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Hearing Disorders and Their Biological Therapy

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In approaching a discussion of hearing disorders and their biological treatment, it is first advisable to briefly call to mind the anatomy and the physiology of the ear. In anatomical terms, we divide the organ of hearing into the external ear (auricle and outer auditory canal), the middle ear (eardrum, tympanic cavity, hearing ossicle, Eustachian tube, mastoid process) and the inner ear. Within the tympanic cavity, we differentiate between a lower part, the so called hypotympanum, a middle part, the so called mesotympanum and an upper part, the so called epitympanum, or cupola area. This upper part is the location of the series of small bones with the hammer, anvil and stirrup. The oval plate of the stirrup-bone terminates in the membrane of the oval window known as the vestibule window. Beneath this lies the round window leading to the cochlea.

The inner ear includes:

1. the vestibule
2. the semicircular canals with the vestibulary apparatus
3. the cochlea with the hearing apparatus
4. the nervus acusticus (auditory nerve)
5. the nervus cochlearis
6. the aquaducts which reflect topographic relations to the interior of the skull

A physiological difference is drawn between the acoustic and static apparatus of the ear. The static apparatus, also referred to as the vestibular semicircular canal or vestibular apparatus, serves the maintenance of balance and need not concern us here. The acoustic apparatus is divided into the sound conduction apparatus with the auricle, the outer auditory canal, eardrum, series of small auditory bones, the stirrup-bone plate, oval window and perilymph, as well as the sound perception apparatus in the cochlea, also known as the *Corti* organ.

More recent research results have added considerably to knowledge concerning the occurrence of the sense of hearing insofar as this probably not only involves mechanical processes but also ionic shifts between potassium and sodium at the membranes of the lymph areas (*Meyer zur Gottesberge* inter alia).

As is known according to the theory of *Helmholz*, the function of hearing comes about as follows: auricle and outer auditory canal form a horn, collect the sound waves and conduct these to the eardrum. The eardrum sets the series of small auditory bones in motion, which also sets in motion the inner ear fluid (or perilymph) through the stirrup-bone plate in the oval window. With its vibrations and the ionic shift these cause, the inner ear fluid arouses the sense cells in the cochlea. The stimuli taken up in the cochlea are now passed on via the auditory nerve to the cerebral cortex. The membrane of the round window serves to compensate pressure within the area of the inner ear upon arrival of the sound waves at the oval window. Good vibratory performance of the membranes, i.e. of the eardrum, the membrane in the oval window with the stirrup-bone plate and the membrane in the round window, depends on balanced air-pressure between the middle ear areas and the outside world. This air pressure balance is ensured by the Eustachian tube which forms an open connection between the middle ear and the

pharynx. From a purely mechanical point of view, important factors for the hearing are good mobility and undisturbed function of the eardrum, the series of small auditory bones, of the membrane in the oval window and the membrane of the round window, and finally, the lack of any restriction in the passage of the Eustachian tube.

After these brief anatomical and physiological comments, I should like to discuss certain significant pathological disturbances in the hearing organ itself. These are generally apparent in the form of deficiency symptoms on the one hand, as well as stimuli symptoms on the other. As one of the most frequent deficiency symptoms, I refer first to hardness of hearing. We can differentiate here between three categories.

1. Sound conduction or middle ear deafness occurs when the sound conduction apparatus, in other words the external ear or the middle ear are displaced or damaged so that the stimuli triggered by the sound waves no longer have unrestricted access to the nerve ends.
2. Sound perception deafness, which can also be described as nerve or inner ear deafness and which is based on damage to the actual end organ itself.
3. Centrally produced hardness of hearing, so called cortex deafness.

Subjective noises on the other hand involve stimuli symptoms which are generally the result of disease of the middle ear or nerves and which can occur in disturbances of the entire hearing apparatus. Stimuli symptoms of this kind as associated with subjective noises are not clinically detectable, however. Diseases of the middle ear not caused by mechanical factors lead in the main to hearing disturbances with lesser or greater occurrence of attendant symptoms of giddiness and buzzing in the ears.

The main diseases of the middle ear are the following: acute and chronic tube and middle ear catarrh, acute and chronic inflammation of the middle ear, complications with middle ear suppurations, allergic mucuous membrane reactions in tube and middle ear, so called congestion catarrh in the middle ear (in cases of heart, kidney and circulation diseases), middle ear tuberculosis, otosclerosis. Diseases of the inner ear can also be associated with hardness of hearing, buzzing in the ears and giddiness.

