FIXED STAPES OR INTERRUPTED OSSICULAR CHAIN?

(AUDIOMETRIC DIFFERENTIAL DIAGNOSIS)

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SINCE the advent of microsurgery of the ear many otologists have found that some of the cases of conductive deafness, considered to be due to otosclerosis, have under the operating microscope proved to be instances of interrupted ossicular chain. When the audiograms of these patients are re-examined in retrospect, they show features which should have made the surgeon suspect, if not diagnose, the possibility of an interrupted ossicular chain behind the intact tympanic membrane *before* operation.

For the purpose of this paper the history of head injury or cortical mastoidectomy (dislocated incus), obvious congenital abnormalities (fusion of deformed ossicles) and the non-progressive character of the deafness are disregarded and an attempt is made to point out the characteristic differential diagnostic features in the audiogram of the two conditions.

The audiogram in cases of otosclerosis is familiar and has been discussed in detail by Woods, Carhart, Langenbeck and many others. Let us only state that it is characterized by an air-bone conduction gap, which varies in degree according to the stage of the disease, showing greater loss for air-conducted sound for the lower than for the higher frequencies and Carhart's notch in the bone conduction audiogram (Fig. 1). This notch consists of impairment of the bone conduction by 5 db. at 512 c.p.s., 10 db. at 1,024, 15 db. at 2,048 and 5 db. at 4,096 c.p.s. (compared with normal), and disappears following fenestration operation. The existence of this notch in otosclerosis is generally recognized and several theories have been suggested for its explanation. They will be discussed later.

The pure tone audiogram in cases of an interrupted ossicular chain behind an intact tympanic membrane has not received adequate attention. Surveying the literature the author found that only some of the writers considered it necessary to reproduce bone conduction audiograms and limited the published audiograms to registration of the air conduction only, before and after operation. A further difficulty is the lack of a standardized bone conduction vibrator used in different makes of audiometers and the difference in masking. In other published cases additional

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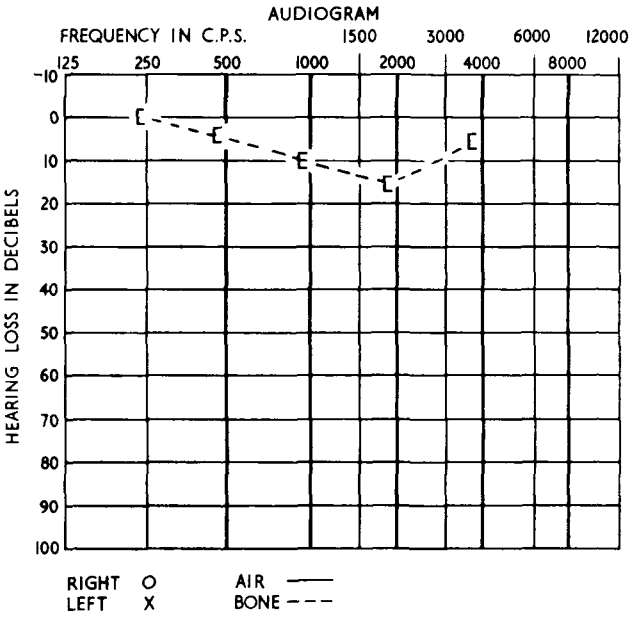


FIG. 1.
Carhart's notch.

abnormalities were found on tympanotomy, e.g. head of the malleus fixed in the attic, incudo-malleal ankylosis, etc., which make them unsuitable for comparison. Nevertheless a number of characteristic audiograms of cases of an interrupted ossicular chain behind an intact tympanic membrane have been collected and are reproduced in Fig. 2. These audiograms have the following characteristics:

1. The loss of hearing by air conduction is very severe, amounting to some 50 db. for the speech frequencies.
2. The bone conduction curve runs roughly parallel to the air conduction curve so that there is no marked or constant difference in the air-bone conduction gap for the low and high frequencies.
3. The Carhart notch in the bone conduction curve is absent.
4. The air-bone conduction gap is large (at least 40 db.).

What is the explanation of these findings?

