New Thinking on Hearing in Noise: A Generalized Articulation Index

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ABSTRACT

The articulation index (AI) theory serves to predict word recognition scores when the number of speech cues have been reduced by noise or lack of audibility. It fails to predict how poorly some subjects do in noise, even when all speech cues have been made audible with amplification. Such subjects require an unusually large signal-to-noise ratio (SNR) for a given performance level, and are said to have a large SNR loss. We have found that the AI can be generalized to predict word recognition scores in the case of missing (speech cue) dots. Some speech cues appear to be lost on the way to the brain even though they were audible. Corliss[1] suggested the term *channel capacity* to describe this phenomenon, and we adopt that term for our use. In this article, the substantial psychoacoustic and physiological evidence in favor of this generalized AI is described. Perhaps the strongest evidence is (1) the SNR loss of subjects is poorly predicted by the degree of their audiometric loss, and (2) their wideband word-recognition performance in noise can be predicted from their channel capacity inferred from filtered speech experiments.

KEYWORDS: Articulation Index, signal-to-noise ratio loss, channel capacity, missing dots, audibility

Learning Outcomes: Upon completion of this article, the reader will be able to define and discuss channel capacity, signal-to-noise ratio loss, the importance of audibility to speech understanding, and choices in sensory aids that impact audibility and SNR losses.

The informal title of this article summarizes the essential argument: Inaudible dots and missing dots (dis)appear all the same to the brain.

The "dots" refer to the graphical representation of speech cues in the Count-the-Dots audiogram method for estimating the Articulation Index (AI) shown in Figure 1. Any of the count-the-dots methods shown by Mueller and Killion,[2] Humes,[3] or Pavlovic,[4] will give similar results. In the Mueller and Killion method, each of the 100 dots represents 1% of

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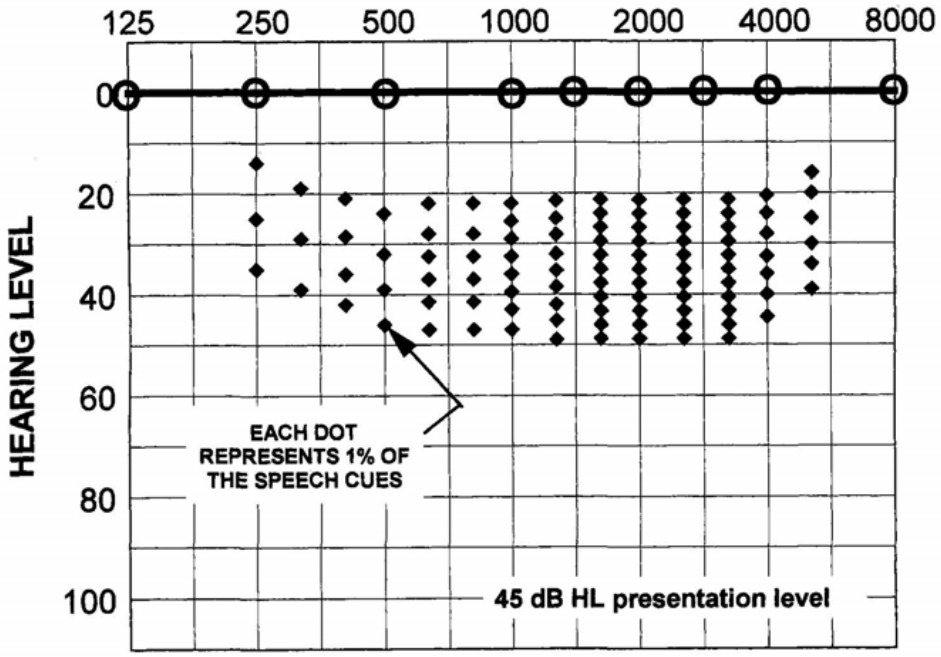

Figure 1 Count-the-dots audiogram for estimating AI (articulation index). Adapted from Mueller and Killion.[2]

the available speech cues in ordinary speech. The relative density of dots at a given frequency reflects the relative importance of the speech cues in that frequency region.

In attempting to explain the difficulties that hearing-impaired persons often experience in understanding speech in noise, previous formulae used addition to describe the two contributions to hearing in noise: (1) audibility and (2) the proportion of undistorted audible information that reaches the brain. A better estimate results if we use multiplication instead of addition. Thus, the essential suggestion of this article is the following formula:

$$\text{perceptive AI} = (\text{audibility AI}) \times (\text{channel capacity})$$

AUDIBILITY AND CHANNEL CAPACITY

The formula stated above can be understood intuitively. If only half the audible speech cue dots actually reach the brain (perhaps half the inner hair cells are missing), then it seems reasonable that such a person will need to have twice as many speech cues made audible in order to reach the same level of performance on a speech-in-noise task as a normal-hearing person. As shown in Figure 2, someone with normal hearing may be able to carry on a conversation with only 25% of the speech cues audible (AI = 25%). When only half the audible dots reach the brain, however, we need to start with an extra 25% (a total of 50%) in order for 25% to reach the brain.

There are several terms that might be used to describe loss of speech cues arriving at the brain. Internal AI suggests itself. The original AI formulae used a proficiency factor that was multiplied by the audibility portion to reach the final AI, which took into account the amount of practice a listening crew had. The use of information theory's term *channel capacity* was suggested some time ago by Corliss[1] in this context. It seems to be the most intuitive of the terms, and is used here.

Because the inner hair cells generate all the auditory signals to the brain, and loss of inner hair cells often accompanies hearing loss, it is perhaps no surprise that hearing in noise is a problem for many hearing aid wearers. This

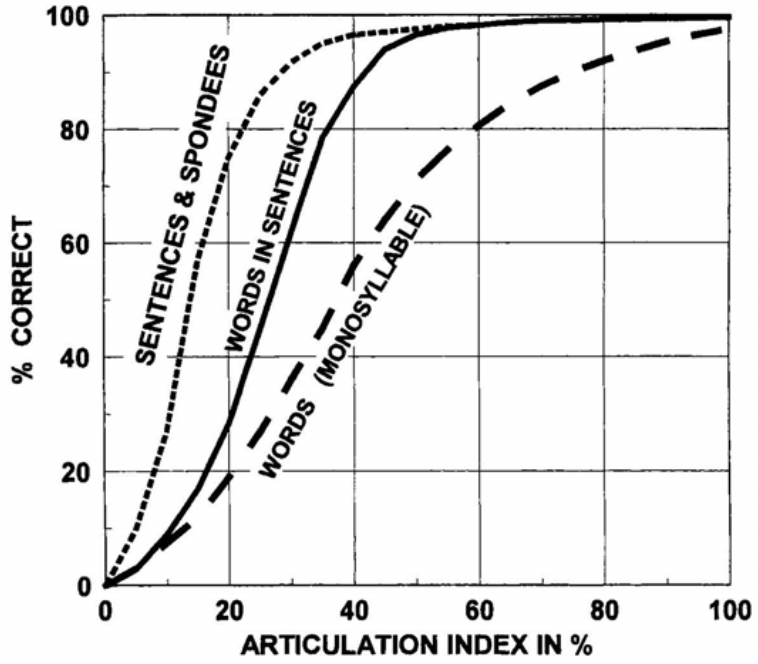

Figure 2 Relationship between AI and percent correct for spondees or sentences, words in sentences, and isolated monosyllables.[18]

article attempts to summarize what we can quantify about the problem and its origins.

Just as we speak of the pure tone loss, we may speak of SNR loss. We define SNR loss as the increased signal-to-noise ratio compared to that normally required by a subject to repeat 50% of words in sentences correctly. In the example shown in Figure 3, normal subjects require 2 dB SNR for 50% correct on the Speech-In-Noise (SIN) Test, while the example subject requires 8 dB. The latter subject thus has a 6 dB SNR loss.

SUBJECT DATA

The Minimal Relationship between SNR Loss and the PTA

Some 30 years ago, Tillman, Carhart and Olsen[5] reported a series of measurements on normal hearing and presbyacousic subjects in which the subjects' ability to understand words in a single-competing-talker noise was measured for different SNRs. One can estimate an average deficit of 12 dB for their subjects with presbyacousis, as shown in Figure 4.

