

Auditory Neuropathy: Why some hearing-impaired listeners can hear but do not understand and how can DSP technology help them?

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1. Introduction

Hearing impairment affects almost 30 million Americans and costs the nation more than \$56 billion annually (Dana Alliance for Brain Initiatives, 1996). To improve communication among these hearing-impaired people, various assistive listening devices from hearing aids to cochlear implants have been developed and achieved remarkable results. However, there have been consistent observations that some hearing-impaired people do not benefit from conventional hearing aids and their degrees of hearing loss may not justify the use of cochlear implants. These people often complain: "I can hear but do not understand." Their speech understanding problem is especially aggregated under realistic listening situations where noise and reverberations are abundant. Recent studies have identified a new type of hearing impairment, auditory neuropathy, which appears to have normal cochlear amplification function but abnormal auditory nerve activities (Worthington and Peters, 1980; Kraus et al., 1984; Starr et al., 1990; 1991; 1996). We have found that suprathreshold temporal processing is a major factor contributing to speech recognition problem in patients with auditory neuropathy (Zeng et al., 1999).

Based on the sites of lesion, hearing loss has been traditionally classified into conductive and sensorineural types. The conductive hearing loss usually indicates blockage or damage of the external and/or middle ears. The simplest form of conductive hearing loss is to plug the ear canal with your fingers, which could result in 20 to 30 dB attenuation. Another common form of conductive hearing loss is due to liquid filled in the middle ear cavity in cases of middle ear infection. Clinically, conductive hearing loss can be distinguished from sensorineural hearing loss based on simple tests such as air- vs. bone-conduction thresholds. Conductive hearing loss may be thought as linear attenuation and can be compensated for by surgery or a simple hearing aid with linear amplification circuits.

The sensorineural hearing loss usually refers to damage in the inner ear or the nerve pathway. The most common hearing loss due to noise exposure, ototoxic drugs, and aging is associated with damaged nonlinear amplification function provided by the outer hair cells in the inner ear (cochlea). Patients with cochlear loss typically demonstrates loudness recruitment (a steeper than normal growth function in loudness) and derives significant benefits from hearing aids using nonlinear compression circuits. Contrary to cochlear loss, auditory neuropathy preserves normal cochlear amplification function, as indicated by the presence of otoacoustic emission, but disrupts synchronous activities in the auditory nerve,

as reflected by the abnormal evoked brainstem responses. These patients can definitely perceive sounds, and usually have normal cortical potentials and negative brain imaging results, indicating normal central auditory structure and functions. At present, the exact pathology that disrupts the neural synchrony is not known and may include loss of inner hair cells, abnormal synaptic function, and/or demyelination in the auditory nerve fiber.

Here we report behavioral evidence for relating neural synchrony to perception in human subjects who had been diagnosed with auditory neuropathy. We use a synthesis-by-analysis technique to simulate various degrees of auditory neuropathy in normal hearing-hearing listeners and obtain speech recognition deficits similar to that found in neuropathy listeners. Finally we discuss novel processing algorithms that could contribute to the diagnosis and treatment of their speech recognition problem.

2. Methods

The methods are briefly discussed here as the detailed methods have been described elsewhere (Zeng et al., 1999). Ten auditory neuropathy subjects, 5 females and 5 males, participated in this study. These subjects were aged from 10 to 53 years old with an average age of 28 years old. Their pure-tone averaged thresholds (500, 1000, and 2000 Hz) ranged from 15 to 82 dB with an average of 48 dB HL. Their speech recognition scores (NU-6 words) ranged from 0% to 66% correct with an average of 22% correct. All subjects, except for AN5, had measurable otoacoustic emissions (3 to 16 dB), but none of them had normal auditory brainstem evoked responses. In addition, the imaging results using MRI and CT were normal in all 5 subjects tested.

In intensity and frequency discrimination, tonal stimuli of 200 ms in duration were used. In temporal processing experiment, sinusoids of different frequencies were used to modulate a broad-band (20-14,000 Hz) white noise. The noise had a 500-ms duration and was presented at a maximal comfortable level on an individual basis. All stimuli had 3-ms, cosine-squared ramps. They were generated digitally using TDT System II and presented to the subject through either an insert earphone (Etymotic ER2). NU-6 word lists were used in speech recognition.

We used a 3-alternative, forced-choice, 2-down, 1-up, adaptive procedure in all psychophysical tests. This procedure produces a 70.7% correct response on the psychometric function. In these psychophysical tests, subjects had to identify which of the three listening intervals contained a signal. The signal was the louder sound in intensity discrimination, had higher pitch in frequency discrimination, and contained modulations in the temporal processing experiment. In speech recognition, percent correct scores were obtained by calculating the number of correctly identified words over the total number of presented words.

