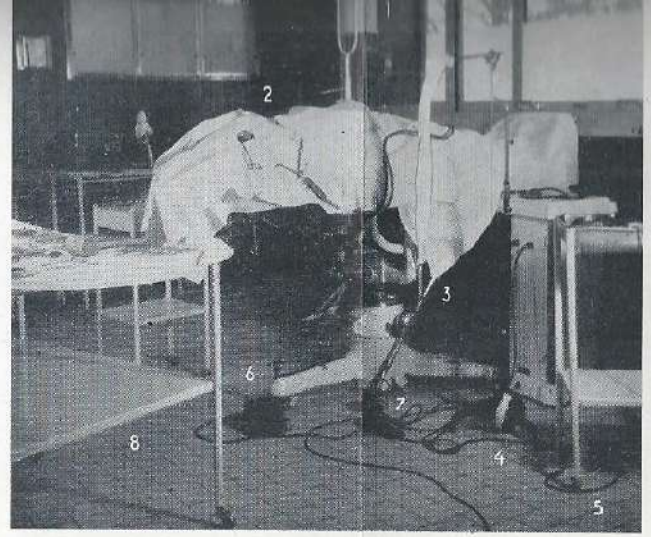


Fig. 1.

1 Stand with cylinder of physiol. salt solution.
2 Patient on the operating table. Suction-flushing tube hooked up.
3 Drilling machine.
4 Diathermic apparatus.
5 Transformer for head lamp.
6 Pedal for drilling mach.
7 Pedal for diathermy.
8 Instrument table.

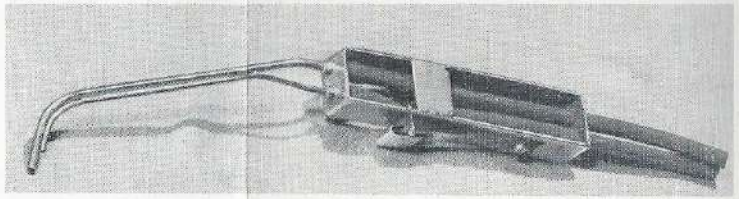


Fig. 2. Suction-flushing apparatus.

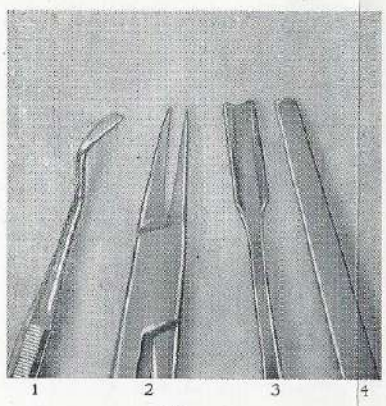


Fig. 3.

1 Raspatory for auditory canal.
2 Forceps for amputation of head of malleus.
3 Swallow-tail chisel.
4 Flat chisel (rounded corners).

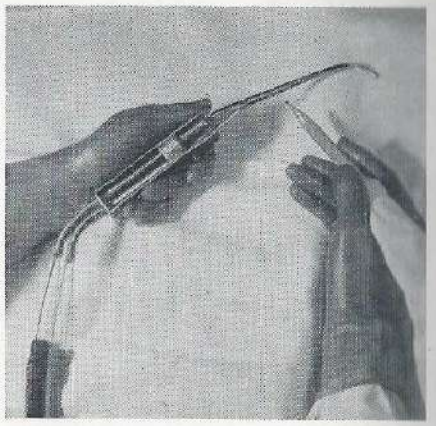


Fig. 4.

Left hand: Suction-flushing tube.
Right hand: Drill.

ration which in course of time occurs in most cases of otosclerosis and is taken by many specialists as a reason for not operating. LEMPERT, SIMSON HALL and others hold a different point of view because they have often attained excellent results even under these supposedly unfavourable conditions. I shall revert to the indication for operation later on in this thesis.

In general I consider the following rules:

Anamnesis.

1. Progressive deafness in both ears, set in after 15 years of age.
2. Deafness in the family.
3. Aggravation during pregnancy. The last two conditions, however, are not essential.

Pathological examination.

1. Internal examination shows no serious defects.
2. Normal tympanic membranes (or possibly atrophy).
3. Normal functioning tubae and no infections in nose and throat.
4. X-ray examination shows normal pneumatisation of the mastoid.

Examination of the ears.

1. Whisper test of both ears: less than ¼—½ meter.
2. With tuning fork: raising of the lower tone limit to 128—256 vibrations.
3. Normal bone conduction with a tuning fork of 512 vibrations.
4. Rinne negative.
5. Audiometric examination:

a. air conduction — as a rule bilateral deafness of about 40 dBl loss or more in the frequencies of 128—4096 vibrations;

b. bone conduction — practically normal up to 512 vibrations, and above that a reduction to 40 dBl for 1024 and 2048 vibrations is permissible.

The ear with the hardest hearing is chosen for the operation in view of the risk that is always involved in the operation; SHAMBAUGH reports 1.2 % of cases with total deafness after this operation.

3. *Preparations for the operation.*

The patients are admitted to hospital a few days before the operation. The ears are thoroughly examined and tested once more, and as pre-operative audiogram the mean is taken of three audiograms recently recorded, which, however, do not as a rule differ much.

In order to shorten the clotting time and bleeding time 300—500 cc blood is drawn from the vena a few days before operating, whilst two hours before the operation 20 cc Sangostop is administered intramuscularly. This reduced clotting time and bleeding time has been proved by the physician, Dr. WENSINCK.

All patients are subjected to a thorough internal examination.

The day preceding the operation alcohol of 70 % proof is dripped into the auditory canal, which is then loosely plugged with a sterile gauze tampon.

4. *Anaesthetics.*

Here a choice has to be made between a local anaesthesia and general narcosis. Local anaesthesia has the drawback that the patient becomes restless, particularly when the horizontal canal is opened, just at the time when the head should be motionless. The advantage lies in the little bleeding. General narcosis is accompanied with more profuse bleeding, and especially laughing gas is not to be recommended. The use of ether has the disadvantage that diathermy cannot be applied to arrest bleeding. If assistance is available then an Evipan, a Eunarcon or Pantothal anaesthesia is to be recommended, because diathermy can then be applied during the whole operation.

So far I have always used ether as an anaesthetic and had to manage without the advantage of diathermy. However, I inject a few cc of a 1 : 100,000 adrenaline solution into the soft tissue and the upper posterior wall of the auditory canal. I have not always found this method quite satisfactory and therefore I now begin the operation with Evipan narcosis or local anaesthesia and apply ether as soon as incisions have been made in the soft tissues and the bleeding vessels have coagulated. Before the operation is begun the patients are injected with 10 mg Morphine and ¼ mg Scopolamine.

LEMPERT, SHAMBAUGH and others operate under local anaesthesia, whilst CAMPBELL, SIMSON HALL and others prefer general narcosis.

5. *Operational technique.*

If the fenestration operation is to be performed accurately — and I consider this absolutely essential, because the success of the whole operation may be affected by minor matters — the technique is decidedly difficult. It is an operation in no respect comparable to any other in otology, and the radical operation in the mastoid, for instance, is a coarse, inelegant and bloody process.

To my mind it is irresponsible to attempt this operation without having previously acquired most intensive practice in all details of the technique, and moreover a good knowledge of the post-operative progress is desired. Faulty technique and injudicious after-treatment involve great risks of endangering the patient's hearing.

The operation may be considered as being divided into two parts:

A. Preparation of the plastic flap.

B. Application of the fistula in the horizontal semicircular canal.

A. Contrary to LEMPERT, I have performed all my operations behind the ear and by this method had a clear view of the operative field, obtained a good entrance to the operative cavity by drawing the ear well forward, experienced no complications in the after-treatment and, finally, achieved a good cosmetic result. I will not enter here into a discussion as to which is the better, the endaural approach or my method, but since the approach behind the ear appeals to me more I have followed that.

The order of the various manipulations in the following technique is of importance, those involving the most profuse bleeding being carried out as far as possible first.

1. After infiltration of the soft tissues behind the ear and in the superoposterior wall of the auditory canal with an adrenaline solution of 1 : 100,000, the incision is made parallel to the junction of the auricle, and the planum mastoideum is exposed.

2. An incision is then made perpendicular to the axis of the auditory canal in the posterior auditory canal skin, exactly medially from the auricle cartilage. This is the incision which later on connects the two incisions in the auditory canal, whereby the plastic flap is formed.

3. A piece of cartilage with perichondrium is then taken from any part of the auricle to serve later on as a stopple in the fistula; until required it is preserved in a physiological salt solution. (This stopple was only used in the first seven operations.)

4. The antrum is then opened with chisel and hammer and the incus resected. No more mastoid cells are opened than is necessary to get a clear field and give good access to the operative cavity.

5. The cells along the posterior auditory canal wall are resected, leaving only a thin plate of bone.

6. The osseous posterior wall of the auditory canal is resected, sparing the soft tissues of the canal. This is a technically troublesome part of the operation. The skin has to be lifted very carefully from the osseous auditory canal with great patience a millimeter at a time, after which it is removed piece by piece mainly with the swallow-tail and sometimes also with the help of small bone forceps. It must be borne in mind that the skin of the auditory canal is extremely thin in some places (0.01—0.03 mm) and is easily torn if the pressure is at all excessive; it very easily tears in the area of the inferoposterior wall, where there is a small bony ridge often making it most difficult to lift the skin.

If the plastic flap should be in the least damaged there is the risk that later on it will not cover the fistula, or it may become necrotic owing to lack of nutrition, with the danger of an osseous closing of the fistula or closure of the canal with tough fibrous tissue (Lempert).

While carrying out the elevation very carefully I use the suction-flushing apparatus already described, so as to give better observation and because by swabbing with gauze the plastic flap is apt to be damaged.

7. The bone around the head of the malleus is widely resected and the ligaments attached to it are severed with a knife. The head is then amputated from the malleus with the special forceps.

8. Two incisions are then made in the auditory canal, the first from the tympanic membrane laterally along the superoanterior wall and the second one from the tympanic membrane laterally along the inferoposterior wall of the auditory canal. This leaves a plastic flap in the form of a segment with its narrowest part connected with Shrapnell's membrane.

9. If the cartilaginous stopple is to be used then the cartilage

preserved in physiological salt solution is scraped clean of its perichondrium and cut approximately to size ready for use.

B. The application of the fistula cannot be performed without using magnifying glasses or Zeiss–Gullstrand glasses. Moreover it is essential to use the suction-flushing installation in this stage of the operation, as otherwise the bone dust cannot be thoroughly removed and no clear view is obtained of the operative field.

For drilling one can use right-hand and left-hand rotating burrs. Should the burr slip through then if a right-handed one is used while working in the left mastoid it will not damage the n. facialis, and vice versa, but this should not happen if the burr is handled under not too heavy pressure. Drilling should be done under light pressure while repeatedly retracting the burr slightly, taking care not to touch the plastic flap. If too heavy pressure is brought to bear on the burr there is, moreover, the risk of it suddenly shooting through the thinned wall into the semicircular canal and, of course, causing irreparable damage to the endolymphatic system. In connection with the possibility of damaging the endolymphatic system, I would point out that when sucking away the bone dust it is dangerous to hold the suction tube immediately against the fistula, because there is a risk of the capillary endolymph canal being sucked up into the tube. I therefore hold the suction tube in a position roughly central with respect to the fistula, which is then just nicely flushed clean of bone dust with the flushing liquid (for the position of the flushing tube, see fig. 2).

In order to avoid damage to the endolymph canal while drilling I apply the following method: when removing the bone dust from the fistula I always noticed that when the flushing liquid was drawn off through the suction tube the endolymph canal subsided to the bottom of the semicircular canal; on the other hand, as long as the semicircular canal was filled with liquid the endolymph canal was rather close to the edge of the fistula, floating as it were in the liquid.

Consequently before starting to drill I always see to it that the fistula is not entirely filled with liquid (see fig. 5), so that the endolymph canal may not be so readily damaged.

Bleeding during the operation is arrested as far as possible by means of diathermic coagulation as regards the soft tissue. Diffuse bleeding from the small bone vessels is arrested by means of hydrogen

peroxide, adrenaline or bone wax. Especially just before opening the fistula it must be ensured that there is absolutely no more bleeding in the operative cavity.

The fistula is applied at the level of the ampulla. Rough surfaces around the site of the fistula are removed with the burr. Then with the polishing burr No. 5 the periosteal bony layer is removed on and around where the fistula is to be applied (SHAMBAUGH). The enchondral bone is then exposed, as may be clearly seen from the speckled structure. The bone dust formed is regularly flushed out and drawn off with physiological salt solution heated to 37° Celsius.

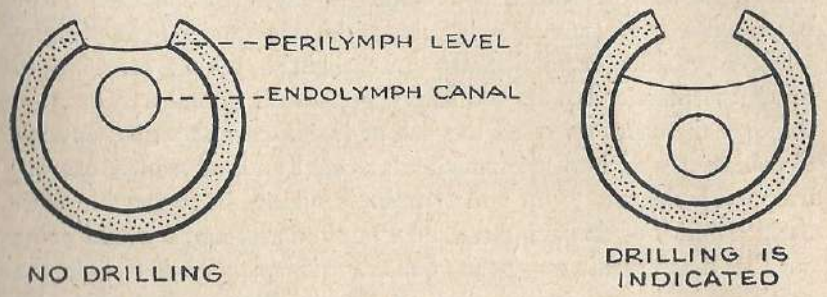


Fig. 5.

As soon as a bluish tinge begins to show through the lumen of the canal and the ampulla, the operative cavity is again examined to see whether there is still any slight bleeding, and if so it has to be arrested. The thin bony layer still covering the lumen of the canal is then pulverised with the burr. Some perilymph then runs out.

In most cases the greater part of the endosteum will already have been removed with the burr, but if there should still be some small pieces left inside the fistula opening they must be removed with needle-shaped instruments and if necessary with a small sharp curette.

The fistula opening is now enlarged with the burr as desired, while taking care, in the manner indicated above, not to damage the endolymphatic system. We can see the endolymphatic system running through the fistula like a somewhat brownish capillary, which, however, does not show up so well against the darker posterior wall near the ampulla. The fistula, situated above the ampulla and extending over the horizontal canal, is about 5 mm long and 1½—2 mm wide.

Above the ampulla the fistula is made slightly wider than above the semicircular canal. If necessary a little bone is removed on both sides of the fistula so that it comes to lie on an elevation.

The cartilaginous stopple is now cut to the right size and made to fit in the fistula. It must protrude a little above and below the level of the canal wall, as indicated by LEMPERT. It must not protrude too far into the canal, because otherwise it might damage the endolymphatic system. The fistula is then covered with the plastic flap, over which a strip of gauze (1 cm wide) saturated in paraffin wax is laid. On top of this a small sponge is inserted in the cavity to bring an elastic pressure to bear upon the plastic flap.

The wound behind the ear is closed with clips and the ear bandaged with a sterilized dressing.

6. *Post-operative treatment.*

During the first five days following the operation the patients get 20,000 units of Penicillin every three hours. During the first 48 hours the patients lie on the operated ear (SHAMBAUGH).

On the second day the dressing is renewed.

On the sixth day the sponge is removed from the operative cavity, and on the eigth day the wax gauze is removed.

The cavity is further treated with Orgasepton powder and sometimes with a few drops of Penicillin. In some cases it is necessary to remove granulations with a small sharp curette.

After-treatment must be performed with great care and it is important that the cavity dries quickly. If a cavity continues to discharge pus for a long time there is a danger of the fistula closing and of purulent labyrinthitis (LEMPERT, SIMSON HALL). MOORHEAD reports for 124 operations 10 % with a chronic light discharge.

The patient leaves hospital between the tenth and fourteenth post-operative days and is then further treated in the out-patients department.

7. *Case histories.*

In the following case histories, for the sake of brevity, only a concise anamnesis is given, with a description of the state of the tympanic membrane and the hearing tests.

None of the patients showed any great defects in the nose, nasal cavities, nasopharynx or throat.

Internal examination, urine and blood tests revealed no abnormalities worth mentioning here; I will revert to this farther on under "General Symptoms".

Where the operations are dealt with mention will only be made of what might have been of importance in the further progress of the case. So far I have performed twenty-five operations, but only the first twelve cases will be discussed in detail; most of the others are recent cases and cannot yet very well be judged.

In the first eight cases the fistula was closed with a cartilaginous stopple. In the last four cases this was not used, the plastic flap being applied to the fistula directly.

