BIOELECTRONIC DEVICES

Why hearing aids are impaired

An analysis of the neural coding of speech sounds in anaesthetized gerbils shows that sound-processing algorithms used by hearing aids can degrade the wearer's ability to discriminate sounds.

John C. Middlebrooks

Hearing aids are often unused, even in the developed countries where they are readily available. Why? The most common answer is that they simply don't work very well[1]. Although hearing aids may help to detect a faint sound or to understand speech at a moderate level in quiet surroundings, they fail to provide clear and comfortable speech recognition in many real-world environments. In young children, mild-to-moderate hearing loss impairs communication skills, social development and performance in school[2]. In the elderly, it can lead to social isolation[3], and is a major preventable cause of dementia[4]. Several hundreds of millions of people worldwide suffer from mild-to-moderate hearing loss, for which the current treatment of choice is a hearing aid[5].

At the most basic level, a hearing aid is an amplifier that boosts sound levels in the ear canal; it replaces the intrinsic amplifier of the inner ear that is lost with deafness. Hearing aids can be customized for individual users, to amplify specific sound frequencies in proportion to the user's degree of hearing loss. Also, most modern hearing aids can be fitted with hardware and software for wide dynamic range compression (WDRC). WDRC amplifies sound proportionally to the levels of the incoming frequencies, with respectively more or less gain for low or high incoming sound levels, thereby replacing some of the compression of the dynamic range of sounds that is provided by normal inner-ear mechanics. Reporting in *Nature Biomedical Engineering*, Nicholas Lesica and colleagues now show that commonly applied sound-processing algorithms used in hearing aids degrade the discrimination of speech sounds at a critical junction of the ascending auditory pathway, and identify sound-processing strategies that can restore sound discrimination[6].

Lesica and co-authors studied the responses of populations of neurons to sounds from human speech (consonants followed by varying vowels, either spoken by one talker or by multiple talkers) in anaesthetized gerbils. Gerbils are a favoured model for human hearing because, unlike most other small animals, their hearing frequency range overlaps with much of the human low-frequency range (sound perception at higher frequency is more specific to the gerbil and not as applicable to human-speech perception). In the authors' study, stimuli were presented to gerbils with normal hearing and to gerbils that were exposed to high sound levels, so as to induce mild-to-moderate hearing loss. The stimuli were unprocessed, processed by simple linear amplification, or processed by combining linear amplification and WDRC. In gerbils, all auditory stimuli are relayed through the inferior colliculus of the midbrain to reach the auditory cortex for hearing perception. The authors examined how speech sounds were coded by the activity of large populations of neurons in the inferior colliculus, by evaluating the features of speech sounds that are transformed to neural activity by the cochlea of the inner ear, conducted to the brain by

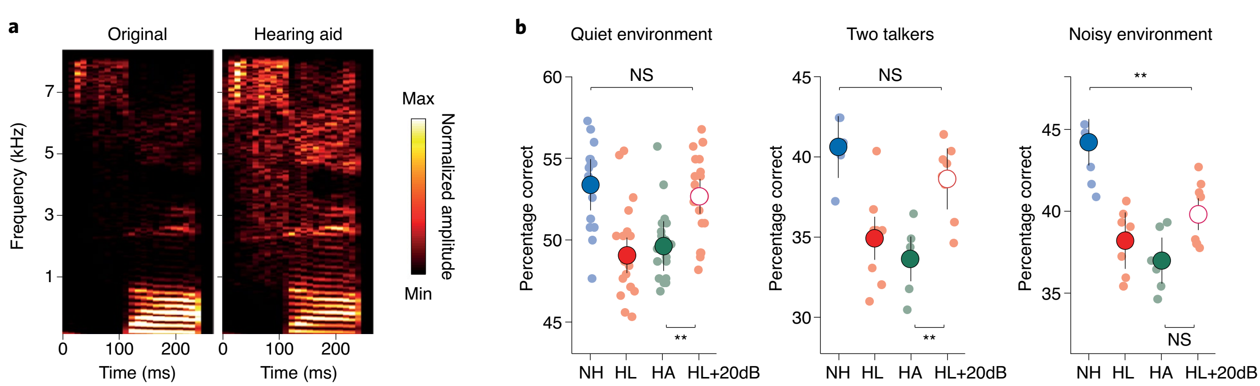

Fig. 1 | Sound compression in hearing aids impairs the discrimination of speech sounds. a, Spectrograms for the syllable "za" before (left) and after (right) sound processing with a hearing aid. **b**, Percentage of instances in which consonants were correctly identified by a classifier (a support vector machine) trained to identify them on the basis of spectrogram data from single-trial responses to speech at 62 dB in quiet conditions (left), in the presence of ongoing speech from a second talker at equal intensity (centre), and in the presence of babble noise at equal intensity (right), for gerbils with normal hearing (NH), gerbils with hearing loss (HL), gerbils with hearing loss and with sound processed by a conventional hearing aid that included compression (HA), and gerbils with hearing loss and with sound processed only by linear amplification (with a fixed gain of 20 dB across all frequencies; HL+20 dB). The error bars denote the mean and 95% confidence intervals derived from bootstrap re-sampling across populations of neurons in gerbils. $^{**}P<0.01$; NS, not significant. Figure reproduced with permission from ref. [6], Springer Nature Ltd.

the auditory nerve, and that emerge intact from the first few stages of the auditory pathway.

