

Occupational Implications by Exposure to Industrial Noise: A Review

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Abstract

The study of so-called occupational diseases and among them noise-induced hearing loss, nowadays is of relevant importance given first by the increase in its incidence and economic cost. The main objective of this review article is to analyze qualitatively the scientific literature available in the databases Science Direct, Scielo, Redalyc and official websites, using as search words: industrial noise, professional deafness, noise induced hearing loss, acoustic trauma. Relevant information related to the proposed objective was obtained, which is presented in six sections: generalities, definition, clinical picture, diagnostic exams and tests, prevention and control, treatment and rehabilitation. We conclude that the main effects of exposure to noise are auditory fatigue in which there is a transient decrease in hearing capacity.

Keywords: Hearing-induced hearing loss, Industrial noise, Productive sector, Professional deafness, Acoustic trauma.

INTRODUCTION

Work and development force us to live in an environment in which the world of sounds becomes aggressive to man, so noise can be considered a significant contaminant today [1]. According to a report by the World Health Organization (WHO) in 2002, noise is included in the five main physical risk factors for health in the workplace [2].

According to Ganime and collaborators [3], industrial noise exists in all industries as a result of the operation of machines of the most varied types, some machines mainly that are equipped with less technology produce excessive noises, beyond tolerable. This type of noise is in conflict with the conditions of human life and is opposed to the increase of the productivity of the work and the quality of health of the worker, that is to say, if the employee is forced to work in noisy environments decreases his productivity by psychophysiological effects, Ranging from simple irritation to hearing loss.

Noise is in most countries the most common harmful agent in the workplace. Its presence in the industrial activities is added to its wide diffusion in the urban and social means, especially in leisure activities. This almost universal diffusion of noise in social and work environments becomes more important if

hearing damage is considered to be irreversible, and exposure causes other disorders - organic, physiological and psycho-emotional - that result in a clear reduction in the quality of life and health of workers [4, 5, 6, 7].

Unlike other pollutants, the effects of noise can be instantly unnoticed and their accumulation can lead to obvious physical, psychological and social deterioration. The best studied effect of overexposure to noise is hearing loss. The problem is that exposed people are scarcely aware of the cause-effect relationship, since it occurs slowly but progressively [8, 9].

The main effect of prolonged exposure to this physical risk factor on workers' health is hearing loss, called hearing loss or professional deafness, which is identified as the most documented and frequently recognized effect as Occupational Illness [10,11,7]. In addition, the noise interrupts the communication and alters the affectivity, which induces the isolation and can trigger neurosis. Other general effects of continuous and uniform noise exposure include mental stress, fatigue, sleep disturbances, decreased alertness and speed of motor reactions, and reduced responsiveness of the autonomic nervous system [12].

Noise is one of the intraorganizational stressors most commonly encountered in work environments that cause various effects on humans [13], states that most industrial workers point to noise as the main environmental condition that Affects their work, studies show the influence of exposure to chronic industrial noise in 1680 men and 688 women participants of the Cordis Study, and reported that men presented job dissatisfaction and post-work-day irritability, while for women somatic disorders , Anxiety and depression were accentuated. All stress symptoms were higher for women, especially those who were exposed to moderate levels of noise (75-84 dBA), usually not considered harmful to hearing.

According to Alonso in 2014 in Spain [14] and through the VII National Survey of Working Conditions conducted by the National Institute of Occupational Safety and Health in 2011 indicates that 35% of workers are exposed to a level of Noise annoying, high or very high, being the industrial workers and mechanics and workshop employees the most affected groups. According to data from the survey, only 42% of workers considered to be exposed to a very high noise level (2% of the

total) stated that it is mandatory to wear hearing protectors at their workplace. On the other hand, of the workers who are considered to be exposed to a high noise level (8% of the total), only 32% show this compulsory. Therefore, noise supported by a large group of workers exceeds subjectively adequate levels without perceiving the need for the use of personal protection, which favors the appearance of hearing loss among those affected and makes necessary the realization of instrumental explorations to detect precociously the onset of the disease.

In the same vein for Fernández and collaborators in 2009[9], they point out that every day millions of European workers are exposed to noise and all their consequent risks in their workplace. One in five workers in Europe must raise their voices to be heard for more than half the working day and 7% of them suffer from work-related hearing problems. According to European data, hearing loss caused by noise is the most common occupational disease in the European Union. Noise can be a clear problem in sectors such as manufacturing or construction, but it can also be a problem in some other working environments. The most obvious parameter for characterizing noise may be its level, measured in decibels, but there are some other important factors to consider, such as duration of exposure, impulsivity, frequency and spectrum, incidence and distribution to throughout the working day.