In all operations a good pneumatization of the mastoid was found, so that there was in every case ample space between the dura of the middle fossa and the horizontal semicircular canal, which facilitated the operation.

Case 1. A man of 41 years (fig. 6).

Progressive hardness of hearing since the age of 20. Left ear worse than the right. No deafness in the family.

Much trouble from tinnitus, particularly in the left ear. The patient used a hearing aid but even this did not enable him to follow a conversation in company.

State of the tympanic membrane: wide auditory canal, no cerumen, both membranes transparently atrophic, not drawn in.

Hearing test:

R. loud speech ad concham.

L. very loud speech ad concham.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: slightly diminished both sides.

Lowest limit: R. 256, L. 512.

Operation L. ear.

Considerable diffuse bleeding, very troublesome at times, causing the operation to take rather long, 2¾ hours.

Result 4 months after operation: very little improvement in hearing. Fistula reaction +.

Case 2. A woman 32 years old (fig. 7).

Progressively hard of hearing since 21st year. R. ear worse than left. Much trouble from tinnitus in R. ear. Patient used no hearing aid and could no longer follow conversation in company. There is a sister hard of hearing. Hearing became worse while carrying first and only child.

Tympanic membrane: wide auditory canal, no cerumen, both membranes atrophic, inferoposterior quadrant transparent pink.

Hearing test:

R. whispering ad concham.

L. whispering heard at 25 cm.

Weber: no laterilization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 128.

Operation R. ear.

No operative complications.

Result 4 months after operation: very good, whispering heard at 5 metres. Fistula reaction +.

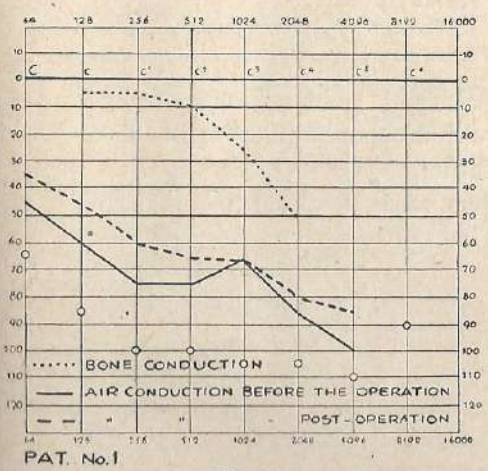


Fig. 6.

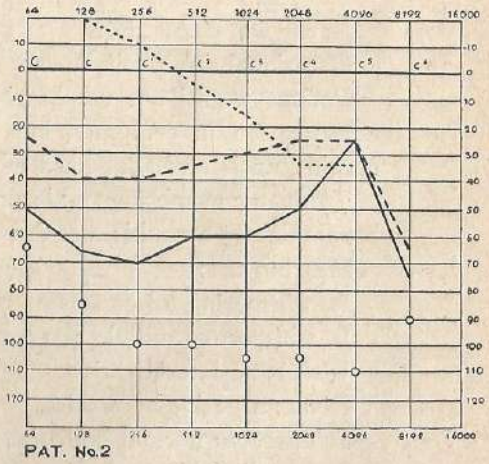


Fig. 7.

Case 3. Woman 23 years old (fig. 8).

Strongly progressive hardness of hearing during several years. L. ear worse than the right.

Occasional tinnitus in L. ear.

Patient used no hearing aid and could no longer follow conversation in company.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes highly transparent and atrophic, not drawn in.

Hearing test:

R. whispering heard at 25 cm.

L. whispering ad concham.

Weber: lat. to left.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 64, L. 64.

Operation L. ear.

No operative complications. In the evening following operation profuse bleeding in wound cavity.

Result 3 months after operation: moderate improvement in hearing, whispering heard at 50 cm. Fistula reaction —.

Case 4. Woman of 40 years (fig. 9).

Progressively hard of hearing since 28th year.

R. ear worse than left.

Occasional tinnitus in R. ear.

Patient would not use hearing aid and could no longer follow conversation in company, but could telephone quite well. Two cousins, uncle and aunt hard of hearing.

Tympanic membrane: wide auditory canal, no cerumen, both membranes lightly atrophic, not drawn in.

Hearing test:

R. whispering ad concham.

L. whispering audible at 25 cm.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 128, L. 128.

Operation R. ear.

When elevating the plastic flap the auditory canal skin was torn over a length of 50 mm. Nevertheless a satisfactory flap was obtained which covered the fistula well.

Result 4 months after operation: good improvement, whispering heard at 3 metres. Fistula reaction +.

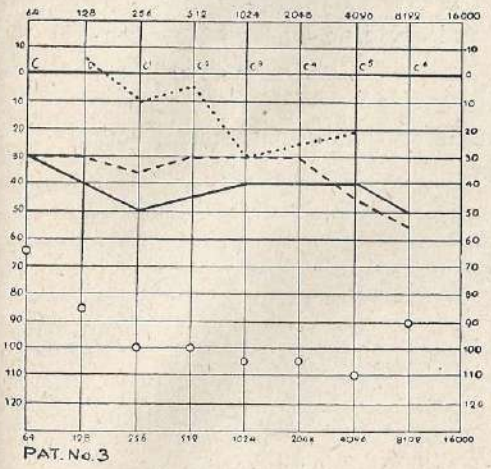


Fig. 8.

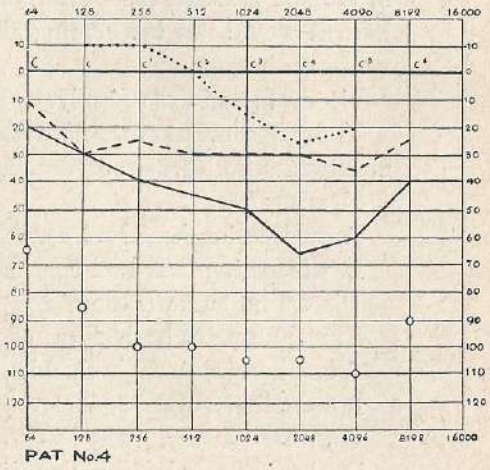


Fig. 9.

Case 5. Man aged 41 (fig. 10).

Hard of hearing since age of 15, alternating progressive and stationary periods. R. ear worse than left. No trouble from tinnitus. Patient would not use hearing aid and could no longer follow conversation in company. Mother and three brothers hard of hearing.

Tympanic membrane: auditory canal only slightly wider than normal, no cerumen, both membranes somewhat dull and not atrophic.

Hearing test:

R. whispering heard ad concham.

L. whispering heard at 25 cm.

Weber: no lateralization.

Rinne: neg. both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 256.

Operation R. ear.

No operative complications.

Result 3 months after operation: marked improvement in hearing, whispering heard at 2 metres. Fistula reaction —.

Case 6. Man 36 years old (fig. 11).

Progressively hard of hearing since 24 years old, though stationary in the last year. R. ear worse than the left. Tinnitus only in R. ear. Patient would not use hearing aid and could no longer follow conversation in company.

No deafness in the family.

Tympanic membrane: normal auditory canal, no cerumen, both membranes dull grey, not drawn in.

Hearing test:

R. whispering heard ad concham.

L. whispering heard ad concham.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 256.

Operation L. ear.

During the whole operation diffuse bleeding difficult to arrest.

Result 3 months after operation: whispering heard at 50 cm. Fistula reaction —.

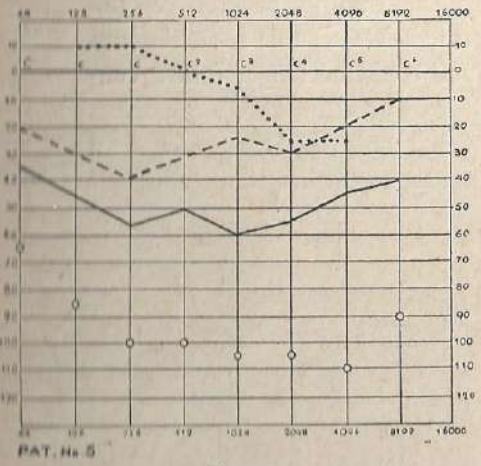


Fig. 10.

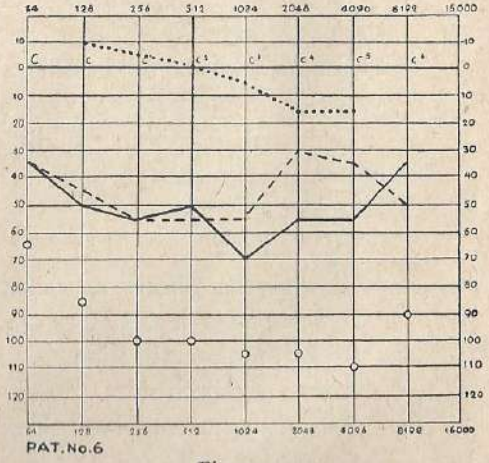


Fig. 11.

Case 7. Woman 42 years of age (fig. 12).

Progressively hard of hearing since 28th year. L. ear worse than right. Much trouble from tinnitus in both ears.

Patient used a bone conduction hearing aid but can only follow conversation in company with difficulty. No deafness in the family.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes slightly atrophic, not drawn in.

Hearing test:

R. very loud talk heard ad concham.

L. very loud talk badly heard ad concham.

Weber: no lateralization.

Rinne: neg. both sides.

Schwabach: slightly diminished both sides.

Lowest limit: R. 512, L. 512.

Operation L. ear.

No operative complications.

Result 3 months after operation: very decided improvement of hearing, whispering heard at 10 cm. Fistula reaction +.

Case 8. Man of 47 years (fig. 13).

Progressively hard of hearing since 25 years old. L. ear worse than right. At times trouble from tinnitus in L. ear.

Patient would not use hearing aid and could only with difficulty follow conversation in company. Three brothers and mother hard of hearing.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes lightly atrophic and dull, not drawn in.

Hearing test:

R. whispering heard ad concham.

L. loud talk heard ad concham.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: slightly diminished both sides.

Lowest limit: R. 256, L. 256.

Operation L. ear.

No operative complication. In the evening following operation some bleeding in wound cavity due to pressure during tendency to vomit.

Result 2 months after operation: whispering heard at 25 cm. Fistula reaction +.

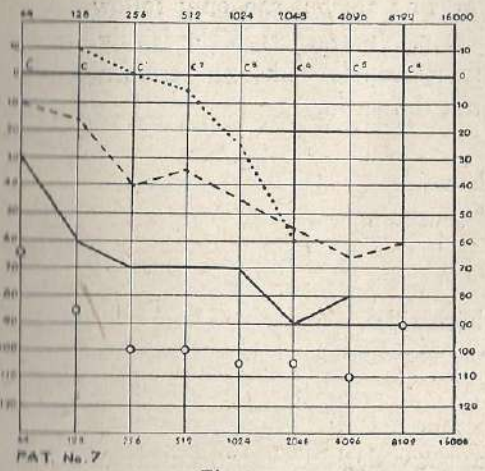


Fig. 12.

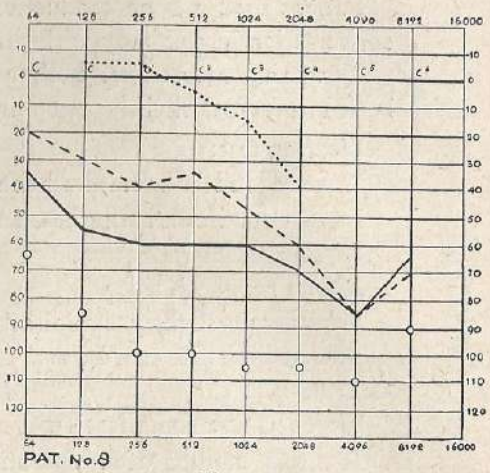


Fig. 13.

Case 9. A woman of 25 years (fig. 14).

Progressively hard of hearing since 4 years of age. L. ear worse than the right. There had been tinnitus in both ears, but not at that time.

Patient would not use hearing aid and could not follow conversation in company. Hearing much worse since pregnancy with only child.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes very transparent and atrophic, not drawn in.

Hearing test:

R. whispering heard ad concham.

L. whispering heard ad concham.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 512, L. 512.

Operation L. ear.

Result 3 months after operation: considerable improvement of hearing. Whispering audible at 4 metres. Fistula reaction +.

Case 10. Man 33 years old (fig. 15).

Progressively hard of hearing since 25th year. L. ear worse than

right. Patient had formerly been troubled with tinnitus. Refused to use hearing aid and had difficulty in following conversation in company. No deafness in the family.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes lightly atrophic and transparent.

Hearing test:

R. whispering heard ad concham.

L. loud talk heard ad concham.

Weber: lateralization to left.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 480.

Operation R. ear.

No operative complications.

Result 2 months after operation: considerable improvement of hearing. Whispering audible at 1 metre. Fistula reaction —.

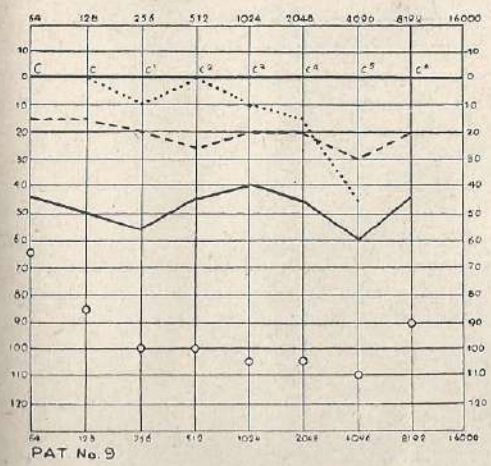


Fig. 14.

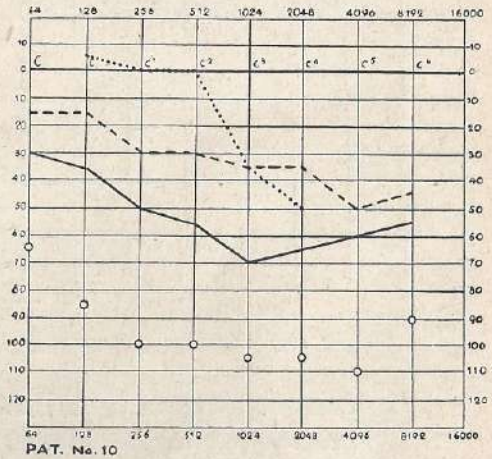


Fig. 15.

Case 11. Man 40 years old (fig. 16).

Progressively hard of hearing since 20th year. Both ears equally bad. Formerly had tinnitus. Patient used bone conduction hearing aid, but could not follow conversation in company. No deafness in the family.

Tympanic membrane: both auditory canals wide, no cerumen, both membranes very transparent and atrophic, not drawn in.

Hearing test:

R. whispering heard ad concham.

L. whispering heard ad concham.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 256.

Operation L. ear.

No operative complications.

Result 2 months after operation: good improvement of hearing. Whispering heard at 1 metre. Fistula reaction +.

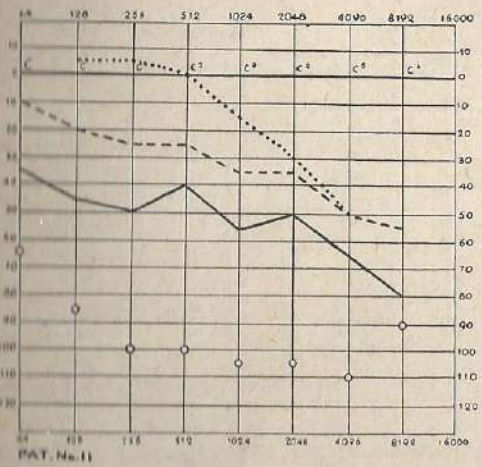


Fig. 16.

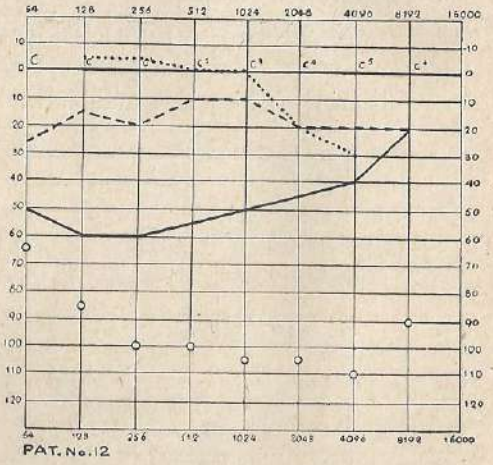


Fig. 17.

Case 12. Woman 37 years old (fig. 17).

Progressively hard of hearing since 23 years of age. R. ear worse than the left. Formerly troubled with tinnitus in R. ear. Patient found hearing aid troublesome and could not follow conversation in company. No deafness in the family.