Somewhat later, Plomp[6] argued that the ability of subjects to hear in noise was reduced by two separate elements: an attenuation A and a distortion D in the cochlea. Present in varying amounts, the D factor helped explain why hearing aids were more successful for some listeners than others in restoring their ability to hear in noise.

Subsequently, Dirks, Morgan and Dubno[7] described an adaptive method for rapid determination of SNR-50 (signal-to-noise ratio required for 50% correct recognition of words) using a 12-talker babble for the noise. By comparing the performance of hearing-impaired subjects with that of normal hearing subjects, they obtained the equivalent of SNR loss, and presented data on two individual subjects: one with a 30 dB pure-tone average (PTA) loss and a 2 dB SNR loss, and the other with a 45 dB pure-tone average and a 12 dB SNR loss. The 20 subjects with SNR loss in this study (Fig. 5) had a relatively narrow range of audiometric loss (actually speech-reception thresholds, typically approximately equal to the pure-tone averages), ranging from 10 dB HL to 49 dB HL, but their SNR loss ranged from 0 dB to 28 dB,

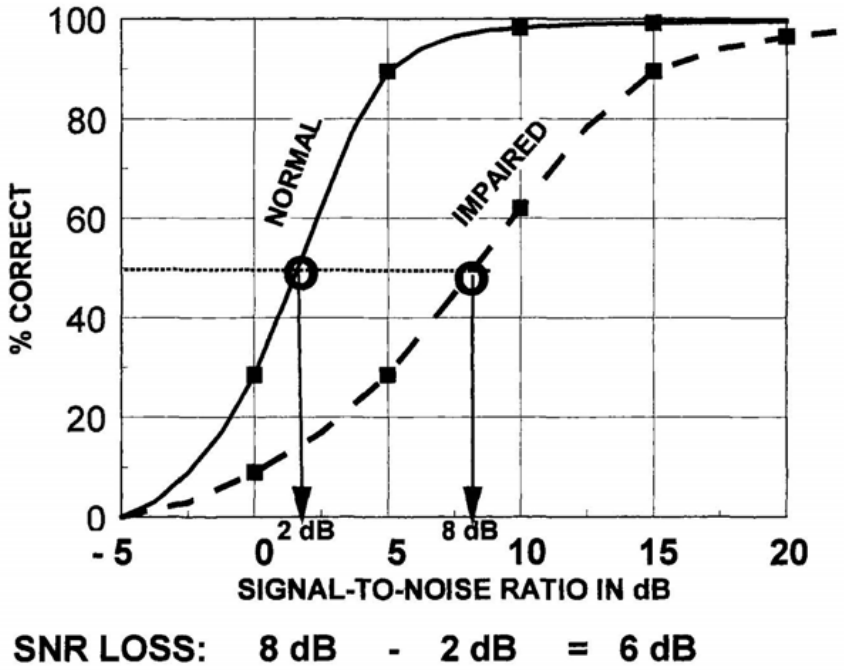

Figure 3 Illustration of SNR loss: The difference between normal and impaired SNR required for 50% correct identification of words in sentences.

even at 76 dB HL (91 dB SPL) presentation level.

Dirks et al[7] stated: "The [SNR] deficit at high presentation levels must be evidence of a characteristic of sensorineural hearing loss other than threshold sensitivity loss for speech."

The same year in which Dirks and colleagues published their study, Lyregaard[8] published the data shown in Figure 6, indicating that subjects with the **same** PTA thresholds could have a 15 dB difference in SNR loss. In other words, there was no way to predict the SNR loss from the audiogram.

More recent data from both the Hearing-In-Noise Test (HINT) and the SIN Test (Fig. 7) both show 15 to 20 dB differences in SNR loss for hearing-impaired subjects having nearly the same PTA—and, in some cases, similar audiometric configurations.

The conclusion is clear: the difficulty a patient will have understanding speech in noise cannot be predicted from the patient's audiogram. As a corollary, we conclude that judging from patients' complaints, we have not been measuring that which is most important to many of our patients: their SNR loss.

Fortunately, estimating SNR loss is now fairly easy. The QuickSIN (quick speech in noise) Test is a recent speeded-up version of the SIN Test that permits an SNR estimate good to ±3 dB in slightly over one minute.[9] Each block contains six sentences presented at sequentially worsening SNRs, starting at 25 dB and dropping in 5 dB steps to 0 dB (Fig. 8).

Another simple test has been introduced by Moore.[10] In this case, a spectrally shaped masking noise is used to identify dead regions in the cochlea (see the following discussion).

THE CASE FOR SNR TESTING

Not having measured the degree of difficulty our patients have hearing in noise, we are surprised when some patients with 50–60 dB loss tell us their hearing aids are absolutely wonderful, and others with the same threshold loss return to our office twice a month to complain

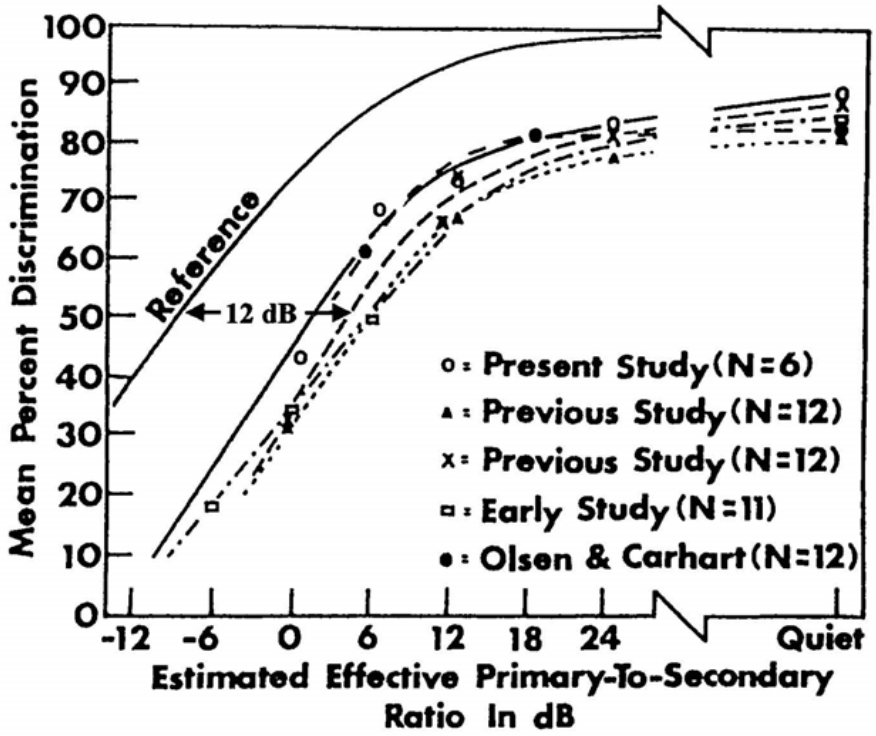

Figure 4 Loss in ability of impaired listeners to hear against competing talker in sound field listening. Results for normals shown as reference. From Carhart and Tillman, [5] with permission.

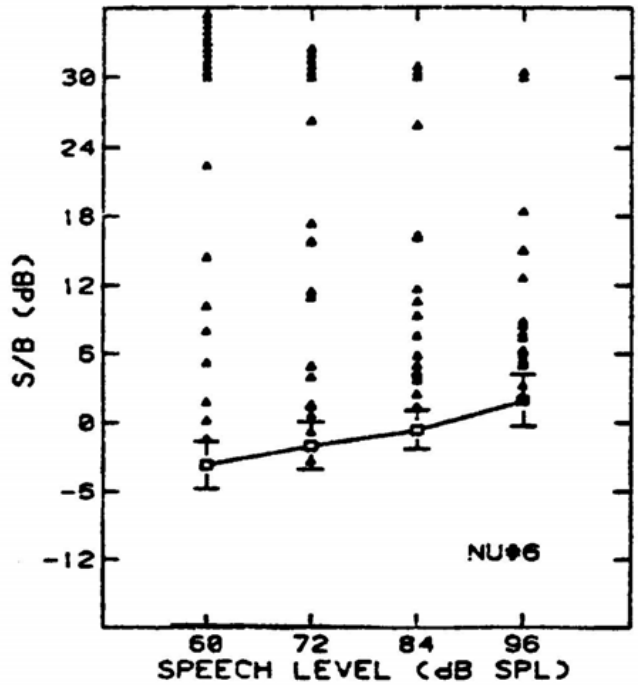

Figure 5 Loss in ability of impaired listeners to hear in noise, earphone listening. Results for normals shown for comparison. From Dirks et al, [7] with permission.