As regards air conduction: If the ossicular chain is interrupted behind an intact tympanic membrane, the resulting deafness is more profound than in cases of radical mastoidectomy, in which in the absence of pressure transformation and isolation of the labyrinthine windows patients hear with their windows only (Zöllner's "Fensterhörer"). In cases of interrupted ossicular chain behind an intact tympanic membrane the hearing by air

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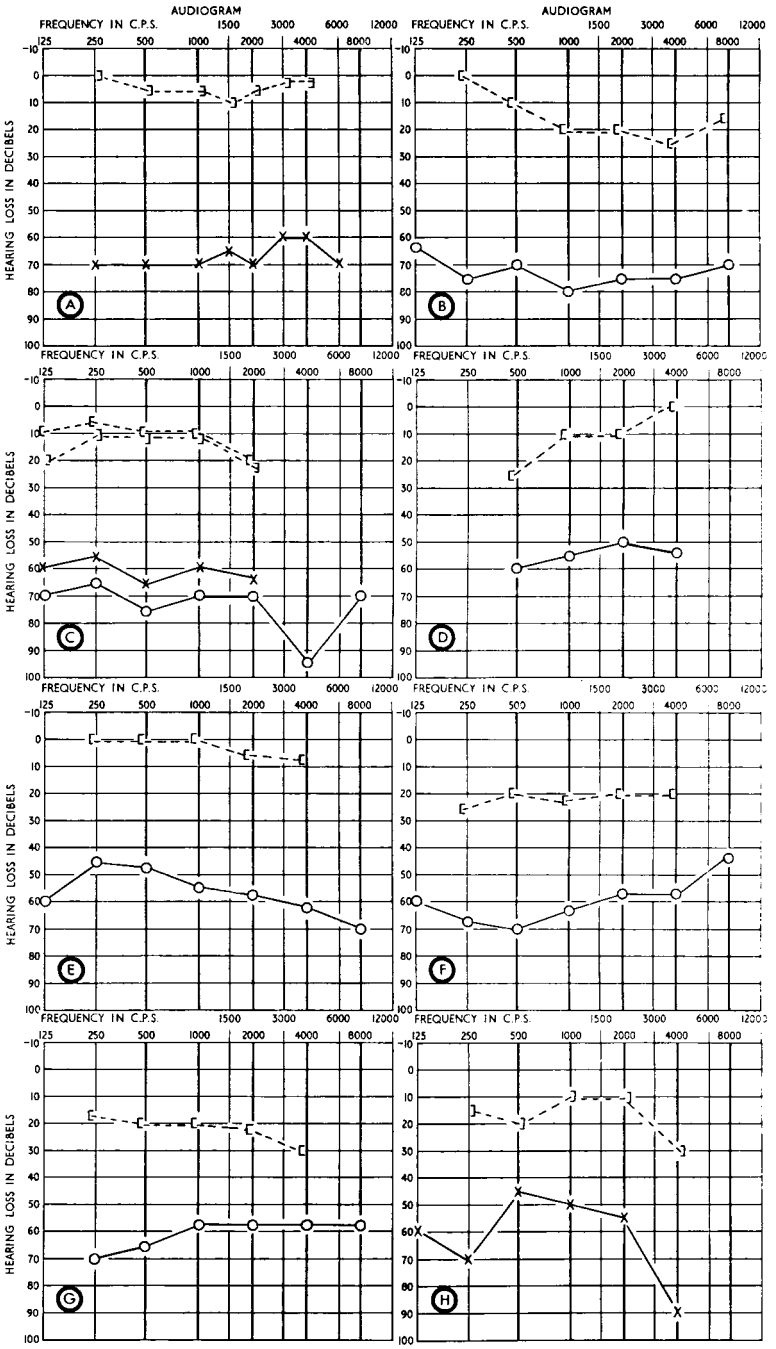


FIG. 2.

