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Loudness and the Auditory Brainstem and Middle Latency Responses

By

Jo Manette Kathleen Nousak

A dissertation submitted to the Graduate Faculty in Speech and Hearing Sciences
in partial fulfillment of the requirements for the degree of Doctor of Philosophy.
The City University of New York

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September 10 2001
Date

David R. Stapells
David R. Stapells, Ph.D.
Chair of Examining Committee

9/10/01
Date

Robert Goldfarb
Robert Goldfarb, Ph.D.
Executive Officer

Supervisory Committee:

Harry Levitt, Ph.D.
Distinguished Professor Emeritus
Graduate Center of CUNY

Stanley Gelfand, Ph.D.
Queens College
Graduate Center of CUNY

External Examiner:

Sharon Sandridge, Ph.D.
Cleveland Clinic Foundation
Cleveland, Ohio

THE CITY UNIVERSITY OF NEW YORK

Abstract

Loudness and the Auditory Brainstem and Middle Latency Responses

by

Jo Manette K. Nousak

Advisor: David R. Stapells, Ph. D.

This dissertation examines the relationship between amplitude/latency changes of the auditory brainstem (ABR) and middle latency (MLR) responses with intensity and behavioral loudness growth measures to 1000-Hz tones. Studies of both normal-hearing and sensorineurally-impaired listeners examined (1) the accuracy of the ABR and MLR in predicting behavioral hearing threshold, and (2) the relation between the rate of ABR/MLR amplitude growth/latency decrease and behavioral loudness growth with increasing threshold shift. Results indicate that the ABR and MLR give equally accurate estimates of hearing threshold for 1000-Hz tones for both groups of subjects. In addition, ipsilateral broadband masking effectively shifted the thresholds of the normal-hearing listeners to 40 and 60 dB nHL levels for both the electrophysiologic and behavioral measures. Relative to the normal response, electrophysiologic measures to supra threshold stimuli reveal (1) differences between the ABR and MLR in subjects with

decreased hearing sensitivity, and (2) the effects of noise masking on the ABR/MLR are different than the effects of cochlear impairment. ABR and MLR intensity-amplitude/latency functions show a slower rate of change with intensity compared to behavioral loudness growth rates for both normal and impaired listeners. However, ipsilateral broadband masking studies of the normal listeners reveal that the relative increase in slope with increasing masker is similar for both the electrophysiologic and behavioral measures, indicating that the ABR/MLR reflect, at least in part, the same neural processes that underlie loudness perception. Hearing loss (masking and cochlear impairment) causes an increase in the rate of ABR/MLR amplitude growth, ABR latency decrease, and loudness growth with intensity increase, and these electrophysiologic and behavioral functions steepen with increasing threshold shift. These supra threshold results indicate that the ABR/MLR may serve as potential predictors/indicators of loudness growth in persons who are unable to report their judgement of the loudness of sound.

Preface

This dissertation is arranged in the following style. Chapter 1 is a brief introduction to the thesis. Chapter 2 is a review of the relevant literature concerning psychoacoustic measures of loudness and physiology of intensity coding, and Chapter 3 reviews the relevant literature on the effects of intensity on auditory evoked potentials. Within each of these sections is reviewed the pertinent findings regarding the normal response to increasing stimulus intensity, and also the effects of (a) noise masking in normal-hearing listeners and (b) sensorineural hearing loss on the responses to increasing signal level. Chapter 4 discusses the rationale for the studies of the present dissertation, including stimulus and recording parameters for obtaining the ABR and MLR. Threshold values for both the behavioral and electrophysiologic measures are compared in Chapter 5, and the ABR/MLR absolute latencies and amplitudes with intensity are described and also compared within and between groups. In Chapter 6, ABR/MLR intensity-amplitude and intensity-latency functions are compared to behavioral measures of loudness growth both within and between groups, (normal-hearing, noise-masked normal-hearing, and sensorineurally impaired subjects), and these findings are discussed in detail. Chapters 5 and 6 are written in the form of independent research papers, so there is some overlap of material among chapters. Chapter 7 presents summary and conclusions, and this section is followed by appendices of the individual subjects' data, and tables of the ANOVAs included in Chapters 5 and 6. The final section lists all of the references cited in this dissertation.

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And last but most – the best is always last. There were five schedules, five siblings, and five minutes to the end: there was 1964, 1976, and 1980: there were many silences, some moments of clarity, and a few good one-liners; there were three measures, three groups of subjects, and three conditions; there were two meeting places for thousands of hours of work over almost 14 years; but as a dear friend of mine used to tell me when the occasion arose: 'in the end, it takes only ONE'. Thank you, Carl.

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Chapter 1

Introduction

The primary focus of these dissertation studies was to examine the relation of the tone-evoked auditory brainstem (ABR) and middle latency (MLR) response to psychoacoustic measures of the auditory dynamic range. The ABR and MLR, which derive from different generators within the auditory system, were recorded simultaneously in order to compare these brainstem and early cortical responses with respect to their accuracy and potential use as predictive measures of hearing sensitivity and loudness growth. Previous studies have compared the rate of change in the ABR or MLR amplitudes and/or latencies to the loudness growth rate for click stimuli (e.g., Madell & Goldstein, 1972; Pratt & Sohmer, 1977). Because neural measures of stimulus magnitude consistently show a slower rate of change than do behavioral loudness results (e.g., Davis & Zerlin, 1966; Wilson & Stelmack, 1982), a different method of evaluating their relation was used in the present studies to determine whether or not the electrical responses reflect activity included in psychophysical experiences of loudness growth. Broadband noise was employed in these dissertation studies to simulate sensorineural hearing loss in normal listeners. Masking noise produces threshold elevations and loudness recruitment that is similar to that seen in sensorineural hearing loss (e.g., Hellman & Zwislocki, 1964; Schlauch et al., 1998; Stevens & Guirao, 1967), including steepening of the loudness growth function with increasing threshold shift (e.g., Gleiss & Zwicker, 1964; Hellman, 1981; Lochner & Berger, 1961; Miskolczy-Fodor, 1960). These effects of the noise masker are thought to be predominantly of cochlear origin (e.g., Oxenham & Plack, 1997; Zwicker & Fastl, 1999), although recent evidence indicates that there are also central contributions to these changes (e.g., Gerken, 1991, 1996; Phillips, 1987, 1990). Two

levels of broadband noise were used to simulate mild and moderate hearing loss in order to assess whether or not the relative changes in the electrophysiologic and behavioral responses to increasing signal level are similar or different (Stevens, 1970). These results were augmented by obtaining the same measures from a group of sensorineurally impaired individuals with mild-to-moderate cochlear loss of hearing sensitivity.

The following review discusses and describes the relevant literature pertaining to the psychoacoustic and electrophysiologic measures that were obtained in the present studies, including description of the underlying physiological mechanisms responsible for the behavioral and evoked potential results. It includes a discussion of pertinent studies reported previously relating electrophysiologic and behavioral results with intensity, including the early work on the Cortical Auditory Evoked Potential which, unlike the conclusions drawn from prior ABR and MLR studies, showed that auditory evoked potentials relate to loudness. Because this dissertation examined three measures (i.e., loudness, ABR, and MLR) in three groups of subjects (i.e., normal-hearing, noise-masked normal-hearing, and sensorineurally-impaired), each section of the literature review contains summary of results obtained from normal-hearing subjects, as well as description and discussion of the effects of masking noise and cochlear pathology on these measures.

Chapter 2

Review of psychoacoustic measures of loudness and physiology of intensity coding

Psychoacoustics of Loudness

Loudness is the psychological attribute of sound associated with an increase in sound pressure level of an auditory stimulus (Davis, 1970, pg. 33). The loudness of a sound is subjective, and, therefore, is measured using psychophysical methods. These behavioral methods require subjects to make judgements of the apparent magnitude of auditory stimuli, and from these data, loudness scales are obtained. "By a scale of subjective magnitude we mean a quantitative scale by which we can predict what people will say when they try to give a quantitative description of their impressions" (Stevens, 1956, pg. 2).

In measuring the relation between sound intensity and subjective magnitude perception, we are interested in mapping the auditory dynamic range. The dynamic range of hearing is that range of intensities between threshold, or the faintest tone a subject can hear (Green, 1972), and the threshold of discomfort, expressed in dB and referred to hearing level (dBHL) or sound pressure level (dBSPL) (Hawkins, Walden, Montgomery & Prosek, 1987). Determination of hearing thresholds – i.e., the intensity level at which a stimulus is just detectable – is a central tool in auditory psychophysical experiments/procedures. For suprathreshold stimuli, two general types of psychophysical scaling procedures have been proposed to measure the relation between perceptual magnitude and stimulus magnitude. In one approach, differential sensitivity thresholds are obtained by measuring the smallest detectable difference in intensity that can be discriminated 50% of the time. It was Gustav Fechner in 1860 (Fechner, 1966) who first suggested that these just-noticeable-difference (JND) thresholds may be used to quantify

the intensity-loudness growth transformation. Fechner based his relation on the earlier work of E. H. Weber who found that the JND increases in direct proportion to intensity increases. Fechner proposed that the sensation of loudness increases by a constant amount each time the stimulus intensity is increased by a JND ($\Delta I/I$, the Weber fraction). According to Fechner, all JNDs could then be added to estimate a scale of the total subjective magnitude of the sensation, and the greater the slope of the loudness function, the smaller is the JND. His relation may be described by the formula:

$$P = k \log S$$

where P is the subjective loudness unit, k is a constant, and S the physical intensity unit (Plack & Carlyon, 1995). This notion of a logarithmic law that describes sensation growth, however, was not supported by experimental evidence from studies employing pure-tone stimuli (e.g., Miller, 1947; Stevens & Davis, 1938). Although intensity discrimination experiments are a primary psychophysical tool used to investigate the way in which intensity is coded by the auditory system (Plack & Carlyon, 1995; Viemeister, 1988), the relationship of loudness to intensity JNDs remains unclear (Plack & Carlyon, 1995; Veimeister, 1988). The reader, however, is referred to recent publications that report new approaches to this classical problem of relating JNDs to loudness (i.e., Allen & Neely, 1997; Johnson, Turner, Zwislocki & Margolis, 1993; Schlauch, Harvey & Lanthier, 1995).

A second approach utilizing more direct methods of scaling was proposed by Stevens in the 1930s (Stevens & Davis, 1938). In these methods, different stimulus intensities are presented within the subject's dynamic range, and subjective estimates are used directly

to form a function relating perceived magnitude of the stimulus to some physical measure of the signal (i.e., the psychophysical function). The form of the loudness function varies with the scaling method employed.

Behavioral scaling tasks used to measure the relationship between loudness and intensity are generally defined by the type of subject response required. These include partition (Keidser, Seymour, Dillon, Grant, Byrne, 1999; Launer, 1995) and magnitude scales (Hellman & Meiselman, 1993; Serpanos, Gravel & O'Malley, 1997). The most commonly used partition procedure is category scaling, in which subjects are asked to label their perception of sensation magnitude according to a set of categories defined by number and/or adjective (e.g., 1 = "soft", 5 = "loud, but OK"), and outlined prior to presentation of the stimuli (Bentler & Pavlovic, 1989; Hawkins et al, 1987; Launer, 1995; Macpherson, Elfenbein, Schum & Bentler, 1991; Pascoe, 1978). This method requires subjects to place a range of stimuli into equally spaced or sized categories, and the resulting data are transformed to derive loudness growth functions (Guirao, 1991). Factors that can influence the form of the function derived from categorical scaling are contextual effects, judgement bias, and number of categories (Poulton, 1989; Stevens & Galanter, 1957). For example, a small number (1-7) of categories yields a much shallower slope for loudness growth compared to the function derived from either a partition scale that includes a larger range of categories (e.g., 1-100) (Guirao, 1991), or from magnitude scales that allow the subject the widest possible range of choices in units (Stevens & Galanter, 1957). Category scales that include relatively small numbers of categories are common in clinical use for defining an individual's auditory area, including

Most Comfortable Level (MCL) and the Loudness Discomfort Level (LDL) (e.g., Robinson & Gatehouse, 1996). More recently, adaptive categorical scaling procedures have been advocated for use in obtaining reliable and valid measures of loudness growth (Allen, Hall & Jeng, 1990; Keidser et al., 1999, Kiessling, Schubert & Archut, 1996; Launer, 1995), some of which have yielded loudness growth functions that are similar to the results obtained using magnitude estimation (Launer, 1995).

The most commonly used magnitude procedure is the method of magnitude estimation (Gescheider, 1988). This approach to measuring sensation magnitude was developed by S. S. Stevens in the 1950s. Stevens posed the question "How do people describe the apparent strength of a 1000-Hz tone when presented at different levels, and the listener describes its loudness in a numerical language instead of adjectives?"(Stevens, 1956). He asked subjects to judge the loudness of a sound on a ratio scale. That is, given a reference stimulus, or modulus, labeled as having a certain loudness (e.g., 10 units), subjects were asked to report their perception of how much louder (or softer) a second stimulus was in relation to the modulus. This is the basis of the sone scale (Stevens, 1957). For example, given a 40 dBSPL 1000-Hz tone (defined as one sone), subjects were presented with a second stimulus and asked to judge whether it was twice as loud, one-half as loud, etc., by assigning a number to the second stimulus presentation. These data, plotted directly along the physical (stimulus intensity) continuum result in a function that shows the relation between loudness and sound intensity: each 10-dB increase in level results in an approximate doubling of loudness, and hence a doubling of the sone value (Stevens, 1955). Stevens found that the assigned numbers (N) were related to stimulus intensity (I)

by a power function, which may be expressed as $N = kI^e$, where N is the assigned number, k is a constant depending on the choice of units, I is the intensity level (referred to Intensity Level in dB), and e is the exponent. Because Stevens contended that the assigned numbers were directly proportional to sensation magnitude, his general psychophysical power law, which describes the power function relationship between sensation magnitude and stimulus intensity, substitutes sensation magnitude (ψ) in the equation expressed as:

$$\psi = kI^e$$

Stevens' Power Law applies not only to loudness, but also to other sensory continua that relate to subjective intensity or magnitude, such as brightness, weight, and smell (Stevens, 1959). When measured using magnitude estimation, the various sensations yield power functions, but the value of the exponent (i.e., the slope of the line in a log-log plot) is characteristic of the particular sensation. For example, the exponent for electric shock is 4.5, whereas brightness shows a more gradual increase in perceived magnitude with increasing light (0.33). The exponent relating loudness to intensity is approximately 0.30 (or 0.60 re: pressure) (Stevens, 1956; Hellman, 1991).

The basic assumption of direct scaling using magnitude estimation is that the observer is able to match numbers to her perceptions. Because Stevens assumed that listeners judge the loudness of a stimulus on a "ratio scale", in accordance with his definition of ratio scales (Stevens, 1951), a subject's numerical judgements should be invariant to a mathematical transformation when the value of the modulus is changed. However, in later experiments, it was found that the arbitrary modulus had a substantial affect on the

form of the loudness function, and therefore resulted in response bias (Hellman & Zwislocki, 1961; Stevens, 1956). That is, the scale values obtained with one modulus were not linearly related by a multiplicative constant to those obtained with a different modulus. Stevens' method was therefore later modified by Hellman and Zwislocki (1961, 1963, 1964, 1968), who suggested that listeners pair loudness and numbers on an absolute basis with a fixed unit (Hellman & Zwislocki, 1961) rather than on a ratio scale with an arbitrary unit. The Absolute Magnitude Estimation (AME) hypothesis (Hellman & Zwislocki, 1961, 1963, 1964) states that subjects tend to assign a number to a stimulus in such a way that their psychological impression of the size of the number matches their impression of the psychological magnitude of the stimulus. Subjects are, therefore, not restricted in their choice of numbers used, but rather are encouraged to assign numbers that are felt to be appropriate without any biases that my be associated with a response system devised by the experimenter. From a group of normal-hearing listeners who gave numerical estimates of the loudness of 1000-Hz tones, Hellman & Zwislocki (1963) obtained a power function with an exponent of 0.54 (re: dB SPL) using magnitude estimation.

There have been criticisms of loudness scaling, many of which have to do with the technique's susceptibility to bias effects such as range of stimuli presented (Parker & Schneider, 1994), instructions to the subject (Keidser et al., 1999), and range of permissible responses (Marks, 1991). However, studies have shown consistency in the data, which some researchers suggest demonstrates validity of the technique (e.g., Zwislocki & Goodman, 1980). Indeed, there is a large body of empirical evidence in

support of the general form of the loudness function obtained with this method from both normal-hearing adults (Fucci, Petrosino, Mcoll, Wyatt & Wilcox, 1997; Gescheider & Hughson, 1991; Hellman, 1978, 1991, 1999; Hellman & Meiselman, 1988, 1990, 1993; Hellman & Zwislocki, 1961, 1963, 1964, 1968; Parker & Schneider, 1994; Rowley & Studebaker, 1969; Stevens, 1957, 1959; 1972; Zwislocki, 1983) and children (Bond & Stevens, 1969; Collins & Gescheider, 1989; Dorfman & Megling, 1966; Zwislocki & Goodman, 1980). In her recent review of 78 studies reporting slope values for loudness obtained using magnitude estimation, Hellman (1991) reported a mean slope of 0.3 (0.6 re: dBSPL); most of the slopes fell within ± one standard deviation, i.e., within the range of 0.255 and 0.345. While these studies include the mean results obtained from subjects tested individually and usually in sound-attenuated rooms, similar findings have also been reported from groups of unpracticed listeners tested in reverberant conditions (i.e., classrooms, auditoriums) (Canévet, Hellman & Scharf, 1986), indicating that inexperienced listeners in any listening environment judge the loudness of sounds in a consistent manner using AME.

Measures of loudness growth in normal listeners obtained using magnitude estimation procedures yield a psychophysical function with a slope less than unity (i.e., 0.54 re: dBSPL), indicating that a strong nonlinear compression along the intensity continuum takes place within the auditory system (e.g., Fletcher & Munson, 1933; Stevens, 1955). Psychological models based on Stevens' power law (1956) have been proposed to account for the compressive characteristics of the auditory system (e.g., Humes & Jesteadt, 1991; Zwislocki, 1965), and have been applied in many research studies

describing a wide variety of loudness data from normal-hearing, noise-masked normal-hearing, and hearing impaired listeners (e.g., Buus, Florentine & Poulsen, 1999; Florentine, Buus & Poulsen, 1998; Florentine, Fastl & Buus, 1988; Hellman & Meiselman, 1990; Humes & Jesteadt, 1991; Launer, 1995; Moore & Glasberg, 1996; Zwicker, 1965; Zwislocki, 1969).

Loudness scaling measurement in normal-hearing subjects also serves practical purposes in the engineering and design of sound systems (Stevens, 1955) and in the clinical utility of establishing norms which serve as reference levels for classifying hearing status. An international standard for loudness (ISO R 131-1959) was adopted in 1959 based on a large-scale study of normal-hearing listeners in several laboratories. Of practical and theoretical interest, also, is the loudness growth of partially-masked tones, because listening rarely occurs in quiet.

Psychoacoustics of loudness: Effects of masking

Broadband noise presented simultaneously with tonal stimuli shifts the threshold of detection for the tone, and causes an increase (i.e., steepening) in the slope of the loudness growth function (Hellman & Zwislocki, 1964; Stevens, 1966; Stevens & Guirao, 1967). For example, white noise presented at a fixed level of 40 dB SPL shifts the threshold of a 40 dB SPL 1000-Hz tone to just audible. The loudness of this 1000-Hz non-masked tone, equal to one sone, is reduced in the masked condition to zero sone. However, when the intensity of the tone in noise is increased to about 80 dB SPL, its loudness is nearly equal to the loudness of the non-masked 80 dB SPL tone. Thus, the masking noise causes a threshold shift, and also changes the loudness growth function to

one that shows a steeper growth rate compared to the non-masked response (Stevens, 1966; Stevens & Guirao, 1967, Zwicker & Fastl, 1999). The greater the threshold shift, the steeper is the loudness growth function (Stevens & Guirao, 1967).

In a study undertaken to provide further evidence of the validity of magnitude estimation scaling methods, Hellman and Zwislocki (1964) compared the results of loudness growth for partially-masked tones obtained by magnitude estimation to those obtained using a loudness balance procedure. In the latter procedure, tones are presented alternately between the right and left ears, and normal-hearing listeners are asked to adjust the loudness of the tones presented to one ear, the variable ear, to the perceived loudness of the tone presented to the other "standard" ear. For tones presented in the quiet, listeners are able to accurately perform this procedure so that the tones are nearly perfectly matched. The loudness growth functions for partially-masked tones obtained using the loudness balance procedure were found to be steeper than the non-masked response (e.g., Lochner & Berger, 1961). This being so, Hellman & Zwislocki (1964) supposed that, if the AME is a valid measure of loudness growth, the loudness growth functions for partially-masked tones obtained using magnitude estimation would be consistent with those obtained from loudness balance. Using a narrow-band masker (600 - 1200 Hz) that shifted threshold for 1000-Hz tones to 40 and 60 dBSL levels, the mean loudness growth functions for the partially-masked tones obtained from nine normal-hearing listeners were steeper than the non-masked functions for both psychophysical methods. In addition, the slopes for magnitude estimation and loudness balance were similar for each noise condition, and the slopes of their functions increased with

increasing masker level (Hellman & Zwislocki, 1964; Lochner & Berger, 1961). More recently, Schlauch, Digiovanni and Riesz (1998) employed a loudness balance procedure to obtain loudness growth functions to 4000-Hz long- (250 ms) and short- (2 ms) duration tones presented in broadband masking noise that shifted thresholds to 30 and 50 dB HL (long-duration) and 35 and 55 dB nHL (short-duration). Their data also show a faster rate of loudness growth for both partially-masked long- and short-duration tones compared to the non-masked responses, and a faster rate of loudness growth with increasing threshold shift.

The increase in the slope of the loudness growth function for partially-masked tones in normal-hearing listeners is similar to that seen in the results obtained from sensorineurally impaired listeners whose threshold is shifted primarily by cochlear dysfunction (Hellman, 1981; Hellman & Zwislocki, 1964; Miskolczy-Fodor, 1960; Stevens, 1966). Indeed, when Hellman & Zwislocki (1964) compared their normal listeners' results for partially-masked 1000-Hz tones to those of Miskolczy-Fodor (1960), who obtained loudness growth measures from patients with 40 and 60 dB HL sensorineural hearing loss, they noted the striking similarity in the loudness growth functions for both groups of subjects. Thus, the phenomenon of abnormally fast loudness growth with increasing intensity, known as loudness recruitment, is seen both in noise-masked normal-hearing subjects and in subjects with cochlear loss of sensitivity (Hallpike, 1976).

Psychoacoustics of loudness: Effects of cochlear impairment

Loudness measures in hearing-impaired persons are important in the diagnosis and treatment of hearing disorders, including differentiation of the nature of hearing loss (i.e., cochlear versus retrocochlear pathology) and, importantly, the habilitative purposes of determining appropriate amplification parameters such as the gain of a hearing aid, type of hearing aid output limiter and amplifier, and maximum output level (Hawkins & Mueller, 1990; Killion & Fikret-Pasa, 1993). The perceptual consequences of cochlear hearing loss with respect to loudness are decreased hearing sensitivity (i.e., increased absolute threshold), loudness recruitment, reduced loudness summation, and reduced frequency selectivity (see Moore, 1995 for review). Here we are mainly concerned with the first two phenomena, that of raised absolute thresholds and loudness recruitment.

The recruitment phenomenon was first identified by Fowler (1928) from studies of patients with unilateral hearing impairment. He developed the Alternate Binaural Loudness Balance (ABLB) test, which requires the subject/patient to match the loudness of a tone presented at a fixed intensity in the normal-hearing ear to that of the same frequency tone presented in the impaired ear (Fowler, 1936). The loudness growth function for the impaired ear revealed a steeper loudness growth function than that obtained from the normal ears. Later, in their studies on patients with hearing loss, Dix, Hallpike and Hood (1948) found that recruitment was present only in subjects with primarily sensory (i.e., cochlear) hearing loss, and absent in those with retrocochlear pathology. Miskolczy-Fodor (1960) collected 200 loudness-level measurements from several investigators in addition to 100 patients of his own who had unilateral

sensorineural hearing loss exhibiting loudness recruitment. His results for patients with 40, 50, 60, and 80 dBHL hearing loss show the characteristic faster rate of loudness growth compared to the normal response, and also that with increasing hearing loss, the loudness function becomes more steep. The ABLB thus serves as a clinical test to both identify cochlear pathology and to assess loudness growth for rehabilitation purposes. As a measure of loudness, however, it has been pointed out that fewer subjects/patients have unilateral impairment compared to the number presenting with bilateral sensorineural hearing loss, thus reducing the usefulness of the ABLB as a clinical measure of loudness growth (Hellman, 1999).

Magnitude estimation techniques have recently been advocated for use in the study and measurement of loudness growth in hearing-impaired adults (Elberling & Nielsen, 1993; Hellman, 1999; Hellman & Meiselman, 1990, 1993; 1993; Knight & Margolis, 1984; Launer, 1995), and as a potential method for hearing aid selection in adults (Geller & Margolis, 1984; Harris & Goldstein, 1985; Margolis, 1985; Purdy & Pavlovic, 1992; Studebaker & Sherbecoe, 1988). Hellman and Meiselman (1993), for example, obtained loudness growth functions and their slope values from a group of normal-hearing listeners, and compared those findings to measures obtained from a group of sensorineurally impaired listeners using magnitude estimation, magnitude production, and cross-modality matching. Magnitude production (AMP) is a procedure that requires the subject to adjust the loudness of a tone to match the perceived magnitude of numbers assigned by the experimenter (Hellman, 1999), and generally yields loudness growth functions that are slightly steeper than those obtained using magnitude estimation

(Hellman, 1981). The results for the normal listeners gave a mean exponent of 0.62, (re: dBSPL). The mean loudness exponents for the impaired subjects with 58 and 69 dB HL sensorineural hearing loss were 1.60 and 2.31 respectively, indicating a steeper loudness growth function compared to the results for the normal listeners; the steepness of the slope depends on the severity of the hearing loss. Intersubject variability increased with increasing hearing loss, which is thought to be mainly due to heterogeneity of individual thresholds and/or the amount and type of cochlear dysfunction (Hellman & Meiselman, 1993; Hellman, 1999).

Stevens (1966) contended that the steeper slope of the loudness functions observed in noise-masked and sensorineurally-impaired listeners is due to a transformation of signal level processing in the auditory system, and that a common underlying physiological mechanism may be responsible for the recruitment effect seen in these two groups of listeners. The power law has been applied in subsequent research to investigate the similarities of and the differences between these effects of noise-masking and cochlear pathology on loudness growth (e.g., Humes & Jesteadt, 1991; Moore & Glasberg, 1996; Oxenham & Plack, 1997; Schlauch et al., 1998) and on related issues, such as temporal integration (Florentine et al., 1988, 1998) and loudness summation (e.g., Florentine & Zwicker, 1979; Zwicker, 1965; Zwislocki, 1969) in an effort to determine, albeit indirectly, the mechanisms involved in the perceptual response transformation. In recent studies, loudness data for partially-masked tones in normal listeners and from cochlear-impaired subjects have been compared to physiologic results of basilar membrane input-output functions to assess the notion that peripheral mechanisms, in the main, determine

the form of the loudness function (e.g., Oxenham & Plack, 1997; Schlauch et al., 1998; Yates, Winter & Robertson, 1990). Others, however, have suggested that central mechanisms play a large role in determining the shape of the loudness function (e.g., Zeng & Shannon, 1994; Zeng, Shannon & Hellman, 1998). In later sections, the physiological mechanisms that underlie loudness perception in normal, noise-masked normal, and cochlear-impaired systems will be described, and their relation to theories of intensity perception will be discussed.

Psychoacoustics of loudness: Pediatric and developmental issues

Little is known about the perception of loudness growth in infants and very-young children (Mueller & Bentler, 1994). Using category scaling with categories defined by pictures considered age appropriate, Kawall, Kopun and Stemalchowicz (1988) demonstrated that hearing-impaired children aged 7-14 yrs responded reliably in reporting their LDLs, and gave similar results as those obtained from a group of hearing-impaired adults. Macpherson, Elfenbein, Schum and Bentler (1991) employed several tasks involving analogical reasoning to train and assess 4-7 year-old children's thresholds of discomfort (LDL). They obtained reliable responses from those children with a mental age (MA) of 5 yrs and older; children who had an MA younger than 5 yrs were unable to respond successfully in the task to assess LDL. Employing several categorical scaling procedures that included pictorial representations of "soft", "loud", etc. that were developed for very-young children (i.e., < 5 yrs), Launer (1998) reported that children younger than 5 years of age are not able to judge reliably the loudness of tones using categorical scaling techniques.

Zwislocki and Goodman (1980) tested children aged 6 yrs and older using magnitude estimation and found similar results for these normal young listeners as those obtained from a group of normal-hearing adults. Collins and Gescheider (1989) also studied the responses of 12 adults and 11 children (aged 4-7 yrs) with this method, and showed no significant differences in the loudness exponents between the two age groups, or in the variability seen within the two groups. Children younger than 4-5 years of age, however, generally are unable to report reliably their perception of sensory magnitude using this method because preschoolers are unable to assign numbers in a manner that describes sensation magnitude (Smith, 1985). Zwislocki and Goodman (1980) suggest that this is due to the fact that the original coupling of numbers with impressions of physical quantities is a concept that children younger than 5 yrs have not yet developed (Fuson, 1988; Michie, 1985).

Presently, there is no recommended behavioral procedure for measuring loudness growth or loudness comfort/discomfort levels in infants or very-young children (Launer, 1998; Mueller & Bentler, 1994), yet children with hearing loss are currently identified in most developed countries at these early ages (e.g., Gravel, 2000). Indeed, with the advent of universal hearing screening programs, children with hearing loss are identified at birth, and hearing aids are fitted by age 6 months (Gravel, 1992, 2000; The Pediatric Working Group, 2000), indicating the need for an assessment protocol that provides a measure, or an estimate, of loudness growth in this young population to ensure the fitting of adequate and comfortable amplification systems for remediation of hearing loss and development of speech and language during these critical years (Pediatric Working Group of the

Conference on Amplification for Children with Auditory Deficits, 1996; Pediatric Working Group, 2000). Currently, the auditory brainstem response (ABR) is recommended for use in assessing threshold estimates and/or confirming behavioral threshold measures of type, degree, and configuration of hearing loss in infants aged 0-36 months (Pediatric Working Group, 2000). The ABR (and MLR) may also be recorded to suprathreshold tonal stimuli. Auditory evoked potential amplitudes and latencies are level dependent and thus may show a dependence on intensity that is similar to that seen in behavioral loudness growth measures (Keidel, 1976). It may be that one or both of these evoked potential recordings could, therefore, be useful in predicting loudness growth in the pediatric population and for others who are unable to participate in behavioral tasks that require their report of the loudness of sounds. Such is the focus of this dissertation.

Before reviewing the literature on the effects of intensity on auditory evoked potentials, it is useful to review first the physiology of intensity coding. This provides description of the physiology that underlies and relates to both the behavioral and electrical output measures examined in this dissertation. Secondly, because the present studies include measures of two electrical responses that reflect neural activity from different levels of the auditory pathway, review of the physiologic responses from various portions of the auditory system to increasing intensity provides background information that is germane to the present dissertation studies.

Physiology of intensity coding

The amplitude of a sound wave is the attribute of the physical stimulus associated with the magnitude of pressure movements produced. Its subjective correlate is loudness. The relation between the perceived output (i.e., loudness) and the physical input (e.g., a 40 dB SPL, 1000-Hz tone) of a sound is determined by the way in which the acoustic signal is processed by the auditory system. The peripheral auditory system transforms and encodes the mechanical energy of sound waves into electrical pulses, which are subsequently transmitted to the auditory cortex via the auditory nerve and several higher-level relay stages of the central auditory pathway. Following is an overview of the physiology of intensity coding in the auditory system.

There are basically three stages of sound transformation in the peripheral auditory mechanism that contribute to auditory processing of stimulus intensity. In the first stage, the outer ear collects sounds and causes attenuation and enhancement of different frequencies that arrive at the tympanic membrane (TM). The middle ear, composed of the TM and ossicles (malleus, incus, and stapes), transforms the variations in sound pressure from the ear canal to a sound pressure variation in the cochlea. The outer and middle ear act as linear systems, such that the output of the stapes footplate increases in proportion to an increase in the input sound pressure level, and no significant nonlinearities (e.g., harmonics or intermodulation distortion products) are seen (Guinan & Peake, 1967).

In the second stage, the inner ear hearing organ (i.e., the cochlea) transforms the mechanical energy of sound waves to neural pulses that are transmitted to the neural

pathways. The mechanical vibrations of the stapes, transmitted to the scala vestibuli via the oval window, result in a fluid motion in the cochlea which causes displacement of the basilar membrane and its associated structure, the organ of Corti. The displacement is characterized as a traveling wave of motion that is initiated at the base of the cochlea. Its amplitude increases as it moves upward along the partition towards its apex, until it reaches a resonant place along the partition after which it abruptly dissipates (von Bekesy, 1960). The place along the partition of maximum peak amplitude depends on the frequency of the stimulus, and at the resonant point, basilar membrane vibration grows as a power function of stimulus amplitude (Ruggero, Rich, Recio, Narayan & Robles, 1997). Displacement of the basilar membrane stimulates the outer hair cells and receptor inner hair cells (OHCs & IHCs) in the organ of Corti. Chemical changes in the receptor potentials in turn initiate the third stage of peripheral processing, an excitatory response in the afferent neurons of the cochlea from which series of action potentials, or electrical pulses, are generated and transmitted via the auditory nerve to the various nuclei of the central auditory system.

