Excerpts from

HEARING: A 21ST CENTURY PARADIGM

including,

ELECTROCHEMISTRY OF THE NEURON

This material is excerpted from the full β-version of the text. The final printed version will be more concise due to further editing and economical constraints. A Table of Contents and an index are at the end of this paper.

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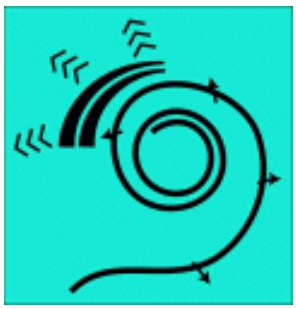

10 Disorders of the Auditory System[1]

10.1 Introduction

10.1.1 Categorizing auditory pathologies

Disorders of the auditory system can be broken into two major categories, mechanical and neural disorders. The former consist of disorders related to the outer, middle and non-neural portions of the inner ear. Clinically, these are generally described as conduction loss diseases. The latter can be subdivided into neural based disorders. Those associated with the sensory neurons (and called sensorineural loss diseases) and those associated with later stages of neural circuitry. These last have not been further categorized within the field of otolaryngology and subjects with these types of problems are frequently referred to a neurologist. Some investigators have divided the above disorders into three distinct categories, conductive, sensory and neural. In this case, the sensory category applies only to the sensory neurons.

An alternate subdivision of disorders of the auditory system occurs at the logarithmic conversion occurring at the axon pedicle. This division places the majority (if not all) of the analog signal processing ahead of this conversion and all of the phasic signal processing subsequent to this demarcation. It is also important to note the major, but poorly named phenomenon of "recruitment" is intimately involved in the logarithmic conversion at this juncture. Recognizing this functional demarcation leads to three major subdivisions within hearing disorders;

1. conductive (defined as stage 0 disorders)
2. sensory neuron (defines as stage 1disorders), and
3. neural (stage 2 and higher disorders), other than sensory.

A curious disorder that may not fit either the conventional (2-section) or the proposed (3-section) categorization is commonly labeled the sea-shell effect. While many investigators attempt to assign this effect to anomolous performance associated with either the mechanical or the neural category, Tonndorf [page 37 in Jahn, 1988] gives an alternate explanation that appears plausible. He attributes it to the Brownian Motion associated with a confined volume of air molecules. He notes that in his experience, the effect is proportional to the volume of the confined air volume. The spectrum of this noise is known to decrease with frequency and the magnitude is known to increase with enclosed air volume. Even the masking associated with this effect has been measured. Bigger shells produce a greater effect, a greater RMS pressure variation on a given size tympanic membrane. Under this interpretation, the effect is an interesting external phenomenon and cannot be considered pathological to the auditory system.

Genetics has unearthed a large number of hearing disorders that can be loosely related to genetic abnormalities but there has not been great success in attributing a specific genetic defect to a particular area or mechanism of hearing. Griffith & Friedman have described the situation[2].

[xxx geisel pg 275-318]

[xxx chapter seven of shambaugh & glasscock on inflammatory diseases]

10.1.2 Background

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[1]Released: 2 June 2009

[2]Griffith, A. & Friedman, T. (1999) Making sense out of sound *Nature Genetics* vol 21

Many auditory disorders affect only one ear and its associated circuitry. This allows both qualitative and quantitative evaluation of the disorder through binaural testing.

The auditory system, like all known sensory modalities, is designed to operate in a constant amplitude signal environment following the sensory neurons. This feature means that most hearing abnormalities, other than those due to trauma, inflamation or other pathological conditions, are associated with the sensory neurons. These abnormalities are generally grouped into sensorineural disorders. They are discussed in **Section xxx** below.

10.1.2.1 DC coupling of neurons introduces a complication

A complication related to the sensorinerual disorders is related to how individual circuits are coupled together in hearing, and all of the neural system. All of the neural signaling channels of biology are DC coupled. There are no capacitors (or transformers) separating the individual circuits (connexuses). Instead, they are coupled by synapses that consist of an Activa and a minimum of associated circuitry. While the Activa are three-terminal devices, the synapses function as two-terminal diodes when properly biased. They form a diode between the axon of one neuron and a neurite of a second serially connected neuron.

> While the morphologist will frequently describe a synapse between an axon of one neuron and the soma of a second, this labeling is inappropriate. Functionally, the receiving circuit element is always a dendrite or podite, even if it is enclosed within the outer envelope of the soma.

The problem in analyzing DC connected serial circuits is that a change in the potential or current flow at one point can cause a ripple effect in subsequent orthodromic circuits. Following a summation process, the source of a single error may be traceable to any of the signaling channels participating in the summation.

10.1.2.2 The continuous audiometer of Bekesy

[xxx pages 81-91 in Bekesy]

10.1.3 Pathologies of the auditory system

[xxx see Geisler chapter 16

This work will differentiate between auditory diseases which can be clinically identified from outside of the patients body from those that cannot (autoacoustic emission versus tinnitus, as defined below, as an example).

10.1.3.1 Meniere's disease

Meniere's disease is also known by the scientific name, endolymphatic hydrops. It is characteristically in one audio channel initially but may become bilateral.

This major syndrome is characterized by four major symptoms, vertigo, hearing impairment, tinnitus and fullness or pressure[3]. A characteristic condition related to the hearing loss is the ballooning of Reissner's membrane. This probably interferes with the launching of the surface acoustic wave within the tectorial membrane due to distortion of the launcher near the oval window.

The tinnitus in this condition is usually at low frequencies and described as a "roaring."

10.1.3.2 Echoes within the auditory system

[xxx consider a misalignment of the IHC and Hensen's Stripe.]

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[3]Shambaugh, G. & Glasscock, M. (1980) Surgery of the Ear. London: W. B. Saunders pg 572

Two different types of echoes occur in the human auditory system. The most obvious are those perceived by the subject. Another type is perceived primarily by the medical staff during examinations. These may or may not be heard by the subject. These echo types occur within the human auditory system under three different conditions. First, some occur within an interval of less than three milliseconds and relate directly to acousto-mechanical reflections associated with the outer and middle ear. Second, many occur within a period of a three to 30 milliseconds and relate directly to the operation of the cochlear partition of the inner ear. These echoes are associated with interference with the propagation of energy along the cochlear partition. Third, a few people have claimed they heard echoes at intervals up to one second delay. Echoes occurring after more than 30 milliseconds must be associated with the neural system and probably the higher cognitive processes.

Any physical deformity or physical interference with the surface of Hensen's stripe can result in an echo that is observable by a clinician. The physical location of the problem is easily computed from the time of the echo. The problem may also result in the loss of hearing by the subject at frequencies below the frequency associated with that location along the stripe.

Unusually sharp bends in the cochlear partition can result in the perception of sounds at frequencies higher than their actual frequency. If severe, the bend can cause termination of hearing at a specific frequency and the perception of sounds of lower frequency as if they were at or near the termination frequency.

Any extraneous material contacting the outside of Hensen's stripe, with an acoustic impedance different from that of the endolymph, can cause a reflection in the dielectric waveguide formed by Hensen's stripe. Such a disturbance is frequently observable by the clinician at a level on the order of 20 dB below the source intensity. The delay between the source pulse and the recorded echo can be related to a specific position along Hensen's stripe.

10.1.3.3 Clinically observable spontaneous sources within the auditory system

[xxx consider ringing gell trapped between input structure and a discontinuity due to the gel or the sensory neurons (even kinocilia).

10.1.3.3.1 Auto acoustic effects EMPTY

[xxx sounds generated by the hearing apparatus and observed by external investigator]

10.1.3.4 Clinically unobservable sources within the hearing system--Tinnitus

[xxx offer a circuit diagram if possible Form into a website page like Snowy Vision]
WV 272 T591-6, 2000 is quite complete on this subject
WV 272 T591 1981 has a good epidemiology section
WV 272 K27p 1983 is good

Most of the above is clinical and empirical psychological data

Kiang 1970, pg 263 and the discussion, pg 268 on

[xxx pages 81-91 in Bekesy]

[xxx see Geisler 1998, pg 292-294
[xxx AT reports her tinnitus]

[xxx develop how the external feedback loop may well play a role in controlling, or suppressing tinnitus. **Section 5.4.6**]

Moller has included an overview of tinnitus[4]. Tyler has provided the most complete and current handbook

[4]Moller, A. (2000) Hearing: its Physiology and Pathophysiology. NY: Academic Press pp 461-470

on Tinnitus[5]. It is limited to clinical situations and offers little on the source of the condition. The epidemiology chapter by Davis & Rafaie is quite complete as is the chapter on the "psychological profiles" of tinnitus patients by Erlansson.

Tinnitus is a disease exhibiting a variety of symptoms in different patients. Like snowy vision, the symptoms reflect the introduction of extraneous noise into the signaling channels of hearing. While snowy vision is suffered by at most a few subjects per million, tinnitus is reported broadly (probably more than 10%) in the population of those over 50 years of age. The author encountered the bilateral form of the problem starting at about 60 years of age. Coincidentally, or not, a variety of drugs were taken beginning in this time period for other reasons. It is quite reasonable to suspect some sensory hair cell damage from these drugs. However, the transient nature of his condition suggests his tinnitus is not due to physical damage but to bias problems based on chemical factors.

Recently, during a night where I encountered a very dry mouth at two different intervals (4 June, 2005), the tinnitus in my left ear disappeared completely. Upon arising, the tinnitus in both ears was absent for a matter of hours. After that period, it became noticeable again but not at its nominal intensity. No tinnitus was noticeable in my left ear after 18 hours. The tinnitus in my right ear was only noticeable when I concentrated on it.

During intervals when the tinnitus is absent from the left ear, the tinnitus in the right ear is perceived as originating at the peripheral portion of the system, somewhere between the cochlea and the outer ear.

Tyler, in his preface, says: "Tinnitus remains one of the most difficult hearing disorders to treat. . . . tinnitus has numerous causes and many neurophysiological mechanisms are likely to be involved." Even defining tinnitus has become a challenge. Davis & Rafaie quote McFadden (1982) that "Tinnitus is the conscious expression of a sound that originates in an involuntary manner in the head of its owner, or may appear to him to do so." In the context of this work, the use of the term "conscious expression of sound" is questioned. This wording seems to imply willful action. In the majority of cases, there is nothing willful about tinnitus. In the context of this work, tinnitus can be defined as,
The awareness of a sound percept not caused by external acoustic sources, and generally not audible to a clinician due to the generation of sound waves by the middle or inner ear. This definition places most sources of tinnitus within the neural component of the hearing and cognitive systems. It is also compatible with the definition of Jacobson (Tyler, page 181), "tinnitus represents the perception of sound when none is present, . . ."

As Davis & Rafaie note, "Classification of tinnitus could play a constructive role in research and treatment. However, the lack of a clear understanding of a mechanism of generation and perception makes a single classification difficult to envisage." They describe the general nature of the classification systems attempted and used to date. A useful feature of tinnitus in some patients is its monaural character, that allows binaural evaluation of the symptom(s). Both Davis & Rafaie and Erlandsson stress the lack of any potent treatment for the condition. They both stress the importance of managing the subjects emotional involvement more than actual medical treatment.

Jacobson offers several pertinent comments. "Investigators have utilized in tinnitus investigations all available auditory assessment techniques. . . ." "Inherent to all investigations has been the idea that tinnitus results from a 'hyperactive' auditory system." "Despite the ready availability of patients, . . . the findings reported by investigators to date have been inconclusive. There has not bee, as yet, unanimous support for any positive finding."

Harrison has provided a general introduction to tinnitus[6]. "To start with, it must be stated that the term tinnitus covers a range of diverse phenomena. Some useful subclassification of tinnitus types have been made, although categorizing is, in most cases, not easy." "The distinction between 'roaring' tinnitus and single pitch or narrow band tinnitus is not easy when most often the patient's description falls between these two extremes."

Four distinct types of tinnitus can be identified from a theoretical perspective:

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[5]Tyler, R. *ed*. (2000) Tinnitus Handbook. Singapore: Singular–Thomson Learning

[6]Harrison, R. (1988) The Biology of Hearing and Deafness. Springfield, Il: Charles C. Thomas pg 57

1. tonal tinnitus (the perception of a narrowband background signal),
2. white noise tinnitus (the perception of a broadband background signal exhibiting equal energy content as a function of frequency over a wide acoustic frequency band),
3. colored noise tinnitus (the perception of a broadband background signal where the energy content varies significantly as a function of frequency), and
4. later stage tinnitus (the perception of a complex signal largely independent of the signals delivered from the peripheral neural system).

Patients have described a wide range of sounds in a clinical environment (Tyler, page 152) that can be associated with these four categories.

Harrison gives some additional information on pages 352-353.

Many musically trained subjects suffering from tonal tinnitus can walk up to a piano and select the key that most nearly represents their perceived signal. AT (80 years old) reports her narrowband tinnitus is quite continuous and is represented by a frequency near B above middle C or nominally 987 Hz.

Many subjects exhibit the second type of tinnitus, a broadband, equal energy spectrum that they describe as a roaring sound, not unlike a waterfall.

The authors tinnitus is of the third type, consisting of a broadband noise with the energy density highest at a frequency near 1000 Hz. The signal is perceived as a buzz.

Tinnitus exhibits the unusual property that its can be perceived intermittently, and frequently exhibits two unusual properties. First, many subjects report it is not observed during periods of concentration on specific tasks. Second, the condition may not mask the sensing of other external acoustic signals. Both of these properties suggest the condition originates in the CNS or is subject to some level of control within the CNS.

The introduction of extraneous signals into the hearing system can occur at a variety of locations. **Figure 10.1.1-1** highlights some of these sites.

Javel has noted that tinnitus often exists when hair cells are completely absent[7]. It is not clear if he means stereocilia are completely absent or the entire cell is absent.

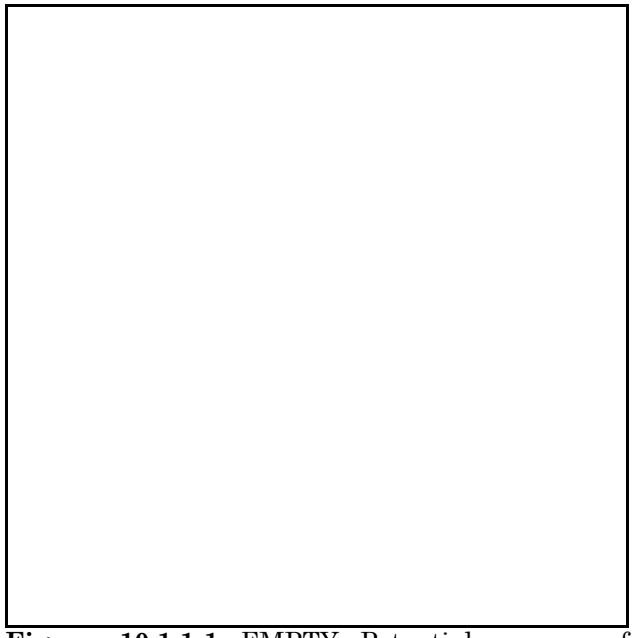

Figure 10.1.1-1 EMPTY Potential sources of tinnitus.

10.1.3.4.1 The character of tintinnitis

The disease known as tintinnitis or ringing in the ears is usually described in terms suggesting a pure tone. Such a pure tone consists of a single narrow frequency suggestive of that from a well made bell. The word tintinnabulum means small bell. However, this occasional sufferer has noted that the illusory internal sound I perceive is not that of a pure tone, nor is it bell-like. The sound exhibits the characteristics of a broad band noise source. In particular, it is very similar to the "colored noise" heard in the speaker of a police or taxi radio receiver (of the type used from about 1950 to the late 1990's) when the squelch circuit is left open. This colored noise is characteristic of the signal produced by the output circuit of a phase

[7]Javel, E. (1986) Basic response properties of auditory nerve fibers *In* Altschuler, R. Hoffman, D. & Bobbin, R. *eds*. Neurobiology of Hearing: The Cochlea NY: Raven Press. Chapter 13, pg 215

modulation system in the absence of any de-emphasis.

With the advance of time, it is becoming common to use the contraction tinnitus (most often with two n's) to represent the word tintinnitis.

Salvi, et. al. writing in Tyler (page 152) have provided a table of tinnitus characteristics based on leading questions to 528 tinnitus patients. The list of qualities of the subject's perceived sound included a series of adjectives and noun names; ringing, bussing, cricket, hissing, whistling, humming, roaring, musical note, steam whistle, pulsing, cracking, clicking, popping, etc. While more than one-third of the subjects selected ringing, ten other names were chosen at least two percent of the time.

In the future, it would be preferable to provide the subjects with a series of more technically defined and carefully prepared prerecorded samples, at an appropriate sound level.

An anonymous blogger has contributed his perception of tinnitus. His condition is obviously different from that reported by most people. It appears to be more like a neural hallucination, like those encountered after an amputation. "The right ear is so silent and surreal with tinnitus sounds it's as if it is wired to listen in to a different planet. Tinnitus is hard to describe, as it always changes. I took some notes on it last night and came up with this:

8-9pm: Three Arias from Don Giovanni mixed with the song of the Southern Pacific blue whale.
9-9:30pm: Trucks downshift while warm rain falls.
9:30-10pm: A rainbow unfurls and flaps in the wind. Japanese geishas titter.
10-11pm: Sound of waves receding. A woman walks by in a dress made of string. Did I forget to turn off the dishwasher?
11-11:30pm: Music for giant space babies."

This blogger's form of tinnitus appears to relate to Stage 4 signal manipulation and subsequent Stage 5 cognition problems more than the typical Stage 1, 2 & 3 signaling problems. He is perceiving sounds that are not introducible by simple errors in the early stages of hearing.

10.1.3.4.2 Previous theories of tinnitus

Eggermont, writing in Tyler, has provided the broadest review of tinnitus from a research perspective. However, it is reasonable to say the literature and the review do not converge on an answer to the fundamental question, "what is the source of the disease known as tinnitus?" This is largely understandable based on the character of the symptoms described by Tyler on page 152. Eggermont explores the possibility that the source is at one of five different levels within the hearing system; the system, network, neuron, synapse or molecular level. He suggests the sources could be virtually anywhere in the morphologically defined auditory system.

Tinevez, Martin & Julicher have recently discussed a potential source of tinnitus related to the chemical environment surrounding neurons in general[8]. Based on this environment, the source they speak of could effect tinnitus, one of its analogs identified as "snowy vision" (see Chapter 19 of Processes in Biological Vision, 2005) and many other possible neural disorders.

Tinevez, Martin & Julicher suggest that actin, a piezoelectric material found in the cilia of hair cells may exhibit a nonlinear transfer characteristic related to the local concentration of Ca^{2+} ions. **Figure 10.1.1-2** shows their concept. Under normal conditions, the transfer function is a straight line. However, under unfavorable conditions, the transfer function may take on a nonlinear form that includes a negative impedance region. As commonly found in relaxation oscillators, such a condition results in a self initiating oscillation about the origin in the absence of any quiescent force. In the presence of an external force, the transfer function is driven into the positive slope region of the transfer function and the spontaneous oscillation is suppressed. In the case of actin, the presence of Ca^{2+} could be considered an irritant.

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[8]Tinevez, J-Y. Martin, P. & Julicher, F. (2008) Active hiar-bundle motility by the vertebrate hair cell *a.k.a.* Unifying the various incarnations of active hair-bundle motility by the vertebrate hair cell *In* Cooper, N. & Kemp, D. *eds*. Mechanics of Hearing Workshop 2008 pg 172

A spontaneous oscillation of this type in the cilia of an IHC could result in the generation of a response perceived as a pure tone by the subject. If sufficiently large, the oscillation could generate an acoustic force that could travel back up the cochlear partition, through the middle ear, and be radiated from the external ear. A similar oscillation in an OHC could result in a similar effect. However, the necessity of crossing an expanse of the tectorial membrane before entering Hensen's stripe and projecting back through the outer ear reduces the probability that an oscillation at the OHC would be strong enough to be observed externally. Such an oscillation could still produce a signal that would be perceived by the subject.

10.1.3.4.3 Cilia loss within the cochlea

[xxx see pg 17 in Jesteadt Best cut at it]

[xxx see fig 16.5 in Geisler]

[xxx See pg 20-21 in Jesteadt]

Harding & Bohne have recently provided extensive data on this problem[9].

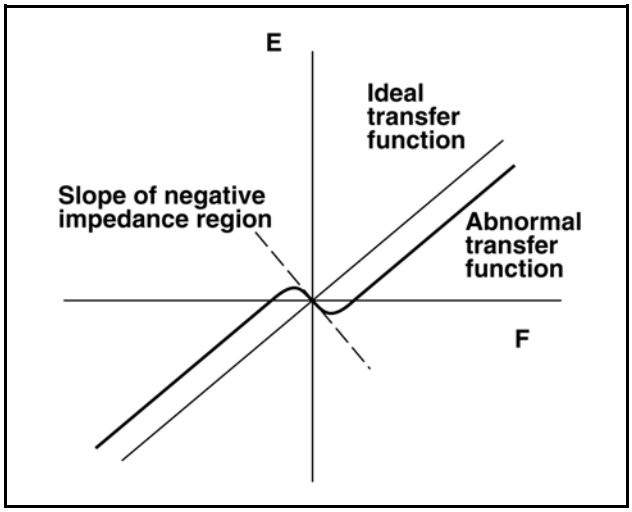

Figure 10.1.1-2 A potential chemical source of tinnitus and similar conditions. The graph plots the potential, E, generated in response to a force, F.