In Colombia, based on Gómez [15], one of the main consequences of exposure to prolonged occupational noise in the workplace is sensorineural hearing loss, defined as hearing loss produced by prolonged exposure to hazardous noise levels. According to the report of professional disease in Colombia, conducted by the Directorate General of Professional Risks of the Ministry of Social Protection published in 2004, sensorimotor sodas is the fourth cause of occupational morbidity in the contributory regime and worldwide after presbycusis, is the most common cause of decreased auditory acuity. The large number of cases of sensorineural hearing loss observed in Colombian and military employees, and the consequences thereof, make it a serious public health problem that significantly affects the hearing capacity of workers.

The measurement of the noise allows a more precise analysis of the components of frequency, amplitude and duration that are necessary to determine their noxiousness. It is important to know the amount of sound energy that an individual accumulates during their working hours in noisy environments. To determine the causal relationship between occupational exposure to noise and hearing loss, the differential diagnosis is conclusive and the occupational physician, who will establish the connection. In the analysis, in addition to the audiometric configuration or sequential evolution, other factors such as the worker's clinical and work history, age; Past time and current exposure to sound pressure

levels; The levels of sound pressure to which the worker is or has been exposed in the course of work; Not occupational exposure to sound pressure levels; Occupational exposure and not occupational exposure to other risk agents for the auditory system [16].

BACKGROUND

Noise-induced hearing loss (HIR) is a health problem that increases, in conjunction with the advancement of civilization. Exposure to high intensity noises causes disorders such as inability to communicate with others, reduces the quality of life of the human being and their socialization, a phenomenon known as socioacusia. Possible causal factors of hearing loss in the workplace should be considered. The nature of noise, noise intensity, frequency of noise, exposure time, individual susceptibility, diseases of the middle ear and different toxic products (eg carbon dioxide, arsenic, toluene and so on) [17,15].

The earliest reference to the effect of noise on hearing is an observation recorded in the first century of n. By Pliny the Elder in his "Natural History," when he mentions that people living near the Nile Falls "were deaf." At the end of the nineteenth century, with the advent of the steam engine and the initiation of the industrial age, noise appeared as an important public health problem. At this stage the deafness of exposed workers, such as smiths and welders, begins to be documented. Fosbroke, in 1831, mentioned the deafness of the blacksmiths and Wittmarck did the same in 1907, when showing the histological effect of the noise in the ear; In 1927, McKelvie and Legge report on the deafness of the cotton farmers; In 1939, Lars described the deafness of shipyard workers and, in 1946, Kristensen referred to the deafness of aviators and submarine crews [18,19].

Automation and mechanization have revolutionized the mass production systems that emerged from the industrial revolution. Since 1980, this period has been called the "postindustrial revolution". This new system is characterized by the use of modern equipment, pesticides and other chemical substances that lead, on the one hand, to greater productivity and, on the other hand, to health problems and environmental contamination [20, 21].

EPIDEMIOLOGY

It is estimated that one-third of the world's population and 75% of the inhabitants of industrialized cities suffer from some degree of deafness or hearing loss caused by exposure to high intensity sounds. PAHO reports an average prevalence of hearing loss of 17% for Latin America, in workers with 8-hour days a day, for 5 days a week with an exposure ranging from 10 to 15 years. In the United States of America, hearing loss induced by industrial noise exposure is one of the most

common occupational diseases. In Europe, an estimated 35 million people are exposed to harmful noise levels [17, 22, 5, 23].

PATHOGENY

Mechanisms favoring noise damage

Microtrauma Theory: The peaks in the sound pressure level of a constant noise, lead to the progressive loss of cells, with the consequent elimination of neuroepithelium in increasing proportions [22, 24].

Biochemical Theory: Postulates that hearing loss originates from the biochemical alterations that the noise triggers, and leads to a depletion of metabolites and ultimately to cell lysis. These biochemical changes are: decrease of O₂ pressure in the cochlear duct; Decreased nucleic acids in cells; Decreased glycogen, ATP; Increase of reactive oxygen elements (ROS), such as superoxides, peroxides, and hydroxyl radicals, which favor oxidative stress induced by noise; (Na⁺), K⁺ - ATPase and Ca²⁺ - ATPase [25, 22].

Theory of intracellular calcium conduction: It is known that the noise is able to depolarize neurons in the absence of any other stimulus. Recent studies have shown that the alterations or distortions suffered by the propagation wave of intracellular calcium in neurons are due to changes in the calcium channels. Low calcium levels in the inner hair cells seem to intervene in the prevention of hearing improvement rate HIR [25, 26].

