Sudden Deafness Caused by Lifestyle Stress: Pathophysiological Mechanisms and New Therapeutic Perspectives

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Abstract: Stress caused by lifestyles in industrialised countries can affect numerous illnesses. Side effects of stress at the level of microcirculation are vasoconstriction, haemoconcentration and vascular occlusion. Reduced or restricted blood flow in the internal auditory artery of the inner ear can lead to sudden deafness. The extent of the damage depends on whether vascular occlusion is momentary or prolonged. The hypothesis put forward is that stressful situations in everyday life can cause differing degrees of sudden deafness. Treatment is based on three main actions, including elimination of the acute stress, re-establishment of blood flow to the inner ear and management of overall patient stress for a complete recovery. Furthermore, another therapeutic option exists, and this alternative is based on the hypothesis that hair cells in the cochlea can be physical rehabilitated with sounds. The acoustic stimulus places pressure on the tympanic membrane, which increases pressure *via* the ossicles (malleus, incus and stapes). The stapes in the oval window move the perilymph, producing waves that move the endolymph. The tectorial membrane moves the cilia of the external and internal hair cells. Massaging the cilia causes hair cells in the cochlea to contract each other, thus emitting an electric impulse. This impulse through the cochlear nerves and the auditory system produces sound perception in the auditory cortex. The sound is applied in the ear with sudden deafness, even though cophosis, a complete loss of hearing, has occurred. In conclusion, sudden deafness may be caused by stress, being the treatment the elimination of stress; re-establish the blood flow to the inner ear and a physical rehabilitation with sounds of cochlear hair cells to reverse sudden deafness.

BACKGROUND

Sudden deafness is a loss of hearing that develops over several hours [1], and it may be accompanied by tinnitus and vertigo. It affects between 5 and 20 out of every 100,000 individuals [2]. Many aetiologies have been proposed for sudden deafness, including cochlear membrane rupture, microangiopathic processes, viral infection, autoimmune disorders, Ménière's disease, schwannoma and meningioma. Processes related to stress have also been investigated in association with sudden deafness. Specifically, studies have examined stressful lifestyles; emotional, psychosocial or occupational disorders and personalities that are prone to stress [3-7]. Taking into account all of the above aetiopathogeneses, no pathophysiological mechanisms have been established to explain sudden deafness. There has recently been an increase in information potentially explaining the pathophysiological problems caused by stress, which could elucidate how sudden deafness progresses.

HYPOTHESIS: STRESS PRODUCES SUDDEN DEAFNESS. PATHOPHYSIOLOGICAL MECHANISMS

Animal studies. Experiments carried out on stressed rats have shown an increased aggregation of red blood cells, which leads to haemoconcentration, with reduced blood flow in the microcirculation [8]. The occlusion of blood flow in the inner ear has a greater impact on more vulnerable structures such as the cochlea. The cochlea is more affected by these types of changes than the vestibular labyrinth, for example. The critical areas in the cochlea include the external and internal hair cells, along with the stria vascularis [9].

Human studies. Potential pathophysiological mechanisms for the changes in the ear include vasoconstriction, with liquids escaping from the bloodstream because of the activity of the sympathetic nervous system. Additionally, hyperviscosity of the plasma and the blood; higher concentrations of proteins, fibrinogen, free fatty acids and interleukin-1 in the plasma; increased platelets and leukocytes; augmented platelet adhesiveness and aggregation; and higher blood pressure are also potential results of stress. The plasma volume is reduced, as is blood flow and filterability [10-20]. Stress produces vasoconstriction, haemoconcentration and reduced blood movement throughout the body. For the purpose of this pathology, stress refers to any type of physical, mental, psychological and psychoemotional stress. Stress may be acute and chronic. There are psychosomatic influences on the lifestyle of each person and therefore the way in which he/she becomes ill. Emotional reactions associated with a stressful situation result in abnormalities in blood rheology, causing blood circulation to deteriorate due to a reduced flow from increased haemoconcentration [21].

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At the microcirculation level within the heart this same process triggers angina and heart attacks [22]. Within the inner ear, this process can trigger sudden deafness (Fig. **1**).

The hypothesis supported by this knowledge explains how stress can trigger sudden deafness.