10.1.3.4.4 Other experimental evidence

[xxx see Tyler, specifically Eggermont and Tyler's chapter 6 and Jacobson's chapter 7 in Tyler]

10.1.3.4.5 A theory of tinnitus

[xxx compare to snowy vision and generate a map or table of potential sources.]
Figure 6-4 in Tyler provides valuable information related to the source of some tinnitus, although the caption appears to contain editorial errors. Referring to the referenced original contains the same awkward wording. The form of these curves suggests that the type of tinnitus they are exploring occurs in the system prior to the internal feedback circuit of the adaptation amplifier of the sensory neurons, and particularly the OHC neurons.

The characteristics described by in Tyler are not limited to simple waveforms. They consist of a broad range of waveforms, ranging from a musical note to various intermittent sounds (e. g., a cricket chirping). Based on the theory of this work, tinnitus is quickly recognized as having multiple sources within the system, not unlike the multiple sources described for snowy vision in the closely related visual modality. Many of the symptoms can be considered similar to the somatosensory responses observed by a subject following amputation of a limb.

The Theory of Hearing [xxx use formal designation] presented here clearly distinguishes between percepts, interps, and other cochleotopic signals within the hearing system. The mechanisms generating each of these classes of signals are theoretically capable of generating extraneous outputs. **Figure 10.1.1-3** displays some of these sites. [xxx see next section]

[9]Harding, G. & Bohne, B. (2004) Noise-induced hair-cell loss and total exposure energy: analysis of a large data set J Acoust Soc Am vol. 115(5) Pt 1, pp 2207-2220

10.1.3.5 Clinically recognizable forms of tinnitus

The record supports three distinct forms of tinnitus.

A.T. & S.W. describe a pure tone type of tinnitus that appears to relate to the failure of an individual signature channel in the tonal portion of the neural system. The failure provides a signature at the CNS that, as far as is known, is present constantly.

The apparently broader band form of tinnitus of J.F. suggests the introduction of a noise source following some degree of processing in the midbrain. Its variability in amplitude and occasional disappearance for intervals of a few hours suggest it is not the result of a permanent circuit failure but more likely is the result of a bias error. The fact that it does not mask external audio inputs, and that it has a finite bandwidth suggests it may be directly related to the "critical band" mechanism. The latter is closely associated with the concept of attention, a feature normally associated with the frontal lobe and possibly the parietal lobe.

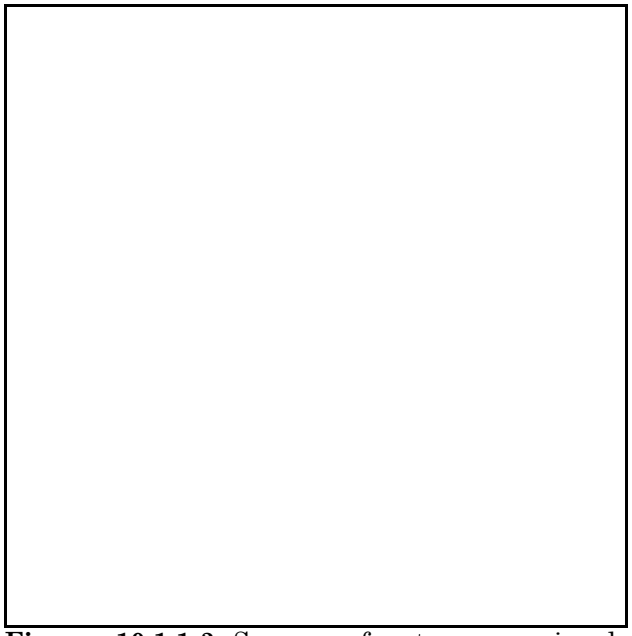

Figure 10.1.1-3 Sources of extraneous signals within the auditory system EMPTY.

The reports of the anonymous blogger suggest a higher level failure. The failure appears to consist of a false percept that is passed to the saliency map of the parietal lobe. The variety of the perceived information suggests the false percept may be due to a false interp combined with other correct information.

10.2 Sensorineural disorders related to intensity level

A wide range of disorders have been loosely identified by clinicians in the absence of a detailed model of the hearing system. Many of these disorders can be combined into one based on a better model. Historically, many of these disorders have been associated with a loss in threshold sensitivity. Others, such as the inappropriately named recruitment, have been associated with an apparent increase in loudness changes in response to a standardized change in stimulus intensity. Hallpike provided an extensive discussion of sensorineural losses in 1976[10]. Many of his conclusions are not supported here based on more recent data and interpretations.

The clinically recognized disorders related to intensity level in hearing can be described based on simple graphs derived from **Figure 10.2.1-1** developed in **Section 5.xxx**. Unfortunately, understanding the basis of this figure requires considerable knowledge of neural electrolytic circuitry and the conventional electrical circuit techniques upon which it is based. This section will not discuss these techniques since their application is discussed in **Chapter 5**, along with references to the underlying literature. This figure describes the operation of the tonal (OHC) sensory neurons in general Using the performance of the OHC neurons with a characteristic frequency and characteristic bandwidth including 1000 Hz.

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[10]Hallpike, C. (1976) Sensori-neural deafness and derangements of loudness function *In* Keidel, W. & Neff, W. *eds*. Auditory system. NY: Springer-Verlag pp 1-25

[xxx relook at stimulus levels and make agree with Standard Ear.]
The upper solid line connecting to the dotted line describes the nominal output voltage at the pedicle of the sensory neuron in the absence of the mechanism known as adaptation. In this figure, the voltage is described in units of loudness called Sones for convenience. Note that both scales in the figure are logarithmic. In the absence of the logarithmic conversion performed at the pedicle, the gain of the overall sensory neuron would continue to rise along the dashed line at the bend in the solid and dotted curve. Such a condition is not sustainable in any electrical circuit. In the sensory neurons, the maximum output potential is as shown, the gain of the circuit decreases with increased stimulus intensity as shown. The effect is described as compression in the vernacular. The point where compression becomes effective is controlled by the base potential associated with the distribution Activa of the sensory neuron. At some point, the Activa associated with the adaptation amplifier is driven into saturation (as shown). No additional signal amplification can occur above this level. Above this level, the shape of the signal representing any stimulus will be greatly distorted. Various forms of pain become apparent as the stimulus conditions drive the sensory neurons farther into saturation.

Negative internal feedback is employed in each of the OHC neurons of hearing. If this feedback was 100% effective, the resulting output characteristic would be shown by the lower (horizontal) solid line. Note that there is no output as a function of stimulus intensity under this condition. It is not a useful condition. In practice, the nominal feedback used in the hearing system approximates xxx %, with the upper solid curve representing 0%. The level of feedback is controlled by the impedance in the common emitter to ground circuit of the sensory neurons.

Not shown in this figure is an equivalent background noise level due to the internal noise performance of the auditory system. For purposes of discussion, this highly variable loudness level (across the population) will be considered equal to 0.01 Sones.

The major sensorineural disorders of tonal hearing are related to abnormalities in these curves due to abnormal conditions within the circuitry of the sensory neuron. Several of these disorders can be described by replotting only the central region of this figure.

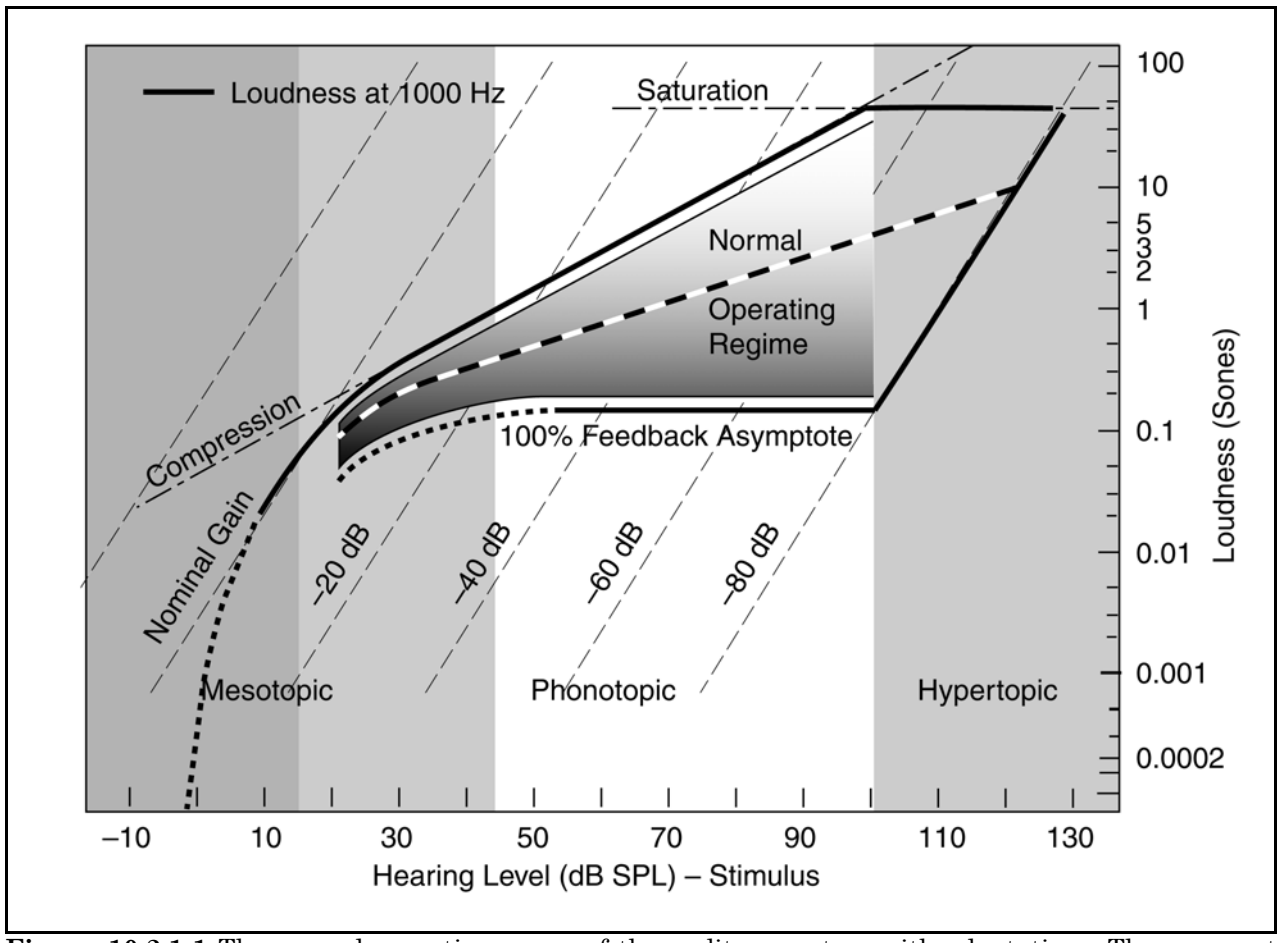

Figure 10.2.1-1 The normal operating range of the auditory system with adaptation. The apparent loudness of a 1000 Hz tone will initially be perceived according to the upper solid line. Following three time constants, the perceived level will correspond to the lower solid line. This figure applies to any individual spectral channel of the auditory system after modification to reflect the relative sensitivity of the channel compared to 1000 Hz. The background level is stabilized near the bottom of the adaptation level shown. Any additional stimulus exhibiting short term acoustic components will rise above this level into the region of normal operation. If the level of the short term stimulus is too high, it will encounter compression as suggested by the previous figure

When this curve is interpreted recognizing the very short time constants associated with hearing and the normal background level of the auditory system, the central portion can be re-ploted as shown in **Figure 10.2.1-2**.
Frame A shows the theoretical overall hearing characteristic. Frame B shows an expansion of only the central portion of the curves in Frame A for the normal ear.

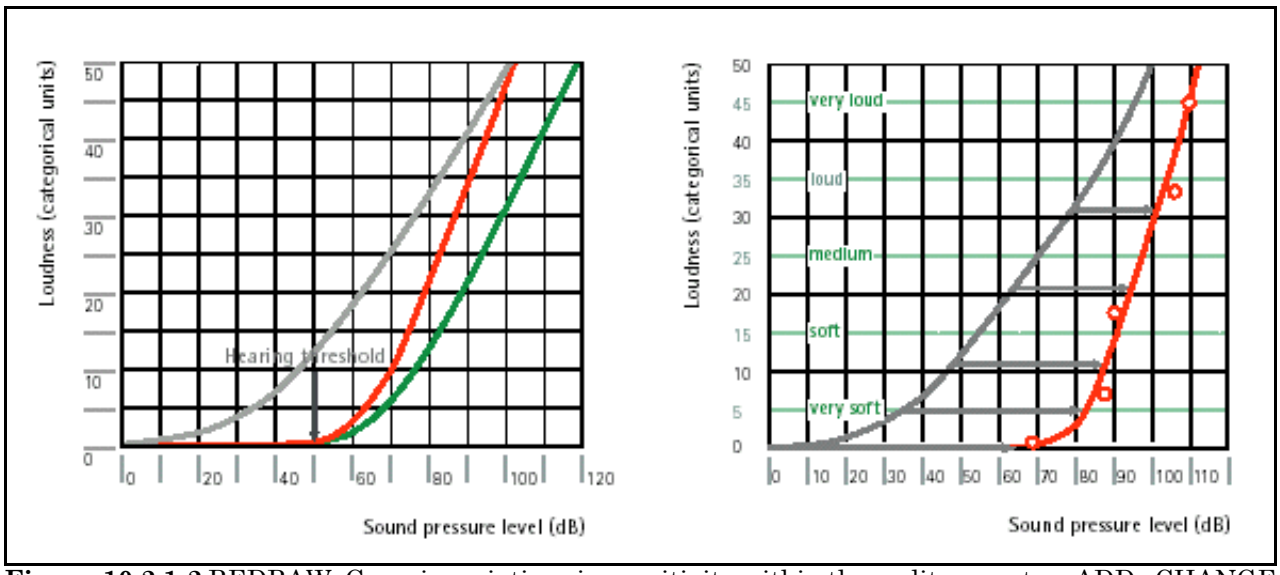

Figure 10.2.1-2 REDRAW, Generic variations in sensitivity within the auditory system ADD. CHANGE or expand Graph to show exponential curve and adaptation function

The commonly encountered conditions involve horizontal shifts in the position of, and changes in the slope of, the fundamental loudness versus stimulus intensity characteristic. Horizontal displacement of the curve is usually described by the term acusis while increases in the slope of the curve have generally been described in the vernacular using the term recruitment. The detailed description of these clinical conditions is frequently difficult in the absence of careful understanding of adaptation and its control during examinations of a subject's hearing capability.

10.1.3.2 Echos within the auditory system

10.1.3.2 Echos within the auditory system

10.2.1 Sensorineural disorders related to intensity range

10.2.1.1 Intensity abnormalities independent of frequency

Describing the perceived quality of hearing in the normal individual is difficult. Relating this quality to abnormalities is even more difficult. Phonak corporation has publicized a scale that describes the intensity of a stimulus in the normal environment. While it is labeled loudness in their literature (which connotes a perceptual characteristic), it more properly describes the actual intensity environment. In this context, the numerical values relate to the equivalent intensity levels expressed in SPL units. **Figure 10.2.1-3** reproduces this scale. The normal phonotopic range on this scale extends from 4 to 10 (nominally 45 dB to 100 dB in this work). The mesotopic range extends from 1 to 4 (nominally 15 to 45 dB). Beyond these ranges, hearing performance is highly dependent on the character of the stimulus.

10.2.1.1.1 Hypoacusis

Hypoacusis is frequently and most simply defined as a loss in auditory sensitivity that is not frequency dependent. The more specific definitions depend on the loudness versus stimulus intensity function of the subject. Two principle types of hypoacusis are found. The most common is that associated with the loss of amplifier gain in the adaptation amplifier. This type of hypoacusis is closely linked to the sensorineural loss of hearing previously labeled recruitment (**Section 11.2.2.1 xxx**). It is primarily involved with hearing at low stimulus intensities. The subject's hearing threshold is raised but his phonotopic regime may be largely unaffected (although it may be shortened with the high intensity shoulder remaining near 100 dB). The causes and potential cure of this condition are discussed in the section just referenced.

In the second type of hypoacusis, the loudness versus stimulus intensity function exhibits a typical shape but the subject complains of poor hearing relative to other people. If confirmable as present and not due to a conduction loss, the following comments probably apply. In this case, the entire loudness versus stimulus intensity function occurs at a lower location on the typical plot. However, this may not be easily documented since loudness is a relative psychophysical measurement. It may be documented in cases of binaural hearing where only one ear is affected.

The sources of this type of hypoacusis is more difficult to pinpoint. If the form of the loudness versus intensity function is normal, it suggests the adaptation amplifier and at least the current passing through the distribution amplifier are functioning normally.

10.2.1.1.2 Hyperacusis

Hyperacusis is frequently defined as an exaggerated sensitivity to sounds at all intensities. Hyperacusis may also be defined as a reduced tolerance to normal environmental sounds. One or both sides of the auditory system exhibit significantly enhanced sensitivity throughout the normal intensity range. However, to achieve this feature, the system loses its ability to adapt to different intensity ranges. As a result, the portion of the system affected operates in saturation much of the time. The term hyperacusis is frequently used synonymously with dysacusis, oxylacusis, and hypersensitive hearing.

Hyperacusis is a disease of the sensorineural class. In its simplest form, the internal feedback associated with the adaptation amplifier is lost.

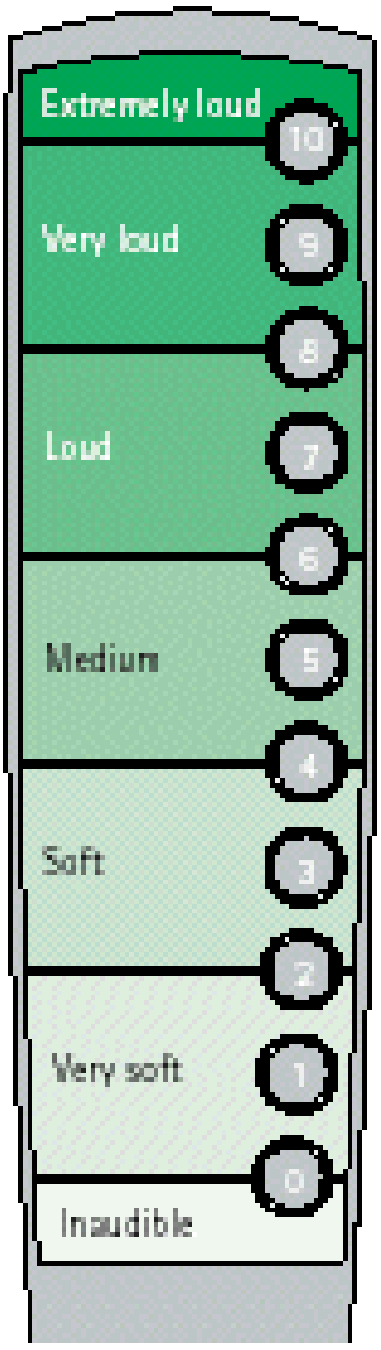

Figure 10.2.1-3 A loudness scale correlated to the conventional SPL intensity scale. From Phonak, 2004.

This results in the gamma of the loudness versus stimulus intensity function remaining close to 1.0 throughout the phonotopic regime (45 to 100 dB). As a result, excessively large amplitude signals are passed to the distribution amplifier where they are significantly distorted by the compression mechanism of the current to voltage converter associated with the axon. Loss of internal feedback is closely related to a reduction in the value of the common emitter impedance of the sensorineuron. A major loss of feedback suggests a short-circuiting of the common emitter impedance. This type of hyperacusis tends to be long term and may occur as a result of aging.

A more limited form of hyperacusis is possible if the gain associated with the avalanche amplification mechanism should become raised. This would raise the sensitivity threshold of the system, and potentially raise the sensitivity of the system in the kaumotopic and mesotopic regimes. This condition can occur if the chemical concentration of glutamic acid (glutamate) is raised in the perilymph of the cochlea or the chemical concentration of GABA is reduced in the perilymph. Either change can stimulate the electrostenolytic process and cause the voltage supplied to the adaptation amplifier to rise. This in turn can cause the avalanche gain mechanism to operate at a higher quiescent gain. The result is an increased sensitivity to low level stimuli by the affected sensory neurons. This disease can affect both the tonal (OHC) and wide band (IHC) sensory neurons. The author experiences this condition occasionally. This form of hyperacusis may also be related to the disease, or be part of the syndrome leading to the disease, known as tinnitus. This type of hyperacusis may be intermittent or long term.

10.2.1.1.3 Chugging

The author has observed an unusual sensitivity related function on many occasions. It is defined as chugging. Chugging is a perceived variation of hearing sensitivity observed in quiet locations and related to the pulse of the individual. The broadband background appears to rise and fall in amplitude in cadence with the pulse. It sounds vaguely like the average sound of freeway traffic except its intensity varies cyclically.

Chugging is almost certainly related to the porosity of the tissue between the arterial system and the points of electrical energy production serving hearing.