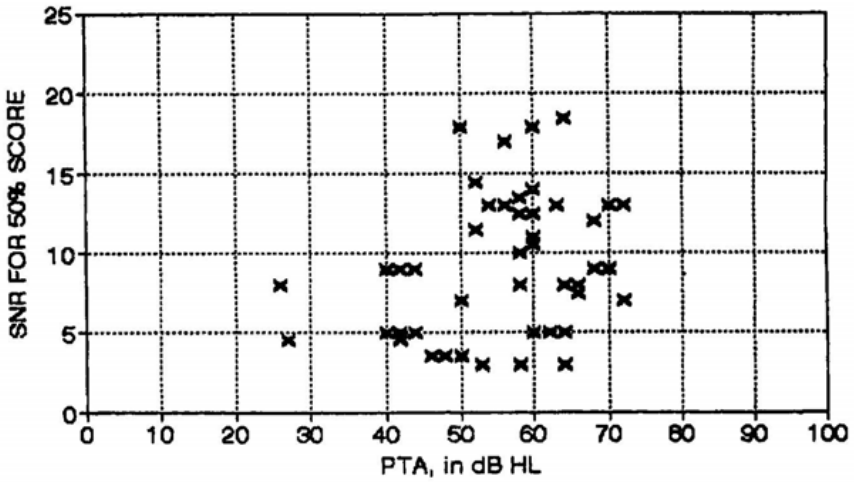

Figure 6 Signal to noise ratio required for 50% words correct vs. hearing loss (three-frequency average). CVCVs in sentences against speech spectrum noise sensorineural-loss subjects. Data from Lyregaard,[8] with permission.

that we do not have their hearing aids adjusted correctly or ask if there is not some new technology that would do a better job. A colleague, Laurel Christensen (personal communication, 2001) reports that when she measured her most satisfied hearing-impaired patient (one who had sent her many referrals), she found he had almost no SNR loss. In contrast, the patient who came in repeatedly for adjustments to her hearing aids, accompanied by complaints about them, had an SNR loss exceeding 20 dB. (At the highest SNR on the original SIN test, 15 dB, that patient could only repeat a few out of the 25 words.)

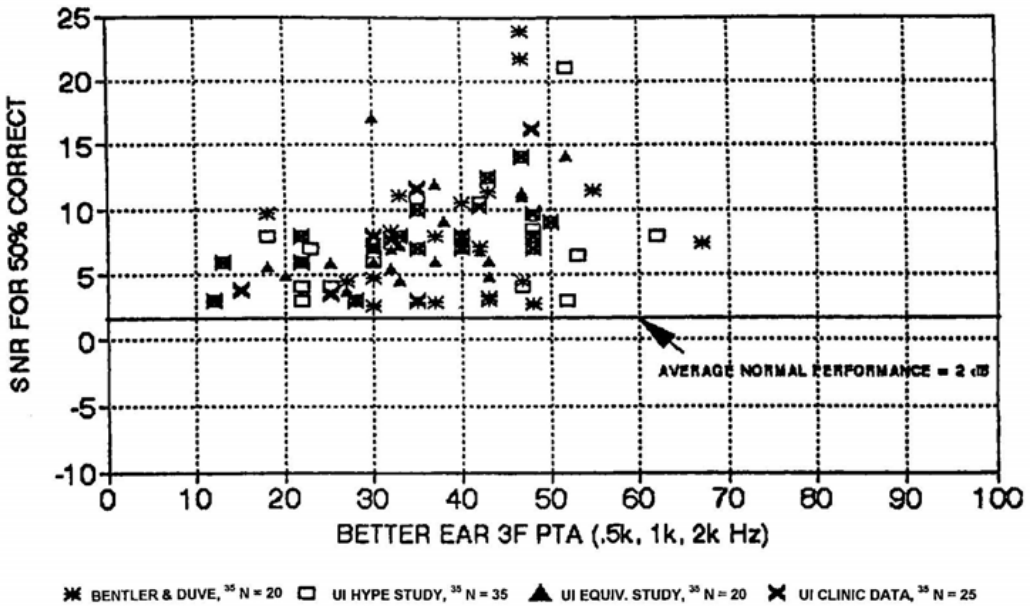

Figure 7 Signal to noise ratio required for 50% words correct vs hearing loss (three-frequency average). SIN test at 83 dB SPL. UI, University of Iowa. From Killion and Niquette,[28] with permission.

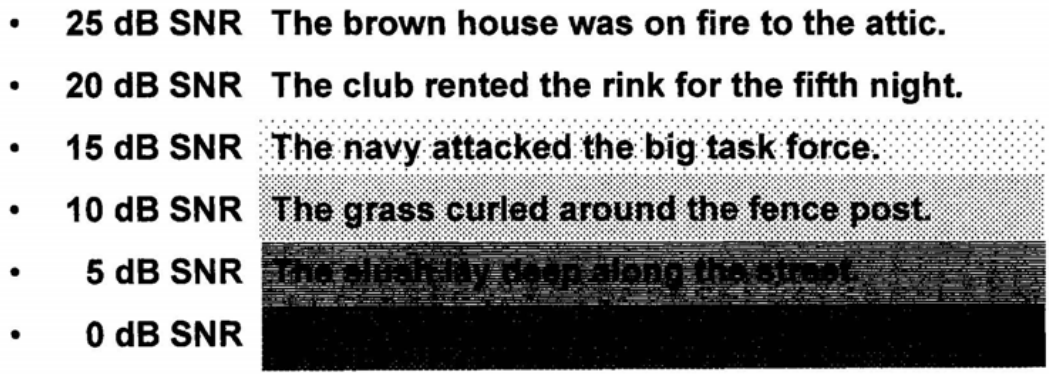

Figure 8 Visual illustration of single block of QuickSIN test, showing progressively poorer signal-to-noise ratio with each succeeding sentence.

My own experience was similar. I took on a difficult patient that no audiologist at a local university would see any longer (she regularly screamed at clinicians, accusing them of all sorts of things including cheating her out of her money, and threatening to go to the president of the university to have them dismissed). Shielded by her belief that I am a wonderful clinician, I regularly see her to check her hearing aids and reassure her that I have done the best I know how. Her SNR loss on the SIN test is 16 dB. Her loss for hearing in noise is such that she will not hear any better no matter what circuit I might try. She cannot understand any speech in common social situations where significant background noise is present, although her audiogram indicates only a moderate-severe flat loss. She has so far declined to purchase the directional-microphone hearing aids that would help her in some situations.

SNR testing has an important additional application in the clinic.[31] After being tested for SNR loss, patients sometimes comment "This is the first time anyone has really tested my hearing!" Counseling is easier when the patient believes you have measured the problem most important to them.

Distribution of SNR loss

As stated previously, we formally define SNR loss as the difference in SNR required to understand 50% of words in sentences by a hearing-impaired subject compared to that of an average normal hearing subject.

A preliminary estimate of the distribution of SNR loss is shown in Figure 9. Taking into account that SNR-50 for normal listeners on the SIN test is 2 dB, it appears that nearly half of hearing aid purchasers have a 4 dB or greater SNR loss. More precisely, half have an SNR loss greater than 4.2 dB. However, the opposite of this is encouraging: half of hearing aid purchasers have an SNR loss less or equal to 4.2 dB, which means that the better of the modern directional microphone hearing aids—that have an effective noise reduction in typical settings of 4 to 6 dB—should be able to bring half of hearing aid wearers back to normal ability to hear in noise, or even better than normal.