A. Dislocated incus (Anklesaria). B. Dislocated incus (Freeman). C. Bilateral dislocation of the incus (Gisselsson). D. Dislocated incus (Bauer). E. Absent lenticular process (Oppenheimer and Harrison). F. Erosion of the tip of the long process of the incus (Beales). G. Absence of the long process of incus (history of otitis media in childhood) (Beales). H. Head injury. Fracture of the postmeatal wall. Incus missing, stapes lying free (Beales).

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conduction is even worse, because here, in addition, the drum acts as an actual hindrance to sound reaching the windows by its absorption and reflection.

The parallel course of the air-bone conduction curves is due to the absence of Carhart's notch.

If we exclude from our consideration cases of active middle ear suppuration, because they do not present a differential diagnostic problem, Carhart's notch occurs in the following conditions: (1) Otosclerosis; (2) Fixation of stapes in tympanosclerosis; (3) Loading of the tympanic membrane (Allen and Fernández). Several explanations of the Carhart notch have been suggested and, as at present there is no universally accepted theory of bone conduction, we have to enumerate the main ones and consider, how the observed facts fit in with these theories. According to v. Békésy "clinical" bone conduction consists of three components:

1. "Compression bone conduction", due to deformation of the skull during vibrations, causing compression or dilatation of the labyrinth with displacement of fluid in the two scalae of the cochlea.
2. "Inertia bone conduction", due to inertia of the ossicular chain in relation to the skull, causing a relative movement between the stapes and the cochlea.
3. "Inertia of the mandible", causing deformation of the external auditory canal and thus pressure changes acting upon the tympanic membrane and transmitted to the inner ear by the ossicular chain in the normal way.

Kirikae considers inertia of the labyrinthine fluids in relation to the bony labyrinth an important factor in the mechanism of bone conduction.

Langenbeck also takes into consideration inertial forces producing movements of the skull relative to the brain: whilst the skull moves forward the brain lags behind, causing compression of contents in the posterior cerebral fossa. This compression is transmitted through the internal auditory meatus to the fluids of the cochlea.

Under pathological conditions, e.g. when the round window is closed by the otosclerotic process, additional factors affect bone conduction: acoustic leak of the scala tympani through the apertures of the cochlear aqueduct and the inferior cochlear vein (Groen and Hoogland).

What is the explanation of Carhart's notch in the light of the above theories?

Carhart himself explains the impairment of bone conduction in otosclerosis by fixation of the stapes, which "becomes loaded by the adjacent bony mass" and this "mass increase affects the impedance formula". Fenestration surgery approximately restores the mechanical constants.

Woods also considers that the bone conduction audiogram in otosclerosis can be explained by stapedial fixation, which prevents vibrations

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of the ossicular chain reaching the labyrinth and impedes the response of the end organ by diminishing mobility of the perilymph. Fenestration frees the perilymph and allows it to vibrate and stimulate the organ of Corti.

Kirikae is of the opinion that fixation of the stapes causes a decrease of the inertia of labyrinthine fluids and thus explains the decrease in bone conduction sensitivity and its improvement following fenestration.

Allen and Fernández believe, as the result of their experiments with drum loading by skin grafts, that the compressional element of bone conduction is the most important. They explain the Carhart notch, which is present in drum loading as well as in fixation of the stapes, by interference with the transmission of energy from bone to cochlear fluids.

According to Langenbeck, Carhart's notch is due to the absence of that part of the bone-conducted sound, which the inertia of the mandible contributes to "clinical" bone conduction. The fixed stapes prevents transmission of pressure changes generated in the external auditory meatus by the inertia of the mandible. This deficit is responsible for the deterioration of bone conduction.

Huizing thinks that under normal conditions vibration of the ossicles reinforces vibrations of the labyrinthine fluids. In fixation of the stapes this reinforcement is absent, causing Carhart's notch.

Now according to all the theories quoted, with the exception of Langenbeck's and Huizing's, in the absence of stapes fixation Carhart's notch would be absent. The audiograms in Fig. 2 show that this indeed happens in cases of interrupted ossicular chain behind an intact tympanic membrane, in which both labyrinthine windows are mobile.