Mechanism mediated by macrotrauma: The shock wave produced by intense discontinuous noise is transmitted through the air generating a force capable of destroying structures such as the tympanum and the ossicles chain [27].

Noise damage protection mechanisms

Neural Mechanism: Studies in guinea pigs confirm the hypothesis that the cochlear efferent system is involved in mechanisms underlying the "hardening effect" at high frequencies. This effect is defined as a progressive reduction of the threshold when repeated exposures to the same noise are applied. Vestibular neurectomy performed through the posterior fossa, ensuring the interruption of crossed and non-crossed olivocochlear fibers in a single ear, before its entrance into the ear canal, causes hearing loss due to noise exposure, compared to the non-operated contralateral ear [28, 29].

Antioxidative Mechanism: The absence of antioxidant substances such as superoxide dismutases (CuZn-SOD) and glutathione potentiate noise-induced damage. They exert a protective mechanism on the cochlea [30, 31, 32].

Sound Conditioning Mechanism: Evidence continues to accumulate that demonstrate the importance of reducing the

deleterious effects of acoustic trauma by sound conditioning, this is a process of exposure to low levels of non-harmful noise, to create long-term protective effects To the detriment of subsequent harmful forms of acoustic trauma. Different conditioned sound paradigms have been successfully tested to prevent pathological changes in the auditory system [33, 34].

DEFINITION

The HIR is defined as the decrease in the hearing capacity of one or both ears, partial or total, permanent and cumulative, of sensorineural type that originates gradually during and as a result of exposure to harmful levels of noise in the working environment, Of continuous or intermittent type of relatively high intensity (> 85 dB SPL) over a large period of time, and should be differentiated from acoustic trauma, which is considered more as an accident, than a true occupational disease [35]. The HIR is characterized by an insidious onset, a progressive course and a predominantly bilateral and symmetrical presentation. Like all sensorineural hearing loss, this is an irreversible condition, but unlike it, HIR can be prevented. From a behavioral point of view and for its better compression and adequate audiological follow-up the HIR can be divided into four phases or stages based on the classifications of Martinez [36] and McBride [37]:

Phase I: (installation of a permanent deficit). Before the introduction of an irreversible HIR, a threshold increase of approximately 30-40 dB occurs at the 4 kHz frequency. This phase has as a characteristic that the cessation of exposure to noise can reverse the damage after a few days.

Phase II (latency): A latency period occurs where the 4 kHz deficit remains stable, increasing to the neighboring frequencies at a lower intensity and increasing the threshold between 40-50 dB, without compromising even the compression of the Word but there is no reversibility of hearing damage. Its discovery is important in prophylaxis.

Phase III: (of subtotal latency). There is not only affectation of the 4 kHz frequency but also of the neighboring frequencies, there is an increase of the threshold between 70-80 dB, thus leading to the incapacity in the compression of the word.

Phase IV: (terminal or manifest hearing loss). Wide auditory deficit, affecting all the acute frequencies, with compromise of serious frequencies and a threshold increase to 80 dB or more.

CLINICAL PICTURE

The HIR requires careful study of all available information, from anamnesis and clinical examination and data obtained in audiometry measurements. The anamnesis should not only

include medical and physical information of the subject but also a careful investigation on personal exposure to noise [38]. It is formed by auditory symptoms, such as hearing loss, tinnitus and vertigo (Usually the reports in the literature suggest that the noise does not produce adverse effects on the vestibular system.) Recent studies indicate the existence of vestibular disorders in asymmetrical hearing loss, being absent in symmetrical hearing loss). Others suggest that the impulse noise causes deterioration of the vestibular system, mainly of the otolithic organ.

Non-auditory effects include: hypertension, tachycardia, tachypnea, hyperacidity, decreased appetite, interfering in spoken communication, may cause distraction and greater propensity to suffer work accidents, decrease in work performance, increase personal level of Stress, irritability and sleep disturbances [39, 40, 41].

DIAGNOSTIC TESTS AND TESTS

Exams and diagnostic tests are of great importance for the study, diagnosis, treatment and rehabilitation of the patient; [11]. In this paper, the most frequently used in audiological practice and occupational medicine will be addressed, with a focus on the most novel diagnostic techniques available today.

Tonal audiometry: A test by which the degree or extent of hearing loss is determined. The goal is to obtain the thresholds for pure tone notes or variable airway and bone frequency. It is recorded on a graph, an audiogram, which shows the level of an individual's hearing threshold as a function of frequency (Hz) and intensity (dB). The role of audiometry is not limited to the mere attainment of thresholds of audibility, but it has a wide use in prevention, diagnosis, therapy and evolutionary follow-up of hearing loss, which sometimes allows an etiological diagnosis of Dhere in 2009[42].