THE MISSING DOTS EXPLAIN EVERYTHING

Since the 1920s, the Bell Laboratory's Articulation Index or AI (now mistakenly—in my view—called the Speech Intelligibility Index in the latest ANSI standard) has been a good predictor of intelligibility in quiet and noise for normal-hearing subjects.[11] For such subjects, the AI nicely predicts word recognition scores when intelligibility has been reduced by filtering, masking, or low presentation levels. How many dots are required for spondees?

Figure 1 shows a "Count the Dot Audiogram" modification of the AI. The dots on the

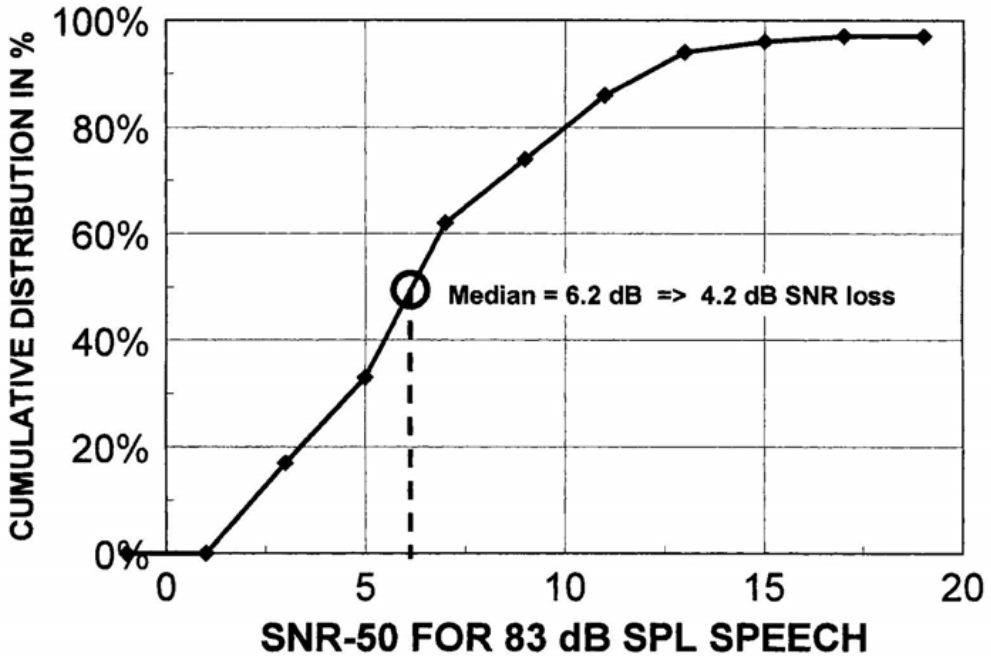

Figure 9 Distribution of SNR loss among 110 hearing aid purchasers.

AI audiogram in Figure 1 correspond to 45 dB HL speech (about 60 dB SPL). A presentation level 10 dB higher would move each dot down 10 dB on the audiogram (to higher HL values). The calibration of the speech audiometer circuit in audiometers is set so that a normal-hearing subject will repeat 50% of spondee words (e.g., hothouse, northwest, sidewalk, birthday) at 0 dB HL (15 dB SPL in the sound field) presentation level. Similarly, if we present 45 dB HL speech to a patient with a 45 dB flat loss (pure tone thresholds of 45 dB at each frequency), they will also be able to repeat 50% of such words correctly if they do not have any SNR loss. In both cases, only the most intense 5 dB of the speech cues are audible, but that 5 dB contains some 15% of the speech cues, which is all that is required for the task. As indicated by the "Sentences & Spondees" curve in Figure 2, spondees are extremely easy. (As a check on understanding, draw a line at 45 dB HL across the audiogram in Figure 1, and confirm that 14 or 15 of the speech-cue dots are left audible by 45 dB HL speech presented to someone with a 45 dB hearing loss.)

As illustrated by the case of spondee thresholds, only a small number of speech cues are needed for speech. Speech is a highly redundant code.[12] For example, Figure 2 indicates that only about 26% of the speech cues are required by a normal-hearing person to understand 90% of sentences. Indeed, the situation at a typical social gathering such as a cocktail party is illustrated in Figure 10, where the diamond symbols represent the desired talker and the nondiamond symbols suggest the masking by other talkers at the party.

While a normal-hearing person can understand speech with 75% of the speech cues masked, someone with a hearing loss may not be able to do so. Plomp[6] suggested that there are two ways to lose speech cues, attenuation and distortion, which we will treat, respectively, as:

1. Lack of audibility (you do not hear the cues because they are masked by noise or are below threshold) and
2. audible but not entirely useful (all the cues do not reach the brain intact).

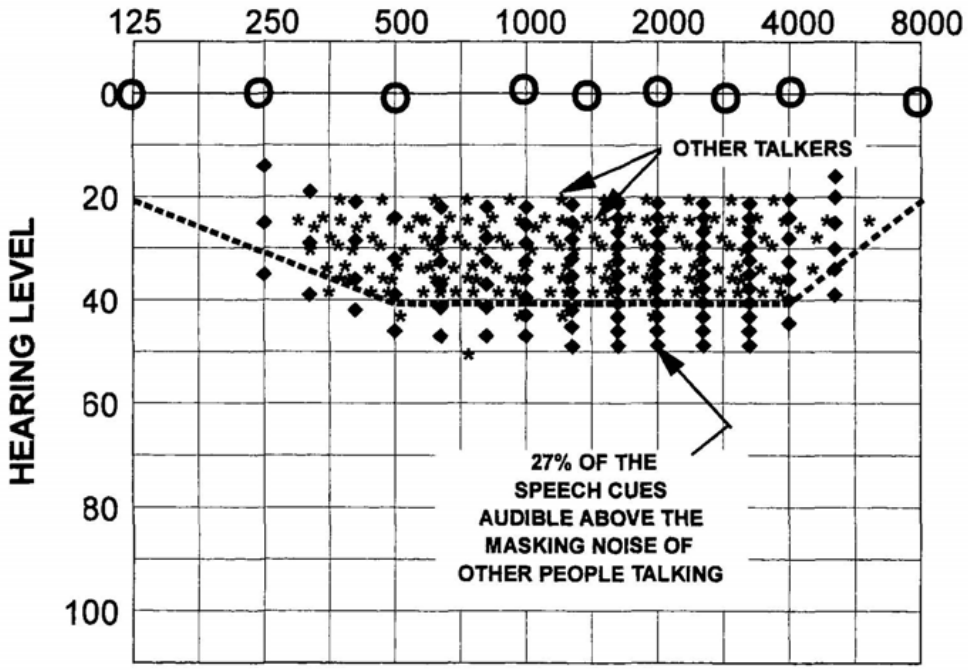

Figure 10 Illustration of masking by other talkers, leaving 27% of the desired talker's speech cues audible.

These two deficits are often combined, so that some speech cues fail to reach the brain because they are not audible while additional ones fail to reach the brain because they are lost along the way. These combined effects are discussed later in this article.

Audibility AI vs. Channel Capacity

We already have illustrated loss of cues in the examples of a flat hearing loss or of masking noise at a party. Here we are dealing with an audibility problem, and we may almost completely describe the problem in terms of the traditional AI, which we will call the Audibility AI to emphasize its essential nature.

Assume a patient has 99% of their inner hair cells missing above 1.5 kHz. The patient with such a loss might be expected to operate much the same as someone with normal hearing listening to speech subjected to low-pass brick-wall filtering at 1.5 kHz. (Inner hair cell loss is an intuitive explanation for SNR loss. A discussion of other possible mechanisms occurs later.) Because about half the speech cues lie above 1.5 kHz, half the speech cues would fail to reach the brain in either case. Assuming our patient's hearing loss was such that no sounds above 1.5 kHz were audible, we would simply say that AI = 50%.