3. Results

3.1 Intensity and frequency processing

Figure 1 shows normative data (shaded areas) and data from several neuropathy subjects (symbols) in intensity and frequency discrimination. When similar sensation levels were used, neuropathy subjects performed generally with the normal range in intensity discrimination. However, their frequency discrimination showed an interesting abnormal

pattern, which was about 1 order of magnitude worse than the normal for frequencies below 2000 Hz but was within the normal range at frequencies at 4000 Hz and above.

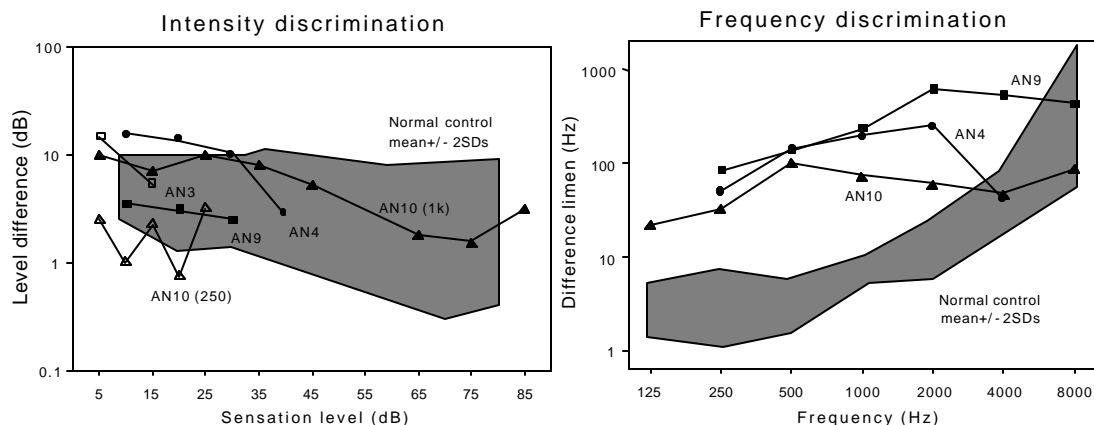


Figure 1 Intensity discrimination (left panel) and frequency discrimination (right panel). The shaded area represents the mean plus and minus 2 standard deviations from 4 normal-hearing subjects. Intensity discrimination data were collected with a 200-ms, 1000-Hz tone. Frequency discrimination data were collected with a 200-ms, 90-dB SPL tone. In subject AN10, intensity discrimination was measured at 250 and 1000 Hz.

3.2 Temporal processing

Figure 2 shows temporal processing measures from 10 neuropathy subjects and three controls including the normal-hearing listeners (white filled area: mean \pm 2 SDs), the cochlear-impaired subject and the normal ear in the unilateral neuropathy subject.

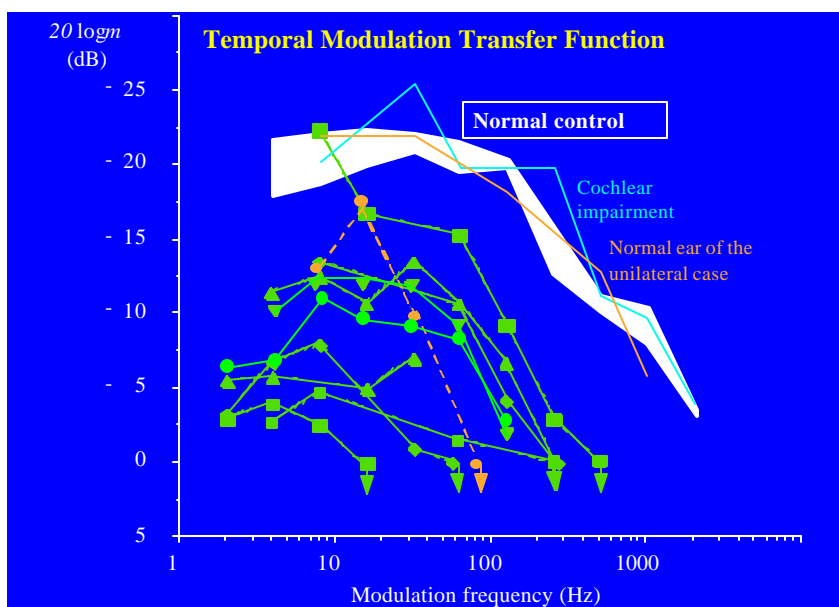


Figure 2 Temporal modulation transfer functions in normal (white areas) and neuropathy (symbols) subjects. The two additional controls (lines) included a cochlear-impaired subject and the normal ear in a unilateral neuropathy subjects. The x-axis is the sinusoidal modulation frequency and the y-axis is a logarithmic function of modulation depth (0 dB indicating 100% modulation and -20 dB indicating 10% modulation).