10.2.1.2 Frequency dependent variations in sensitivity EMPTY

10.2.2 Sensorineural disorders related

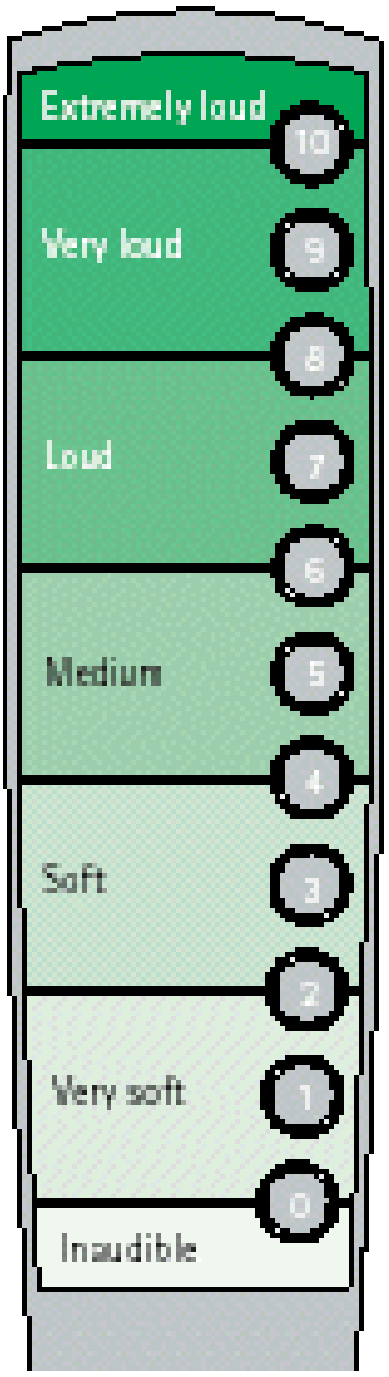

Figure 10.2.1-4 A loudness scale correlated to the conventional SPL intensity scale. From Phonak, 2004.

to differential sensitivity

10.2.2.1 Loudness Recruitment (loss in adaptation amplifier gain)

[xxx much of the following belongs in Chapters 8 or 9]
The variation in sensitivity with intensity has long been studied empirically, generally associated with the descriptor "recruitment" based on an early theory of the proposed underlying process. A wide variety of explanations for it appear in the general and clinical literature. These are generally described using the vernacular. When addressed in the scientific literature, the condition lacks a clear definition. Because of the confusion, recruitment is frequently described as a paradoxical phenomenon. Pierce & David described it as a peculiar phenomenon and then failed to define it in words that were consistent with other investigators[11].

Moody has provided a hypothetical explanation of recruitment based on its relationship to the latency of a response[12]. This quantity is not the latency of the transduction process. It includes both cognition and motor response time and tends to be in the hundreds of milliseconds. It is difficult to uncover any theoretical mechanisms that would support the proposed theoretical interpretation.

[xxx should define gamma before this comment]
Moore provides a two-page discussion of recruitment circa 1977[13]. No graphical treatment or tabular data is provided. The word picture suggests a gamma of 2.5 for the average situation. It is simplistic. It ignores any role for the adaptation mechanism in normal or abnormal hearing.

Plack & Carlyon have provided a recent one-page discussion of the subject using a graph with loosely defined relative scales and no data points[14]. They associate the problem with sesorineural impairment and note that it can be evaluated binaurally if present in only one ear. Their list of citations is limited. They did note an early study by Hood (1950) that said the ear exhibiting recruitment frequently exhibited an abnormally fast loudness adaptation characteristic.

Allen has provided one of the broader discussions of recruitment[15]. His multiple definitions and his figure 6.4 are converging on the model and definitions of this work. His figure 6.3 from Fletcher is fundamental to understanding recruitment. Recruitment is ***not*** the increase in something; normal hearing, within the phonotopic regime, is a reduction in something (the gamma of the amplification process due to feedback).

The above treatments have not discussed the change in slope of the hypothesized recruitment mechanism when illustrated on a (perceived) loudness versus intensity graph. The slope of such a curve on a log-log plot of this type is generally called γ (gamma). A gamma of 1.0 implies a linear process. Any other value of gamma implies a nonlinear process (in the absence of changes in the signal content in the frequency domain that can have a similar effect). The value of gamma can be evaluated on a local basis. It need not be a constant. A value of gamma below one is generally associated with a feedback mechanism, particularly if the value of gamma remains constant over a significant range.

Moore & Glasberg[16] and Allen, et. al[17]. have provided papers describing various clinical evaluations designed to improve loudness assessment and recruitment description. The Moore & Glasberg paper

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[11]Pierce, J. & David, E. (1958) Man's World of Sound. NY: Doubleday. pg 175
[12]Moody, D. (1973) Behavioral studies of noise-induced hearing loss to primates: loudness recruitment *In* Adv Oto-Rhino-Laryng, Vol. 20 Basel: S. Karger. pp 82-101 Figure is shown in Smith & Vernon, 1976.
[13]Moore, B. (1977) Introduction to the Psychology of Hearing. London: Macmillan Press pgp 83-85
[14]Plack, C. & Carlyon, R. (1995) Loudness perception and intensity coding *In* Moore, B. ed. Hearing. NY Academic Press pp 127-128
[15]Allen, J. (1997) Derecruitment by multiband compression in hearing aids *In* Jesteadt, W. *ed*. Modeling sesorineural hearing loss Mahwah, NJ: Lawrence Erlbaum Associates pp 99-112
[16]Moore, B. & Glasberg, B. (1993) Simulation of the effects of loudness recruitment and threshold elevation on the intelligibility of speech in quiet and in a background of speech *J Acoust Soc Am* vol. 94(4), pp 2050-2062
[17]Allen, J. Hall, J. & Jeng, P. (1990) Loudness growth in ½-octave bands (LGOB)–a procedure for the assessment of loudness *J Acoust Soc Am* vol. 88(2), pp 745-753

makes the following comment. "Our method for simulating the effects of loudness recruitment is based upon a consideration of its probable underlying cause." It does not appear they feel they have determined that cause with confidence. However, their later comments are very useful. If we accept this account of recruitment as a working hypothesis, then we may regard the normal ear as containing a compressive nonlinearity (a kind of automatic gain control, AGC), whereas the impaired ear lacks this nonlinearity. The exact time cours of operation of the compressive nonlinearity is a normal ear does not appear to be known. However, it clearly works very rapidly, within a few ms." These specific points are supported by this work over a portion of the hearing range. However, a more complete and detailed model covering the entire dynamic range of hearing is offered here. Their conclusion is relevant. "The main conclusion of this study is that threshold elevation combined with loudness recruitment is sufficient to produce a substantial decrease in the ability to understand speech in the presence of a single competing talker, especially when the recruitment is associated with a sloping hearign loss. Linear amplification of the type typically used in hearing aids compensates for this effect only to a limited extent, and does not restore performance to normal."

Carney has provided a mathematical model attempting to describe recruitment for modeling purposes[18]. While the model includes many of the elements expected in such a model, no details of the individual elements showing they are directly related to the underlying physiology are offered. This is particularly true of his time-varying narrowband filter prior to the traveling wave delay associated with the cochlea.

Heinz & Young have provided considerable empirical data in a recent paper[19]. Their introduction does provide a wide bibliography. However, they did not focus on the mechanism underlying their "recruitment." They adopt a simple definition of the effect and refer to the review to a 1995 review by Moore. Their figure 1A clearly and correctly describes the sensorineural hearing loss generally described by the clinical term recruitment, except the label on the ordinate mis-assigns the source of the problem. The root cause is the loss of neural amplification within the sensory neurons as developed in the following figure.

Buus & Florentine have provided a well defined data set and related discussion[20]. They illustrated the difficulty in working without a physiologically compatible theoretical model by their comparison of two definitions of recruitment in item 6 of their conclusions:

1. Recruitment– An abnormally large loudness at an elevated threshold. (Buus & Florentine, 2003)

2. Recruitment– An abnormally rapid growth in loudness above an elevated threshold. ("Classical")

These two definitions continue to miss the subtle details of the condition as shown most clearly in their figure 3, reproduced here as **Figure 10.2.1-5**. The figure has been supplemented by construction based on the theoretical model of this work. A premise of this work is that the perceived loudness is proportional to the intensity of the stimulus within the mesotopic regime between 15 dB and 45 dB SPL. Above this level, adaptation introduced a significant reduction in the slope of the loudness to intensity relationship that extends up to the 100 dB SPL level. Moore made an astute observation in 1977 related to normal vision[21]. "At low sensation levels (around 10–20 dB SL) the loudness of a complex sound is roughly independent of bandwidth. This is also easy to explain. At these low levels firing rates change relatively rapidly with intensity and so does loudness. The loudness of a single critical band changes almost in direct proportion to intensity, . . ." This is precisely the condition described in the figure.

The range from 45 dB to 100 dB defines the phonotopic regime of hearing. A series of diagonal lines have been drawn for a gamma of 1.0. A system performing linearly and at a constant gain will follow one of these lines. Also shown is a dashed line sloping up to the right at a gamma of 0.3 and passing through the measured data points of the normal subject. This is the slope usually associated with the plateau in the human response defined as the phonotopic regime. This slope is controlled by the internal feedback factor associated with the adaptation amplifier of the "normal" sensory neurons. This regime extends to a

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[18]Carney, L. (1994) Spatiotemporal encoding of sound level: models for normal encoding and recruitment of loudness *Hear Res* vol. 76, pp 31-44

[19]Heinz, M. & Young, E. (2004) Response growth with sound level in auditory-nerve fibers after noise-induced hearing loss *J Neurophysiol* vol. 91, pp 784-795

[20]Buus, S. & Florentine, M. (2003) Growth of loudness in listeners with cochlear hearing losses: recruitment reconsidered *J Assoc Res Otolaryngol* vol. 3, pp 120-139

[21]Moore, B. (1977) The Psychology of Hearing. London: Macmillan pg 97

nominal stimulus intensity of 100 dB SPL where the adaptation mechanism is no longer functional. At higher stimulus intensities, the response of the normal subject (in terms of the current passing through the distribution amplifier of the sensory neuron proceeds upward along a gamma equal 1.0 line. However, the rise in the voltage at the pedicle of the sensory neuron is subject to further limitation by saturation in the diode-based current to voltage conversion circuit associated with the axon.

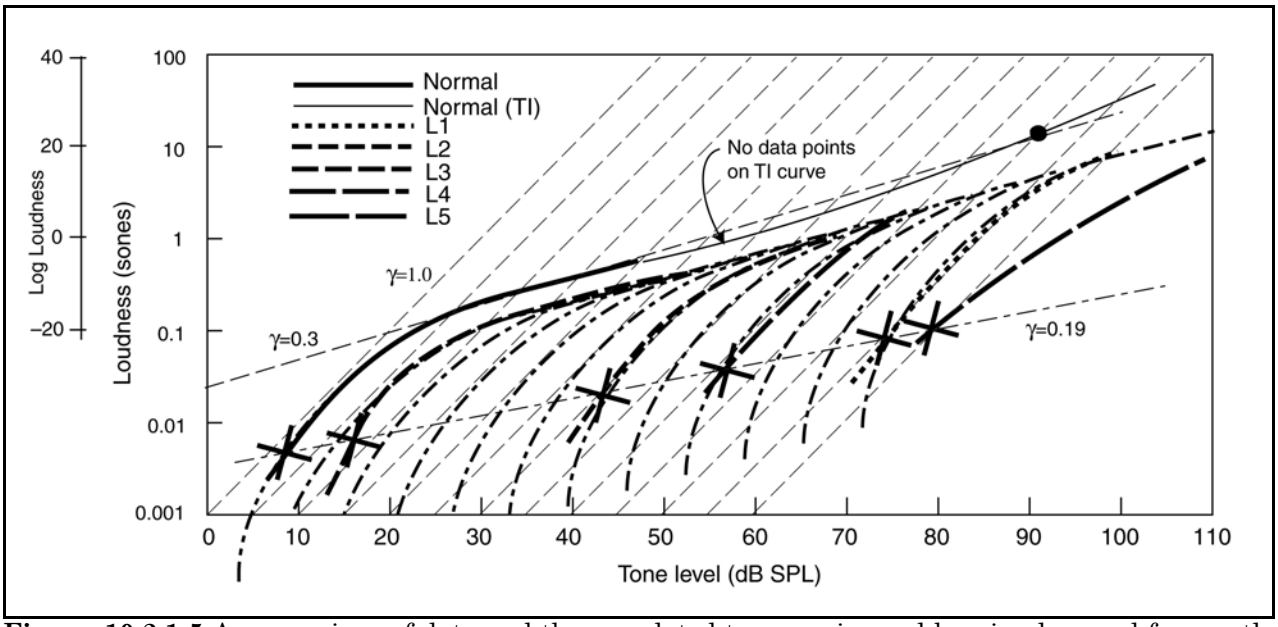

Figure 10.2.1-5 A comparison of data and theory related to sensorineural hearing loss and frequently described using the label recruitment. Empirical data from Buus & Florentine, 2003). Theoretical constructions based on this work. Note a lack of data points in the extended normal (T1) line of Buus & Florentine. The straight diagonal dashed line represents a linear system with a $\gamma = 1.0$. A line at a slope of $\gamma = 0.3$ is shown drawn through the normal curve for reference. This work suggests the "normal (T1)" curve reverts to a slope of $\gamma = 1.0$ above 100 dB SPL. The light set of dash-dot lines represent a copy of the measured "normal" response lowered by a factor of two and replicated along a $\gamma = 0.3$ diagonal at intervals of five dB. These replicas match the empirical data well. See text.

There is an important difference in concept between "the common comment that the ***loudness rises exceptionally rapidly*** in the mesotopic and kaumotopic regimes compared to the phonotopic regime" and the alternate, "the ***rise of loudness is reduced*** in the phonotopic regime relative to the rise as a function of intensity observed in the kaumotopic and mesotopic regimes." The latter concept recognizes that the mechanism of adaptation was introduced into hearing with the specific purpose of extending the overall dynamic range of the system. The same adaptation mechanism is found in vision and presumably all other sensory modalities.

A similar difference occurs between the concept of ***a level-dependent attenuation at low intensity levels***[22] and ***a level dependent amplification at phonotopic intensity levels*** as a fundamental mechanism of hearing and relevant to a source of sensorineural hearing loss.

This work will show it is the amplification factor of the sensory neuron that is reduced in both normal adaptation (leading to a reduced rise of loudness in the phonotopic regime) and in the most common form of sensorineural hearing loss (leading to a loss in sensitivity at low stimulus

[22]Neely, S. & Allen, J. (1997) relationship between the rate of growthof loudness and the intensity DL. *In* Jesteadt, W. *ed*. Modeling Sensorineural Hearing Loss. Mahwah, Nj: Erlbaum pp 21-222

intensities.

The data of Buus & Florentine can be interpreted with considerable specificity based on their data and this theoretical construction. First, the reduction in amplitude of the response by a factor of two in sones will be ignored temporarily. Under this condition, the presumed sensorineural hearing losses associated with subjects L1 through L4, and probably L5, can be described as a loss in maximum adaptation amplifier gain. The loss predicted by this work for L1 is 55 dB using the weighted curve of Buus & Florentine. They give this subjects loss as between 50 and 65 dB HL for frequencies between 1 and 8 kHz and ignoring their values for 250 and 500 Hz. 55 dB approximates the total gain associated with the adaptation mechanism in the Standard Ear of this work. The loss for L2 is 30 dB (versus the un-weighted 40 dB of Buus & Florentine at 1000 Hz). The loss for L3 is only 5 dB (versus 10 dB at 1000 Hz). L4 shows a loss of 40 dB (which equals the value of Buus & Florentine). The responses of these subjects all follow the dash-dot replicas of the normal ear quite well. Subject L5 is the only one showing a significantly different shape than that of the replicas. This work suggests his loss was 60 dB versus the un-weighted 70 dB of Buus & Florentine. It would be interesting to see the actual data points for the experiment using this subject and see if any of them could be considered out outliers.

Look now at the small vertical displacement in the data points of the normal versus the subjects with sensorineural hearing loss that was ignored earlier. Buus & Florentine describe their explanation for this displacement on page 132. "The model predicts a small loudness-level loss owing to loudness summation near threshold because it assumes that the loudness increases more rapidly than intensity (i. e., the local exponent is larger than unity) near a threshold that is elevated by masking. If the local exponent is unity, no loudness-level gain (or loss) owing to loudness summation should be obtained, ..." No mechanism for achieving a local exponent (gamma) greater than one has been identified in the circuitry of the hearing system in this work. Buus & Florentine did not identify any mechanism providing a gamma greater than one. They introduced a fitting function in their mathematical model that allowed the local exponent (gamma) to be greater than one. This work suggests that any calculated local exponent greater than one is due to statistical problems (and possibly preconceived ideas based on earlier proposals) unrelated to the underlying mechanisms.

The effect of the loss of adaptation amplifier gain is to narrow the plateau region associated with phonotopic hearing. As a result, the subject encounters a higher gamma experience over most of his auditory range. At the simplest level, the overall response can be averaged to approximate the pedagogical graph by Plack & Carlyon in Moore (page 127). However, this action obscures the potential causes of the problem.

Buus & Florentine struggle with the gamma of the hearing mechanism at levels near threshold and specifically with their interpretation of the perceived intensity (loudness) expressed in sones (page 122). They note, "Although many authors have proposed loudness functions according to which loudness at threshold is zero (for review, see Buus et al. 1998), our finding of nonzero loudness at threshold should hardly be surprising." Reviewing this referenced paper[23] surfaces a fundamental problem found in the literature. All of the equations cited are first order mathematical equations that were proposed based on very limited empirical data and no significant model of the physiological processes involved. They are examples of curve fitting a single (simple) continuous equation to a compound response due to multiple underlying mechanisms. This procedure is obviously inadequate. None of the discussed equations are sufficiently flexible to describe the actual loudness-intensity relationship or the phenomenon labeled recruitment. Any mathematical treatment describing loudness (perceived intensity) as a function of stimulus intensity must recognize the roles of avalanche gain, non-linear negative internal feedback and the nonlinearity of the current-to-voltage conversion process in that function.

Buus & Florentine show a threshold for each of their measured loudness versus stimulus functions (indicated by the X symbols). The thresholds appear to align along a slope they describe as $\gamma = 0.19$. This value of gamma was one of four free parameters plus a scale factor in their final empirical equation. These parameters could not be interpreted as applying to any physiological model. They did note that this parameter "showed substantial differences across listeners. They ranged from 0.14 to 0.62 for the three listeners with hearing losses that varied considerably across the range of component frequencies, which indicates that loudness at threshold increases systematically with the amount of hearing loss within each listener, but the rate of increase differs across listeners." Their threshold values will not be addresses

[23]Buus, S. Musch, H. & Florentine, M. (1998) On loudness at threshold *J Acous Soc Am* vol. 104, pp 399-410

further here.

The 1998 paper also suffers from a lack of mathematical rigor in its mathematical framework. One of the keystones of the paper concerned their proposal that the perceived intensity (loudness) in sones did not begin at zero value at threshold, a common assertion in the pedagogy of recruitment. Although it recognizes both a loudness parameter and a loudness function which is defined as the logarithm of the loudness parameter (the perceived intensity), they are inconsistent when discussing them. Loudness is traditionally been plotted using a logarithmic scale, in consonance with the conventional intensity scale. These are both relative scales that exhibit no absolute zero value. This fact is illustrated in their figures 1 and 3. In the caption to figure 5, they use the term loudness function in the vernacular, instead of their technical meaning, to describe a relationship shown by a line on a graph. When the logarithm of the intensity parameter is taken, the resulting scale is given a zero value corresponding to an intensity of 20 micropascals. When the logarithm of the loudness parameter is taken, the resulting loudness function can be assigned a value of zero wherever desired. It is absolutely arbitrary. It can be taken as zero at one sone—which is itself arbitrarily defined relative to 40 dB SPL—which is itself arbitrarily defined relative to 20 micropascals). Alternately, it can be taken at a "threshold value" read from a graph. The paper also speaks of the sum and difference of random variables in the simplest language. They describe an "absolute threshold" instead of a threshold defined relative to the RMS value of a random noise source.

All of their data was manipulated extensively in arriving at the average estimates of loss versus intensity plotted in their figure. This discussion is based largely on the assumption that their results still contain the essence of the loudness versus intensity relationship. It would be useful to repeat their experiments using the protocol developed in their paper except using only one stimulus frequency at a time and plotting that data without using an arbitrary summation protocol.

The loss of hearing sensitivity associated with the term recruitment does not involve neural circuits orthodromic to the adaptation amplifier. In particular, the internal negative feedback introduced by the common emitter impedance is not affected (except for the fact that the current through the impedance is reduced at a given level of stimulus intensity). The effect of this lower current level is to shift the transition between a gamma of 1.0 and a nominal gamma of 0.3 to the right along with the threshold intensity level.

10.2.2.1.1 Redefinition of the phenomenon previously known as recruitment

Based on this data and this interpretation, the vernacular term recruitment can be defined scientifically and very precisely.

3a. Recruitment– (as a phenomenon) The reduction in the maximum average amplification factor achievable within the adaptation amplifiers of a group of sensory neurons forming a single signaling channel and responding within a specific critical bandwidth.

3b. Recruitment– (as a clinical condition) A loss of auditory sensitivity recognized by a horizontal shift in the loudness versus intensity function.