However, let us assume we can provide enough high frequency gain that all of the speech cues somehow become audible to our patient. This seems unreasonable at first, but a sufficiently intense signal can perhaps stimulate inner hair cells in remote regions, or a few surviving inner hair cells provide audibility but no useable information to the brain.

Such things do happen. Years ago, I was fitting a friend who had ski-slope hearing loss that dropped to 70–80 dB above 2 kHz. No attempt to provide amplification in that frequency region seemed to help. I finally thought to ask him what he heard when I presented tones at 3 and 4 kHz. Depending on frequency and level, he reported a hum, screech, or buzz, but never a tone.

More recently, Skinner,[13] Rankovic,[14] Turner[15] and Moore[10] all reported cases in which providing the high-frequency amplification re-

quired to make the high-frequency speech cues audible reduced word recognition scores. Not only does audibility without recognition not increase intelligibility, it can decrease it. Such patients, when queried, often report that a high-frequency tone does not sound like a tone. In this case, we can speak of "black hole dots" or "minus dots." Making speech cues more audible in that frequency region **decreases** the information reaching the brain.

After sufficient amplification, our hypothetical patient with virtually no inner hair cells above (basal to) 1.5 kHz may have a calculated AI of 100%. This is calculated on the basis of the patient's detection response to high-frequency stimuli. When presented with normal speech, however, he responds as if he has an AI of 50%. When presented with speech having intense high-frequency emphasis, he may respond as if he has an AI of less than 50%.

These observations suggest that we introduce a new AI that we will call "Perceptive AI." This better estimates the number of speech cues that actually reach the brain. We can then write:

$$\text{Perceptive AI} = (\text{AI}) \times (\text{Channel Capacity}) \quad \text{(Formula 1)},$$

where Channel Capacity gives the percentage of **audible** speech cues that are accurately passed on to the brain. In this equation, AI and Channel Capacity are expressed as decimal fractions. If broadband masking reduces the AI to 50% and the Channel Capacity is reduced to 50% because half of the inner hair cells are missing, the Perceptive AI becomes $0.5 \times 0.5 = 0.25$ or 25%.

Even before we had audiometers, it was known that some people had an inordinate amount of difficulty in noise and could "hear but not understand." For a shorter formula, we can rewrite formula 1 as:

$$\text{pAI} = \text{AI} \times \text{CC} \quad \text{(Formula 2)}$$

Three examples: A musician friend of mine had an eighth nerve schwanoma that gradually reduced his ability to understand speech, but his thresholds stayed at 0 dB HL. In fact, he exhibited a 0 dB HL detection threshold at all audiometric frequencies until well after his speech recognition score had dropped to zero. At the end he could detect the presence of sound, but nothing else. In this case the traditional audibility AI was 100%, but the channel capacity was close to 0%.

With the introduction of carboplatin, it has become possible to induce a loss of inner hair cells in animals while sparing many of their outer hair cells. Wynne (personal communcation, 2001) reported that (human) patients given carboplatin as part of a chemotherapy treatment often end up with hearing loss and word-recognition difficulties. One of Wynne's patients, whose chemotherapy was successful for its intended purpose, had normal hearing and 100% word recognition scores bilaterally before treatment. After treatment, he had a pure-tone loss of 80–100 dB and word recognition scores in quiet of 16% in the left ear and 10% in the right ear. What was unusual about this patient was that his outer hair cell function—as assessed by transient-evoked otoacoustic emissions—remained significant (although not normal) after treatment. From the curve for "Words" in Figure 2, we can estimate his loss of channel capacity as 80–90%. That is, if everything is made audible, 10% correct on monosyllabic words in quiet corresponds to a channel capacity of only 10%.

More recently, Jardine et al[16] used carboplatin to reduce inner hair cell count while retaining outer hair cells in chinchilla in order to estimate the number of inner hair cells required for normal threshold. They reported that only a small portion of inner hair cells were required to maintain thresholds in quiet, but that thresholds in noise rose substantially with loss of inner hair cells. Following Corliss'[1] general approach, they suggest that inner hair cell damage reduces the number of transmission lines.

Third, patients with cochlear implants may exhibit pure-tone thresholds consistent with an AI of 100%, yet they do not perform normally in noise. Having said that, the performance of some persons with today's cochlear-implant processors and electrode arrays seems almost miraculous. Indeed, some

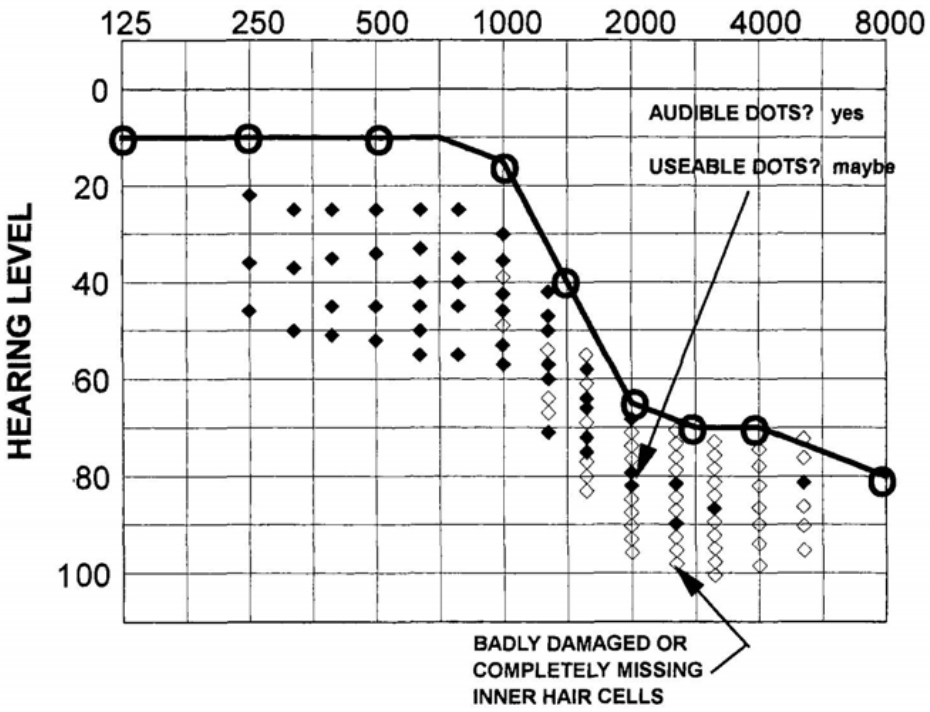

Figure 11 Illustration of amplified speech cues that might be audible, but only half reach the brain because of damaged or missing inner hair cells.

cochlear implant patients' performance in noise is so good that they behave as if they had a channel capacity of 25 to 50% (Skinner, personal communication of SIN test results, 2001). By Formula 1, if 100% audibility is assured, a channel capacity of about 25% is required to carry on a telephone conversation. In other words, 25% audibility and 100% channel capacity can be expected to give the same performance as 100% audibility and 25% channel capacity.

Figure 11 shows a graphical representation of loss of channel capacity. In that figure, solid squares represent audible speech cues that are passed on accurately to the brain, whereas open squares represent speech cues that have been made audible with amplification but do not reach the brain with sufficient accuracy to be useful.

The effect of reduced channel capacity on SNR loss can be readily calculated theoretically. Figure 12, from Killion and Christensen,[17] shows the SNR loss expected from missing dots (left ordinate) or remaining dots (channel capacity) of 20 to 100%.