Intensity coding in the cochlea depends upon the mechanics of the basilar membrane and the cochlear fluids, and their interactions with the hair cells. The traveling wave, initiated by the vibrations of the stapes, moves from base to the apex of the cochlea. A basic property of the cochlea is tonotopicity. This refers to the regular order of best frequency along one dimension. The resonance properties of the basilar membrane change systematically as a function of distance due to decreasing stiffness in the cochlear partition with increasing distance from the stapes, such that the motion of the cochlear

fluids and peak displacement of the basilar membrane in response to low-frequency stimuli travel the length of the cochlea, peaking toward its apex (Geisler, 1998, pg. 63; von Bekesy, 1960). Waves of displacement resulting from high-frequency stimuli peak toward the base of the cochlea and do not propagate farther. Tonotopy is seen also in primary auditory neurons and all central auditory nuclei, and serves as a basic scheme for describing the response of the auditory system to changes in sound pressure level.

The passive traveling wave initiated in the cochlea is altered by the action of the OHCs which inject mechanical energy into the traveling wave, creating a larger vibration than would otherwise be present (Patuzzi, 1996). The resulting active wave grows relative to the passive wave as long as the active outer hair cells are adding energy to it. Thus, low-level sounds are effective in producing a particular amplitude of vibration or particular hair cell or neural response. There is a gradual transformation of the vibratory pattern from the highly sensitive and localized active pattern at low stimulus levels to a less sensitive and broader passive response pattern at high stimulus levels (Patuzzi, 1996). This progressive change in basilar membrane mechanics is responsible for much of the nonlinearity in cochlear mechanics. For a given stimulus frequency, basilar membrane vibration grows in a compressive, or nonlinear fashion when the peak of the traveling wave arrives at the characteristic place along the partition tuned to that frequency. In this region, basilar-membrane displacement grows at about 0.2 dB/dB (Ruggero et al., 1997), implying that the mechanical nonlinearity determines cochlear dynamic range (Yates, Winter & Robertson, 1990).

When the basilar-membrane vibration amplitude at a single location is plotted as a function of frequency for a pure-tone stimulus, the data are represented as frequency tuning curves (FTCs). FTCs show that the mechanical response of the basilar membrane acts as a band-pass filter and has a high degree of frequency selectivity at low stimulus levels (Johnstone et al., 1986; Ruggero, 1992). Basilar membrane tuning curves have low-intensity sharply-tuned tips at the place of characteristic frequency, a steeply sloping high-frequency side, and a less-steep low-frequency edge. At moderate-to-high stimulus intensities, the more broadly tuned response shows that the tip of the FTC shifts towards lower frequencies (Baker, Rosen & Darling, 1998; Dallos, 1996; Johnstone et al., 1986). Intracellular recordings from mammalian inner and outer cells show that the shape of the FTCs for OHCs and IHCs corresponds to those of the mechanical response of the basilar membrane (Cheatham & Dallos, 1998, 2000; Russell & Sellick, 1978; Patuzzi & Robertson, 1988).

The majority of auditory nerve fibers innervate the IHCs, and their response reflects the vibratory patterns of basilar membrane displacement and the active amplification process of the traveling wave by the OHCs. Each auditory nerve fiber (ANF) is sensitive to a limited range of frequencies and intensities, and there are two dimensions of ANFs (and central neurons) that are important for understanding their response to increasing sound pressure level (Delgutte, 1996). The first corresponds to cochlear place, or that place along the cochlear partition that is best tuned to a particular or "characteristic" frequency (CF), and the second corresponds to their sensitivity or threshold.

When stimulated by pure tones, the response area of auditory nerve fibers may be characterized by their threshold as a function of tone frequency. Each nerve fiber is most sensitive to a single frequency; an increment in its firing (discharge) is just detectable at the lowest stimulus intensity at CF. When stimulation is repeated for different tone frequencies above and below CF, a frequency-threshold or tuning curve (FTC) is derived. These curves show that each auditory nerve fiber has a low threshold at one frequency (CF), and that threshold rises as stimulus frequency is changed. The V-shaped threshold-tuning curves of high-CF fibers (e.g., 4000 Hz) have low-intensity sharply-tuned tips at CF, steeply-sloping high-frequency edges, and less steeply sloping low-frequency tails; FTCs of low-CF fibers (e.g., 500 Hz) have more symmetric low- and high-frequency edges (Ruggero, 1992).

The second important feature of auditory nerve fiber response is threshold, which is related to their spontaneous discharge rate, or the rate of discharge in the absence of external stimulation. ANFs differ from one another in their spontaneous discharge rate, and this measure is predictive of their sensitivity to pure tones at CF (Kiang et al., 1965). Auditory nerve fibers (ANF) with high spontaneous rates (SR) of discharge have low thresholds at CF, and those with low SR have comparatively higher thresholds at the same characteristic frequency. High-SR fibers are likely used for detection of tones in quiet (Delgutte, 1996). The width of the tuning curve is smallest at low intensities at CF; at high stimulus intensities, the high-SR fibers are driven into saturation and their frequency resolving power deteriorates and is shifted toward lower frequencies. Low-SR fibers show essentially no change in their frequency resolving power at high intensities

(Møller, 1977). Thus, as stimulus intensity is increased, frequency resolution of the auditory nerve is preserved.

Several researchers have investigated the effects of increasing stimulus intensity on the discharge rate of primary and central auditory neurons in mammalian species (e.g., VIIIth nerve: Sachs & Abbas, 1974; Winter, Robertson & Yates, 1990; brainstem: Eggermont, 1989; Ehret & Merzenich, 1988; Irvine & Gago, 1990; Phillips & Burkard, 1998; Rhode & Greenberg, 1994a,b; Schoffner & Young, 1985; cortex: Heil, Rajan & Irvine, 1994; Phillips, Hall & Hollet, 1989; Rouiller, de Ribaupierre, Morel & de Ribaupierre, 1983; Suga & Manabe, 1982). The data from these studies provide description of the intensity dynamic range of auditory neurons for tonal stimulation in the form of rate-intensity functions. Pure-tone stimulation is always excitatory in VIIIth nerve fibers, thus their rate-intensity functions are always monotonic. Some central auditory neurons also show monotonic rate-intensity functions. That is, with increasing SPL, their average discharge rate increases approximately in proportion to the logarithm of sound pressure, but over a limited intensity range (Ruggero, 1992). At CF, low-threshold, high-SR primary auditory neurons show a saturating effect in response to high-intensity stimuli; their rate-level function at CF is sigmoidal in shape. Some high-threshold low-SR auditory nerve fiber rate-intensity functions, on the other hand, do not saturate in response to high-intensity stimuli (Winter et al., 1990); their rate-level functions have a comparatively less-steep slope and wide dynamic range (Ruggero, 1992), and probably reflect the nonlinear input-output functions of the basilar membrane mechanical response (Sachs & Abbas, 1974; Sellick, Patuzzi & Johnstone, 1983; Winter et al., 1990). For stimulus frequencies below

CF, rate-level functions are linear and thus steeper than at CF, while the response to frequencies above CF become progressively flatter with increasing frequency (e.g., Geisler, et al., 1974; Sachs & Abbas, 1974). Importantly, the dynamic range of most primary auditory neurons is only 30 to 40 dB SPL (Ruggero, 1992), and this is true also of most neurons at higher stages of the auditory system exhibiting monotonic rate-level functions (Clarey, Barone & Imig, 1992; Irvine, 1992), although larger dynamic ranges on the order of 60 to 80 dB have been recorded in some neurons in the VIIIth nerve (Geisler, 1998, pg. 190), medial geniculate body and cortex of mammalian species (e.g., Rouiller et al., 1983). The combined effects of high-, mid- and low-SR neurons and convergence of excitatory and inhibitory input at the higher loci of the auditory system are thought to contribute to the coding of intensity over the normal wide dynamic range (120-130 dB) estimated from psychophysical measures (Henderson & Salvi, 1998; Viemeister, 1988).

In central auditory nuclei, beginning at the level of the cochlear nuclei, many afferent responses are characterized by nonmonotonic rate-intensity functions. The degree of nonmonotonicity varies (Irvine & Gago, 1990), but generally there is an increase in discharge rate up to a certain sound pressure level (the "best SPL"), and a decrease in the total response at higher SPLs. These results are due to inhibition, which is the reduction in spontaneous or stimulus-evoked response by excitation of neighboring neurons. That is, with increasing intensity, inhibitory processes shape the discharge rates of these central neurons at CF as a result of stimulation from a neuron(s) of similar CF but with higher threshold (Wang, Salvi & Powers, 1996), from neurons of different CF whose response area for intense stimuli overlaps that of the input providing the nonmonotonic cell's CF

(Shofner & Young, 1985), and also from inhibitory inputs from the low- and/or high-frequency domains flanking the response areas of the neuron (Phillips, 1988). In addition, the cochleotopic representation of stimulus frequency in cortex is extended to include a wider array, or strip of cell units tuned to a particular frequency that lie orthogonal to the frequency axis. The increase in the number of excitatory cell units at this level combined with the multiple inhibitory processes that serve to shape the rate-intensity functions of some central auditory neurons indicates that the identity of the discharging elements and their respective discharge rates likely play as large a role in determining the way in which intensity is coded at these higher level stages as does the size of the discharging population. Evidence of an amplitopic, or place representation of stimulus intensity has been observed in the discharge patterns of the cat (Heil, Rajan & Irvine, 1994), the echolocating mustached bat (Suga & Manabe, 1982), and more recently in positron emission tomographic recordings in humans (Lockwood et al., 1999).

Physiologic models of intensity perception have been based mainly on Fletcher and Munson's (1933) hypothesis that loudness is proportional to the total discharge rate for the ensemble of auditory nerve fiber responses. The total discharge rate is dependent on both the number of excited fibers, and on the rate-level functions of individual fibers. Two classes or models of encoding sound pressure level have been advanced. The rate-place model is based mainly on the response of primary auditory neurons (e.g., Viemeister, 1988) in which the acoustic information is carried by the rate or frequency of the discharge of a neuron as a function of SPL and the spread of excitation across the population of fibers as SPL is increased, and recent findings support this hypothesis

(Cheatham & Dallos, 1998, 2000; Doucet & Relkin, 1997; Relkin & Doucet, 1997). However, because many central auditory neurons respond to sounds with single onset spikes or with complex temporal patterns (Brugge, 1992; Heil, 1997a,b), a rate-place code scheme is insufficient for resolving the problem of intensity coding. A second class of physiologic modeling of intensity coding is based on timing cues; phase-locking of auditory stimuli preserves the temporal information in both simple and complex low-frequency signals. This is limited, however, to low-frequency (i.e., <5000 Hz) CF fibers for pure-tone stimuli (Young & Sachs, 1979; Evans, 1981). More recently, Carney (1994) proposed a model based on both place and timing properties of neural responses, or their spatiotemporal discharge pattern, which refers to the simultaneous discharge of a population of auditory nerve fibers with different CFs. While not yet tested psychophysically, this model holds promise for explaining intensity coding because it incorporates the way in which both primary and central auditory neurons process changes in sound pressure level (Delgutte, 1996). Presently, however, the phenomenon of intensity encoding of sound pressure level is not completely understood (Carney, 1994; Delgutte, 1996, Irvine, 1992).

Physiology of intensity coding: Effects of masking

Broadband noise activates the entire basilar membrane because it contains all frequency components. The noise is filtered by the cochlear partition, and this action creates an inner-hair-cell receptor potential with a quasi-oscillatory waveform that resembles the waveform of a characteristic-frequency tone whose amplitude is randomly waxing and waning (Geisler, 1998, pg. 208). The properties of the synapse provide a

compressive stimulus-response curve that is similar to the FTC for a pure tone at low stimulus levels, and the FTCs of basilar-membrane motion widen as masker level increases (Evans, 1977; Geisler, 1998, pg. 210). Simultaneous presentation of a tone signal at levels just above masker level causes initial mechanical suppression of the tone and masker (Ruggero et al., 1992). Suppression of a signal by simultaneous presentation of a second sound is another manifestation of the mechanical nonlinearities of the cochlea (Patuzzi, 1996). Introduction of a second stimulus decreases the amplitude of the response to both stimuli (in this case, a tone and broadband noise); the basic characteristic of suppression is accounted for by the compressive nature of the OHCs' motile forces (Geisler, 1998, pp 151). As signal level increases, the interaction of tone and noise is altered, such that now the excitatory tone stimulates the region of the basilar membrane tuned to that particular frequency, but with the tip of its FTC shifted apically (Ruggero et al., 1992). The amplitude of the tone's response at suprathreshold levels, however, is reduced from its non-masked response even at the highest stimulus levels, due to suppression by the noise. In addition, for moderate-to-high intensity tones, the traveling wave becomes more like a passive traveling wave and the FTC is broadened, incorporating activity from a larger area of the cochlear partition. Thus, basilar membrane nonlinearities are reduced or eliminated, and the response to high-intensity stimuli is linear. This pattern of response is transmitted via the IHCs to primary auditory nerve fibers.

The sensitivity and response of auditory neurons to the SPL of pure tones presented simultaneously in broadband noise have been studied by many investigators (e.g., Abbas

& Sachs, 1976; Burkard & Palmer, 1997; Cooper, 1996; Costalupes, Young & Gibson, 1984; Geisler & Sinex, 1980; Gerken, 1991; Greenwood & Goldberg, 1970; Nuttal & Dolan, 1993; Phillips, 1990; Phillips & Cynader, 1985; Rees & Palmer, 1988; Rhode, Geisler & Kennedy, 1978; Rhode & Greenberg, 1994a,b; Smith, 1979). Peripheral mechanisms include excitation (via the "line-busy" effect), adaptation to the masker (which lowers sound-evoked responses), and suppression (masking without excitation) (Delgutte, 1990). Excitatory masking occurs when one stimulus, in this case a broadband masker, obscures or reduces the response of another ("line busy" or "strong signal capture" effect) by increasing the background rate of neural responses. Superimposed stimuli (i.e., tones) will add negligible activity of their own (Smith, 1979), and thus the response to the tones will be reduced. Adaptative masking also compresses neural responses by reducing the plateau (or saturated) rate of nerve fibers' responses (Ruggero, 1992). That is, when the tones are sufficiently strong to produce an increase in firing, their asymptotic firing rate (i.e., non-masked response) is reduced. Suppressive masking refers to the decrease in the neural response to a sound stimulus when another stimulus is introduced (Delgutte, 1990). In this case, the masker produces no increase in discharge rate over spontaneous activity, but shifts the rate-level function for the signal toward higher intensities, resulting in a threshold elevation (Delgutte, 1990). When a simultaneous wideband noise masker is used, the net effect is suppression of the response (Delgutte, 1992; Pickles, 1988, pp 103-106). The rate-level functions for ANFs in response to pure-tones in continuous noise generally show a reduced dynamic range, with a slope equal to or less steep than the non-masked response (Costalupes et al., 1984).

Costalupes et al. (1984) found that some high-threshold low-SR fibers that have comparatively broader dynamic ranges show some resistance to the noise masker (e.g., Costalupes et al., 1984; Sachs & Abbas, 1974); depending upon the SPL of the noise, the response of these cochlear afferents may have no effective net decrease in discharge rate for suprathreshold tones. It has been suggested that the convergence of such input to the cochlear nucleus and higher-level loci may result also in some resistance to saturation in central neurons (Phillips, 1987).

In central auditory nuclei, the rate-level function for tones in a noise mask differs, to some extent, from that of primary ANFs. For example, Rhode and Greenberg (1994a) found that the high-frequency slope of FTCs in some units of the cochlear nucleus (CN) increases in the presence of a background noise, indicating activation of inhibitory input. Inhibitory processes cause a shift in the dynamic range of some units in the CN, a truncation of the rate-level function to suprathreshold tones, known as "strong signal capture" (Geisler & Sinex, 1980), or suppression of response (Rhode & Greenberg, 1994b). Burkard and Palmer (1997) observed facilitation, or response enhancement, of chopper units in the cochlear nucleus in response to clicks presented in low-level wideband noise. Gerken (1991) and Rees and Palmer (1988) reported response enhancement, or an increase in discharge rate for units in the inferior colliculus (IC) in response to tone bursts masked by a continuous tone or broadband masker. Although most of the noise effects in the IC reflect peripheral mechanisms, these findings indicate that centrally-based processes serve to enhance detectability of increases in tonal sound pressure level in the presence of noise. Cortical neuron rate-level functions for tones in a

background of noise show little or no change in their basic discharge rate due to adaptation to the masking noise (Phillips, 1990, 1987; Phillips & Cynader, 1985). The main effect of wideband noise on the rate-level functions of cortical neurons is a shift of the dynamic range to higher SPLs (i.e., increased threshold); the magnitude of the shift increases with increasing noise SPL (Phillips & Cynader, 1985). Some cortical neurons show a steepening of the rate-intensity function in response to tones in wideband noise, presumably due to the fact that many afferent neurons of similar CF and varying thresholds are stimulated, thereby increasing the number of activated neurons for tones in noise compared to the response for tones of the same SPL presented in the quiet (Phillips, 1987; Phillips & Hall, 1986). In addition, the effective thresholds of cortical monotonic and nonmonotonic units to tones in a noise masker may be modified by adaptive, suppressive, as well as inhibitory mechanisms, so that a larger population of neurons are excited by suprathreshold masked tones (Phillips, 1987).

Loudness recruitment, as manifested by steeper loudness growth functions for tones in noise, is therefore likely a result of the composite processes underlying the neurophysiologic changes in the responses to tones in wideband noise, including linearization of the BM response, resistance of some neurons to the noise masker, as well as disinhibition in central neurons that cause signal enhancement at higher loci, although the details of where in the auditory system this response transformation occurs remain unresolved. Auditory evoked potential recordings, such as the ABR and MLR, that reflect neural activity from different levels of the auditory system pathway in response to

tones in noise likely would provide additional evidence about how and where masked signals are processed in the peripheral and central auditory system.

Physiology of intensity coding: Effects of cochlear impairment

The physiological basis and causes of cochlear injuries have been studied extensively (see Geisler, 1998; Patuzzi, 1996; Ruggero, 1992; Saunders, Cohen & Szymko, 1991). Cochlear hearing loss can result from a variety of different causes, including acoustic trauma, long-term exposure to high-level noise (Schmiedt, 1984), ototoxic drugs (e.g., Spongr, Boettcher, Saunders & Salvi, 1992), chemical solvents (Franks & Morata, 1995), endolymphatic hydrops (Harrison & Prijs, 1984), and genetic/hereditary causes, among others. Such factors produce initial damage in the cochlea, specifically the organ of Corti, which affects the mechanical transduction processes in inner and outer hair cells (Saunders et al., 1991), although the morphological changes resulting from the various etiologies differ.

The main consequence of IHC damage is elevated thresholds; frequency selectivity may remain relatively normal, but the spontaneous and driven discharge rates of ANFs are lower than normal (Liberman, 1978; Wang, Powers, Hofstetter, Trautwein, Ding & Salvi, 1997). Total destruction of IHCs results in a loss of spontaneous activity in the ANFs innervating them, and no response to suprathreshold stimuli even at very intense levels (Kiang et al., 1965). Loss or damage of the OHCs results in decrease or loss of the active amplification process (Geisler, 1998, pg. 301; Patuzzi, 1996). The consequences of OHC loss/damage are loss of sensitivity (elevated thresholds), loss of sharp tuning of FTCs (or broadened auditory filters), loss of compressive nonlinearities in the basilar

membrane input/output functions (Saunders et al., 1991), and absence or reduction of other nonlinearities such as two-tone suppression (e.g., Schmiedt & Zwislocki, 1980) and otoacoustic emissions (Eddins, Zuskov & Salvi, 1999).

The changes in peripheral auditory processing due to IHC and OHC loss/damage are thought to determine, in large part, the psychoacoustic performance of individuals with sensorineural impairment in their response to increasing signal SPL. Specifically, linearization of basilar-membrane motion and increased excitation resulting from stimulation of activity in the upper, wider portions of FTCs in response to suprathreshoid signals are thought to be the primary bases for the more linear and steeper loudness growth functions observed in sensorineurally-impaired persons (Geisler, 1998, pg. 301). In addition, recent reports indicate that changes in central auditory processing occur also as a result of cochlear impairment (e.g., Wang, Salvi & Powers, 1996), and that alteration of signal processing in central nuclei likely plays a role in determining the outcome of behavioral-based measures.

In cochlear pathology, the absolute threshold of cochlear afferents is raised, and their FTCs become more broadened (Evans, 1978). Based on the assumption that perceived loudness is related to the number of activated ANFs, Evans (1978) proposed that because the tuning curves in impaired cochleae are broadened and therefore overlap, moderate- to high-intensity tones stimulate a larger number of neurons than are stimulated in normal cochleae at CF. It is this abnormally rapid recruitment of nerve fiber response for suprathreshold stimuli that Evans (1978) suggested underlies loudness recruitment, a notion supported by both psychoacoustic tuning curve studies in cochlear-impaired

subjects (e.g., Thornton & Abbas, 1980), and evoked potential recordings of subjects with cochlear impairment, which reflect shorter latencies and a steepened intensity-latency function (e.g., Don et al., 1998; Galambos & Hecox, 1978). However, several psychophysical studies of normal and sensorineurally-impaired listeners (e.g., Hellman, 1978; Moore, Glasberg, Hess & Birchall, 1985) have demonstrated that the loudness of a pure tone is reduced when spatial spread of excitation is restricted. These results indicate that although spread of excitation is a neural consequence of cochlear pathology, it is not the main factor producing recruitment (Relkin & Doucet, 1997).

A second physiological model of loudness coding and recruitment is based on the rate-intensity functions of impaired cochlear afferents, which some researchers have shown are steeper at CF than those in normal cochleae (Evans & Klinke, 1982; Sachs et al., 1989; Schmiedt & Zwislocki, 1980; Yates, 1990). Cochlear-impaired ANF rate-level functions are shifted toward higher sound pressure levels and their dynamic range is reduced, but their maximum or peak firing rate is unchanged (e.g., Evans & Klinke, 1982). Thus, the steeper rate-level functions and maximum firing of neurons tuned to the test signal in impaired cochleae may be the neurophysiological basis of the changes in the loudness percept in persons with primarily cochlear hearing impairment (Phillips, 1987; Yates, 1990). More recent evidence indicates, however, that response recruitment is present in OHCs and exists at the level of a single hair cell, indicating that the basis for recruitment is entirely or in part due to partial or complete elimination of the active feedback mechanism in the cochlea (Zhang & Zwislocki, 1995). Loss of the active feedback mechanism causes linearization of the basilar membrane, which is thought by

many researchers to be responsible for recruitment (e.g., Moore, 1996; Pang & Guinan, 1997; Zhang & Zwislocki, 1995).

The effects of cochlear pathology on central auditory neurons have only recently been investigated in single- and multi-unit recordings in animals with compromised cochleae (Boettcher & Salvi, 1993; Gerken, 1992; Qui, Salvi, Ding & Burkard, 2000; Salvi, Wang & Ding, 2000; Syka & Rybalko, 2000; Syka, Rybalko & Popelar, 1994; Szczepaniak & Møller, 1996; Wang, et al., 1997; Wang, Salvi & Powers, 1996; Willott & Turner, 2000). A major finding from these studies is a central neuronal response "enhancement" which is thought to be due to disinhibition, or reduction in the inhibitory processes of neurons contributing to the normal neuron's response area (Gerken, 1993; Wang et al., 1996, 1997). Measures of the neural discharge patterns of central nuclei show an increase in their discharge rate, with nonmonotonic cells showing a greater increase in discharge than monotonic cells (Wang et al., 1996), and also an increase in the amplitudes of gross evoked potential recordings (cochlear nucleus: Boettcher & Salvi, 1993; inferior colliculus: Gerken, 1992; Wang et al., 1997; auditory cortex: Syka et al., 1994). For example, in their studies of the response patterns of auditory neurons in the inferior colliculus (IC) of the chinchilla, Salvi and colleagues (Qui, Salvi, Ding & Burkard, 2000; Salvi et al., 2000; Wang et al.,1996, 1997) found that, following exposure to intensive tonal stimulation, the FTCs of a majority of cells, including low-SR high-threshold fibers widen considerably, and that the discharge rate of nonmonotonic cells increases significantly. The data from these authors' pre- and post-acoustic trauma studies showed that damage to OHCs in the periphery reduces inhibitory effects, but the location of

inhibitory circuits is unknown; they may involve inputs from central nuclei such as the cochlear nucleus or inferior colliculus (Wang et al., 1996). Selective IHC loss reduces cochlear output, but the gross evoked potential recording in the IC shows only a modest amplitude reduction (Qui et al., 2000). The cortical response from primary auditory cortex, however, is enhanced suggesting a central auditory "gain factor" that may be regulated by an ascending or descending mechanism, but which compensates for decreased neural activity arising from the periphery due to cochlear pathology (Salvi et al., 2000). Syka and Rybalko (2000) found increases in the amplitudes of the middle latency response (MLR) in rats following exposure to intense noise. These authors suggested that the temporary threshold shift observed in their recordings is related to peripheral changes, and that MLR amplitude enhancement is "probably connected with a change in the processing of auditory information in the central nervous system." Indeed, Gerken (1993) suggested that the central mechanism(s) underlying response enhancement of gross evoked potential recordings in cochlear-impaired animals is/are possibly the same mechanism(s) that is/are activated in normal-hearing masking experiments that also show increased amplitudes for transient signals masked by a continuous tone (Gerken, 1991, 1992). He also proposed that the human MLR wave Pa recorded from subjects with cochlear impairment would likely show a response enhancement relative to the response from normal-hearing listeners. A review of the relevant findings led Gerken (1993) to suppose that the region of altered neural function for both factors (hearing loss and masking) is centered in the upper brain stem, but the actual site has yet to be determined. It may be that, while etiology of hearing loss differs, the central "gain factor"

may be a common neurophysiologic mechanism underlying the loudness recruitment seen in both masked normal-hearing and cochlear-impaired listeners.

Few data are available on the human MLR in sensorineurally-impaired (McFarland et al., 1977) or in noise-masked normal-hearing listeners (Gott & Hughes, 1989). Study of the changes in this early cortical response with intensity in comparison with those observed from brainstem recordings (ABR) obtained from these two groups of subjects is needed in order to address Gerken's (1993) supposition, the findings of which may serve to elucidate further the underlying mechanisms of intensity coding in hearing-impaired listeners. This issue is addressed in the present dissertation studies.

Chapter 3

Literature review of intensity effects on auditory evoked potentials

Auditory Evoked Potentials: An overview

Many different electrical potentials can be recorded from the human scalp in response to auditory stimulation, and each may provide important information about auditory function in relation to the loudness percept. Transient auditory evoked potentials (AEP) are most commonly employed in objective audiometry. A transient AEP is elicited by a change in the auditory stimulus, such as the onset or offset of a tone, or a change in its frequency or intensity (Picton & Fitzgerald, 1983). The transient AEPs used in clinical practice are onset responses that are elicited effectively by relatively short-duration stimuli (i.e. <50 ms) having fast rise/fall times, such as clicks or tonebursts, presented at a rate sufficiently slow that the response to one stimulus has finished before the next stimulus occurs (Regan, 1982). Because of their small amplitudes (i.e., <5 μV), evoked potentials can only be recognized by averaging several hundred responses in order to increase the signal-to-noise ratio (i.e., the ratio of response to background electroencephalographic activity). The assumption of averaging is that the evoked potential remains relatively constant, whereas the background noise is random (Picton, Linden, Hamel & Maru, 1983). Within a few milliseconds of stimulus onset, the earliest potentials appear and form a series of waves that extends for several hundreds of milliseconds. The characteristics (amplitude and latency) of the "exogenous" or stimulus-dependent (Hillyard & Picton, 1987) potentials are primarily defined by the physical characteristics of the stimulus, and are presumed to reflect neural activity from ascending portions of the auditory system (Squires & Hecox, 1983).

Auditory evoked potentials provide unique measures of auditory system integrity from various levels of the peripheral and central auditory system pathway that enable researchers to make inferences about the neurophysiological processes underlying human auditory perception. In addition, they can demonstrate the sequence of processes that lead to behavior, thereby allowing some measurement of when and where a disorder is occurring. The transient auditory evoked potentials commonly used in audiological assessment are categorized according to their generators and three primary time epochs: the auditory brainstem responses (ABR) (2-20 ms post-stimulus onset) which represent activity from the auditory nerve and brainstem structures (Allen & Starr, 1978; Durrant, Martin, Hirsch & Schwegler, 1994; Jewett, 1970; Legatt, Arezzo & Vaughan, 1988; Møller, 1994; Moore, 1987), the middle latency responses (MLR) (10-80 ms following stimulus onset) which represent activity from the auditory cortex and possibly subcortical structures (Hashimoto, Mashiko, Yoshikawa, Muzuta, Imada & Hayashi, 1995; Kuriki, Nogai & Hirata, 1995; Liégeois-Chauvel, Badier, Marquis & Chauvel, 1994; Scherg & von Cramon, 1986), and the cortical auditory evoked potential (CAEP) (50-250 ms post-stimulus onset) which represents activity from the primary auditory cortex, the temporal auditory association cortex, and possibly the frontal areas (Näätänen & Picton, 1987; Picton, Alain, Woods, John, Scherg, Valdes-Sosa, Bosch-Bayard & Trujillo, 1999; Picton, Hillyard, Krausz & Galambos, 1974; Scherg, 1990; Scherg & von Cramon, 1986; Woods, 1995).

In response to moderate-to-high stimulus intensities, the ABR is characterized by a series of vertex-positive and negative "fast" waves, reflecting action potentials,

superimposed on a slow wave component which reflects low-frequency dendritic activity (Legatt, Arezzo & Vaughan, 1988; Suzuki, Hirai & Horiuchi, 1977; Takagi, Suzuki & Kobayashi, 1985). The major positive peaks of the ABR are labeled by Roman numerals I-V after a classification scheme developed by Jewett and Williston (1971). The most robust positive peak is wave V, which occurs about 6 ms after the onset of a moderately-intense stimulus and has an amplitude of about 0.7 μV. In response to tones and low-intensity stimuli (clicks and tones), the ABR consists primarily of a "slow" positive peak, wave V, and the following negative trough, variously labeled SN10 (i.e., slow negative wave at 10 ms, Davis & Hirsh, 1979), or V' (Stapells & Picton, 1981).

The middle latency response to moderate-to-high stimulus levels is characterized by a sequence of slow negative and positive waves, reflecting dendritic potentials in the central auditory system (Hashimoto et al., 1995; Liégeois-Chauvel et al., 1994; Picton et al., 1974). The primary peaks of the MLR are labeled according to their vertex orientation (Positive or Negative) and arbitrarily in alphabetical order, such that wave Na is the first negative wave, Pa is the first positive wave, and Nb is the second negative wave; wave Na occurs approximately 20 ms following stimulus onset, wave Pa occurs at about 30 ms following stimulus onset, and wave Nb is seen within the 40-60 ms time frame after onset of a moderately-intense stimulus. Wave Pa has an amplitude of about 1.2 μV, and is the most robust waveform of the MLR complex (Kraus et al., 1994).

The cortical auditory evoked potential is characterized by a series of positive-negative slow waves, reflecting low-frequency EEG activity and postsynaptic potentials (Davis & Yoshie, 1963; Picton et al., 1974). The peaks of the CAEP are labeled also according to

their polarity at the vertex (Positive-Negative) and to their placement (e.g., 1, 2) and/or their timing with respect to stimulus onset. Thus, wave P1 (or P50) is the first positive peak occurring at about 50 ms post-stimulus onset. This wave is followed by a robust negative wave, N1 (or N100 for the fact that it occurs about 100 ms post-stimulus onset), which has an amplitude of about 4.0-10.0 μV for a moderately-intense stimulus (Davis, Mast, Yoshie & Zerlin, 1966; Picton et. al., 1974). Wave P2, or P200, is the second positive peak that occurs about 200 ms after stimulus onset in response to clicks, tones, and speech stimuli presented at moderate-to-high stimulus levels (Davis et. al., 1966; Picton, Woods & Proulx, 1978).

Each of the above evoked potential measures has been advocated for use in predicting hearing thresholds, primarily in the neonatal and infant populations, and also in older patients who are unable or unwilling to participate in basic audiometric tasks (e.g., ABR: Stapells, Picton & Durieux-Smith, 1994; MLR: Kraus, McGee & Stein, 1994; CAEP: Hyde, 1997; Stapells, in press). In practice, AEP thresholds are typically estimated from making present/absent judgements on response averages obtained at a few stimulus levels. Similar to conventional behavioral techniques (Carhart & Jerger, 1959), a bracketing procedure is used, and threshold is based on detectability, or the lowest stimulus intensity that yields a present response (Hyde, 1994). With increasing sensation level, AEP amplitudes increase and their latencies decrease (ABR: Davis & Hirsh, 1976; Eggermont & Don, 1980; MLR: Thornton, Mendel & Anderson, 1977; CAEP: Picton, Woods & Proulx, 1978). From these data, intensity-amplitude/latency functions provide an index of the rate of change in the amount and timing of neural activity at the various

stages of the auditory pathway as signal level is increased. One or both of these AEP input-output functions may relate to the loudness growth function obtained from subjective magnitude estimates, but it remains unclear whether or not the values obtained from the two methods provide measures of similar or different processes (Picton et al, 1978). It has been demonstrated, for example, that the ratio between onset neural activity and steady-state (i.e., adapted) neural activity is constant over a wide intensity range (Smith & Zwislocki, 1975). This suggests that loudness (which is proportional to steady-state neural function, as measured in conventional audiometric procedures using long-duration tones) may be related to AEP amplitude (which is proportional to onset neural activity) (Eggermont, 1983; Smith, 1988). Study of the relation of AEP amplitude growth and latency decrease to psychophysical measures of sensory magnitude is the focus of this dissertation, and the relevant literature to date regarding each of the transient evoked potentials and loudness will now be reviewed.

