Significant hearing loss is probably not part of normal aging

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ABSTRACT

Significant (25-40 decibel (dB)) hearing loss is probably not part of normal aging but rather represents sociocusis or pathological aging due to cumulative noise exposure. Seven lines of evidence support this conclusion: 1) preservation of auditory sensitivity throughout life in primitive populations; 2) correlations between occupational noise exposure and hearing loss; 3) better hearing in women than in men, presumably from less noise exposure; 4) significant variations in the prevalence of hearing loss in different population groups; 5) hearing loss occurs in the sound frequencies to which the ear is exposed 6) the prevalence of hearing loss increases with lifetime noise exposure; and 7) basic science research showing noise-induced changes in auditory cells leading to cell damage and death.

Many changes considered part of normal aging, e.g., wrinkled skin or tooth loss, are common but not physiologically normal. These changes result from exposures, poor quality diet, disuse, or suboptimal medical care. Significant hearing loss with age can likely be prevented by reducing lifetime noise exposure.

INTRODUCTION

Physicians, audiologists, and the public think that significant hearing loss is part of normal aging. This is reflected by use of the terms "presbycusis" [1] and "age-related hearing loss" [2], supported by epidemiologic studies showing increasing prevalence of hearing loss with age. [3] Analysis of existing and generally longstanding research, however, strongly suggests that while significant hearing loss may be very common with older age, it is probably not part of normal physiological aging.

This analysis incorporates information from biology, epidemiology, audiology, occupational medicine, geriatric medicine, and other disciplines. The auditory sense appears to have started out as a primitive vibration sense, to help predator species find food and to help prey species avoid getting eaten. Even animals without ears can hear. [4] Over time, this primitive vibration sense evolved into complex ear structures, and animals developed the ability to make sound and to communicate. In primate species, these communications can be quite complex, culminating with the development of speech and language in humans.

The biological bases of aging are not precisely known, with many theories based on genetic or biochemical factors in cells and subcellular structures. In contrast, aging itself is readily apparent even to the most casual observer. Men and women gain weight, hair thins and turns gray, menopause occurs, skin gets wrinkled, hearing gets worse, and eventually everyone dies. The question is: what is normal aging and what is abnormal aging? [5]

The goal of normal aging, also called healthy aging, has been called “compression of morbidity” or “the squaring of the mortality curve”. [6,7] Humans should live full, active, healthy lives well into old age, until they take ill and die quickly, ideally in their mid-to-late-80s or even in our 90s. The fact that disease, disability, and early death are not part of normal aging is shown by the existence of “Blue Zones”, where many live long and active lives, often into their 90s or even over 100. [8] Genetics may play a role, but diet, exercise, social relationships, and a little bit of alcohol help prolong healthy functional life. Obesity [9], hypertension [10], diabetes [11], and heart disease [12] are not part of normal physiological aging. For the auditory system, the goal should be the preservation of hearing throughout life.

Why does it matter if significant hearing loss is probably not part of normal aging, but is really preventable noise-induced hearing loss? Hearing loss has a much greater impact on health than graying hair or wrinkled skin, and it would be much better and far cheaper both for individuals and societies if hearing loss could be prevented. The impacts of hearing loss are underestimated for several reasons. First, except for those paying for hearing aids, the economic impact of hearing loss is limited because it becomes common only in the seventh decade of life, [3] when people are largely out of the work force. Second, hearing loss is invisible. Unlike those with visual impairments, persons with hearing loss are able to walk, drive, and complete their activities of daily living without apparent difficulty. Third, people engage in a variety of compensatory strategies as their hearing worsens: cupping a hand over the ear, trying to find a quiet corner of a restaurant, eating early or late when the restaurant is quieter, asking someone to repeat what was said, pretending to understand by nodding or saying “Uh, huh”, and finally, when communication becomes too difficult, limiting social interaction. Fourth, it is commonly believed that “nobody dies from hearing loss”.