The chief diseases of the inner ear to be mentioned are tympanogenic labyrinthitis, meningeal labyrinthitis, *Meniere's* disease, as well as toxic damage to hearing. These occur following infectious diseases such as flu, measles, typhoid, scarlet fever, mumps, etc., in particular following use of certain medications such as quinine, acetylsalicylic acid and streptomycin, and also following the effect of toxins such as nicotine, alcohol, lead, carbon monoxide and many others. Focal toxic causes (teeth, tonsils) and food-poisoning (fish, meat) etc., can be the cause of toxic damage to hearing.

Indirect traumatic inner ear damage is also known in relation to labyrinth concussion (fracture of the base of the skull), explosion and noise damages (boiler-maker), as well as with neuritis of the nervus facialis, trigeminus, and of the nervus acusticus (through a virus, rheumatic or toxic condition or a cold).

Toxic damage to hearing is often followed by degenerative processes on the auditory nerve itself. Finally, deafmutism should also be mentioned. There are therefore a great many diseases in which hearing disturbances with giddiness and buzzing in the ears can occur.

I should now like to refer in detail to otosclerosis as being of most interest for our theme here.

Otosclerosis is the term given to a hereditary progressive hardness of hearing with anatomical changes which are characterized by atypical formation of new bone in the bony labyrinth capsule and a bony fixation of the auditory ossicle and the stirrup-bone plate in the oval window. This leads above all to disturbance of the sound conduction. In pathological and anatomical terms, this process of new formation and reshaping of bone can best be compared to osteodystrophia fibrosa. According to homotoxin theory, degeneration and impregnation phases are mainly involved here.

The sound conduction disturbance is generally also accompanied by degenerative processes affecting the auditory nerve itself as well as the nervous end organs of the cochlea, leading to inner ear deafness. No uniform explanation has as yet been found for these processes, although homotoxin theory suggests that the effects of homotoxins and of retoxins in particular are to be assumed. Both ears are often more or less equally affected. The disease frequently first becomes apparent in the second decade of life. It is a characteristic phenomenon that sufferers from otosclerosis can hear better in noisy surroundings as well as by pressing the receiver hard against the ear when telephoning, the explanation for which is that the bone conduction of sound is not affected in cases of otosclerosis.

Combined hardness of hearing is often accompanied by highly unpleasant, tormenting and purely subjective noises in the ear which patients frequently find more disturbing than the hardness of hearing itself. These noises in the ear can reach such proportions that they lead to mental depression, and even attempted suicide.

Treatment of hardness of hearing as a result of otosclerosis can be both operative or conservative. An operative intervention can involve mobilization of the stapes, whereby the stirrup-bone plate is loosened by shaking with small hooks and other instruments if it has not become too firmly fixed. The so called stapes graft can also be carried out, involving formation of a window and a graft on the oval window and stirrup-bone. The actual window-forming operation consists of the removal of part of the bony labyrinth capsule thus incurring a window-like exposure of the endolymph tube. All operative measures only have chances of success if sufficient inner ear function still prevails, however.

Conservative treatment of hardness of hearing has up to now consisted primarily of air showers, tube catheters, eardrum massages, fever cures, foot contrast baths, head light baths, ray-treatment.

For psychological reasons, sedatives of all kinds have mainly been prescribed up to now against noises in the ears.

Since it is the view of homotoxin theory that otosclerosis should not usually be considered as a deposition but rather as an impregnation or degeneration phase, the following therapeutic guideliness ensue in consideration of homotoxin theory:

If the otosclerosis is acquired, that is to say, is based on retoxic impregnations, various different therapeutic interventions can be attempted which, in the case of the degenerative form of the disease will prove to have as little success as with prevailing methods, i.e. only rarely leading to a positive outcome.

I recommend the following standard treatment:

One tablet each of *Barijodeel* and *Cocculus compositum* are to be taken at the same time three times daily.

In cases of therapy resistance, it is worth adding *Graphites-Homaccord*, in particular if there has been a progressive vicariation from dermatitis or eczema.

Osteoheel has also proved successful in some cases specifically when the bony enclosure of the auditory smal bones and the stirrup-bone plate have been involved.

In the case of reverberation of sound in the ear, *Phosphorus-Homaccord* can also be tried with occasiona success. *Schwef-Heel* is indicated for the treatment of any retoxic phases.

The result of the tests carried out by me is encouraging We have possibilities here of applying homotoxin theory t the treatment of otosclerosis with its tiresome attendan symptoms, above all tormenting buzzing in the ears and unpleasant giddiness and of exercising a favorable influenc under certain circumstances.

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