Langenbeck's explanation is unlikely for the following reasons: In cases of interrupted ossicular chain the mandibular component of bone conduction does not reach the labyrinth either, and yet the Carhart notch is absent. If the bone conduction is impaired, the impairment does not take the shape of Carhart's notch and does not improve, when continuity of the chain is restored. It represents true cochlear involvement.

Further, Allen and Fernández have made measurements of bone conduction on a very interesting case, relevant to the present discussion: In a young man with clinically normal hearing, who has lost the ramus and the condyle of his mandible as a result of childhood osteomyelitis, Weber's test showed no lateralization. As the absence of the mandible should eliminate the mandibular component of bone conduction on that side, if Langenbeck's theory were correct, there should have been lateralization, actually to the side of the missing mandible. According to Langenbeck the mandibular component arrives at the cochlea later than the cranial component and therefore the phase of the remaining cranial component leads and causes lateralization. Thus it seems that from the clinical point of view the mandibular component of bone conduction is unimportant.

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Huizing's theory does not explain the observed facts either. If the ossicular chain is interrupted, its reinforcing influence on the vibration of the cochlear fluids is lost too, and yet Carhart's notch is absent.

Ossicular inertia seems less important than inertia of the labyrinthine fluids in the mechanism of bone conduction: by the interruption of the ossicular chain, as in dislocation of the incus, the inertia of the chain is diminished and yet the clinical bone conduction is unaffected. This agrees with Bárány's observation that under high degrees of conduction deafness inertial bone conduction is eliminated.

It is, of course, very difficult to assess clinically the relative importance and interaction of compression bone conduction, labyrinthine inertia and probably other, still unknown components of bone conduction, e.g. sound energy given off by the walls of the middle ear to the enclosed air and acting on the labyrinthine windows and on the tympanic membrane from the inside. For practical purposes it is sufficient to state that if the bone conduction audiogram does not show Carhart's notch, the oval window is not likely to be blocked by stapes fixation. If, in addition, there is a large and uniform air-bone conduction gap, then these findings should alert the otologist to the possibility of an interrupted ossicular chain behind an intact tympanic membrane and prompt him to search for further confirmation from the patient's history. The final diagnosis may not always be possible from audiometric findings alone, because combinations of stapes fixation with an interrupted ossicular chain in cases of tympanosclerosis and post-inflammatory adhesions in the middle ear do occur. In these cases a probatory tympanotomy will be necessary, but then the surgeon will have his instruments ready to deal with any eventuality.

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I wish to thank Mr. Philip Beales of Doncaster for his ready co-operation in allowing me to peruse the records of his patients. The audiograms of three of his patients are reproduced in Fig. 2.

REFERENCES

Allen, G. W., and Fernández, C. (1960) *Ann. Otol. (St. Louis)*, **69**, 5.
Anklesaria, D. M. (1963) *J. Laryng.*, **77**, 528.
Bárány, E. (1938) *Acta oto-laryng. (Stockh.)*, Supp. 26.
Bauer, F. (1958) *J. Laryng.*, **72**, 676.
Beales, P. Personal Communication.
Békésy, G. v. (1954) *J. acoust. Soc. Amer.*, **26**, 106.
Carhart, R. (1950) *Arch. Otolaryng.*, **51**, 798.
Freeman, J. (1959) *J. Laryng.*, **73**, 196.

https://doi.org/10.1017/S0022215100062216 Published online by Cambridge University Press

GISSELSSON, L. (1958) *J. Laryng.*, **72**, 329.
GROEN, J. H., and HOOGLAND, G. A. (1958) *Acta oto-laryng.* (*Stockh.*), **49**, 206.
HUIZING, E. H. (1960) *Acta oto-laryng.* (*Stockh.*), Supp. 155.
HOOD, J. D. (1962) *J. acoust. Soc. Amer.*, **34**, 1325.
KIRIKAE, I. (1959) *Acta oto-laryng.* (*Stockh.*), Supp. 145.
LANGENBECK, B. (1963) Lehrbuch der praktischen Audiometrie, G. Thieme, Stuttgart.
WOODS, R. R. (1950) *Arch. Otolaryng.*, **51**, 485.

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