This is not true. Hearing loss worsens social isolation [13] and is correlated with depression, [14] dementia, [15] falls, [16] and death. [17] Hearing aids are only a partial solution for hearing loss in the elderly. Health insurance coverage for hearing aids is limited in the US, and coverage under national health programs in other countries may require meeting strict criteria. Many people with hearing loss do not get hearing aids for reasons of cost, ignorance, or stigma. [18] Hearing aids provide only limited assistance in understanding speech, especially in noisy environments. Because of this, up to 40% of those who have obtained hearing aids do not use them. [19]

ABNORMAL AGING

Discussion of selected topics will illustrate that what is often thought to be normal aging is *not* normal aging but is rather pathological aging or abnormal aging. Many changes occurring in animals over time, most accurately described by physiological and biochemical measures, appear to be normal. In males, testosterone levels begin to fall once maturity is reached. In females, reproductive hormone levels change and eventually the ability to reproduce ceases. The basal metabolism rate begins to fall. The cumulative effect of these physiological changes can be seen in measures of performance. Even for superior athletes, the fastest times

reported for each event decline as athletes get older. But many changes that are thought to be normal aging in humans are not physiologically normal.

Absent specific genetic syndromes, abnormal aging can be ascribed to four basic causes: 1) exposures, 2) poor quality diet, both quantity and quality, 3) disuse atrophy, and 4) suboptimal medical care. The term suboptimal medical care refers not to patients getting poor quality medical care, but rather to patients collectively receiving recommended or standard treatments that are later found to have been suboptimal.

Exposures are the easiest example to understand and perhaps most relevant to noise and hearing loss. The best analogy for the ear being damaged by noise exposure may be the skin being damaged by sun exposure. Ultraviolet light in sunlight damages the skin by causing changes in DNA in dermal cells. [20] The cumulative effect of ultraviolet light on the skin is called photoaging: deep wrinkles, age spots, and skin cancers. [21] Some sagging (ptosis) is the result of changes in collagen over time, but deep wrinkles, color and texture changes, and vascular changes are caused by the sun. Approximately 90% of skin cancers are caused by sun exposure. [22] The reader can compare his or her own exposed to unexposed skin to see the effect of the sun on the skin. Genetic factors are involved, with darker pigmentation protecting the skin from the effects of sun exposure, but the sun affects even the darkest skin.

Another example of an exposure causing abnormal aging is the effect of air pollution on the lung. Lung function decreases with age, but exposure to air pollution is correlated with worse lung function. [23] The effect is most marked in those who smoke, an extreme form of self-induced air pollution. [24] This effect may be compared to hearing loss in those who deliberately expose their ears to loud noise.

Poor quality diet, both in terms of quantity and quality, also causes abnormal aging. Obesity is associated with increased morbidity and mortality, with increased mortality correlated with greater weight. [9] The optimal body-mass index (BMI) is 22-24. [25] Severe caloric restriction has not been well studied in humans, but prolongs life in animal models. [26] Dietary composition is also important, with vegetarian or primarily plant-based diets offering many health benefits. [8,27] Ample research supports the health benefits of the so-called Mediterranean diet. [28,29] Maintaining an ideal body weight, exercising, eating a healthy diet, and not smoking reduce rates of diabetes, heart disease, stroke, and cancer. [30]

Disuse atrophy is also a cause of changes associated with aging. The clearest example is muscle atrophy, which can be prevented by exercise [31]. A recent report, which received widespread media coverage, showed a training effect of exercise in a 105-year-old French cyclist. [32]. Declines in intellectual function may be prevented by good general health, intellectual activity, and maintenance of social networks [33]

Finally, suboptimal medical care due to inadequate and constantly evolving medical knowledge at any point in time can contribute to changes normally thought of as part of aging, which can be prevented in later years by better medical care as new knowledge becomes available. Three examples are hypertension and its treatment, heart disease, and dementia. For decades it was thought that hypertension was part of normal aging, treating hypertension in the elderly was dangerous, and strokes and heart disease couldn’t be prevented. Research is helping refine hypertension treatment goals and strategies, but there is no longer any doubt that treating hypertension even in older people prevents stroke and heart disease. [34]

It was also thought that heart disease was an inevitable part of aging, but studies showing that diet, smoking, and lack of exercise caused coronary artery disease led to dramatic changes in physician practice and patient behavior. Coupled with more aggressive treatment of hypertension, new drugs to treat elevated blood lipids, and a decline in smoking, the rates of heart disease and death from heart disease declined dramatically. [12] Similarly, dementia

was thought to be an invariable part of aging, but recent studies show that treating the risk factors for heart disease also led to decreases in rates of dementia. [35,36].