The derivation of the curves in Figure 12 is straightforward: For any Audibility AI (abscissa), only half of speech cue dots reach the brain of someone with a 50% channel capacity. (Applying Formula 2 in this case, Perceptive AI = AI × CC = AI × 0.5). Thus, if a Perceptive AI of 26% is required for 50% words correct, someone with normal hearing needs an AI of only 26%, someone with a channel capacity of 50% needs an Audibility AI twice as large, or 52%, and someone with a channel capacity of 25% needs an impossible Audibility AI of 104%. Nonetheless, Figure 12 underscores the first order of business for hearing aid design and fitting: Increase audibility. As Villchur[18] stated, ". . . probably the most important approach to the noise problem is to empower the selective-listening ability of hearing-impaired persons by restoring to their perception [audibility] acoustical speech cues they have lost." And in his now classic statement,

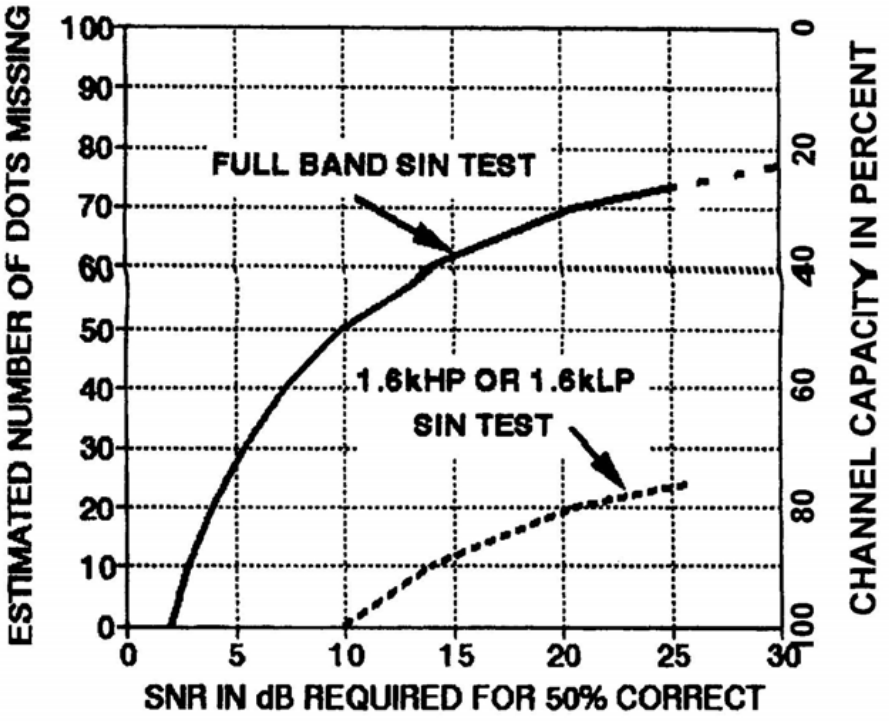

Figure 12 Graph for estimating missing dots or channel capacity from full-band and 1600-Hz low pass or high pass SIN test.

Pascoe[19] observed: "Although it is true that mere detection of a sound does not ensure its recognition, it is even more true that without detection the probabilities of correct identification are greatly diminished."

It is easy to show theoretically that a channel capacity loss of 50% produces an expected 8 dB SNR loss, and a slope for word recognition scores vs. AI half that of normal, assuming everything is audible. This agrees almost perfectly with our experimental data.[17]

There is a third perceptive loss that often is discussed—the loss of central processing ability or processing speed, usually with age. We will ignore that loss here, and assume the patients under discussion have an intact central processor with adequate clock speed.

SEARCH FOR MECHANISMS

There are two types of hair cells in the cochlea (Fig. 13). The outer hair cells almost certainly provide mechanical amplification of quiet sounds.[20]

A relatively innocuous pure tone loss can come from a loss of outer hair cells only; innocuous in the sense that SNR loss will be minimal and the audibility loss can be corrected by amplification.

All the signals to the brain, however, appear to come from inner hair cells.[20] As discussed above, assuming 75% of the inner hair cells are missing or inoperative (and assuming a one-to-one correlation between inner hair cell function and speech cues that make it to the brain intact), someone with a 75% inner hair cell loss should be able to carry on a conversation in quiet but not in noise.

Figures 14 and 15 show a model of the normal ear and of an ear with inner hair cell loss only—in the example illustrated—above 1.6 kHz. A failure of the cochlea to transmit speech cues to the brain should cause difficulty hearing in noise.

But a stronger statement can be made. The word-recognition score for speech in noise appears to be predicted entirely by the number of AI dots reaching the brain (speaking figuratively). It does not appear to matter whether

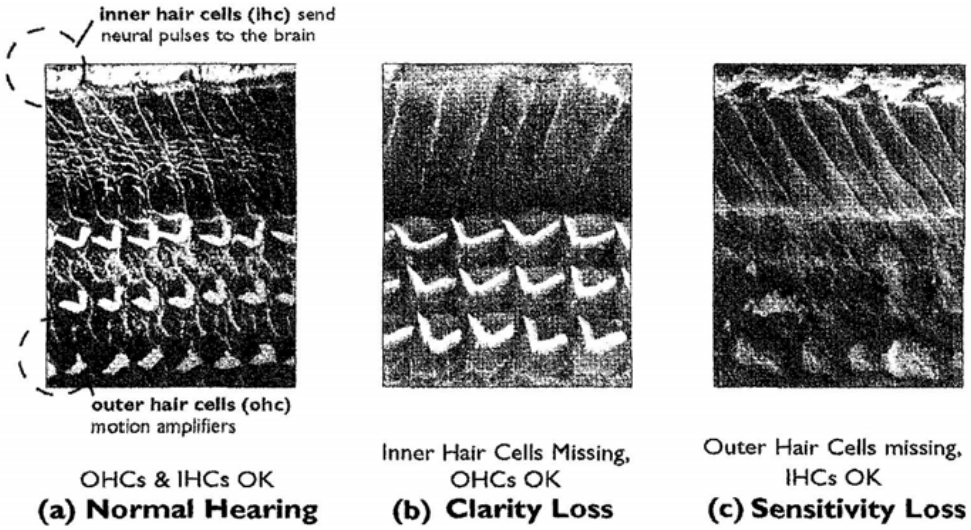

Figure 13 Electron microphotographs of a small section of the cochlea showing (A) normal inner and outer hair cells, (B) missing inner hair cells, and (C) missing outer hair cells. Photograph A is by Derek Dunn, used with permission of NHCA and NIOSH; photographs B and C were supplied by and used with the permission of Robert Harrison, University of Toronto.

the dots are lost to audibility (signals near threshold or masked by noise) or to a loss of channel capacity.

Psychoacoustic Evidence

Although three examples that might be included as evidence were given previously in the section *Audibility AI vs. Channel Capacity*, a more careful analysis is warranted.

When speech is subjected to low-pass or high-pass filtering at 1.6 kHz, we obtain the expected result with normal hearing subjects: with half of the speech cues removed, normal hearing individuals required the expected 8 dB increase in SNR for 50% recognition. (The reason the number is 8 dB is explained later.)

In our experiments,[17] our normal-hearing subjects showed the expected 2 dB SNR for 50% correct for the broadband condition, with a 7 dB increase in SNR for the low-pass condition and a 9 dB increase for the high-pass. (The fact that both were not equal to 8 dB presumably came about because the 1.6 kHz filter

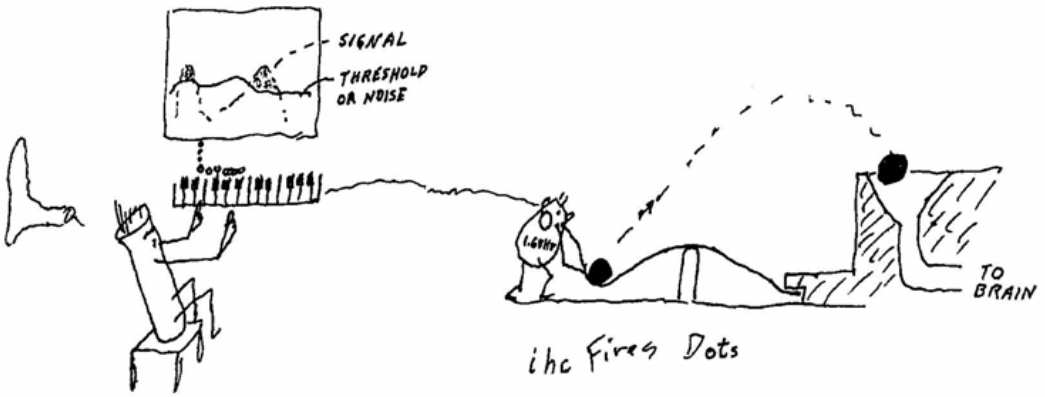

Figure 14 Cartoon model of a normal ear (IHC = inner hair cells).

