case of congestive heart failure with a blood pressure of 140/0 and an abnormal electrocardiogram showing no specific pattern. Since aortic incompetence was not present the diagnosis was one of high-output failure with a collapsing pulse which was not due to an aortic lesion. Clinical and laboratory findings ruled out hyperthyroidism and anaemia (radio-iodine uptake and metabolic rate were normal, there were 4,8000,000 r.b.cs. per ml and 92% Hgb) and we were therefore left with the following possibilities of high output failure:

1. A—V fistula; 2. Severe liver failure; 3. Beri-beri heart disease. At this stage the following investigations were carried out: skeletal X-ray survey, radio-iodine uptake, metabolic rate estimation, liver function tests, electrophoretic pattern study, serum cholesterol, serum electrolytes, serum calcium and phosphorous estimation, alkaline phosphatase, transaminase and haemochromatosis tests all being normal. Wassermann test was negative. Pyruvic acid estimation showed an abnormal rise after glucose: 25 mgm, normal being 1 mgm. Blood urea was 10 mgm. per 100 ml.

A diagnosis of beri-beri heart disease was made and the patient was treated accordingly. He was put on vitamin B^1 100 mgm by intramuscular injection three times daily for one week, followed by 150 mgm. orally daily. He was also given Digoxin 0.25 mgm. twice daily and 100 mgm. hydrochlorticoside weekly.

After one month's treatment the patient felt much better, was able to return to work, did not get out of breath, the murmurs disappeared and his blood pressure was 130/85. His blood urea had risen to 30 mgm. per 100 ml. The electrocardiogram now showed a normal T-wave and higher voltage of the QRS complex.

Mechanism of heart failure in Vit. B^1 deficiency: Since B^1 is the co-enzyme of carboxylase and is required for normal carbohydrate metabolism and utilisation, its absence renders the heart muscle unable to utilise lactate pyruvate normally. This leads to diminution of myocardial O^2 extraction, insufficient energy production and functional failure. In addition there is an accumulation of the vasodilating intermediate catabolites of glucose (pyruvic, lactic and other keto-acids) which cause widespread peripheral arterioral dilatation. This acts like a large arterio-venous fistula augmenting the venous return to the right ventricle and making demands upon the left ventricle for increased output, thus putting a burden on a heart already working at a disadvantage (Konstam G. & Sinclair H. M. 1940). Cardiac catheterisation in a patient with beri-beri heart shows an average cardiac output of 16 litres per minute (normal 4—7 litres) and an O^2 consumption of 355 ml/m (normal average 760 ml/m). Alcoholics who take more of their calories in the form of alcohol (which requires B^1 for its metabolism) and do not eat enough B^1 containing foods, eventually develop heart failure through the mechanism described above.

I would like to thank Dr. R. I. S. Bayliss M.D., F.R.C.P., Physician in the Westminster Hospital, London, for the biochemical tests carried out in the investigation of this case.

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STAPEDECTOMY WITH A SPRING PROSTHESIS

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In deafness due to otosclerosis the footplate of the stapes becomes ankylosed to the niche of the oval window in such a way that the stapedovestibular joint progressively disappears.

The treatment for this form of deafness is surgical. The stapes is removed and the gap between the incus and the oval window is bridged by means of a prosthesis. The operation is called a stapedectomy and the prosthesis may be either a polythelene tube or Teflon. The oval window is usually closed by means of a vein graft ,but Prof. H. Schuknecht uses as his prosthesis a plug of fat taken from the lobe of the ear (which occludes the oval window) and stainless steel wire which is attached to the incus. Another

method that is nowadays used is the Teflon rod or the McGee piston, both of which are attached to the incus and go down into the labyrinth through a hole bored in the stapes footplate.

Results from these various techniques are usually good, but they all carry a risk of sensory-neural changes occuring in the labyrinth which come on suddenly days, weeks or months after a successful operation. The patient hears a rushing noise in the ear and his hearing deteriorates rapidly until a "dead" labyrinth develops. Vertigo may or may not be complained of.

This problem has worried otologists since stapedectomy came into general use and no specific reason can be adduced to explain it.

However two important factors have to be taken into consideration because they may be responsible for this tragedy.

1. Before removing the stapes, the stapedius muscle, which is attached by its tendon to the neck of the stapes, is cut. It is known that one of the roles of the muscles of the middle ear is to protect the internal ear from excessive stimulation. Whenever the ear is subjected to a loud noise, the tympanic muscles contract and the strength of the stimulus reaching the labyrinth is reduced.

In stapedectomy with prosthesis, this damping effect of the muscle is lost and the artificial stapes is free to "pump" the labyrinth strongly or weakly according to the strength of the stimulus. Excessive "pumping" action by a prosthesis may be one of the factors which produce sensory-neural changes in the labyrinth.

2. Another consideration is a leak of perilymph which occurs after all forms of stapedectomy techniques. This has been proved by injecting radio-opaque substances immediately after a stapedectomy into the spinal column and taking serial X-rays of the middle ear. The radio-opaque substance was almost invariably shown in the middle ear. This was found to be most abundant when a Teflon rod or a McGee piston was used, and least when the oval window was closed by means of a vein graft (J. Shea, personal communication).

In an endeavour to minimise the above factors, I developed in March 1965 a stapedectomy technique which has so far proved very satisfactory.

After removing the stapes, the oval window is closed by means of a vein graft taken from the dorsum of the foot. The gap between incus and vein is bridged by means of a spring made of stainless steel wire which could, in theory, attenuate a loud stimulus.

This spring prosthesis has now been used in over 40 cases in Malta and, except for one case which showed no improvement, all the others show marked improvement, normal or above-normal hearing being the rule. No "dead" labyrinths have so far occurred.

The spring prosthesis is also being used in the United Kingdom and in the United States of America, and, in fact, it is being produced commercially by a well known firm of surgical manufacturers.

Reports from various centres show that the spring prosthesis does all that is claimed for it, and what is more important, is that to date no sensory-neural changes in the labyrinth have been observed following its use.

Summary

The causes of sensory-neural changes in the labyrinth following stapedectomy operations are discussed. A new technique is described and its advantages outlined.

OCULAR TOXOPLASMOSIS: A report on a case discovered in Malta

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Before the discovery of toxoplasmic retino-choroiditis, all cases of fundus diseases in infants and young children were diagnosed as foetal chorioretinal infection of unknown origin, or congenital developmental anomaly of the retina and choroid