Cortical Auditory Evoked Potentials (N1-P2) and Loudness

The advent of electric response audiometry occurred in the mid-1960s, shortly after S. S. Stevens proposed his general power law which quantified input-output relations in sensory psychology. The slow cortical auditory evoked potential, which was the first electrical response at the time that was seen clearly using electronic averaging and summating techniques (Davis & Yoshie, 1963), was subsequently examined by many researchers in an effort to find values of the size of the N1-P2 complex versus stimulus intensity that were in accord with the values established from psychophysical data (Antinoro, Skinner & Jones, 1969; Beagley & Knight, 1967; Botte, Bujas & Chocholle,

1975; Butler, Keidel & Spreng, 1969; Butler, Spreng & Keidel, 1969; Clayton & Rose, 1970; Davis, Bowers & Hirsh, 1968; Davis & Zerlin, 1966; Henry & Teas, 1968; Keidel & Spreng, 1965; Knight & Beagley, 1969; Onishi & Davis, 1968; Picton, Goodman & Bryce, 1970). Electrophysiologists substituted the magnitude of the evoked potential (i.e., amplitude) for sensory magnitude in Stevens' formula, $\psi=k\phi^c$, and measured the dynamic range for various sensations (e.g., pain, vision, vibration, audition) which they then compared to the perceptual results (see Keidel, 1976).

The early findings of the auditory evoked potential recordings indicated fair agreement between the exponents obtained for amplitude growth of the CAEP and loudness (Davis & Zerlin, 1966; Keidel & Spreng, 1965; Knight & Beagley, 1969). For example, Keidel & Spreng (1965) obtained an exponent value of 0.36 for the CAEP intensity-amplitude function derived from recordings to 1000-Hz tones presented at a rate of 0.03/s, which they suggested compared favorably with Stevens' (1959) psychophysical results. Davis & Zerlin (1966), on the other hand, recorded the cortical auditory evoked potential to 2400-Hz tones presented at a rate of 3.2/s from six normal-hearing subjects, and obtained a mean amplitude exponent of 0.24 (re: dBSPL), indicating a smaller slope and exponent for the CAEP compared to the established results for loudness (i.e., 0.60; Stevens, 1959). In addition, these authors reported the results of electrical response recordings to tactile stimuli (300-Hz tones presented at 1/s) obtained from 20 deaf children, which also gave slope averages in double logarithmic coordinates that were "much flatter than the loudness function" (Davis & Zerlin, 1966). Reports from other studies confirmed this difference in exponent value between the amplitudes of the slow cortical potential and

loudness growth (Botte et al., 1975; Henry & Teas, 1968; Onishi & Davis, 1968; Rau, 1968), but a general consensus at the time was that there was "similarity between the amplitude of the evoked response and the loudness function" (Knight & Beagley, 1969), and that Stevens' power law is successfully applied to electric response audiometry (Keidel, 1976).

Other experimental results from this early period provided further evidence in support of the notion that the amplitude of the slow cortical evoked potential relates to the loudness percept. For example, the amplitude of the CAEP to binaural stimulation is larger than that to monaural stimulation (Butler, Keidel & Spreng, 1969; Davis & Zerlin, 1966). Davis & Zerlin (1966) found that an increase of 6 dB in stimulus level presented monaurally gave voltage values that were equal to those obtained to the lower level stimulus presented binaurally. These findings are in agreement with psychophysical results re: the difference (6 dB) in stimulus level that produces equal loudness for monaural vs binaural stimulation (Reynolds & Stevens, 1960). Also, recordings of the CAEP with intensity from subjects with hearing loss gave steeper intensity-amplitude functions compared to the normal response (Clayton & Rose, 1970; Davis, Bowers & Hirsh, 1968; Henry & Teas, 1968; Knight & Beagley, 1969; Shimizu, 1968; Uziel & Seneclaus, 1978), similar to behavioral measures of loudness growth (e.g., Hellman & Zwislocki, 1964; Miskolczy-Fodor, 1960). This was true also for the masked cortical auditory evoked potential recorded from normal-hearing subjects (Davis et al., 1968), and for subjects with sensorineural hearing loss (Clayton & Rose, 1970; Henry & Teas, 1968; Knight & Beagley, 1969; Shimizu, 1968; Uziel & Seneclaus, 1978). Knight & Beagley

(1969), for example, reported good agreement between the growth in loudness measures obtained using the ABLB from subjects with unilateral sensory loss, and the growth in amplitudes of the slow cortical evoked potential recorded to 20-ms 1000-Hz tones presented at a rate of 0.125/s. Others reported that the slope of the N1 intensity-amplitude function increases with increasing hearing loss (Davis et al., 1968; Uziel & Seneclaus, 1978), a finding also seen in the psychophysical results (e.g., Miskolczy-Fodor, 1960). The latter results are especially pertinent, because a major goal of electric response audiometry is its application as a useful "objective" measure of auditory sensation and perception for clinical purposes in the diagnosis and treatment of persons with hearing loss.

In addition to changes in stimulus intensity, the slow cortical auditory evoked potential is affected by other stimulus parameters, including repetition rate, stimulus envelope, and frequency (see Hyde, 1997 for review). The early studies that investigated the effects of manipulating these parameters on the CAEP reported some findings that reveal a dissociation between the slow cortical auditory response and the loudness percept (e.g., Antinoro et al., 1969; Beagley & Knight, 1967; Butler, 1968; Butler, Spreng & Keidel, 1969; Davis et al., 1966; Keidel & Spreng, 1965; McCandless & Lentz, 1968; Onishi & Davis, 1968; Picton et al., 1970, 1978; Rapin, Schimmel, Tourk, Krasnegor & Pollak, 1966; Skinner, Antinoro & Shimota, 1972; Tyberghein & Forrez, 1969). For example, the amplitude of the N1 is inversely related to stimulus repetition rate, such that the response is maximal at rates of 0.1/s or less, and declines with increasing repetition rate by as much as 75% (e.g., 2/s) of its maximum amplitude (Butler et al., 1969; Davis et al.,

1966; Picton et al., 1970). Repetition rates of 0.5 to 1.0/s are commonly used, however, providing a reasonable compromise between response amplitude and efficiency of data collection (Hyde, 1997). Increasing repetition rate also causes a decrease in *rate* of amplitude growth with intensity (Rau, 1968), which is the opposite effect from that seen for behavioral loudness growth measures (Darling & Price, 1989; Garner, 1948; Picton et al., 1978; Zwicker & Fastl, 1999). Also, N1 amplitude saturates at high stimulus intensities (e.g., 80-90 dBSL) for relatively rapid repetition rates (e.g., 2/s) (Picton et al., 1970; Butler et al., 1969), whereas loudness continues to increase up to 120 dBSL (Fletcher & Munson, 1933). The amplitude of the N1 also varies with stimulus frequency independent of any change in loudness, such that high-frequency tones evoke smaller responses than low-frequency stimuli of equal loudness (Antinoro et al., 1969; Picton et al., 1978; Rapin et al., 1966). These frequency effects are due, in part, to the position and orientation of the generator sources contributing to this gross evoked potential (e.g., Elberling, Bak, Kofoed, Lebech & Saermark, 1982; Jacobson, Lombardi, Gibbens, Ahmad & Newman, 1992).

The cortical auditory evoked potential is an obligatory response elicited maximally over frontal cortex, with the largest response observed at the vertex (Picton et al., 1974). Recent investigations of source activity underlying the scalp-recorded CAEP indicates that several concurrent sources contribute to both the N1 and P2, which are now known to be two distinct events (Picton et al., 1999). The N1 is composed of at least three components, and possibly more, arising from several neural sources in the temporal and frontal lobes (Näätänen & Picton, 1987; Picton et al., 1999; Scherg, 1990; Wood &

Wolpaw, 1982; Woods, 1995), but it is not yet clear whether its major source is localized (e.g., Woods, 1995) or represents a widespread activation of auditory areas (Picton et. al., 1999). The most robust negativity that occurs at about 100 ms post-stimulus onset (N1) derives from generators in the supratemporal plane, probably in or slightly posterior to the primary auditory cortex (Picton et al., 1999), with maximal source activity arising from the temporal lobe contralateral to the ear of stimulation. It has been established that the frequency effects on the N1 noted above are due to differences in the orientation and position of the sources that are activated for low- vs high-frequency stimuli (Elberling et al., 1982; Jacobson et al., 1992; Picton et al., 1999). The functional significance of each sub-component of the N1, however, is not completely understood. It has been postulated that the N1 (and possibly P2) may reflect detection of sensory information (Hillyard & Kutas, 1983), formation of a sensory memory of a stimulus, or possibly a widespread transient arousal which facilitates sensory and motor response to the stimulus (Näätänen & Picton, 1987). Because the CAEP is recorded using standard EEG electrodes that are relatively few in number, experimental and clinical paradigms generally do not allow examination of each of the several components that contribute to this evoked potential.

The CAEP was the first auditory evoked potential used to estimate hearing sensitivity, and many studies reported good agreement between the cortical auditory evoked potential and behavioral pure-tone thresholds (e.g., Beagley & Kellog, 1970; Davis, Hirsh, Shelnutt & Bowers, 1967; Hyde, Alberti, Matsumoto & Li, 1986; Jones, Harding & Smith, 1980; McCandless & Best, 1966; McCandless & Lentz, 1968; Prasher, Mula & Luxon, 1993; Suzuki & Origuchi, 1969; Taguchi, Picton, Orpin & Goodman, 1969). The pure-tone

audiogram can be closely approximated, in part because the toneburst envelope parameters used to obtain the CAEP are relatively frequency-specific. N1-P2 amplitudes are constant for stimulus rise/fall times of up to 30 ms and remain stable for durations longer than 30 ms (Onishi & Davis, 1968). Maximum response amplitudes may be generated for a given stimulus level using a 5- to 10- cycle rise/fall time and 30 ms duration (Hyde, 1997), and such stimuli are effective in eliciting the CAEP with the advantage that spectral splatter is greatly reduced. The slow cortical evoked potential, however, is significantly affected by subject state, including level of arousal, as well as cognitive processes of attention, expectation, and stimulus evaluation (Picton, Hillyard & Galambos, 1976). Amplitudes are reduced and waveform morphology changes with decrease in subject/patient's alertness, and during sleep and barbiturate anesthesia (Celesia & Puletti, 1971; Williams, Tepas & Morlock, 1962). This is the primary reason why the CAEP was supplanted by the emergence of the ABR in the 1970s as the AEP of choice for clinical use in obtaining estimates of hearing threshold in infants and young children who are unable or unwilling to remain passive and/or alert during evaluation. Unlike the N1-P2, the ABR is unaffected by subject state (Picton et. al., 1977). Measurement of the N1-P2, however, remains a central tool in current research endeavors that focus on other suprathreshold psychoacoustic measures, such as masking level differences (Fowler & Mikami, 1992), and speech-sound processing (Martin & Boothroyd, 2000). As this review shows, N1-P2 amplitude is strongly correlated with loudness, although normative data for clinical application have not, as yet, been established.

Middle Latency Response and Loudness

One early study investigated whether or not the power function for the amplitude of the middle latency response correlated with the power function for loudness (Madell & Goldstein, 1972). These authors obtained MLR recordings and magnitude estimates to clicks presented at 9.6/s from 24 normal-hearing subjects. The power functions for MLR amplitudes Po-Na, Na-Pa, and Pa-Nb yielded mean exponent values of 0.11, 0.09, and 0.07 respectively, which did not correlate with the mean loudness exponent of 0.28 obtained from subjective magnitude estimates. However, these authors found significant correlations between the individual subjects' absolute amplitudes and their loudness estimates, and concluded that a possible physiologic relation exists between the two phenomena. They cautioned that the normal individual variability found in their subjects' MLR recordings may preclude applying these measures for clinical determination of pathology.

As might be suggested from the foregoing review of the only previous study that examined the relation of the MLR to psychoacoustic measures of loudness, the body of literature on the effects of intensity on the MLR is much smaller than that for either the slow cortical auditory evoked potential or, as will be seen in the next section, the brainstem response. Indeed, although the MLR has been investigated as a potential tool for objective measurement of hearing for more than 30 years (Hall, 1992; Kraus et al., 1994), clinical exploitation of this electrophysiologic response has been limited (e.g., Jerger, Oliver & Chmiel, 1988) for a number of reasons that prompt a somewhat more expanded review of this evoked potential than is provided for the CAEP and ABR. As

will be discussed below, factors contributing to the lack of widespread use of this evoked potential include, among others, initial controversy over whether or not the MLR is of myogenic or neurogenic origin (Bickford, Jacobson & Cody, 1964; Harker, Hosick, Voots & Mendel, 1977), conflicting findings regarding its presence/absence in the pediatric population (Mendel, Atkinson & Harker, 1977; Stapells, Galambos, Costello & Makeig, 1988; Suzuki & Hirabayashi, 1987), subsequent findings regarding the influence of filtering and filter roll-off slopes on the components of the MLR and their amplitudes and latencies (e.g., Kraus, Reed, Smith, Stein & Cartee, 1987; Scherg, 1982a; Suzuki Kobayashi & Hirabayashi, 1983; Suzuki, Hirabayashi & Kobayashi, 1984), and, for much of this period, lack of firm evidence and controversy in defining the neural generators contributing to this response (e.g., Fischer, Bognar, Turjman & Lapras, 1995; Liégeois-Chauvel, Badier, Marquis & Chauvel, 1994).

The MLR is produced from activation of spatially overlapping but distinct neuronal subpopulations within the auditory cortex (e.g., Liégeois-Chauvel et al., 1994). The position and orientation of the dipole sources contributing to this response have been determined from scalp distribution studies (e.g., Cacace, Satya-Murti & Wolpaw, 1990; Goff, 1977; Goff, Matsumiya, Allison & Picton et al., 1974; Wood & Wolpaw, 1982), epidural and direct cortical recordings in humans and animals (e.g., Celesia, 1976; Hashimoto, 1982; Liégeois-Chauvel et al., 1994; McGee, Kraus, Comperatore & Nicole, 1991), magnetic recordings (e.g., Hashimoto, Mashiko, Yoshikawa, Mizuta, Imada & Hayashi, 1995; Kuriki, Nogai & Hirata, 1995; Scherg et al., 1990), and from case studies of patients with cortical and subcortical lesions (e.g., Fischer, et al., 1995; Ibañez, Deiber

& Fischer, 1989; Kileny, Paccioretti & Wilson, 1987; Scherg & Von Cramon, 1986; Waring, Ponton & Don, 1999; Woods, Clayworth, Knight, Simpson & Naeser, 1987). Generally, findings indicate that the sources of middle latency responses are in the primary auditory pathway in primary auditory cortex. These sources are distributed from medial to lateral sites on the supra temporal plane. Specifically, wave Na reflects activity primarily from a source located at or near the postero-medial part of Heschl's gyrus in primary auditory cortex, and possibly some activity from the midbrain (subcortical) (Fischer et al., 1995; Hashimoto et al., 1982; 1995; McGee et al., 1991). Wave Pa reflects activity originating in the lateral part of Heschl's gyrus in primary auditory cortex (e.g., Hashimoto et al., 1995; Scherg et al., 1990). Evidence from animal studies (e.g., Kraus, Smith & McGee, 1988), lesion studies (e.g., Kaga, Hink, Shinoda & Suzuki, 1980), and a recent study of two subjects with auditory brainstem implant (stimulating the cochlear nucleus) (Waring, Ponton & Don, 1999) indicates that the MLR may also derive from contributions and interactions of activity in non-specific auditory pathways. Non-primary pathways include intralaminar, midline, and reticular nuclei of the thalamus that project to widespread cortical areas (Steward, 2000, pg. 488). They are multimodal, broadly tuned, and show only mild binaural interaction (i.e., enhanced amplitude to binaural stimulation vs monaural stimulation) (see Kraus & McGee, 1995 for review).

During the first 15 years in which research on the MLR was conducted, many researchers investigated the effects of stimulus and recording parameters and subject state on this evoked potential (e.g., Galambos, Makeig & Talmachoff, 1981; Goldstein, Rodman & Karlovich, 1972; Madell & Goldstein, 1972; McFarland, Vivion & Goldstein,

1977; McFarland, Vivion, Wolf & Goldstein, 1975; Mendel et al., 1977; Mendel & Goldstein, 1969; Mendel, Hosick, Windman, Davis, Hirsh & Dinges, 1975; Mendelson & Salamy, 1981; Picton et al., 1974; Skinner & Antinoro, 1971; Thornton, Mendel & Anderson, 1977; Vivion, Hirsh, Frye-Osier & Goldstein, 1980). Studies showed that the transient MLR is maximally recorded for stimulus repetition rates from 1 to 15/s (Goldstein et al., 1972; McFarland et al., 1975); at higher rates, MLR amplitudes decrease and latencies increase (McFarland et al., 1975; Picton et al., 1974), but there is an exception to this general finding. At presentation rates greater than 30/s, a steady-state response (SSR) is produced, such that there is an overlapping of the responses to successive stimuli resulting in enhanced amplitudes (Galambos et al., 1981; Stapells, Linden, Suffield, Hamel & Picton, 1984; Stapells & Picton, 1981). The largest enhancement of the MLR occurs for presentation rates near 40/s, and this MLR has been labeled the "40-Hz auditory evoked potential" or MLR-Steady State Response (Galambos et al., 1981; Galambos & Makeig, 1992). The MLR-SSR derives from the superimposition of successive negative and positive peaks of the response which, at 40/s, occur in phase and add together to produce a response with a larger amplitude (Stapells et al., 1984a, 1988). Presentation rates of ≤15/s may be used to elicit the transient click- or tone-evoked MLR (Picton et al., 1974; Thornton et al., 1977). Brief acoustic transients with rise/fall times of 5 ms or less have been shown to be the most effective stimuli for recording the MLR (Lichtenstein & Stapells, 1997; Vivion et al., 1980). Stimulus frequency effects on the MLR are similar to those found for the slow cortical and brainstem responses such that amplitudes and latencies increase with decreasing signal

frequency (McFarland et al., 1977; Oates & Stapells, 1997a,b). In an early report, Mendel et al. (1975) demonstrated that the latencies and amplitudes of the MLR are altered as a function of sleep state, and later reports confirmed that latencies are prolonged and amplitudes decrease during sleep stages 2-4 (after Rechtschaffen & Kales, 1968; e.g., Kraus, McGee & Comperatore, 1989; Osterhammel, Shallop & Terkildsen, 1985). Most of the early studies cited above used narrow (e.g., 25-175 Hz) EEG filter settings and steep (e.g., 48 dB/octave) roll-off slopes, recording parameters that were later found to cause distortion of the MLR waveform (e.g., Scherg, 1982a).

Technical reports published in the early 1980s regarding the frequency content of the MLR and the acquisition parameters that influence the amplitudes and latencies of this response led to renewed efforts for defining/determining the appropriate recording techniques for optimizing this evoked potential for clinical application. Suzuki and colleagues (Suzuki, Kobayashi & Hirabayashi, 1983), for example, demonstrated from their spectral analysis of the MLR that the main spectral energy of the adult response to 85 dB peSPL clicks is between 30 and 50 Hz, with peak response at 40 Hz, and also some energy in the 90 to 180 Hz range. These researchers and others examined the effects of bandpass filtering on this low-frequency electrophysiologic response, and demonstrated that high-pass filtering produces significant changes in MLR amplitudes and latencies such that increasing high-pass filtering from 1 to 40 Hz shortens Na, Pa, and Nb latencies and decreases the amplitudes of these major components (e.g., Kraus, Reed, Smith, Stein & Cartee, 1987; Scherg, 1982a; Suzuki, et al., 1983a; Suzuki, Hirabayashi & Kobayashi, 1984). These decrements in latency and amplitude are due to phase shifting produced by

the analog filtering as well as the elimination of low-frequency contributions to the response (Scherg, 1982a,b; Suzuki et al., 1983a, 1984b).

Low-pass filtering also produces significant effects on the MLR (Kileny, 1983; Scherg, 1982a). Phase shifting of the response by analog filtering is seen when the low-pass filter is reduced to 200 Hz or less causing an increase in the latencies of waves Na, Pa, and Nb (Scherg, 1982a,b). In addition, use of low-pass filters below 200 Hz with steep analog-filter slopes (e.g., 48 dB/octave) distorts the MLR waveform and artificially produces new components in the response following wave V/Na (Kileny, 1983; Scherg, 1982a). The results of these technical reports helped to resolve the outstanding question of whether or not the MLR is present in the pediatric population which arose from conflicting reports published early on (e.g., Mendel et al., 1977; Mendelson & Salamy, 1981; Sprague & Thornton, 1982). The positive findings of clear-cut MLRs obtained in infants and young children reported by Mendel et al. (1977) and Mendelson & Salamy (1981), for example, were a result of their use of narrow bandpass settings (20-175 Hz) and steep roll-off slopes (24 dB/octave). Later reports from studies that employed wide bandpass filter settings (e.g., 20-2000 Hz), low high-pass filter settings (e.g., 10 Hz), and shallower (e.g., 6 dB/octave) roll-off slopes showed a lack of consistency in obtaining the MLR in infants and young children (e.g., Kraus, Smith, Reed, Stein & Cartee, 1985; Sprague & Thornton, 1982; Stapells et al., 1988). This is due to the more restricted frequency content of the infant MLR (Suzuki et al., 1983b) and to the absence of this response in infants during sleep stages 2-4 (Kraus, McGee & Comperatore, 1989), possibly reflecting immaturity in their central auditory system (Kraus & McGee, 1992).

Suzuki et al. (1983) reported that 85 to 90% of infants and young children (aged 1-7 years, N = 26) had a detectable Pa for 60 dB nHL 1000-Hz tones only when a high-pass filter of 20 Hz was used; a lower (10 Hz) or higher (30 Hz) high-pass filter setting reduced wave Pa detectability to 50% or less in this younger group. Later studies also demonstrated that the MLR may be recorded in some infants and young children during certain stages of natural sleep (Kraus, McGee & Comperatore, 1989; Tucker & Ruth, 1996), mildly sedated sleep (Suzuki et al., 1983b), and during wakefulness (McPherson, Tures & Starr, 1989), but its absence in some normal babies precludes its use in clinical assessment of this young population.

The MLR has been advocated as an adjunct to the ABR for estimation of hearing threshold (Harker & Backoff, 1981; Kraus & McGee, 1990; Scherg & Volk, 1983) and/or as an alternative test of hearing sensitivity when the brainstem response is absent (e.g., Kavanaugh, Domico, Crews & McCormick, 1988; Kraus, Özdamar, Stein & Reed, 1984). Some researchers have suggested that the tone-evoked MLR yields more frequency-specific measures than the ABR (Kraus & McGee, 1990; Musiek & Donnelly, 1983; Scherg & Volk, 1983), but recent findings have shown that this is not the case (Mackersie, Down & Stapells, 1993; Oates & Stapells, 1997a,b; Wu & Stapells, 1994). That is, there are no significant differences in the frequency selectivity/specificity between the ABR and MLR for low- and high-frequency tones of low and moderate intensity. Most studies investigating the accuracy of tone-evoked MLRs for behavioral threshold estimation have recorded this response to 500-Hz tones from normal-hearing subjects (e.g., Barajas, Exposito, Fernandez & Martin, 1988; Kavanagh, Harker & Tyler,

1984; Kileny & Shea, 1986; McFarland et al., 1977; Musiek & Geurkink, 1981; Scherg & Volk, 1983; Thornton et al., 1977; Wu & Stapells, submitted). These authors found good agreement between MLR threshold estimates and behavioral pure-tone thresholds for 500-Hz tones. There are few reports, however, on the accuracy of the MLR for threshold estimation to frequencies other than 500 Hz in normal-hearing adults (McFarland et al., 1977; Thornton et al., 1977; Wu & Stapells, submitted; Xu, De Vel, Vinck & Cauwenberge, 1995) and children (Kavanaugh et al., 1984), or in hearing-impaired listeners (McFarland et al., 1977; Suzuki et al., 1981; Xu, et al., 1995). Such studies are needed for clinical application of this evoked potential.

MLR intensity series to tonal stimuli have been reported for normal listeners (Scherg & Volk, 1983; Thornton et. al., 1977; Wu & Stapells, submitted) and hearing-impaired listeners (McFarland et al., 1975; Suzuki et al., 1981), but the findings from the early studies are compromised by the use of inappropriate EEG bandpass filter settings (Thornton et al., 1977; McFarland et al., 1977) and included data from subjects with conductive and sensorineural hearing loss of diverse degree and configurations (McFarland et al., 1977; Suzuki et al., 1981), and the later studies recorded the tone-evoked MLR to a limited range of intensities above threshold (Scherg & Volk, 1983; Suzuki et al., 1981; Wu & Stapells, submitted). There does not appear to have been a *systematic* study of the rate of amplitude growth/latency decrease of the tone-evoked MLR in normal-hearing or hearing-impaired subjects, nor is there a comparative study of this electrophysiologic measure and psychoacoustic measures of magnitude perception in hearing-impaired individuals. Such findings would add substantially to the paucity of

data available on the MLR, and to our knowledge of how sound intensity is processed at this early cortical level of the auditory system.

Auditory Brainstem Response and Loudness

The auditory brainstem response has been compared to loudness measures using clicks as stimuli (Darling & Price, 1990; Howe & Decker, 1984; Pratt & Sohmer, 1977; Serpanos et al., 1997; Wilson & Stelmack, 1982). Most of this research has been done in normal listeners and, generally, the limited and largely negative findings/conclusions reported to date relating the ABR and loudness contrast with both the amount of data available on the CAEP and loudness, and the view that the neural activity recorded at the scalp does provide a measure of processes involved in loudness perception (e.g., Keidel, 1976).

Studies of the ABR and magnitude estimates reveal that the amplitude and latency changes with intensity may be described by a power function, but findings consistently show a slower growth in amplitude and decrease in latency compared to the function obtained from psychoacoustic observations (Howe & Decker, 1984; Pratt & Sohmer, 1977; Serpanos, et al., 1997; Wilson & Stelmack, 1982). Pratt & Sohmer (1977) obtained brainstem recordings and magnitude estimates to clicks presented at a rate of 10/s from 22 normal-hearing adults. These authors concluded that, although the exponents derived from the intensity-amplitude functions were similar to the loudness exponents, both for individual subjects and for the group, the greater inter-subject and inter-session variability observed in the subjective estimates compared to the stability of the brainstem response suggested lack of a true correspondence between measures. In addition, the wave V

intensity-latency exponent (-0.03) correlated poorly with the exponent of 0.26 derived from magnitude estimates. Wilson and Stelmack (1982) obtained comparable findings from their investigation of the amplitudes and latencies of the click-evoked ABR and subjective magnitude estimates in 36 normal-hearing subjects, and supported the conclusion proposed by Pratt & Sohmer (1977) that the electrical response does not represent the neural components involved in loudness estimation.

More recently, Serpanos et al. (1997) examined wave V latency as a function of intensity and behavioral loudness measures obtained from magnitude estimation/production procedures from 10 normal-hearing adults and two groups of hearing-impaired subjects; 10 of these adults had flat configuration sensorineural hearing loss, and 10 subjects had high-frequency (above 1000 Hz) sensorineural hearing loss. The click-evoked ABR was recorded using a presentation rate of 61/s, and magnitude estimates to 1-minute presentations of the same stimuli were made. These authors obtained mean intensity-latency exponents of -0.04 from the normal listeners and -0.05 from each group of impaired listeners, which were significantly different from the behavioral loudness exponents of 0.51, 1.10, and 0.58 obtained from the normal-hearing, impaired-flat configuration, and impaired-high-frequency-loss groups, respectively. Serpanos et al. found significant correlations between wave V latency and loudness for the normal-hearing subjects and the impaired listeners with flat configuration of hearing loss, but not for the group with high-frequency hearing loss. The findings for the normal listeners are consistent with the results from the earlier reports (Pratt & Sohmer, 1977; Wilson & Stelmack, 1982), and together with the results from the impaired listeners,

indicate that click-evoked ABR latency, which varies with intensity, is also dependent on configuration of hearing (loss). Serpanos et al. (1997) noted that the lack of a significant relationship between wave V latency and loudness in the group with high-frequency hearing loss is likely due to the use of clicks, and that "ABR recordings elicited using frequency-specific stimuli may be more useful".

Howe & Decker (1984) investigated the relation between ABR latency and magnitude estimates to clicks for monaural and binaural stimulation from 10-12 normal-hearing adult subjects in three experimental procedures. They reasoned that, because the ABR to binaural stimulation is larger than the monaural response, this difference would be related to the difference between binaural and monaural measures of loudness growth that have been demonstrated in psychophysical studies which show that the loudness of tones presented binaurally grows at a faster rate compared to monaural listening (Reynolds & Stevens, 1960). Binaural loudness summation was observed in the exponents obtained from a loudness matching procedure wherein subjects were asked to match the loudness of 500-ms trains of clicks (rate: 22/s) presented binaurally at four intensities, 20-80 dBSL (re: monaural threshold) to the loudness of the clicks presented monaurally. Comparison of the monaural vs binaural wave V intensity-latency functions, however, revealed no significant difference between the exponents of the monaural (-0.02) and binaural (-0.02) brainstem responses. These authors concluded that the brainstem response does not reflect directly the processes of loudness summation or psychophysical experiences of loudness growth. Latencies of the binaural responses, however, were shorter than either of the monaural responses as seen in their Figure 5, suggesting that the amplitudes are,

indeed, larger than the monaural response, but this difference was not analyzed, nor were the ABR amplitudes.

Darling and Price (1990) examined the influence of loudness summation on the click-evoked ABR to determine whether or not the brainstem response provides a direct link to loudness perception. Loudness summation refers to a physiological process within the auditory system by which the power of the stimulus or the neural activity in response to the stimulus is summated over time, such that stimuli of longer duration are louder and therefore result in decreased (i.e., better) hearing thresholds (Eddins & Green, 1995). Temporal summation occurs also for trains of repetitive stimuli (Garner, 1948), and Darling & Price (1989) investigated this phenomenon by assessing the loudness of clicks presented at various repetition rates. Eight normal-hearing adult subjects were asked to match the loudness of 500-ms click trains presented at four repetition rates (11, 31, 51, & 91/s), to the level of 70, 80, and 90 dBSPL 1000-Hz long-duration tones using a loudness balance procedure. The intensity (dB peSPL) of the clicks judged to be equally loud to 70, 80, and 90 phons decreased with increasing repetition rate, indicating that temporal integration of these brief repetitive stimuli occurs for suprathreshold stimuli as it does for near-threshold stimulus levels (e.g., Zwislocki, Hellman & Verillo, 1962).

In their study, Darling and Price (1990) recorded the ABR to clicks at the same repetition rates from these same subjects at the individual intensities that each had judged to be equal to 70, 80, and 90 phons, and examined the amplitudes and latencies as a function of repetition rate and loudness. Their results did not support the hypothesis that the brainstem responses measured at equal loudness levels would have the same

amplitudes, latencies, and waveform morphology. The ABR recorded to clicks presented at fast (e.g., 51/s) repetition rates and lower intensities showed reduced amplitudes, prolonged latencies, and reduced waveform clarity compared to the response to slower rates of presentation (e.g., 11/s) and higher stimulus levels, even though the stimuli were judged to be equally loud. In addition, regression analyses of the amplitude growth rate showed an inverse relationship with increasing repetition rate, consistent with Rau's (1968) findings for the N1. The individual subjects' data revealed the opposite effect for loudness, such that the slope of the loudness function becomes steeper with faster repetition rates. These results indicate a dissociation between sensory magnitude and AEP amplitude (Näänänen & Picton, 1987).

In their investigation of ABR amplitudes and loudness, Davidson, Wall, and Goodman (1990) compared the absolute amplitudes of click-evoked ABRs recorded from 10 normal-hearing adults to their subjective judgement of the magnitude of these stimuli obtained using a magnitude estimation procedure (Stevens, 1956). The individual subject's ABR amplitudes and the numbers assigned during the magnitude estimation procedure were rank ordered from largest to smallest values, and rank correlation of these values was analyzed for individuals and the group within and across test sessions. The results from single sessions were quite variable, but across sessions, findings revealed a strong correlation between ABR amplitude and loudness for individual subjects as well as for the group. The authors concluded that their findings supported Kiessling's (1982, 1983) assumption that ABR amplitude is directly related to loudness.