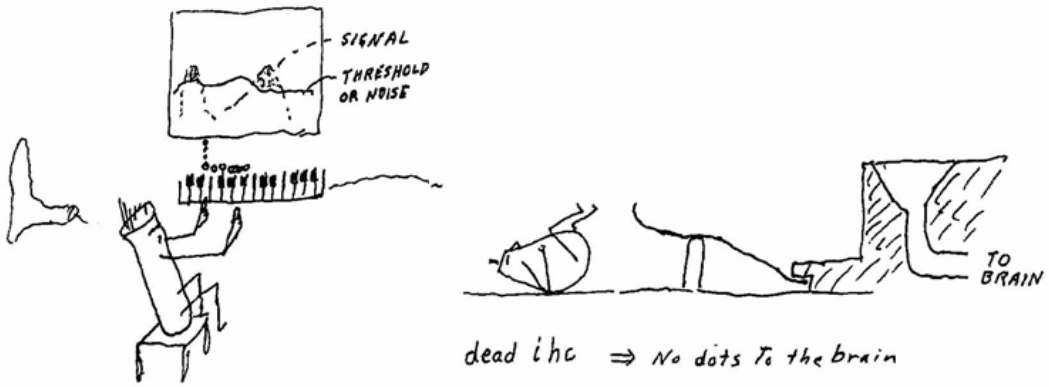

Figure 15 Cartoon model of a channel-capacity-impaired ear (IHC = inner hair cells).

cutoff we used was not the exact center of importance for those test materials.)

Subjects with high-frequency hearing loss required an average 5 dB SNR for the broadband condition, corresponding to a channel capacity loss of 27% overall, as shown in Figure 12. They required a 15 dB SNR for the low-pass condition, and a 22 dB SNR for the high-pass condition. Using the graph of Figure 16, these figures correspond to a channel capacity loss of 12% in the low band and 21% in the high band. These numbers add up to nearly the same number estimated from the broadband SNR measurements.

The findings above lend support to the basic thesis described here. Normal hearing individuals require 26 AI dots (again speaking figuratively) to obtain 50% word recognition score (WRS) in noise in the SIN test. High-pass or low-pass filtering to remove half the dots means that only 13 dots remain at the original signal-to-noise ratio. To restore the other 13 dots from those available within the low-pass or high-pass condition requires an 8 dB increase in SNR. (To

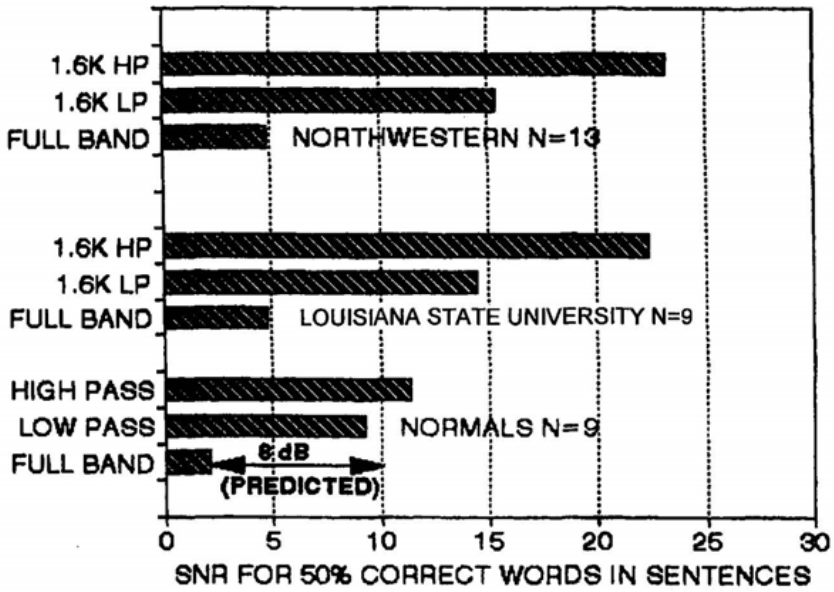

Figure 16 Results of full-band, 1600-Hz low-pass, and 1600-Hz high-pass SIN tests with three groups of subjects, two with mild-moderate sloping loss.

explain, the speech cues cover a 30 dB range, so to pick up 13 dots requires 13 dots × (30 dB/50 dots) × (50 dots in half band) = 7.8.

The best explanation for audibility without intelligibility would be a few inner hair cells sending enough information to the brain for audibility but not enough for intelligibility. Interestingly enough, Corliss et al[21] reported a case that suggests such a patchy inner hair cell loss. One of the authors was a physicist who noticed that 4 kHz presented to one ear at threshold gave two tones—neither of which equaled the pitch of a 4 kHz tone heard in his other ear. As the level was raised, he heard four separate tones. One plausible explanation would be that tonality at high frequencies is determined largely by the place of excitation on the basilar membrane, his inner hair cells at the 4 kHz place were missing, and patches of progressively remote inner hair cells became excited by the increasingly intense 4 kHz input.

More recently, Moore et al[22] introduced the term "dead regions" to describe regions in the cochlea with nonfunctioning inner hair cells. They described a threshold-equaling noise (TEN) that produced equal masked thresholds at all frequencies from 250 to 10,000 Hz in normal-hearing listeners. (Ordinary room noise does about the same thing between 250 and 4000 Hz.[23]) Subjects with apparent dead regions in the cochlea exhibited elevated thresholds at frequencies corresponding to the dead region. A subject with normal hearing and a 75 dB high-frequency loss at 4 kHz, for example, would exhibit the same 75 dB loss in the presence of a 50 dB HL TEN noise if his or her inner hair cells were intact. The presence of a threshold shift to a value greater than 75 dB HL indicates that the original threshold was probably a result of off-frequency detection. For the sake of example, assume that detection occurred at the 3 kHz place in the cochlea where, let us further assume, the threshold was nearly normal at 30 dB HL. Since the TEN noise raised the 3 kHz threshold to 50 dB, we might expect the 4 kHz threshold to rise by nearly 20 dB. Moore and colleagues found subjects with more or less similar audiograms, some having no apparent dead regions and some having apparent dead regions in the cochlea. This finding is consistent with the SNR data shown in Figures 5, 6, and 7, where a wide variety of abilities to understand speech in noise was found for subjects with similar audiograms.

Further strong evidence for dead regions in the cochlea, this time from psychophysical tuning curves, was reported by Moore and Alcántarao.[24] In one case, for example, the shift in the tuning curve indicated that a 1500 Hz signal was detected at a place in the cochlea tuned to approximately 2500 Hz, indicating a dead region near 1500 Hz.

In the near future it would be interesting to follow up on another suggestion of Corliss[25] to explore the relationship between SNR loss and loss of asymptotic loudness for very loud sounds. Mild-to-moderate cochlear loss is sometimes but not always accompanied by complete recruitment (return to normal loudness) for sufficiently intense sounds. We might expect such patients to have only a mild SNR loss. If we accept one popular belief that loudness is more or less proportional to the total number of neural firings reaching the brain, we might expect a good correlation between the degree of loudness deficit and the degree of SNR loss. Those whose asymptotic loudness never reaches normal no matter how intense the stimulus is presented might be expected to have substantial SNR loss.

PHYSIOLOGICAL EVIDENCE

Physiological data on the causes of SNR loss provide further support for the basic arguments contained here. Margolis[26] did a retrospective study comparing the amplitude of N1, the action potential, with the speech recognition scores of a group of several hundred subjects. Because the action potential is presumably generated by the firing of cochlear neurons triggered by inner hair cell activity, we hoped to find a strong relationship between the two. There was a clear positive correlation between the higher speech scores and AP amplitude, but also a large amount of scatter: the use of word scores in quiet resulted in a large ceiling effect.

