MENIERE’S DISEASE
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Meniere’s disease is characterised by attacks of vertigo, associated with deafness (unilateral in 80% cases), tinnitus and vomiting. The attacks tend to appear in groups with intervals of complete freedom. Vertigo is typically of rotational type; as it wears off deafness becomes prominent and tinnitus may be distressing. There is distortion rather than loss of hearing, usually unilateral. Otoscopy reveals normal tympanic membrane. Deafness, if present will be of perceptive type. Tuning fork test will reveal positive Rinne and Weber lateralised to the better ear. Pure tone audiometry shows greater loss of hearing to low tones in the early stage. Recruitment phenomenon is usually demonstrable by loudness balance test. Speech audiometry measures percentage of words repeated correctly against the loudness of speech, i.e. intelligibility. Owing to distortion there is a limit to an increase in loudness which will facilitate understanding. Caloric test shows a reduced late response on the affected side.

Meniere’s must be differentiated from acoustic neuroma, labyrinthitis, vestibular neuronitis, positional vertigo and neurological lesions like cerebellar and brain stem lesions and disseminated sclerosis. In acoustic neuroma true vertigo is rare and recruitment phenomenon is absent. Late cases show widening of internal auditory meatus with bone destruction on the affected side. Romberg test is positive, and neurofibromatosis may be present elsewhere in the body. In labyrinthitis obvious lesions are present in the ear. In the remaining conditions deafness is not present.

The first thing in the management of Meniere’s is to give reassurance to the patient, and advise avoidance of excessive changes in the posture and movement of neck. Any septic focus present must be suitably dealt with.

As the aetiology of Meniere’s is not definitely known, treatment of a case is not definitive. However it should be treated first medically, and then surgically if necessary.

Medical treatment is aimed at controlling the symptoms through influencing the endolymph electrolyte and water balance, strial permeability and sensory end-organ activity.

Electrolyte water balance

The aim is to keep the body electrolytes at a reasonably constant level by low salt intake and restriction of fluids to 2½ pints per day. These measures can sometimes suppress attacks.

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Strial permeability

Sympathetic overactivity causes increased strial permeability due to ischaemia. To counteract this, nicotinic acid is given. The average dose is 100 mg three times a day.

Antihistamines have also been tried with good results; in these cases the hydrops formation is believed to have an allergic basis. The commonly used drugs are Dramamine, Avomine, (both contain antihistamine and theophylline), Diligan (contains antihistamine and nicotinic acid) and Stemulil.

Sensory end-organ activity

Dramamine and Avomine are labyrinth sedatives. They suppress vertigo. Their mode of action—whether they act directly upon end-organs or through the central nervous system—is not known.

General sedatives have also proved useful, particularly phenobarbitone.

SURGICAL TREATMENT is indicated when medical treatment fails, vertigo is disabling and distortion of hearing is intolerable.

Conservative Surgery attempts to modify the labyrinthine behaviour, without any effect on cochlear function. The attempts are directed towards control of endolymph formation, absorption and sensory nerve impulse.

Control of endolymph formation—This is based on an Ischaemic theory of hydrops formation; stellate ganglion block and cervical sympathectomy have been tried. These do not always control vertigo.

Control of endolymph absorption—This is done by decompression of the labyrinth by opening of succus endolymphaticus. The drawback is that the opening tends to close by fibrosis.

Recently, in a few cases, endolymph has been shunted into subarachnoid space through an indwelling silicone rubber tube.

Control of sensory nerve impulse—This is attained by the destruction of vestibular function without loss of hearing.—

1 Electrocoagulation of the lateral semicircular canal has been tried. It was not successful as the electrocoagulation was either inadequate for fear of cochlear destruction or when adequate, cochlea was also destroyed.

2 Recently, selective destruction of vestibular end-organs by means of an ultrasonic beam is being used with good results.

3 Intracranial division of vestibular portion of eighth cranial nerve has also been tried. This is a major neurosurgical procedure and is not without risks.

Radical surgical treatment is indicated when hearing in the affected ear is nil, symptoms are severe, and when the disease is unilateral. This is achieved either by total destruction of the affected labyrinth through injection of alcohol into it or by Membranous labyrinthectomy.