These definitions are specifically applicable to OHC neurons. The loss can be evaluated on a channel by channel basis where each channel exhibits a distinct critical frequency. A loss of 10 dB in this maximum amplification factor results in a sensorineural loss (measured clinically) of 10 dB.

The term recruitment is completely inappropriate based on the underlying physiological problem. It is antiquated and should be discarded by the community. The deficit does not involve the recruitment of signals from separate individual sensory (or signaling) channels as originally proposed.

The new definition has little in common with the earlier definitions given above.

Based on this theoretical interpretation, the scientific literature concerning recruitment prior to 1998 should be discarded. The description of recruitment by Phonak shown on the world wide web, in conjunction with their Claro product line, should also be updated when more than a consumer version of the effect is needed.

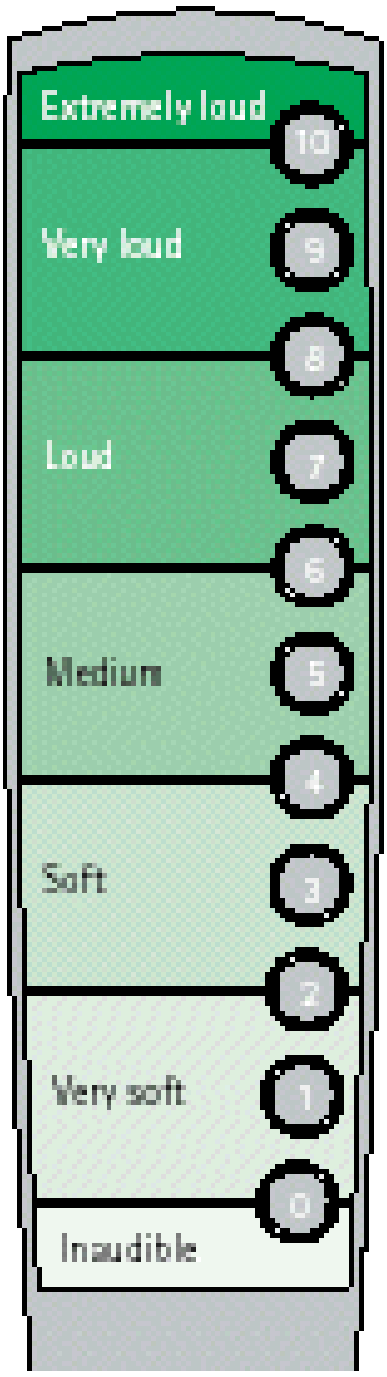

Figure 10.2.1-6 A loudness scale correlated to the conventional SPL intensity scale. From Phonak, 2004.

[xxx what to do with this figure]
Figure 10.2.1-7 illustrates the possible forms of recruitment. The upper frame shows the general situation as proposed by this theory. The lower frame shows an expanded view of the region between the presumed noise floor and hypertopic operation.

10.2.2.1.2 Potential causes of XXX alias Recruitment

The potential causes of sensorineural hearing loss of the type previously associated with the concept of recruitment. can be described based on the Electrolytic Theory of the Neuron and the associated model. It involves the partial to complete failure of the gain mechanism associated with (at least) the OHC sensory neurons. It is likely the loss is also found among the IHC. Two primary causes of the problem are readily identified. The first would involve a physical deformation (primarily thickening, of the type 2 dendroplasm in the immediate vicinity of each stereocilia of the sensory neuron. This could involve the replacement of the triglyceride molecule normally found there with one of greater axial length. This would entail a genetic code-based change that is unlikely to occur in mid to later lifetimes.

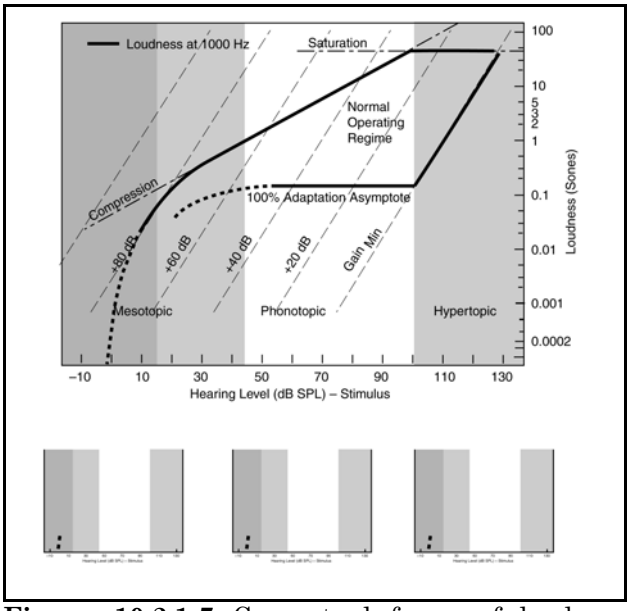

Figure 10.2.1-7 Conceptual forms of loudness recruitment EMPTY ADD.

The second likely cause involves a change in the electrical potential supplied to the collector fo the Activa of the adaptation amplifier within each sensory neuron. This failure can be caused by several changes in the metabolic system supporting the electrostenolytic process on the surface of the type 2 dendroplasm. Currently, they can only be placed in an un-ordered list.

1. Isolation of the stereo-specific electrostenolytic sites due to occupation by “foreign” molecules.

2. Insufficient concentration of the neuro-facilitator, glutamic acid, in the perilymph fluid supplying the sensory neurons.

3. Excessive concentration of the neuro-inhibitor, GABA, in the perilymph fluid supplying the sensory neurons.

4. The presence of other neuro-inhibitors within the perilymph.

The cause of the incorrect chemical concentrations can obviously be referred back along the cardiovascular supply chain to the blood stream.

10.2.2.2 Minimally invasive data on human hearing losses

Yoshie and Ohashi have obtained minimally intrusive data reflecting the cumulative static performance of the human hearing modality at the pedicle of the sensory neurons to clicks[24]. Unfortunately, their notation is dated. They describe the signals they recorded as consisting of action potentials (which have

[24]Yoshie, N. & Ohashi, T. (1969) Clinical use of cochlear nerve action potential responses in man for differential diagnosis of hearing losses *Acta Oto-laryng Supp* 252, pp 71-87

constant amplitude). Their data actually reflects the generator potential at the pedicles of the sensory neurons upon excitation. Their latency data is also based on the clinical protocol of measuring delay to the top of the waveform instead of to the beginning of the response (as in the relevant physiology and mathematics). Their data was collected by averaging the responses to 500 clicks at each intensity level in order to isolate the low signal level from the high background. **Figure 10.2.1-8** illustrates their data with additional annotation.

The upper dotted line labeled A.F. represents a subject with normal hearing. It has been overlaid by a diode characteristic and two lines with slopes of 1.0 and 0.3. Together, they illustrate that the normal first order human generator response follows the diode characteristic proposed as the load impedance of the sensory neuron distribution amplifier. The response also exhibits a slope in the 1.0-1.5 region for stimulus intensities below 45 dB SPL and a slope in the region of 0.3 for stimulus intensities above 45 dB SPL. The response clearly exhibits a slope associated with recruitment by many in the clinical community. However, it is a normal feature of all auditory systems.

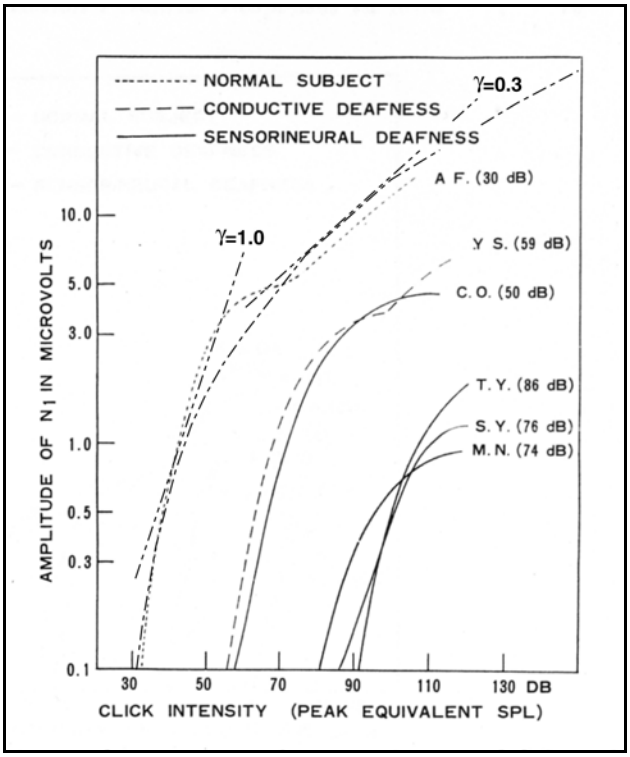

Figure 10.2.1-8 Variations in the cumulative human performance of stages 0 & 1 measured by the generator potential, N1, as measured at the promontory of patients with different types of hearing loss. See text. Data from Yoshie & Ohashi, 1969.

As noted in **Section 5.5xxx**, the absolute magnitudes shown on the ordinate are not reliable because they vary with the precise location of the sensing electrode. However, it is interesting to note the similarity of the response of Y. S. (dashed line) to that of A.F. The loss appears to be a pure amplification loss of 29 dB occurring before the diode element in the circuit.

Interpretation of the other subjects is more difficult from this figure. Yoshie & Ohashi indicate they all represent sensorineural hearing losses. However, the curves do not extend to high enough levels to confirm the ultimate shape of their responses. The curves appear to follow a diode characteristic until they begin to show an even more severe saturation at high levels. This would be the expected result if the distribution amplifier Activa went into saturation earlier than expected. Such a condition could occur if the active area of the base of the Activa was reduced. It may also be associated with a higher than normal emitter potential in the distribution amplifier. This condition could be caused by a change in the common emitter impedance shared with the adaptation amplifier. The conditions in this figure do not suggest a bias error between the pedicles of the sensory neurons and their orthodromic synapses.

Figure 10.2.1-9 reproduces Figure 4 in the Yoshie and Ohashi paper. It suggests the sensorineural loss may occur even earlier in the signal chain. It shows latencies significantly longer in those subjects than for those with normal hearing. Their latency measurements for normal hearing agree quite well with the latencies predicted by the P/D Equation. By shifting the brackets enclosing the latency of normals to the right by the magnitude of the hearing loss of the subjects, good agreement is seen between the data and the expected delay if the loss is due to a reduction in absorption cross-section of the piezoelectric material in the cilia. These long latencies would suggest an inadequate number of cross links available in the piezoelectric material of the cilia or an abnormality in the electrical bias supplied to the piezoelectric material by the endolymph. The latter abnormality could easily be generated in a fluid leakage path between the endolymph and the perilymph of the scala vestibuli, a condition frequently associated with Meniere's disease. It could also be caused by a electrical leakage path (or fluid leakage path) between the endolymph and the perilymph of the scala tympani.

10.2.2.3 XXXEchos within the auditory system

5.5.4 Revisiting the concepts of recruitment and tinnitus

Because of their close association with the interface between the sensory neurons and the subsequent orthodromic neurons, the diseases labeled "cochlear hearing loss with recruitment" and "tinnitus" will be briefly discussed. Functionally, these diseases closely parallel similar diseases in vision where the symptoms are "snowy vision" and "achromatopsia."

Cochlear hearing loss with recruitment has long been recognized in the clinical environment as the loss of sensitivity at low stimulus levels without a similar loss at high stimulus levels. The term recruitment was associated with this type of loss based on an analogy with a conceptually similar phenomena in the the neuro-muscular system. In fact, the analogy is a poor one.

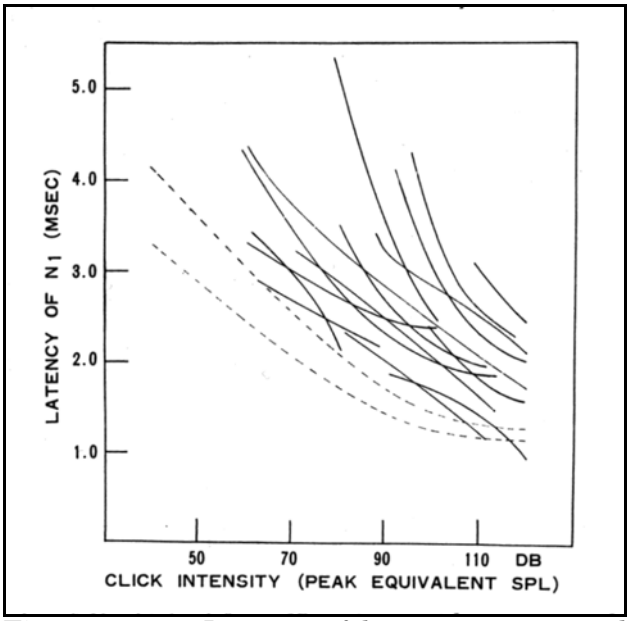

Figure 10.2.1-9 Latencies of the generator potential response obtained from 15 subjects with sensorineural deafness. Each solid line was averaged by eye from three sets of measurements based on identical stimuli. The dashed lines bracket the typical response of those with normal hearing. Yoshi & Ohashi, 1969.

Defining recruitment precisely in words has proven particularly difficult. Such expressions by leading experts in the recent literature are frequently contradictory at the detailed level. Use of a graphical description is far more satisfactory. Unfortunately, no physical or schematic description (beyond the most gross psychophysical models) of this disease has been presented in the research literature.

The disease known as tinnitus has a similar nebulous foundation. The symptoms of this disease vary widely but generally are associated with a nominally continuously perceived random or quasi-random background sound. The origin of this sound have not been isolated in the prior literature.

The laboratory work on the subjects of recruitment and tinnitus have been long on psychophysics and short on electrophysiology up to this time.

Summarizing the conclusions of this section, these two diseases can be defined more specifically based on this work. They are both manifestations of an electrolytic bias error between the axons of the sensory neurons (node E) and the dendrites of the orthdromic neurons (node E') in [**Figure 5.xxx**]. Recruitment is not associated with any failure within the sensory neurons. Hearing loss with recruitment and tinnitus are both caused by non-genetic variations in the hydraulic and chemical systems supporting the electrostenolytic mechanisms generating voltages within the neurons. In the authors case, these hydraulic and or chemical variations associated with tinnitus are only quasi-permanent. The symptoms do disappear periodically for intervals of one hour or more.

5.5.4.1 Background

The archaic concept of recruitment is discussed in detail in Hallpike and in Eggermont, both writing in Keidel & Neff[25]. The material by Hallpike is quite extensive and allows a clear understanding of the mechanisms underlying the concept based on the modern model used here. Both the monoaural and

[25]Keidel, W. & Neff, W. *eds*. (1976) Auditory System: Clinical and Special Topics. Vol. V/3 of Handbook of Sensory Physiology. NY: Springer-Verlag Chapters 1 & 15

binaural aspects of recruitment are discussed. [Xxx add words based on Hallpike figures.]

Eggermont, writing in 1976 asserted, recruitment is the abnormally rapid increase in the loudness sensation with increase in intensity of an acoustic signal.

Harrison has also discussed recruitment briefly[26]. [xxx not very important unless I find other pages on subject]

Allen provides an alternate historical review of recruitment[27]. The first page of his material provides a different perspective on recruitment versus the common wisdom. It is in consonance with that provided here. He notes the shaky introduction of the concept in the 1930's and says it is "generally misdefined as *the abnormally rapid growth of loudness*. It is not well known that "loudness" in sones does *not* grow more rapidly in the recruiting ear; rather, it is the "loudness-level" in phons that grows more rapidly." To justify this statement, he proceeds to define his terms in a less than simple manner. Subsequent material in the Allen paper relies upon the conventional wisdom regarding the sensory neurons and is not supported here. His assertions in the caption to figure 6.4 requires further discussion. "The belief that the loudness slope in the damaged ear is greater led to the belief that the JND in the damaged ear should be smaller. Both conclusions are false." He defends his position based on his simulation of the psychophysical situation and an interesting observation; the algebraic difference between two pairs of real positive numbers is always smallest for the pair with the smaller maximum number [algebra 101]. However, the ratio between the same two pair of numbers is largest for the pair with the smaller maximum number. Two questions arise. First, are algebraic differences or ratios are of primary interest in hearing? The empirical work of Fletcher and his contemporaries were clearly based on ratios. Second, should the sone be defined in algebraic units or in dB? The assertion in the final paragraph of the paper is far from accepted by those suffering from the disease and from those studying the auditory system.

Neely & Allen put another face on the subject in the same volume[28]. They expand the framework by introducing the term "loudness recruitment" and exploring the relationship between this term and the just noticeable difference (JND) in normal and diseased systems. Initially, it must be noted that they made no effort to empirically determine the location of the dominant noise source in the hearing modality. The analysis of this work suggests the signal processing neuron orthodromic to each sensory neuron presents a threshold that limits the lowest perceived level of hearing while simultaneously preventing the lower level noise contribution from the sensory neuron being passed up the auditory signaling chain. Because of this threshold, the dominant noise contribution to the system originates in the decoding circuit, generally associated with a stellate neuron, in stage 3. It is this source that establishes the JND in the healthy system. The JND of systems exhibiting the symptoms of tinnitus is controlled by a different noise source. The source becomes the internal noise associated with the sensory neuron.

In their last paragraph, they drew a specific conclusion. "Loudness recruitment should not be defined as 'a more rapid growth in loudness.' Loudness recruitment represents a loss of the dynamic-range compression normally provided by outer hair cells." This conclusion is inherently based on the assumption that the outer hair cells are active moto-neurons rather than sensory neurons. In fact, both the inner hair cells and the outer hair cells incorporate the logarithmic conversion of current to voltage that is interpreted as dynamic-range compression in their analysis. Their final sentence addresses the question of how the sone is defined. "This results in a steeper log-loudness slope; however, it does not imply that the impaired ear has a steeper loudness slope (with loudness plotted on a linear coordinate, ed.) when the comparison is made at the same intensity."

Buus & Florentine reviewed the argument over loudness change based on inadequate agreement on definition in 2001[29]. Buus & Florentine have continued the debate over the definition of recruitment[30].

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[26]Harrison, R. (1988) The Biology of Hearing and Deafness. Springfield, Il: Charles C. Thomas pp 353-355

[27]Allen, J. (1997) Derecruitment by multiband compression in hearing aids *In* Jesteadt, W. *ed*. Modeling sensorineural hearing loss. Mahwah, NJ: Lawrence Erlbaum Assoc. pp 99-112

[28]Neely, S. & Allen, J. (1997) Relation between the rate of growth of loudness and the intensity DL *In* Jesteadt, W. *ed*. Modeling sensorineural hearing loss. Mahwah, NJ: Lawrence Erlbaum Assoc. pp 213-221

[29]Buus, S. & Florentine, M. (2001) Growth of loudness in listeners with cochlear hearing losses: recruitment reconsidered *JARO* vol 3, pp 120-139

[30]Florentine, M. & Buus, S. (2002) It's not your father's recruitment: a new view of loudness growth in cochlear hearing loss. Abstract in *J Acoust Soc Am* vol 111(5), p 2353

They maintain, "listeners with cochlear hearing losses not only have reduced dynamic range of audibility; they also have reduced dynamic range of loudness."

Moore entered this discussion by testing the Buus & Florentine proposal according to his understanding. He asserts that their proposed "softness imperception" is not realized[31]. "The results show that, for levels very close to absolute threshold, a given SL leads to approximately the same loudness for an ear with a hearing impairment at the test frequency and an ear with normal hearing at the test frequency." "This occurs for Sls in the imparied ear up to 4-10 dB, depending on the subject. For Sls above 4-10 dB in an impaired ear, the rate of growth of loudness with increasing level is greater than normal; this is the main basis of the loudness-recruitment effect."

The problem with this tempest in a teapot is that the different groups are talking about different situations and frequently do not define their terms and framework adequately. The sensory modalities of biology all face wide ranges of signal intensity in their environments and have been designed specifically to handle this range. As a result, they contain an intrinsic logarithmic transformer plus a number of adaptive elements. Even using this transformer, the range of signals is large with respect to the dynamic range of the individual neurons. As a result, virtually all of the neurons operate under large signal conditions. Nonlinear operation, signal compression and saturation are to be expected within the system. To appreciate the performance of these systems requires the use of logarithmic scaling in most descriptions of their performance. In the logarithmic environment, it is the ratio of two signals that is most often relevant. In the rare case where small signals are of primary interest, it is easy to use the mathematical approximation relating small arguments of a logarithm to the logarithm of that argument. The proposal by Allen that the loudness slope, dL/dI plotted against logI, should be used rather than the conventional d(logL)/d(logI) plotted against logI appears to offer little. The lower right frame of his figure 13.1 does show the conventional slope is continuing to rise near 0 dB SPL in the normal system, as predicted by this theory. His function can be overlaid with the same exponential used to describe the diode at the axon of the sensory neuron. It is this exponential function that defines the phenomenon associated with the term recruitment. No other mechanism is required. The slopes (local exponents) of 1.25 and 1.31 noted by Buus & Florentine for those with and without hearing loss both correspond to a stimulus of about 20 dB SL and their difference is probably not statistically relevant.

With the above considerations in mind, a stimulus consisting of a stair step waveform where the ratio of the stimulus level of one step to the next is a fixed value is preferred.