Tympanic membrane: both auditory canals rather wide, a trace of cerumen, both membranes dull grey and opaque.

Hearing test:

R. whispering heard at 10 cm.

L. whispering heard at 25 cm.

Weber: no lateralization.

Rinne: negative both sides.

Schwabach: normal both sides.

Lowest limit: R. 256, L. 256.

Operation R. ear.

No operative complications.

Result 3 months after operation: good improvement of hearing. Whispering audible at 3 metres. Fistula reaction +.

8. *Post-operative complications.*

I will first mention here the damaging of the N. facialis during the operation. This is a complication that can be avoided if the chiselling and burring is done carefully and the surgeon has adequate anatomic knowledge of the mastoid and ensures that the operative field is kept clear for good visibility.

Paresis of the N. facialis is also apt to set in three of four days after the operation, as described by CAMPBELL. Probably this paresis is due to oedema in the facialis canal, because usually the nerve recovers after six weeks.

Another complication, which is very serious, is purulent labyrinthitis. That this is a complication that must be reckoned with is evident from SHAMBAUGH's report of 1.2 % totally deaf cases after the operation. This stresses the necessity of the utmost sterility in working and taking care not to operate while there is any infection in the nose and nasopharynx, whilst furthermore the postoperative administration of penicillin will certainly be indicated.

The above mentioned complications have fortunately not occurred in my cases, but there has been the occurrence of traumatic labyrinthitis, as it is called by KINNEY. It appears from literature that other surgeons also experience this complication as a rule. Immediately after the operation the patients have a horizontal rotatory nystagmus towards the side not operated upon, and this nystagmus is usually of the third degree. It lasts mostly from two to four days, with an exceptional case of a few days longer. It was probably this labyrinthitis that was the cause of some of my patients not showing a positive fistula reaction although the fistula was open. FOWLER demonstrated experimentally that the cause of this lies most probably

in a local labyrinthitis around the fistula immediately after the operation. Among CAMPBELL's patients there were a number with a negative fistula reaction. SHAMBAUGH likewise reports a much higher percentage of successful cases than of patients with a positive fistula reaction.

Finally I would mention the post-operative serous labyrinthitis as it is called by LEMPERT, which he ascribes mainly to an infection arising from the plastic flap. SHAMBAUGH lays more emphasis upon the bleeding from this skin flap and upon the blood penetrating into the fistula during and after the operation. Whereas the complication last mentioned concerned mainly the vestibular part of the labyrinth, this complication concerns principally the cochlea. In how far these two complications are related one to the other I cannot at the moment say. SHAMBAUGH was able to demonstrate experimentally that blood that had entered the fistula penetrated via the perilymphatic space into the cochlea, deposited itself on the REISSNER membrane and formed there a fibrinous layer, this being taken by SHAMBAUGH as the cause of the original improvement in hearing being partly lost again. Already the second day following the operation this complication manifests itself in loss of acuity in the high notes whilst the low notes are still heard quite well. The turning point, remarkably enough, always lies in the region of 1024—2048 vibrations (see figs. 18 and 19); it must be remembered that these audiograms are recorded without dressing but with the sponge and wax gauze still filling the cavity.

On the fourth day following the operation the gain in hearing for the low notes also suddenly disappears, to return again between the tenth and the twenty-first post-operative day, as also reported by LEMPERT. Sometimes this is a gradual process but cases are known where it takes place rather suddenly.

Recovery from the post-operative reduced perception of the high notes is much slower and is not complete until the ear has dried (see figs. 20 and 21). When the ear is dry and thus the cavity epithelialised hearing is as a rule at its best, as is also concluded by SIMSON HALL and KINNEY. SIMSON HALL points out the importance of getting the cavity dry as quickly as possible, as otherwise the gain in hearing may be lost. He gives no explanation for this, but two reasons could be mentioned: firstly the fact, pointed out by FOWLER,

that in case of infection in the wound cavity there is a greater tendency of the fistula closing; and secondly the toxic influence apparently emanating from the infected wound cavity, clearly noticeable from the fact that the perception of high notes — apparently the most sensitive — remains poor or but little improved until suddenly it becomes much better as soon as the wound cavity has dried up.

As a rule hearing recovers after a post-operative serous labyrin-

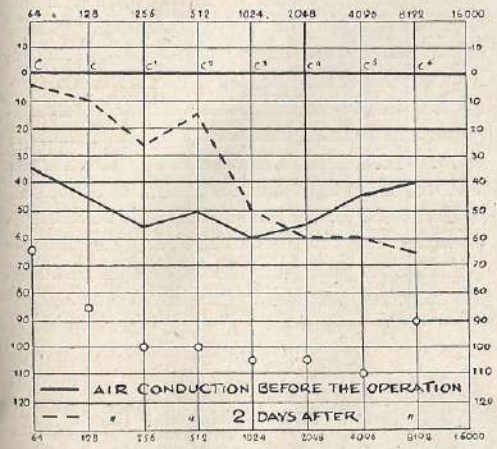


Fig. 18.

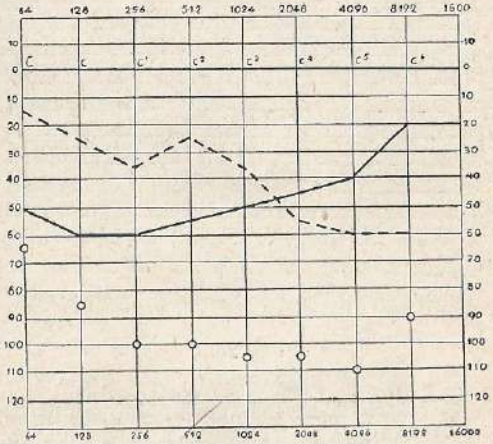


Fig. 19.

thitis, but cases are known where it is left as it was before the operation.

LEMPERT tried to avoid post-operative serous labyrinthitis by completely filling up the wound cavity with paraffin wax. Later on he tried to do this by inserting a cartilaginous stopple in the fistula. As already remarked, he believes the cause of this labyrinthitis lies in an infection from the plastic flap.

SHAMBAUGH is more inclined to ascribe the cause of this complication to the penetration of blood into the perilymphatic space, which he tries to prevent by applying an elastic pressure to the plastic flap, using pieces of sterilised sponge to fill up the traumatic cavity. Further, he places the patient with the head in such a position that the cochlea lies higher than the fistula in the horizontal canal.

Thus for 48 hours after the operation the patient lies on the operated ear.

CAWTHORNE fills up the traumatic cavity with serum into which trombine is injected. It has the advantage that it does not need removing afterwards, because it dissolves of itself.

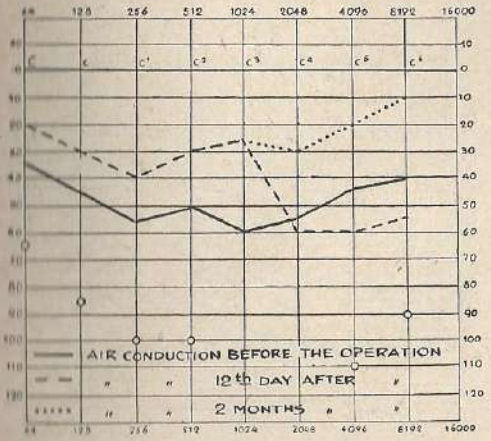


Fig. 20.

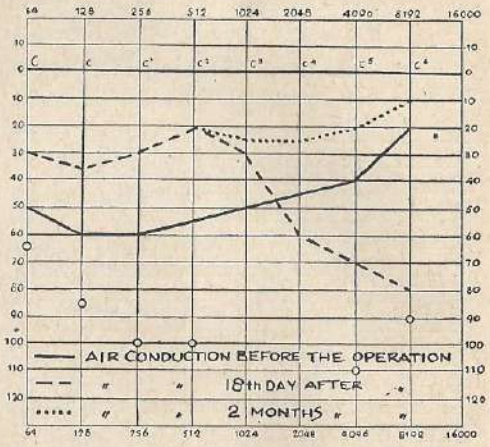


Fig. 21.

In this stage of labyrinthitis most patients are troubled with tinnitus, at times with throbbing in the ear and a perceptible diplacusis. It is very difficult to take an audiogram of these patients, because sometimes the different notes continue to ring a long time after; the patient often declares he can hear the sound while the signal lamp is burning, thus when the sound has been switched off.

All these symptoms disappear as soon as hearing improves. A corrective operation is only called for when an osseous closure of the fistula is suspected. Should there be no immediate improvement in hearing after the post-operative serous labyrinthitis there is of course no use in performing a corrective operation, especially if we are to ascribe the cause to the changes in the cochlea described by SHAMBAUGH.

9. *Post-operative course of own operational cases.*

All patients showed the symptoms of post-operative traumatic labyrinthitis. In all cases there was a horizontal rotatory nystagmus,

sometimes of the third degree, and directed towards the non-operated side. In most cases this lasted three days, and in one case a few days longer. In two cases the nystagmus had already disappeared 24 hours after the operation. In the cases where the fistula had been shut off with a cartilaginous stopple, during the first fourteen days the patients had attacks of dizziness when suddenly turning the head in a horizontal plane. There was one case, where the fistula had not been shut off with the cartilaginous stopple, where for four weeks the patient felt very unsteady on his legs when walking.

In all cases the hearing immediately after the operation was much better, but on the third or fourth day a relapse set in owing to the post-operative serous labyrinthitis. In only two cases (No. 2 and No. 11) was this phenomenon appreciably less marked.

Those patients who had been troubled with tinnitus before the operation and showed a considerable improvement in hearing after the operation were no longer troubled with tinnitus.

There was no post-operative elevation of temperature or any particular complication.

After a few weeks patient No. 8 had a rather narrow entrance of the auditory canal which I had to widen operatively.

On an average the traumatic cavity had dried up in 8—9 weeks, in one case already after 5 weeks and in another not until after 12 weeks. As already stated, hearing — particularly perception of the high notes — is at its best when the cavity dries up. The same is reported by other surgeons, i.a. CAMPBELL and G. PASSE. Subsequently the hearing diminishes again by a few decibels, after which it remains stationary.

It is not the intention to discuss at length the results of my own operative cases in this thesis, because they cannot be properly judged until a longer period of time has elapsed. CAMPBELL thinks the result cannot be judged before 8 months after the operation, whilst SHAMBAUGH holds to 12 months and CAWTHORNE even two years.

I will therefore speak only of the direct operative results. Of my 12 cases 8 were successful, 2 gave slight improvement and in 2 other cases the hearing remained practically the same as before. Where I speak of successful cases I mean those where the patients are satisfied with the result and whose hearing for speech has improved, so that my judgment is not based only on the question whether after

the operation the audiometric test showed 30 decibels or more loss of hearing. As a matter of fact, considering the loss of hearing prior to the operation, this was not always to be expected.

Can any reason be advanced as to why in two cases there was no improvement? In both these cases there was during the operation diffuse bleeding from small bone vessels which was difficult to arrest. I suspect that a small quantity of this blood penetrated into the fistula.

Of the two cases where there was only a slight improvement in hearing one had rather profuse bleeding in the cavity in the evening following the operation, while the other patient had heavy vomitting attacks after the operation and owing to the pressure there was likewise some slight bleeding in the cavity. Most probably in both cases some blood penetrated underneath the plastic flap into the fistula.

It would therefore be of great help if we could dispose of some means of immediately sealing the plastic flap in situ. This means has perhaps been found in the solutions of fibrinogen and thrombin with which experiments are now being made in plastic surgery in England. These two solutions mixed together and exposed to the atmosphere give a very hard substance within one minute. In England it is believed it can be used for skin grafting. It should, however, also be suitable for our branch of surgery and may provide the solution to the problem of post-operative labyrinthitis.

VASOMOTOR SYMPTOMS WITH OTOSCLEROSIS AND THE OTOSCLEROSIS OPERATION.

I. *Improved hearing in the non-operated ear.*

In the foregoing no mention has been made of the phenomenon that in every case immediately after the operation there was improved hearing in the non-operated ear.

It is most remarkable, when standing by the bedside, how much easier one can talk with the patient than before the operation. And notwithstanding the post-operative dizziness the patients are greatly pleased with what they regard as the success of the operation. Sounds they have not heard for many a year are perceived again and many of them can hardly bear the sudden noise. Patient No. 8, who before the operation was very bad of hearing (whispering heard ad concham), heard, in the evening following the operation, a nurse walking along at the end of the corridor (with rubber floor covering) at a distance of quite 30 metres, whilst the door to his room was closed. Others hear a clock ticking, a tap running, motorcars passing by the hospital. Many more instances could be given, but what I have mentioned here will suffice.

On the face of it one is inclined to think that these are already the results of the operation, but when an audiogram is taken of the non-operated ear and of the operated ear still with the dressing on, it is soon found that in most cases it is the non-operated ear that has the better hearing than before. The operated ear perceives the various notes of the audiometer at a level that is in any case lower than that of the other ear. This is understandable when we bear in mind that the operative cavity is filled with paraffin wax, gauze and sponge, whilst this filling is furthermore saturated with secretions and some blood, and over it all is a thick dressing.

Figs. 22—33 show the improvements in hearing of- the non operated ear of the various patients one or two days after the operations. In some cases there is a difference of 10 to 15 decibels, and considering that it occurs in all cases I do not think that it is a question of a psychic phenomenon, as SHAMBAUGH and also LEMPERT are inclined to believe. Moreover an intelligent patient notices this himself.

Literature mentions some cases of better hearing in the non-operated ear. GRAY observed in 1935 that in cases where hearing was improved by his thyroxine treatment there was also improvement in the non-treated ear. URBANTSCHITSCH reports the same phenomenon, in his "Lehrbuch der Ohrenheilkunde" (1910, page 375). He gives instances where after operation on one ear also the hearing in the other ear was favourably affected. This occurred after teno-

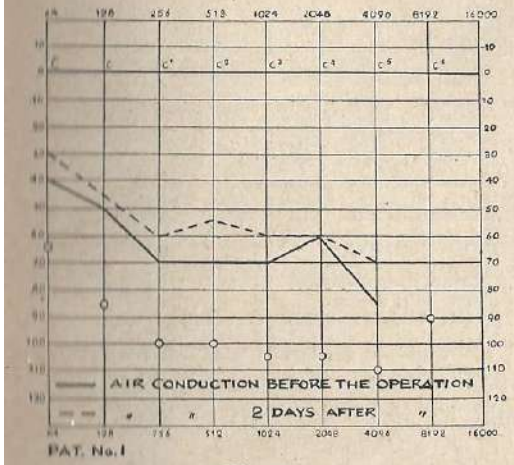


Fig. 22.

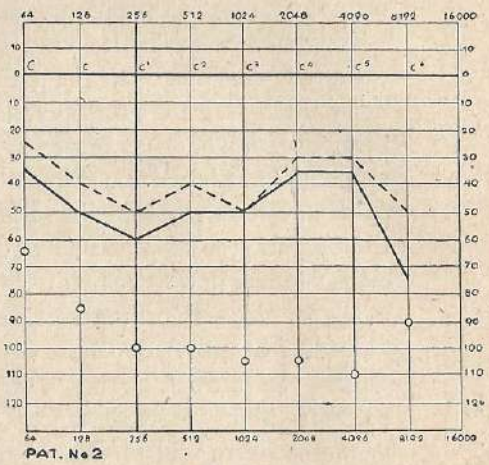


Fig. 23.

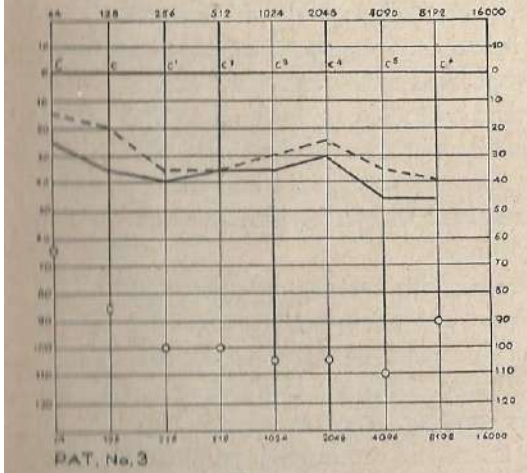


Fig. 24.