THERAPEUTIC PERSPECTIVES

Therapeutic measures must be established in the following points:

1. Acute stress elimination: Medical-therapeutic advice may be used, along with relaxation techniques and sedative medication (hydroxyzine, halazepam, alprazolam).

2. Re-establishment of blood flow to the inner ear: In addition to removing the acute stress, haemodilution [23], blocking the cervical sympathetic chain [10], corticosteroids [24] and vasodilators [25] can be utilized to help return normal blood flow to the inner ear.

3. Stress management: The patient must be able to manage stressful situations. The elimination of prolonged stress is essential for recovery from sudden deafness. If the patient continues to experience stress during the post-acute phase, the prognosis gets worse since the factors causing the episode are maintained. The individual's personality is therefore extremely important. Cognitive-behavioural therapy is advised.

4. Inner ear hair cells physical rehabilitation. This new approach is based in animal studies. Sound stimulus leads to improvements or recovery in hearing. Chinchillas were subjected to acoustic deprivation following an acoustic trauma, which consisted of an ossiculectomy on one of their ears. The elimination of the ossicles results in receiving less ambient sound to that ear. In the remaining non-surgical ear, the acoustic trauma resulted in damage to the hair cells and hypoacusia. However, increased damage occurred to the hair cells in the operated ear, as well as greater hypoacusia [26]. In a second study on guinea pigs suffering from bilateral acoustic trauma, the group of animals that received sound conditioning seemed to recover normal hearing, while the group that did not receive sound conditioning developed hypoacusia [27]. In a third study on mice with a genetic predisposition for sensorineural deafness as adults, one group was subjected to a moderately intense sound while the other group had no sound treatment. The group who received sound stimulation presented less presbycusis and less damage to the external and internal hair cells, the spiral ganglion and the ventral cochlear nucleus [28]. Finally, a fourth study on cats demonstrated that animals exposed to acoustic trauma and sound stimulation did not undergo reorganization of the tonotopic map. Therefore, they did not suffer from deafness, tinnitus or hyperacusis. In contrast, in the group of cats that was not subjected to sound stimulation, acoustic trauma caused reorganization of the tonotopic map, along with deafness, tinnitus and hyperacusis [29].

HYPOTHESIS: PHYSICAL REHABILITATION OF INNER EAR HAIR CELLS AIDS RECOVERY

Physical rehabilitation consists of moving the cilia of the external and internal hair cells. These may be reached through the liquids contained in the inner ear. The endolymph bathes both types of cells. The endolymph may be moved by placing pressure on the tympanic membrane. This pressure may be applied by sounds introduced through the ear canal (Fig. **2**). The type of sound applied may be white noise, which would stimulate all auditory frequencies at an intensity sufficient to produce wave movements in the endolymph. These changes in the endolymph will in turn lead to movement to the tectorial membrane, causing action in the cilia of the cochlear hair cells. If the intensity of the sound is too high, it may cause acoustic trauma, and if it is too low, the resulting movement of the cilia may be insufficient. Average values of 60dB SPL seem to be adequate. White noise will evenly stimulate all frequencies at this intensity.

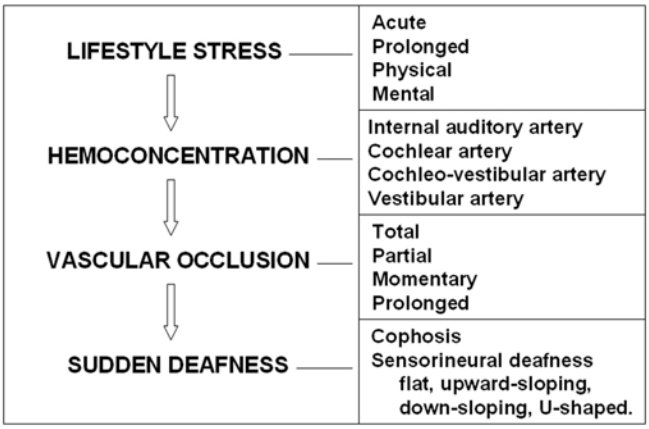

Fig. (1). Hypothesis: lifestyle stress produces sudden deafness. The pathophysiological mechanisms, induced by stress, triggering sudden deafness include vasoconstriction, haemoconcentration and vascular occlusion. The degree of sudden deafness depends on predetermined factors of blood flow, which are shown in the right part of the figure.