The following material will demonstrate that the slope of the perceived intensity, or loudness, in sones in dB as a function of the stimulus in dB SPL is finite at and near threshold and that this slope is higher than the slope in regions of higher stimulus level due to the compression normally encountered. This effect is defined by the logarithmic relationship between the stimulus and the loudness, or perceived intensity. The slope of the response in the impaired ear is typically similar to that of the unimpaired ear near threshold.

Heinz et al. have recently provided an extensive paper on hearing loss and recruitment in cats[32]. It is important to note they equate noise induced hearing loss (NIHL) with sensorineural hearing loss (SNHL). This places an inappropriate limitation on the scope of SNHL. They also rely upon the common frequency (or rate) histogram which distorts their data at the detailed level (Section xxx).

Moore & Glasberg have written extensively on the disease associated with the label recruitment. However, their work has been limited to abstract mathematical models describing their conceptualization of the mechanisms involved[33]. The 2004 paper, and its predecessor[34], have employed the concepts of broad filters

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[31]Moore, B. (2004) Testing the concept of softness imperception: loudness near threshold for hearing-impaired ears *J Acoust Soc Am* vol 115(6), pp 3103-3111

[32]Heinz, M. Issa, J. & Young, E. (2005) Auditory-nerve rate responses are inconsistent with common hypotheses for the neural correlates of loudness recruitment *JARO* vol 6, pp 91-105

[33]Moore, B. & Glasberg, B. (2004) A revised model of loudness perception applied to cochlear hearing loss *Hear Res* vol 188, pp 70-88

[34]Moore, B. * Glasberg, B. (1997) A model of loudness perception applied to cochlear hearing loss *Audit Neurosci* vol 3, pp 289-311

within the system first studied by Zwicker. Unfortunately, they did not localize these filters or explain how the narrow bandwidth of the individual sound channels described by others are obtained. They did note an unusual feature of these filters and reference Zwicker & Fastl, 1990. The masking noise, which ostensibly defines the filter, is effective in masking the signal frequency even when the signal frequency is several octaves higher than the masker frequency. This feature is key to understanding the correlation mechanism found within stage 4 of hearing (**Chapter 8**).

The above researchers have not defined a viable physical or physiological model of the cochlear partition.

5.5.4.2 Framework for discussion of hearing abnormalities

The neurology community has separated the types of sensory system failures into those that are organic and those that are nonorganic[35]. Organic failures are either anatomic or chemical. Nonorganic are associated with psychiatric or behavioral symptoms untraceable to anatomic or chemical problems. This segregation leaves a large range of problems associated with electrophysiological abnormalities without a home. Additional classifications relating to failures at the molecular level (frequently genetic in origin), involving chemical transport (frequently a hydraulic problem), or involving electrical circuit failures are needed.

The hearing community has segregated hearing failures in to what are called conductive hearing loss and neural hearing loss. The simple analogy is that conductive losses occur in the acousto-mechanical portion of the system prior to any neural activity while neural losses occur within the neural system. The conductive losses are frequently associated with signal amplitude losses independent of the frequency. Unfortunately, the largest potential losses in both the acousto-mechanical signal level and the electrophysiological signal amplitude occur within the sensory neurons. Segregation of hearing losses into conductive losses and neural losses does not result in independent sets.

The ISO has now defined the sone as a unit of *loudness* or psychologically perceived intensity in a Standard (ISO Resolution 131-1959). One sone is defined at 1000 Hz as 40 dB above threshold. The loudness function (in sones) is defined as the sound pressure raised to the power of 0.6 for intensities above 40 dB (above threshold). Therefore, at 70 dB above threshold, the loudness is ten sones. Below 40 dB, the exponent varies significantly. This definition is restricted to the applicable region of Steven's Law. A more precise definition would follow Fechner's (logarithmic) Law without restriction as to source intensity level.

This work defines the *loudness level*, in phons, in object space as the sound pressure at a given frequency that equals the sound pressure of a 1000 Hz tone of specified SPL as perceived by a standard human ear. If the given frequency is 1000 Hz, the loudness level in phons equals the Standard Pressure Level in dB. This definition follows Fletcher, although the reader is cautioned that his paper contains a remarkable number of typographical errors and inconsistencies[36].

5.5.4.3 Threshold level hearing losses

Figure 10.2.2-1 describes the clinically recorded condition known as recruitment. The data in the middle and lower frames was obtained at 1000 Hz. Note the labels on the left frame in the middle row. They describe the major functional operating regimes of normal hearing. The nominal operating system operates at full amplification throughout the region below 45 dB SPL. However, below about 15 dB SPL, the nominal system is performance limited by noise introduced in the later stages of the system (nominally stage 3). Between 0 and 45 dB, the system normally operates at constant amplification. As a result, the internal signal level increases in proportion to the stimulus level. Above 45 dB, the system introduces compression into the signal stream by at least two separate mechanisms. The sensitivity of the piezoelectric transducer is reduced in proportion to the stimulus level and the diode load of the distribution amplifier further compresses the largest signal amplitudes. This is the phonotopic operating regime where signals are perceived as increasing only slowly in intensity as the stimulus level increases. Above 90-100 dB SPL, the amplification available within the sensory neurons reaches a minimum and the system goes into an advanced stage of saturation. The system encounters considerable distortion in this hypertopic regime.

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[35]Trobe, J. (2001) The Neurology of Vision. NY: Oxford University Press pg 369

[36]Fletcher, H. (xxx) Speech and Hearing in Communications. NY: Van Nostrand

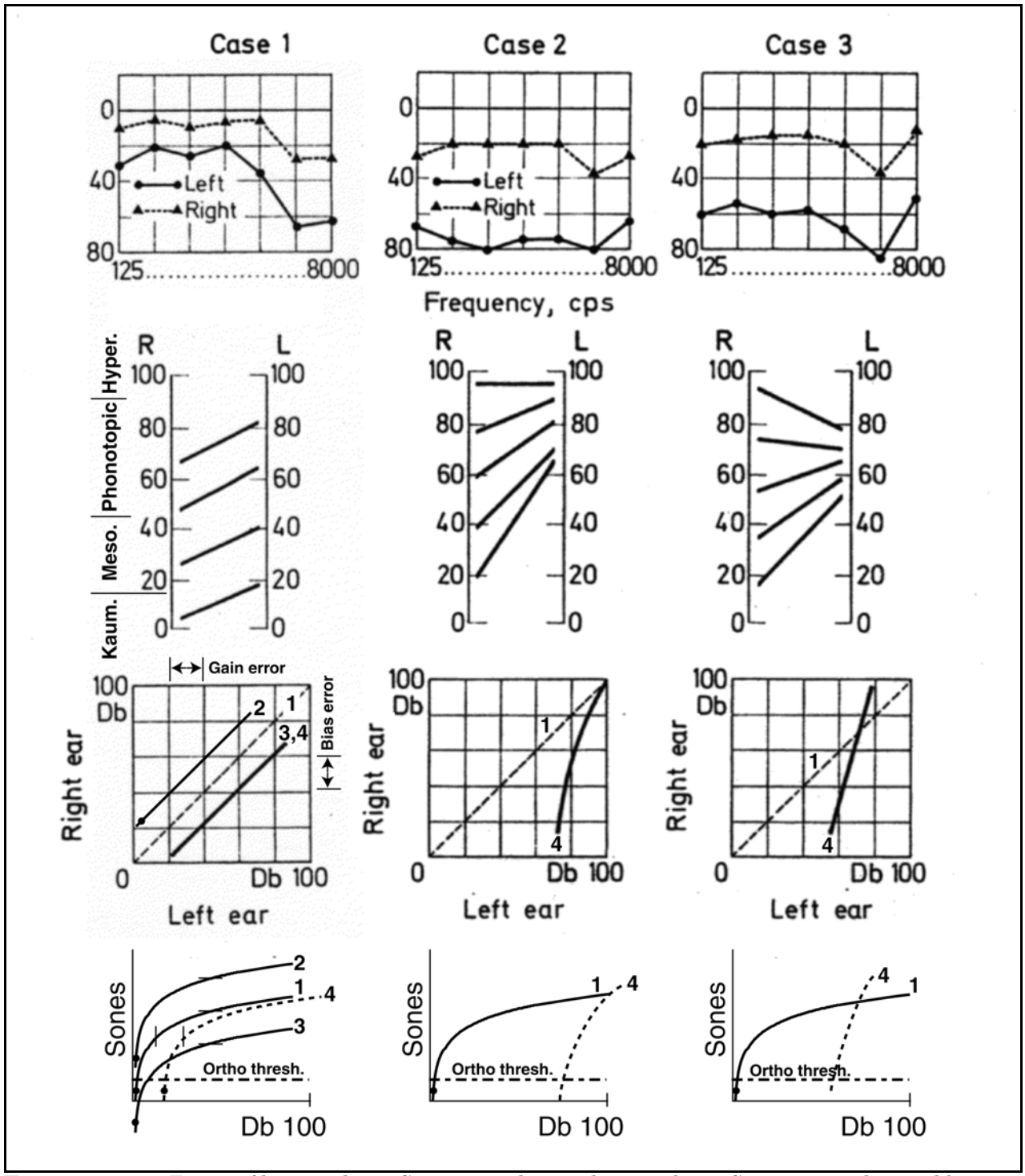

Figure 10.2.2-1 Forms of hearing loss. Case 1, conduction hearing loss. Case 2, typical neural hearing loss. Case 3, atypical neural hearing loss (or over-recruitment) showing additional compression or the presence of other mechanisms. Upper frames; conventional spectral scans. Middle frames; ABLB tests. The nominal regimes of hearing are labeled on the left graph. Next lower frames; Steinberg-Gardner graphs. Convention is to plot the affected ear on the abscissa in the lower frames. Bottom frames; Overall transfer function for two ears. Trace 1 is the normal ear. See text. Data included from Hallpike, 1976.

The first clear indication of a neural disease is the center frames based on the Alternate-Binaural-Loudness-Balance (ABLB) test". However, the more graphic description of the disease is shown in the bottom frames where the data is plotted in the Steinberg-Gardner graphic format. Case 1 shows a clear case of conduction based hearing loss. The ABLB lines are parallel and the Steinberg-Gardner graph shows a straight line parallel to the healthy locus (dashed line). Case 2 shows the most common form of the disease. The ABLB lines converge in the affected ear with the values at high intensity frequently approaching horizontal. Case 3 is a special case where the lines converge toward the affected ear at both low and at high intensities.

The cause of conduction deafness may be in either the acousto-mechanical or the post-sensory neural portion of the auditory system.

The next lower frames, known as Steinberg-Gardner charts, show the comparative performance of the two ears when subjected to tones of different intensity. It is conventional to plot the affected ear on the abscissa. Any time the data plots a locus below the equality locus, some disease is present. If the data plots as a line parallel to the equality locus, a loss in gain is involved in the disease. If the data plots at an angle of greater than 45 degrees with the abscissa, some form of variability in gain is involved. The angle is frequently, but not generally, frequency dependent.

- - - -

The Steinberg-Gardner chart for case 1 has been annotated to describe two different potential situations based on the circuitry of the sensory and initial orthodromic neurons. Trace 1 remains the equality locus for the two ears. This diagram can reflect two different errors in the diseased ear. A shift to the right reflects a reduction in signal amplitude at the synapse with the orthodromic neuron. This reflects a gain loss somewhere antidromic to the synapse (trace 4). A vertical shift can represent a mismatch between the dynamic range of the axoplasm and the dynamic range of the orthodromic neuron transfer function. A negative shift of the axoplasm potential (trace 2) threatens the maximum signal capability of the system. It also provides an additional capability at very low signal levels. Such a shift may also cause the noise source associated with the sensory neuron to move into the operating range as indicated by the filled dot. This will be discussed below. A positive shift of the axoplasm potential (trace 3) introduces a reduction in sensitivity at the low stimulus level that is difficult to distinguish from trace 4 unless very accurate measurements are recorded and the recording is extended to higher stimulus levels.

The lower row of frames illustrate the potential changes suggested by the Steinberg-Gardner charts. An arbitrary threshold level for the transfer function of the orthodromic neuron is also shown. In the left frame, the traces equivalent to the Steinberg-Gardner chart are shown. A pure gain loss moves trace 4 to the right without affecting the compression achieved at high stimulus levels. If the sensory neuron noise source is ahead of the gain loss, this noise source will be suppressed further. If it occurs after the gain loss, the equivalent amplitude of the noise source at the axoplasm will not be affected. In either case, the noise will remain below the threshold. If the disease is caused by a bias shift, a positive shift will move the graph up to trace 2. This will raise the compressed part of the curve up. This may result in additional compression or saturation at high stimulus levels by the orthodromic neuron. This shift will also raise the absolute level of the sensory neuron noise level to a value potentially above the threshold. If this occurs, the subject will report both more acute hearing sensitivity at low stimuli and a potential RMS noise source of unknown internal location. This condition is one form of the disease known as tinnitus in hearing. In vision, the name associated with the same disease is even more descriptive, snowy vision. If the bias shift causes the axoplasm to become more negative, the effect is illustrated by trace 3. A loss in perceived signal intensity will be reported near threshold and a reduction in maximum reported signal as a function of the stimulus will also be reported. The level of the equivalent sensory neuron noise will be pushed farther below threshold and will not be perceived.

Trace 4 in case 2 suggests a much greater loss in amplitude of the signal due to low stimulus levels at the axoplasm but no equivalent loss at 100 dB stimulus levels. The convergence suggested in the Steinberg-Gardner chart requires additional testing at high levels to ascertain whether the traces cross in both of the lower figures.

Trace 4 in case 3 suggests a considerable loss in amplitude of the signal due to low stimulus levels at the axoplasm and either a large positive bias shift or a loss in effectiveness of the compression mechanism. No single cause for such a condition is obvious. While the overall figure may be useful for pedagogical purposes, it is not clear that case 3 occurs frequently in practice. This condition also highlights the fact

that the Steinberg-Gardner chart may represent problems in the nominally good ear (compared to the diseased ear) when the trace crosses the diagonal into the upper quadrant.

- - - -
The bottom row of caricatures show the slope of the overall transfer function of hearing, from the stimulus in dB SPL to the perceived intensity (loudness) in sones, near threshold is finite and larger than it is for larger stimulus levels. This is the expected response based on the detailed performance of the system. It is consistent with the logarithmic processing of the stimulus within the sensory neurons. The effect is not recognized by Steven's Law because that law only applies to levels nominally 40 dB above threshold and higher.
- - - -

While recruitment is frequently associated with a concept labeled excess gain, both terms require further clarification. The term excess gain is usually used to describe the necessity of reducing an excess gain to a nominal level in order to accommodate loud signals. However, this gain is not in excess, it is absolutely required in order to sense signals at reduced stimulus levels. The term would be more appropriate if it were changed to full gain, the condition used in the mesotopic region and below.

A change in the concept to full gain within the mesotopic region places a different interpretation on the concept of recruitment. The concept of recruitment has been difficult to define and has suffered greatly in the literature over the years. Neely & Allen attempted to place a different face on recruitment in the conclusion of one of their articles in 1997[37].

> "Loudness recruitment should not be defined as 'a more rapid growth in loudness.' Loudness recruitment represents a loss of the dynamic range compression normally provided by outer hair cells. This results in a steeper log-loudness slope; however, it does not imply that the impaired ear has a steeper loudness slope when the comparison is made at the same intensity."

Unfortunately, they did not relate their semantics to a specific model of the auditory system. Their statement does little to reduce the confusion. A simpler explanation is available if the concatenation of the individual mechanisms is recognized. Anything that causes a lower level signal to be presented to the logaritmmic conversion mechanism will necessarily result in the performance recorded orthodromic to the pedicle of the sensory neuron to be shifted in the direction of higher required stimulus intensity.

Recruitment is specifically concerned with the voltages involved in the transfer of the signals from the sensory neurons to the first orthodromic neurite. Two related but distinct mechanisms are potentially involved. The first involves a change in the portion of the output characteristic of the distribution amplifier used for signaling. The second is related and involves a mismatch between the operating voltage range of the pedicle and the acceptance voltage range of the orthodromic neurite. Related to these two possibilities is the problem of biasing of the synapse. If the bias of the synapse should fail to maintain forward biasing of the input diode and reverse biasing of the output diode, the passing of signals across the synapse will cease. [xxx not a reported problem] [xxx add figure]

[xxx edit together with previous paragraph]
Recruitment results from the movement of the entire auditory response of an individual to the right due to a loss in performance of the auditory system prior to the neural system. The result is a measured performance that exhibits a higher slope in the region normally associated with phonotopic region of healthy ears. Nothing has changed in the operation of the neural portion of the subjects hearing. The term recruitment should be abandoned in favor of a more accurate description of its cause, the loss in signal gain prior to the neural system.

A further description of this postulate, the proposed abandonment and the relative data is offered in Section xxx where internal damage to the auditory system is discussed. [xxx move the quote to the other Section xxx leave the analysis.]

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[37] Allen, J. & Neely, S. (1997) Relation between the rate of growth of loudness and the intensity DL *In* Jesteadt, W. *ed*. Modeling sensorineural hearing loss. Mahwah, NJ: Lawrence Erlbaum Assoc. pp 213-222

Loudness is a psychophysical perception related to the intensity of a stimulus.

The **intensity** of the stimulus is defined in engineering units. This intensity is specified in terms of the absolute intensity scale (not power) and known as the specific pressure level (SPL) relative to an intensity of 20 micro-Pascal.

Volume is a measurement of the intensity of the stimulus.

The relationship between the loudness (perceived intensity) and the actual applied intensity as a function of frequency was first described in 1927-29. The fundamental relationship is that at threshold level. This relationship is reproduced faithfully within the mesotopic region. However, the relationship becomes time dependent as well as frequency dependent in the phonotopic region. It is greatly distorted in the hypertopic region. Iso-loudness levels are given using a phon scale based on the threshold level as a reference. They describe the intensity of two or more tones that will be perceived as equal in loudness. This relationship is a complex one. For tones separated in frequency by more than a specific percentage, different OHC and signaling channels will be excited. As a result, the tones will be subject to different levels of adaptation based on events immediately prior to the test. In addition, the level of the tones will be perceived differently in the mesotopic region than in the phonotopic region.

> During the 1950's high quality audio amplifiers, such as those made by Harmon-Kardon, provided both a volume control and a loudness compensation control on their front panels. The manufacturer recognized that the distance of the listener from the speakers introduced a variable in the perceived sound.

[xxx Work in Allen pg 99 in Jesteadt '97 on "de-recruitment"]

5.5.4.3 xxx (aka recruitment) is a definable disease

[xxx broaden the following]
Figure 10.2.2-2 describes the source of the disease known as recruitment. The lower portion of the figure shows a simplified circuit diagram at the output of the sensory neurons; the distribution amplifier on the left, the orthodromic neuron on the right, and the intervening synapse acting as a virtual diode. To maintain a signal path through the synapse, it is absolutely mandatory that the emitter to base potential, E_{EB}, be positive at all times. Since the neural circuitry is direct coupled and uses distributed power supplies, the impedance between the local grounds, R_G, is critical to the operation of this interface.

The upper portion of the figure describes the output characteristic of the distribution amplifier (upper left) under two bias conditions, and the transfer characteristic of the orthodromic neuron (upper right) also under two conditions. The voltage scales in the middle of the upper portion are defined as relative. The absolute potentials of these two elements are critically important to the operation of the overall circuit.

The convention of using a sine wave is followed in this figure as it allows easier understanding of any distortions involved. Except at stimulus frequencies well below the integration frequency of the neuron, the actual signal is the envelope of the stimulus.

The waveform of example #1 represents the nominal operating condition for the system. The current through the collector of the distribution amplifier is shown as 15 pA. The output at upper right is a good facsimile of the input at lower left. On the other hand, example #2 shows two potential problems. First, at the nominal current of 33 pA, the transfer function exhibits a nominal slope of 1.0 instead of the typical 0.3. This results in a considerably larger output voltage amplitude at the collector than otherwise expected (a hallmark of the disease called recruitment). As a result, part of the signal voltage waveform is outside of the acceptance range of the transfer function of the orthodromic neuron. The result is considerable distortion in the output waveform at the collector of this neuron.

To illustrate the potential for various conditions, the upper right portion can be moved up or down relative to the upper left portion. The distortion in example #1 can be alleviated by moving the upper right figure down relative to the upper left portion. This entails a change in the absolute potential between the two halves of the figure. As a result, the example #2 waveform will not be as distorted. However, its output at the collector of the orthodromic neuron will be considerably larger than in example #1 (the principle symptom of the disease).

The lower portion of the figure shows several potential sources of the distortion and or excess amplification associated with the diseased condition of example #1 above. First, the potential source, E_B, and its associated diode play a major role in determining the quiescent current through the distribution amplifier. While the potential E_B is set largely by genetics, the impedance of the diode is a function of the vascular supply system. Its impedance is susceptible to the effects of aging. Second as noted above, a significant change in the absolute value of the quiescent potential of the distribution amplifier can cause the synapse, acting as a virtual diode, to become non-conductive over part or all of the operating cycle associated with the output of the distribution amplifier. This is particularly true if the impedance between the local ground of the synapse and the local ground of the distribution amplifier are at different potentials. The result is significant distortion or cutoff of the signal. Third, the quiescent potential at the emitter (neurite) terminal of the orthodromic neuron can be outside the operating range of its transfer function. This condition can also contribute to significant distortion. The experimental literature does not provide information as to which of these potential problems are actually encountered in the disease.