The notion that ABR amplitude, or the magnitude of the electrical response, is related to sensory magnitude led Kiessling (1982, 1983) to develop a procedure for predicting hearing-aid gain and output characteristics based on normative intensity-amplitude measures obtained from normal-hearing and hearing-impaired children and adults. Kiessling demonstrated that the slope of the intensity-amplitude function was steeper for hearing-impaired subjects, and used comparative graphs of the intensity-amplitude functions from normal-hearing and hearing-impaired subjects to predict the appropriate amount of hearing aid gain and maximum power output required to restore hearing to normal levels within the impaired subjects' reduced dynamic range. A similar type of predictive procedure is commonly employed in current clinical practice, except that behavioral loudness measures are used instead of voltage values (e.g., Humes, 2000; Killion & Fikret-Pasa, 1993). In addition to their studies of normal-hearing subjects described above, Davidson et al. (1990) also evaluated three subjects with three different configurations of sensorineural hearing loss: sloping, flat, and rising. They compared the prescribed hearing-aid gain and output characteristics for these subjects obtained from ABR amplitudes and Kiessling's (1982) projection procedure with those obtained from a standard hearing-aid prescription procedure based on loudness measures (NAL-R, Byrne & Dillon, 1986). The prescribed hearing-aid gain data derived from the two formulae were similar for the subjects with flat and rising hearing loss, but for the subject with high-frequency (above 2000 Hz) sloping configuration of hearing loss, the ABR projection procedure overestimated hearing-aid gain, whereas the NAL-R procedure predicted an appropriate gain level. The details of the intensity-amplitude functions for

the impaired subjects were not provided, but the finding of overestimation of hearing aid gain requirements suggests that the ABR recordings for the subject with high-frequency hearing loss yielded small absolute amplitudes and/or little steepening of the growth function. Davidson et al. (1990) concluded that the lack of frequency specificity of the clicks used to elicit the ABR therefore limits its use as an objective measure for predicting hearing-aid parameters.

Clicks are the most effective -- and most often used -- stimuli for obtaining ABRs because their fast rise/fall time and short duration provide for excitation of a large number of cells and the best synchronization of activity contributing to the response (Eggermont, 1982a; Stapells, Picton & Smith, 1982). Use of such a broad-frequency stimulus for ABR threshold estimates and/or suprathreshold measures, however, is analogous to using a speech stimulus to acquire behavioral thresholds and loudness measures, a practice that is generally not accepted for making definitive statements about type, degree, and configuration of hearing loss (Roeser, Buckley & Stickney, 2000) nor for acquiring loudness measures (Keidser et al., 1999; Skinner, 1993). The effective frequency-specific stimuli used to elicit the ABR are tones with durations of less than 10 ms (Kodera, Marsh, Suzuki & Suzuki, 1983; Stapells & Picton, 1981). Such short-duration tones have maximal energy at the nominal frequency and also sidebands of energy above and below the nominal frequency of the stimulus (i.e., spectral splatter) (Elberling & Don, 1979; Picton et al., 1979). The effects of rise/fall time on tone ABRs vary with the frequency of the test tone such that longer rise/fall times (i.e., > 5 ms) cause a greater increase in latency and decrease in amplitude for the response to 500-Hz tones compared to the

results to 2000-Hz tones (Kodera et al., 1983; Stapells & Picton, 1981; Suzuki & Horiuchi, 1981); a constant cycle rise/fall time of < 5 ms results in smaller inter-frequency differences (Takagi, Suzuki & Kobayashi, 1985) and is recommended for routine clinical use (Kodera et al., 1983; Lichtenstein & Stapells, 1997; Stapells, 2000a; Takagi et al., 1985).

Tone-evoked ABRs are frequency-specific at low-to-moderate intensities for air-conducted (e.g., Davis, et al., 1984; Kodera, Yamane, Yamada & Suzuki, 1977; Picton et al., 1979; Oates & Stapells, 1997a,b) and bone-conducted tones (Kramer, 1992; Nousak & Stapells, 1992); at higher intensities, ipsilateral masking procedures may be used to eliminate contributions to the response from regions away from the nominal frequency of the test tone for frequency-specific threshold estimation (e.g., Picton et al., 1979; Stapells et al., 1994). Latency of wave V varies inversely with tone frequency, such that the response to 500-Hz tones occurs later than the response to 2000-Hz tones (e.g., Kodera et al., 1977; Oates & Stapells, 1997a; Stapells & Picton, 1981), reflecting the longer travel time of the low-frequency excitation wave on the cochlear partition from base to apex, as well as the longer rise/fall time of the lower-frequency stimulus (Davis & Hirsh, 1979; Stapells & Picton, 1981). The ABR latency difference between low- vs high-frequency tones is greater for low-intensity stimuli than for high-intensity stimuli because responses originate from place/frequency-specific regions of the cochlea as intensity is decreased (Don, Eggermont & Brackmann, 1979; Stapells et al., 1994); at higher intensities (i.e. > 60 dBHL), the ABR includes additional contributions from more basal regions of the cochlea in response to both low- and high-frequency tones (Eggermont, Spoor &

Odenthal, 1976; Folsom, 1984). The amplitude of wave V is similar across frequencies when constant cycle rise/fall times are used (e.g., Oates & Stapells, 1997a; Takagi et al., 1985), but the morphology of the brainstem response varies with frequency, such that wave V to high-frequency tones is sharply peaked and narrower than the response to low-frequency tones (e.g., Oates & Stapells, 1997a). These effects of frequency on wave V morphology are due to the dual structure (i.e., fast and slow components) of the ABR (Takagi et al., 1985) and the neural sources contributing to this response.

The ABR is produced from neural activity arising from ascending fiber tracts and/or nuclei from the distal auditory nerve through the pons and midbrain (Moore, 1987). The structures responsible for the generation of each of the scalp-recorded peaks I-V observed in the ABR to clicks at moderate-to-intense stimulus levels have been determined from near-field recordings in animals and scalp-recordings in humans (Achor & Starr, 1980; Jewett & Williston, 1971; Legatt, Arezzo & Vaughan, 1986a,b; Martin, Pratt & Schwegler, 1995; Møller, 1994; Møller & Jannetta, 1982, 1985; Møller, Jannetta & Sekhar, 1988; Zaaroor & Starr, 1991), including methods employing modeling of activity of equivalent dipoles of the ABR generators from multiple-channel scalp recordings (e.g., Martin, Gardi & Jewett, 1987; Scherg and von Cramon, 1985). Findings indicate that multiple generators contribute to the majority of the vertex-positive peaks that comprise the "fast" components of the ABR (Legatt, Arrezzo & Vaughan, 1988; Moore, 1987; Zaaroor & Starr, 1991). Wave I reflects the compound action potentials generated in the distal portion of the auditory nerve. Wave II arises from anatomical structures in the posterior fossa, specifically at the point the neural activity crosses a conductivity

boundary from which this stationary potential is generated (Martin et al., 1995). Wave III reflects axonal activity in the trapezoid body and from activity arising from the superior olivary complex. Wave IV is generated also in the pons and reflects activity in the lateral lemniscus (Legatt et al., 1988; Moore, 1987). Wave V originates in the midbrain in the region of the inferior colliculus, and reflects activity from an array of generator sources in the lateral lemniscus and inferior colliculus (Durrant, Martin, Hirsch & Schwegler, 1994; Hashimoto, 1982; Møller, 1994). The "slow" component of the ABR represents summation of graded postsynaptic potentials in the inferior colliculus (Hashimoto, 1982; Legatt et al., 1988) and the "fast" waves reflect action potentials within the auditory nerve and brainstem pathways (Legatt et al., 1988). Thus, the ABR is compounded activity that represents neural responses initiated in the brainstem by cochlear activation over time (Elberling, 1979).

The latency of the ABR is determined by several mechanical and physiological processes in the cochlea, auditory nerve, and brainstem which have been identified, in part, from studies of cochlear place-specific measures obtained using the high-pass/derived-response technique (Eggermont, 1976; Eggermont & Don, 1978; Parker & Thornton, 1978a,b). These processes include cochlear transport time, cochlear filter build-up time, the synaptic delay between IHCs and auditory nerve fibers, and the neural conduction time and intervening synaptic delays that occur up to the point in the brainstem pathway responsible for peak activity (Don, Ponton, Eggermont & Kwong, 1998). Amplitude is a measure of response strength, and is determined mainly by the

level of stimulation and the number and amount of synchronized neuroelectric sources, as well as by cochlear travel times (Don, Ponton, Eggermont & Masuda, 1994).

The latency of the ABR is a highly stable measure both within and between individuals, and many studies have evaluated the changes in the peak latencies of the suprathreshold ABR to determine its use in identifying and quantifying hearing loss (e.g., Arslan, Prosser & Rosignoli, 1988; Brackmann & Selters, 1977; Brookhouser, Gorga & Kelly, 1990; Chisin, Gafni & Sohmer, 1983; Don, Ponton, Eggermont & Kwong, 1998; Fria & Sabo, 1980; Galambos & Hecox, 1978; Gorga, Worthington, Reiland, Beauchaine & Goldgar, 1985; Kirsh, Thornton, Burkard & Halpin, 1992; Oates & Stapells, 1992; Prosser & Arslan, 1987; Rupa & Dayal, 1993; Serpanos et al., 1997; Suter & Brewer, 1983; van der Drift, Brocaar & van Zanten, 1987; Yamada, Kodera & Yagi, 1979; Yamada, Yagi, Yamane & Suzuki, 1975; Watson, 1996, 1999; and see Hall, 1992 for review). For example, a clear response recorded at a mild or moderate intensity assists the clinician in determining threshold, because wave V latency is successively displaced along the time axis as stimulus level is decreased (e.g., Picton et al., 1981). Many investigators have suggested that displacement of the intensity-latency function along the time and intensity axes, relative to the normal response, provides a means of determining the nature of hearing loss (e.g., Fria & Sabo, 1980; Picton, 1990; Yamada et al., 1979). For example, prolonged latencies at suprathreshold intensities that yield a function parallel to the normal intensity-latency function and shifted to higher intensities suggests primarily conductive hearing loss (e.g., Fria & Sabo, 1980; Stapells et al., 1985; Yamada, et al.,1975), whereas a steepening of the intensity-latency function relative to the normal

response, such that the response only at low sensation levels is prolonged, has been associated with sensorineural hearing loss (e.g., Galambos & Hecox, 1978; Yamada, Kodera & Yagi, 1979). Prolonged latencies (and reduced amplitudes) in the case of conductive hearing loss are due mainly to reduced signal intensity arriving at the cochlea (Fowler & Durrant, 1994). It has been suggested that the steeper intensity-latency function observed in individuals with sensorineural hearing loss is indicative of recruitment, such that moderate-to-intense stimuli (i.e., above 20 dBSL) yield normal ABR latencies (e.g., Galambos & Hecox, 1978). Most of these reports on the changes in the intensity-latency function in subjects/patients with hearing loss, however, are limited to results of ABR recordings to non-masked clicks which, due to their frequency content, yield latency values that are unreliable indicators of degree, type, and/or configuration of hearing loss (Eggermont, 1982a). An early study by Yamada et al. (1979), for example, demonstrated the variety of sensorineural hearing losses (by degree, configuration, and type/etiology) that yield similar intensity-latency functions when the ABR is recorded to clicks alone. Use of frequency-specific stimuli (i.e., tones) is more appropriate and may provide more reliable measures of wave V latency changes for suprathreshold stimuli in individuals with hearing loss.

In contrast to latency, ABR amplitude has largely been ignored (Mahoney, 1985) except for the wave V/I amplitude ratio, which is considered a useful diagnostic index of normal/abnormal brainstem functioning (Schwartz & Berry, 1985). ABR amplitudes have not received clinical attention, at least in North America (Hall & Ruth, 1985), because of early reports of its variability in the normal-hearing population (Chiappa,

Gladstone & Young, 1979; Rowe, 1981; Starr & Achor, 1975), its susceptibility to myogenic activity and noise level, and also reports of difficulty in replicating waveforms (Schwartz & Berry, 1985). Picton, Stapells, and Campbell (1981) noted, however, that although ABR amplitudes are variable between subjects, individual subjects "may show quite consistent steps in the amplitude-intensity functions." Recent reports confirm this supposition (e.g., Jiang, Zhang, Wu & Liu, 1993; Lauter, Oyler & Lord-Maes, 1992), and also show that the amplitude variations observed in the normal-hearing population are partially a result of individual differences in cochlear transport time (Don et al., 1993).

There are several biological variables that can influence AEP response amplitudes, such as the thickness of the calvarium, the impedance properties of the intervening brain tissue (Hecox & Burkard, 1982), and, as has recently been shown, gender differences in cochlear anatomy. Females have shorter cochleae (Sato, Sando & Takahashi, 1991). The anatomical data coupled with their electrophysiologic findings from high-pass/derived-response studies led Don et al. (1994) to suggest that a steeper stiffness gradient along the basilar membrane in female cochleae likely causes the unmasked amplitude differences between males and females. The consequence of a steeper stiffness gradient is faster response times across the cochlea to the generators of wave V that results in better synchrony and therefore, larger amplitudes. Don and colleagues also indicate that wave V amplitude variations in the normal population are a result, also, of irregularities in cochlear response times which arise from individual differences in the mechanical properties of the basilar membrane, differences in the sharpness of tuning resulting from variation in feedback gain of the cochlear amplifier, as well as individual differences in

threshold along the cochlea (Don et al., 1994). It may be that these individual differences in anatomy and physiological processes that produce variability in ABR amplitudes also give rise to the some of the variability observed in subjective magnitude estimates. Comparisons of the effects of hearing loss on the tone-evoked ABR intensity-amplitude function and its relationship to psychophysical measures of magnitude estimation in normal-hearing listeners or in groups of impaired listeners with homogeneous degree, configuration, and type of hearing loss have not yet been reported. Such findings may provide a means of establishing a functional relationship between the two measures that could be applied in both basic and clinical endeavors.

Chapter 4:

Rationale for the dissertation studies

General Rationale

The goal of research in evoked potential audiometry has been to establish the relationship of these measures to behavioral indications of auditory system capabilities and particularly, their application for clinical purposes. In current clinical practice, behavioral audiometry includes tests that evaluate three main features of hearing: frequency-specific sensitivity measures to air- and bone-conducted tones, loudness growth, and speech recognition ability (Humes, 2000; Roeser et al., 2000; Thibodeau, 2000). The goal of applying electrophysiologic measures, then, is to obtain the same information with AEPs as that obtained using behavioral methods. AEPs, especially the ABR but also the MLR, are now used to predict hearing sensitivity, primarily for the early identification of hearing loss in infants who are unable to perform the tasks required in behavioral audiometry. Presently, however, we cannot obtain reliable loudness data behaviorally in children younger than five years of age (Launer, 1998; Mueller & Bentler, 1994). It may be that the ABR and/or MLR can predict loudness. Loudness increases with increasing stimulus level, and so do AEP amplitudes. As the preceding literature review suggests, however, ABR studies relating the characteristics of this evoked potential and loudness have, to date, produced equivocal results, and there is a paucity of information on the MLR to suprathreshold stimuli in normal-hearing or hearing-impaired listeners. A study of ABR/MLR latency and amplitude measures of intensity dependence obtained in normal-hearing and hearing-impaired persons, and their relationship to psychoacoustic measures of sensory magnitude obtained from these same subjects may

provide evidence indicating that these AEPs do reflect the neural events involved in loudness perception as has been shown for the slow cortical evoked potential.

The results from the earlier studies of the ABR/MLR and loudness have limited application because most authors reported findings from normal-hearing subjects only, recorded the ABR or MLR only to clicks (i.e., to broadband stimuli), used narrow EEG filter settings, focused only on the changes in latency, obtained a limited number of trials, or some combination of these factors (Davidson, Wall & Goodman, 1990; Howe & Decker, 1984; Madell & Goldstein, 1972; Pratt & Sohmer, 1977; Serpanos et. al., 1997; Wilson & Stelmack, 1982). Because clicks contain equal energy across a broad frequency spectrum, their use in hearing assessment is limited, both for threshold- as well as for loudness-based measures (Eggermont, 1982a; Stapells et al., 1990). In order to provide estimates of the auditory response from specific frequency regions and thereby approximate behavioral audiometric measures, frequency-specific stimuli such as short-duration tones (Hyde, 1997; Kodera et al., 1977; Kraus et al., 1994; Stapells et al., 1994), or place-specific procedures such as combining clicks in high-pass noise to derive narrow-band responses (Don, Eggermont & Brackmann, 1979; Eggermont, 1982a) must be used. Many studies have shown, for example, that tone-evoked ABR thresholds for the frequencies 500, 1000, 2000, and 4000 Hz approximate well the behavioral pure-tone thresholds in normal and impaired listeners of all ages (for review see Stapells, 2000b). Obtaining intensity-amplitude/latency functions of the ABR/MLR to tonal stimuli would provide frequency-specific measures (Stapells et al., 1994) that are comparable to the existing literature on behavioral measures of loudness growth, which predominantly

employed frequency-specific stimuli (pure-tone or 1/3-octave narrow-band noise) (e.g., Hellman, 1991; Hellman & Meiselman, 1990, 1993; Humes, 2000; Keidser et al., 1999; Skinner, 1993). As well, ABR/MLR recordings to suprathreshold tonal stimuli may prove to be clinically applicable as predictive measures of loudness growth for diagnostic and habilitative purposes. To date, there has been no study comparing tone-evoked ABR and/or MLR intensity series with psychoacoustic measures of loudness growth in either normal-hearing or hearing-impaired listeners.

Previous studies comparing the ABR/MLR and magnitude estimates have consistently shown that the exponents for the AEP latencies and amplitudes are smaller than the exponent of the loudness function (e.g., Madell & Goldstein, 1972; Wilson & Stelmack, 1982). This has been taken as evidence that the neural sources reflected by the electrophysiologic recordings are not the same as those that give rise to perceptual magnitude (Howe & Decker, 1984; Pratt & Sohmer, 1977). Several early studies, however, demonstrated that the relative size of the psychophysical power functions for different modalities usually approximates the relative size of the evoked potential power functions (e.g., Keidel & Spreng, 1965; Walsh, 1979). Thus, while the size of the exponents differs within a modality, the consistency of the *relative* exponent size across modalities for the evoked potentials and the psychophysical measures indicates that the two measures have something in common (Stevens, 1970).

An alternative way to determine whether evoked potential measures reflect a similar pattern of change as that observed from psychophysical measures is to evaluate both sensation magnitude and evoked potentials in the same subjects in several conditions

within the same modality. This may be accomplished in the auditory domain by evaluating a group of normal-hearing subjects in several different conditions: tones presented alone and in the presence of continuous maskers at several different intensities. Noise-masked normal-hearing subjects demonstrate loudness recruitment comparable to that observed in ears with sensorineural pathology (e.g., Hellman, 1970; Stevens, 1966). That is, the function for loudness growth in noise-masked normal-hearing subjects is steeper than that observed for those subjects in the non-masked condition, and steepens with increasing threshold shift (e.g., Gleiss & Zwicker, 1964), similar to the findings reported by Hellman and Meiselman (1993) which showed a faster rate of loudness growth for those hearing-impaired listeners with greater hearing loss. By using different levels of masking, several functions may be obtained from a group of normal listeners for both the behavioral and ABR/MLR measures. If the relative exponent size is similar between conditions for the evoked potentials as that seen in the psychoacoustic observations, then it would suggest that both measures reflect similar neural processes. If the changes in loudness growth are reflected reliably in the ABR and/or MLR measures, then the evoked potential measures may be used to predict the loudness exponent in subjects/patients who are unable to perform behavioral loudness scaling procedures. Presently, there are no studies that have compared the effects of masking noise on the rate of amplitude growth/latency decrease of the ABR/MLR with intensity and behavioral assessment of loudness growth in noise-masked normal-hearing subjects.

We also elected to obtain the electrophysiologic and behavioral measures from a group of subjects with primarily cochlear loss of hearing sensitivity to determine whether or not

the changes in their loudness growth functions are reflected also in their ABR/MLR measures, and also to compare their results to those of the masked normal listeners. No previous study has examined the relation between the tone-evoked ABR or MLR and loudness in a group of hearing-impaired listeners; such studies are needed for clinical application of these evoked potentials.

Goals of the Dissertation

The specific aims of the present studies were (1) to assess and compare the accuracy of the tone-evoked ABR and MLR in estimating behavioral hearing thresholds in hearing-impaired listeners, (2) to evaluate the effects of hearing loss on the brainstem and early cortical responses to increasing stimulus level, (3) to examine the relationship between the changes observed in the major components of the brainstem and middle latency electrophysiologic response measures with increasing intensity, and behavioral measures of loudness growth for frequency-specific stimuli, and (4) to inquire whether or not there is a consistent pattern of change in the response measures between normal-hearing and hearing-impaired adults for the electrophysiologic measures.

The long range objectives of this research are threefold: (1) to contribute to the basic science of understanding the neurophysiologic mechanisms which implement processes of sensory and perceptual experience, (2) to use electrophysiological data to address psychoacoustic and physiologic theories of loudness growth, and (3) to provide preliminary information on the potential application of electrophysiologic response measures for study of developmental issues of loudness growth, and for determining appropriate parameters of amplification systems for hearing-impaired infants, children,

and older persons who cannot report their subjective experience of the loudness of sounds.

Rationale underlying selection of specific parameters

Subjects. Normal-hearing and sensorineurally hearing-impaired subjects participated in these dissertation studies. The normal listeners participated also as "hearing-impaired" listeners when ipsilateral broadband noise was presented simultaneously with the signal to assess masker effects on their behavioral and electrophysiologic measures. Adults with sensorineural hearing loss of mild-to-moderate degree with flat or gradually sloping configuration of hearing loss, primarily cochlear in nature, were chosen in order to obtain results from a homogenous group of hearing-impaired persons re: configuration and degree (Skinner, 1988).

Stimuli. The stimulus used in the dissertation studies was a 1000-Hz short-duration tone (5 ms exact-Blackman windowed) which is effective for eliciting the ABR and MLR (Kodera et al., 1977; Lichtenstein & Stapells, 1997; Stapells & Picton, 1981). This stimulus frequency was chosen for four reasons: (1) because most of the extant literature on psychoacoustic measures of loudness growth employed 1000-Hz tones (Hellman, 1991), (2) because there are few studies of the MLR to 1000-Hz tones for either threshold estimation or suprathreshold measures in normal listeners and/or hearing-impaired listeners, (3) because few studies have compared the ABR and MLR to 1000-Hz tones (Suzuki et al., 1981; Kodera et al., 1977), and controversy exists as to whether or not the MLR to low-frequency tones is superior to the ABR for threshold estimation (e.g., Beatty et al., 1996; Musiek & Geurkink, 1981), and (4) because there are no reports of

normative data (amplitude, latency, waveform morphology) from intensity series of the MLR recorded to this frequency stimulus from hearing-impaired subjects using appropriate acquisition parameters.

The ABR/MLR and loudness estimates were obtained at 8-10 intensities above the individual subjects' thresholds that included equal sensation and equal hearing levels. For example, all subjects were tested at the following equal sensation levels (dB SL) re: behavioral thresholds: 5, 10, 15, 20 and 30 dBSL and at the following equal hearing levels: 55, 70 and 80 dB nHL. The levels for all subjects, determined prior to data collection, were chosen for four reasons. First, the number of levels was chosen to ensure that we obtained a function that describes well both the changes in the ABR/MLR amplitudes and latencies and the changes in the psychoacoustic measures (loudness growth) from which a power function may be approximated. Second, equal sound pressure levels were chosen for all subjects in order to eliminate differential stimulus level comparisons between groups (Humes, 1982). If recruitment is present, equal sound pressure levels will correspond to equal loudness at moderate-to-high sensation levels (Hellman & Zwislocki, 1961). Third, we considered it important to obtain functions for a wide dynamic range for both the psychoacoustic and ABR/MLR measurements in order to assess the utility of using the ABR/MLR measures as predictors of loudness growth. Finally, the 3- 4 sensation levels at and just above threshold were included to assist in determining ABR/MLR response presence/absence at and near threshold.

The tonal stimuli were presented alone or in simultaneous broadband noise (normal-hearing subjects only). Ipsilateral noise was presented to mask the responses to 40 and 60

dB nHL tones, and the level of the noise masker that Effectively Masked the tones 50% of the time (i.e., 40 and 60 dBEM) was determined individually. We chose threshold shift levels of 40 and 60 dB nHL based upon two considerations. First, we considered the threshold levels included in the range of those for the hearing-impaired group. Obtaining noise-masked functions for similar threshold levels from both groups allowed us to compare the psychophysical and electrophysiologic functions between groups in addition to within-group comparisons. Secondly, in seeking to demonstrate whether or not the relative difference between psychoacoustic and electrophysiologic measures is similar within the auditory domain, we expected that a difference of at least 20 dB between thresholds for the normal listeners would yield effective and clear comparisons within the scope of these studies. Several previous studies demonstrated a clear trend toward steeper functions with increased threshold shift by comparing threshold elevations separated by 20 dB (Gleiss and Zwicker, 1964: 40 and 60; Hellman, 1970: 58 and 78; Hellman and Zwislocki, 1964: 40 and 60; Lochner and Burger, 1961: 35 and 55; Schlauch et al., 1998: 30 and 50).

In order to ensure that subjects would not be exposed to excessive signal levels (i.e., uncomfortably loud) during any portion of the experimental procedures, Loudness Discomfort Levels (LDLs) of the 1000-Hz tones were determined individually for each condition (i.e., in the quiet and in noise) using an established LDL procedure (Hawkins et al., 1987). These levels were used to determine the individual subjects' maximum level for response assessment for both their psychoacoustic and electrophysiologic measures.

The intensity of the white noise required to mask 40 and 60 dB nHL tones was assessed prior to commencing the study by reviewing the masker levels used in previous studies using stricter (i.e., 75%) criterion than that employed in the present studies (50%) (e.g., Oates & Stapells, 1997a,b; Purdy et al., 1989), and also from obtaining masked thresholds from five normal-hearing subjects. This was done to ensure that the highest masker level used in this study would be well below the level which may cause temporary or permanent threshold shift (Ward, Glorig & Sklar, 1958).

Psychoacoustic Measures. The behavioral measures evaluated in this dissertation are threshold and loudness growth. Threshold was defined as the point on the psychometric function that yields 50% yes-response obtained using a bracketing procedure (Carhart & Jerger, 1959) that was modified to a 2-dB step size in the search of threshold for all subjects. Thresholds were obtained to long-duration and short-duration tones in the quiet for both groups of subjects (dB HL/dB nHL). The broadband noise level that effectively masked the 40 and 60 dBnHL tones in the normal listeners was obtained by using a 2-dB step size bracketing procedure, and the same criteria for threshold as that for the tone thresholds was applied. The accuracy of ABR/MLR threshold estimation has not been previously assessed using individually determined masked thresholds in normal-hearing subjects. These results will therefore expand the existing data set regarding tone-evoked ABR/MLR/behavioral threshold agreement in normal-hearing and hearing-impaired listeners (Stapells, 2000b; Suzuki et al., 1981; Wu & Stapells, submitted).

Loudness growth was assessed using the Absolute Magnitude Estimation (AME) (Hellman & Zwislocki, 1961) procedure which has been established as a standard metric

of individual loudness growth in normal and impaired listeners (Hellman, 1991; Hellman & Meiselman, 1990, 1993). Previous studies have demonstrated that the slope of the loudness function obtained using the AME becomes more steep with increasing threshold shift for both noise-masked normal listeners (e.g., Hellman & Zwislocki, 1963; Schlauch et al., 1998) and for sensorineurally-impaired listeners (e.g., Humes, 2000; Mizkolczy-Fodor, 1960).

ABR/MLR Measures. The auditory evoked potentials examined in this dissertation are ABR wave V-V' and MLR Na-Pa. The ABR and, to a lesser extent, the MLR are used currently in clinical practice, and study of these two electrophysiologic responses allows for examination of auditory processing of signal level at different levels of the auditory system. The ABR derives mainly from neural activity in cochlear and brainstem structures to the level of the inferior colliculus (Legatt et al., 1988; Møller, 1994) and the MLR reflects activity rostral to the inferior colliculus, primarily in structures in primary auditory cortex (Scherg & von Cramon, 1986). The ABR and MLR were recorded simultaneously in order to compare/contrast (1) the changes in their amplitudes and latencies with increasing intensity and psychoacoustic measures of magnitude perception and (2) their accuracy in estimating behavioral measures of hearing threshold.

Electroencephalographic (EEG) signals were amplified (gain = 50,000) and analog filtered using a bandpass of 10-1500 Hz and a roll-off slope of 12 dB/octave. The high-pass filter setting of 10 Hz was chosen because the spectral content of both the ABR and MLR to low-frequency tones (i.e., 1000 Hz) and to low-intensity stimuli includes predominantly low-frequency energy (Malinoff & Spivak, 1990; Suzuki & Horiuchi,

1977; Suzuki et al., 1983) and we wanted to maximize these responses' amplitudes which decrease with increasing high-pass filter settings (Stapells & Picton, 1981; Suzuki et al., 1983, 1984). In their studies on the ABR with intensity and psychoacoustic magnitude estimation, Howe and Decker (1984; 300-3000 Hz), Pratt and Sohmer (1977; 250-3000 Hz), Serpanos et al. (1997; 100-3000 Hz), and Wilson and Stelmack (1982; 100-3000 Hz) used high-pass EEG filter settings which have been shown to reduce substantially the amplitudes of the ABR to clicks (Cacace, Shy & Satya-Murti, 1980; Elberling, 1979; Stockard, Stockard & Sharbrough, 1978) and tones (Stapells & Picton, 1981; Suzuki & Horiuchi, 1977; Suzuki, Hirai & Horiuchi, 1977). Madell and Goldstein (1972) used narrow EEG filters (25-150 Hz) which can cause waveform distortion, increased latencies, and decreased amplitudes of the MLR (Kileny, 1983; Scherg, 1982a; Suzuki, Kobayashi & Hirabayashi, 1983).

The short duration of most ABR peaks indicates that they derive from action potentials (Legatt et al., 1988). The slow wave component of the ABR on which these brief-duration peaks ride, however, shows a large positivity at about 5-6 ms (in response to 80 dBnHL click) followed by a slow negativity (Davis & Hirsh, 1979; Suzuki & Horiuchi, 1977). The prolonged time course of the slow wave compared to the earlier "fast" components suggests that it reflects predominantly postsynaptic (dendritic) potentials (Legatt et al., 1988; Møller, 1994). Similarly, the MLR, composed of a slow negative wave (Na) followed by a prolonged positivity (Pa) likely reflects postsynaptic activity (Hashimoto et al. 1995; Scherg & von Cramon, 1986). It is the slow negative wave that is most useful in the interpretation of the ABR/MLR, especially at low intensities (Davis

& Hirsh, 1979; Fabiani, Sohmer, Tait et al., 1979; Picton, Woods, Baribeau-Braun, & Healey, 1977) and for low-frequency stimuli (Stapells, 1994; Stapells & Picton, 1981; Suzuki & Horiuchi, 1977). It may be that electrophysiologic recordings that include this postsynaptic activity reflect the neural processes that contribute to subjective reports of loudness growth. Therefore, the bandpass filter settings and roll-off slopes chosen for the present studies are appropriate for obtaining the ABR and MLR to 1000-Hz tones (Kileny, 1983; Kraus et al., 1987; Stapells & Picton, 1981; Suzuki & Horiuchi, 1977). The tones were presented at a rate of 10/second so that the transient ABR and MLR could be obtained simultaneously without overlap of components (e.g., Stapells & Picton, 1981) in order to compare their response measurements to suprathreshold stimuli, and also to compare their accuracy in estimating behavioral sensitivity measures to 1000-Hz tones.

Chapter 5

Auditory brainstem and middle latency responses to 1000-Hz tones in noise-masked normal-hearing and sensorineurally hearing-impaired adults

Introduction

The auditory brainstem (ABR) and middle latency (MLR) responses have been studied extensively during the past 30 years, particularly to investigate their potential clinical application in audiologic assessment. Both the ABR, which occurs 1-20 ms following stimulus presentation, and the MLR, which occurs within the 10-80 ms post-stimulus time epoch, have been advocated for frequency-specific audiometry (for reviews, see: Kraus, McGee & Stein, 1994; Stapells, Picton, & Durieux-Smith, 1994). That is, each of these evoked potential options may be used to estimate the pure-tone audiogram. Within the literature, however, are conflicting reports about whether or not one of these evoked potential measures is superior to the other for estimating pure-tone thresholds, especially in response to low-frequency stimuli (i.e., 500 and 1000 Hz) (Laukli, Fjermedal, & Mair, 1988; McGee & Kraus, 1996; Musiek & Geurkink, 1981; Scherg & Volk, 1983; Stapells, Picton & Durieux-Smith, 1984b). How these two measures compare in this regard is an important consideration for the clinician when the choice of appropriate (or alternative) electrophysiologic measure for a given patient population and/or assessment protocol must be made.

The ABR is used routinely in clinical practice as the evoked potential of choice for threshold estimation because it is a highly stable and replicable response, unaffected by subject state (Picton, 1990; Picton, Woods, Baribeau-Brāun, & Healey, 1977). Many researchers have shown good response detectability and estimation of hearing sensitivity using tone-evoked ABRs for the frequencies 500, 1000, 2000, and 4000 Hz in normal-hearing and hearing-impaired adults, children, and infants (Beattie, Garcia & Arthur,

1996; Hyde, Matsumoto & Alberti, 1987; Munnerley, Greville, Purdy & Keith, 1991; Sininger, Abdala, & Cone-Wesson, 1997; Stapells, Gravel & Martin, 1995; Suzuki, Kodera & Yamada, 1984; Werner, Folsom & Mancl, 1993; see Stapells, 2000b for review). A few researchers, however, have reported that ABR recordings to 500- and/or 1000-Hz tones yield unacceptably elevated thresholds (i.e., ≥25 dB re: behavioral threshold), which could preclude their use as an objective audiometric measure to these low-frequency tones (e.g., Beattie, Garcia & Johnson, 1996; Gorga, Kaminski, Beauchaine & Jesteadt, 1988; Laukli, 1983; Laukli et al., 1988; Scherg & Volk, 1983).