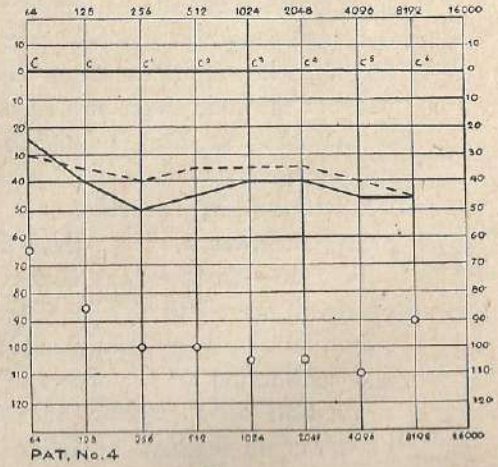


Fig. 25.

tomy of the musculus tensor tympani, after tenotomy of the tendon of the musculus stapedius, after resection of the malleus or of the malleus together with the incus.

URBANTSCHITSCH likewise found that dilatation of the tuba with a bougie on one side may result not only in improved hearing but also reduced tinnitus in the other ear. He writes (on page 308):

"Dementsprechend kommt auch den Bougierungen des Tuben-

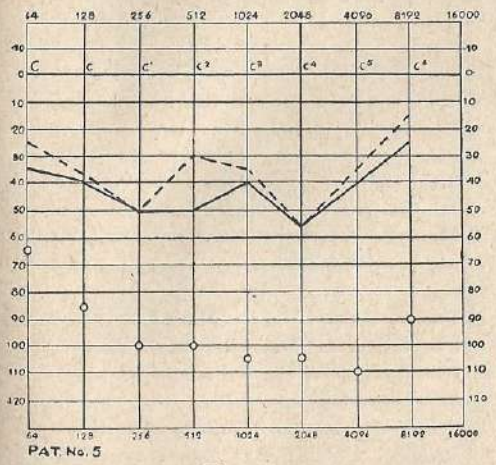


Fig. 26.

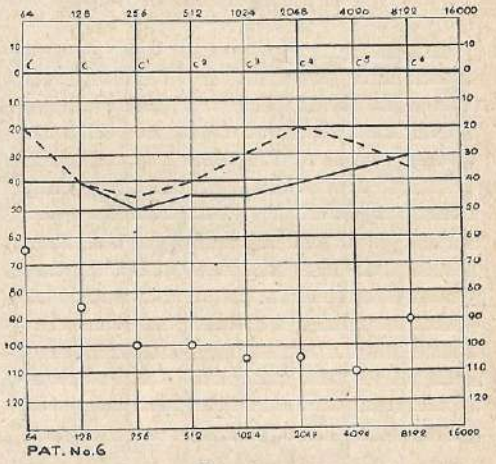


Fig. 27.

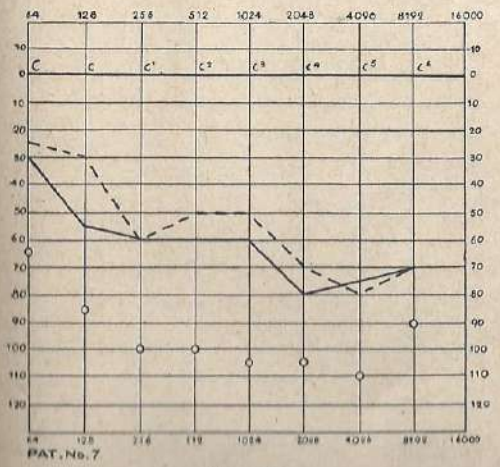


Fig. 28.

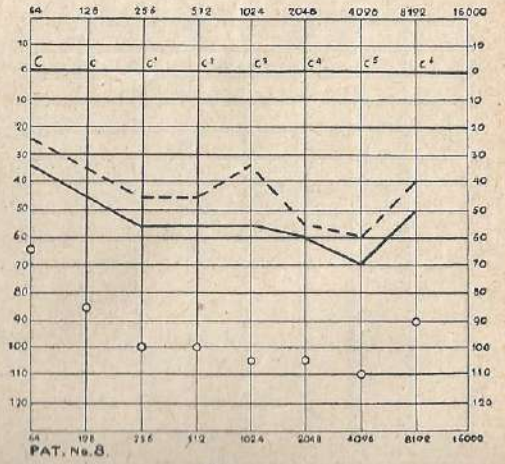


Fig. 29.

kanales nicht nur eine direkte locale, sondern auch eine irradiiert ausgelöste centrale Wirkung zu. Diese letztere erstreckt sich nicht nur auf die behandelte, sondern auch auf die andere Seite, so dasz also z. B. eine Bougierung der rechtsseitigen Ohrtrompete nicht nur eine Steigerung des Hörsinnes am rechten, sondern auch am linken Ohr veranlasst."

URBANTSCHITSCH has thereby in mind the excitation of some sensory trigeminus branch.

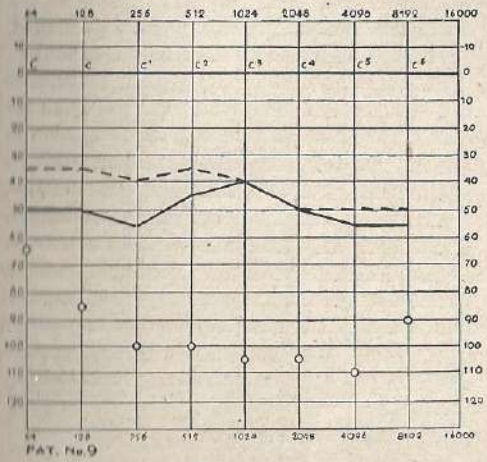


Fig. 30.

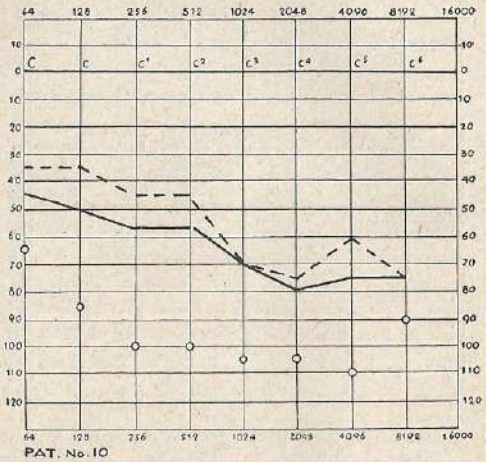


Fig. 31.

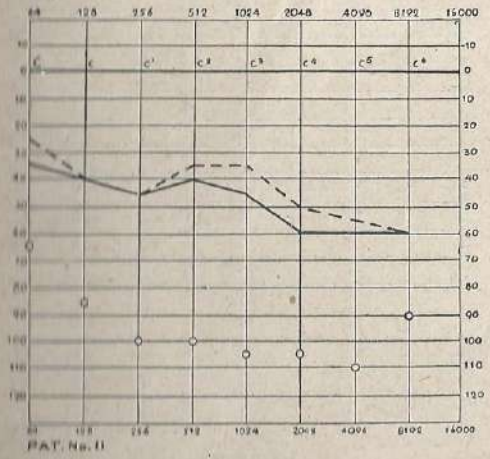


Fig. 32.

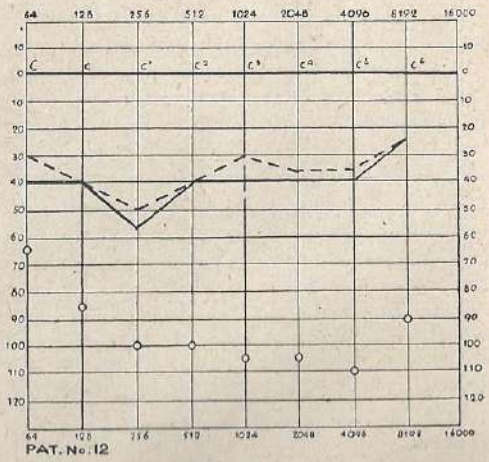


Fig. 33.

SOURDILLE, too, has observed this phenomenon and speaks of a "reflexe oto-otique" and questions whether it is a trophic or a vascular effect. He calls this phenomenon "symphatie auriculaire".

HOLMGREN likewise observed improved hearing in the non-operated ear, but he does not give any explanation for it.

CAMPBELL found an average gain in hearing of 3.6 decibels in the non-operated ear and 6.2 decibels at the moment that the hearing in the operated ear was at its best. He does not give any explanation either.

KOPETZKY believes that the improved hearing of the non-operated ear is due to an "awakening of the cortical hearing centre", leaving it at that and giving no further explanation.

NASIELL ascribes this phenomenon to vibrations in the skull caused by the chiselling open of the mastoid. As proof he exposed the mastoid of an otosclerosis patient and imitated the chiselling by tapping on a punch. The result was improved hearing in both ears, which was maintained for a month. In a second experiment he did not succeed in improving the hearing. My opinion is that in his experiments he brought about hyperaemia, thus finding an improvement in hearing that way. That his theory of skull vibration is not correct is evident from the fact that GRAY observed the same thing after a simple injection of thyroxine in the tympanic cavity, without exciting any vibration in the skull at all.

From this review of the literature on the subject it appears that nobody has really been able to give a good explanation of the phenomenon.

I have endeavoured to account for this remarkable symptom and believe an analogous phenomenon is to be found in GRAY's otosclerosis paradoxica. Before going into this question further, however, I would briefly recall to mind GRAY's theory of otosclerosis.

In his paper read before the Royal Society of Medecine on 4th May 1934, GRAY began with a detailed pathological review in connection with some post-mortem examinations. The conclusions at which he arrived are: that the spreading of the osseous changes in the capsule of the cochlea has little to do with the seriousness of the deafness, the tinnitus or the duration of the disease. He found a remarkable symmetry in the changes in the bone of both the organs of hearing. GRAY found changes in the N. cochlearis, degenera-

tive changes in the medullary sheath and the neurilemma. The N. vestibularis was normal. In some cases these changes were found even before fixation of the stapes. As regards the clinical symptoms, GRAY called attention to the fact of otosclerosis paradoxica occurring also when amyl-nitrite is inhaled, when there is a sudden improvement in hearing due to changes taking place spontaneously in the blood circulation (dilatation of the blood vessels). These phenomena, taken together with the defective vasomotor reflex of the tympanic membrane, lead GRAY to think that a defective vasomotor reflex must play a part in the otosclerosis.

A greater supply of blood is needed when a tissue is functioning than when it is at rest, and the increased circulation is regulated by the vasomotor system. The stimulus acting upon a certain tissue and activating it is at the same time a stimulant for the vasomotor system supplying blood to that tissue. As a consequence of the vasomotor reflex there is a dilatation of the arteries and arteriolae, accompanied as a rule by an increase in the volume of the tissue. In the case of arteries situated in bone, as in the organ of hearing, this is not possible; these arteries can only dilate outside the osseous part, with the result that there is an accelerated flux through the vessels in the bone.

When the vasomotor reflex of an organ does not function properly then the cells may undergo degeneration or may be destroyed by neighbouring cells, or what is most usual, the tissue may remain normal in appearance, but the cells do not immediately die for want of nourishment.

The stimulus for the organ of hearing is sound, and this also stimulates the vasomotor system. GRAY presumes that a permanent defect in the vasomotor reflex of the whole organ of hearing is the cause of otosclerosis. This must then be reconciled with the clinical and pathological symptoms.

1. The vascular reaction to stimulation of the tympanic membrane is much less in cases of otosclerosis than in persons with a normal sense of hearing (FROESCHELS).

2. Tinnitus indicates that the nerve structures are involved in the disease and sometimes it precedes deafness. On the other hand in some cases it does not occur even with the most serious stapes ankylosis.

3. When amyl-nitrite is inhaled there is a considerable improvement in hearing, as occurred more or less in all cases of otosclerosis investigated by GRAY. This means of dilating the vessels furnishes proof that the organ of hearing can indeed still function satisfactorily if there is a sufficient supply of blood. This cannot be any regenerative process in the damaged nerve cells, because it takes place too quickly for that — in a few seconds. It is a matter of the function of complete living tissues being restored by a rapid improvement in the blood supply.

4. Otosclerosis paradoxica is closely related to this. It is the phenomenon of a patient hearing better under conditions where in many cases one would even expect it to be otherwise. These conditions arise suddenly and are likewise due to a drastically improved blood supply (consumption of alcohol while in a state of fatigue; riding in an open car without windscreen; hyperaemia in the head). They are conditions mostly of short duration!

As to the pathological anatomic changes, GRAY believes that these, too, are to be accounted for by his theory of the defective vasomotor reflex. The osseous changes in the labyrinthine capsule particularly arise just there (Foramen ovale) where both the function and the traction (ligaments of the moving stapes) require an abundant supply of blood, which is lacking. The nerve, too, requires extra blood supply when functioning, and when this is lacking the consequence is the degeneration described. The cells of Corti's organ and of the spiral ganglion show no changes, except perhaps in the last stage of this disease.

GRAY draws attention to the symmetry of the structural changes in both organs of hearing. This is due to the fact that the vasomotor nerves controlling the blood supply to the organs of hearing are anatomically symmetrical and belong to one and the same system. Whenever structural changes take place as a result of defective functioning of these nerves then those changes are bound to be symmetrical too. So much for GRAY's theory.

Support for GRAY's theory is found in the experiments conducted by RAINISCH, who in cases of otosclerosis observed deterioration of hearing when adrenaline was injected but not in cases of other forms of deafness.

LEIDLER, in an extensive treatise, explains how the vegetative nervous system of the organ of hearing and the vestibulary apparatus

work. The art. auditiva interna supplies the organ of hearing with blood and originates in the art. basilaris. Fibres lead from the gangl. cervic. sup. to the art. carotis interna and together with the latter enter the skull. From the gangl. stellatum fibres lead to the art. vertebralis and in this way reach the cranial fossa. The two plexus carotici connect up with each other in the circulus arteriosus *Willisii*, so that — according to LEIDLER — even when there is stimulation of the sympathicus only on one side there may well be innervation of the vessels on both sides in the same sense! It is in this network of nerve fibres that the vasomotors of the membranous labyrinth have their origin.

My line of thought as regards these processes is the following:

The stimulus for the vasomotor system of the organ of hearing is sound. In the case of otosclerosis there is a defect in this vasomotor reflex system causing changes to take place in the osseous capsule of the cochlea and the nervous tissues. As a consequence of the bone growth of the round window there is fixation of the stapes, with the result that fewer and naturally weaker sound stimuli reach the cochlea. The already badly functioning vasomotor reflex will therefore function still worse. The worse this reflex, the greater will be the structural changes and the stronger the fixation of the stapes, and so on. Thus there arises a vicious circle with an ever-increasing aggravation of the process.

Now when the fistula has been applied in the semicircular canal this vicious circle is suddenly interrupted and, since the perilymph has been remobilized, a condition is brought about which is comparable to that of a non-fixed stapes. A new expansion outlet having been made, the sound waves are again enabled to penetrate into the whole of the labyrinthine system [1]) and the stimulus for the vasomotor reflex system suddenly becomes much stronger. Furthermore, the operative trauma of the mastoid likewise stimulates the vasomotor system, especially in the immediate post-operative period, for a post-traumatic hyperaemia is observed in this region. There are thus two stimuli for the vasomotor system, to be regarded as one

[1]) TULLIO, P., "....Das Ohr und die Entstehung der Sprache und Schrift", Berlin and Vienna, 1929. See also the interesting experiments and literary summary by A. J. H. v. EUNEN, "....TULLIO's reaction in relation to the function of the middle ear", Acad. thesis, Groningen 1942.

entity for both ears (*cf.* GRAY and LEIDLER). Consequently a stimulation of this system in one of the auditory canals must also affect the other. The abovementioned stimuli acting upon the operated ear then cause a better flow of blood to the non-operated ear, so that the hearing on that side is better after the operation. This is borne out by the observation — of which an instance will be given — that the improvement in hearing of the non-operated ear fluctuates with the post-operative fluctuations in the hearing of the operated ear (*cf.* also figs. 34 and 35).

Of course if this theoretical consideration is to carry any conviction it must be supplemented with clinical observations. I have therefore sought for proofs and believe to have found them in the following points:

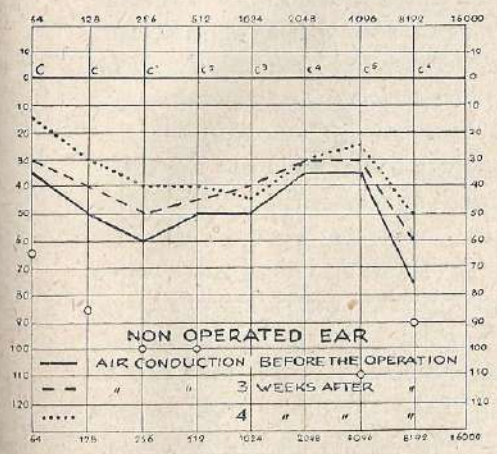


Fig. 34.

