CONCERNING THERAPEUTICAL PRINCIPLES
IN ACUTE LOSS OF HEARING

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Key-words: acute loss of hearing – treatment – etiology

The problem of acute loss of hearing (ALH) is actual in otorhinolaryngology because of various etiology and difficult restoration of hearing damage. That is why it is reasonable to look for new means and methods for diagnostics and treatment despite of certain difficulties in this respect.

According to numerous literature data available, there are various reasons for ALH, e.g.:

1. Most frequently (in 2/3 of the cases), ALH is caused by vascular disorders resulting in circulatory disturbances of the inner ear. They induce hypoxia and thus cell respiration reduction. Then metabolic products accumulate, glycogen and phosphate metabolism is destroyed, electrolyte composition of endo- and perilymph undergoes changes. Partial \( O_2 \) pressure in the inner ear decreases and causes disorder of oxidative phosphorylation which is closely related to metabolic processes of inner ear receptor apparatus. Damaged metabolism induces loss of function and later on acoustic cell death (4, 11).

2. Traumatic inner ear lesion: a) labyrinth membrane rupture; b) temporal bone fracture; c) covered head and neck injuries without fracture; d) diving damages; e) detonation wave damages; f) surgical trauma (1-3, 6, 13, 14).

3. Labyrinthitis, labyrinthineuritis: a) toxic — by kanamycin, gentamycin, etc.; b) toxoinfectious — in cases of parotitis, herpes zoster, influenza, morbilli, etc.


5. Endocrine disturbances: diabetes mellitus, thyrotoxicosis, etc.

6. Sudden cryptogenic loss of hearing developing without any general or local symptoms.

During the last 8 years, we treated a total of 34 ALH patients. Of them 24 were males and 10 females. Their age distribution was as followed: between 10 and 20 years — 4 cases; between 20 and 30 years — 6; between 30 and 40 years — 15; between 40 and 50 years — 5, and between 50 and 60 years — 4.

According to etiological factors, ALH patients’ distribution was as followed: loss of hearing set in suddenly in completely healthy persons in 25 cases; 7 patients were with infectious diseases and 2 ones with meningoencephalitis. Of 25 cases with sudden loss of inner ear function, in 4 ones ALH was caused by an acute acoustic trauma: in one after diving and in 2 other ones with cranial basis fracture. ALH was accompanied by vestibular disorders (ataxia, vertigo, fistular symptom). According to H. Ludman (9), labyrinth membrane rupture could be suspected in such patients. This possibility should be had in mind with: a) patients with perception ALH related to or preceded by head injury, barotrauma or uncommon physical efforts. If fistular test was positive diagnosis was almost sure; b) patients with vertigo after cerebral commotion (5, 7, 8). Loss of hearing set in with 15 completely healthy individuals (without any anamnestic data about a given reason) (however, ignoring smoking in 11 of them) with normal paraclinical examinations. Proceeding from the concept that ALH could be due to preceding change of altitude above sea level, cough, sneeze, overstrain when having a bowel movement, painful micttition, etc. (7, 12) we could explain the “causeless” inner ear function loss. Smoking-induced changes were also important: hypoglycemia, enzyme system activity inhi-
bition, resulting in metabolic disturbance in the cochlea. Besides almost 20 per cent of haemoglobin was bound up with an other substance in the form of carboxyhemoglobin in smokers' blood and that was why oxygen blood supply to cells was insufficient (10).

ALH patients were hospitalized in the Clinic after different time intervals, namely: between the 1\textsuperscript{th} and 5\textsuperscript{th} day — 3 patients; between 5\textsuperscript{th} and 10\textsuperscript{th} day — 5 patients; between 10\textsuperscript{th} and 15\textsuperscript{th} day — 2 patients, and between 15\textsuperscript{th} and 30\textsuperscript{th} day — 3 patients. The rest 21 patients were hospitalized after a period between 1.5 month and one year. This late admitting to hospital was due not only to patients themselves but also to underestimation of their state by physicians who did not consider them requiring urgent treatment.