The controversy about using tone-evoked brainstem response recordings to estimate low-frequency hearing sensitivity has led several researchers to investigate and propose the use of the MLR as an objective electrophysiologic measure of low-frequency hearing (Kavanagh et al., 1984; Kileny & Shea, 1986; Kraus & McGee, 1990; McGee & Kraus, 1996; Musiek & Geurkink, 1981; Scherg & Volk, 1983). Although historically the MLR preceded the ABR as a possible electrophysiologic technique for hearing evaluation, it was not until the 1980s that several technical papers were published defining appropriate acquisition parameters for this response (Kraus, Reed, Smith, Stein & Cartee, 1987; Kraus, Smith & McGee, 1987; Scherg, 1982a,b; Suzuki, Kobayashi & Hirabayashi, 1983a,b, 1984b). The MLR, dominated by low-frequency EEG energy, is significantly affected by analog filtering (Kraus et al., 1987; Scherg, 1982; Suzuki et al., 1983a,b, 1984b), and requires a low high-pass filter setting (~ 10-30 Hz), and a wide passband for optimum response measures (Kraus et al., 1987; Suzuki et al., 1984b), parameters that are

the same as those recommended for use in obtaining the ABR (Stapells, 1994, 2000a; Suzuki & Horiuchi, 1977; Takagi, Suzuki & Kobayashi, 1985).

Several researchers have shown good threshold prediction from MLR recordings to 500-Hz tones in normal-hearing adults and children (Barajas, Exposito, Fernandez & Martin, 1988; Kavanagh et al., 1984; Kavanagh, Domico, Crews & McCormick, 1988; Kileny & Shea, 1986; Scherg & Volk, 1983). Only a few studies, however, have reported on the accuracy of the tone-evoked MLR for threshold estimation to frequencies other than 500 Hz in either normal-hearing subjects (Stapells, et al., 1984b; Suzuki et al., 1981) or in hearing-impaired subjects (Stapells et al., 1984b; McFarland, Vivion & Goldstein, 1977). Studies of MLR estimates of hearing threshold at other frequencies important for communication (e.g., 1000, 2000, and 4000 Hz) using appropriate acquisition parameters and in various hearing-impaired groups (by degree, type, and configuration of loss) are needed for clinical application of this response.

Some researchers have compared the detectability of the ABR vs MLR by obtaining both responses from the same subjects. The conclusions drawn from these reports are conflicting. For example, Musiek and Geurkink (1981) obtained ABRs and MLRs to clicks from 15 normal-hearing adults and suggested that the MLR was superior to the ABR for threshold estimation. Conversely, Kavanagh et al. (1988) recorded ABRs and MLRs to clicks from 48 children and judged the ABR to be superior to the MLR in response detectability. Stapells et al. (1984b) obtained both responses from normal-hearing and hearing-impaired adults, and reported that the ABR yielded lower thresholds (i.e., better accuracy) than the MLR. In all of these studies, however, ABR and MLR

recordings were made separately, usually using different EEG filter settings (e.g., ABR: 150-3000 Hz vs MLR: 10-100 Hz), stimuli, presentation rates, and/or a different number of trials. Thus the conflicting conclusions drawn from these studies may reflect the differences within each study in the acquisition parameters used rather than differences in the ABR vs MLR regarding their accuracy in threshold estimation.

As noted above, the ABR and MLR may be recorded to low-frequency tones using the same filter settings, stimuli, and presentation rate, although the MLR requires a longer analysis time (70-100 ms). Direct comparison of their response properties is best made by recording these responses simultaneously (Suzuki et al., 1981). Comparative descriptions of the ABR/MLR recorded in this manner were first reported in the 1970s. Picton, Hillyard, Krausz, & Galambos (1974) obtained ABR/MLRs to clicks from normal-hearing adults. With increasing stimulus intensity, amplitudes increased and latencies decreased at a similar rate for the two responses. Suzuki and colleagues (Kodera, Yamada, Yamane & Suzuki, 1978; Kodera, Hink, Yamada & Suzuki, 1979; Yamada, Yamane & Kodera, 1977) investigated intensity (30 and 50 dBSL), rate, and rise time effects on the ABR/MLR to 500- and 1000-Hz tones in normal-hearing adults. They noted larger amplitudes and greater variability of the MLR compared to that seen for the ABR wave V, but found similar effects of rise time on both components. They also demonstrated that a slow (e.g., 10/s) stimulation rate yielded robust ABR/MLRs from all subjects at both moderate and low intensities. More recently, Stapells and colleagues investigated ABR/MLRs to clicks and/or tones from adults and showed: (1) no difference in the frequency specificity of the ABR and MLR to 500- and 2000-Hz tones (Mackersie,

Downs, & Stapells, 1994; Oates & Stapells, 1997a, b; Wu & Stapells, 1994), (2) no large differences in the detectability of the ABR and MLR in response to clicks, or to 500- and 2000-Hz tones in normal-hearing adults (Wu & Stapells, submitted), and (3) no difference in rise-time (Lichtenstein & Stapells, 1997).

Although simultaneous recording of the ABR/MLR has been recommended for routine clinical use (Hood, 1995; Kraus & McGee, 1990), very few studies exist describing the ABR/MLR waveform morphology and their amplitudes and latencies at various intensities, or ABR/MLR threshold estimates in subjects with decreased hearing sensitivity. These data are important for clinical application of this recording technique. Scherg and Volk (1983) recorded the ABR/MLR to clicks and to 500-Hz tones at 20, 30 and 70 dBSL from a group of subjects with high-frequency hearing loss. They found that near threshold (20 and 30 dBSL), ABR wave V amplitude was lower and its detectability was poorer than the MLR, but they suggested that simultaneous ABR/MLR recordings likely provide more reliable threshold estimates than the ABR alone. Suzuki et al. (1981) obtained ABR/MLRs to 500, 1000, 2000, and 4000-Hz tones from normal-hearing adults and from adults with various configurations, type (conductive and sensorineural) and degree of hearing loss. They concluded that the ABR was more consistently observed in both groups of subjects, but that the larger amplitude of the MLR allowed for better response detectability for many subjects. For 500- and 1000-Hz tones, detectability was similar for the ABR and MLR, whereas at the higher frequencies (2000 and 4000 Hz), the ABR gave somewhat better (lower) thresholds compared to the MLR. These authors,

however. did not provide complete information concerning their results, thus further examination of the ABR/MLR in groups of hearing-impaired subjects is needed.

One means of assessing and comparing the ABR/MLR for their accuracy in threshold estimation and detectability in subjects with decreased hearing sensitivity is by recording these responses simultaneously from a group of normal-hearing subjects with tones presented alone and in the presence of a masking noise. An effective masker (EM) refers to the threshold shift in decibels produced in the masked ear by a given amount of noise (Sanders, 1972). Determined behaviorally, this shift in threshold should also be seen in the electrophysiologic responses. Obtaining masked behavioral and ABR/MLR thresholds from a group of subjects who serve as their own control can reduce some of the variability in response recordings, and provide for consistent threshold elevations allowing for assessment of the similarities and/or differences between the ABR and MLR.

The effects of simultaneously presented broadband ipsilateral masking noise on the click-evoked ABR are reasonably well known. Reports by several investigators have shown that, in general, with increasing noise level, the latency of wave V increases and amplitude decreases (Beattie & Kennedy, 1992; Beattie, Thielen & Franzone, 1994; Burkard & Hecox, 1983; Picton et al., 1979; Stapells, 1984). Few studies have reported on the effects of masking on the MLR (Beattie & Boyd, 1984; Gott & Hughes, 1989). Gott & Hughes (1989) recorded the ABR and MLR to 60 dB nHL clicks in normal-hearing subjects, and found little or no change in MLR latencies and amplitudes with increasing masker level. Beattie & Boyd (1985) reported similar rates of detectability for the ABR and MLR to 500, 1000, and 2000 Hz tones presented simultaneously with white

noise. However, they presented the tones at a rate of 31.1/s, which is too high for obtaining the transient MLR (Stapells et al., 1984a, 1994), and their recordings and analyses (25 ms) did not include wave Pa, the most robust positive peak of the MLR (Kraus et al., 1994). There are no studies that have assessed the accuracy of the tone-evoked ABR or MLR in estimating threshold from normal-hearing subjects whose behavioral threshold has been shifted with broadband masking noise.

In light of the above review indicating (i) the need for further studies directly comparing the ABR and MLR for their accuracy in estimating behavioral thresholds to low-frequency tones, (ii) the need for studies that include results from hearing-impaired subjects, and (iii) the lack of information concerning the effects of noise masking on the tone-evoked MLR, the present study was undertaken to examine the ABR vs MLR recorded simultaneously to 1000-Hz tones from noise-masked normal-hearing adults and from a group of subjects with sensorineural hearing impairment. Thresholds were shifted in the normal-hearing group to levels that were similar to the those of the sensorineurally-impaired subjects. Comparative evaluation of ABR/MLR response properties included analyses of recordings to tones presented over a range of stimulus intensities, including threshold levels, in order to investigate the changes in ABR/MLR amplitudes and latencies with increasing intensity.

Methods and Procedures

Subjects

Twenty-four subjects participated in this study, ranging in age from 18 to 63 years (mean = 42.1 years). Only one ear was tested per subject. Twelve subjects had normal

hearing, with pure-tone behavioral thresholds equal to or less than 20 dBHL (ANSI, 1989) at octave frequencies 250 through 8000 Hz for at least one ear (the test ear). The other 12 subjects had moderate sensorineural hearing loss. Configurations of hearing loss were either flat or gradually sloping, with no greater than a 10-dB change in threshold between successive half octaves within the frequency range of 500 through 4000 Hz. Their mean thresholds at 500, 1000, 2000, and 4000 Hz were 35.8, 45.4, 52.5 and 55 dB HL respectively (s.d. = 10.6, 9.6, 10.6, 12.8). Thresholds at 1000 Hz ranged from 30 to 60 dBHL. All subjects had normal middle-ear function as measured by immittance testing, and no significant neurologic histories.

Stimuli and Equipment

The stimuli were short-duration 1000-Hz tones presented alone or simultaneously with broadband noise. The tones were generated and amplified (Intelligent Hearing Systems, SmartEP), mixed with noise (when appropriate) and attenuated (Med Associates, ANL918), and directed to an earphone. The tones were 5 ms in total duration (5 cycles, no plateau, exact-Blackman windowed), alternated in onset polarity, and presented at a rate of 10.9/s. The broadband masking noise was generated by a white noise generator (Med Associates, ANL-912), filtered (Wavetek model 852; 20-8000 Hz, 48dB/octave), attenuated, and mixed (Med Associates ANL 918) with the tones. Stimuli were presented monaurally via an insert earphone (ER-3A). Latencies were corrected by -0.93 ms for the acoustical delay imposed by this earphone.

Stimulus intensities were calibrated relative to the behavioral thresholds of 10 normal-hearing adults (mean = 36.1 years), one of whom also participated in the experimental

portion of this study. Behavioral thresholds were obtained in 2-dB steps using an ascending approach. The intensity of their average threshold (0 dB nHL) was determined acoustically using a 2 cm^3 acoustic coupler (Bruel & Kjaer, model DB-0138) and sound level meter (Bruel & Kjaer, Type 2209). The peak-to-peak equivalent sound pressure level (ppeSPL) for 0 dB nHL was 25 dB. Maximum output for the 1000-Hz tones was 100 dB nHL. The linearity of the attenuators was checked during the initial calibration.

The electroencephalogram (EEG) was amplified (50,000X) and filtered (10-1500 Hz, 12 dB/octave) by the SmartEP system. EEG activity was monitored on an oscilloscope by the examiner to ensure appropriate subject state. Single-channel recordings of the ABR and MLR were obtained with gold-plated cup electrodes placed at the vertex and earlobe ipsilateral to the stimulus. The SmartEP system employed a preamplifier with optical isolation and did not require a ground electrode. The inverting electrode was placed on the earlobe to minimize postauricular muscle reflex which could distort the MLR and the later components of the ABR, especially at high stimulus intensities (Stapells & Picton, 1981). Interelectrode impedances were kept below 2000 Ohms. Signal averaging was carried out using an 80-ms analysis window with no prestimulus baseline. Trials containing amplitudes exceeding ±50 μV were rejected automatically. Four replications of 1000 trials each were obtained for intensities ≥ 70 dB nHL; four replications of 2000 trials each were obtained for intensities < 70 dB nHL. At higher intensities (≥ 70 dB nHL), fewer trials were needed to evoke clear responses. Obtaining four replications for each intensity, however, provided an equal number of waveforms per intensity for subsequent response rating.

Procedures

Acoustic immittance testing was performed at the beginning of each test session to ensure normal and unchanged middle-ear status. Following audiometric evaluation, one ear was selected as the subject's test ear and used for all test conditions. The nontest ear of the normal-hearing subjects was occluded by an ER-3A earphone during the experimental procedures. Subjects were seated in a recliner chair in a double-walled sound-attenuated room, and were tested individually.

Threshold of audibility and loudness discomfort levels (LDL) for the 1000-Hz tones were obtained individually to determine the intensities for each subject/condition, and to establish the maximum level for assessment for all conditions. Threshold for the 1000-Hz tones was obtained using a modified Hughson-Westlake procedure (Carhart & Jerger, 1959) using 2-dB steps. For the masking conditions, broadband noise was simultaneously introduced to mask the 40 and 60 dB nHL tones. The masking noise level was determined individually. Noise increments of 10 dB were used initially until "no response", and then a 2-dB step descending approach was employed to obtain the effective masker (EM) level, which was defined as the masker intensity that just masks the stimulus 50% of the time. On average, noise levels of 67.3 and 82.6 dB SPL were required to mask the 40 and 60 dB nHL 1000-Hz tones, respectively. These correspond to spectrum levels of 28.4 and 43.7 dB SPL/Hz.

Maximum intensity levels for the masked and non-masked 1000-Hz tones for each subject were obtained using previously established Loudness Discomfort Level procedures (Hawkins, Walden, Montgomery & Prosek, 1987). For the normal-hearing

subjects, maximum intensity levels tested were 90 (n=9), 85 (n=2), and 80 (n=1) dB nHL. These levels were generally 5-10 dB below the subjects' LDLs. The hearing-impaired subjects gave results that allowed for response measurement at maximum levels of 90 (n=3), 85 (n=2), 80 (n=6), and 77 (n=1) dB nHL. For this group, the maximum intensity levels were generally 3-10 dB below subjects' LDLs. The levels for stimulus presentation were determined individually, based on threshold measures and LDLs. Results for 9-10 intensities were obtained for each subject, with different randomized orders of presentation for each condition. For the normal-hearing subjects, intensities generally ranged from 0 to 90 dB nHL in the non-masked condition, from 45 to 90 dB nHL in the 40 dBEM condition, and from 65 to 90 dB nHL in the higher-level masking condition (60 dBEM). The hearing-impaired subjects were tested at 8-9 intensities ranging from threshold (about 40-to-60 dB nHL) to 80 - 90 dB nHL.

Subjects were asked to sit quietly and relax, either reading a book or resting comfortably. They were not allowed to sleep, and were monitored visually, by EEG, and via intercom to ensure that they did not fall asleep. The stimulus intensity initially tested, however, was 70 dB nHL both for the normal-hearing subjects in the non-masked condition and for the hearing-impaired subjects to ensure that an individual's evoked potentials did not include excessive noise and were replicable. Test conditions (non-masked, 40 dBEM, and 60 dBEM conditions) were randomized for each normal-hearing subject.

Data analysis

Three very experienced judges familiar with ABR/MLR recordings and their measurement were asked to score independently the waveform peaks based on visual observation of the appropriate windows surrounding the wave V, Na, and Pa peaks. There were four conditions: (1) 0 dBEM or non-masked normal hearing (NH); (2) 40 dBEM and (3) 60 dBEM [noise-masked normal-hearing (NMNH)]; (4) non-masked SNHL. The ABR/MLR waveforms were evaluated by judges in random order (within and across conditions), without knowledge of the intensity of the stimulus, the masking condition, or the subject's hearing status (NH/NMNH/SNHL), and in a different condition order for each judge (Stapells et al., 1990; Oates & Stapells, 1997a,b; Wu & Stapells, submitted). Each of the raters scored the ABR and MLR peaks on a scale of 1-4, where a rating of "1" = no response, "2"= possible response, "3"= probable response, and "4"= definite response. The three judges' scores were averaged and the decision regarding the presence or absence of the electrophysiologic responses (ABR/MLR) was based on the averaged scores (Oates & Stapells, 1997a; Stapells et al., 1990; Wu & Stapells, submitted). Peaks with a combined score of ≥ 2.5 were considered responses, and measures of latency and amplitude were obtained from the average of the replications of those recordings. The measures obtained were: the latency of waves V and Pa, and the peak-to-peak amplitudes of V-V' and Na-Pa. Wave V was defined as the maximum vertex-positive peak occurring between 6 and 20 ms following stimulus onset. Wave V' was defined as the maximum negativity following wave V and within 8 ms of wave V (Nousak & Stapells, 1992; Oates & Stapells, 1997a; Stapells & Picton, 1981). Peak Na

was the largest negativity preceding Pa in the 12-20 ms window following stimulus onset. Wave Pa was defined as the maximum vertex-positive peak occurring 25-50 ms following stimulus onset (Wu & Stapells, 1994).

Threshold difference measurements were calculated by subtracting the pure-tone (in dB HL) or brief-tone (in dB nHL) behavioral thresholds from the ABR/MLR thresholds (in dB nHL). Threshold and intensity-amplitude/latency results were analyzed using descriptive statistics, linear regressions, and two- and three-way analyses of variance (ANOVA). Because not all subjects were tested at the same stimulus levels, the ANOVAs for latency and amplitude included, in some cases, estimates from individual regression equations. Where these estimates yielded negative amplitudes, a value of .001μV was assigned. Results from the ANOVAs were considered significant if $p < .01$. Where appropriate, Huynh-Feldt epsilon (ϵ) correction factors were applied to the degrees of freedom (Huynh & Feldt, 1970). Newman-Keuls post-hoc comparisons were carried out only for significant main effects and interactions, and these results were considered significant if $p < .05$.

Results

Normal-Hearing Subjects

Representative ABR/MLR recordings to 1000-Hz tones for one normal-hearing subject are displayed in Figure 1. Each column includes data obtained over several intensities for each of three conditions: 0 dBEM , 40 dBEM, and 60 dBEM noise. ABR wave V and MLR wave Pa are seen in the waveforms, from the highest intensities down to threshold (indicated by the asterisks) in all three conditions. Their latencies increase

and amplitudes decrease with decreasing intensity within each condition. For this subject, ABR and MLR thresholds were the same for each condition.

Mean latency and amplitude results for the normal-hearing subjects are plotted in Figure 2, describing the changes in the ABR and MLR with intensity for tones presented alone (0 dBEM) and in masking noise (40 and 60 dBEM). In this and subsequent figures, each plotted point represents an average of at least eight or more subjects' responses, except at the lowest (i.e., threshold) and highest (90 dB nHL) intensities within each condition, where means include 6-7 subjects' results.

The ABR/MLR intensity-latency functions on the left of this figure show that for all conditions, latencies decrease with increasing intensity. Three-way repeated-measures ANOVA [intensity (3) × wave (2) × noise condition (3)] that included the responses to 80, 70, and 60 dB nHL tones indicated that these changes in latency with intensity are significant [intensity main effect: $F(2,33) = 9.22$, $p = .001$], and *post hoc* assessment showed that this is true for all mean comparisons ($p < .05$). At higher intensities, latencies for the masked ABR and MLR approach the values for the non-masked response. Broadband noise effectively prolonged ABR and MLR latencies [noise condition main effect: $F(1,51) = 20.18$, $\varepsilon = .78$, $p < .001$], but *post hoc* assessment revealed that these effects are significant only between 0 and 40 dBEM ($p < .01$) and 0 and 60 dBEM ($p < .01$) conditions, and not between the 40 and 60 dBEM noise conditions ($p = .946$). The MLR occurs significantly later than the ABR [wave main effect: $F(1,33) = 3702.39$; $p < .001$], and there is a significant wave × noise condition interaction [$F(2,66) = 5.04$, $p = .009$]. *Post hoc* analysis indicated a different effect of

noise masking on the ABR and MLR, such that ABR latencies are significantly different between the 0 and 40 dBEM ($p < .01$) and 0 and 60 dBEM conditions ($p < .01$), whereas for the MLR, latencies are significantly different only between the 0 and 40 dBEM conditions ($p = .04$).

On the right of Figure 2 are shown the ABR and MLR intensity-amplitude functions. The same stimulus levels used to assess the effects of noise and level on response latencies were included in the three-way repeated-measures ANOVA [intensity (3) × wave (2) × noise condition (3)] to analyze the effects of intensity and noise condition on ABR and MLR amplitudes. These results show the expected significant increase in response amplitudes as stimulus level increases [intensity main effect: $F(2.33) = 18.25$, $p < .001$]. *Post hoc* analysis showed that the amplitude increase is not quite significant between 60 and 70 dB nHL tones ($p = .07$), but that all other means comparisons show significant differences ($p < .001$). Increasing noise level effectively decreases the evoked potential amplitudes [noise condition main effect: $F(1.56) = 117.64$, $\epsilon = .85$, $p < .001$], and this is true for all means comparisons ($p < .001$). The results of the ANOVA also show that MLR amplitudes are significantly larger than those for the ABR [wave main effect: $F(1.33) = 8.69$, $\epsilon = .85$, $p = .006$].

The mean (± standard deviation) ABR and MLR thresholds in the 0 dBEM condition are 3.3 (± 5.4) and 4.2 (± 4.7) dB nHL, respectively. With increasing masking, results show the expected shift in threshold levels: in the 40 dBEM condition, average ABR and MLR thresholds are 40.4 (± 3.3) and 41.7 (± 5.4) dB nHL, respectively. In the 60 dBEM

condition, mean thresholds are 59.2 (± 3.6) dB nHL for the ABR and 59.9 (± 4.8) dB nHL for the MLR.

The relationship between the behavioral brief-tone and AEP thresholds for the normal-hearing group for the three masking conditions is shown in Figure 3. There is excellent agreement between each of the electrophysiologic responses and the behavioral measures. Ninety-one percent (33/36) of the ABR and 83% (30/36) of the MLR thresholds are within 5 dB of the behavioral thresholds for the short-duration tones. This point is made further by comparing threshold agreement between the two evoked potential measures. In almost two-thirds of all conditions (22/36), threshold levels for the ABR and MLR are equal. In the remaining 1/3, the ABR vs MLR threshold differences are 5 dB (N=10), 7 dB (N=2), and two subjects' thresholds in the 40 dBEM condition show a difference of 10 dB. The correlations for threshold estimates for (1) ABR vs behavioral threshold for brief tones, (2) MLR vs behavioral threshold for brief tones, and (3) ABR vs MLR thresholds are .98 or better.

In order to compare threshold estimation between all conditions, difference measurements were calculated for these normal-hearing subjects by subtracting their behavioral threshold for brief-tones from that obtained electrophysiologically. The average difference measurements, shown in Table 1, decrease slightly, but significantly, with increasing masker level [main effect noise condition: $F(2,22) = 6.40$, $\epsilon = .88$, $p = .007$]. *Post hoc* analyses show threshold difference measurements decrease significantly only between the 0 and 40 and 0 and 60 dBEM noise conditions. The decrease in behavioral-electrophysiological threshold difference scores, or improved threshold

estimation with increasing masking, indicates that ABR/MLR thresholds are not affected by increasing masker level as much as are behavioral thresholds. There is no difference in the difference scores between waves [$F(2,22) = 1.79$, $p = .208$], and no condition × wave interaction [$F(2,22) = .05$, $p = .950$].

Sensorineurally-Hearing-Impaired Subjects

Figure 4 displays waveforms from another normal-hearing subject (0 dBEM condition) and two sensorineurally-impaired subjects, one with a mild sloping configuration and one with a moderately-severe flat configuration of hearing loss. The results for these two hearing-impaired subjects are typical of those for the sensorineurally hearing-impaired (SNHL) group. Six subjects had gradual sloping configuration, and six subjects had flat configuration of hearing loss.

Mean latency and amplitude results for the hearing-impaired subjects are plotted in Figure 5. Within each graph, solid symbols show the average intensity-latency and intensity-amplitude functions of the hearing-impaired subjects. Also plotted are the normal non-masked mean and the standard deviation of their results from Figure 2, as well as the mean results from the normal-hearing group in the 40 and 60 dBEM conditions. Three-way ANOVA [group (2) × wave (2) × intensity (3)] assessed the effects of intensity and group (non-masked NH and SNHL) on the latencies and amplitudes of the ABR/MLR. These analyses included measures from the responses to 70, 55, and 40 dB nHL tones for nine subjects from each group (not all of the subjects had a response at 40 dB nHL). These mild (40 dB nHL), moderate (55 dB nHL), and high (70 dB nHL) stimulus levels were chosen because they are representative of the

range of intensities assessed, and include levels that are of interest for evaluating the differences between these two groups. The results of the ANOVA reveal, as expected, that ABR and MLR latencies decrease with increasing intensity [main effect intensity: $F(1,26) = 40.66$, $\epsilon = .80$, $p < .001$]. *Post hoc* analysis revealed significant differences between all means comparisons ($p < .02$). ABR/MLR latencies are not significantly different between groups [main effect for group: $F(1,16) = 0.43$, $p = .523$], which is probably due to the fact that MLR latencies for the SNHL subjects are the same or shorter than those of the normal listeners, whereas their ABR latencies are prolonged relative to the normal response. When latencies are collapsed across groups, the wave × intensity interaction term is significant [$F(2, 32) = 7.19$, $p = .003$], such that ABR latencies, which are shorter than MLR latencies [wave main effect: [$F(1,16) = 786.45$, $\epsilon = .80$, $p < .000$], decrease at a faster rate with increasing intensity than do MLR latencies. *Post hoc* assessment revealed significant differences between all means comparisons ($p < .05$), except between 70 and 55 dB nHL for the MLR ($p = .10$).

The latencies of the hearing-impaired subjects' recordings to 80, 70, and 60 dB nHL tones were also compared to the normal listener's responses in the 40 and 60 dBEM conditions [group (2) × wave (2) × intensity (3)]. Two 3-way ANOVAs assessed the effects of intensity and group on ABR/MLR latencies of (1) the normal listeners in the lower-level noise condition (40 dBEM) and the SNHL subjects, and (2) the normal-hearing subjects in the higher-level noise condition (60 dBEM) and the SNHL listeners. As expected, the results show that latencies decrease significantly with increasing intensity [main effect for intensity: 40 dBEM & SNHL: $F(1,42) = 82.17$, $\epsilon = .96$,

$p < .001$; 60 dBEM & SNHL: $F(2,44) = 74.45$, $\epsilon = 1.00$, $p < .001$], and this is true for all means comparisons ($p < .001$). Although the differences between groups is not significant [main effect group: 40 dBEM and SNHL: $F(1,22) = 4.215$, $p = .052$; 60 dBEM and SNHL: $F(1,22) = 4.54$, $p = .045$], there are significant group × intensity interactions [40 dBEM and SNHL: $F(1,42) = 10.04$, $\epsilon = .96$, $p < .001$; 60 dBEM and SNHL: $F(2,44) = 12.97$, $\epsilon = 1.00$, $p < .001$], indicating a different effect of intensity on the ABR/MLR latencies between groups (i.e., 40 dBEM and SNHL, and 60 dBEM and SNHL). *Post hoc* analyses reveal that the noise-masked normal-hearing (NMNH) subjects' latencies are more delayed with decreasing intensity compared to the results for the SNHL listeners ($p < .001$).

In the right panels of Figure 5 are shown the intensity-amplitude functions. Three-way ANOVA that assessed the effects of intensity and hearing group (i.e., normal and sensorineurally-impaired listeners) on the ABR and MLR amplitudes for 70, 55, and 40 dB nHL tones show, as expected, that ABR and MLR amplitudes increase with increasing intensity [main effect intensity: $F(2,32) = 47.32$, $\epsilon = 1.00$, $p < .001$], and that MLR amplitudes are larger than those for the brainstem response [$F(1,16) = 15.75$, $\epsilon = 1.00$, $p = .001$]. There is no significant difference between the amplitudes of the normal listeners and the SNHL subjects [main effect group: $F(1,16) = .98$, $p = .337$]. However, when amplitudes are collapsed across waves, there is a significant group × intensity interaction [$F(2,32) = 5.35$, $\epsilon = 1.00$, $p = .01$], such that the impaired listeners' amplitudes tend to increase at a faster rate with increasing intensity than do those of the normal-hearing subjects. *Post hoc* assessment revealed that the SNHL group's

amplitudes at 70 dB nHL are not significantly different from the normal listeners' results at the same intensity ($p = .392$), nor are there significant differences between the impaired listeners' amplitudes at 55 dB nHL and the normal-hearing subjects' results at 55 ($p = .226$) and 40 dB nHL ($p = .590$). All other interactions are not significant [intensity × wave $F(2,32) = .523$, $p = .598$; group × wave $F(1,16) = .526$, $p = .479$; intensity × group × wave $F(2,32) = 1.061$, $p = .358$].

Two 3-way ANOVAs [group (3) × wave (2) × intensity (3)] evaluated the effects of intensity and group on the ABR/MLR amplitudes of (1) the normal listeners in the lower-level noise condition (40 dBEM) and the SNHL subjects, and (2) the normal-hearing subjects in the higher-level noise condition (60 dBEM) and the SNHL listeners for 80, 70, and 60 dB nHL tones. There are significant group effects [40 dBEM and SNHL: $F(1,22) = 14.45$, $p < .001$; 60 dBEM and SNHL: $F(1,22) = 31.85$, $p < .001$], such that ABR/MLR amplitudes for the SNHL subjects are significantly larger than the masked normal listeners' responses ($p < .001$). There is also the expected significant increase of ABR/MLR amplitudes with increasing intensity [main effect intensity: 40 dBEM and SNHL: $F(1,39) = 51.00$, $\epsilon = .91$, $p < .001$; 60 dBEM and SNHL: $F(1,35) = 59.03$, $\epsilon = .81$, $p < .001$], which was true for all mean comparisons ($p < .01$). There are no significant interactions.

The relationship between the long-duration pure-tone behavioral and ABR/MLR thresholds for the non-masked normal-hearing and sensorineurally hearing-impaired subjects is shown in Figure 6a, and on the right, in Figure 6b, are shown the results for the behavioral short-duration and ABR/MLR thresholds. Threshold agreement between the

behavioral and electrophysiologic measures for 1000-Hz tones, both for normal-hearing and hearing-impaired subjects, is excellent. In all, 98% of the non-masked ABR/MLR thresholds are within 10 dB of the pure-tone behavioral thresholds for these 24 subjects. Eighty-three percent of the ABR and 79% of the MLR thresholds are within 5 dB of these behavioral thresholds. Only one (ABR-SNHL) threshold estimation is 15 dB different than the pure-tone behavioral threshold. There also is good agreement between the two evoked potential threshold measures for the group of hearing-impaired subjects (SNHL): 60% (7/12) of their ABR and MLR threshold levels are the same; the remaining 40% (5/12) show only a 5-dB difference in threshold between the two evoked potentials. Across both groups and all conditions, including the noise conditions, 92% (44/48) of the ABR and MLR thresholds are within 5 dB of each other.

Table 2 lists the mean sensorineurally hearing-impaired subjects' threshold difference scores for brief-tones (ABR/MLR threshold minus brief-tone behavioral threshold) compared to those of the normal listeners (non-masked). There are no differences, as the mean difference scores are the same for both groups. For the normal-hearing (non-masked) and SNHL subjects, ABR and MLR accuracy in estimating their pure-tone thresholds was determined by subtracting the subjects' behavioral pure-tone thresholds from their electrophysiologic thresholds. These mean difference measurements for each group and for all subjects combined are also shown in Table 2. On average, threshold difference scores were slightly lower for the hearing-impaired group (SNHL), but this difference is not significant between groups [$F(1,22) = 1.165$, $p = .292$] or AEP measure [$F(1,22) = 1.571$, $p = .223$].

Discussion

This study assessed several issues concerning ABR/MLR threshold estimation for 1000-Hz tones, and their amplitude and latency changes with increasing intensity. Comparisons of these measures was made between (i) non-masked and masked normal-hearing listeners, (ii) normal-hearing and sensorineurally hearing-impaired listeners, and (iii) masked normal-hearing and non-masked sensorineurally hearing-impaired listeners. Briefly, the results from this study indicate no difference between the ABR and MLR in their accuracy of estimating behavioral hearing threshold in normal-hearing, masked normal-hearing, and sensorineurally hearing-impaired adults, at least for 1000-Hz tones. There also is no difference between the ABR and MLR amplitude and latency changes with increasing intensity for the non-masked normal-hearing subjects. Broadband masking noise presented simultaneously with the stimuli to the normal-hearing subjects effectively shifts their thresholds to levels that are in the range of those of the sensorineurally hearing-impaired subjects. ABR/MLR recordings from both groups of subjects with elevated thresholds, however, reveal differences between the ABR and MLR latency and amplitude changes with intensity, compared to the non-masked normal listeners' responses. ABR/MLR suprathreshold results for the noise-masked listeners are different also from those of the subjects with cochlear impairment.