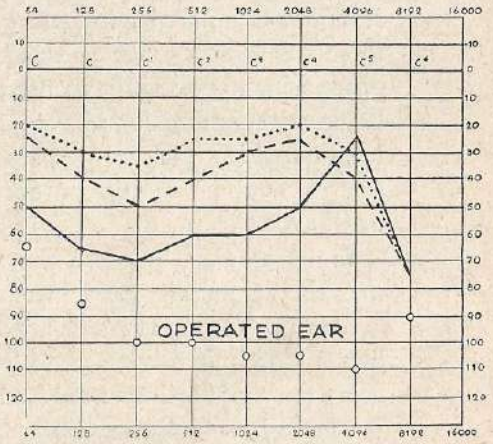


Fig. 35.

1. *Vasomotor defects in the auditory canal.*

FROESCHELS has described the remarkable phenomenon that in cases of otosclerosis the auditory canal becomes less sensitive as deafness increases. GRAY likewise found reduced vascular reaction of the tympanic membrane upon the application of pneumomassage or excitation with a swab, as compared with persons of normal hearing. As already remarked, GRAY takes this phenomenon as a support for his theory.

My assumption was that if, after the operation, there is an improved flux of blood through the non-operated ear, then there must be also improved vascular reaction of the tympanic membrane. I therefore experimented by injecting 100 cc water of 37° C. against the processus longus of the malleus of the non-operated ear, with a thin needle and at constant pressure. I waited two minutes before making an examination (wearing glasses of a power of 3) and carefully recording the vascular reaction of the tympanic membrane. In 15 of the 18 cases I found a stronger vascular reaction after the operation than before it. Often before the operation there was no vascular reaction at all, whereas after the operation there was some reaction. Compared with test persons of normal hearing, however, there was in every case a decided difference to be noticed. The same phenomenon apparently occurred when exciting the tympanic membrane with swabs, but in view of the small differences in vascular reaction, these tests are not absolutely reliable, owing to the difficulty of applying in this way excitations of exactly the same intensity in all cases.

In my opinion, however, these observations do indeed go to prove that the vasomotor reflex in the non-operated ear is improved.

Since, as GRAY was able to demonstrate, in cases of otosclerosis better blood circulation gives better hearing, we must take this as being the cause of improved hearing in the non-operated ear.

The following phenomena, though not to be taken as proof, make it quite probable that as a result of stimulation of the vasomotor system there must be a better blood supply in the operated ear.

2. *The disappearance of tinnitus after the operation.*

One of the most important subjective phenomena is tinnitus. Many patients have it, or at least have had it before, while some have it periodically. According to GRAY the cause is to be sought in the degenerating cells of the acoustic nerve. Apart from the fact that this tinnitus may cease spontaneously through some cause or other, we find that it also disappears under the following conditions:

a. Otosclerosis paradoxica.

One of my patients told me that when she was in the mountains her hearing became much better all at once and that also the trouble-

some ringing in the ears stopped. It is to be assumed in this case that the lack of oxygen in the blood caused the vessels to dilate, resulting in hyperaemia of the organs of hearing.

b. The fenestration operation.

In all my cases where there was improved hearing after the operation and the patients had had trouble from tinnitus, this disappeared entirely. The same phenomenon has been recorded by other operators, among whom SOURDILLE, CAMPBELL, LEMPERT, SHAMBAUGH, G. PASSE, and SIMSON HALL. HOLMGREN reports having observed that tinnitus stopped as soon as the endosteum of the horizontal canal was exposed.

c. Other treatments of otosclerosis patients where hearing has been improved without activating the perilymph.

GRAY mentions the disappearance of tinnitus with improved hearing after injection of thyroxin in the tympanic cavity, administered with the object of effecting hyperaemia.

M. MEYER observed the same thing when hearing was improved as a result of sub-occipital punctures in cases of otosclerosis.

RUBARSCHOW performed a sympathectomy of the cervical sympathetic ganglia of a patient with otosclerosis in view of tinnitus trouble and also had success; in fact there was even a temporary improvement of hearing. The hyperaemia occurring must, in my opinion, have been the cause of the improvement.

In the cases sub *a* and *c* we therefore find that tinnitus disappears as a consequence of hyperaemia, accompanied by improved hearing, while the perilymph continues to be wholly or partly immobilised.

In the case sub *b* tinnitus disappears at the moment that the fistula is applied, thus when the perilymph is again mobilized. The sound stimuli penetrating better into the cochlea are the stronger stimuli for the vasomotor reflex system.

From *a* and *c* as well as *b* I think it is to be concluded that improved blood supply to the nerve cells immediately causes tinnitus to stop.

3. *Raising of the upper tone limit after the operation.*

In three of my cases (patients 7, 9 and 12) I observed a raising

of the upper tone limit, also the non-operated ear sometimes recording higher notes than before the operation:

	Patient No. 7		Patient No. 9		Patient No. 12	
	before	after	before	after	before	after
operated ear	6,000	10,000	9,500	10,500	12,000	12,500
non-op. ear	8,500	9,500	9,200	9,500	12,000	12,000

In agreement with the foregoing it is obvious to assume that before the operation that part of the nervous system responsible for the perception of these higher notes must have been in a sort of comatose, and as a result of the operation was again able to function.

I have not been able to find anything written about this in the literature, but from a study of the various cases published it is to be seen that in several instances there has been a post-operative improvement in hearing amounting to 20—30 decibels above the level prior to the operation where no perception could be recorded with the audiometer.

4. *Improved bone conduction after the operation.*

Among my eight successful cases there were five with improved bone conduction between 512 and 2048 vibrations, the difference amounting to 5—15 decibels. The same phenomenon has also been reported by LEMPERT, HOLMGREN and KOPETZKY.

Bone conduction is generally regarded as a measure for the intactness of the acoustic nerve. When as a result of the operation this nerve receives a better flow of blood it is able to function better, and this manifests itself in improved bone conduction.

5. *A phenomenon observed in one of my otosclerosis cases.*

Some years previously the patient had undergone elsewhere a mastoid operation on the left ear. Hearing was worse in that ear than in the right one and moreover the patient was seriously troubled with tinnitus in the left ear. Upon the left mastoid being exposed a cavity was found which proved to be the antrum enlarged in the previous operation. A large part of the osseous posterior wall of the auditory canal had been removed, as a result of which the skin of the auditory canal had become fibrous and easily tore at once. I then

decided not to operate further, because there was no possibility of making a proper plastic flap. So really only antrotomy was performed on this patient.

Before I was able to tell the patient that I had been unable to perform the operation intended, she said how delighted she was with the result (see figs. 36 and 37). Further she said she was so glad that the troublesome ringing in the left ear had gone. The improvement in hearing of both ears continued for four weeks.

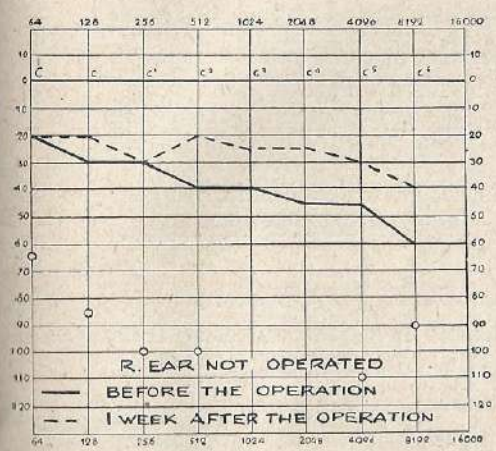


Fig. 36.

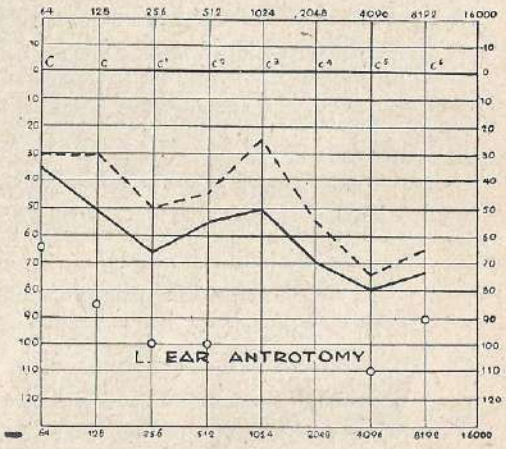


Fig. 37.

Apparently the trauma of the mastoid gave hyperaemia and resulted in the temporary improvement of hearing. Here was another case where the stimulation started in one organ of hearing affected the vasomotor system of both ears.

It has struck me that the improved hearing in the non-operated ear may continue for several months. As already remarked, it fluctuates with the variations in hearing of the operated ear.

The improved hearing has nothing to do with "overhearing"; this can be precluded by masking. It appears, inter alia, that soon after the operation the perception of high tones is often improved in the non-operated ear (see the audiograms), whereas in the operated ear it is mostly greatly reduced!

I believe that in this theory based on a vasomotor reflex an

explanation has been found for the improved hearing of the non-operated ear.

II. *General Symptoms.*

In the treatment of patients with otosclerosis, and especially during the time they are in hospital, I have noticed that they are mostly a particular type of person. Generally speaking, they are highly nervous patients, highly susceptible to changes. They are very often troublesome post-operative patients.

At first I thought this characteristic might be due to their dull hearing, but it appears to exist often also in cases of otosclerosis in its first stages, thus with patients only slightly hard of hearing.

Of the 25 patients upon whom I operated 19 were of the highly nervous type.

One patient has a mother suffering from schizophrenia. Another patient, not operated, is afflicted by the same disorder and is being treated with shock therapy.

In cooperation with the physician, my colleague WENSINCK, I have been seeking for other vasomotor defects in patients suffering from otosclerosis. The symptoms observed are the following: migraine, nervous headaches, nervous heart attacks, fainting fits and many other suchlike symptoms. Our investigations cover too few patients as yet to give any conclusive results.

Also other investigators, inter alia O. MAYER, BAUER and STEIN, FREY and ORZECHOWSKY mention general vasomotor defects.

BAUER and STEIN have found many degenerative stigmata among their patients.

According to M. SCHWARZ, ZORBACH has found mainly asthenic types among the sufferers of otosclerosis.

III. *Otosclerosis paradoxica.*

One is already familiar with the publications of GRAY and ASHCROFT describing otosclerosis paradoxica, so called because the patients hear better at a moment when the contrary would be expected. This phenomenon is due to a vascular dilatation brought about by external conditions. According to GRAY it is not so rare if one only carefully studies the anamnesis of the patients.

I would add here a few cases out of my own practice:

1. There is that of the patient already mentioned, who always heard much better when she was in the mountains and was immediately relieved of her troublesome tinnitus. I gave as an explanation of this phenomenon the lack of oxygen as causing vascular dilatation. [1])

2. There was a similar case of a man 45 years of age, suffering from otosclerosis, who upon stepping out of the plane from Amsterdam to Groningen was so delighted because, as he thought, his hearing had suddenly become normal again. After a few hours, however, it returned to the former level.

3. One of my patients underwent a minor nose operation in an outpatients' department elsewhere and upon his return home he thought his hearing was quite normal again, but the next morning there was no longer any sign of the improvement.

The very same evening after the operation subsequently performed on this patient for otosclerosis there was a most remarkable improvement in hearing. When during the post-operative treatment the entrance to the auditory canal had closed up a little I performed a small plastic operation under local anaesthesia and filled up the traumatic cavity with a tampon. In the evening the patient, who was living in a hotel during the post-operative treatment, had to leave the restaurant because he could not bear the noise; unfortunately on both these occasions he did not inform me at once, so that I was unable to take an audiogram. Thus each time this patient had undergone an operation phenomena occurred which indicated otosclerosis paradoxica.

Apparently each operation must have given a considerable stimulus to his vasomotor system.

4. Then I would mention a sufferer from otosclerosis who experienced a very marked improvement in hearing immediately following the birth of her first child (10 minutes after); within the following weeks this improvement gradually disappeared. At her second confinement five years later exactly the same phenomenon occurred.

5. Mrs. H., 37 years old, had been attended by me at intervals

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[1]) LANDOIS and ROSEMANN, Lehrbuch der Physiologie, 1928.

during the last five years. She has been completely deaf in the left ear since birth. There had been progressive deterioration in the right ear for some years, while patient is also troubled with tinnitus in the right ear.

Examination 2nd July 1946: both tympanic membranes slightly atrophic, auditory canal normal, no cerumen. Left ear inactive in the cochlea and the vestibular part of the labyrinth. Whispering heard in the right ear at a distance of 1 metre. Bone conduction diminished, lower limit 256, *Rinne* positive, *Weber* lateralized to the right. Considering the positive *Rinne* test and the diminished bone conduction I had always thought the patient was suffering from a progressive internal ear deafness on the right side. On 27th February 1947 patient informed me that three days before she had over-exerted herself while bending down and pulling one of her boy's clogs out of a frozen heap of snow. She felt a rush of blood to her head, became rather dizzy and suddenly was able to hear much better. I then tested her hearing again: whispering was heard in the right ear at a distance of three metres, lower limit 128, *Rinne* ±, *Weber* lateralized to the right. This improvement resembled so much that experienced with otosclerosis that I wondered whether after all the patient might not be afflicted with otosclerosis and not with labyrinthine deafness. I therefore applied the GELLÉ test, which proved to be negative. My opinion is that patient is probably suffering from an otosclerosis with strong degeneration of the acoustic nerve. This again demonstrates the importance of the GELLÉ test.

Figs. 38 and 39 show the audiograms for air and bone conduction taken on 2nd July 1946 and 27th February 1947. On 1st March 1947 patient telephoned to say that her hearing had diminished again. Another test and new audiogram showed the same picture as that which existed on 2nd July 1946.

The phenomenon of otosclerosis paradoxica is all the more remarkable when considering that the fixation of the stapes does not change. As M. MEYER also observed, the fixation of the stapes therefore does not determine only the deafness in otosclerosis.

IV. *Vasomotor symptoms in the tympanic membrane and auditory canal.*

GRAY mentions as one of the clinical symptoms found with otosclerosis the slight vascular reaction occurring when the tympanic membrane is excited.

HOLMGREN and PEYSER carried out an extensive investigation into the occurrence of reduced vascular reaction, diminished cerumen secretion and reduced cough reflex. They arrived at the conclusion that the older the case of otosclerosis the more pronounced is this trio of symptoms. This agrees with what FROESCHELS found.

HOLMGREN bases these three symptoms on what I consider to be

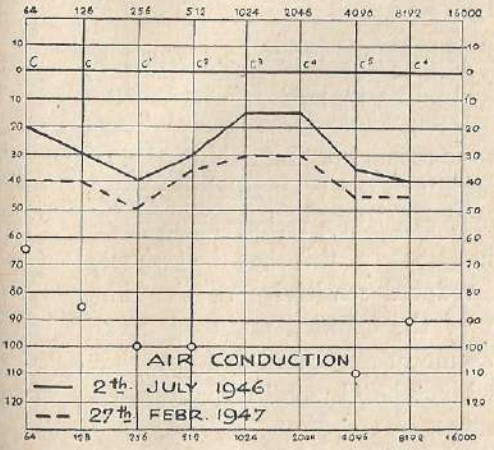

Fig. 38.

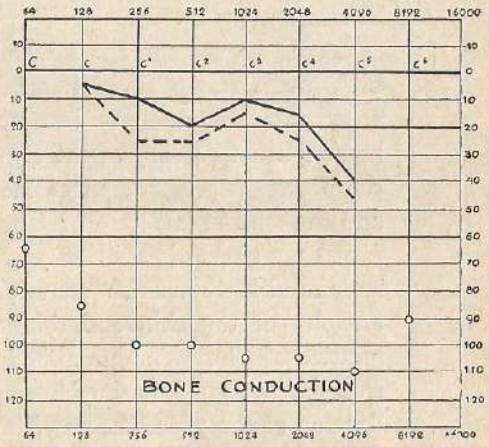

Fig. 39.

— 27th Febr. 1947
--- 2nd July 1946

a fallacious assumption that in otosclerosis the intralabyrinthine pressure is increased. There is no reason whatever for assuming this to be the case. In fact we see an acute improvement of hearing in otosclerosis paradoxica, thus a sudden hyperaemia of the organ of hearing just when we might expect an increase of the intralabyrinthine pressure. According to HOLMGREN's theory the hearing should then diminish. He comes to assume this intralabyrinthine pressure because hearing improves when the fistula is opened and the perilymph begins to flow. This, in my opinion however, is due to a sudden improvement of the vasomotor reflex and, as NASIELL also remarks, to the fact that at that moment the perilymph is suddenly mobilised. HOLMGREN has not furnished proof of this increased pressure under these conditions.