For hearing loss, the most relevant example of suboptimal care causing changes once thought to be part of normal aging may be found in dentistry. In the 1950s, tooth loss with age was so common as to be considered part of normal aging. In 1960, half of all Americans over 65 years old were edentulous. [37] By 2011, as a result of better diet, better dental care, and perhaps economic progress, only 18.6% of Americans over 65 were edentulous. [38] Another analysis showed 34% of adults age 65-74 living below the U.S. federal poverty level to be edentulous, compared with 13% of adults age 65-74 at or above the federal poverty level. [39] Clearly, being edentulous in old age and needing dentures to eat is not part of normal aging.

Significant (25-40 dB) hearing loss with age is not normal aging, but is most likely the result of lifelong noise exposure causing cumulative auditory damage. Incontrovertible evidence shows that noise exposure causes hearing loss [40,41]; that most people are exposed to excessive noise in everyday life [42]; and that avoidance of noise exposure or use of hearing protective devices prevents hearing loss. Just as the skin should stay unblemished and unwrinkled if not exposed to the sun, hearing should remain normal if the ears are not exposed to loud noise. Just as dentures should not be needed in old age if good dental care is obtained throughout life, hearing aids should not be needed if loud noise is avoided and adequate hearing protection is used throughout life.

EVIDENCE THAT SIGNIFICANT HEARING LOSS IS PROBABLY NOT PART OF NORMAL AGING

Studies of auditory sensitivity in primitive populations

A number of studies of auditory sensitivity in primitive populations done in the 1960s found hearing to be well preserved with age in populations not exposed to noise. By today's standards, the studies are also primitive in terms of design and execution, and need to be replicated, but they offer the only insights available about what happens to human hearing without noise exposure. The best known may be that of Rosen, who studied the Mabaan population in the south Sudan. [43] He reported that the Mabaan lived in conditions approximating the late Stone Age. Rosen found the ambient noise in the Mabaan village to be the lowest he had ever recorded. Older Mabaans had almost as good hearing as younger ones. He reported that two adult Mabaans could converse normally at a distance of 100 meters. [44]

Several studies were done of hearing in different tribal groups in South Africa. Dickson et al studied the Bantu and the Kalahari Bushmen. [45] He reported observations made by another researcher, stating that a Bushman could hear a single engine airplane at a distance of 70 miles (102 km). His own study showed little hearing loss with age in both groups, but that Bushmen did not have better hearing than other groups. Jarvis and van Heerden [46] and van der Sandt, Glorig, and Dickson [47] also studied hearing in the Kalahari Bushmen. Jarvis and van Heerden concluded, "The most striking finding was the absence of presbycusis even in old age." Van der Sandt et al. found better hearing than in other groups, but a high prevalence of chronic ear infections causing hearing loss in younger individuals. They also did not find "super natural hearing" in the Bushmen.

In 1983, Goycoolea et al. reported that natives of Easter Island who had never left the island had better hearing than islanders who had spent time on the South American mainland. [48] It is impossible to determine how much noise exposure those who lived on the mainland

experienced, but Goycoolea et al. reported that length of time on the mainland correlated with the degree of hearing loss. No difference was noted in hearing between men and women who had never left Easter Island.

These studies were all done with what would now be regarded as primitive audiometry equipment, using inadequate testing protocols, and should be repeated with modern equipment and modern protocols if primitive populations not exposed to noise can still be found. But all showed that in populations not exposed to loud noise, hearing did not decline dramatically with age. A 10 dB decrement with age may be normal physiological aging; a 25-40 dB decrement is not.