Two audiometries should be performed with a minimum separation of one week. If there is more than 10 dB difference in the auditory averages found between one test and another, a third test must be performed. In cases where the audiometric examination was not sufficient to make an accurate diagnosis of the auditory damage, of occupational origin, it should be complemented by other audiological examinations [21].

Periodicity of audiometries: There is no consensus on this topic, but the following deadlines are considered reasonable:

- Annual audiometry for workers exposed to sound pressure levels (NPS) equal to or greater than 90 dB (A), 8 h daily.
- Audiometric monitoring every 2 years for those exposed to NPS between 85 and 89 dB (A), 8 h daily.
- Audiometric monitoring every 3 years to those exposed to NPS between 80 and 84 dB (A), 8 h daily.
- Recall audiometry for all workers who have been exposed

to NPS equal to or greater than 80 dB (A), 8 h daily.

However, NPS are not the only or the most important factor in defining the frequency of audiometries. The medical judgment may modify the timing in relation to factors such as age, exposure time, use of hearing protectors and previous audiometric results [43, 9].

Classification of audiometries: In this aspect there is a wide range of criteria, which do not always inform us of the existing reality. To unify the classification methods many health and labor institutions have adopted the one offered by the teacher. Hermann ER for being considered useful, practical and easy to calculate. This method classifies the audiometries according to the deterioration in the main conversational frequencies, by means of the system SAL (of English Speech Average Loss), and according to the loss in 4000 Hz, by the system ELI (of the English Early Loos Index).

Brain stem auditory evoked potentials (PEATC): Electrophysiological test, from the brain response to a given stimulus. It differentiates the origin of sensorineural hearing loss (cochlear or retrocochlear) and is used to assess the integrity of the brain stem in neurological syndromes and also in the search for auditory thresholds in patients who do not collaborate or simulate hearing loss.

The interpretation of the PEATC from an audiological profile is characterized by:

- The absence of response to 30 dB nHL reveals the presence of hearing loss.
- The presence of the 3 main peaks with values of prolonged absolute latencies and interpeak latencies within normal limits to 70 dB nHL is a typical sign of conductive hearing loss.
- Absence of responses (no occurrence of any component), when they do not influence technical problems, is a typical sign of severe hearing loss due to receptor injury.
- The presence of only peak V with absolute latency within the normal or slightly prolonged limit at 70 dB nHL suggests a sensorineural hearing loss, as well as the presence of peaks I, III, and V with absolute and interpeak latency values at 70 DB nHL, with electrophysiological threshold above 30 dB nHL

Multiple-frequency steady-state auditory evoked potentials (PEAeeMF): A reliable electrophysiological audiogram is possible with the PEAeeMF technique, which can be used as a new alternative in the study of noise-induced hearing loss.

In particular, the steady-state response that is obtained in the frequency range between 80-110 Hz is probably generated by the overlapping of the PEATC and therefore is little affected by sleep and sedation. This gives it great value as an instrument of audiometric exploration since the cooperation of

the subject is not required.

Otoacoustic emissions: Otoacoustic emissions are currently the objective, non-invasive and low cost test that gives us data of the acute frequencies so necessary for speech and language. Attias et al, [44] looked for the relationship between auditory thresholds by audiometry and the presence of otoacoustic emissions, in patients with or without HIR, and found that in patients exposed to noise the emissions were greatly reduced, even when the thresholds Hearing aids did not show significant changes, demonstrating that otoacoustic emissions represent a more accurate measure of cochlear damage that exposure to noise is producing even before the patient can see it, confirming that otoacoustic emissions offer high sensitivity (79-95%) and specificity (84-87%), and often provide indispensable information in medical-legal cases, in which the configuration of the audiometric thresholds are necessary to obtain an accurate diagnosis of hearing loss and that the Compensation is proportional to the severity of this.

These studies demonstrate that otoacoustic emissions provide high objectivity and certainty, complementing the audiogram in the diagnosis and monitoring of cochlea status after exposure to a noisy environment [45, 46, 47].

PREVENTION AND CONTROL

According to CDC [48], in the decade of the 70 the implantation of systems of prevention and control more complete, the denominated programs of hearing conservation begins. The use of these programs has the following objectives:

- Prevent hearing loss induced by industrial noise (100%).
- Prevent health effects from exposure to industrial noise
- Reduce absenteeism.
- Improve industrial productivity.
- Improve the administrative conditions of companies.