Threshold Estimation

In this study, no difference was found between the ABR and MLR in their accuracy of estimating behavioral threshold. For the 24 normal-hearing and hearing-impaired listeners, 98% of the pooled ABR and MLR threshold estimations are within 10 dB of

their pure-tone behavioral thresholds, and 81% are within 5 dB. These results are similar to those of previous studies in which similar recording techniques (simultaneous ABR/MLR) and stimuli were used, and which showed no significant differences in threshold estimations between the ABR and MLR in normal-hearing (Beattie & Boyd, 1984; Suzuki et al., 1981; Wu & Stapells, submitted) and hearing-impaired adults (Suzuki et. al., 1981). For example, Suzuki et al. (1981) reported mean ABR vs MLR threshold differences of 5, 0, 5, and 10 dB from recordings to 500, 1000, 2000, and 4000-Hz tones, respectively, in normal-hearing adults. More recently, Wu and Stapells (submitted) reported findings for the ABR/MLR to clicks, and to 500 and 2000-Hz tones and also demonstrated no significant difference in threshold between the ABR and MLR for their normal-hearing adult subjects. Their mean ABR vs MLR threshold differences were 7 dB for 500 Hz, 2 dB for 2000 Hz, and 3 dB for clicks.

Suzuki and colleagues (Suzuki, Hirai & Horiuchi,1981) also recorded ABR/MLRs from subjects with various type and degree of hearing loss. They found that for their subjects with either gradually sloping or flat sensorineural hearing loss, ABR and MLR threshold estimations were within 10 dB of their behavioral pure-tone thresholds. In the present study, all the hearing-impaired subjects had flat or gradually sloping sensorineural hearing loss, and 92% of their ABR/MLR threshold estimations were also found to be within 10 dB of the behavioral pure-tone threshold.

The findings from this study directly comparing the ABR and MLR re: their accuracy in estimating threshold for 1000-Hz tones do not support the suggestion by some investigators that one or the other of these responses yields better threshold estimation for

low-frequency tones (Kavanagh et. al., 1984; Musiek & Geurkink, 1981; Scherg & Volk, 1983; Stapells et al., 1984b). Neither do the findings from this study support the notion that the MLR is more useful for threshold detection due to the larger amplitude of the Na-Pa component compared to the ABR V-V' (Musiek & Geurkink, 1981; Scherg & Volk, 1983). Larger amplitudes for the MLR were also observed in our data, but this factor did not yield better threshold estimations for the MLR compared to the ABR. Both are equally good for assessing hearing thresholds in older children (e.g., Kraus, Smith, Reed, Stein & Cartee, 1985) and adults, but likely not for infants and young children (e.g., Stapells et al., 1988).

Masked Threshold Estimations, normal-hearing subjects. This study appears to be the first to provide results comparing behavioral and ABR/MLR measures of threshold shifts produced by broadband noise in normal listeners. Several researchers who have studied the effects of simultaneously presented broadband noise on the click- or tone-evoked ABR or MLR held stimulus intensity constant and varied the noise level (Beattie et al., 1992, 1994; Burkard & Hecox, 1983a,b, 1987; Gott & Hughes, 1989; Hecox, Patterson & Birman, 1989; Owen & Burkard, 1991; Picton et. al., 1979), or varied both the stimulus and noise at a fixed signal-to-noise ratio (Stapells, 1984). None of these reports, however, included the individual or group masked ABR or MLR threshold estimations of their subjects' behavioral masked thresholds.

The ipsilateral broadband masking noise effectively shifted the normal-hearing subjects' behavioral thresholds for brief tones to 40 and 60 dB nHL levels, and the electrophysiologic results show the same threshold elevations. That is, the ABR and

MLR gave highly accurate threshold estimates of the masked behavioral thresholds for brief tones. Noise-masked thresholds are within 5 dB of the behavioral threshold for 100% of the ABR and 83% of the MLR estimates. The accuracy in threshold estimation found in the masked normal data is likely a result of the fact that we determined masker levels for both the behavioral and electrophysiologic measurements for each individual rather than using a mean masker level. Comparison of the masked electrophysiologic threshold measures also reveals no differences between the ABR and MLR for threshold estimation. All of the ABR and MLR threshold differences are within 10 dB for the masking conditions, 87% were ≤5 dB, and 62% are the same (0 dB difference).

Effects of increasing intensity on the ABR/MLR

Non-masked normal-hearing and sensorineurally-impaired subjects. The suprathreshold measures obtained in this study show that with intensity, latencies and amplitudes of the non-masked normal-hearing listeners change similarly for the ABR and MLR. ABR and MLR latencies decrease and their amplitudes increase with increasing intensity at about the same rate. These results for 1000-Hz tones are consistent with previous investigations that reported the effects of increasing intensity on normal listeners' latencies and amplitudes for the click- and tone-evoked ABR (e.g., Picton et al., 1979; 1974; Stapells, 1984b), the MLR (e.g., Madell & Goldstein, 1972; McFarland et al., 1977; Thornton et al., 1977), and the ABR/MLR (Picton et al., 1974; Stapells & Wu, submitted; Suzuki et al., 1981; Yamada, Yamane & Kodera, 1977).

The effects of sensorineural hearing loss are seen in both the ABR and MLR data. At the brainstem level, wave V latencies are prolonged slightly and, in the 40-60 dB nHL

range, the ABR V-V' amplitudes are reduced compared to the non-masked normal listeners' responses. At high intensities (≥65 dB nHL), the results for the sensorineurally impaired subjects show ABR amplitude and latency values in the normal range. The rapid decrease in the latency of wave V with increasing intensity in subjects with sensorineural hearing loss has been well established (e.g., Hall, 1992 pp. 356; Hyde,1985; Picton et al., 1977; Stapells et al., 1994), and this "abnormal" intensity-latency function has been used clinically as an indicator of cochlear hearing loss (Galambos & Hecox, 1978). The effects of sensorineural hearing loss on ABR amplitudes has received scant attention in the literature. However, smaller amplitude values in these subjects have been reported (e.g., Fowler & Durrant, 1994), and presumably are due to the loss of some neural contributions.

In contrast, MLR wave Pa latencies for the hearing-impaired listeners are the same as those of the normal response, and their latencies decrease with intensity increase at the same rate as the normal response. Their Na-Pa amplitudes are smaller only at threshold compared to the normal response. At moderate-to-high intensities, the Na-Pa amplitudes of the hearing-impaired subjects are the same or larger, and their amplitudes increase at a faster rate than those of the normal listeners. These findings are consistent with previous reports on the MLR in subjects with hearing loss (McFarland et al., 1977; Squires & Hecox, 1983; Suzuki et al., 1981). For example, McFarland et al. (1977) obtained MLRs to 500, 1000, and 3000 Hz tones from ten adults with various degree, configuration, and type (conductive, mixed) hearing loss, and from normal-hearing subjects. For stimulus levels of 0 - 50 dBSL, they noted that the results from the hearing-impaired listeners

were "essentially indistinguishable . . . from comparable responses elicited from normal-hearing subjects". They also reported larger amplitudes for the hearing-impaired subjects compared to those of the normal listeners. The MLR, therefore, does not show the same effects of peripheral (i.e., cochlear) pathology as does the ABR.

The effects of cochlear hearing loss on the ABR and MLR may be understood from our knowledge of the physiologic changes that occur as a consequence of cochlear pathology and the generator sources that subserve these evoked potential responses. It is known, for example, that these evoked potentials derive from different generators within the auditory system. Specifically, the ABR reflects contributions from the cochlea, auditory nerve, and brainstem to the level of the inferior colliculus, sites specific to the primary auditory pathways (Allen & Starr, 1978; Legatt, Arezzo & Vaughan, 1988; Møller, 1994; Moore, 1987). The characteristics (longer latencies and smaller amplitudes) of the impaired subjects' recordings at and just above threshold likely reflect cochlear disease and/or damage, which cause a decrease in the number of active elements, decreased synchronization of the neuroelectric sources, and/or effective reduction in stimulus intensity (Davis, 1983; Durrant & Fowler, 1994; Salvi et al., 1983). These effects are attributed to the loss of active cochlear mechanisms due to outer hair cell loss/damage. Loss of the "cochlear amplifier" results in broadening of cochlear tuning and threshold elevation (Davis, 1983; Geisler, 1998, pg 281; Saunders et al., 1991), decreased impulse response times (e.g., Ruggero, 1994), and shorter cochlear response times in listeners with cochlear impairment (Don, Ponton, Eggermont & Kwong, 1998). Shorter latencies in response to suprathreshold stimuli have been documented by

Eggermont and colleagues in the derived response (i.e, narrow-band) recordings of subjects with cochlear impairment for both VIII th nerve (Eggermont, 1979) and brainstem (Don et al., 1998) responses compared to those of normal-hearing listeners. At stimulus intensities of at least 20-30 dB above threshold, a sufficient number of neural elements are activated to yield near-normal ABR latency and amplitude values in subjects with mild-to-moderate sensorineural hearing impairment.

The MLR reflects source activity from bilateral generators in primary auditory cortex (Hashimoto, Mashiko, Yoshikawa, Muzuta, Imada & Hayashi, 1995; Kuriki, Nogai & Hirata, 1995; Liégeois-Chauvel, Badier, Marquis, Chauvel, 1994; Scherg & von Cramon, 1986). At this cortical level, these evoked potentials include contributions from the periphery and brainstem as well as from neural sources rostral to and including the inferior colliculus. The temporal and spatial distribution of source activity at these levels is more complex and not completely understood (e.g., Heil, 1997a,b; Lockwood, Salvi, Coad, Arnold, Wack, Murphy & Burkard, 1999; Webster, 1992). The normal latencies and amplitudes of the impaired listeners' MLRs probably reflect, in part, the neural redundancy of the central nervous system. That is, due to neural branching as the auditory pathway is ascended, small numbers of active cells at the periphery can elicit activity in very large numbers of more central units (e.g., Heil, Rajan & Irvine, 1994; Lockwood et al., 1999), a number sufficient to yield normal MLR responses in hearing-impaired listeners. It may be also that the structural and physiologic changes in central neurons resulting from peripheral pathology (e.g., Bilak, Kim, Potashner, Bohne & Morest, 1997; Melcher, Guinan, Knudson & Kiang, 1996; Qui, Salvi, Ding & Burkard,

2000; Willott, Bross & McFadden, 1994) alter the processing of the converging excitatory and inhibitory inputs in these higher loci (e.g., Qui et al., 2000; Wang et al., 1996), thereby resulting in normal latency and amplitude measures of gross evoked potentials in impaired listeners (Gerken, 1993). The MLR results in this study show that tone levels at and just above threshold provide sufficient stimulation to elicit "normal" early cortical evoked potentials in adults with mild-to-moderate cochlear loss of sensitivity. Similar findings have been reported for the slow cortical auditory evoked potential (CAEP) (Hyde, 1994), which also derives from generator sources in primary auditory cortex (e.g., Picton, Alain, Woods, John, Scherg, Valdes-Sosa, Bosch-Bayard & Trujillo, 1999). Further, the MLR results for the hearing-impaired listeners in this study support the notion advanced by Gerken (1993) that the human MLR Pa in subjects with sensorineural hearing loss would likely show similar "response enhancement" (i.e., increase in amplitude) as that seen in recordings from within cortical and upper brainstem (inferior colliculus) structures in animals with cochlear damage/hearing loss (Gerken, 1992; Gerken, Saunders & Paul, 1984; McFadden, Kasper, Ostrowski, Ding & Salvi, 1998; Salvi, Powers, Saunders, Boettcher & Clock, 1992; Salvi, Wang & Ding, 2000; Syka, Rybalko & Popelar, 1994). The differences between the ABR and MLR amplitude/latency results for supra threshold stimuli in listeners with SNHL contrast with the similar threshold accuracy results, and these findings may have important clinical applications/implications. Further studies in other groups of subjects with various types and degree of hearing loss are needed.

Masked ABR/MLR with intensity; normal-hearing subjects. The results for suprathreshold recordings show that simultaneous masking caused an increase in the latencies and a decrease in the amplitudes of the ABR and MLR, consistent with previous reports of the effects of broadband masking on the ABR to tones (Beattie et al., 1992; Burkard & Hecox, 1983a,b; 1987; Picton et al., 1979) and the MLR to clicks (Gott & Hughes, 1989). The masker effectively delayed both responses, but there is no further increase in latency when masker level was increased from 40 to 60 dBEM for either the ABR or MLR. ABR latencies are more prolonged by masking, showing an average delay of about 3 ms for 40-70 dB nHL tones compared to masked MLR latencies, which were prolonged by only about 1 ms in this intensity range. In contrast, masked amplitudes are reduced to a similar degree for both the ABR and MLR, and both become smaller with increasing noise masking.

With increasing stimulus level, masked ABR/MLR latencies decrease and their amplitudes increase, and approach those of the responses in Quiet at high ($\geq$75 dB nHL) stimulus intensities due to the increasing signal-to-noise ratio and, possibly, upward spread of excitation for these high-level stimuli. At the highest intensities (i.e., 80-90 dB nHL), masked ABR latencies approach non-masked values, but the early cortical MLR latencies at these high SPL levels are *shorter* than the non-masked response. ABR/MLR amplitudes, however, approach or exceed normal values only at the highest (90 dB nHL) stimulus intensity assessed.

Differential effects of broadband masking noise on the behavior of wave Pa compared to that seen for wave V were also reported by Gott and Hughes (1989) who recorded the

ABR/MLR to 105 dBSPL clicks and simultaneously presented broadband masking noise of 0, 55, and 75 dB SPL. They found that, for the masked ABR, amplitudes decreased and latencies were prolonged with increasing noise level, but there was no significant effect of increasing noise on MLR wave Pa amplitude or latency. Gott and Hughes (1989) employed a comparable masker level (75 dB SPL) as that used in the present study in the 40 dBEM condition (67 dB SPL), yet failed to show a masker effect on MLR amplitude. This may be due to their choice of peaks used to measure the amplitude of this response or possibly, to the differences in stimuli used (clicks vs tones). However, in the waveforms shown in their Figure 3, the amplitude of the masked MLR appears to be smaller than the non-masked response. At a SNR of +30 dB, Gott and Hughes' results and those of the present study show that, relative to the non-masked response, masked ABR latencies are prolonged whereas MLR latencies are not. Galambos and Makeig (1992) recorded the click-evoked ABR and 40-Hz steady-state response (MLR-SSR) with ipsilateral broadband noise. The 40-Hz response represents the superimposition of several waves that make up the ABR/MLR (Galambos, Makeig & Talmachoff, 1981; Stapells et al., 1988). While not directly comparable to the present study, their data reveal that for moderate noise levels (i.e., ≥ 50 dBSPL), masked ABR and MLR-SSR amplitudes decrease, wave V latency increases, and the large phase change seen in the masked MLR-SSR corresponds to a *decrease* in the timing of this response. They suggested that these MLR-SSR phase changes may represent "progressive changes" in signal level processing that arise at sites central to where wave V is generated in the brainstem.

The effects of masking on the behavior of waves V and Pa behavior are attributable to both peripheral and central physiological mechanisms. Peripheral mechanisms include mechanical suppression in the cochlea and VIII[th] nerve which causes a shift in threshold to higher levels (Costalupes, Young & Gibson, 1984; Ruggero, Robles & Rich, 1992), excitation of nerve fibers by the noise signal which increases their baseline discharge rate and reduces or obscures the response to the tone (Smith, 1979), and adaptation to the continuous noise which compresses the neural response by reducing the saturation discharge rate of nerve fiber response to the tone (Delgutte, 1990; Ruggero, 1992). As the noise background is increased in level, the operating range shifts to a higher level, the baseline discharge rate increases, and the saturated discharge decreases (Costalupes et al., 1984; Palmer, 1995). These masker effects are seen in both evoked potential peaks. Masked ABR/MLR amplitudes are reduced and become smaller with increasing noise level, reflecting the decrease in both the effective stimulus level and the number and synchrony of neuroelectric sources contributing to the responses as masker level increases. These effects of the noise that cause a decrease in cochlear/VIII[th] nerve output are also observed in the ABR/MLR response delay, although the peripheral masker mechanisms involved are reflected to a greater extent in the more caudal (i.e., ABR) response, which derives from VIIIth nerve and lower brainstem generator sources (e.g., Legatt et al., 1988; Zaaroor & Starr, 1991). Masked MLR latencies are not as prolonged as those of the ABR, possibly due to timing differences between cochlear and central neuron responses to these short-duration stimuli (Heil & Irvine, 1997; Phillips & Burkard, 1999), and/or to central masker mechanisms that reduce the effects of the noise at the

cortical level (e.g., Phillips, 1987; and see below). ABR/MLR latencies are also prolonged due to cochlear place-shift mechanisms (Burkard & Hecox, 1983b). The broadband masker effectively removes the responses originating from higher- and lower-frequency regions that contribute to the non-masked response, thereby limiting the region of the cochlea from which the masked response can emanate (Burkard & Hecox, 1983a,b; Picton et al., 1979). The result of the shift in response from more apical regions of the basilar membrane is a response delay, which is reflected in both the ABR and MLR latency results. Masked ABR and MLR latencies, however, are not delayed further with increasing noise level, possibly due to central masker mechanisms.

In central auditory nuclei, suppression effects are also seen in the response to tones in noise (e.g., Gibson, Young & Costalupes, 1985; Rhode & Greenberg, 1994), but at these levels, inhibitory effects shift the rate-level functions to higher levels at a faster rate than that observed in the cochlear nerve (e.g., Arle & Kim, 1991; Gibson et al., 1985). Some cells in the cochlear nucleus show excitation to the noise, but in others, suppression effects are reduced (e.g., Rhode & Greenberg, 1994) or signal processing is facilitated by inhibitory effects (e.g., Burkard & Palmer, 1997), resulting in enhancement of signal processing. Further, at both the inferior colliculus and cortex, the compression of the neural response to the tone is less extreme than in the VIIIth nerve because the noise is not excitatory at these levels, the reduction of the saturated firing rate is smaller (Phillips, 1990; Phillips & Cynader, 1985; Rees & Palmer, 1988), and the distribution of source activity is larger than in the cochlear nerve (Heil & Irvine, 1997). These effects of central mechanisms that shape the response of central neurons to tones in noise may explain, in

part, why the masker effect on MLR latency, which derives from generators rostral to those that generate the ABR wave V, is less than that seen for the ABR latency, and also why masked MLR latencies to high-intensity tones (i.e., 80-90 dB nHL) are *shorter* than the non-masked response. These differential effects of the masker on ABR and MLR latencies may be due to the same (central) mechanisms that affect the early cortical response in subjects with cochlear impairment (Gerken, 1993). That is, the greater response delay for the masked ABR vs that seen for the MLR relative to the non-masked response is similar to the results of the subjects with sensorineural hearing loss. Masked MLR amplitude was enhanced only at a high SNR (SNR = 50 dB) in the 40 dBEM condition, which suggests that central enhancement or facilitory effects may be dependent on the level of the noise masker and/or the signal-to-noise ratio (Burkard & Hecox, 1983a). Further study of the masked ABR/MLR in humans is needed.

Effects of increasing intensity on the ABR/MLR: Masking vs SNHL. Overall, the major differences between the noise-masked normal-hearing and hearing-impaired ABR/MLR results with intensity are longer delays in latencies and greater decreases in amplitude for the noise-masked normal-hearing subjects. These differences are not surprising given that the decrease in hearing sensitivity, although similar by degree, arises from different etiologies for these two groups of subjects. A general finding, however, is that ABR latencies are more prolonged compared to those for the MLR (relative to the normal non-masked response) for both groups of hearing-impaired subjects. ABR/MLR amplitudes are reduced to a greater degree by masking than by cochlear impairment, but it remains to be seen whether or not a similarly greater decrease in response amplitude is

seen also in subjects with greater hearing loss. These results may have important research and clinical implications. For example, a recent study investigating the effects of broadband masking on the cortical auditory event-related potentials to supra threshold speech stimuli found increases in the latencies and decreases in the amplitudes of N1, P2, and P3 with increasing masker level (Whiting, Martin & Stapells, 1998). These authors suggested that their findings may be applicable clinically in that similar results may be seen in subjects/patients with sensorineural hearing loss. As the present study indicates, this may be true only for threshold results. Future investigations of cortical event-related potentials for clinical application, such as the MMN or P3, require examination of these waves from subjects with various degrees, levels, and types of hearing loss.

Summary

The similarity between the tone-evoked ABR and MLR for threshold estimation found in this study indicates that either or both responses provide equally accurate and valid estimates of threshold in normal-hearing and sensorineurally hearing-impaired adults, at least for 1000-Hz tones. Therefore, one may choose to record the ABR or MLR alone, or simultaneously to tonal stimuli from adults, and obtain reliable predictive measures of threshold. Recording paradigms/technological advances now allow for this type of recording and analysis which has been recommended for routine clinical use. Overall, the findings from this study provide evidence that (i) the ABR and MLR are equally effective in measuring hearing sensitivity in adults, (ii) threshold shifts imposed by broadband masking in normal listeners are the same for both behavioral and electrophysiologic measures, (iii) sensorineural hearing loss and masking affect the ABR

and MLR intensity-amplitude/latency functions differently relative to the normal listeners' non-masked responses, and (iv) ABR and MLR latency and amplitude measures with intensity are affected differently by broadband masking noise compared to sensorineural hearing loss, with one exception: the rate of increase in amplitude of both the ABR and MLR as intensity increases is similar for both noise-masked normal-hearing and sensorineurally hearing-impaired subjects. It may be that ABR/MLR intensity-amplitude functions reflect the neural correlate to loudness perception, a possibility that will be explored in the following paper.

Acknowledgments

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Table 1. Mean (and standard deviation, s.d.) brief-tone difference measures from the normal-hearing subjects to1000-Hz tones presented in three levels of broadband noise.

Noise Condition	Difference Measures (dB) ABR	MLR
0 dBEM		
Mean	4.2	5.0
s.d.	4.7	3.7
40 dBEM		
Mean	0.4	1.7
s.d.	3.3	5.4
60 dBEM		
Mean	-0.8	-0.1
s.d.	3.6	4.8

dBEM = Effective Masker level

Difference measures = ABR/MLR threshold (dB nHL) minus brief-tone behavioral thresholds (dB nHL).

Table 2. Mean difference measures (dB) for 1000-Hz non-masked tones obtained from the normal-hearing and hearing-impaired subjects.

	Normal-hearing (N = 12)		Hearing-Impaired (N = 12)		All Subjects (N = 24)	
	ABR	MLR	ABR	MLR	ABR	MLR
Difference scores: brief tones						
Mean	4.2	5.0	4.2	5.0	4.2	5.0
s.d.	4.7	3.7	4.2	3.7	4.3	3.6
Difference scores: long-duration tones						
Mean	0	0.8	-2.5	-1.7	-1.3	-0.4
s.d.	5.2	5.6	6.6	6.2	6.0	5.9

Difference measures = ABR/MLR threshold (dB nHL) minus brief-tone/long-duration behavioral thresholds (dB nHL/dB HL).

Figure Captions

Figure 1. Representative waveforms obtained from one normal-hearing subject. 1000-Hz brief tones were presented over a range of intensities in quiet (left column), or simultaneously with broadband noise that Effectively Masked (dBEM) 40 dB nHL (center column), and 60 dB nHL (right column) tones. Each waveform is the average of 4000 (≥70 dB nHL) or 8000 (<70 dB nHL) trials. The ABR wave V and MLR wave Pa are labeled for a high-level response in all three conditions. Threshold levels (in dB nHL) determined by judges for each condition are denoted with an asterisk. Positivity at the vertex is shown as an upward deflection.

Figure 2. The effects of stimulus intensity on the normal-hearing subjects' ABR wave V (lower panels) and MLR wave Pa (upper panels) latencies (left) and amplitudes (right) in the three masking conditions. The dotted lines show ±1 standard deviation for the mean non-masked responses.

Figure 3. Threshold estimation for normal-hearing subjects using the ABR and MLR to 1000-Hz brief tones presented in quiet, and in broadband noise. The Effective Masker (dBEM) noise level was determined individually, producing behavioral threshold shifts of 40 and 60 dB nHL. Diagonals (solid lines) represent perfect ABR behavioral threshold correspondence and are not regression lines. Points with multiple subjects have symbols offset (± 2 dB) to show clearly the overlapping subjects.

Figure 4. Representative ABR/MLR recordings to 1000-Hz tones over a range of intensities from one normal-hearing subject (left column), and two sensorineural hearing-impaired subjects. The waveforms in the center column were obtained from a subject with mild sloping configuration of hearing loss, and in the right column are waveforms obtained from a subject with moderately-severe flat configuration of hearing loss. Each waveform is the grand mean of 4000 (≥70 dB nHL) or 8000 (<70 dB nHL) trials. Threshold levels for each subject are denoted by symbols: * = ABR/MLR; ** = ABR; ~ = MLR.

Figure 5. The effects of stimulus intensity on the sensorineurally-impaired subjects' ABR wave V (lower panels) and MLR wave Pa (upper panels) latencies (left) and amplitudes (right). The solid lines indicate the mean normal-hearing non-masked response, with ± 1 s.d. as shown by the dotted lines. The smaller open symbols are those of the mean normal masked responses from Figure 2.

Figure 6. Threshold estimation using the ABR and MLR to 1000-Hz tones. (A) The relationship between the electrophysiologic (in dB nHL) and behavioral pure-tone (in dBHL) thresholds are shown for the normal-hearing and hearing-impaired subjects. (B) The relationship between the electrophysiologic (in dB nHL) and behavioral thresholds for short-duration tones (dB nHL) are shown for the normal-hearing and hearing-impaired subjects. The diagonals (solid lines) represent perfect ABR/MLR/behavioral threshold correspondence and are not regression lines.