To evaluate the neural code, Lesica and co-authors recorded neuronal spike patterns, from 512 electrode channels implanted bilaterally in the gerbil inferior colliculi, in response to sets of the processed and unprocessed speech sounds. That yielded a 512-dimensional spatiotemporal trajectory for each sound (Fig. 1a). A support vector machine (a machine-learning classifier) was then used to identify the consonant that corresponded to each trajectory. The classification accuracy was high for recordings from normal-hearing gerbils in response to unprocessed speech. Some consonants were more recognizable than others, in agreement with studies of human speech perception[7]. The performance of the classifier was less successful in gerbils with hearing loss; it led to a reduction in neural-spike counts and to impaired sound detection. However, simple tone-evoked spike counts and sound detection were restored by conventional hearing-aid processing. Similarly, a conventional hearing aid with amplification and WDRC restored the identification of the frequencies of pure tones in the hearing-loss recordings.

In contrast to the identification of pure tones, the identification of complex sounds (in this case, consonants) was severely degraded in recordings from the hearing-impaired animals (Fig. 1b). Moreover, consonant identification could not be restored by amplification combined with WDRC, whereas amplification alone restored consonant classification to normal levels in conditions equivalent to those of a quiet background or in the presence of one other talker (Fig. 1b). To identify aspects of the neural code that were disrupted by sound compression, Lesica and colleagues decomposed the neural code into distinct noise and signal components. The noise consisted of 'internal noise', which was related to basic limitations of neural coding, and 'nuisance noise', which reflected variation in the talkers and in the vowels that followed the consonants. The signal consisted of a 'common signal', which was common to all the consonants, and a 'differential signal', which distinguished the consonant sounds. In the animals with hearing loss, all of the noise and signal components except the differential signal were restored (but reduced in magnitude) by conventional hearing-aid processing. This suggests that it was the compression of the differential signal that was responsible for the degradation of consonant identification in speech processed by a conventional hearing aid.

If simple linear amplification without compression restored consonant identification in hearing-impaired animals, whereas WDRC degraded identification, why not simply switch off the WDRC? The answer is that, without compression of the dynamic range, amplification fixed at a level sufficiently strong to make quiet sounds audible would also amplify moderate-level sounds to unacceptably high levels. Not only would these high sound levels be physically uncomfortable, but discrimination of speech sounds would be degraded owing to 'rollover', a well-known phenomenon at high sound levels that is especially a problem for speech discrimination in the presence of background noise. The mechanisms of rollover are not well understood, but probably reflect a loss of sensitivity to spectral contrast, owing to the broadening of the frequency tuning of neurons at high sound levels. Lesica and colleagues found that, as a result of rollover, consonant identification at high sound levels was equally poor in both normal-hearing gerbils and hearing-impaired gerbils.

Hence, without dynamic-range compression, hearing aids can provide either minimal amplification, in which case low-level sound would be inaudible, or strong amplification, in which case speech discrimination would be degraded by rollover. Nevertheless, for people with mild hearing loss, a little linear amplification from a simple hearing aid could do a lot of good[8]. The user might have difficulty understanding speech at high sound levels in the presence of background noise, but even those with normal hearing find it difficult in such conditions. Extrapolating Lesica and colleagues' analysis of neural coding of sounds in the gerbil midbrain to human perception suggests that many people with mild hearing loss would be better off without WDRC. Moreover, hearing aids comprising only the essential elements for amplification could be produced at lower cost, and thus made more widely available.

Lesica and colleagues' findings seem to warrant a call for simpler hearing aids. Yet more work is needed to improve hearing-aid technology. For example, people with greater degrees of hearing loss need higher levels of sound amplification, and with that comes the problem of rollover. Today's sound-compression algorithms for the suppression of rollover solve the problem of uncomfortable loudness in conditions of high ambient sound levels, but in doing so they flatten the spectral contrast between adjacent frequency components. We need algorithms that can enhance the spectral contrast in the signal received by an impaired auditory system. Then, enhanced cues for speech-sound discrimination might arrive intact at the level of the midbrain to be passed on to the forebrain for effective speech recognition and comprehension. Because such contrast enhancement is not yet available for hearing aids, a credible solution for hundreds of millions of people with mild-to-moderate hearing loss is to use hearing aids without sound compression. ❐

John C. Middlebrooks ✉

Departments of Otolaryngology, Neurobiology & Behavior, Cognitive Sciences, and Biomedical Engineering, University of California at Irvine, Irvine, CA, USA.

✉*e-mail: j.midd@uci.edu*

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Competing interests

The author declares no competing interests.