Components of an Auditory Conservation Program (PCA)

- 1) Initial and annual audits of procedures.
- 2) Diagnosis of the problem (noise evaluation).
- 3) Methods of noise control (source and transmission medium).
- 4) Individual hearing protection (selection and use of hearing protectors).
- 5) Audiometric assessment and monitoring of workers' hearing.
- 6) Health education about noise and hearing loss.
- 7) Information recording system.

- 8) Evaluation of the effectiveness of the program.

TREATMENT AND REHABILITATION

Many types of treatments have been proposed with the aim of delaying the occurrence of hearing loss or reducing the individual susceptibility resulting from exposure to noise; Mention may be made of the use of vitamin A, vitamin B12 (cyanocobalamin), nicotinic acid, papaverine hydrochloride, ascorbic acid, dextran, etc. Other studies show the effectiveness of the use of hyperbaric oxygenation (HBO) as a single treatment or combined with steroids, favoring the morphological and functional recovery of damaged hair cells. Studies in rabbits with the use of ascorbic acid prior to the noxious noise event raise the possible protective effect of the cochlea by inhibiting lipid peroxidation and oxidative damage of the proteins in rabbits exposed to noise. The use of antioxidants such as NL-acetylcysteine (NAC) and alpha-tocopherol in guinea pigs with a partial protective function of the cochlea to impulse noise damage were tested in rats [49, 50].

Investigations in animals using magnesium (Mg²⁺) to increase the activity of the outer hair cells, have demonstrated the utility of the same to verify that the pre-existence of low levels of this increase the levels of noise-induced hearing loss, while That the existence of elevated levels provide a significant biological-protective cochlear effect. Other studies have confirmed that magnesium is a novel natural and biological agent effective for the prevention and possible treatment of noise-induced damage in humans [51, 52].

Studies with stem cells in the ears of mice show that these could lead to the repair of deafness in humans. French and Swedish scientists have found stem cells in the ears of adult mice that have the potential to develop into hair cells to replace those that no longer exist or have been damaged. The findings of the Bionic Ear project are very promising; more research is needed to prove that these new differentiated hair cells could effectively replace the damaged hair cells in the inner ear of humans. Several clinical strategies have been proposed as new treatment alternatives, including gene therapy and embryo stem cell implantation [53, 54].

Certain patients, with a mean of 27 dB of loss (tones 500-1000 and 2000) may present a social or labor disadvantage and may be favored with prosthetic equipment, taking into account the correct selection of the prosthesis, a calibration and adaptation Adequate counseling before and after starting work [43,29].

Another treatment alternative is the use of cochlear implants, an electronic device intended to provide auditory information and improve communication to people who have a severe-deep hearing loss, who can not understand spoken language with conventional hearing aids [55, 14].

The conventional auditory rehabilitation where the audiologic rehabilitator and the patients affected by hearing loss intervene, is proposed to be replaced by a new individualized rehabilitation strategy made up of 3 components:

- Increased penetration and knowledge.
- Education and counsel with the ability to focus on the problem of communication with the partner.
- Motivation for change through discussion groups and reflective conversation.

New research in this field allows to increase the knowledge about the HIR; new diagnostic and treatment alternatives are envisaged that will improve the quality of life of affected patients, which is of vital importance in the prevention of hearing conservation programs.

CONCLUSIONS

From the results shown, from its analysis and its discussion, we can obtain the following conclusions about the auditory alterations in workers exposed to industrial noise: Among the main effects to the noise exposure we have hearing fatigue in which there is a decrease Transient auditory capacity. There is no organic injury and the hearing recovers after a resting time. Hearing loss is probably the most important effect of noise on the person. It is hearing loss caused by exposure to high intensity noise or long-term fatigue that does not allow recovery. The typical evolution shows a first phase with loss of about 40 decibels in the receiving zone of the frequency of 4000 Hz. At a later stage this loss does not recover even though there are no communicative difficulties. If the noise aggression continues, the lesions extend towards the sensory cells that pick up the vibrations of the frequencies close to the 4000 Hz frequencies and thus a progressive deterioration of the auditory-verbal communicative abilities begins. The hearing loss is stabilized if the worker stops being in contact with the noise. Acute Acoustic Trauma is a disease caused by the impact of high intensity but short duration noise. It requires a great acoustic energy and appears in certain professionals like miners, military, technicians in explosives or in special situations like in random explosions. The clinical symptomatology manifests immediately after the acoustic impact, in the form of tinnitus and hearing loss that can evolve towards its disappearance or remain constant. Chronic acoustic trauma is the auditory deficit caused by continued exposure to noise during work. The presence of deafness depends on the intensity and duration of exposure to noise. This situation is progressive if the noise persists, although factors such as personal susceptibility, age or simultaneity with other pathologies can alter its evolution.

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