Figure 1

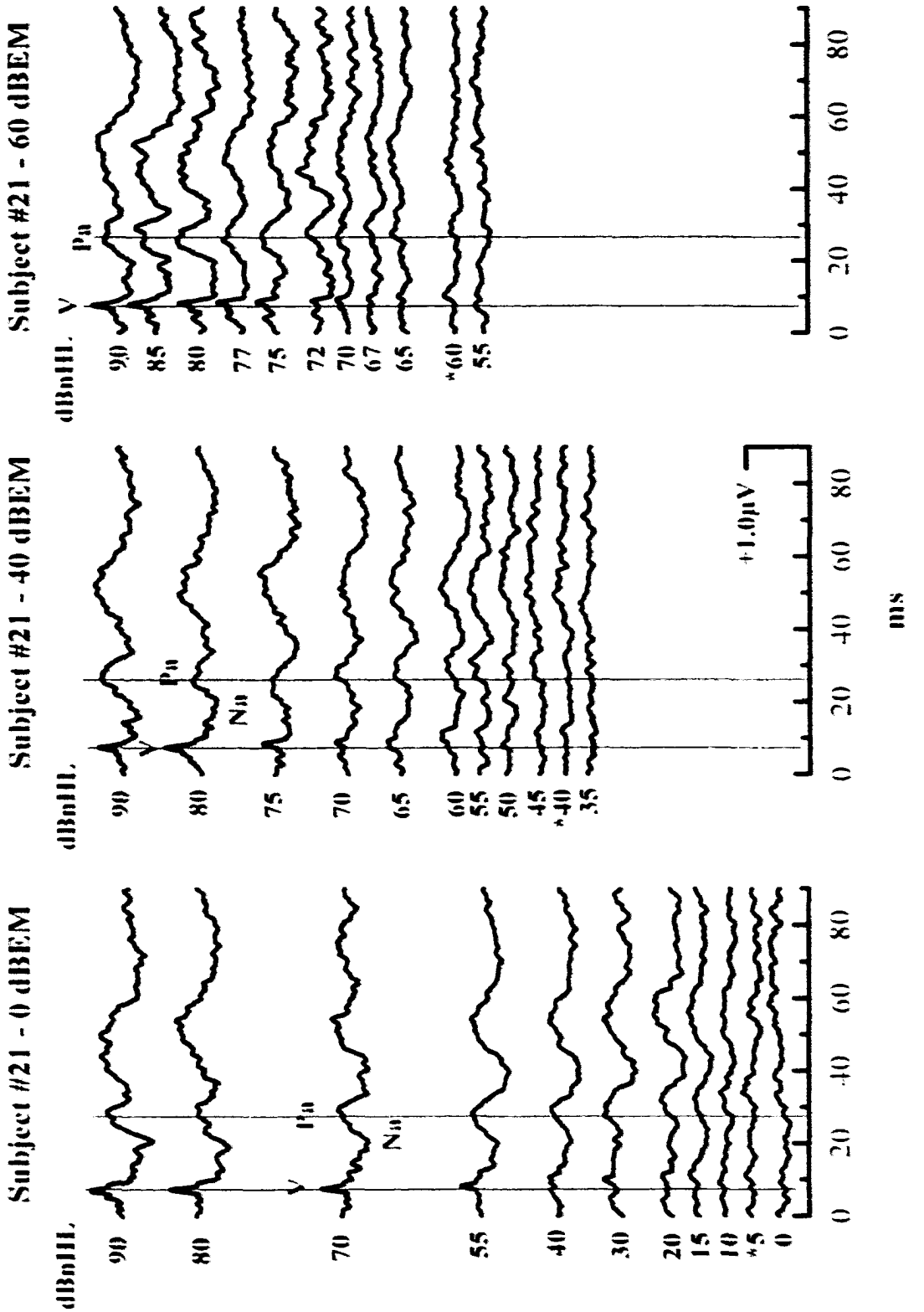


Figure 2

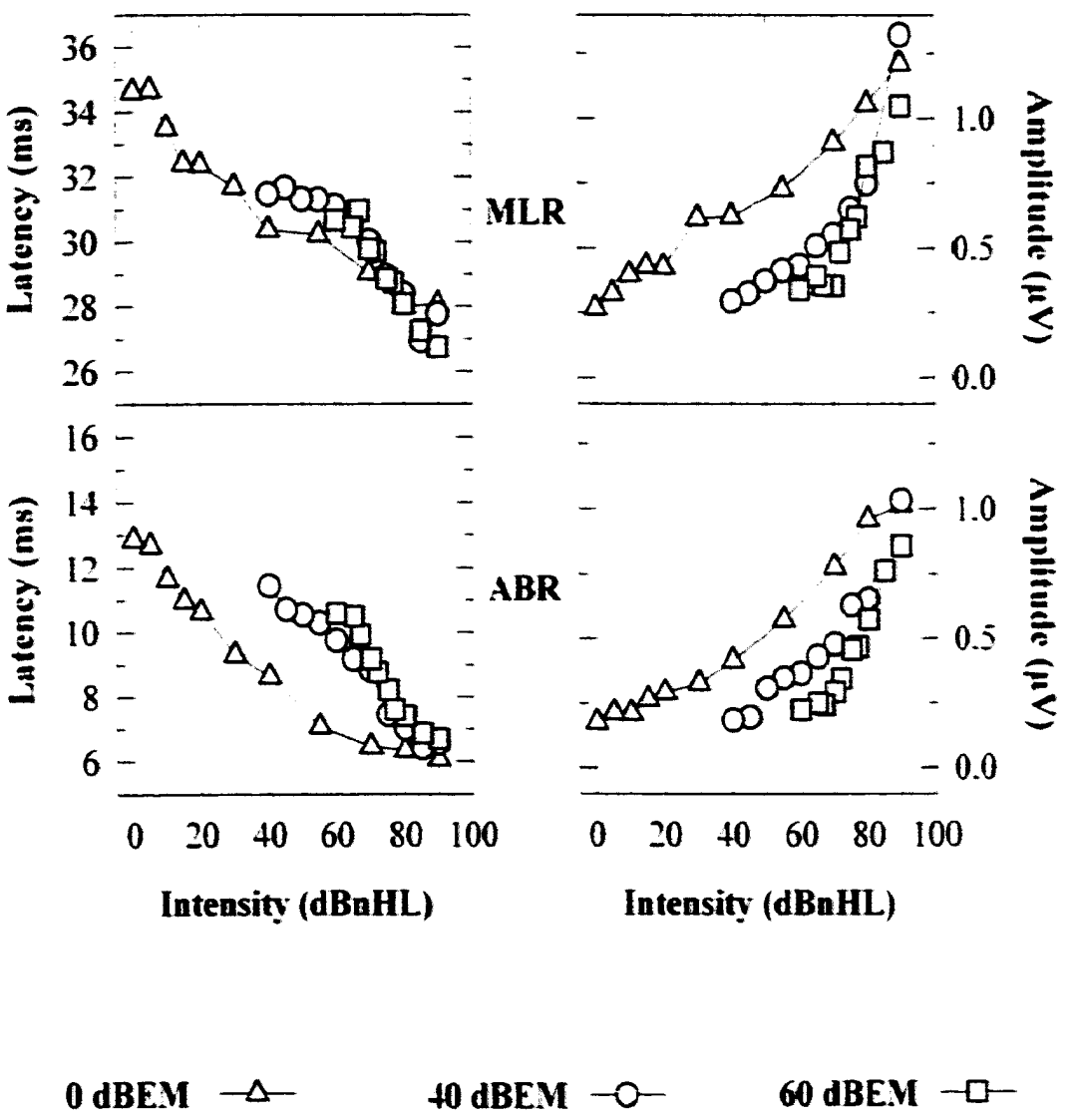


Figure 3

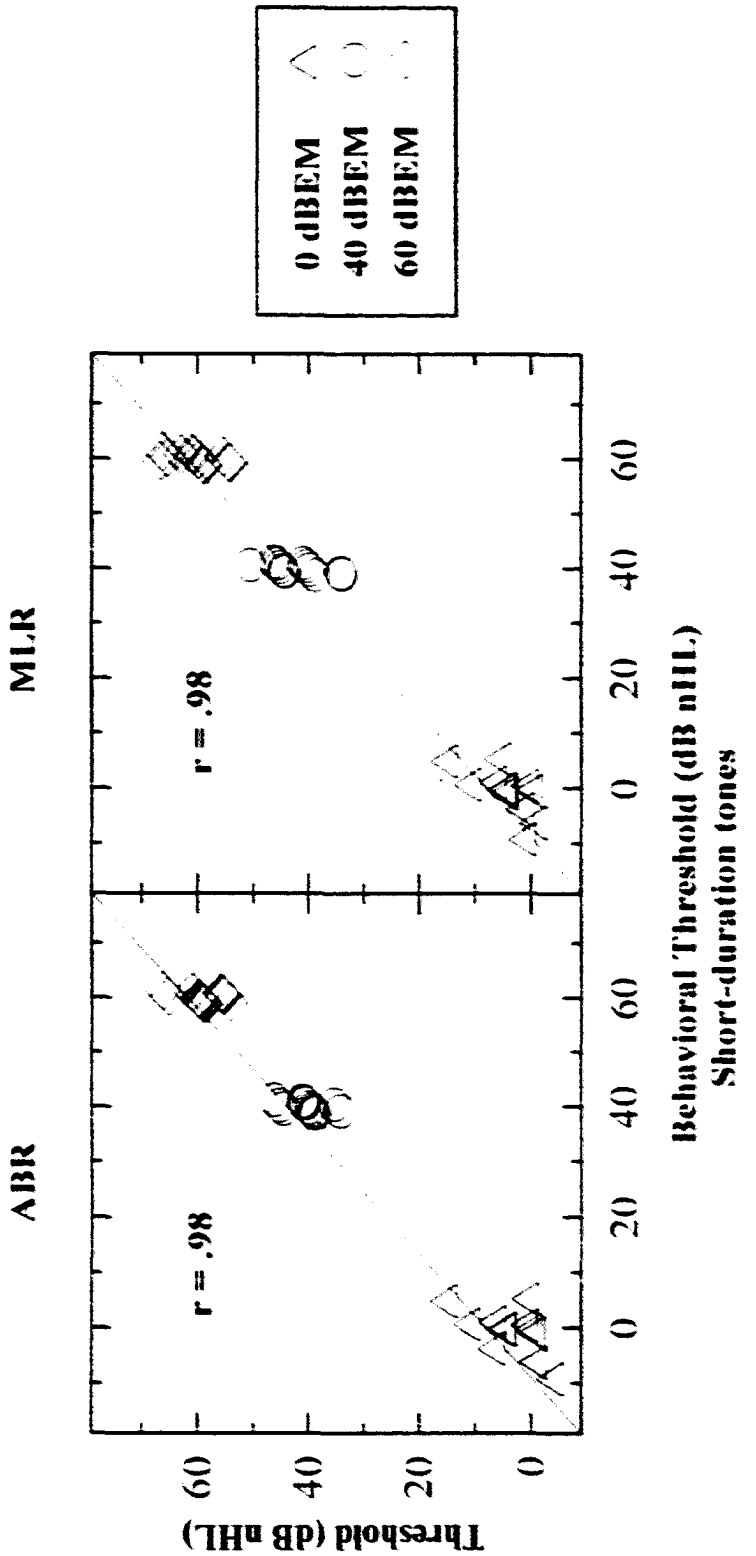


Figure 4

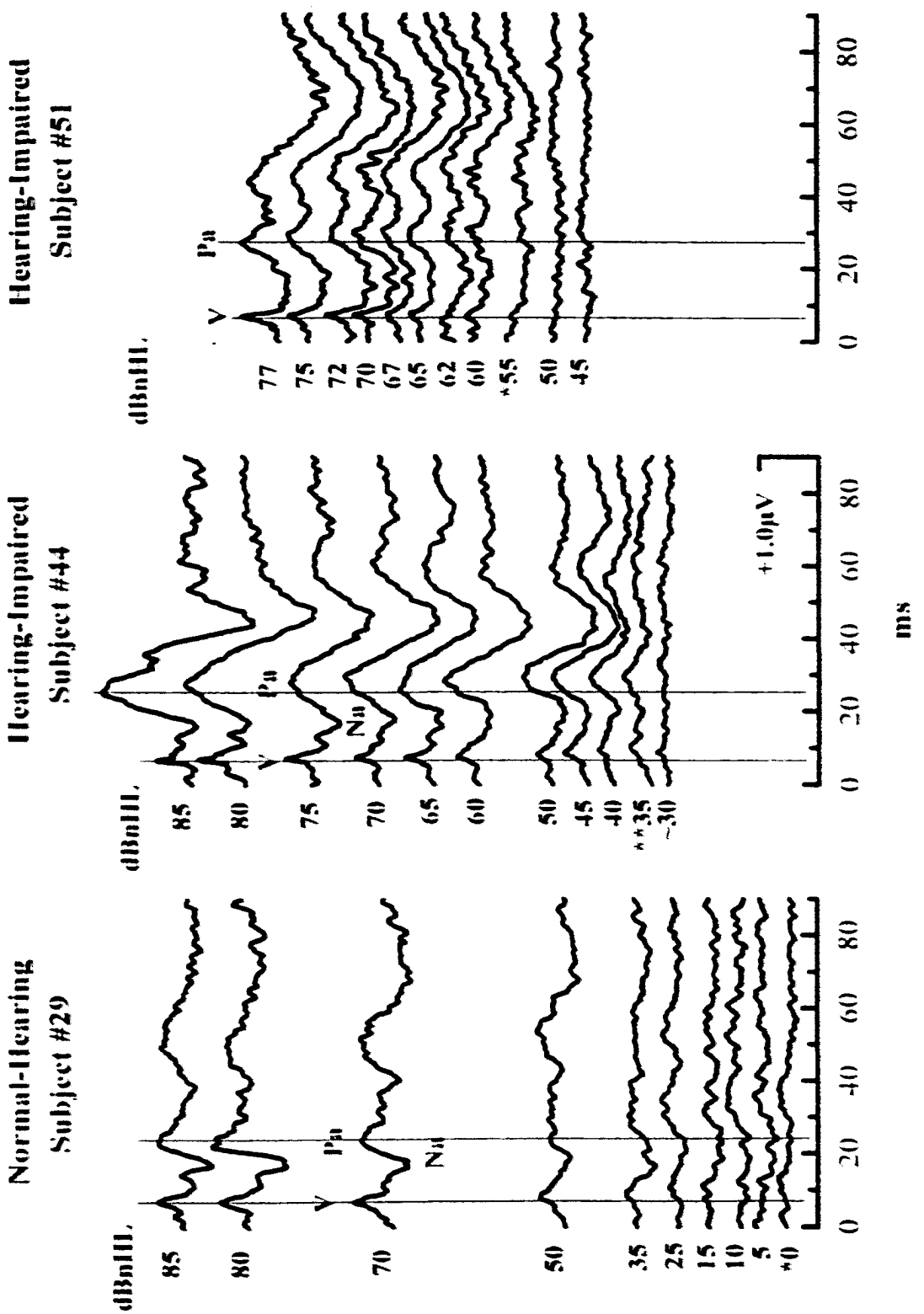


Figure 5

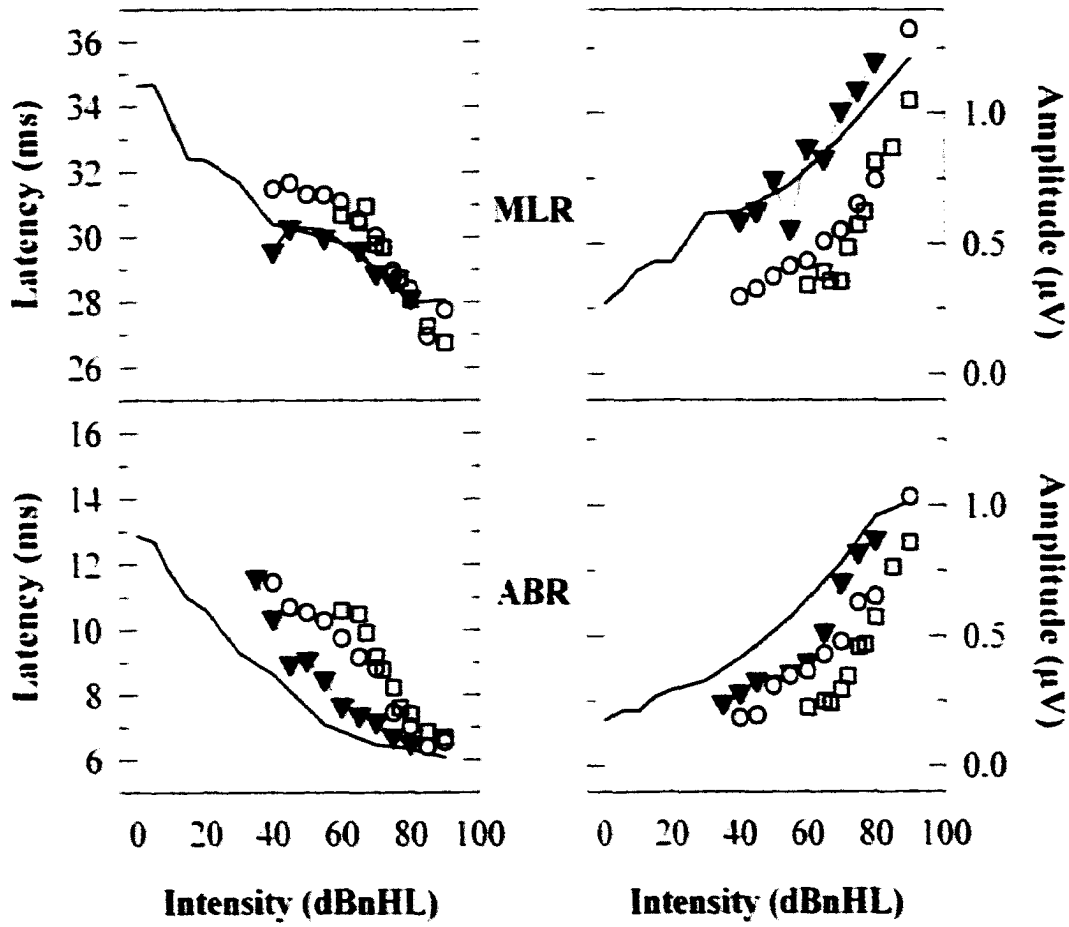
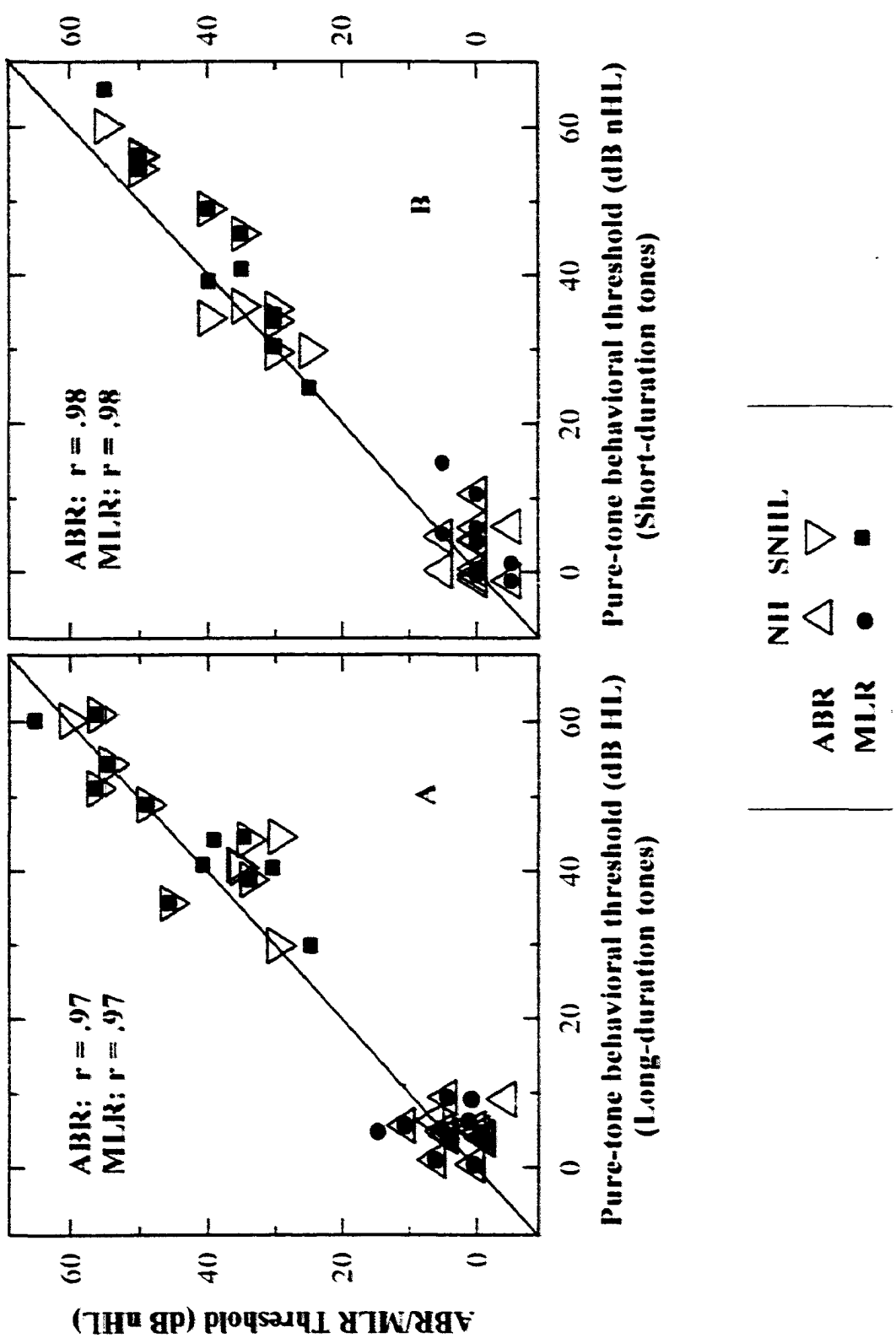


Figure 6



Chapter 6

Auditory brainstem and middle latency response measures of loudness growth: Normal-hearing, noise-masked normal-hearing and sensorineurally-impaired adults

Introduction

Among the different dimensions of auditory sensation which may be compared with auditory evoked potentials (AEPs), loudness is one of the most important. Loudness increases with stimulus level, and the amplitudes and latencies of the AEPs are also level dependent. Determining the nature of the relationship between behavioral and AEP input-output functions can serve both theoretical and practical purposes. Knowing how the electrical activity recorded at the scalp as a function of intensity relates to listeners' perception of stimulus magnitude may contribute to our understanding of the neurophysiological mechanisms which implement sensory and perceptual processes in both the mature and developing auditory systems. The major practical purpose is to provide a useful index for diagnosis and treatment of hearing loss.

Behavioral measures of loudness are obtained routinely from older children and adults with hearing loss, both to assess the nature of hearing loss (e.g., Killion & Fikret-Pasa, 1993), and to determine appropriate amplification parameters (e.g., Cox, Alexander, Taylor & Gray, 1997; Schum, 2000). Children younger than five years, however, are unable to participate reliably in behavioral loudness scaling procedures (Ellis & Wynne, 1999; Israelsson, Sandh & Leijon, 1995; Kawell, Kopun, & Stemalchowicz, 1988; Launer, 1998; Macpherson, Elfenbein, Schum & Bentler, 1991; Stuart, Durieux-Smith & Stenstrom, 1991; Zwislocki & Goodman, 1983), and, presently, there are no data that describe loudness growth in this young population (Mueller & Bentler, 1994). An electrophysiologic measure that is predictive of the loudness growth function would be a useful addition to the audiologist's repertoire for assessment of auditory system

capabilities in very young patients as well as older subjects who are unable to provide reliable judgements of loudness.

Loudness growth is measured using psychophysical scaling methods such as category scaling (e.g., Cox et al., 1997; Keidser, Seymour, Dillon, Grant & Byrne, 1999) and magnitude estimation (Hellman, 1999; Hellman & Meiselman, 1990, 1993; Israelsson et al., 1995). These procedures require subjects to make judgements of the apparent magnitude of sounds which are usually frequency-specific stimuli (i.e., pure tones or narrow-band noise). In magnitude estimation, the subject's task is to give a verbal report of how loud each sound is by assigning a number to each stimulus. These data, plotted as a function of intensity, are described by a power function, developed from the work of Stevens (1957) and later modified by Hellman and Zwislocki (1961, 1963, 1964, 1968). In log-log coordinates, the power function is a straight line with a slope equal to the value of the exponent. Using Absolute Magnitude Estimates (Hellman & Zwislocki, 1961), the loudness of a 1000-Hz tone is a power function of sound pressure with a slope (i.e., exponent) of about 0.54 for normal listeners (Hellman & Zwislocki, 1964), although considerable intersubject variability in the size of the exponent has been reported (e.g., Canévet, Hellman & Scharf, 1986; Hellman, 1981; Hellman & Mieselman, 1988; Stevens & Guirao, 1964; Viemeister & Bacon, 1988). The form of the loudness function, however, remains invariant and to a first approximation, provides a good description of the individual as well as the group results (Hellman & Mieselman, 1988; Stevens & Guirao, 1967). For subjects with decreased hearing sensitivity, the loudness function is steeper compared to those of normal listeners, indicating a faster rate of loudness growth.

This is true for both normal-hearing subjects whose threshold is shifted by masking noise (e.g., Lochner & Berger, 1961) as well as for subjects with sensorineural hearing loss (e.g., Hellman & Meiselman, 1993). In addition, the greater the threshold shift, the steeper is the loudness growth function both for noise-masked normal-hearing (e.g., Gleiss & Zwicker, 1964; Schlauch, DiGiovanni & Riesz, 1998) and cochlear-impaired subjects (e.g., Hellman, 1981; Hellman & Meiselman, 1993). The form of the behavioral loudness power function may be compared to AEP intensity-latency and intensity-amplitude functions, both of which may also be described by a power function (Keidel, 1976). Indeed, if a relation exists between these behavioral and electrophysiologic measures, then the increase in the rate of loudness growth seen in subjects with decreased hearing sensitivity (i.e., masked normal-hearing and impaired subjects) should be reflected also in AEP intensity-latency and/or intensity-amplitude functions.

The auditory brainstem response (ABR), and possibly the middle latency response (MLR), are the AEPs of choice in current clinical practice for estimating hearing sensitivity in subjects who are unable to participate in behavioral audiometric tasks (Hyde, Matsumoto & Alberti, 1987; Kileny & Shea, 1987; Kraus & McGee, 1990; Kraus, McGee & Stein, 1994; Scherg & Volk, 1983; Stapells, 2000a; Stapells, Gravel & Martin, 1995; Stapells, Picton, Durieux-Smith, Edwards & Moran, 1990). The ABR and MLR occur within 100 ms following onset of auditory stimulation, and represent the response of generators in the peripheral (VIIIth nerve = ABR wave I), and central auditory system (brainstem = ABR wave V; primary auditory cortex = MLR wave Pa) (Hashimoto, Mashiko, Yoshikawa, Muzuta, Imada & Hayashi, 1995; Legatt, Arezzo & Vaughan,

1988; Møller, 1994; Scherg & von Cramon, 1986). Both are noninvasive measures that may be obtained simultaneously (Chapter 5, present volume; Mackersie, Down & Stapells, 1994; Oates & Stapells, 1997a,b; Scherg & Volk, 1983; Suzuki, Hirai & Horiuchi; 1981; Tucker & Ruth, 1996; Wu & Stapells, 1994; submitted), a recording technique that has been recommended for routine clinical use (e.g., Hood, 1995; Kraus & McGeee, 1990). Examination of the ABR/MLR with intensity in comparison to loudness growth measures may show whether one (or both) of these evoked potential options is useful for estimating loudness growth.

The ABR and MLR have previously been compared to behavioral measures of sensation magnitude in normal-hearing adults using clicks as stimuli (Davidson, Wall & Goodman, 1990; Howe & Decker, 1984; Madell & Goldstein, 1972; Pratt & Sohmer, 1977; Serpanos, O'Malley & Gravel, 1997; Wilson & Stelmack, 1982). In the main, findings consistently show a slower growth in amplitude and decrease in latency with intensity compared to the behavioral loudness growth function. Measures to clicks presented alone (i.e., without noise masking), however, cannot provide an estimate of the auditory response from specific frequency regions (Eggermont, 1982b; Stapells et al., 1994), and the intensity-latency/amplitude results may reflect changes in cochlear place rather than loudness *per se* (Don, Ponton, Eggermont & Kwong, 1998; Eggermont, 1982a). Behavioral loudness measures are typically obtained using frequency-specific stimuli such as pure tones or 1/3- and 1/2-octave-wide bands of noise (e.g., Allen, Hall & Jeng, 1990; Cox et al, 1997; Ellis & Wynne, 1999; Hellman, 1999; Hellman & Meiselman, 1990, 1993; Keidser et al., 1999; Zwislocki, 1983). ABR/MLR intensity

series to tonal stimuli would be comparable, then, to the existing literature on loudness growth, and more in line with current clinical practices. To date, there has been no study comparing tone-evoked ABR and/or MLR intensity series with psychoacoustic measures of loudness growth in either normal-hearing or hearing-impaired persons.

In addition to the choice of stimuli, filter settings influence these AEP recordings. In the earlier studies on the ABR with intensity and psychoacoustic magnitude estimation, Pratt and Sohmer (1977: 250-3000 Hz), and Howe and Decker (1984: 300-3000 Hz), for example, used high-pass EEG filter settings which have been shown to substantially reduce the amplitudes of the ABR to clicks (Elberling, 1979; Sininger, 1995; Spivak, 1993) and tones (Sininger, 1995; Stapells & Picton, 1981; Suzuki & Horiuchi, 1977; Suzuki, Sakabe & Miyashita, 1982). In their study on the MLR, Madell and Goldstein (1972) used narrow EEG filters (25-150 Hz) which are known to result in waveform distortion, increased latencies, and decreased amplitudes of the MLR (Kileny 1983; McGee, Kraus & Manfredi, 1988; Suzuki, Hirabayashi & Kobayashi, 1984b; Suzuki, Kobayashi & Hirabayashi, 1983; Takagi, Suzuki & Kobayashi, 1985). In their spectral analyses of the ABR and MLR to tones, T. Suzuki and colleagues (Suzuki et al., 1982, Suzuki et al., 1983a) showed that these responses' main components are predominantly low-frequency energy (ABR: 50-100 Hz; MLR: 30-50 Hz). The ABR has a dual structure, consisting of a series of successive waves (I, III, V) superimposed on a slow positive deflection (slow-wave component). It is the slow-wave component that is most useful in the interpretation of the ABR, especially at low intensities (Davis & Hirsh, 1979; Stapells, 2000b; Suzuki et al., 1982). Examination of the ABR/MLR intensity-

latency/amplitude functions as they relate to loudness judgements require recordings that include the dominant low-frequency energy in these responses which consistently appears even with low-frequency stimuli of low intensity (Suzuki et al., 1982).

Previously, the intensity-latency/amplitude functions of these neural responses were compared to the function obtained using the Absolute Magnitude Estimation (AME) procedure. In their studies of subjects with normal hearing, Pratt and Sohmer (1977) and Wilson and Stelmack (1982) found that, generally, the ABR intensity-latency function yielded negatively accelerated power functions, while the amplitude exponents increased as stimulus intensity increased and thus "resembled the loudness power function more closely than exponents derived from the latency values" (Wilson & Stelmack, 1982). However, because the exponents of both the intensity-latency and intensity-amplitude power functions were smaller than those derived from behavioral magnitude estimates, these authors concluded that the ABR does not reflect the neural correlate of loudness. Stevens (1970) points out, however, that whereas there are differences in the exponents between behavioral and electrophysiologic measures within a modality, the consistency of the *relative* exponent size should be the same for the evoked potentials as for the psychophysical measures if the two measures have something in common. Indeed, several earlier studies demonstrated that the *relative* size of psychophysical power functions for different modalities usually approximates the relative size of the evoked potential power function exponents (e.g., Keidel & Spreng, 1965; Walsh, 1979). Keidel and Spreng (1965), for example, obtained neural (cortical N1 evoked potential) responses and magnitude estimates for sound, vibration, and electric shock. Although the neural

exponents were smaller than those obtained from the psychophysical measures, the relative size across modalities was consistent for both types of measurements. Walsh (1979) compared ABR and Visual Evoked Responses (VER) to loudness and brightness balance measures and found that the ratio of the ABR to VER exponents is similar to that obtained using cross-modality matching.

It is possible to evaluate both sensation magnitude and evoked potentials in the same subject for several conditions within the same modality to examine whether or not the AEPs reflect a similar pattern of change as that observed from psychophysical measures. This may be accomplished in the auditory modality by evaluating a group of normal-hearing subjects in several different conditions: tones presented alone and in the presence of noise maskers. As indicated above, loudness grows at a faster rate for tones presented in noise (e.g., Hellman, 1970; Hellman & Zwislocki, 1964; Stevens, 1966), and the slope of the loudness function for noise-masked normal-hearing subjects steepens further with increasing masker level (Gleiss & Zwicker, 1964; Hellman, 1970; Hellman & Zwislocki, 1964; Lochner & Burger, 1961). By using different levels of masking, several functions may be obtained from a group of normal-hearing subjects for both loudness and ABR/MLR measures. If the *relative change* in exponent size is similar between conditions for the evoked potentials as that seen in the psychoacoustic observations, then it would suggest that both measures reflect similar neural processes.

Studies of the click-evoked ABR and MLR in noise masking have described, in general, the changes in the characteristics of these responses with masking (e.g., Chapter 5, present volume). That is, masked ABR and MLR absolute latencies increase and their

absolute amplitudes decrease compared to the non-masked response. However, no studies have reported on whether or not there is a change in the *rate* of ABR and MLR latency decrease or amplitude increase with intensity in masked normal listeners compared to their rate of change for tones (or clicks) presented alone. Also, no study to date has compared the effects of increasing masker noise level on both ABR/MLR with intensity and behavioral loudness growth measures obtained from a group of normal-hearing subjects.

Another means of establishing whether or not the neural responses relate to loudness is to examine these measures in a group of subjects with sensorineural hearing impairment. As noted earlier, the slope of the loudness function is steeper in both noise-masked normal (e.g., Stevens & Guirao, 1967) and cochlear-impaired listeners (e.g., Hellman & Meiselman, 1993) compared to the normal response in the quiet, and the steepness of the slope is dependent upon the amount of threshold shift (Zwicker & Fastl, 1999). By obtaining the ABR/MLR and behavioral magnitude estimates from a group of impaired listeners whose hearing thresholds are within the range of the masked normal responses, we may extend the findings obtained from the noise-masked normal listeners in order to determine the electrophysiologic indices of loudness that may be useful for both basic and applied purposes. In the only previous study that investigated the relation between the ABR and loudness magnitude estimates in hearing-impaired subjects, Serpanos et al. (1997) obtained measures for clicks from a group of normal listeners and two groups of sensorineurally-impaired subjects, one group with mild-to-moderate flat configuration of hearing loss and a second group that presented with sloping high-frequency hearing loss.

These authors found no significant difference in the slope (i.e., exponent) values of the click-evoked ABR intensity-latency functions among the three groups of subjects. A significant correlation between wave V latencies and loudness measures was found for the normal listeners and the impaired subjects with flat configuration of hearing loss, but not for the group with sloping high-frequency hearing loss. These findings suggest that the changes in click-evoked wave V latency with increasing intensity may be more related to the configuration of hearing (loss) and not an index of the loudness percept, as previously suggested by Eggermont (1982b) and shown by others (e.g., Suter & Brewer, 1983; Yamada et al., 1979). Serpanos et al. (1997) did not examine the relation between loudness and ABR amplitudes, measures of AEP magnitude that many researchers consider an index of magnitude perception (e.g., Davidson, Wall & Goodman, 1990; Davis & Zerlin, 1965; Eggermont, 1977,1982b; Keidel, 1976; Kiessling, 1982; Madell & Goldstein, 1972; Picton, Woods & Proulx, 1978; Uziel & Seneclause, 1978). There do not appear to be any studies that have compared the changes in ABR amplitudes with increasing intensity and loudness growth measures in a group of hearing-impaired subjects, nor are there any studies that have compared the changes in the MLR with increasing intensity and measures of magnitude perception in hearing-impaired listeners.

The purposes of the present study were (1) to investigate the relationship between tone-evoked ABRs and MLRs and subjective loudness estimates by examining the ABR/MLR intensity-amplitude, intensity-latency and behavioral loudness growth functions for normal-hearing listeners in several masking conditions, and (2) to examine the relation between ABRs and MLRs and loudness growth in a group of listeners with

sensorineural hearing loss. Both the electrophysiologic and behavioral responses to1000-Hz tones were obtained from the two groups of adult subjects. For the normal listeners, tones were presented also in two levels of simultaneously presented broadband masking noise. The Absolute Magnitude Estimation (AME) procedure was employed to obtain behavioral loudness measures. Results of the intensity-amplitude and intensity-latency functions were compared to the loudness growth functions. It was hypothesized that, with increased threshold shift, the slopes of the ABR/MLR intensity-amplitude and/or intensity-latency functions would steepen in a manner proportional to that seen in the behavioral loudness functions.

Methods and Procedures

Subjects

Twelve normal-hearing subjects (six female), and twelve adults with sensorineural hearing loss (seven female) participated in this study. The age range of the normal-hearing subjects was 23 to 53 years (mean = 36.8 yrs), and that of the hearing-impaired subjects was 18 to 63 years (mean = 47.5 yrs). The normal-hearing subjects all had pure-tone behavioral thresholds equal to or better than 20 dBHL (ANSI, 1989) at octave frequencies 250 through 8000 Hz for at least one ear (i.e., the test ear), and no significant otologic history. The hearing-impaired subjects had mild-to-moderate sensorineural hearing loss (SNHL), with thresholds at 1000 Hz in the range 30 - 60 dBHL (test ear). Their configuration of hearing loss was either flat (six) or gradually sloping, with no greater than a 10-dB change in threshold between successive half octaves within the frequency range 250 through 8000 Hz. Their mean thresholds at 500, 1000, 2000, and

4000 Hz were 35.8 (s.d. = 10.6), 45.4 (9.6), 52.5 (10.6), and 55 (12.8) dBHL, respectively. All subjects had normal middle-ear function as measured by immittance testing, and no significant neurologic histories. None of the subjects had prior experience with magnitude estimation or knowledge of the decibel scale.

Stimuli and Equipment

The stimuli were short-duration 1000-Hz tones presented alone or simultaneously with broadband noise. Tones were 5 ms in total duration (5 cycles, no plateau, exact-Blackman windowed), alternated in onset polarity, and presented at a rate of 10.9/s. These stimuli were generated and amplified (Intelligent Hearing Systems, SmartEP system), attenuated (Med Associates, ANL918), and directed to an insert earphone (EAR-3A) for monaural stimulation. Latencies were corrected by -0.93 ms for the acoustical delay imposed by this earphone. The tones were presented either in Quiet (normal- and hearing-impaired subjects) or in simultaneously presented masking noise (normal-hearing subjects). Broadband masking noise was generated by a white noise generator (Med Associates, ANL912), filtered (Wavetek Model 852: 20-8000Hz, 48 dB/octave), attenuated, and mixed (Med Associates, ANL918) with the tones.

Stimulus intensities were calibrated relative to the behavioral thresholds of 10 normal-hearing adults (mean age = 36.1 years), one of whom also participated in the experimental portions of this study. Thresholds were obtained in 2-dB steps using an ascending approach. The intensity of the average threshold (0 dB nHL) was determined acoustically using a 2 cm^3 acoustic coupler (Bruel & Kjaer, model DB-1038) and sound level meter (Bruel & Kjaer Type 2209). The peak-to-peak equivalent sound pressure

level (ppeSPL) for 0 dB nHL was 25 dB. Maximum output for the 1000-Hz tones was 100 dB nHL. The linearity of the attenuators was checked during the initial calibration.

The electroencephalogram (EEG) was amplified (50,000X) and filtered (10-1500 Hz, 12 dB/octave) by the SmartEP system. Single-channel recordings of the ABR and MLR were obtained with gold-plated cup electrodes placed at the vertex and earlobe ipsilateral to the stimulus. The SmartEP system employs a single-ended preamplifier with optical isolation, therefore a ground electrode was not required. The earlobe location was chosen in order to minimize postauricular muscle reflex which could distort the MLR and the later components of the ABR, especially at high stimulus intensities (Stapells & Picton, 1981). Interelectrode impedances were kept below 2000 Ohms. Signal averaging was carried out using an 80-ms analysis window with no prestimulus baseline. Trials containing amplitudes exceeding ±50 μV were automatically rejected. At high intensities (≥ 70 dB nHL), fewer trials were needed to evoke clear responses. Therefore, four replications of 1000 trials each were obtained for intensities ≥ 70 dB nHL, and four replications of 2000 trials each were obtained for intensities <70 dB nHL. Obtaining four replications at each intensity provided an equal number of waveforms per intensity for subsequent response rating.