In connection with this symptom the experiments conducted by

Sasz on dogs and rabbits are of importance. Upon inserting a cannula in the round window he found an increased intralabyrinthine pressure with vasodilatation and decreased intralabyrinthine pressure with vasocontraction.

Portmann and Terracol, when performing peri-arterial sympathectomy of the arteria carotis, found in their patients a hyperacusis in the case of hypotension of the labyrinth and a hypacusis in the case of hypertension. Hypotension occurred in cases of vasocontraction in the labyrinth and hypertension in cases of vasodilatation.

Owing to the increased intralabyrinthine pressure, according to Holmgren stapes fixation arises and this in turn leads to stapes ankylosis. The result is fixation of the whole system of auditory bones and immobility of the tympanic membrane. Immobilization of the processus brevis changes the supply to the tympanic membrane and the wall of the external auditory canal in a reflectoric manner. Holmgren takes it as a logical consequence of this that there should be diminished cerumen secretion, reduced sensitivity of the skin, atrophy of the skin and dilation of the external auditory canal, reduced cough reflex, diminished vasomotor reflex and thin, atrophic, transparent tympanic membrane with reduced mobility or immobilization of the processus brevis malleus.

Lempert and Wolff carried out a pathological examination of the auditory bones resected in fenestration operations and found changes that are to be ascribed to defects in the blood supply.

Thus we see that both Holmgren and Gray as well as Lempert and Wolff ascribe the symptoms of the tympanic membrane and external auditory canal to vascular changes, though the first two investigators mentioned assume these to be due to a different cause.

A number of my patients drew my particular attention because their cases did not correspond to the descriptions of Holmgren and Peyser just given. I will quote here briefly their histories and audiograms.

J. D. — a man of 48.

Deafness began at age of 28, progressing up to 32nd year, then remaining stationary. Never had any trouble from tinnitus. No deafness in the family.

Examination:

Both tympanic membranes dull grey, opaque and apparently thickened. Auditory canal on both sides normal width. Some cerumen R. and L. Vascular reflex of both membranes apparently normal. X-ray showed mastoid pneumatization. Whispering heard on both sides ad concham. *Weber* not lateralized. *Rinne* negative both sides. Lower limit both sides 256. Audiograms — see figs. 40 and 41.

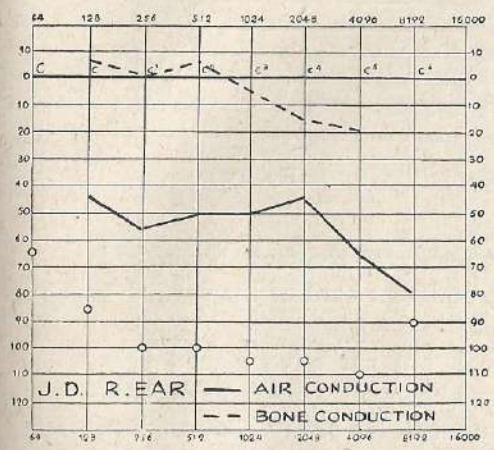

Fig. 40.

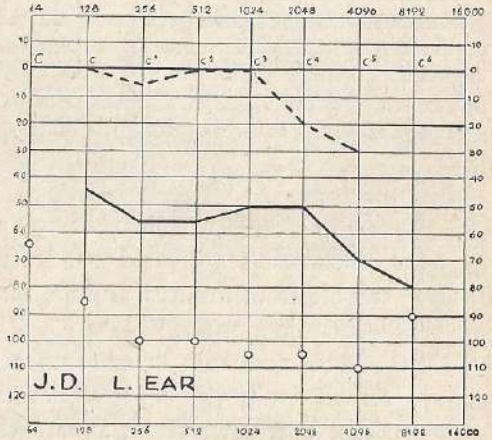

Fig. 41.

G. E. — a woman of 47.

Deafness set in at 20 and progressed up to 26, then becoming stationary. In the last month some trouble from tinnitus in the R. ear, the first time since some years. Mother, two aunts and an uncle hard of hearing.

Examination:

Both tympanic membranes somewhat retracted, dull grey, opaque. Both auditory canals rather narrow; no cerumen. Vascular reflex apparently normal on both sides. X-ray showed good pneumatization of mastoids both sides.

Whispering heard both sides at about 10 cm. *Weber* not lateralized. *Rinne* negative both sides. Lower limit 256 both sides. Audiograms — see figs. 42 and 43.

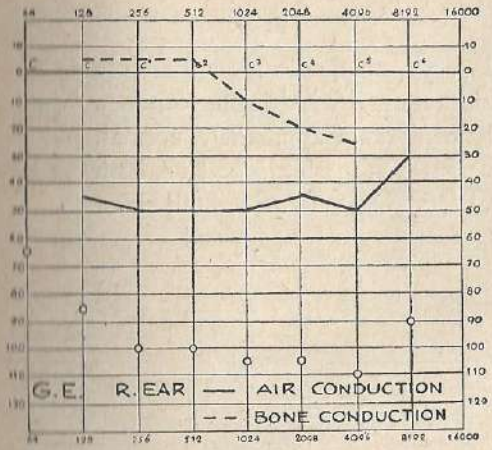

Fig. 42.

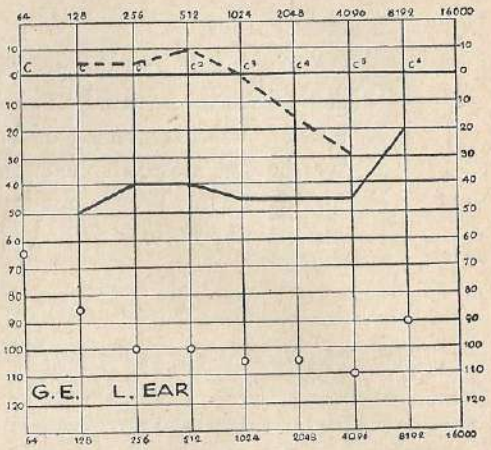

Fig. 43.

M. C. S. — a woman of 39.

Deafness set in at 25 and after 30th year constant. Never had trouble from tinnitus. Cousin, uncle and aunt hard of hearing.

Examination:

Both tympanic membranes normal, but not a very good light reflex. Auditory canals of normal width, with some cerumen both sides. Vascular reflexes apparently normal. No X-ray photos taken. Whispering heard in R. ear at 50 cm and in L. ear at 2 metres. *Weber* not lateralized. *Rinne* negative both sides. Lower limit 256 both sides. Audiograms — see figs. 44 and 45.

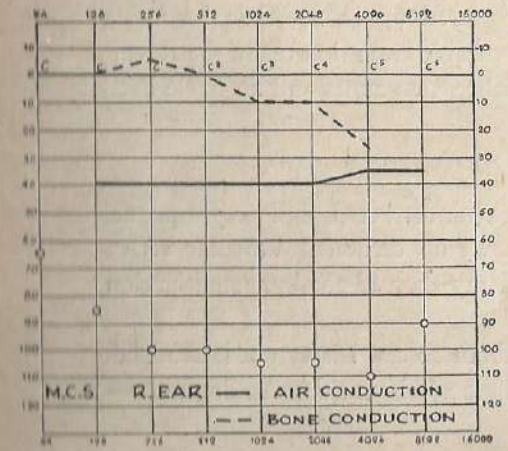

Fig. 44.

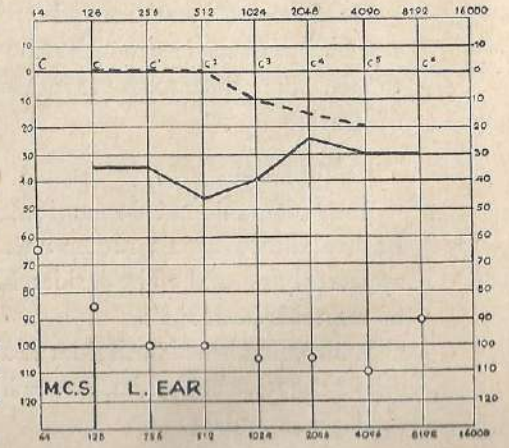

Fig. 45.

L. v. K. — a woman of 57.

Deafness set in at 30 and after 42nd year constant. No trouble from tinnitus. Grandmother hard of hearing.

Examination:

Both tympanic membranes dull grey, opaque, apparently slightly thickened. Both auditory canals rather narrow; no cerumen. Vascular reflexes apparently normal. X-ray showed well pneumatized mastoids. Whispering audible in R. ear ad concham. In L. ear only loud speech ad concham. *Weber* not lateralized. *Rinne* negative both sides. Lower limit L. 256, R. 128. Audiograms — see figs. 46 and 47.

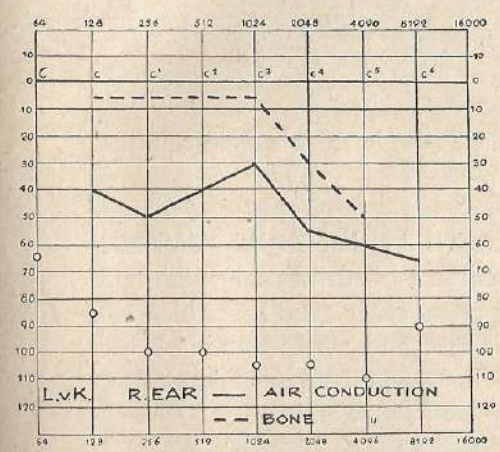

Fig. 46.

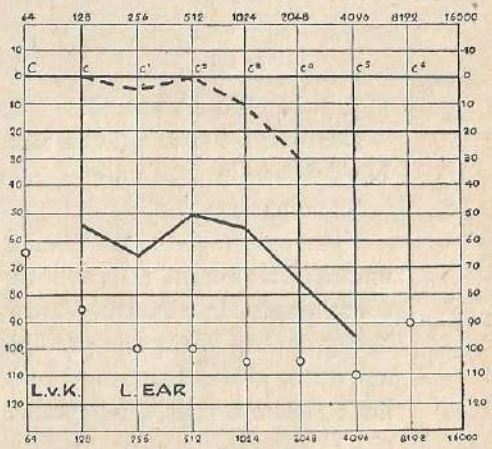

Fig. 47.

C. J. B. — a woman of 62.

Deafness set in at 24, constant since 36. Occasionally very slight tinnitus in R. ear. Father, mother and sister hard of hearing.

Examination:

Both tympanic membranes dull grey, apparently thickened, not transparent. Both auditory canals rather narrow, with some cerumen. Vascular reflex good both sides. X-ray showed normal pneumatized mastoids. Whispering ad concham in R. ear and at 25 cm in L. ear. *Weber* lateralized to the R. *Rinne* negative both sides. Lower limit R. 512, L. 256. Audiograms — see figs. 48 and 49.

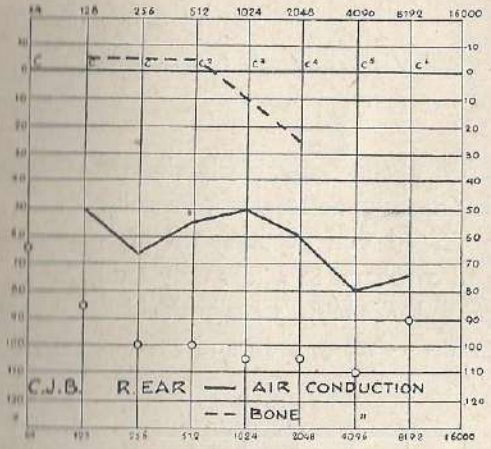

Fig. 48.

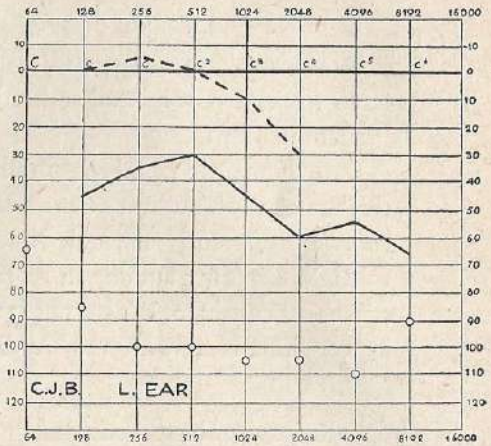

Fig. 49.

J. J. K. — a man of 56.

Deafness set in at 26, constant since 40. No trouble from tinnitus. No deafness in the family.

Examination:

Tympanic membranes normal both sides, but slightly dull and unsatisfactory light reflex. Both auditory canals normal width and containing some cerumen. Vascular reflex fairly good both sides. No X-ray photos. Whispering audible in R. ear ad concham and in L. ear at 10 cm. *Weber* lateralized to R. *Rinne* negative both sides. Lower limit 512 both sides. Audiograms — see figs. 50 and 51.

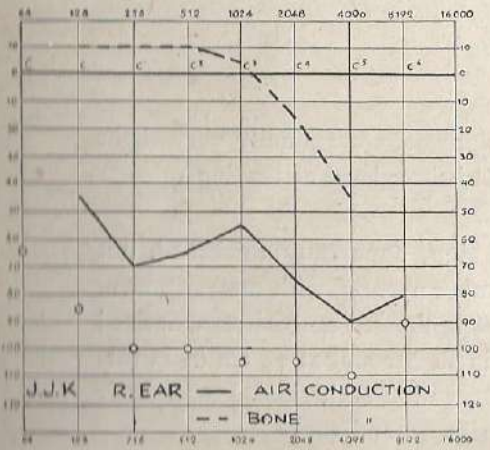

Fig. 50.

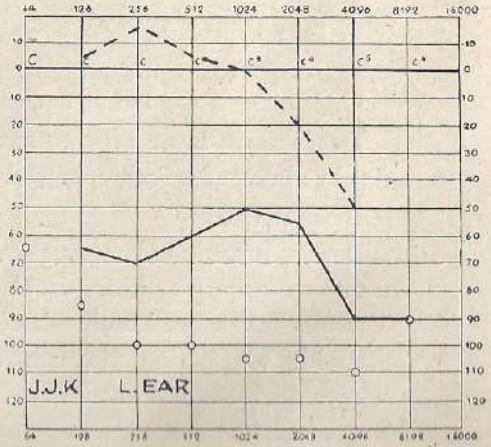

Fig. 51.

What these patients have in common, therefore, is a deafness of the middle ear type, with good pneumatization of the mastoids (X-ray photos). We can therefore fairly safely rule out any adhesive process of the middle ear. In my opinion they are cases of otosclerosis where the auditory canals and tympanic membranes do not give the same picture as usually found with otosclerosis. A symptom common to all the patients is that their sense of hearing has remained constant in recent years. If, therefore, we are to assume that the atrophic symptoms are due to vascular disorders following upon a defective vasomotor reflex, then obviously in these patients the reflex has been restored in some way or other. As a result the tympanic membranes and auditory canals have received a better blood supply (good vascular reflex!), causing the atrophic symptoms to disappear.

In none of the cases just described did I see the patient in the stage when the deafness was progressive. The question is, whether in that stage there were indeed atrophic symptoms in the auditory canal and tympanic membrane. Though of course I cannot prove it, I presume this was the case, for the following reasons:

Since I first observed this phenomenon I have been devoting more attention to these tympanic membrane symptoms and have carefully questioned my patients as to whether their deafness has been progressive or stationary in recent years. In the progressive cases I found there were always atrophic disorders, whereas in the stationary cases conditions were always practically normal.

In this connection I would again point out that GRAY sometimes found increased secretion of cerumen again among his cases of improved hearing after tyroxin treatment.

GRAY brought about in his otosclerosis patients a temporarily improved hearing (about 2 min.) by means of inhalation of amyl nitrite, causing strong dilatation of the arteries. He says that all his otosclerosis patients showed this phenomenon more or less. I have tried this experiment with amyl nitrite myself and since the results are of importance in connection with the foregoing I mention them here.

It is not always easy to make an audiometric test after the inhalation of amyl nitrite because there is generally strong tinnitus and some patients do not feel at all well. I first experimented on 10 persons with normal hearing and was unable to detect any improvement

at all in any case. I had exactly the same results with 7 patients suffering from internal ear deafness. Neither could I find any such effect in every one of the otosclerosis patients.