Occupational studies correlating noise exposure with hearing loss

There can be no rational doubt about the causal relationship of occupational noise exposure to subsequent hearing loss, with clear dose-response curves. Exposure to greater amounts of noise for longer periods of time causes more hearing loss. This body of work, summarized in books [40,41] and monographs, [49] forms the basis of occupational noise exposure limits in many countries including the United States and the European Union.

Studies showing that human females have better hearing than males

At birth, male and female infants have similar hearing, but beginning in the second decade of life, auditory sensitivity in males declines. This decline continues throughout life. [50,51,52] Also Chapter 4 and Table 4.2 in [41] The explanation is thought to be greater noise exposure in boys and men, initially from recreational activities such as hunting, playing loud musical instruments, or motor sports, and later through occupational noise exposures. Men historically have worked in factories, used power tools, or used heavy construction equipment. It was rare for women to work in noisy environments, except perhaps in textile mills. Flamme's study of noise exposure in everyday life also found that men had greater noise exposure than women. [42] Studies of mice models show no gender difference in hearing loss with age in CBA/CaJ mice. [53]

Significant variations in the prevalence of hearing loss in different population groups

If hearing loss was part of normal physiological aging, one would expect to find similar rates of hearing loss in all populations, just as almost all older individuals anywhere in the world have gray hair, often thinning gray hair. When studies show significant variations in the prevalence of hearing loss in different populations, there clearly must be some factor or factors at work. These could include genetics, ear infections in younger persons, or exposures to ototoxic drugs or chemicals, but the most likely cause in older populations is different rates of noise exposure. The longer the exposure, the greater the hearing loss. ([54,55] and Chapter 4 and Table 4.2 in [41])

Hearing loss occurs in the frequencies to which the ear is exposed

If hearing loss was part of normal aging, one might expect that it to be distributed across all frequencies. Instead, hearing loss occurs in the frequencies to which the ear is exposed, which unfortunately include the frequencies of human speech. (Chapter 4 in [41]). Specific exposure to noise in certain frequency bands causes hearing loss in those frequencies, as shown in studies of high frequency hearing loss in dentists from long and repeated exposure to turbine powered equipment. [56,57]. Animal studies show both hearing loss and anatomic

changes in cochlear cells specific to the frequency range to which the animals are exposed, and these changes are similar to those seen in humans. [58,59] There may be other factors involved, relating to the structure of the cochlea with the parts of the cochlea sensing higher frequencies being closer to the tympanic membrane, where they may receive impulses of greater intensity, but this also implies an effect of noise on hearing loss.

The audiometric notch is a decrement in hearing at 4000 Hertz (Hz) readily seen on pure-tone audiometry and is considered characteristic of noise induced hearing loss. [55, 60] Human noise exposure is usually broadband in frequency, and incoming sound is shaped by passage through the external and middle ears. [61] For humans, resonance in the ear canal produces amplification of acoustic frequencies at 4000 Hz, which is the location of the audiometric notch seen on audiograms. [62] The presence of the audiometric notch in those without occupational exposure [63] strongly implies that everyday noise exposure [42] is causing hearing loss. The audiogram of “presbycusis” shows a gradual decline in hearing in higher frequencies, without a notch. This does not mean that hearing loss with age is not caused by noise. As McBride [60] states, “the notch broadens with increasing exposure, and may eventually become indistinguishable from the changes of aging (presbycusis) where the hearing shows a gradual deterioration at the high frequencies.”

The prevalence of hearing loss increases with lifetime noise exposure

The prevalence of hearing loss increases with age [16,55] as the ear is exposed to more noise over time. Stated another way, hearing loss with age is likely the result of the cumulative total noise dose received. Of course, just the passage of time could account for this, with multiple other factors having a potential effect on hearing, including genetic differences, aging, viral and bacterial infections, ototoxic drugs and chemicals, and interactions between these factors. [64] As noted earlier, this is the common wisdom, embodied in the terms *presbycusis* and *age-related hearing loss*. But many lines of evidence strongly suggest that the dramatic rise in the prevalence of hearing loss with age is unlikely to be just part of normal aging. There is no ready explanation for the extreme prevalence of hearing loss in persons in the ninth and tenth decades of life. This may include factors common in late life that may make the aged ear less able to recover from auditory trauma, such as diabetes, hypertension, vascular disease, and decreases in anti-oxidant enzymes, or it may just be additional years of cumulative noise exposure.