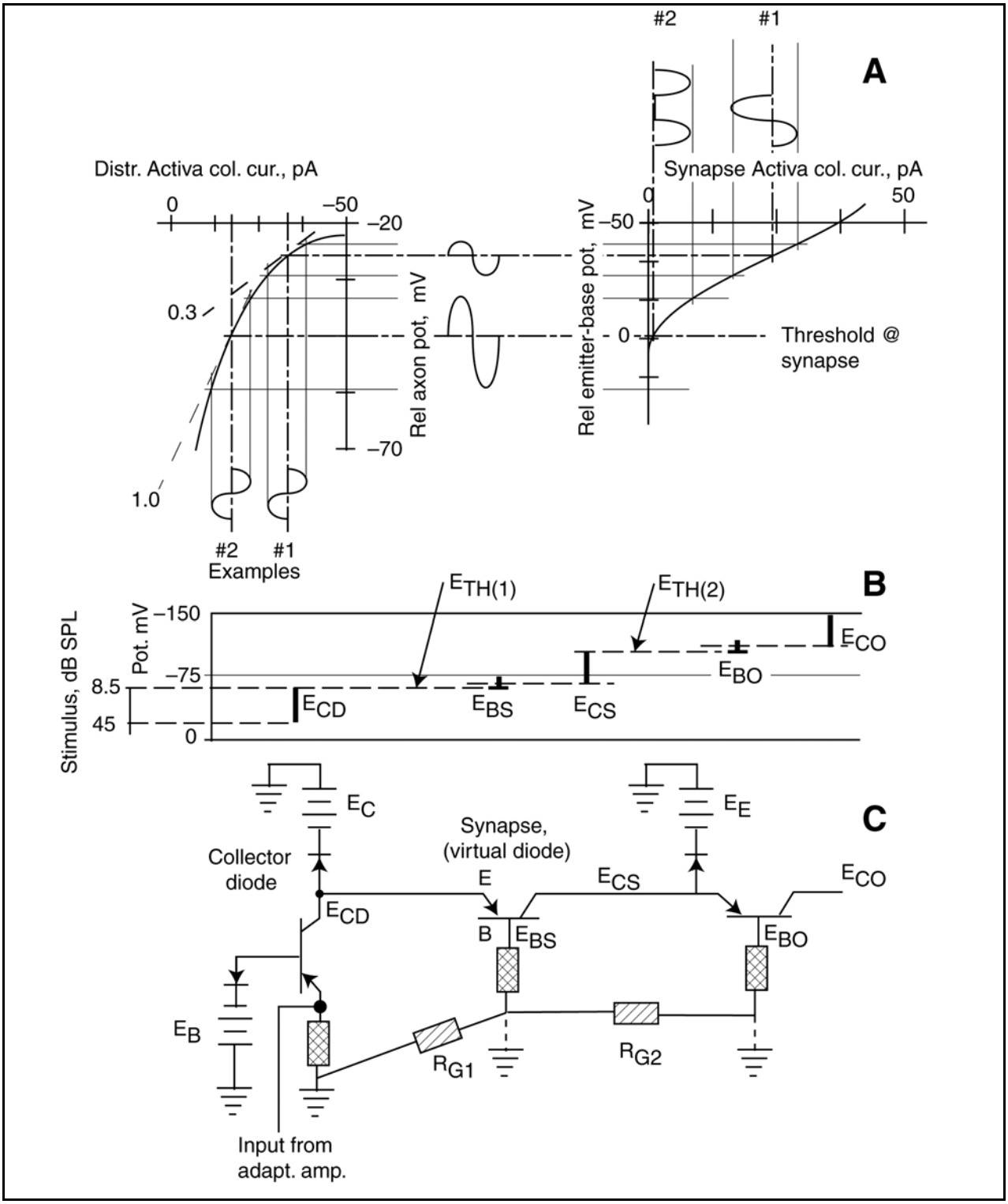

Figure 10.2.2-2 The source of the disease described as recruitment. Bottom; simplified circuit diagram at the distribution amplifier of the sensory neurons. Note the potential impedance, R_G, between the base impedance of the synapse and the local ground of the distribution amplifier. Upper left; output characteristic of the distribution amplifier. Upper right; input characteristic of the orthodromic neuron. See text.

10.3 Sensorineural disorders related to frequency

10.4 Sensorineural disorders related to pharmacology

Damage to the hearing system based on inappropriate pharmacology is difficult to quantify. Several families of widely used pharmaceuticals are known to adversely affect the hearing system when used at high dosage. However, the definition of high is unclear. The family of aminoglycosides is of particular concern[38]. These are the -mycins of pharmacology. Their value in medicine is great. However, the ototoxicity of streptomycin was first reported in 1945, shortly after its development. Neomycin has a well documented and marked ototoxicity potential.

The ototoxicity potential of aspirin has been recognized since its introduction in 1899.

10.5 Selected results of surgical intervention

10.5.1 Effect of total commissurotomy EMPTY

Sidtis has provided a summary of the effect of surgical severing of the corpus callosum on human hearing[39]. The results appear very similar to the effect on vision.

The discussion in Sidtis is based on the conventional wisdom; that all of the interconnections between the two hemispheres are via the corpus callosum. As shown in Section xxx and in "Processes In Biological Vision," this is not the case, especially for the more important information processed probably exclusively in the diencephalon. This material is shared between the two halves of the pulvinar via the corpus principia. Such neural pathways are not affected by a commissurotomy. Sidtis did not address the role of the diencephalon and the corpus principia in hearing.

The Sidtis material is well organized. As a result, he uncovered a variety of peculiarities not explained satisfactorily by the conventional wisdom. As he noted, "With respect to auditory function, unilateral temporal lobe damage does not result in deafness in one ear nor does it result in sensory deficits analogous to the scotomas or hemianopsias than may be observe with lesions to the primary geniculo-striatal visual system." The first point emphasizes the secondary role of the temporal lobes in extracting information related to hearing. The role of these lobes is focused more on the quality and the coloring of the auditory stimulus than on the abstract information it carries. The extraction of the more important information is carried out within the diencephalon. The second point is probably stated too broadly. While a scotoma of either lateral geniculate nucleus, LGN, typically affects either the left or right half of the peripheral field of vision, it does not cause major loss of vision relative to the foveola. As in hearing, the more critical portion of visual information extraction is carried out within the pulvinar and perigeniculate nuclei, PGN, of the diencephalon. Area 17 of the cerebral cortex is usually associated with his reference to the striated area. This area plays a role analogous to the role the temporal lobes play in hearing. It is now documented that total functional loss of this so-called primary visual area of the cortex does not result in total blindness. Such a loss is functionally similar to that documented by Sidtis for the temporal lobes and described above.

Quoting Sidtis again, "After commissurotomy, verbal report of stimuli presented monaurally to either ear is typically normal." He describes the more complex effects when the stimuli are presented to subjects dichotically. At that point, the term paradoxical enters his discussion. This suggests a lack of an adequate model underlying the remainder of his discussion. In fact, he introduces a series of rhetorical questions as subject headings. He says, "Unfortunately there is little or no direct data on the variability of callosal auditory function in humans." The paper concludes with some excellent data on psychophysical

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[38]Lee, K. (1989) Textbook of Otolaryngology and Head and Neck Surgery NY: Elsevier pp 106-109

[39]Sidtis, J. (1988) Dichotic listening after commissurotomy *In* Hugdahl, K. *ed.* Handbook of Dichotic Listening: Theory, Methods and Research NY: Wiley pp161-184

experiments using complex speech as a stimulus.

10.6 Auditory prosthetic devices up to 2009

White, et. al. {analog multichannel cochlear prosthetics }

Loeb 85

[xxx page 168-169 of Roederer on potential uses of a prosthesis containing a programmable random memory capable of sending signals down a particular lead to the auditory nerve].

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The auditory community has investigated prosthetic devices focused on three discrete points of incursion into the system;

1. Augmentation of the overall system through amplification and signal shaping prior to stage 0 (external hearing aids).

2. Introduction of electrically simulated signals prior to stage 2 using cochlear implants

3. Introduction of electrically simulated signals following stage 2 and before stage 4 using electrically simulated signals applied to the brainstem.

All of these approaches have suffered from a lack of an adequate schematic and circuit model of the auditory modality. This fact is epitomized by the difficulty of compensating for the effect known as "recruitment" that is actually the natural logarithmic compression at the axon pedicle of the sensory neurons.

While spectacular results have been obtained with implantable audio prostheses recently from the patients perspective, it is still a very early day in the field. The devices take advantage of the easy physical access to the scala tympani via the round window. The procedure is to introduce a long thin probe with electrical terminals placed periodically along its length. These terminals are used to apply a high electrical pulse between the individual terminals and a common ground, or between individual pairs of terminals. The pulses are meant to shock the system and cause some response in the optic nerve.

10.6.1 Prostheses introduced at stages 0 & 1 EMPTY

Using electrical augmentation to hearing via fully functional hair cells is described as electrophonic hearing by Hartmann and Kral (page224).

DiGiovanni has provided a catalog of virtually all commercially available stage 0 prostheses[40].

10.6.2 The pre-stage 2 cochlear implant prosthesis

While a variety of cochlear implants have been rushed to market to satisfy a considerable patient demand for the devices, the field is only beginning to emerge from a long period of exploratory research. As of 2009, not a single cochlear implant has been designed based on an adequate knowledge of the auditory modality to insure operational success.

10.6.2.1 An annotated history of cochlear prosthetic devices

The New York Academy of Science provided a special edition of their journal on this subject in 1983[41].

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[40]DiGiovanni, J. (2008) Hearing Aid Handbook: 2008-2009. Clifton Park, NY: Delmar

[41]- - - - (1983) Cochlear Prostheses: An International Symposium. *Ann New York Acad Sci* vol 405

34 Biological Hearing

Lee provides a useful chronology of the early work in implanted prostheses[42].

Miller and Spelman edited a text on cochlear implants circa. 1990. However, it included very little histology or cytology and is largely out of date[43]. It does include some useful histograms of neural signals in the auditory nerve and cochlear nucleus. Clark et al. provided an broad overview of the state of the art in audio prosthetics[44]. Zeng et al have provided a more recent overview of the state of the art in audio prosthetics[45]. The discussion in Chapter 6 by Hartmann and Kral is very useful.

Clark et al. discuss a variety of features of the cochlea important to the surgical community (page 78) and the electrical community (page 87). One to five volt pulses are typically used although the neural system typically employs voltages below 50 mV. As discussed in Clark et al., The driver developing these pulses is frequently of the current driver type to assure uniform operation in the face of variable interface impedance. **Figure 10.6.2-1** shows the nominal equivalent circuit of the tissue interface. Experience suggests the interface is largely ohmic.

Clark authored a followup volume in 2003[46]. It provides a very comprehensive summary of the entire clinical field of implant development. It provides considerable discussion of the neurological elements of hearing. However, it lacks a detailed analysis of the auditory modality as a neurological system. The summary of the original fundamental objections and the many solutions developed is very good (pages xxxiii-xxxviii). His reproduction of a figure from Shepherd et al on page 505 shows recent progress in placing the electrode array more precisely within the scala tympani. The importance of placing the electrode near the peripheral processes of the ganglion cells is apparent[47]. However, the figure shows the lack of placement near the actual Organ of Corti. Many details of current prosthetic units are also included.

Waltzman et al. authored a volume in 2006. [xxx add comments after reviewing]

607/2 and 607/55, 607/56 & 607/57 are the appropriate class/subclass numbers for patents issued by the United States Patent Office. Patent 7,333,858 applies to ramped pulses. 7,321,797 applies to an incremental feed processor. 7,317,944 applies to xxx. The early patent 4,495,384 is assigned to W. F. House.

Current US FDA regulations require the prosthetic stimulus contain no DC component to prevent chemical deposition at the interface and possible tissue damage. Thus, the pulse shape shown is for illustration only and can be used in practice only if balanced around a zero current level not shown.

Brummer and Turner studied the electrode tissue interface in 1977[48]. They recognized the charge buildup near the surface of the electrodes as forming a double layer of . This was the origin of the term double layer capacitor in implant discussions. They also discussed the real electrode surface area for a cylindrical electrode versus the effective surface area. "Usually 1.0 geometric cm^2 of a shiny Pt electrode is equivalent to 1.4 real cm^2."

Huang et al. have provided recent data on the electrode tissue interface under a variety of conditions[49].

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[42]Lee, K. (1989) Textbook of Otolaryngology and Head and Neck Surgery NY: Elsevier pp 176-183

[43]Miller, J. & Spelman, F. (1990) Cochlear Implants: Models of the Electrically Stimulated Ear N:Y: Springer

[44]Clark, G. Tong, Y. & Patrick, J. *eds*. (1990) Cochlear Prostheses. NY: Churchill Livingstone

[45]Zeng, F-G. Popper, A. & Fay, R. *eds*. (2004) Cochlear Implants: Auditory prostheses and electric hearing. NY: Springer-Verlag

[46]Clark, G. (2003) Cochlear Implants: Fundamentals and Applications. NY: Springer

[47]Shepherd, R. Hatsushika S. Clark G. (1993) Electrical stimulation of the auditory nerve: the effect of electrode position on neural excitation *Hearing Res* vol 66, pp 108-120

[48]Brummer, S. & Turner, M. (1977) Electrical stimulation with Pt electrodes: I. A method for determination of "real" electrode areas *IEEE Trans Biomed Eng* vol 24, pp 436-439

[49]Huang, C. Shepherd, R. Seligman, P. & Clark, G. (1998) Reduction in excitability of the auditory nerve following acute electrical stimulation at high stimulus rates: III. Capacitive versus non-capacitive coupling of the stimulating electrodes *Hear Res* vol 116(1-2), pp 55-64

They quantify the damage inherent in DC stimulation.

This network represents a volumetric impedance. Charge injected into the tissue has been found to spread significantly according to Black and Clark[50]. Their studies were of a simple resistor-only equivalent network that did not represent the heterogenous nature of the cochlear partition well. More recent data appears in the special edition of Ear & Hearing, vol 23(1), 2002. The data appears clear that the inserted electrode array should hug the surface of the basilar membrane immediately below Corti's tunnel. to be close to the first Nodes of Ranvier or other ganglion neuron components. This presumes it is the first Node of Ranvier is of primary efficacy because of its unmyelinated pre-nodal axon. Figures 2.3.4–3 through 2.3.4–5 detail this area. Based on the theory of this work, the array should be as close as possible to the first Nodes, individually located directly below both the IHC and the OHC, and always before the habenula perforata (the area labeled "d" in the figure of Shepherd et al.). Placing the array between the habenula perforata and the area closest to the modiolus reduces the effectiveness of the stimulation. This is shown for the "bipolar" configuration of Shepherd et al. where a current nearly an order of magnitude less is required in the area labeled "dendritic" versus other areas in the scala tympani to cause an evoked brainstem response.

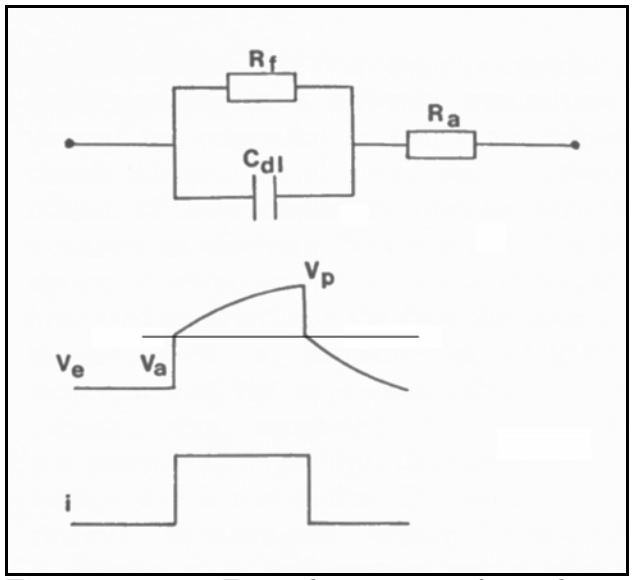

Figure 10.6.2-1 Equivalent circuit of prosthesis-tissue interface. The electrode voltage is shown for a square current pulse, i. V_a represents dissipation across the resistor R_a. Modified from Clark et al., 1990.

The challenge of transmitting the sensed signals through the skin to the implanted circuitry was a problem in the early days but has now been largely resolved using inductive techniques.

Several signal generation strategies have been developed and implemented (see Wilson in Zeng, et. al. for a review). No significant attempts to emulate the encoded pulse streams normally generated by ganglion neurons has been reported in the literature to date.

Clark reviewed, "Feature extraction: for and against" in 1990 (page 54). He compared the speech feature extraction and presentation approach (using vocoder principles) versus the maximum signal information approach (using multiple band pass filters). The features involved were typically the first formant (F1) and second formant (F2) with the option of including an estimate of the fundamental frequency of the speaker (F0).

Clark's assertion related to the feature extraction approach, "Each selected feature is then separately coded in a way that is known to be robust in transmission through the neural interface and into the perceptual system of the implantee." is open to considerable argument. In fact, the neural interface using amplitude modulated pulses was entirely non-functional. Later investigators accepted the fact the feature extraction approach failed. The end-to-end emulation of the implant and the implantee presented by Blamey et al in 1984 in no way represented a realistic situation as suggested by Clark (page 58). As Wilson notes in Zeng, et. al. (2004, page 40), "All present processing strategies for cochlear implants use a "filter-bank" or "waveform" approach. Explicit extraction and representation of specific features of speech was abandoned in the early 1990s."

Gantz, Turner & Gfeller have addressed the virtue of implanting an electrode array in those subjects with

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[50]Black, R. & Clark, G. (1980) Differential electrical excitation of the auditory nerve *J Acoust Soc Am* vol 67(3), pp 868-874

some residual hearing[51]. Writing in 2006, they note the limited spectral resolution achieved with current electrode arrays. They developed a short electrode array that was initially placed in the basal turn to augment the subjects residual low frequency hearing. A complaint was the perceived augmentation was too high pitched and even unpleasant. Subsequent testing placed a 6-element, 10 mm long augmentation array farther into the cochlear partition. The authors provide their views concerning the performance achieved. They stress the need of surgeons to preserve any residual hearing available to the subject when providing an augmented capability.

Gantz, Turner & Gfeller appear to have demonstrated the ability to excite the higher frequency (basal) OHC neurons as well as the basal IHC. Their work also suggests it is important to excite the basal IHC to achieve good temporal performance while it is more important to excite the mid-range OHC to achieve good mid frequency (1500-6000 Hz) tonal performance[52].

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Edwards has recently provided another review of the exploited technology approaches[53]. He focuses on the design of the various applications rather than discussing the hearing mechanism or its failings that are the subject of auditory loss compensation. Edwards noted, "A more important factor has been the absence of a scientific consensus on precisely what a hearing aid should do to properly compensate for a hearing loss."

Graeme Clark has provided a discussion of cochlear implants in the same volume[54]. This discussion also avoids discussing the physiology of the Organ of Corti and concentrates on the circuitry chosen for the loss compensation device. No discussion of the geometry of the electrode bundle inserted into the Scala Tympani was provided.

As a result of the above strategies, the subject tends to perceive sound as if it were generated by non-voiced utterances (from shocking the temporal IHC channels) accompanied by tonal artifacts (from shocking the tonal OHC channels). It is not clear whether the artificial signals stimulate the sensory neurons or the ganglion neurons of the cochlear nerve. The very limited dynamic range encountered in practice (6-12 dB, Clark, et. al., pg 107) suggests the shock is effective in generating signals beyond the point of adaptation. It appears the shock primarily affects the ganglion neurons or one of the Nodes of Ranvier associated with those ganglion neurons.

The current used in the early Clark work resulted in charge densities of up to 25.7 $\mu C/cm^2$ geom. per phase (page 86). Several recent units capable of generating up to 914 $\mu C/cm^2$ geom. per phase (Clark, 2003, page 490). More recent investigations have used bipolar excitation with the charge limited to xxx in accordance with FDA guidelines.

A major problem of cochlear implants up to this time has been their inability to pass the temporal fine structure of acoustic waves needed for speech interpretation[55]. This work demonstrates that proper stimulation to achieve this result requires precise electrode array placement and specific electrical stimulation waveforms.

Drennan and Rubenstein proposed three criteria for passing the fine structure in acoustic speech via an implant satisfactorily based on only their unstated conceptual model (page 44). The first is to use pulse excitation at greater than 2000 pps. This work stresses the fact that exciting the neural system at pulse rates greater than about 1000 pps is of negligible value. The normal action potential interval, action potential duration plus the following refractory period, leaves the regeneration of action potentials at rates

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[51]Gantz, B. Turner, C. & Gfeller, K. (2006) Acoustical and electrical speech processing *In* Waltzman, S. & Roland, J. *eds*. Cochlear Implants, 2nd Ed.. NY: Thieme Chapter 19

[52]Turner, C. Gantz, B. Vidal, C. Behrens, A. & Henry, B. (2004) Speech recognition in noise for coclear implant listeneres: beefits of residual acoust hearing *J Acoust Soc Am* vol 115, pp 1729-1735

[53]Edwards, B. (2004) Hearing aids and hearing impairment *In* Greenberg, S. Ainsworth, W et al. *eds*. Speech Processing in the Auditory System. NY: Springer Chap 7

[54]Clark, C. (2004) Cochlear Implants *In* Greenberg, S. Ainsworth, W et al. *eds*. Speech Processing in the Auditory System. NY: Springer Chap 8

[55]Drennan, W. & Rubinstein, J. (2006) Sound processors in cochlear implants *In* Waltzman, S. & Roland, J. *eds*. Cochlear Implants, 2nd Ed.. NY: Thieme page 44

significantly higher than 1000 pps very inefficient and undependable. Hartmann & Kral suggest the limit is closer to 600 pps[56]. The Drennan and Rubenstein discussion also suggests stochastic pulse firing within the neural system is useful. This work stresses the deterministic character of all (information carrying) neural pulses. The only action potentials in the neural system that *appear* to be non-deterministic are those due to long step excitation applied to the low frequency encoding (ganglion) neurons. The result can be multiple pulses indicating the duration of the excitation, not its intensity (See **Section xxx**).