Let us first classify the tympanic membrane and auditory canal pictures, viz:

Type I Practically normal tympanic membrane, narrow auditory canal, possibly some cerumen.

Type II Moderate atrophy of tympanic membrane and auditory canal.

Type III Transparent atrophic tympanic membrane, wide atrophic auditory canal.

Improvement of hearing after inhalation of amyl nitrite:

	None	slight	good (± 10—15 decibels)
Type I	4	1	0 patients
Type II	1	6	2 „
Type III	0	2	3 „

From this table it appears that the greatest improvement of hearing occurs with type III cases, thus where the process has been most progressive. With type I, on the other hand, we find practically no improvement of hearing, and these are cases where the process has been stationary for a number of years.

I think the explanation of this is to be found in what has been said in the foregoing pages. In the cases of type III there is a pronounced defect in the vasomotor reflex, with a gradual transition via type II to type I, where we have a vasomotor reflex almost normal, and thus a fairly normal blood supply to the ear, as compared with a most unsatisfactory supply in type III.

For the cases of type III I would classify the nerve cells of the organ of hearing as follows:

a. those still functioning normally;

b. those whose functions can be immediately improved by better nutrition (transient hyperaemia such as brought about with amyl nitrite);

c. those cells which can be caused to function better after some time, through better blood supply;

d. cells which are absolutely dead.

Among the cases of type I we find other conditions because there the blood supply is functioning much better. There we have the true stapes ankylosis and, further, only the cells of class *a* and *d*, the latter having become moribund in the period when the process was progressive and thus the patient had a bad vasomotor reflex. Cells of the classes *b* and *c* are absent or at most scarcely present owing to a good blood supply, and it is just these cells which in the experiments with amyl nitrite bring about an improvement in hearing.

The stationary cases of otosclerosis are not directly to be considered for operative treatment. Should such a case be operated upon, and given that the foregoing considerations are correct, then one would hardly expect any improvement of hearing in the other ear after the operation. [1])

I would recall in this connection the case of Mrs. H. described under "Otosclerosis paradoxica" (case 5). As regards the conditions of the tympanic membrane and auditory canal this patient belongs to type II. During the time that her hearing showed improvement the inhalation of amyl nitrite gave no further improvement. At the time of the examination on 1st March 1947, when her hearing had again diminished, amyl nitrite produced an improvement in hearing in the speech zone from 6 to 10 decibels. On 27th February 1947 there was apparently a hyperaemia of the ear which had brought about a better functioning of the cells classified under *b* and *c* — note also the improved bone conduction. The vascular dilatation caused by the inhalation of amyl nitrite had at that time no longer any effect upon the already improved state of the nerve cells. On 1st March, however, the hyperaemia had apparently disappeared and the nerve cells were again in a state as described under *a, b, c* and *d*; the inhalation of amyl nitrite then did, indeed, have its effect and there was a slight improvement in hearing.

The results attained in this case with amyl nitrite would seem to support my theory. On 27th February 1947 this patient showed signs of a stronger vascular reflex of the tympanic membrane than on 1st March.

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[1]) At the time of going to press I am able to report having recently operated successfully on the L. ear of Mrs. L. v. K. (see p. 53). There is, however, no improvement in the R. ear, which goes to confirm my theory.

With stationary cases of otosclerosis tinnitus occurs sporadically. This is quite comprehensible, because according to GRAY tinnitus is caused by degeneration of the nerve cells. As we have just seen, degenerated cells are hardly ever found to exist in stationary cases, and this would therefore account for the sporadic occurrence of tinnitus among these patients.

It is a known fact, as also reported by NAGER, that otosclerosis is often slowly progressive. In many cases stationary periods alternate with progressive periods, whilst in other cases there is a constant stationary condition (NAGER found 60 such cases out of 1146).

From the foregoing I therefore draw the following conclusions:

1. Otosclerosis is based upon a defective vasomotor reflex of the entire organ of hearing.

2. This vasomotor reflex may sometimes be restored wholly or partly, thereby rendering the deafness stationary.

3. There is not, therefore, always a permanent defect in the vasomotor reflex, as is concluded by GRAY.

CONCERNING THE RESULT OF THE OPERATION.

There is a great improvement in hearing immediately after the operation.

HOLMGREN showed how this improvement took place directly after opening the horizontal canal. As already remarked, he attributed the acute improvement in hearing for a large part to the relief of the elevated intralabyrinthine pressure.

KATZ holds the opinion that vascular defects play an important role in otosclerosis. He believes that the immediate improvement in hearing following the operation is due to a suction action set up by the flow from the labyrinth; the greatly improved hearing could only continue to exist so long as the perilymph is running.

In my opinion the acute improvement of hearing depends for a large part upon vasomotor reflexes, the factors that count being the following:

1. The perilymph, being mobilised again, forms a great stimulant for the vasomotor system, because the sound waves are better able to penetrate into the cochlea.

2. The post-operative hyperaemia resulting from the operative trauma. This is illustrated in the case of the patient upon whom I performed only antrotomy.

3. Possibly also of importance is the effect of the histamine-like substances (SHAMBAUGH) formed in the plastic flap during the first four days following the operation. It is not unlikely that some of these substances reach the labyrinth via the fistula and likewise cause hyperaemia.

CAMPBELL noticed, and I have found too, that the decided improvement in hearing obtained during the first few days usually returns after cessation of the post-operative labyrinthitis in a less degree. It is possible that this is due to the labyrinthitis, but it is certain, too, that after a time important stimuli for the vasomotor system also cease, namely the post-traumatic stimulus and that brought about by the action of the histamine.

The fact that vasomotor reflexes may exercise a strong influence

long after the operation has been proved in the examination of one of my patients four months after the operation. In the dry operative cavity was some cerumen that I had great difficulty in removing owing to the hyperaesthesia in the cavity. The auditory canal of the other ear however was rather insensitive. Immediately the tympanic membrane in the operative cavity was touched there was a strong vascular reaction, whereas in the non-operated ear there was hardly any reaction at all when the membrane was touched. I found the same phenomenon to exist post-operatively in several patients. My conclusion is, therefore, that the result of the operation depends not only upon the mobilisation of the perilymph but also for a large part upon the better blood supply.

The following case will show that the post-operatively improved vasomotor reflex may again be disturbed.

Six months ago I operated upon a patient (case No. 2) with excellent results, and right to this very day she has a decidedly positive fistula reaction. At the time of the operation she was not aware that she was pregnant. Hearing is now somewhat reduced in both ears, but in the operated ear less so than in the other (see figs. 52 and 53).

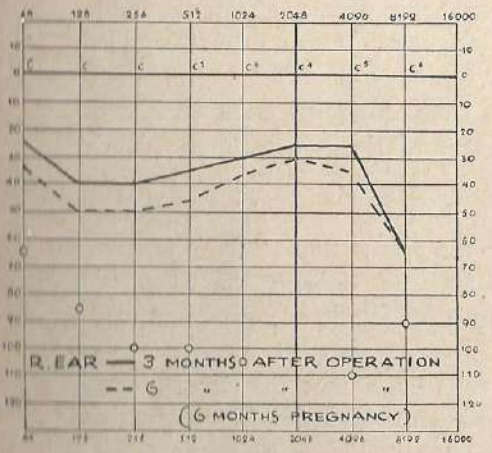

Fig. 52.

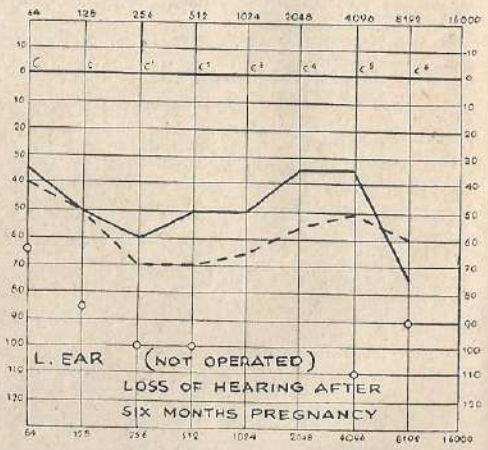

Fig. 53.

In a case of otosclerosis the open fistula is apparently the best weapon against vasomotor defects in pregnancy.

The frequent occurrence of aggravated deafness during pregnancy in otosclerosis cases is ascribed by GRAY to the influence exercised

by the genital system upon the vasomotor system under these conditions. The otosclerosis patient is more susceptible to such influences because there is already a defect in the vasomotor reflex.

In this connection it is also interesting to note that RAINISCH brought about an improvement in hearing for a few days in cases of different kinds of deafness by means of Pilocarpine injections. This effect was noticeably reduced in the case of women during menstruation.

SIMSON HALL and DAY found several cases among their patients operated for otosclerosis where the hearing in the operated ear did not decline during pregnancy.

By what process does the patient hear after the operation?

As we have seen in the discussion on otosclerosis paradoxica, for improved hearing in cases of otosclerosis it is not essential that the perilymph should be mobilised. It is at least to be supposed that in most cases of advanced otosclerosis the stapes will be fixed. In this connection it is very remarkable that five months after the operation KOPETZKY found in one of his patients that although the fistula was closed the hearing was still good.

Does the patient, then, hear via the new fenestra? What is the part played by the tympanic membrane and the round window?

In one of his first operations HOLMGREN applied a fistula in the superior vertical semicircular canal on the eminentia arcuata, thus inside the cranial fossa. The result was satisfactory, though only of short duration. In this case it can hardly be imagined that the sound stimuli penetrated this new fenestra inside the cranial fossa. The most obvious assumption, according to TULLIO, is that these stimuli enter via the round window.

LEMPERT and SHAMBAUGH observed reduced hearing after the operation when the tympanic membrane had been damaged, but as soon as the tear had healed the hearing improved.

After laying a piece of cotton wool with sterilized vaseline on the tympanic membrane in the operative cavity I found in several cases a loss of 10—15 decibels in hearing.

In these last two examples it is a question, however, in how far the function of the round window was obstructed.

Is the tympanic membrane in communication with the applied

fistula, and are the sound waves communicated from the tympanic membrane to the fistula via the plastic flap, as SHAMBAUGH and SOURDILLE believe to be the case? Opposed to this are the results obtained by KOPETZKY. He very successfully applied a fistula in an old dry radical cavity, where there was no tympanic membrane. In another case, that of an otosclerosis patient, where he applied a fistula but the plastic flap did not hold, the fistula remained open and the result, as far as hearing was concerned, was very good. In these cases, therefore, there was a good improvement in hearing without any communication between the fistula and the tympanic membrane.

Several operations performed by HOLMGREN, where he covered the fistula with Thiersch grafts, also yielded good results.

CAMPBELL reports as one of his greatest successes a case where the plastic flap was torn and laid loosely on the fistula.

Thus also in these cases there was no communication between the tympanic membrane and the fistula.

What seems to be most probable is that the sound waves penetrate the round window and that the applied fistula serves as an expansion outlet for the perilymph.

I will briefly quote here the results reported by various operators.

SIMSON HALL considers his cases to be a success if there is an improvement in hearing of at least 15 decibels. He has had most successes with young people: 80 % under 19, 77 % between 19 and 24, 70 % between 24 and 29, 62 % between 29 and 34, 61 % between 34 and 39, and above that about 50 %.

LEMPERT operated in 1000 cases, of which 815 were diagnosed before the operation as cases of otosclerosis. In 571 cases there was such an improvement in hearing that they were socially and economically absolutely restored.

In 92 cases there was partial improvement. In 18 cases there was no improvement whilst in 34 cases hearing was worse. Of 185 cases with otosclerosis showing reduced bone conduction, he achieved a very good result in 39 cases, no improvement in 93 and reduced hearing in 11 cases. Of 309 patients operated more than 3 years ago, 187 retained a very good improvement in hearing, 37 a fair improvement, whilst in 65 cases there was no improvement and 20 were harder of hearing than before the operation.

SHAMBAUGH reports 88 % successful cases with a gain averaging 28.9 decibels during 2 years or more.

DAY performed 100 operations and found a very good improvement in hearing in 80 cases.

SULLIVAN performed 299 operations. In 246 cases there was a very good improvement, in 17 cases partial improvement, in 21 cases the hearing was as before and in the other 15 cases there was a loss in hearing; all these operations had been performed more than a year before.

GREENFIELD obtained in 21 cases, out of a total of 28, a good improvement, in 4 cases fair improvement and in 1 case none at all, whilst one patient's hearing was worse and another lost all sense of hearing.

INDICATIONS FOR THE OPERATION.

To arrive at a diagnosis in a case of otosclerosis, and thus also to judge the indications for the operation, it is often necessary to exclude the possibility of an adhesive process. The tympanic membrane does not always give a sufficiently clear picture to be able to decide this point, and for that reason it is advisable to make an X-ray photograph. Where an adhesive process exists one usually finds arrested pneumatization, whereas in the case of otosclerosis as a rule there is good mastoid pneumatization.

SHAMBAUGH reports, for his successful cases, an average improvement in hearing of 28.9 decibels, LEMPERT 24.3 decibels and CAMPBELL 21.9 decibels. Generally speaking, the result after successful operations is such that the audiogram is roughly parallel to that taken before the operation. Consequently it is possible to predict roughly what the hearing is likely to be after the operation. Assuming for the moment that an improvement of 25 decibels is to be expected in the speech zone, then it is obvious to assume that if a patient registers a loss of hearing of the order of 50 decibels prior to the operation this will be reduced to a level of about 25 decibels. For all practical purposes this is worth more than operating on a case to reduce the loss in hearing from a level of say 75 to one of 50 decibels, for such a patient would still be unable to hear whispering at a distance of a few metres. Should this consideration, however, constitute a reason for not operating? In my opinion an operation should be worth while even in such relatively adverse cases. As an instance I would mention my patient No. 7: loss in hearing in the speech zone prior to the operation was 70—90 decibels and after the operation 35—55 decibels, thus a gain of more than 25 but still leaving a loss in hearing of more than 30 decibels. And yet this patient is highly pleased with the result, as may be understood considering that whereas before she could only hear loud speech ad concham now she can hear whispering at a distance of 10 cm; before the operation with a hearing aid she could only just catch

what her husband was saying, but now she can converse with him without the hearing aid.

Admittedly such patients will often still have need of a hearing aid. As DAY observes, this is disappointing for the patient and cannot really be regarded as an operational success, and he believes, therefore, that such patients should not be operated. I, myself, however, think otherwise, especially if the operation should prove to have arrested the otosclerotic process.

CAWTHORNE does not operate in cases where the patient has previously had paracusis *Willisii* and can no longer manage with a hearing aid because the perception of sound is distorted. This would indicate a too severe lesion of the acoustic nerve. It happens that my case No. 7 is a patient who had had paracusis *Willisii* and could not use a hearing aid owing to distortion of sound.

WALSH and SILVERMAN find improved bone conduction in successful cases of fenestration operations, and for that reason do not attach much value to bone conduction as an indication for the operation.

LOWY fixed the columella of pigeons by filling the external auditory canal with bone wax, then making a fistula in the horizontal semicircular canal. He found no difference in bone conduction before and after the fenestration. In my opinion, however, an artificially created conductive deafness in a pigeon is not comparable with otosclerosis and a defective vasomotor reflex in the human being.

LEMPERT, in the beginning, did not think it worth while to operate in cases where the loss in bone conduction in the speech zone amounted to more than 30 decibels, but now he thinks otherwise. Bone conduction is often improved after the operation, so that it does not give a direct indication as to the condition of the acoustic nerve.

In this connection NASIELL remarks that a true stapes ankylosis may give perception deafness. The same has been observed by MARKMANN, BEKESY, CROWE, GUILD and SHAMBAUGH. Therefore the state of bone conduction is not of such very great importance for the operation. In regard to this I would once more call attention to the case of my patient No. 7, who showed a loss of 60 decibels for c4 in bone conduction.

In many cases the improved vasomotor reflex after the operation is apparently capable of restoring for a large part the acoustic nerve that had seemingly lost its function.

Generally speaking, the operation yields the best results with young people (see SIMSON HALL's analysis of results according to age). These are the cases where the loss of hearing is not usually so great and the acoustic nerve has not yet suffered any considerable degenerative changes.

The fact that improved hearing is possible in all cases of true otosclerosis has been established in the first days after the operation, when all my patients showed an improvement, even in those cases where prior to the operation there was unsatisfactory bone conduction. In my opinion, therefore, it is due to the still imperfect technique that this improvement in hearing is not maintained in all cases.

The operation means for the cochlea a large trauma, as is evident from the post-operative reactions. It need cause no surprise that the least satisfactory results are obtained in those cases where a severe degeneration of the acoustic nerve is to be expected. The defective nerve cells will be less able to bear this trauma than the healthy cells.