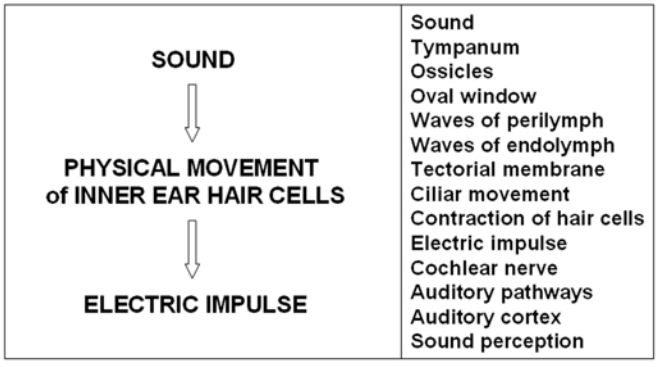

Fig. (2). Hypothesis: physical rehabilitation of inner ear hair cells. The cilia of the hair cells are massaged using sounds. Movement of the cilia produces cellular contraction and nerve impulses. Physical movement of the cell helps recover its hearing function. The entire sequence is shown in the right side of the figure, starting with the sound and ending with the sound perception.

This mode of treatment may be adequate for patients who suffer from incomplete sudden deafness or in cases of sudden cophosis. Patients with sudden cophosis will not initially

hear the sound applied. They will begin to hear it as they recover their hearing.

Sound stimulation can be personalised according to the audiometric configuration, which consists of cophosis (complete loss of hearing) and a flat curve, an upward-sloping curve, a downward-sloping curve or a U-shaped audiometric curve. In cases of cophosis and a flat audiometric curve, uniform sound stimulation at all frequencies may be adequate. In upward-sloping configurations, low and medium frequencies are principally stimulated. In downward-sloping configurations, high and medium frequencies are more intensely stimulated. Finally, medium frequencies are stimulated for U-shaped configurations (Fig. **3**). Alternative sounds that may be used, instead of white noise, include more complex musical sounds or sounds of nature. The practical application of this hypothesis is simple. Sounds should be added to the routine treatment of sudden deafness.

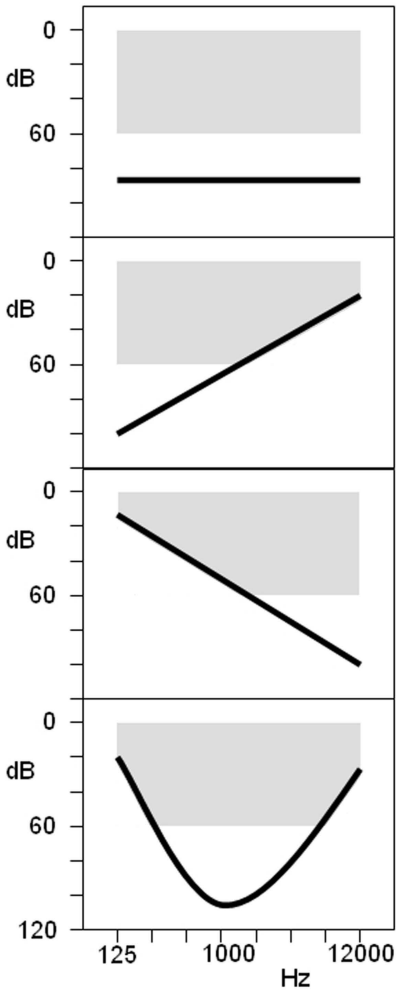

Fig. (3). Personalising sound stimulation. Sounds are applied in accordance with the audiometric configuration. From top to bottom, flat audiometric configuration and cophosis receive the same type of sound. An upward-sloping configuration receives a less intense sound in the better hearing acute frequency zone. A downward-sloping configuration receives a less intense sound in the better hearing bass frequency zone. A U-shaped configuration receives a less intense sound in the better hearing high and low frequency zones. The shaded zones correspond to the sound applied.

CONCLUSIONS

Sudden deafness can be triggered by stress, which leads to vasoconstriction, haemoconcentration and microcirculation occlusion in the inner ear. Treatment consists of eliminating the stress, as well as managing future stress to achieve a full recovery, re-establishing blood flow and physically rehabilitating the cochlear hair cells using sounds.

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