Normal-hearing listeners showed a low-pass characteristic in their modulation transfer function: being able to detect about 10% modulation depth for modulation frequencies lower than 100 Hz and dropping off at a rate of -3 to -6 dB per doubling frequency above 100 Hz. The two controls, the cochlear-impaired and the normal ear in the unilateral case, showed a normal modulation transfer function. On the contrary, all 10 neuropathy subjects, including the affected ear (dashed orange line) in the unilateral case, showed abnormal modulation transfer functions. Not only did they require greater degrees of modulations than normals (as much as 20 dB difference, or an order of magnitude difference) to detect the temporal envelope fluctuations, but they could not follow the high frequency modulations as well as the normals (the average 3-dB cutoff frequency was 238 Hz for normals and 52 Hz for neuropathy subjects).

3.3 Simulation of auditory neuropathy

The obtained modulation transfer functions provide a quantitative measure of the functional deficit in temporal processing in neuropathy subjects and also allow us to simulate various degrees of auditory neuropathy in normal-hearing listeners. Figure 3 shows a block diagram of the simulation using an analysis-by-synthesis approach (Drullman, 1994; Zeng et al., 1999). Briefly, speech sounds were divided into 1/3-octave bands and within each band, the fine structure was extracted by an LPC-based method while the temporal envelope was extracted by a half-wave rectifier and a low-pass filter. To simulate different degrees of auditory neuropathy, the gain and the low-cutoff frequency of the envelope low-pass filter were manipulated according to the modulation detection functions in neuropathy subjects. For example, to simulate the severe neuropathy case, the modulation depth was reduced by 12 dB and the low-cutoff frequency was lowered to 25 Hz. Normal-hearing listeners were tested with this simulation to see whether similar speech recognition deficits can be observed. Audio demonstrations of the simulation can be found on the web site (<http://www.bsos.umd.edu/hesp/zeng/>) and will be presented at the meeting.

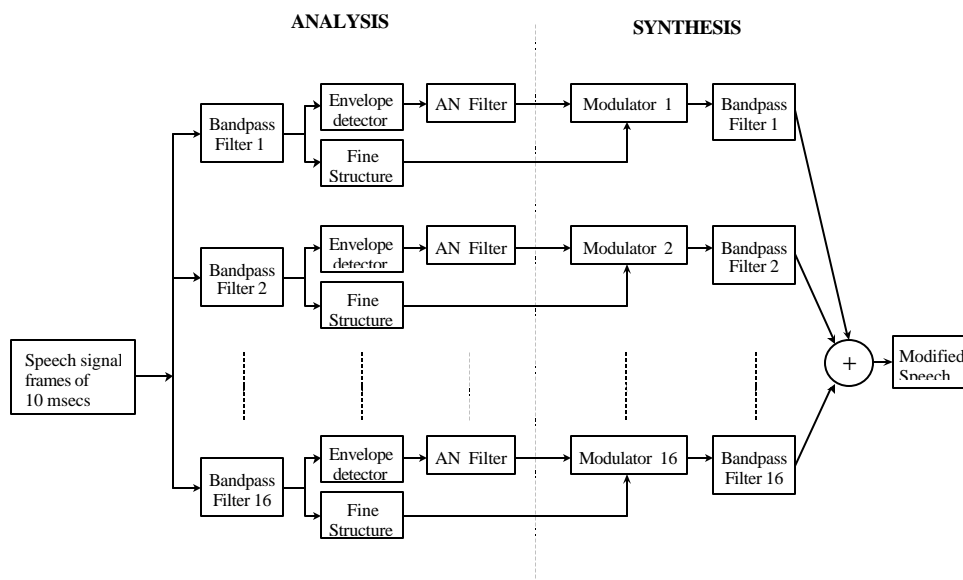


Figure 3. Simulation of auditory neuropathy. The simulation was implemented using MATLAB and can be implemented using a real-time DSP technique.

Figure 4 shows speech waveforms based on simulation of various degrees of auditory neuropathy. The original waveform (leftmost) has clearly identifiable peaks and valleys, which are smoothed out as the severity of neuropathy is increased from mild (about 3 dB reduction in the modulation depth) to profound (18 dB reduction).