Possible exceptions may be those cases where considerable structural changes have taken place in the cochlea as a result of the otosclerotic process; but this is difficult to diagnose before the operation.

Another point of importance for the indication and the prognosis is the retardation of the otosclerotic process after the operation. Interruption of the circulus vitiosus, which I described when dealing with the improved hearing in the non-operated ear, is apparently a means of arresting the process. Various operators arrive at the conclusion that the operation arrests the otosclerotic process — I would mention here LEMPERT, HOLMGREN, SOURDILLE, G. PASSE, FRASER and FREMEL. When operators with such vast experience as HOLMGREN, SOURDILLE and LEMPERT express such an opinion then there is all the more reason to widen the scope of indications for the operation.

Summarizing, we therefore find that:

1. the greatest successes are attained with young persons;
2. bone conduction is not a decisive criterion for the state of the acoustic nerve;
3. in successful cases bone conduction is improved after the operation;
4. presumably the operation arrests the process.

The indication for the operation can only be extended if the principle is maintained that only the worst ear is to be operated and

that the other ear should never be operated until sufficient time has elapsed to show whether the improvement in the operated ear is permanent. It can never be justified, in my opinion, to perform the operation on both sides at once, because there is always the possibility that instead of an improvement there may be an aggravation or even a total loss of hearing after the operation. Fortunately the number of such cases is very small. Anyhow the patient should be told in advance of the possible favourable or adverse consequences of the operation. Another point to be considered is whether in a particular case there is any benefit in using a hearing aid or whether it has its disadvantages.

NEW METHOD OF TREATMENT.

On the grounds of pathological changes in the bone, NAGER and M. MEYER conclude that otosclerosis is to be counted among the osteodistrophia fibrosa. Its aetiology, however, is not clear to them. NAGER believes that inflammation in the vascular wall may be the cause of the bone changes taking place in otosclerosis.

WITTMAACK is inclined to ascribe the cause to a congestion and maybe even a back-flow of the blood in the veins of the ear. He believes to have established this from experiments with fowls.

From what has been stated in the foregoing pages, however, there seems no doubt that vasomotor changes must play a part. In a certain sense this is in agreement with the opinions of WITTMAACK and NAGER, for they, too, seek the cause in changes in the blood supply.

In view of the defect in the vasomotor reflex I am inclined to place otosclerosis in the same group of diseases as the disease of BUERGER and that of RAYNAUD, thus among the group of peripheral vascular diseases.

Consequently the therapy for otosclerosis should be analogous to that applied for the peripheral vascular diseases just mentioned. The object, therefore, is to bring about a permanent hyperaemia of the organ of hearing.

I will briefly review the arterial blood supply system and the vegetative nervous system.

Arterial blood supply system.

The cartilaginous part of the external auditory canal is supplied by the arteria temporalis superficialis and the arteria auricularis posterior. The bony part of the external auditory canal is supplied by the arteria tympanica and the arteria auricularis profunda, the latter being a branch of the arteria maxillaris interna.

The middle ear is fed by the arteria stylomastoidea (a branch of the arteria auricularis posterior), the arteria tympanica (a branch of the arteria maxillaris interna), a branch of the arteria meningea media, the arteria pharyngea (a branch of the arteria carotis externa) and the arteria carotico-tympanica (a branch of the arteria carotis interna).

The bony cochlea is fed by some branches of the arteria stylomastoidea and some anastomotic vessels leading from the cavum tympani to the vestibulum through the intermediate wall.

The membranous labyrinth is supplied by the arteria auditiva interna, a branch of the arteria basilaris, reaching the internal ear via the internal auditory canal, at the bottom of which it splits up into some 20 branches passing through the apertures of the tractus spinalis forminosus into the modeolus.

The vegetative nervous system of the organ of hearing.

The cervical sympathicus consists of three ganglia: the ganglion cervicale superior, the ganglion cervicale mediale and the ganglion cervicale inferior or stellatum.

From the ganglion stellatum there extend the sympathic fibres for the plexus brachialis and a number of vascular branches, the most important of which is the one leading to the arteria vertebralis. The fibres which together with the two arteriae vertebralis penetrate into the cranial fossa join up around the arteria basilaris. From there fibres lead to the two labyrinths.

From the ganglion cervicale superior sympathic fibres lead to the arteria carotis communis. Together with the arteria carotis interna, these sympathic fibres penetrate into the cranial fossa. The fibres of these two arteriae carotis converge in a plexus around the circulus arteriosus *Willisii*. This sympathic plexus joins up with that of the arteria basilaris.

From the plexus around the arteria carotis interna the nervus carotico-tympanicus leads through the front wall of the tympanic cavity to the promontorium, where it communicates with the nervus *Jacobsoni* and a branch of the nervus *Vidianus* and also branches leading to the third, fourth and sixth brain nerves.

The sympathic nerve plexus on the promontorium is called the plexus tympanicus. Sympathic fibres from this plexus supply the vessels of the middle ear.

Parasympathic fibres for the organ of hearing probably run in the nervus acusticus (*Racine*).

As regards the surgical treatment of the peripheral vascular defects (diseases of *Raynaud* and *Buerger*), periarterial sympathectomy and

removal of the sympathic ganglia are indicated for vasodilatation. In order to bring about hyperaemia of the organ of hearing it is therefore necessary to remove the cervical ganglia or to perform a periarterial sympathectomy of the arteria carotis.

These operations have already been performed and described, but not with the object of improving the hearing in cases of otosclerosis.

LANNOIS examined patients who had been operated by JABAULEY for *Basedow*'s disease and by whom the ganglion cervicale superior had been removed. He found on the operated side hyperaemia of the tympanic membrane and hyperacusis.

In 1924 RUBARSCHOW resected the ganglion stellatum on the left side of a patient with otosclerosis because of severe tinnitus. The tinnitus disappeared temporarily and there was even a perceptible improvement of hearing on the operated side. The reason why the success of that operation was only of a temporary nature is probably that the sympathic fibres on the other side took over the function from the operated side via the plexus around the circulus arteriosus *Willisii*.

According to MONTANDON extirpation of the ganglion cervicale inferior has the following results:

1. disappearance of the pilomotor reflexes of head, neck, upper half of the thorax and arm;
2. suppressed perspiration in the same part of the body;
3. a *Claude Bernard–Horner* syndrome of shorter duration than when the ganglion cervicale superior is extirpated;
4. vasomotor symptoms in four phases:

a. vasoconstriction, the first one or two hours after the operation;

b. hyperaemia of face, ears, mucous membrane of the nose and conjunctiva, and hyperthermia of one half of the face due to paralysis of the vasoconstrictors, this phase lasting several weeks;

c. a phase of latent compensation, where the symptoms can be revived by a cold or warm stimulus, with a stronger reaction on the operated side;

d. a final phase where the vasomotor reflexes in the respective region are suppressed by remote stimuli.

Excision of the ganglion cervicale superior results in the following:

Arresting of the perspirative and pilomotor reactions of head and neck, a *Claude Bernard–Horner* syndrome and a pronounced vaso-

dilatation of the face, conjunctiva, ear, mucous membranes of the nose and mouth, the meninges and the cortex ceribri. On the paralysed side these symptoms ultimately become latent, whilst the eye symptoms diminish.

PORTMANN and TERRACOL performed periarterial sympathectomy of the arteria carotis and came to the following conclusions in regard to the organ of hearing: Periarterial sympathectomy of the arteria carotis communis yields an improvement in hearing on the operated side, hyperaemia of the tympanic membrane and a hypotension of the inner ear with reduced irritability of the vestibular organ. (According to them, vegetative hypertonia and hypertension of the inner ear cause hyper-irritability of the vestibular organ and hypacusis.)

Periarterial sympathectomy of the arteria carotis communis and of the arteria carotis externa brings about a still more pronounced hyperaemia.

Periarterial sympathectomy of the arteria carotis interna produces less hyperaemia in the middle ear, but on the other hand the gain in hearing and the vestibular symptoms are the same.

PORTMANN, in cooperation with DESPONS and MAISONOBE, performed operations on the cervical ganglia of patients affected with vestibular defects. They arrived at the same conclusion as MONTANDON and found also hyperacusis and reduced irritability of the labyrinth on the operated side; in these cases of sympathectomy on one side the two symptoms last mentioned lasted about fifteen days.

In my opinion, in order to produce a prolonged hyperaemia of the organ of hearing it is desirable to perform sympathectomy on both sides. The fact that the labyrinth is innervated by the vegetative system on both sides seems to me to be borne out by the results obtained by PORTMANN and TERRACOL. The hyperacusis lasted only about fourteen days and in cases of one-sided periarterial sympathectomy PORTMANN found reduced irritability of both labyrinths, though more pronounced on the operated side. These two phenomena can very well be explained by the bilateral innervation.

If, as we have seen, hyperacusis occurs with persons of normal hearing, then we can certainly expect an improvement in hearing of otosclerosis patients when the cervical sympathicus is treated bilater-

ally. Both GRAY's experiments and mine with amyl nitrite have proved that owing to the creation of hyperaemia in the organ of hearing a more pronounced improvement is obtained with otosclerosis patients than with normal persons or patients with labyrinthine deafness.

A drawback attaching to the removal of the cervical ganglia is the occurrence of the *Claude Bernard–Horner* syndrome, but this treatment should nevertheless be considered, because fenestration, though technically a very neat operation, involves much mutilation of the organ of hearing and there is always some risk for the patient as regards his sense of hearing.

Good results may be attainable also with other treatments applied for peripheral vascular diseases, such as the intravenous injection of hypertonic saline solutions (BAX) or typhoid vaccine.

We have seen that the sympathic fibres for the arteries of the middle ear and the bony capsule of the cochlea converge in the plexus tympanicus on the promontorium. The severance of these sympathic fibres will therefore result in a dilatation of the arteries supplying blood to the bony labyrinth through their branches. Consequently any improvement in hearing is hardly to be expected by this means. Vascular communications between the middle ear and the cochlea are very few in number, so that this treatment is not likely to bring about hyperaemia in the cochlea. What we can hope for is that no further structural changes will take place in the bony labyrinth, and if this treatment is applied in the early stages it might perhaps prevent further fixation of the stapes.

The advantage of severance of these sympathic fibres as compared with sympathectomy lies in the local hyperaemia, in contrast with the hyperaemia of the entire head with the *Claude Bernard–Horner* syndrome. Moreover, the latter operation has to be performed bilaterally if any success is to be attained.

Resection of the plexus on the promontorium is a very small operation but it has the disadvantage that it does not lead to dilatation of the arteria auditiva interna. The technique of this treatment is not so difficult and can best be performed under a light narcosis. LEMPERT has already applied this treatment and described the technique; he calls it the "tympano-sympathectomy". This, however, he

applied in cases of various kinds of deafness where there was trouble from tinnitus, because he believes that the cause of tinnitus is an inflammation in the plexus tympanicus. After sterilization of the auditory canal an incision is made in the skin of the auditory canal at a distance of about 2 mm parallel to the lower half of the tympanic membrane. The skin is then lifted with a fine raspatory down to the tympanic membrane and the annulus tympanicus is loosened. The lower half of the tympanic membrane is then folded back upwards, exposing the plexus tympanicus, which is clearly visible under magnifying glasses. The plexus tympanicus is then resected with a fine raspatory and the tympanic membrane replaced in its original position. The auditory canal is loosely plugged. Should the auditory canal appear to be too narrow for a large ear speculum it can be enlarged at the beginning of the operation by making an incision in the soft parts of the upper wall.

I have applied this treatment to some patients but the hearing remained the same in all cases; the tympanic membrane healed without any infection.

It is not yet possible to judge the results of this treatment.

SUMMARY.

After a review of the literature on the operative treatment of otosclerosis, a description is given of the technique of the operation, the instrumentarium required, the post-operative treatment and the post-operative complications.

As part of the instrumentarium, a combined suction and flushing apparatus is described which may be very useful also for other ear operations.

An account is given of twelve operational cases and the results thereof several months after the operation.

By means of audiograms it is demonstrated that there is a slight improvement in hearing also in the non-operated ear. This is explained with the help of GRAY's theory. The gain in hearing probably arises from improved blood supply due to a stimulation of the vasomotor reflex. In most cases there was evidence of a stronger vascular reflex of the tympanic membrane of the non-operated ear.

A number of cases of otosclerosis paradoxica are described, in one of which it was possible to take a detailed audiometric recording.

The histories are given of a number of cases where the otosclerosis had not been progressive in recent years. These patients were found to have a good vascular reflex of the tympanic membrane, whilst there were no symptoms of atrophy of the auditory canal or the tympanic membrane, such as is found with cases of progressive otosclerosis. This would indicate that in some cases of otosclerosis it is apparently possible that the vasomotor reflex is wholly or partly restored, so that the deafness becomes stationary.

The immediate results of the operation and the indications are discussed.

Finally the possibility is considered of improving the perception of sound by means of hyperaemia of the organ of hearing, such in view of the important part apparently played by vasomotor defects in otosclerosis. In connection herewith, the various operations performed on the cervical sympathicus are discussed.

SAMENVATTING.

Na een literatuuroverzicht der otosclerose-operatie, volgt de beschrijving van de techniek hiervan, het benoodigde instrumentarium, de nabehandeling en de postoperatieve complicaties.

Bij het instrumentarium wordt een zuig- en spoel-apparaat beschreven, dat ook bij andere ooroperaties van groot nut kan zijn.

Twaalf operatie-gevallen worden beschreven met vermelding van de resultaten enkele maanden na de operatie.

Met audiogrammen wordt aangetoond, dat ook in het niet-geopereerde oor een lichte verbetering van het gehoor optreedt. Dit verschijnsel wordt verklaard met behulp van de theorie van GRAY. De verbetering van het gehoor berust waarschijnlijk op een betere bloedvoorziening tengevolge van een versterkte vasomotorische reflex. Een verbeterde vaatreflex van het trommelvlies van het niet-geopereerde oor kon in de meeste gevallen worden vastgesteld.

Een aantal gevallen van otosclerosis paradoxus wordt beschreven, waarbij in één geval het gehoor nauwkeurig kon worden opgenomen.

Een aantal patienten wordt beschreven, waarbij de otosclerose de laatste jaren geen progressief verloop had gehad. Bij hen werd een goede vaatreflex van het trommelvlies gevonden, terwijl geen atrophie van de gehoorgang of het trommelvlies werd waargenomen, in tegenstelling met de progressieve gevallen van otosclerose. Dit zou er op wijzen, dat het defect in de vasomotorische reflex bij otosclerose (GRAY) zich in sommige gevallen blijkbaar geheel of gedeeltelijk kan herstellen, waardoor de doofheid dus stationnair blijft.

Het directe resultaat der operatie en de indicatie wordt besproken.

Aan het slot wordt overwogen, om in verband met de vasomotorische stoornissen, die blijkbaar een groote rol spelen bij de otosclerose, door middel van een hyperaemie van het gehoororgaan een verbetering van het gehoor te verkrijgen. In verband hiermede worden de verschillende ingrepen aan de halssympathicus beschreven.

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STELLINGEN.

I.

Bij de scheelzienoperatie verdient de peesplooiing de voorkeur boven de peesopschuiving.

II.

Het gebruik van sulfapyridine en sulfathiazol is verre te verkiezen boven sulfaguanidine bij bacillaire dyssenterie.

III.

De vrije verkoop van gehoorapparaten dient verboden te worden.

IV.

Bij de vroege belasting na operatieve behandeling van de fractura colli femoris, is het gewenscht de breede pen volgens FELSENREICH te gebruiken.

V.

Bij de fenestratie-operatie biedt de endaurale methode geen voordeelen boven de postaurale methode.

VI.

Wanneer een haemorrhagische diathese een gevolg is van een essentieele idiopathische thrombocytopenie, die niet spontaan geneest of aanleiding geeft tot levensgevaarlijke bloedingen, is miltexstirpatie de eenige juiste therapie.

VII.

Macrocytaire anaemieën, die gepaard gaan met neurologische afwijkingen, behandele men niet met folinezuur, maar met leverextract.

VIII.

Na de mastoidoperatie volgens SCHWARTZE is de bijna totale sluiting van de wond te verkiezen boven het geheel openlaten hiervan.

IX.

De epilepsie is geen ziekte-eenheid, doch een syndroom.

X.

In de chronische stadia na schedeltraumata, bestaat geen correlatie tusschen de mate van liquorverandering en de ernst van het organisch-psychisch syndroom.