10.6.2.2 The use of formant filters in previous implants

Speech processors were explored during the 1970's and 1980's that extracted formants from the applied acoustic signals and then used signals based on those formants to drive multi-electrode arrays implanted in the cochlear partition. The features involved were typically the first formant (F1) and second formant (F2) of vocal signals with the option of including an estimate of the fundamental frequency of the speaker (F0). F1 and F2 were the most obvious spectral features of the signals within the lower frequencies displayed in a spectrogram. Little effort was made to extract the variation in frequency of the individual formant versus time. The formant extractors were typically based on fixed filter characteristics.

As noted above, efforts to develop formant based speech processors ahead of an implanted electrode array dissipated at the end of the 1980's. The reason was fundamental. The human auditory modality incorporates a very sophisticated formant extraction mechanism as described in **Section 9.xxx**. The use of a crude formant extractor ahead of a frequency and temporally indiscriminate electrode array, based primarily on position within the cochlear partition, led to considerable confusion within the hearing modality. The signals presented to the natural feature extraction correlator of the CNS did not resemble the signals it was evolved over many millennia to process.

As a minimum, an auxiliary feature extractor, used to augment the natural feature extractor, must maintain the octave-based harmonic relationships of the original signal. It must also maintain the relative delays associated with those harmonic relationships when reintroduced into the neural signaling system. Finally, the reintroduced signals must excite the appropriate frequency-based sensory channels based on the signals frequency content. Finally, the auxiliary feature extractor must be able to reproduce the frequency variations with time of the original formants. This requirement calls for the use of frequency tracking formant extractors as active filters in place of fixed bandwidth filters.

Otherwise the signals from the man-made feature extractor must be introduced at a point in the hearing modality after the natural feature extractor of the perigeniculate nucleus (PGN). This will remain an unrewarding clinical approach for a long time, at least until we learn to understand the detailed output of the natural feature extractor.

10.6.2.3 Signal compression in the analog circuitry of previous implants

Acoustic signal compression has been widely used in cochlear implants based on a fundamentally erroneous assumption.

The fundamental assumption has been that the neural signals of the auditory nerve are some form of amplitude modulated representations of individual portions of the applied acoustic signals. In such modulation, the frequency of the signal is analogous to the frequency of the stimulus and the amplitude of the signal is analogous to the amplitude of the stimulus. This is incorrect. The neural system employs a form of pulse position modulation where the amplitude of the pulses carries no information and the pulse interval is proportional to the amplitude of the stimulant.

No information about the frequency of the stimulus is carried within the neural signaling channels. Frequency information is related to the place of the sensory neuron along the cochlear partition and the equivalent place of the delivery axon in the auditory correlator portion of the audio portion of the perigeniculate nucleus.

The fact that the experimentally verified susceptibility of the Nodes of Ranvier to amplitude variations (Section 10.xxx) is very limited provides two important pieces of information. First, it demonstrates the

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[56]Hartmann, R. & Kral, A. (2004) Central responses to electrical stimulation *In* Zeng, F-G. Popper, A. & Fay, R. *eds*. (2004) Cochlear Implants: Auditory prostheses and electric hearing. NY: Springer-Verlag page 236

stimulation provided by laboratory probing and by early implants is affecting the signal regeneration Nodes of Ranvier (subscripts labeled O,2 or I,2 and higher in [Figure 10.xxx]) and not the signal encoding Nodes of Ranvier (subscripts labeled O,1 or I,1 in [Figure 10.xxx]). If the stimulation was affecting the signal encoding NoR, the expected dynamic range of the circuit would be as large as the normal stimulus dynamic range (at least 30 dB) at the pedicle of the stage 1 sensory neurons. Hartmann and Kral define this dynamic range as mostly between 20 to 60 dB[57].

Second, it shows the signal regeneration Nodes of Ranvier are very efficient in their operation. They require only a small change in amplitude above their threshold value (less than a factor of two, 6 dB) to generate a full amplitude action potential that is temporally correlated with the driving signal.

A compressed analog (CA) strategy is still available in many current commercial speech processors designed to support implants (in the form of one of multiple software routines). The use of CA strategies by patients has been limited in day-to-day usage.

10.6.2.4 The record of a cochlear implant prosthesis user

Chorost published a comprehensive book in 2005 on his experiences as an implant recipient[58]. One of his initial statements is striking and confirms the operating methodology described above. He noted that during his first session designed to interface the prosthesis to his neural system, he could clearly interpret the ticking of a clock across the room. At the same time, the sounds and sentences presented by the audiologist were completely un-interpretable. The prosthesis was obviously exciting the neurons associated with the temporal (LOC) channel of hearing. It may or may not have been exciting neurons of the tonal (MOC) channels. In his book, Mr. Chorost says he can now discriminate between tones with a frequency spacing on the order of 30 Hz at 2,000 Hz, down from 70 Hz initially. Mr Chorost's one-hour oral presentation, as recorded by C-SPAN, is available for purchase or viewing over the internet[59]. His website is www.rebuilt-thebook.com

10.6.3 Prostheses introduced beyond stage 2 EMPTY

[xxx see chapter 20 in Waltzman & Roland]

xxx Kuchta et. al. {multichannel brainstem implant.}

St. Clair, Golfinos & Roland have reported on efforts to provide a "brainstem implant" for those with severe bilateral damage to the auditory nerve[60]. Their goal was to stimulate the cochlear nucleus (which is part of the brainstem). Their efforts employed an electrode structure that was laid upon the exterior of the cochlear nucleus. It excited a poorly defined portion of the cochlear nucleus. The results were of limited value but showed feasibility of stimulation. Several undesireable side effects were noted with this approach.

Their discussion of the steps needed to achieve a degree of tonotopic stimulation in the future is useful. What they do not discuss is the multiple tonotopically and tempotopically oriented regions of the cochlear nucleus. These regions are tailored to support different feature extraction engines within the CNS as illustrated in **Section 2.4.2**. Thus, to avoid causing vertigo, anxiety and similar problems, it is important that only the appropriate neurons be stimulated. These are the neurons associated with the MOC path and the awareness mode of hearing. The neurons associated with the alarm and source location tasks should be avoided.

Conventional speech processors were modified for this application (although few details were provided). They also address the subject of non-auditory perceptions as a result of cochlear nucleus stimulation,

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[57]Hartmann, R. & Kral, A. (2004) Central responses to electrical stimulation *In* Zeng, F-G. Popper, A. & Fay, R. *eds*. (2004) Cochlear Implants: Auditory prostheses and electric hearing. NY: Springer-Verlag page 219

[58]Chorost, M. (2005) Rebuilt : How Becoming Part Computer Made Me More Human. NY: Houghton-Mifflin

[59]www.booktv.org Search for material by Chorost.

[60]St. Clair, E. Golfinos, J. & Roland J. (2006) Auditory brainstem implants *In* Waltzman, S. & Roland, J. *eds*. Cochlear Implants. NY: Thieme Chapter 20

including dizziness and other interferences with the vestibular signaling channels. On the order of 200 patients have been treated with one or more of a variety of brainstem implants as of the article publication date.

10.7 Proposed future generation pre-stage 2 cochlear prostheses EMPTY

10.7.1 The state of the prosthesis art based on this work

The literature as of 2009 reports on efforts to build implantable prosthetic hearing devices designed without detailed knowledge of how to interface to the neural system of the hearing modality. As a result, the implantable devices have achieved a minimal level of performance primarily as a supplement to lip reading. While recipients are overjoyed by their ability to hear anything after implantation, their ability to hear in the broader sense is not significantly increased.

The failure described above is highlighted by two events. First, the abandonment of efforts to use electronic signal processing to optimize the interface with the neural system. Second the failure of multi-electrode interfaces spaced along the cochlear partition to achieve any significant perceived level of tonal hearing quality.

These failures are primarily due to four facts.
1. The researchers have failed to understand the transduction mechanism of the hearing modality. In particular,

A. They have failed to understand that the place-frequency relationship does not mean that a certain acoustic frequency is processed as an equivalent electrical frequency beginning at the same place. The "place" is mapped to the brain by anatomical computation. The only signal from that place represents the amplitude of the envelope of the acoustic signal measured at that point.

B. They have failed to recognize that there are two distinct groups of sensory neurons in hearing, the IHC (which are temporally selective) and the OHC (which are tonally selective).

2. They have failed to understand that no frequencies greater than 600-1000 Hz are passed from the sensory (stage 1) neurons to the signal projection (stage 3) neurons. The sensory neurons incorporate a low pass filter with a critical frequency of 600 Hz.

3. They have failed to appreciate that the maximum acoustic frequency that can be represented by the stage 3 signal projection neurons is less than 600 Hz (in accordance with Shannon's Sampling Theorem).

4. The scale of the current man-made electrode arrays is not compatible with the scale of the auditory sensory mechanisms. Clark (page 508) shows a free-fitting silastic electrode carrier inserted into the scala tympani of a cat. The carrier fills the scala while the individual neurons are invisible (by at least a factor of ten) at the scale of the figure.

The diameters of the electrode arrays are typically 0.3 to 0.6 mm while the diameter of the neurons they are attempting to influence are typically 0.3 to 0.6 microns. As a result, the ratio of the size of the electrodes to the size of the neurons (1000:1) is like the size of a cottage to a key on a computer keyboard (an order of magnitude worse than wearing boxing gloves while typing on a keyboard).

With measured fields with attenuation parameters measured in millimeters, exposing one thousand or more neurons to a single electrical pulse is a brute force approach. This is especially true since the pulse exhibits a single temporal characteristic while attempting to excite neurons where temporal differences of 0.01 seconds are important. With an octave represented by a distance of nominally 2.3 mm along the partition, a single pulse can excite neural paths a full octave apart.

These represent formidable problems in prosthesis development. The ultimate goal will require several major development cycles to approach a true prosthesis offering significant restoration of the hearing function. The community must adopt an entirely different model of the hearing modality if it hopes to aid the profoundly deaf through the use of implantable prosthetic devices. Similarly, the electrode arrays must move from miniature wires grouped for insertion into the scala tympani, to microcircuits on substrates

capable of positioning much closer to the tunnel of Corti. Closer positioning reduces the electrical field strength, and hence charge required per unit area, to achieve stimulation of the desired neurons[61]. Individual wires of 25 micron diameter are used currently to provide the necessary flexibility in the implant array. Printed wires on the order of five microns are easily achievable with current technology.

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Several researchers have prepared lists of limitation on current cochlear implants. Most are poorly conceived. The major limitation of cochlear implants listed by Drennan and Rubinstein in 2006 is erroneous[62].

The current state of the art in implantable cochlear prosthetic devices is being limited by several false assumptions.

1. The assumption that the individual neural channel must propagate both a frequency and amplitude parameter to represent the signal applied to it. The brain relates the place of origin of the neural signal by means other than the information within the signal (just like a telephone mainframe identifies an incoming telephone pair by a physical label, not by the frequency and amplitude of the message content (or even an assigned telephone number).

> The information carried by the auditory neuron, like all pulse neuron signals is propagated by pulse-to-pulse interval (PPI) modulation. The greater the stimulus amplitude, the shorter the interval between pulses. Frequency modulation is a form of phase modulation, that is in turn a form of pulse-to-pulse interval modulation.

2. The assumption that the dynamic range available via implants is highly limited. This assumption is based on the assumption that the amplitude of a stimulus is the variable parameter. In fact, it is the pulse frequency of the action potentials that is the variable representing sound amplitude. This frequency varies between a low of about 10 pps and 500 pps. The system is pliable, it is the ratio between the two preceding pulse rates and not their absolute values that are important. The signal to noise in the channel is about 30 dB or better and the dynamic range is about 50:1 on a logarithmic increment scale. All of the channel is available to a properly designed prosthetic device.

> There are three distinct types of stage 3 circuits, the analog to pulse converters formed either within the ganglion neurons (or in their first Node of Ranvier in the case of hearing), the pulse repeater circuits formed by all subsequent Nodes of Ranvier, and the pulse to analog converters described as stellate neurons in this work. The implant designer must determine whether he intends to excite an analog to pulse conversion circuit or a pulse repeater circuit. The signaling requirements are grossly different. Past efforts have focused on exciting pulse repeater circuits. These circuits exhibit a very narrow amplitude dynamic range (defined by their threshold sensitivity). Their mode of signaling is pulse frequency variation, not pulse amplitude variation.

3. The lack of ability of cochlear implants to pass the temporal fine structure of acoustic waves to the auditory nerve is another limitation. In fact, the cochlear implant is able to provide more fine structure than the auditory nerve can process. However, it must do that with the proper phasing. The challenge here is to limit the longitudinal range of any stimulation applied by the implant.

10.7.1.1 Parametric limits on stimulation

A scala tympani electrode has to have a geometric shape and mechanical properties that enable the surgeon to place contacts as close to excitable nerve structures as possible and avoid insults to the basilar partition or to the bony spiral lamina.

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[61]Shepherd, R. Hatsushika, S. Clark G. (1993) Electrical stimulation of the auditory nerve, the effect of electrode position on neural excitation *Hear Res* vol 66, pp 108-120

[62]Drennan, W. & Rubinstein, J. (2006) Sound processors in cochlear implants *In* Waltzman, S. & Roland, J. *eds*. Cochlear Implants. NY: Thieme page 43

Clopton et al. have provided a summary of the literature circa 1983 that includes a variety of useful citations[63]. The following is from Brummer & Turner.

"An absolute limit on stimulus encoding is the charge density at which hydrogen is evolved at the surface of the electrode. This has been estimated at 300 pC/cm^2 per phase for platinum electrodes in simulated cerebrospinal fluid at 37 Celsius. This limit holds for the real surface area, which invariably exceeds the geometric surface area. In our laboratory, spheres flamed at the ends of 1mil platinum-iridium wires show real surface areas of approximately 0.001 cm^2 or greater. The gassing limit estimated for this surface area is 6.7 pA rms with 10-Hz stimulation increasing at 6 dB/octave with frequency."

Brummer, Robblee and Hambrecht have provided a good background on electrode selection criteria circa 1983[64]. Their data is all from non-biological solutions (1 N of $HClO_4$) rather than actual tissue. They use a largely conceptual model of the neuron. They also assume it is the *current* passed through neural tissue that affects the generation of action potentials by stage 3 neurons. This work proposes the generation of action potentials at a Node of Ranvier by external means is controlled primarily by the *potential* at the poditic terminal of the Node (**Section xxx**). Brummer, Robblee and Hambrecht provide an annotate generic voltage characteristic for their electrode referenced to a reversible hydrogen electrode (RHE).

Hochmair et al. have provided a good overview of molded electrode array design, stimulation performance and surgical performance[65].

White et al. have reported on a practical polyimide based flexible electrode leading to the more sophisticated next generation design using smaller contact areas and described below[66].

10.7.2 A strategy for the next generation cochlear implant prosthesis

[xxx probably not looking far enough out]
The experience of the last 30 years should define the goals of a next generation cochlear implant prosthesis. The first requirement appears to be to provide a viable signal to the signal paths associated with the IHC neurons closest to the round window. This will provide at least an initial temporal signal to that system. The second, and probably lesser, requirement is to provide a viable signal to at least a few tonal channels associated with the OHC neurons. An ideal solution to this second requirement would be to provide a series of tonal signals covering at least one octave of the musical scale. An alternate solution would be to provide a set of viable signals to OHC spaced by an octave plus one note on the musical scale. This approach would require a longer, but more easily manufactured stimulus array.

The operating block diagrams, circuit diagrams and figures 4.5.1–2, 4.3.3–6, 4.5.4–2, 4.6.1–2 & 6.1.1–2 of this work should be reviewed before attempting to design the next generation cochlear implant prosthesis.
These figures define an entirely different design philosophy for the implant and its interface with the native neural circuits. A dual channel approach is optimum, exciting the appropriate sensory neurons of the temporal signaling subsystem and the appropriate sensory neurons of the tonal signaling subsystem.

Chapter 14 of Clark, 2003, should also be reviewed for thoughts about the future from other investigators with more direct laboratory experience.

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[63]Clopton, B. Spelman, F. Glass, I. Pfingst, B. Miller, J. Lawrence, P. & Dean, D. (1983) Neural encoding of electrical signals *Ann NY Acad Sci* vol 405, pp 146-158
[64]Brummer, S. Robblee, L. & Hambrecht, F. (1983) Criteria for selecting electrodes for electrical stimulation: Theoretical and practical considerations *Ann NY Acad Sci* vol 405, pp 159-172
[65]Hochmair-Desoyer, I. Hochmair, E. & Burian, K. (1983) Design and fabrication of multiwire scala tympani electrodes *Ann NY Acad Sci* vol 405, pp 173-182
[66]White, R. Roberts, L. Cotter, N. & Kwon, O-H (1983) Thin-film electrode fabrication techniques *Ann NY Acad Sci* vol 405, pp 183-190

It will be difficult to get any industrial institution to undertake an entirely new design based on this work. Therefore, it is prudent to define an ideal strategy for the next generation device and a less demanding compromise strategy.

10.7.2.1 Relevant parameters

All future strategies will be based on a variable frequency pulse pattern being employed at each electrode structure based on the measured sensitivity of the hearing system to pulse frequency within the auditory nerve. This approach offers more controllable performance than approaches based on amplitude-based approaches. **Figure 10.7.2-1** shows the performance of one of four electrodes of an array acquired by Tong[67]. Each data point represents the mean of 20 measurements. The bar represents one standard deviation each side of the mean. Only the circle data points are of interest. The multi-pulse data is at an excessive clock rate for reliable interpretation. The circle data shows a conventional roll off from a straight line represented by a single RC filter. The response is down to the 50% point at just under 500 pps. It is proposed the lowest test frequency could have been reduced to below 20 pps with the data retaining the linear characteristic shown by the sloping solid line. **Section xxx** describes the underlying circuit performance in more detail. [xxx stage 3 chapter, encoder.]

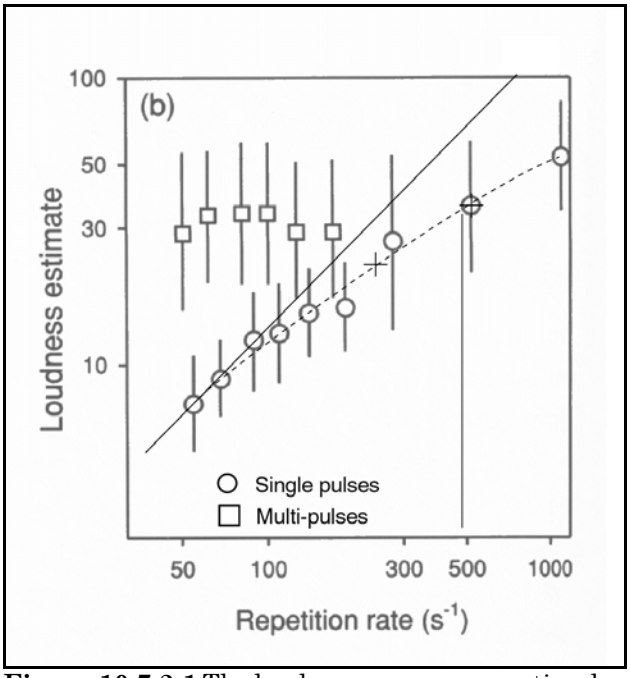

Figure 10.7.2-1 The loudness versus mean stimulus rate for fixed current levels. Circles; single biphasic pulses. Squares; biphasic 5-pulse groups with a constant interior pulse rate of 1000 pps. While not always noted in other reproductions, the circles are displaced to the right by half of one symbol width for clarity. Dashed line; roll off in sensitivity with half-amplitude point just below 500 pps. From Tong et al., 1983.

[67]Tong, Y. Blamey, P. Dowell, R. & Clark, G. (1983) Psychophysical studies evaluating the feasibility of a speech processing strategy for a multiple-channel cochlear implant *J Acoust Soc Am* vol 74, pp 73-80

The complete data set of Tong et al., including their 1982 paper[68], was difficult to interpret at the time it was acquired due to a lack of a realistic model of the auditory system. The data in the 1983 paper is less relevant to this discussion because it centered on efforts to extract speech features from a study using multiple pulse groups as a stimulus. The multiple pulse groups introduce additional variables that confuse the issue.

They provided simpler data in the 1982 paper. The data show the perceived responses of two cochlear implant patients were proportional to both the pulse frequency and the pulse amplitude of stimulation. **Figure 10.7.2-2**, repeated from **Section 2.3.4** with additional notation, provides a clearer interpretation of their results. Multi-element arrays were used and the elements could be and were activated separately. Biphase stimulation was used with the first pulse negative going relative to ground. Each phase was approximately 180 μs long. For an unknown reason, the investigators stressed their pulses had zero rise and fall times. A sharpness ranking study using time-invariant signals suggested that the hearing sensations produced by different electrodes varied from dull to sharp in an apical to basal direction in the scala tympani. A categorization study showed that the hearing sensations produced by two adjacent electrodes (1.5 mm apart) were rarely confused for a restricted range of time-invariant pulse rates.