Dallos and Berlin[20] provided the common view that the primary cause of SNR loss is a result of inner hair cell loss. Other explanations have been suggested for loss of channel capacity and its resultant SNR loss.

McKenna and Adams[27] suggest that the recirculation of potassium, essential for the operation of the cochlea, can become blocked, stating that "inflammatory processes within the cochlea can lead to hearing loss by disruption of cells within the spiral ligament. This, in turn, leads to disruption of potassium recirculation, depression of the endocochlear potential and sensorineural hearing loss."

A puzzling result came from Killion and Niquette's[28] study of the 63 ears presented by Schuknecht.[29] Although the average loss of inner hair cells grew linearly from 0 below 40 dB of hearing loss to nearly 100% for profound hearing losses, none of the 44 ears suitable for study showed a partial loss at any location. The 255 locations (subjects × frequency) Niquette studied each showed an all-or-none response. There was no evidence of partial inner hair cell loss (although partial loss of outer hair cells was not unusual).

Most other investigators have found evidence of patchy degeneration of inner hair cells. The classic Bredberg[30] study showed a cochlea with dead inner hair cells alternating with intact cells. He found the percentage of inner hair cell loss increasing toward the basal turn. Bohne and Clark[31] reported similar results in noise-damaged chinchillas, with narrow lesions of missing inner hair cells interspersed among intact cells. The reason for Schuknecht's findings[29] are not known, but may have been a result of the preparations he used.

Mills[32] suggested that insufficient endocochlear potential—caused in turn by stria vascularis deficiencies—may explain a significant portion of hearing loss. Figure 17 shows loss of stria vascularis as a function of pure-tone-average hearing loss, taken from Killion and Niquette's summary of Schuknecht's findings.[28] What is interesting here is that even mild pure tone losses can show stria vascularis loss, similar to the behavior of SNR loss.

Finally, loss of neural function, while rare, can cause loss of channel capacity.

APPLICATION

The fact that SNR loss can now be estimated to within 2 to 3 dB in 1 to 2 minutes makes it readily available for guidance as to how much SNR improvement the patient needs.

Although the option most often mentioned for improving the intelligibility of speech in noise is digitally filtering out the noise, this will require circuits that can identify the desired talker among several possibilities and then track his or her speech accurately in the midst of noise. Neither of those technical achievements is presently available.

Fortunately, we can still provide dramatic help for someone with SNR loss. Directional microphone hearing aids, array microphones conveniently arranged to wear over the ear and couple to the hearing aid telecoil, and subminiature FM systems can provide 5 dB, 10 dB, and 20 dB of SNR improvement, respectively.

A 20 dB SNR improvement can provide someone with only 25% channel capacity the ability to carry on a conversation at social gatherings. Extending a hand-held microphone to the lips of the talker in one-on-one conversations—which often provides more than 20 dB improvement in SNR—is now pleasantly commonplace on the exhibit floor of Self Help for Hard of Hearing People (SHHH) conventions, allowing members with severe loss of channel capacity to carry on conversations with little apparent effort. Similarly, FM and infrared broadcasts of convention speakers and stage performers is becoming more and more common in public places.

The first line of defense, now and always, is to make as much of the speech audible as possible. Noise reduction schemes can reduce noise (as does turning off the hearing aid), but do no good and often do harm in real-world situations if good is defined as improving intelligibility in the face of noise.[33]

Maximum intelligibility for most patients comes when everything—speech and noise—are made completely audible.[18] In many cases, this means increasing the amount of audible noise. In a situation where 50% of the speech cues are masked by noise, the person with nor-

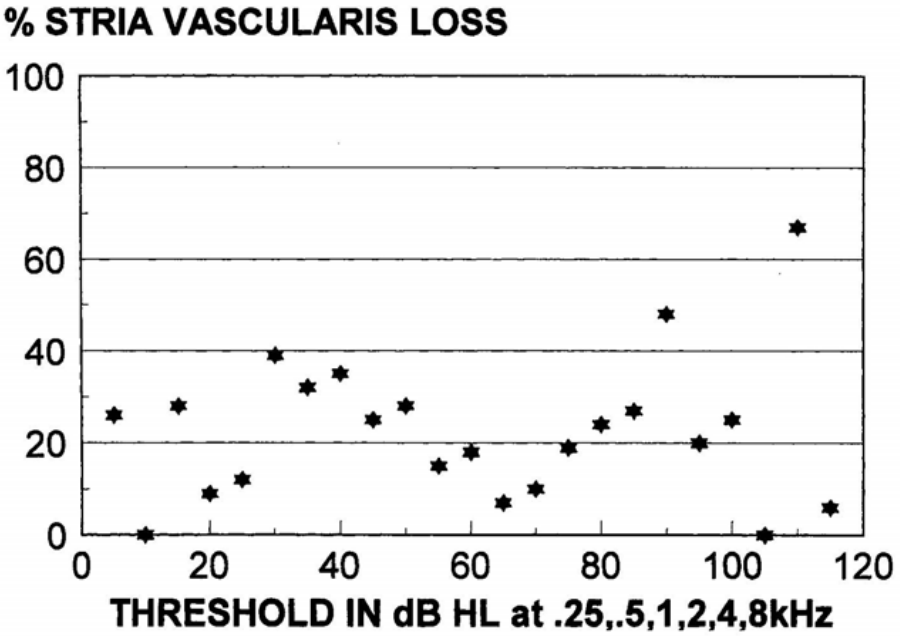

Figure 17 Percentage loss of stria vascularis vs. pure-tone hearing loss in 44 human temporal bones from hearing-impaired patients. Data from Schuknecht.[27]

mal hearing will have no trouble understanding sentences. If only half of those speech + noise cues are made audible to someone because of inadequate gain or bandwidth in a hearing aid, however, then the percentage of audible speech cues drops to 25%. If that person has a channel capacity of only 50%, then only 12.5% of the speech cues will reach their brain. As Villchur[18] illustrated, improved intelligibility in noise can result when all of the speech (and of the noise) is made audible, so that the full 50% of the speech cues left unmasked are audible. In this case the person with 50% channel capacity starts with twice as many audible cues, so that 25% of the speech cues reach the brain even after half are lost to diminished channel capacity. (As shown in Figure 2, 25% of the speech cues are generally adequate for the understanding of 90% of sentences.)

We exclude from the above example the special cases mentioned previously where increased audibility degrades performance. Fortunately, these are relatively uncommon, being a subset of ski-slope hearing losses.

SUMMARY

Whatever the explanation, it is clear that SNR loss is surprisingly unrelated to pure-tone-thresholds, and must be measured separately. With modern SNR-loss tests, it is practical to measure SNR loss as quickly as, or even more quickly than, audiometric loss.

Knowing the SNR loss, it is possible to estimate channel capacity, based on the often-measured relationship between word recognition scores and (audibility) AI. This theorized relationship is shown in Figure 12. Knowing channel capacity (CC), we can extend the application of articulation index theory to include losses in the internal auditory system.

Stated informally, inaudible dots and broken dots (dis)appear the same to the brain. More formally,

$$\text{perceptive AI} = \text{AI} \times \text{CC}$$

Whether speech cues are lost to inaudibility (threshold or masking) or to loss of internal channel capacity (inner hair cells and auditory

neurons), the effect on one's ability to hear in noise appears to be the same.

Two means of improving the ability of hearing-impaired patients to hear in noise are presently available: (1) Make everything audible, and (2) improve the signal-to-noise ratio of the incoming signal with directional and/or close-talking microphones.

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ABBREVIATIONS

AI	Articulation Index
CC	channel capacity
CVCV	consonant-vowel-consonant-vowel
HINT	Hearing-in-Noise Test
IHC	inner hair cells
PAI	perceptive Articulation Index
PTA	pure-tone average
QuickSIN	Quick Speech-in-Noise
SIN	speech in noise
SNR	signal-to-noise ratio
TEN	threshold-equaling noise
WRS	word recognition score

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