
Sketches of Otohistory

Part 10: Noise-Induced Hearing Loss

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The Good Old Days: Immortal Hair Cells

The human ear is well designed for sensitivity and accuracy in receiving and distinguishing among the myriad sounds that reach it, be they informative, pleasurable or otherwise. Probably because injuriously intense sounds are so rare in nature, the ear's own means of protecting itself against overstimulation are feeble and of little effect against cochlear injury by the noises of modern recreation, industry, and warfare.

In ancient days, the noisiest occupations must have been those of the metal forger and armorer. Paradoxically, there is no Mosaic or Homeric record that either the Hebrews' Tubal Cain or the Greeks' god Hephaestos (the Romans' Vulcan) ever complained of deafness. Tubal's record is minimal; he gets only a single line in the book of Genesis. As for Hephaestos, we are told that his forge was in the bowels of a volcano, where he made the thunderbolts for Zeus and the armor for Achilles, as well as implements for agriculture. If he was not deafened by the blows of his own hammer on the anvil, or the subterranean tumult of his abode, we can only infer that his ears had the damage-proof hair cells of a deity. At least in Mediterranean mythology, immortal hair cells long preceded the much-heralded hair cell lines derived from the 'immortomouse'.

The Coming of Middle Age: Boilermakers' Deafness

In past centuries, the civilian ears of blacksmiths and millers, like those of cannoneers, were most likely to be impaired in the pursuit of their noisy professions. Francis Bacon (1571–1625) [see Hawkins, 2004] was one of the first to write of the association between noise and hearing loss stating that 'an object of surcharge destroyeth the same; a violent sound the hearing'. Chaucer's miller may still have had normal hearing, but Robert Stevenson writes, in one of the verses of his *Child's Garden*, of a miller he once knew, 'deaf were his ears from the moil of the mill'. Thus it seems hardly fair that when occupational deafness finally attracted serious otologic research, it was named for the boilermakers, whose ears had merely become its latest and most familiar victims.

It was St. John Roosa [1873] of New York who observed that 'Workmen employed in hammering large iron plates, such as are used in making the boilers of steam engines, are very apt to lose much of their hearing power. So many of these cases were found that at one time "Boilermakers' Deafness" figured as a separate disease in the reports of one of our institutions where aural disease was treated.' In a paper on the etiology of diseases of the inner ear, he sought the lesion responsible for deafness in the labyrinth and attributed it to a concussion of the fibers of the auditory nerve [Roosa, 1874]. In Munich, Friedrich Bezold [1887] blamed target shooting, lues, and mental

overexertion for hearing losses; in Turin, Giuseppe Gradenigo [1889] called such deafness in stonemasons and millers ‘professional otitis interna’.

Cures for deafness were also available. Toynbee [1865] in his *Diseases of the Ear* describes several cases of noise-related hearing loss recognizing that ‘the most common cases are those which follow the long-continued sport of shooting’. As treatment he uses the application of leeches, or cupping, and the administration of mild aperients (laxatives). Although he claimed success in restoring hearing

in several of his patients, his suggestions may not find much credence today.

The cochlear pathology of noise-induced deafness was first described by the Austrian Habermann [1890] in his study of the temporal bones of a single patient. They had been taken from an elderly ex-boilermaker who had been too deaf to heed the loud warnings of the oncoming train that struck him down. Habermann also examined the impaired hearing of living boilermakers from two different shops, and found that men from the larger and noisier one were the more severely deafened.

Bells and Whistles: Experimental Studies in Animals

Wittmaack [1907] conducted the first experimental study of noise-induced deafness in animals, and other studies followed, including those of Yoshii [1909], Marx [1909] and Hössli [1912]. Wittmaack used an electric bell with its arm vibrating against the metal cage floor of his guinea pigs. Although he employed whistles, sirens, organ pipes, and pistol shots as well, he concluded from the more effective noise of the vibrating floor that the cochlear lesions he found had been produced by bone rather than air conduction. Hössli [1912] disproved that conjecture by showing that removal of the incus could protect the animals’ ears from injury. He had used his ‘Hammerwerk’ (fig. 1) to expose guinea pigs to noise that must have come close to that of a busy boiler shop. Hössli captured his results in several elegant plates that show the effects of noise trauma as convincingly as any modern investigation (fig. 2).