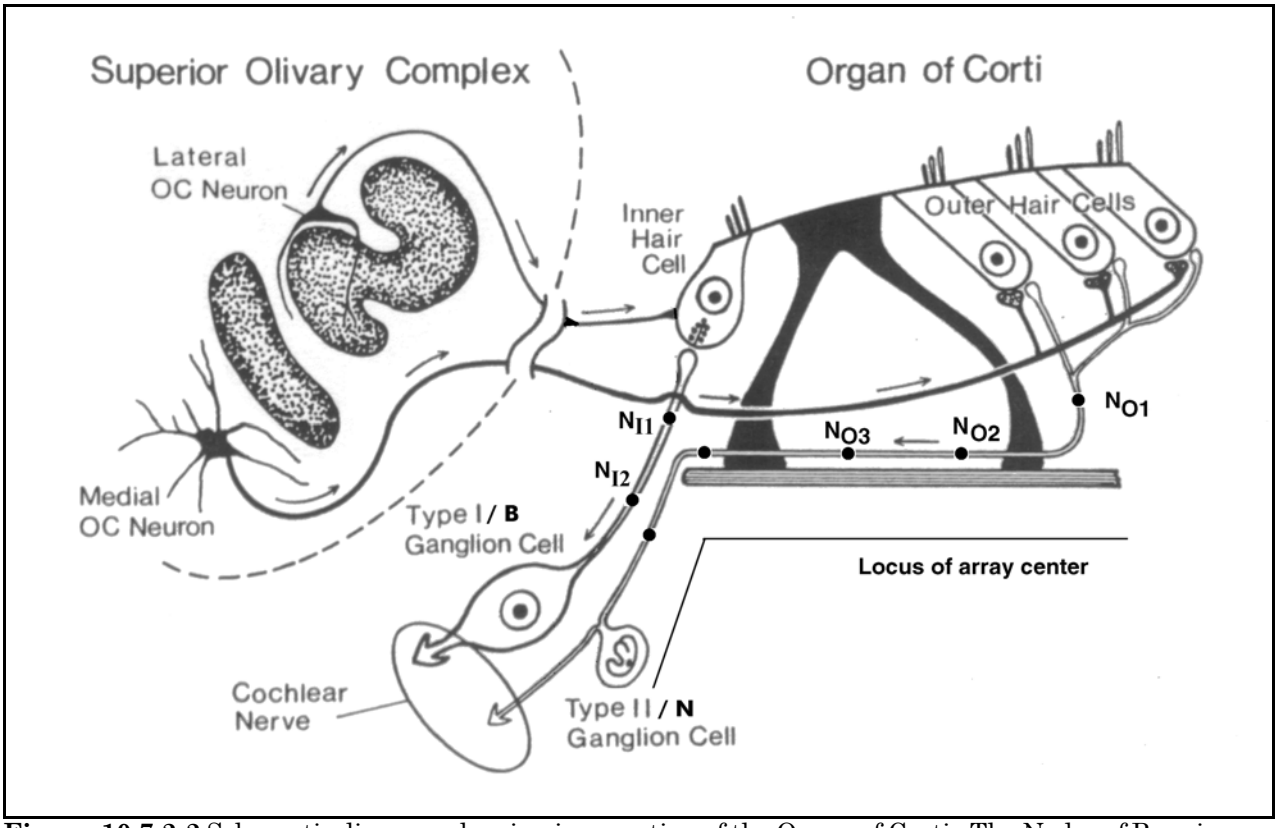

Figure 10.7.2-2 Schematic diagram showing innervation of the Organ of Corti. The Nodes of Ranvier are numbered along both the IHC and OHC paths. See text. Modified from Warr, et. al., 1986.

The precise location of the electrode array in the scala tympani was unknown. It center could be anywhere along the locus shown diagrammatically. It is now known the Nodes of Ranvier labeled N_{O1} in the narrow band channels emanating from OHC, and N_{I1} in the broadband channels emanating from

[68]Tong, Y. Clark, G. Blamey, P. Busby, P. & Dowell, R. (1982) Psychophysical studies for two multiple-channel cochlear implant patients *J Acoust Soc Am* vol 71, pp 153-160

IHC are stage 3 encoding nodes. They accept analog signals and generate pulse trains. The nodes labeled –2 and higher are pulse repeating nodes. They are designed to be receptive to pulse signals and regenerate those pulse signals at full amplitude for further propagation.

This schematic offers insight into the data of Tong et al. Excitation of any of the repeater nodes is easily accomplished by a pulse (or pulses) with amplitudes at a node only slightly above its threshold level. The pulse will be regenerated as a standard width action potential and propagated toward the brain. It will be indistinguishable from any other action potential, except for its timing and the specific neural path it is on. If excited by a pulse stream of varying frequency, the node will regenerate that precise pulse stream over a wide dynamic range[69]. The amplitude dynamic range of the excitation is extremely small[70] since the node is designed as a repeater of low amplitude pulses. Raising the excitation intensity only marginally affects the timing of the regenerated pulse(s).

Nodes labeled –1's are designed as analog signal encoders. They accept an analog signal (or treat a pulse as an analog signal) and generate a series of pulses at their output with a frequency proportional to the amplitude of the stimulation. An excitation pulse must have a minimum pulse duration because these encoding circuits typically have a low pass filter in their input structure. Thus, the output of the node is proportional to the amplitude of the pulses exciting it and a function of the excitation pulse duration.

The nodes labeled with the subscript O– are associated with the narrowband OHC neurons. Stimulation of these nodes will elicit responses related to specific acoustic frequencies. The comments of Tong et al on page 155 are instructive. "The results revealed that the hearing sensations produced by electrode 1 and electrode 2 were rarely confused within the two ranges of pulse rates: 105 to 165 pps, or 180 to 240 pps." This statement shows their tonal character as well as confirming the progression of neural signals from low frequencies near the apex to higher frequencies near the base. It is also of interest that their tonal range between the two groups was less than an octave as expected from the model. An octave would require electrodes approximately 2.3 mm apart and would likely introduce confusion as discussed in **Section 9.xxx**. The ranges also suggest the frequency range of neural paths excited by one electrode was on the order of 0.66 to 0.75 octaves. This range is too wide to support most speech feature-based signal processing approaches. Bringing the electrodes closer to the basilar membrane surface would be helpful.

The nodes labeled with the subscript I– are associated with the broadband IHC neurons. Stimulation of these nodes will elicit responses related to specific temporal characteristics with sharp responses associated with excitation at basal locations and dull responses associated with more apical locations (as reported in Tong et al.) Tong et al. noted one case where an electrode number 9 was very close to the round window and did not follow this pattern (page 154). It is proposed this electrode was beyond the effective start of the cochlear partition as discussed in **Section xxx**.

Using the above figure and analyses, the data of Tong et al. can be interpreted in much greater detail. The following summary of conclusion from Tong et al. (page 158) are presented without the embedded citations.

> "One of the most important observations made from the electrical stimulation of the human cochlea is the orderly increase in pitch and sharpness with the position of the intracochlear electrodes. There is little doubt (from our results and those reported by other researchers) that at least for postlingually deaf patients this phenomenon is real. The most likely mechanism for this phenomenon is the activation of different groups of residual auditory nerve fibers by electric current, and subsequent processing of this "place" information in the more central nuclei of the auditory system.
>
> In addition to electrode position, pulse rate is another electrical parameter influencing the pitch percept produced by an electrical stimulus. The major psychophysical results are summarized as follows: (1) The discrimination performance for electrical pulse rate is much poorer than that for

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[69]Schreiner, C. & Raggio, M. (1996) Neuronal responses in cat primary auditory cortex to electrical cochlear stimulation. II. Repetition rate coding *J Neurophysiol* vol 75(3), pp 1283-1300

[70]Raggio, M. & Schreiner, C. (1994) Neuronal responses in cat primary auditory cortex to electrical cochlear stimulation. I. Intensity *J Neurophysiol* vol. 72(5), pp 2334-2359

acoustic signals such as pure tones and multicomponent tone complexes in normal hearing subjects (Sec. IV), (2) the discrimination performance is poorer for pulse rates above 250 pps in comparison to that for lower pulse rates, (3) the increase in (scaled) pitch with pulse rate is less pronounced above-250 pps, and (4) the pitch sensation produced by a single-electrode stimulus with pulse rate below 250 pps can be matched to that for an acoustic pure tone with the same frequency in a cochlear implant patient with residual hearing in the unimplantcd ear."

10.7.2.1.1 Electrode configuration data EMPTY

Hartmann and Kral have noted, "Electrical stimulation of a node of Ranvier with a monopolar cathodic extracellular electrode leads to a depolarization . . . generating action potentials (page 225)." This quotation supports the position that most electrical stimulation by implants causes a more negative potential to be applied to the poditic terminal of the NoR. This application results in a positive change in the axoplasm potential leading to the generation of one or more positive-going action potentials.

Black et al. have provided data showing the advantages of bipolar stimulation over monopolar[71]. Longitudinal excitation patterns in humans were narrower by a factor of 4:1.

Clark et al. have discussed the performance of various orientations of full and split ring multi-electrode arrays[72] with a better graphic presented in Clark, 2003, page 268.. Their 1983 results "indicate that while there are inevitable variations in thresholds and growth responses with a free-fit electrode of this type, these will be significantly greater with a half-band electrode. Therefore, unless the placement and rotation of the half-band electrode can be closely controlled, the full-band electrode has advantages in providing minimum threshold and growth response variations." Many of these problems have now been overcome and various split ring configurations offer great potential in separately exciting OHC and IHC neurons.

Hartmann and Kral have provided useful data describing the frequency selectivity achievable with mono, bi- and tripolar electrode configurations (page 225). The best selectivity was obtained with the tripolar configuration. However, even this configuration led to selectivity only about one critical band wide. Although not specified, the context of their remarks suggests the electrodes were inside the scala tympani near the modiolar wall. This level of performance leaves little chance of achieving performance adequate for music appreciation of identification of a communicants voice.

10.7.2.2 The ideal strategy

The first challenge is to reliably excite as few IHC neurons as possible near the round window with a wideband temporal representation of the acoustic environment. The precise IHC excited is not critical. Optimally, a broadband signal extending to at least four kilohertz would be provided. As noted in the discussion accompanying figure 4.5.1–2, the energy in this signal will be integrated into a low pass equivalent channel of 600–1000 Hertz before being used to encode the signal by stage 3 neurons propagating to the cochlear nucleus. It is the timing of the first action potential generated by the stage 3 neurons that is critically important to the perception of unvoiced features in human speech.

The second challenge is to excite as few OHC neurons as possible at specific spacings along the cochlear partition. Ideally, the excitation would be optimized to excite OHC already wired together as a group as suggested in Figure 6.1.1–2 adapted from Lorento. In general, about four OHC in a given row of the cochlear partition are wired together. The goal should be to excite one or multiples of these four cell groups. The difficulty arises from the echelon arrangement of these groups relative to the longitudinal axis of the cochlear partition.

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[71]Black, R. Clark, G. Tong, Y. & Patrick, J. (1983) Current distributions in cochlear stimulation *Ann New York Acad Sci* vol 405, pp 137-145

[72]Clark, G. Shepherd, R. Patrick, J. Black, R. & Tong, Y. (1983) Design and fabrication of the banded electrode *Ann New York Acad Sci* vol 405, pp 191-201

It is mandatory that the individual signals exciting the groups described above be properly phased relative to the earlier OHC groups and relative to the IHC excitation. Otherwise, the brain will not be able to perceive the desired relationships.

10.7.2.3 A compromise strategy

It may not be possible to design an electrode array in the short term able to separably excite the IHC and OHC neurons, much less optimal size groups of each type of neurons. Therefore, a compromise strategy is desired demonstrating the majority of the long term goals using more readily available components from the current industrial base.

A minimalistic electrode array can be defined. It would have at least nine individual electrodes (or electrode groups).

> The intent of this design is to achieve both the perception of music by the recipient of this cochlear implant for the first time, and to pass to the hearing system the temporal fine structure that supports speech interpretation for the first time. To achieve this capability, it is important that the stimulating electrodes for the OHC neurons be spaced at submultiples of an octave along the longitudinal length of the cochlear partition. Currently, an octave is estimated to occur at distances of 2.3 mm along the partition (except at very low frequencies where the apical hook modifies this spacing below about 500 Hz as illustrated in **Section 4.5.4**. The use of an imprecise spacing will lead to non-harmonic perceptions of acoustically harmonic signals.

1. An electrode array with at least one electrode designed to carry a wideband signal for stimulating a group of IHC. The IHC oriented electrode would be inserted to approximately 6 mm from the round window.

2. An electrode array with a set of electrodes designed to excite OHC neurons along not less than 4.5 mm of the cochlear partition and beginning between 11 mm and 15 mm from the round window. The set should be designed to have two electrodes separated by as close to one octave along the cochlear partition as possible. See discussion accompanying [**Figure 4.5.4–2**]. The spacing between the other electrodes should be nominally equal. A nice feature would be to have them spaced in accordance with the notes of the musical scale.

The organization of each electrode should be optimized for exciting the fewest possible number of neurons longitudinally.

A key feature in future cochlear implants will be a structure that naturally seeks to place the electrodes as close as possible to the Tunnel of Corti. This will probably require an array of the complexity found in the Nucleus Contour perimodiolar array (page 535 in Clark, 2003) but re-targeted to place the array quite far from the modiolar. The ideal array would be placed adjacent to the basilar membrane and centered on the spiral artery.

The rapid advance in the manufacture of printed circuits both as microcircuits and as interconnecting circuit boards makes a very sophisticated cochlear implant possible now. Fifty micron wide traces (printed wires) are currently used on the circuit boards in commercial cell phones. 0.5 micron traces (as well as 0.5 micron square transistor gates) are used in current consumer market microcircuits (100:1 finer than traces on circuit boards). The 50 micron technology can be adapted to printed circuitry on flexible polyimide substrates of limited size with minimum difficulty. White et al. documented some of the specifics of this process in 1983 (although they used contacts five times larger that reflected the state of the art of that time).

Figure 10.7.2-3 shows a potential printed circuit array overlaid on a graphic from Jahn & Santos-Sacchi (page 179). The array will be formed of printed circuits on a polyimide substrate that curls to follow the scala tympani while positioning itself adjacent to the tunnel of Corti. The precise curl is determined by the silastic mold capturing the polyimide substrate, and any alignment fixturing needed for insertion purposes.. It will employ a multi electrode configuration that can excite the OHC when desired and the IHC when desired. It will minimize excitation of the complementary set of neurons by treating the electrodes nearest that group as a monopolar ground. Simultaneously, it will excite the desired set of neurons at a given longitudinal location using a biphasic (and potentially bipolar) electrode configuration on that side of the array.

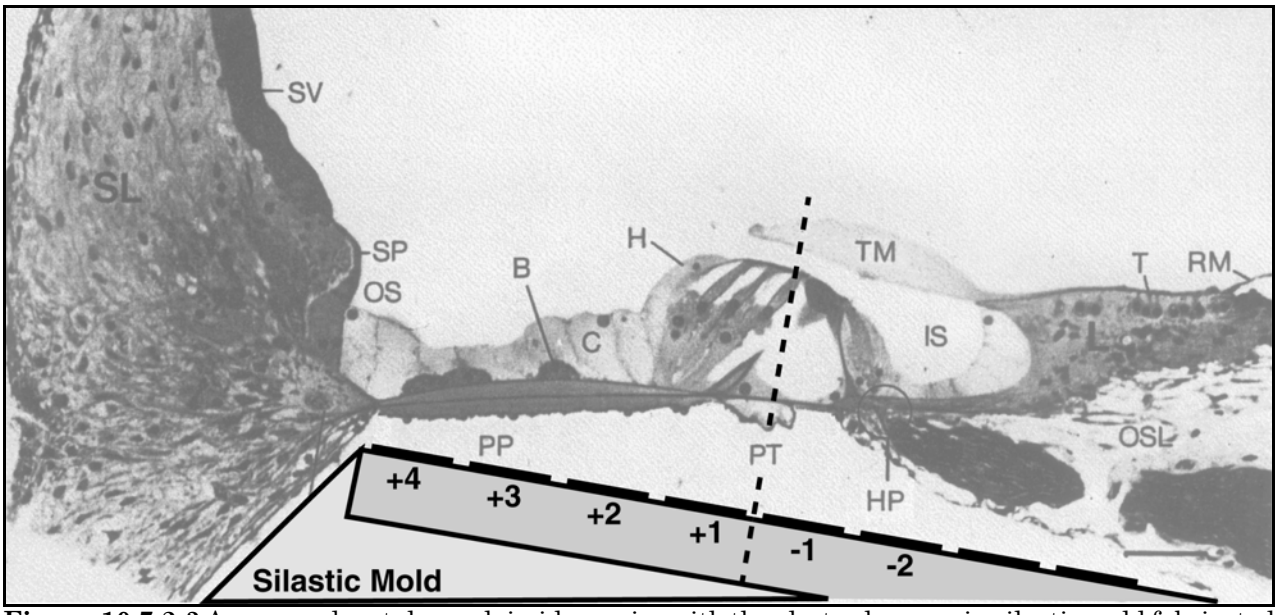

Figure 10.7.2-3 A proposed metal on polyimide carrier with the electrode array in silastic mold fabricated using microcircuit or nanomaterials technology. The silastic mold insures alignment of the carrier over the organ of Corti within the cochlear partition. The electrode array is split along a line optimally positioned to bisect the Tunnel of Corti (dashed line). Individual electrodes are shown at 60 micron spacing (50 micron pads and 10 micron spacing. Additional circuitry can be incorporated below the active pad area or in areas along the longitudinal axis as required. HP; habenula perforata (circled). IS; inner sulcus. OSL; osseous spiral lamina. PP; pars pectinata. PT pars tecta (incorporating the spiral artery). TM; tectorial membrane. Scale bar at lower right; 50 microns. Micrograph from Jahn & Santos-Sacchi, 1988.

The pads, numbered –2 to +4 in the figure, are shown above the surface of the polyimide substrate for clarity. In the actual device, these would be at or below the surface of the polyimide layer. The pads would also be overlaid with a high permittivity dielectric coating that would isolate the biological tissue from any net direct current flow during device operation.

10.7.2.3.1 Electrical operation of the implant EMPTY

The goal of the cochlear implant is to allow excitation of the OHC and IHC at a given longitudinal location independently and independently of other locations along the cochlear partition.

The array is shown with an excess of pads at a given location. The specific pads to be used in excitation can be selected after implant positioning to compensate for any lack of perfect alignment relative to the pars tecta.

The pads, and the potential of the signal applied to the selected pads will be optimized to cause maximum excitation of the desired sensory signal paths and minimum excitation of the adjacent signal paths. Once optimized, it is anticipated the circuitry will be operated in pulse mode at a constant pulse amplitude (at least for individual sections of the longitudinal array.

The system is designed to change the electrical potential of the poditic terminal of the Nodes of Ranvier following the first node (that performing the analog to pulse conversion). By changing this potential, action potentials in synchronization with the exciting pulses will be propagated to the next Node of Ranvier. The propagated pulses will be indistinguishable from those propagated in a totally healthy equivalent ear.

It should be noted, the artificially generated action potentials are not stochastic in any sense. They are totally deterministic and in synchronization with the applied

stimulus.

The pulse frequency applied to a given set of pads will vary in frequency as a function of the desired perceived amplitude. The frequency may be varied continuously in accordance with the amplitude of the acoustic energy to be simulated, or the frequency may be set for intervals of nominally 30 ms (based on the average value of the acoustic stimulus during similar intervals.

The option of pulsing each array pad using a biphasic pulse will be preserved until demonstrated to be unnecessary.

The instantaneous electrical field designed to excite either the OHC or the IHC can be tailored by using signals applied to multiple pads. The optimization of the electrical fields have not been attempted at this time. This optimization may involve bipolar pad operation as explored by Brummer, Robblee and Hambrect.

10.7.2.3.2 Fabrication of the operational implant EMPTY

Maintaining the flexibility needed for implanting the proposed cochlear implant without damaging the electrical circuitry is a non-trivial task. It is assumed the array must exhibit a straight planar configuration prior to insertion and follow the curvature of the cochlear partition afterward (over a distance of at least 20 mm). While the change in curvature of the polyimide substrate and attached wiring is minimal in the plane perpendicular to the substrate surface (parallel to the modiolar axis), the change in curvature is significant in the plane of the substrate. This may require the longitudinal wiring traces be in zigzag form in the intervals between active pad locations. These active pad locations are expected to be concentrated at intervals of 1/8th of 2.3 mm or 287.5 microns along the length of the implant for a nominal 20 mm.

Roland, Huang & Fishman provide interesting details of current electrode array practices[73].

- - - - -

[73]Roland, J. Huang, T. & Fishman, A. (2006) Cochlear implant electrode history, choices and insertion techniques *In* Waltzman, S. & Roland, J. *eds*. Cochlear Implants, 2nd Ed.. NY: Thieme Chapter 9

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