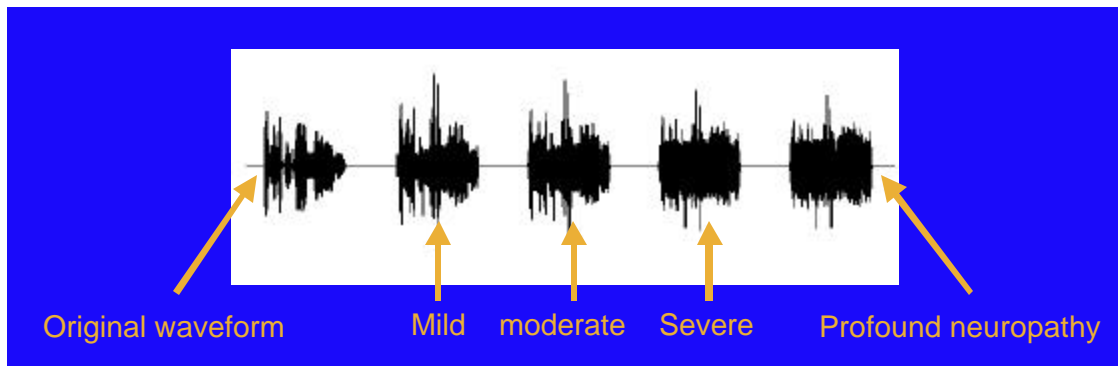


Figure 4. Speech waveforms based on simulations of different degrees of auditory neuropathy.

Figure 5 contrasts the actual data obtained in the neuropathy subjects (numbers) and the simulation results in normal-hearing listeners (shaded area). The simulation results show that speech recognition approaches 0% correct level under simulated severe to profound neuropathy conditions. Except for neuropathy subjects, #8 and #9, the actual data all fall within the normal range, indicating that, to a large extent, the temporal processing deficit can account for the speech recognition problem in auditory neuropathy subjects.

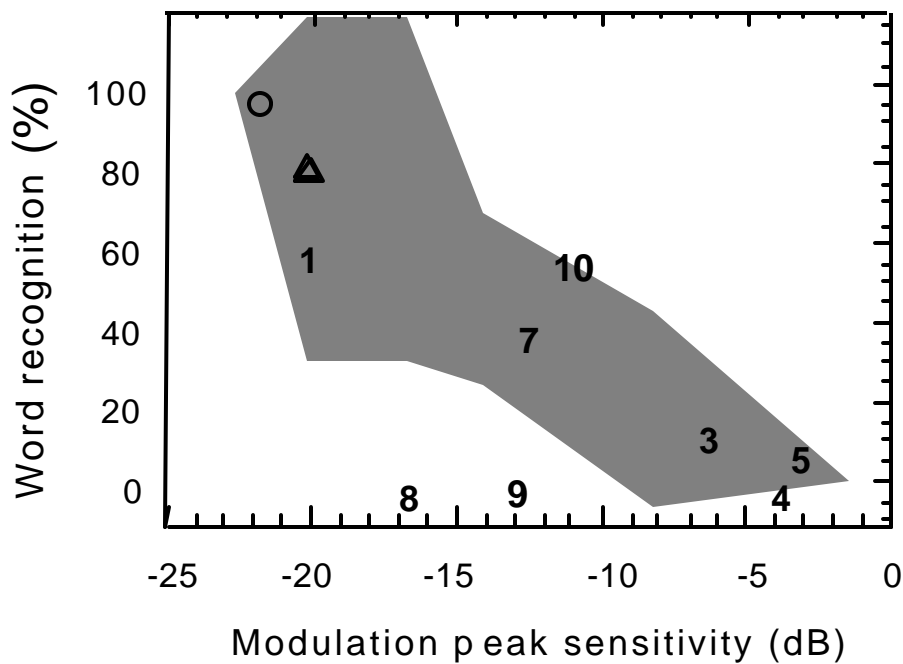


Figure 5. Simulation results of auditory neuropathy in normal-hearing listeners (shaded area). Word recognition (y-axis) decreases monotonically as a function of modulation peak sensitivity (x-axis). The circle represents the normal ear in the unilateral case and the triangle represents the cochlear-impaired subject.

4. Discussion

Our preliminary data show that intensity processing is not significantly affected by auditory neuropathy. Frequency discrimination is significantly affected at low frequencies but not at high frequencies, possibly reflecting the time versus place principle of pitch coding at these frequencies. The demonstrated temporal processing deficits in auditory neuropathy provide direct evidence for an important role of neural synchrony in auditory perception. The present data can also account for the speech recognition deficit that is disproportional to pure-tone hearing loss. They can further suggest new directions for aural rehabilitation of auditory neuropathy. According to the most recent report on young child hearing screening in Australia, about 10% of the children with permanent hearing loss had auditory neuropathy (Rance et al., 1999). Since the available hearing aids either do not change the temporal envelope (linear amplification) or reduce the modulation depth (compression circuits), they offer little help to patients with auditory neuropathy. New hearing aids that accentuate the temporal envelope or cochlear implants that produce highly synchronous neural activity may be more effective than the conventional hearing aids in the clinical management of auditory neuropathy. Real-time DSP technology should be able to implement such an envelope expansion algorithm and may help solve the “I can hear but do not understand” problem.

5. Acknowledgements

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