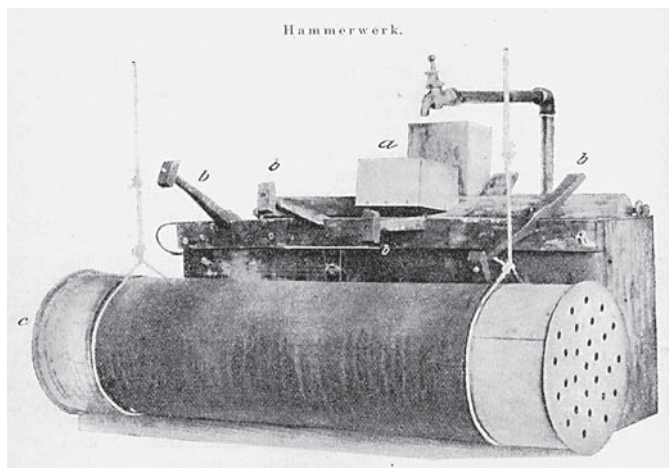


Fig. 1. Hössli’s ‘Hammerwerk’ device. In Hössli’s ingenious contraption, four hammers were driven by water (note the faucet) to pound on a piece of drain pipe. Five guinea pigs could be exposed simultaneously. From Hössli [1912].

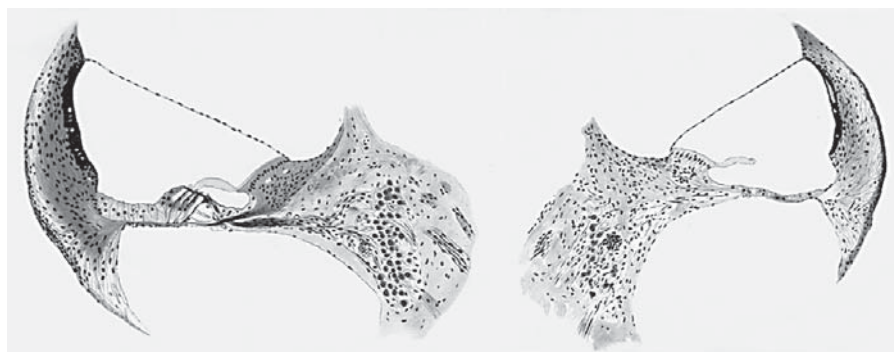


Fig. 2. Sections from the same guinea pig exposed in the Hammerwerk. Left, the ear was protected by incudectomy; right, cochlea from the unprotected ear. Note the almost complete disappearance of the organ of Corti following noise trauma. The original plates are in color. From Hössli [1912].

Human Guinea Pigs: The Harvard Experiments

None of the investigators before the First World War had any satisfactory way of measuring the intensity of the various types of noise exposure they used, or of estimating the degree of auditory change produced. When Hallowell Davis undertook studies of noise-induced hearing loss at Harvard in 1943 [Davis et al., 1943], with human as well as real guinea pigs, both electroacoustic and audiometric equipment had become available. Exposures to tonal frequencies of 0.5–4 kHz or broad-band noise, at 110–140 dB SPL, lasted from 1 to 64 min: the greater the intensity, the shorter the exposure time. Pure-tone thresholds were measured audiometrically, and speech tests were attempted, using recorded word-lists. For hours, or even days, the course of audiometric recovery was followed, and recently exposed ears were not re-exposed until thresholds had returned to normal (fig. 3).

The severity of the hearing losses varied with the intensity, duration, and frequency of the exposure sound. As a rule, the thresholds for tones about one-half octave above the exposure frequency were most affected, but those for lower tones were seldom changed. Broadband noise produced losses over a much greater range of frequencies than did pure tones. Exposure at 4 kHz produced the greatest shift, at 0.5 kHz the least. Thresholds for tones near 4 kHz were the last to return to normal. Subjects differed considerably in their susceptibility to hearing loss, and in the time they needed for recovery. Loudness balance tests clearly showed recruitment. Test tones at the affected frequencies all evoked the same sensation of impure higher pitch, i.e. the phenomenon of *dipacusis binauralis dysharmonica*. Similar wartime studies of noise-induced hearing loss in human and animal subjects were carried out in Switzerland by Rüedi and Furrer [1947], with comparable results.

Dommage à Trois: Cells, Capillaries and Neurons

Loss of hair cells, primarily outer hair cells, in the cochlear region near the exposure frequency is one of the first pathological effects of noise trauma. Following the loss of sensory cells, degeneration of cochlear neurons occurs, the extent of which may determine the failure or success of a potential cochlear implant. Capillary vasoconstriction in the microvasculature has also been a much observed phenomenon that may contribute to its overall manifestations, including tinnitus (fig. 4).

The Louder the Better: Noise-Induced Hearing Loss Today

While in the past the exposure to traumatic levels of noise was confined to small and specific groups of workers, the advent of modern technology has changed this picture. Noise-induced hearing loss has become the most common occupational injury.

The World Health Organization estimates that more than 10% of the world's population are exposed to potentially injurious levels of noise each day. Today, not only are people who operate machinery the victims but also bystanders who experience the racket of street traffic, construction sites or airports. It is no wonder that about 4% of the US population suffer permanent noise-induced hearing loss.