Basic science research showing changes in auditory cells caused by noise, leading to cell damage and death.

In addition to observational studies correlating noise exposure with hearing loss, ample basic science research shows that noise exposure causes biochemical, genetic, and structural changes in cells and subcellular structures in the auditory system, leading to cell and organelle damage and death. The understanding of exactly how noise damages the structures of hearing, specifically cochlear hair cells and auditory synapses, leading to measureable noise-induced hearing loss, is well advanced. [65,66] There is hope that better understanding of these biochemical, genetic, and structural effects of noise will eventually lead to the development of drugs to prevent hearing loss, or even to restore hearing.

CONCLUSION AND RECOMMENDATIONS

The combination of observational and epidemiologic studies showing a clear dose-response relationship between noise exposure and hearing loss, combined with basic science studies explaining how noise damages auditory cells, strongly suggest that significant hearing loss with age is *not* a part of normal physiological aging, but rather the cumulative effect of noise exposure on the ear over time. The proof of this hypothesis would require well-done longitudinal studies measuring noise exposure, ideally continuously, and auditory sensitivity. Periodic sampling of noise exposures in populations, combined with serial audiometry, would be more feasible, but might miss infrequent loud noise exposures, especially impulsive or intermittent noise.

Those studies are unlikely to be done. In their absence, we are left with the evidence summarized in this paper. Based on this evidence, significant hearing loss with age is the result of noise exposure, and should be called “sociocusis” or “noise induced hearing loss.” [50] The terms “presbycusis” and “age-related hearing loss” should be relegated to the dustbin of history.

More research is always needed to better understand relationships among exposures, dietary intakes, activity levels, genetic differences, smoking, and other factors contributing to overall health or to a specific health measure. What should public health authorities recommend while additional research on aging and hearing loss is done? In 1969, in the first publication on noise and public health, Dr. William Stewart, a former Surgeon General of the United States, said. “In protecting health, absolute proof comes too late. To wait for it is to invite disaster or to prolong suffering unnecessarily.” [67]
In public health practice, prevention is almost always better and cheaper than treatment, which in turn is better and cheaper than rehabilitation. [68] To prevent hearing loss, the only evidence-based safe noise exposure, calculated by the US Environmental Protection Agency, is a time weighted average of 70 decibels for 24 hours. [69,70] This noise dose is the same as 75 decibels for 8 hours, or 85 decibels for one hour. [63] Greater noise exposure will cause hearing loss.

If something sounds too loud, it *is* too loud. Accurate sound level meter applications are available for smart phones [71] but if one can’t carry on a normal conversation, the ambient sound level is above 75 decibels (dBA) [Figure D-1 in 68] and auditory damage is occurring. If the sound causes temporary muffling or tinnitus, permanent auditory damage has been sustained. [72] Government health and environmental agencies must issue strict noise regulations for indoor places, power tools, consumer appliances, vehicles, and aircraft to protect the public’s hearing. Limiting government efforts to making better hearing aids and hearing health care available more cheaply and more easily to older people with hearing loss [73,74] is entirely insufficient.

No more research is needed to know that noise causes hearing loss, that significant hearing loss in old age is probably not part of normal aging, and that reducing noise exposure will preserve hearing well into old age. The time for action to make the world quieter is now.

Acknowledgements

I want to acknowledge the support and editorial suggestions of Jamie Banks MS PhD, Gina Briggs JD, Bryan Pollard EE, and David Sykes; the assistance of Janet Wulf and Rosie Murray at the Cedars-Sinai Medical Library, Cedars-Sinai Medical Center, Los Angeles, CA in obtaining references to older literature not available online; and my wife, Ruth Cousineau, MD. My only goal is to find a quiet restaurant in which to enjoy the conversation and the meal with her.

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