Acoustic damage was unilateral in 20 and bilateral in 14 of our 34 patients with ALH. One patient with unilateral ALH had an accompanying lesion of the function of ipsilateral vestibular apparatus.

Acoustic and cochlear structural disorders set in depending on the character of damaging agent during a period from several hours up to 10 days (4). Therefore, every delay of treatment of ALH patients could be fatal concerning the end result.

On the basis of considerations mentioned above treatment of ALH patients has to work on the following principles:

1. Early beginning of treatment, after several hours up to 1-5 days.
2. Performing of labyrinth rehabilitation including: elimination of energetic blockade, restoration of circulation, stopping of the action of the aggressive factor.
3. Intensive treatment in order to restore metabolic comfort in labyrinth.
4. Performing of maintenance therapy directed towards reservation of optimal levels of metabolic processes and improvement of microcirculation in labyrinth.
5. When there is doubt about membrane rupture first of all a conservative treatment is required. It is not allowed to increase intracranial pressure when straining, coughing, etc. If spontaneous healing of fistula does not occur a surgical intervention has to be done. The period on 10\textsuperscript{th}-12\textsuperscript{th} day after trauma is most appropriate for investigation (7).

Treatment must start as early as possible and then be rather long-lasting (up to 6 months).

We apply complex treatment independently of the etiological factor and at the same time by a selective way, in dependence on the cause supposed (e.g. traumatic lesion).

During the first stage (10-14 days) we include: 1. Medicaments with peripherically vasodilatory action in order to improve inner ear circulation: sadamine, dusodril, acidum nicotinicum, A.T.P. These means can reduce vascular resistance and thus improve inner ear microcirculation by their direct myc tropic vasodilatory action. Carbogen (i.e. 5-7 per cent of CO\textsubscript{2} and O\textsubscript{2}) possesses the best vasodilatory effect improving inner ear circulation. Every inhalation is 15-20 min long twice daily with interval of 3-4 hours.
2. Medicaments influencing upon cell metabolism: vitamins of B-group (B\textsubscript{1}, B\textsubscript{6}, B\textsubscript{12}), cocarboxilase, vitamins C, A, E. Vitamin B\textsubscript{1} as well as cocarboxilase together with their favourable influence on carbohydrate metabolism are related to processes of transfer in neuronal synapses.
3. Drugs reducing intrinsic tenseness and psychic stress (thioridazin, etc.).
4. Dehydratants (mannitol, furothril, etc.).

In cases of inflammatory diseases we administer antibiotics of tetracycline group as well as other non-toxic chemotherapeutic drugs for 8-10 days, antiallergic drugs (allergosan, sandostren calcium, etc.) and corticosteroids. 15-20 days after begin of the disease we administer biogenic stimulators (fib, aloe) for 30-40 days and ultraphonophorese (magnitophonophorese) with calcium iodatum or electrophorese with nivalin as well.
REFERENCES


ОТНОСИТЕЛЬНО ПРИНЦИПОВ ЛЕЧЕНИЯ ПРИ ОСТРОЙ ПОТЕРЕ СЛУХА

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РЕЗЮМЕ

Авторами проводится анализ причин острой потери слуха у 34 больных. Применяются основные принципы лечения острой потери слуха: 1. Раннее начало лечения — от нескольких часов до полтора дня; 2. Проведение лабиринтной реанимации; 3. Интенсивное лечение с целью восстановления метаболитного комфорта в лабиринте; 4. Улучшение микровообращения в лабиринте; 5. Оперативное лечение через 10–12 дней в случае сомнения о руптуре мембран, если не наступит спонтанное заживление фистулы.

Авторами рекомендуется парентеральное (венозное) применение лекарственных средств: вит. В1, В2, В6, В12, С, А, АТФ, кокарбоксилаза, никотиновая кислота, стугерон, радекол, компламин, карбоженотерапия, нивалин, антибиотики и кортикостероиды.