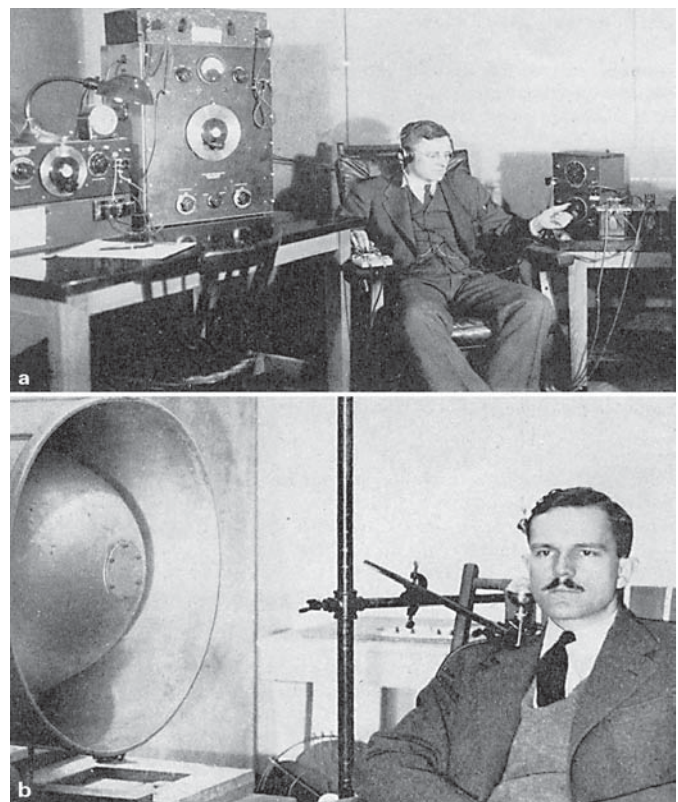


Fig. 3. The Harvard Experiments. **a** Hallowell Davis taking his own audiogram after noise exposure. **b** Subject JEH undergoing exposure of right ear to high-level noise from bull horn, monitored by a sound level meter with a microphone just below the right pinna. The left ear is protected. From Davis et al. [1943].

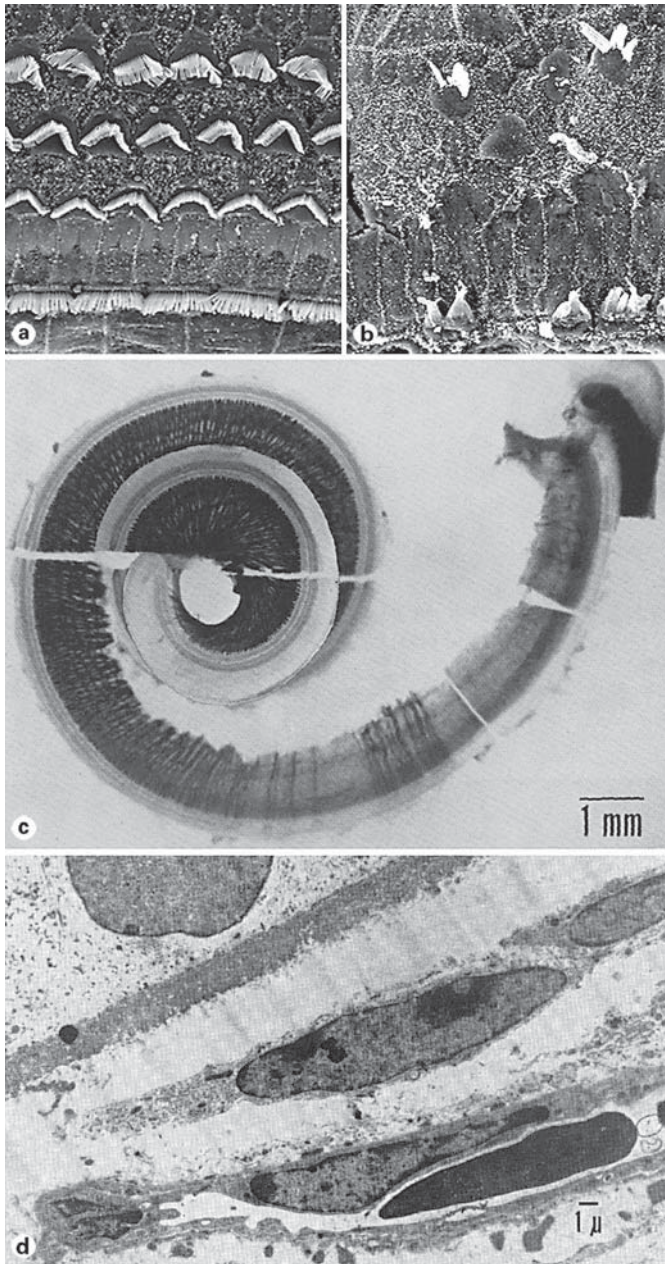


Fig. 4. The morphological triad of noise trauma. **a** Scanning electron micrograph of a section of an intact organ of Corti of guinea pig showing regularly arranged stereocilia of three rows of outer hair cells and one row of inner hair cells. **b** Distorted stereocilia, loss of hair cells and phalangeal scars following noise exposure. Photos courtesy of Drs. Masahiko Izumikawa and Yehoash Raphael. **c** Surface preparation showing loss of Corti's organ and afferent neurons in the basal turn of a cochlea from a 25-year-old man who used shotguns and rifles regularly. From Johnsson [1974]. Reprinted with permission. **d** Capillary vasoconstriction is seen mostly in the microvasculature of the basilar membrane and the supra-strial ligament. A red blood cell is trapped in an outer spiral vessel of a guinea pig. From Hawkins et al. [1971]. Reprinted with permission.

As if occupational stress and the risks of war were not enough, modern times have created new hazards in the environment. Ostensibly intended to relieve stress, *recreational activities* are now contributing to the relentless attacks on our fragile hair cells. The impact of these activities on our hearing is a testimony to our technological achievements.

Firecrackers (up to 170 dB SPL), toy guns (155 dB SPL) or a shotgun (160–170 dB SPL) develop sufficient energy to damage the cochlea mechanically by vibrating the organ of Corti beyond its limits. A single discharge from a high-powered rifle equals the energy of almost 40 h of continuous exposure at 90 dBA. In the US, where there are as many guns as people, recreational shooting is the most important source of noise exposure outside the workplace.

Music has long been an annoyance when played by incompetent musicians, on instruments out of tune, or by children whose maltreatment of classical pieces is sweet only to their parents' ears. Today, music no longer has the connotation of quaint chamber pieces or a pan flute played in the open air. Rock concerts average 103 dB SPL, providing an exposure level of noise illegal in any work place. But then, we may argue that Beethoven already set this trend in 1813 with his composition 'Wellingtons Sieg', only at that time, the orchestral battle sounds lacked the intensity available now in concert halls, stereo systems and the walkman.

Radicals at Work: Mechanisms of Damage and Rescue

Recent decades have brought us an understanding of the underlying mechanisms that doom the hair cells. They can be classified into two broad categories: (1) excessive vibration of the organ of Corti and (2) an increase in the metabolic activity of the sensory cells as a result of the stimulating physical challenge. The idea of mechanical damage is not novel and was already mentioned by the famed French architect and physicist Claude Perrault. In one of his *Essais de Physique* [1680–1688] entitled 'Du Bruit' he expressed the notion that strong vibrations would shatter the hearing organ like glass. The nature of the metabolic events remained more elusive but it is now widely accepted that traumatic noise exposure leads to 'oxidative stress'. The initial events of formation of reactive oxygen species then signal to the molecular machinery that determines cell death or survival. The activation of transcription factors, caspases and other components

of signaling pathways have been confirmed as part of the biochemical cascade leading to the ultimate demise of hair cells.

Protection from noise-induced hearing loss has, in most cases, a simple solution: ear protectors. These, however, are neither fashionable with large segments of society (they appear to be virtually absent from clubs) nor completely effective in harsh environments. However, we can take clues from the studies of the mechanism of noise damage and devise rational approaches to the prevention of hair cell loss.

Three basic, interrelated strategies have been pursued. The first deals with the prevention of the initial events of noise exposure, the generation and action of reactive oxygen species, for example through antioxidant therapy. The second approach intervenes in the downstream events of apoptotic cascades by inhibiting crucial steps in these pathways, for example the activation of c-jun-related transcription factors or caspases. The third method is based on the ever-growing evidence of homeostatic bal-

ances and interactions between cell death and survival pathways. Survival factors, such as growth hormones and neurotrophins, trigger reaction cascades that culminate in the activation of 'survival genes' and the inhibition of apoptotic events. All approaches have yielded promising experimental results.

Such success in animal experimentation should make us hopeful for a clinically applicable procedure. The major problem in translating animal experiments on noise-induced hearing loss to the clinical situation, however, is the 'window of time' allowed for intervention. Most experimental strategies employ drugs given before the noise exposure. This rarely happens in the 'real world' where the otologist as a rule will seldom see the patient before a traumatic noise exposure. When he/she does, hair cells may already have died or be committed to cell death. The window of time that is available for a clinical post-factum intervention is currently unknown, but animal data suggest that at least a small window of time may exist.

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