Procedures

Acoustic immittance testing was performed at the beginning of each test session to ensure normal middle-ear status. Following audiometric evaluation, one ear of each normal listener was selected randomly as the subject's test ear for all test conditions. For the SNHL subjects, the ear with the hearing loss of interest was evaluated. The nontest

ear of the normal-hearing subjects was occluded by an EAR-3A earphone during the experimental procedures. Subjects were seated in a recliner in a double-walled sound-attenuated room. They were tested individually and were unable to observe the examiner during the experimental procedures. The behavioral magnitude scaling and ABR/MLR data were collected from the normal-hearing subjects in 2-to-3 sessions, for a total test time of 9-12 hours, and from the hearing-impaired subjects in one session that lasted about 3 - 4 hours.

Threshold of audibility and loudness discomfort levels were obtained individually to determine the intensities for each subject/condition. A modified Hughson-Westlake procedure (Carhart & Jerger, 1959) was employed to obtain threshold for the 1000-Hz tones using 2-dB steps. Mean behavioral thresholds were -0.8 (s.d. = 4.2) and 39.2 (s.d. = 9.1) dB nHL for the normal-hearing and hearing-impaired subjects, respectively. For the masking conditions, normal-hearing subjects only, broadband noise was simultaneously introduced to mask the tones presented at 40 or 60 dB nHL. Masking levels were determined for each individual. Noise increments of 10 dB were used initially until a simultaneous tone presentation yielded "No Response", and then a 2-dB step descending approach was employed to obtain the Effective Masker level (dBEM), which was defined as the masker intensity that just masks the stimulus 50% of the time. Average (across 12 subjects) noise levels were 67.3 dBSPL and 82.6 dBSPL for 40 and 60 dB nHL 1000-Hz tones respectively. The corresponding spectrum levels are 28.4 and 43.7 dB/Hz, respectively.

Maximum test levels for each subject/condition were determined by measuring each subject's Loudness Discomfort Levels (LDL) using an established LDL procedure (Hawkins, Walden, Montgomery & Prosek, 1987, Appendix A). Subjects rated the loudness/comfort of an approximate 1-second presentation (stimulus rate = 10/s) of the 1000-Hz short-duration masked and non-masked tones. A descending method of limits with a 2-dB step size was used, and the initial stimulus intensity level was 75 dB nHL. When a level of discomfort, i.e., "Uncomfortably Loud", was reached, the intensity of the stimulus was decreased by a randomized amount, ranging from 5-15 dB (Bentler and Pavlovic, 1989), and the stimulus was again presented in an ascending (2-dB step) manner. The LDL was defined as the lowest intensity minus 1 dB that evoked a judgement of uncomfortably loud on two of three ascending trials (Hawkins et al., 1987). For the 12 normal-hearing subjects, the mean LDLs were 96.0, 95.5 and 95.6 dB nHL for the 0 (non-masked), 40, and 60 dBEM conditions, respectively. The maximum intensities subsequently used for these 12 subjects were 90 (n=8), 85 (n=3), and 80 (n=1) dB nHL. The mean LDL for the hearing-impaired group was 91.0 dB nHL, and the maximum intensity levels used for these 12 subjects were 90 (n=3), 85 (n=2), 80 (n=6), and 77 (n=1) dB nHL. Generally, the maximum intensity level used for all subjects was 3-to-10 dB below their LDL.

The levels for stimulus presentation were determined individually for each condition, based on threshold measures and LDLs, and included both equal sensation levels (re: behavioral threshold for the short-duration tones) and equal hearing levels (dB nHL). Within each condition, the same 8-to-10 intensities were used for a subject to obtain both

their psychoacoustic and electrophysiologic measures. Stimulus levels were presented in random order, different for each subject and condition. For Absolute Magnitude Estimation (AME), subjects were given a set of written instructions (see Appendix B) asking them to match an appropriate number, any number, to the loudness of the tones regardless of the number assigned to the previous stimulus (Hellman & Meiselman, 1988). For this procedure, three different randomized sequences of tones were presented within each condition (e.g., Zwislocki, 1983), however the first stimulus of a sequence always occurred within the range between 40 and 70 dB nHL for the normal-hearing group, non-masked condition, and between 60 and 75 dBHL for the hearing-impaired group and for the normal listeners in the masking conditions. This restriction minimized biases resulting from starting stimulus sequences with extreme values (Zwislocki & Goodman, 1980). Subjects were given as many repetitions of the 1-second presentation of tones as they wished before they assigned a number indicating their judgement of sound magnitude. They were encouraged, however, to be spontaneous about their decisions, and to make them as quickly as possible. Any positive number, regardless of its magnitude, was acceptable, including repeated use of the same number. The geometric mean of the last two numerical judgements made at each intensity was used for each subject for data analyses.

For the ABR/MLR recordings, subjects were asked to sit quietly and relax, either reading a book or resting comfortably. They were not allowed to sleep, and were monitored visually, by EEG, and via intercom to ensure that they did not fall asleep. ABR/MLR stimulus levels were administered randomly within a condition, using a

different order for each condition. However, all subjects were tested initially at 70 dB nHL (non-masked, 2 replications) to ensure that an individual's evoked potential recordings did not include excessive noise and were replicable. The other two replications at this intensity were obtained in random order. The order of test condition for the normal-hearing subjects (non-masked, 40 dBEM, and 60 dBEM masking conditions) and test type for all subjects (AME and ABR/MLR) was randomized.

Data analysis

The decision regarding the presence or absence of the electrophysiologic responses (ABR/MLR) was made by combining the ratings of three raters who were very familiar with these responses and their measurement. The ABR/MLR waveforms were evaluated by these raters in random order (within and across conditions), without knowledge of the stimulus intensity, masking condition, and hearing status. Each of the raters independently scored the ABR and MLR peaks on a four point scale, based on visual observation of the appropriate time window surrounding wave V, Na, and Pa peaks. A rating of "1" = no response, "2" = possible response, "3" = probable response, and "4" = definite response. The three raters' scores were averaged and rounded to the nearest integer to obtain a combined score. Peaks with a combined score of ≥ 2.5 were considered responses, and measures of latency and amplitude were obtained from the average of the four replications of those recordings (Oates & Stapells, 1997a,b; Stapells et al., 1990; Wu & Stapells, submitted). The peaks measured were the latency of waves V and Pa, and the peak-to-peak amplitudes of V-Va and Na-Pa. Wave V was defined as the maximum vertex-positive peak occurring between 6 and 20 ms following stimulus onset.

Wave V' was defined as the maximum negativity following wave V and within 8 ms of wave V (Nousak & Stapells, 1992; Oates & Stapells, 1997a,b; Stapells & Picton, 1981). Peak Na was the largest negativity preceding Pa in the 12-20 ms window following stimulus onset. Wave Pa was defined as the maximum vertex-positive peak occurring 25-50 ms following stimulus onset (Wu & Stapells, 1994).

Loudness growth functions obtained from magnitude estimates (AME), and intensity-amplitude/latency functions obtained from ABR/MLR measures were analyzed for the four conditions (0, 40, 60 dBEM and SNHL). ABR/MLR individual and group slope values (i.e., exponents of the functions) were obtained using the amplitudes and latencies measured from the same intensities as those used to obtain the loudness exponents. Because the characteristic change in latency with intensity yields a negative sloping function (i.e., as intensity increases, latencies decrease), absolute latencies were transformed by calculating their reciprocal in order to compare them (visually and statistically) with the behavioral findings. The individual subjects' MLR absolute latencies were corrected first by subtracting the normal listeners' average MLR-ABR latency difference at 80 dB nHL in the 0 dBEM condition (i.e., 20.68 ms). This shifts the MLR latencies to the same starting point as the ABR wave V, but preserves the slope so that the ABR and MLR intensity-latency functions could be compared. Regression lines for the V-V' and Na-Pa amplitudes, the V and Pa latency reciprocals, and the behavioral magnitude estimates were obtained according to a power relationship of the response to stimulus intensity. Exponents were calculated for each subject. The data were subjected to analysis of variance and the results of the ANOVA were considered significant if

$p < .01$. The more-strict 99% confidence interval was chosen due to the number of ANOVAs carried-out and to ensure any "significant" effects are reliable. Huynh-Feldt epsilon (ϵ) correction factors were applied to the degrees of freedom where appropriate for repeated measures. Probabilities presented reflect these corrections. Amplitudes and latencies were analyzed separately. Where appropriate, Newman-Keuls *post-hoc* comparisons were carried out only on significant main effects and interactions (i.e., $p<.01$), and these *post hoc* results were considered significant if $p < .05$.

Results

Normal-hearing subjects

Figures 1 and 2 show two representative subjects' ABR/MLR waveform intensity series as well as behavioral (loudness) magnitude estimates for the three conditions. For each condition, each subject's estimate of loudness magnitude (AME) is listed to the left of the intensities (in dB nHL), with the corresponding ABRs/MLRs shown to the right. The measured peaks of the waveforms for the ABR (Wave V) and MLR (Na & Pa) are indicated for a high stimulus level within each waveform series for clarity. Within each condition it can be seen that, with increasing intensity, magnitude estimates increase and the ABR/MLR amplitudes increase and latencies decrease.

The intensity-amplitude/latency and loudness (AME) functions for the two subjects in Figures 1 and 2 are graphically presented in Figures 3 and 4. Each subject's ABR/MLR amplitudes (upper panels), latency-reciprocal values (lower panels), and the behavioral AME values (right panel) are plotted on log-log coordinates, and the respective exponents representing the slope of the functions are included within each panel. The amplitude

functions for both of these subjects steepen with increasing noise level (0 vs 40 vs 60 dBEM) for both the ABR (left panels) and MLR (right panels), and so also do their loudness growth functions (AME) shown at right. Also, the pattern of change in slope with increasing noise masker level for these three measures is similar for both subjects. Indeed, all 12 subjects' masked ABR/MLR amplitude and loudness growth functions are steeper than their respective non-masked results.

In the lower panels of Figures 3 and 4, the latency-reciprocal functions for these two subjects show somewhat different results. Latencies show some prolongation with masking, but no clear pattern of change in slope with increasing masking (i.e., 40 vs 60 dBEM) emerges from these single-subject latency data for either evoked potential. The results for the subject in Figure 3 show an increase in the slopes of both ABR and MLR intensity-latency functions with increasing masker, but the latency results for the subject shown in Figure 4 are quite different. This subject's masked ABR intensity-latency function increases slightly compared to the non-masked response, but no further increase in slope is apparent for the higher noise condition. Conversely, her masked MLR intensity-latency function is *less steep* than the non-masked response. This finding of no change or a decrease in the slope of the masked MLR intensity-latency function was obtained in about half of the subjects, whereas all subjects' ABR latency functions increase with masking.

The mean data for 11 of the 12 subjects' electrophysiologic and behavioral measures are graphically presented in Figure 5. One subject's[1] AME exponent (0.90) in the non-masked condition is substantially larger than the other subjects' results, therefore

descriptive statistics and subsequent analyses include only the remaining 11 subjects, except where noted. With increasing intensity, the average results of the changes in ABR and MLR amplitude and loudness (AME) are fairly well-fitted by a straight line when plotted on log-log coordinates, and the masked functions for all measures are steeper. Amplitude functions (right panels) become steeper with increasing masking and, within each condition, the rate of amplitude growth is about the same for the ABR and MLR. The left panels of Figure 5 show that ABR and MLR latencies decrease at about the same rate in the 0 dBEM condition. The slopes of the ABR and MLR intensity-latency functions appear to be slightly steeper in the 40 dBEM condition, and are clearly steeper than the non-masked response for the higher-level masker. ABR and MLR intensity-latency functions are less steep in all conditions compared to their corresponding intensity-amplitude functions, indicating a much slower rate of change in the timing of waves V and Pa with intensity compared to the rate of increase in ABR and MLR response magnitude as indicated by the scale differences of the graphs. In the lower panel are shown the mean loudness functions obtained from behavioral magnitude estimates. AME functions show a faster rate of growth in all conditions compared to the results of the neural responses (again, note scale differences). As expected, the slope of the loudness growth function increases with increasing masker level. This pattern of change in loudness growth with masking is similar to that seen in the ABR/MLR amplitude results.

Regression analyses were performed on the ABR, MLR, and AME results in each condition, yielding exponent (i.e., slope) values for each subject's functions. The average

exponents for each noise condition obtained from these normal-hearing subjects are shown in Table 1. The mean exponents for all (ABR, MLR, and AME) measures increase with increasing masker level. Within each condition, the mean ABR and MLR amplitude exponents are about the same. ABR and MLR latency exponents are similar also within each condition, but the variability of MLR latency exponents is greater than the results for the ABR. AME exponents are larger than all of the neural exponents. Two 2-way ANOVAs [measure (3) × noise condition (3)] were carried out, one on the amplitude and AME exponents, and the other on the latency and AME exponents, in order to assess the effects of noise condition on these measures, and to compare each of the electrophysiologic measures with the behavioral results. The ANOVAs confirm the expected significant effects of measure, such that AME exponents are larger than those of the neural responses [main effect measure: Amplitude & AME: $F(1,10) = 15.61$, $\epsilon = .52$, $p = .002$; Latency & AME: $F(1,10) = 29.47$, $\epsilon = .51$, $p < .001$]. The noise effects are significant also, such that increasing masker level causes an increase in the rate of (1) ABR/MLR amplitude increase and loudness growth [main effect noise condition: $F(1,11) = 18.39$, $\epsilon = .55$, $p = .001$] and (2) ABR/MLR latency decrease and loudness growth [main effect noise condition: $F(1,11) = 9.45$, $\epsilon = .56$, $p = .009$]. The increase in the rate of loudness growth for masked tones, however, is greater compared to the results for the ABR/MLR responses [measure × noise condition interaction: Amp/AME: $F(4,40) = 3.91$, $p = .009$; Lat/AME: $F(4,40) = 6.37$, $p < .001$], which is due, in part, to the larger exponent values obtained for the behavioral measures compared to the ABR

and MLR results ($p < .001$). *Post hoc* analyses show also that AME is more affected by increasing masker level than are the electrophysiologic responses ($p < .001$).

Due to the large size of the AME exponents compared to those of the neural responses, ANOVAs were recalculated on only the ABR/MLR amplitude and latency exponents. With AME results excluded from the ANOVAs, the effects of increasing noise level on the evoked potential exponents are significant for ABR/MLR amplitude [noise condition main effect: $F(1,17) = 59.15$, $\epsilon = .88$, $p < .001$] and latency [$F(1,12) = 14.08$, $\epsilon = .64$, $p = .002$]. *Post hoc* analysis reveals that these noise effects are significant for all amplitude exponent comparisons ($p < .002$). However, the slopes of the ABR/MLR intensity-latency functions in the 40 dBEM condition are not significantly different from the non-masked (0 dBEM) response ($p = .113$). There are no significant differences between the ABR and MLR in their rate of amplitude growth [main effect wave: $F(1,10) = 2.81$, $p = .125$] or in their rate of latency decrease [wave main effect: $F(1,10) = 0.07$, $p = .797$], and no significant wave × noise condition interactions [Amplitude: $F(2,20) = .37$, $p = .694$; Latency: $F(2,20) = .16$, $p = .856$]. AME exponents were also analyzed separately, and the effect of noise condition on these behavioral measures did not quite reach significance [noise condition effect: $F(1,11) = 7.41$, $\epsilon = .56$, $p = .017$], probably due to the variability in the masked responses.

The finding that AEP and behavioral measures yield different size exponents is not unexpected (e.g., Madell & Goldstein, 1972; Pratt & Sohmer, 1977). In order to minimize these differences, we normalized the exponents for each measure by calculating the ratio of ABR, MLR, and AME exponents with noise masking to their respective

results in the non-masked condition. That is, each subject's exponents in the 40 and 60 dBEM conditions were divided by their exponent in the 0 dBEM (Quiet) condition. This gives values indicating the amount of increase in the rate of amplitude and loudness growth and latency decrease for the 40 and 60 dBEM conditions, which may then be used to compare the electrophysiologic and behavioral measures. The scatter plots in Figure 6 show the individual ratios of each subject's 40 and 60 dBEM exponents relative to their respective results in the quiet (0 dBEM) condition. Figure 6a shows the amplitude and AME results. The mean values are shown by the solid line within each column of individual values. For each measure, the mean 60 dBEM ratio is larger compared to the 40 dBEM ratio, reflecting the steeper slopes for the higher masker level seen in the raw data. The change in slope is > 1 for both the 40 and 60 dBEM conditions, indicating the steeper slope with masking for each measure relative to the results in the 0 dBEM condition. For example, the mean ABR amplitude ratio for the 40 dBEM condition is 1.51, indicating a 51% increase in the rate of amplitude growth compared to the non-masked response. The mean ABR amplitude ratio of 2.73 for the higher noise level condition indicates that the rate of amplitude growth for tones presented in 60 dB effective masking noise increases 173% compared to the rate of amplitude growth for this response when the tones are presented alone (0 dBEM). These effects of noise condition are seen also in the MLR amplitude and loudness results.

One subject's AME ratio value in the 60 dBEM condition lies well outside the group results, greatly increasing the variability of this measure. Subsequent analyses of these values thus include only the remaining 10 subjects. A 2-way ANOVA [measure (3) ×

noise condition (2)] was performed using these normalized values to assess the effects of the amount of increase for 40 and 60 dBEM noise conditions on these evoked potential and behavioral measures. The results reveal significant changes with increasing masker level for ABR/MLR amplitude and loudness growth [main effect noise condition: $F(1,9) = 81.87$, $p < .001$]. There is a trend toward a difference between measures [measure main effect: $F(1,16) = 4.99$, $\epsilon = .91$, $p = .023$], as can be seen in Figure 6a, such that overall the AME normalized exponents are larger than the ABR and MLR exponents, and the overall amount of increase in the MLR amplitude exponents is greater, also, than those for the ABR. However, the interaction term (measure × noise condition) is not significant [$F(2,18) = 0.12$, $p = .890$], indicating that, as masker level is increased, the amount of increase in the slopes of both the ABR/MLR amplitude and loudness exponents is not significantly different.

In Figure 6b, the results for ABR/MLR latency vs AME are shown. Each of these measures shows an increase in slope for both masker levels. On average, the latency results for the ABR 40 dBEM condition shows a modest increase of 40%, and an increase of 109% in the 60 dBEM condition, compared to 196% and 338% for the AME. ABR latency results are quite uniform across subjects, especially for the lower masker level condition, reflecting the highly stable nature of this brainstem response measurement. In contrast, MLR latencies are the most variable among all measures, especially in the 60 dBEM condition. Half of the subjects show no change or a slight *decrease* in the slope of their masked MLR intensity-latency function for the 40 dBEM condition, and about half showed no change or a decrease in the slope for the 60 dBEM condition compared to

their results for the lower-level noise condition. All of the ABR intensity-latency functions, on the other hand, increase with increasing masker level, as do all but one of the AME exponents. The results of the ANOVA show a strong trend of increasing slope with increasing masker level for the ABR/MLR latencies and loudness growth [main effect noise: $F(1,9) = 10.37$, $p = .011$], a trend toward an effect of measure [$F(1,12) = 5.07$, $p = .032$], and no measure × noise condition interaction [$F(2,18) = .51$, $p = .61$], indicating no difference between measures for the amount of slope increase with increasing masker level. Because the MLR latency exponents are so variable and several individual results show little or no change in these normalized exponents, and because the individual and mean data for the ABR latency and AME exponents suggest that these two measures change similarly, a second analysis of variance of only the ABR latency exponent increase and the AME results was conducted. Although the overall amount of exponent increase for the AME was significantly greater than for ABR latencies [measure effect: $F(1,9) = 18.63$, $p = .002$], there was a significant effect of noise condition [$F(1,9) = 20.68$, $p = .001$], and no significant measure × noise interaction [$F(1,9) = 2.13$, $p = .178$], indicating that masking noise causes a similar amount of increase in the rate of ABR latency decrease and loudness growth for these normal listeners.

The above analyses provide a means of evaluating the effects of masker level on the slopes of the ABR/MLR and loudness functions, and also a means by which the electrophysiologic and behavioral exponents for the partially-masked tones could be compared. One further step was taken to analyze the effects of masker level across measures by calculating a single value of the relative amount of increase in slope with

increasing masker level using the normalized data from Figure 6 for 10 subjects. This gives values indicating the proportional amount of increase in the neural and behavioral exponents for the higher noise level relative to the amount of increase for the 40 dBEM condition, which may then be used to assess whether or not the relative amount of change between masker level conditions is similar or different among measures. Each subject's ratio obtained for the higher noise level was divided by the subject's respective lower noise level ratio (i.e., 60÷0/40÷0). Table 2 presents results of these analyses for the10 remaining normal-hearing subjects. Both the mean and median proportional increase are slightly larger for the ABR and MLR amplitudes compared to the latency and mean loudness values, but generally the results are similar across measures, except for the median MLR latency values. The results for MLR latencies show the smallest median increase (26%) and the greatest variability among all measures. Review of the individual data reveals that the slope of the MLR intensity-latency function decreased for the higher level noise condition for four of these 10 subjects compared to their results for the lower-level noise condition. One-way analyses of variance indicate no significant differences between ABR/MLR amplitudes and loudness in the relative amount of slope increase with increasing masker level [measure effect: $F(2, 18) = 0.38$, $\varepsilon = 1.00$ $p = .688$], nor is there a significant difference between ABR/MLR latencies and loudness [measure effect: $F(2,18) = 0.17$, $\varepsilon = 1.00$, $p = .844$]. The finding of no significant difference between the ABR/MLR latency and loudness exponents may be due, in part, to the variability in MLR latencies, which decreases the power of this statistical analysis. With MLR latencies excluded from the analysis, the ANOVA shows no significant difference between ABR

latencies and loudness for the amount of increase in their slopes with increasing masker level [measure effect: $F(1,9) = .29$, $p = .605$]. Thus, the finding that the *relative* amount of increase in slope with increasing masker level is similar for these electrophysiologic and behavioral measures indicates that the ABR/MLR relate to the loudness percept.

Sensorineurally-hearing-impaired (SNHL) subjects

Figure 7 shows the waveform intensity series and magnitude estimations from one normal-hearing and two representative hearing-impaired subjects. Each subject's estimates of loudness magnitude (AME) are listed to the left of the intensities (in dB nHL), with the corresponding ABRs/MLRs shown to the right. The subject in the center panel had mild (40 dBHL) sensorineural hearing loss, and the subject in the right panel had moderate (55 dBHL) sensorineural hearing loss with significantly reduced dynamic range of hearing (35 dB). With increasing intensity, each subject's ABR/MLR amplitudes increase and latencies decrease, and the magnitude estimates increase.

Intensity functions for the three measures (amplitude, latency reciprocal, and AME) were determined for each of these subjects, and are shown in Figure 8. The results of the two hearing-impaired subjects are depicted within each panel by solid symbols and plotted on log-log coordinates with those of the normal listener for amplitudes (upper panels), latency reciprocals (lower panels), and AMEs (right panel). These hearing-impaired subjects' ABR/MLR amplitude and AME functions are steeper than those for the normal listener. Also, the amplitude-intensity and loudness functions are steeper for the subject with greater hearing loss (right panel, Figure 7) compared to the subject with mild hearing loss (center panel, Figure 7), consistent with the previous section's finding

of an increase in slope with increasing threshold shift. These results are also seen in the individual exponent values shown within each panel. In contrast, the slopes of the ABR and MLR intensity-latency functions for both of these impaired listeners are essentially the same as those of the normal-hearing subject, and this can be seen also by comparing the ABR and MLR latency exponents for these three subjects.

Table 3 shows the average exponents for the 12 hearing-impaired subjects compared to the results for the normal listeners' non-masked responses from Table 1. ABR amplitude exponents for the impaired listeners are larger, but not significantly, than their respective MLR amplitude exponents [wave main effect: $F(1,11) = 7.96$, $p = .017$], and there is no difference between their ABR and MLR latency exponents [wave main effect: $F(1,11) = 4.44$, $p = .06$]. Similar to the findings for the normal listeners, the hearing-impaired subjects' behavioral loudness exponents are larger than their respective neural responses [main effect measure: Amplitude & AME: $F(2,22) = 7.70$, $p = .003$; Latency & AME: $F(2,22) = 79.68$, $p < .001$], and *post hoc* analysis reveals that this is true for all means comparisons ($p < .01$), except between ABR amplitudes and loudness ($p = .09$). In order to assess the differences between the normal and SNHL groups' ABR/MLR amplitude, latency, and loudness exponents, three separate analyses of variance were performed. The 2-factor ANOVA [wave (2) × group (2)] shows that the evoked potential amplitude exponents for the impaired listeners are larger than those of the normal group [main effect group: $F(1,21) = 17.73$, $p < .001$], and *post hoc* analysis reveals that this is true for both the ABR ($p < .001$) and MLR ($p = .001$). ABR/MLR latency exponents for the hearing-impaired listeners, however, are not significantly different from those of the

normal-hearing subjects [group main effect: $F(1, 21) = 2.70$, $p = .115$]. One-way analysis of variance on the behavioral measures shows that the SNHL subjects' AME exponents are larger than those of the normal-hearing subjects [main effect group: $F(1,21) = 13.29$, $p = .002$].

In Figure 9 are shown the individual AME (left panel) and ABR/MLR amplitude (upper panels) and latency (lower panels) exponents for 11 normal-hearing listeners and 12 sensorineurally-impaired subjects, plotted as a function of their 1000-Hz pure-tone behavioral thresholds (dBHL). Generally, AME, ABR/MLR amplitude, and ABR latency exponents increase with increasing threshold shift, as does the variability of response measures. ABR latency exponents tend to increase with increasing hearing loss, but to a smaller degree than the behavioral loudness and ABR/MLR amplitude results. The exponents of the impaired listeners with mild hearing loss are within the range of those of some of the normal-hearing subjects for each of these measures. The relation between the electrophysiologic and behavioral exponents and the degree of hearing loss is shown by the regression line in each panel. Analyses of the correlation of these values show a moderate-to-strong relation between AME slope values and threshold ($r = 0.67$, $p < .001$), ABR latency exponents and threshold ($r = 0.60$, $p = .003$), and ABR/MLR amplitude exponents and hearing level (ABR: $r = 0.75$, $p < .001$; MLR: $r = 0.82$; $p < .001$). For MLR latency exponents, however, the correlation coefficient between the individual exponent values and threshold level does not differ significantly from zero ($r = 0.03$, $p = .87$). The proportion of predicted variance, indicated by r^2 within each graph, is lower for the behavioral measures compared to the ABR/MLR amplitudes.

indicating the greater variability of this subjective measure, and also reflecting the increasing variability in the slope of the loudness function with increasing hearing loss (Hellman & Meiselman, 1990).

The relation between AME exponents and the electrophysiologic exponents was also analyzed. Correlation of these values shows a significant relation between the slopes of the behavioral loudness growth functions and the amplitude exponents of the ABR (r = .53, p = .01) and MLR (r = .61, p = .002). By contrast, nonsignificant correlation coefficients of 0.41 (p = .052) and 0.13 (p = .539) were obtained between the loudness exponents and ABR latency exponents, and between AME exponents and MLR latency slope values, respectively.

Figure 10 shows mean latency reciprocals and amplitudes for the non-masked normal-hearing group (N = 11), and for two subgroups of the SNHL subjects. Eight of the hearing-impaired subjects had mild hearing-loss (40-50 dB nHL), and four subjects' thresholds were in the moderate hearing loss range (55-60 dB nHL). To illustrate the effects of degree of sensorineural hearing loss on the electrophysiologic and behavioral responses with intensity in comparison to the results for normal listeners, amplitudes, latencies, and magnitude estimates were averaged separately for these two subgroups. Each data point represents the average of at least three subjects' responses (in most cases, four) from the smaller group (n=4) of hearing-impaired subjects with moderate hearing loss, and six or more subjects' responses are included in the average results for the group with mild hearing loss. The results for ABR and MLR amplitude and loudness functions show an increase in slope with increasing hearing loss, a pattern that is seen also in the

normal listeners' responses to masked tones (Figure 5). ABR intensity-latency functions (left panels) are slightly steeper for the mildly-impaired subjects compared to the normal response, and become slightly more steep with increasing hearing loss. The slope of the MLR intensity-latency function, however, is steeper only for the subjects with greatest hearing loss; for the impaired listeners with mild degree hearing loss, the MLR intensity-latency function is actually *less* steep than the normal results. This pattern of results for the impaired listeners' ABR/MLR latencies relative to the normal listeners' non-masked response is seen also in the results for the noise-masked normal-hearing subjects (Figure 5). Thus, relative to the normal non-masked response, sensorineural hearing loss results in changes in the rate of amplitude (ABR/MLR) and loudness growth, and latency decrease (primarily ABR), that are somewhat similar to the changes in the electrophysiologic and behavioral measures caused by masking in normal listeners.

Figure 11 shows the individual impaired listeners' exponent values for the electrophysiologic and behavioral measures plotted relative to the mean results from the non-masked normal subjects. That is, for each measure, each hearing-impaired subject's exponent value was divided by the mean exponent derived from the non-masked normal-listeners' responses (N = 11). Values greater than 1.0 indicate an increase in the slope of the impaired subjects' results relative to the mean normal non-masked response. The results in this figure show that most of the impaired subjects' exponents for ABR/MLR amplitude, ABR latency, and AME exponents are larger than the normal response, indicating a faster rate of change in these measures with increasing intensity for these sensorineurally-impaired listeners with mild-to-moderate hearing loss. For example, the

mean ABR amplitude ratio for the impaired listeners is 2.11, indicating a 111% increase in their rate of amplitude growth compared to the normal listeners' non-masked response. AME slope values show a smaller mean increase of 58% for these impaired listeners, but all of these subjects' loudness functions are steeper than the non-masked normal response. Conversely, MLR latency exponent values show only a mean increase in slope of 6%, indicating no change in the rate of timing of the early cortical response with increasing intensity. About half of the impaired subjects' intensity-latency functions *decrease* relative to the normal non-masked response, and inspection of the individual data reveals no pattern to these results. Steeper functions are seen in some of the mildly-impaired subjects, while others in this group have the same slope or a less-steep slope as that of the normal-hearing subjects. The moderately-impaired subjects show similar variability in their results. These findings indicate that cochlear hearing loss has no consistent effect on the timing of the early cortical response for supra threshold stimuli.

One-way analysis of variance indicates that the relative amount of increase in the exponents for the hearing-impaired subjects is not significantly different between ABR/MLR amplitudes and loudness [measure effect: $F(2,22) = 3.99$, $\epsilon = 1.00$, $p = .033$], although the trend may indicate a somewhat greater relative increase in the evoked potential amplitudes compared to their behavioral loudness results. ABR/MLR latencies and loudness also show no significant differences [measure effect: $F(2,22) = 4.18$, $\epsilon = 1.00$, $p = .029$]. With MLR latencies excluded due to their considerable variability, the ANOVA shows no significant difference between ABR latencies and loudness [$F(1,11) =$

.35, p = .56]. Taken together, these results suggest that the evoked potential results, with the exception of MLR latencies, relate to loudness.

Discussion

Relation between loudness and the ABR/MLR to brief tones

Normal-hearing subjects. The brainstem and early cortical (MLR) auditory evoked potentials increase in amplitude and decrease in latency with increasing stimulus intensity (Chapter 5, this volume). As well, the loudness of 1000-Hz brief-tones grows as stimulus level increases. The mean exponents derived from the individual functions relating the electrophysiologic and behavioral responses to stimulus intensity suggest that these stimulus-response relationships may be described by a power function. The exponents for the neural responses are smaller, however, than those obtained from subjective loudness estimates, indicating a slower rate of change in these electrophysiologic responses with increasing intensity compared to loudness growth.

The results of the present study on the relation between ABR/MLRs and loudness for non-masked tones are consistent with previous studies that examined the click-evoked ABR and MLR and loudness in normal-hearing subjects (Howe & Decker, 1984; Madell & Goldstein, 1972; Pratt & Sohmer, 1977; Serpanos et al., 1997; Wilson & Stelmack, 1982). For example, Pratt & Sohmer (1977) obtained power functions for both latency and amplitude changes of the click-evoked ABR in response to increases in intensity from a group of normal-hearing subjects, which they compared to the mean loudness exponent of 0.26 obtained from subjective magnitude estimates. These researchers obtained an exponent of 0.14 for the amplitude growth of wave V, and a flatter slope for the decrease

in ABR latency which gave an exponent of -0.03 (reflecting the inverse relationship between latency and intensity). These results are similar to those of the present study, which show a mean non-masked loudness exponent of 0.29, and mean exponents of 0.18 and 0.08 for ABR amplitudes and the wave V latency reciprocal (i.e., latency decrease), respectively. The ABR latency exponent of 0.08 obtained in the present study is somewhat larger than those previously reported, possibly reflecting the fact that our data extend further into the lower intensity range (i.e., 0 to 20 dB nHL) where the slope tends to increase (Picton, Stapells & Campbell, 1981). This result also reflects, in part, the transformation of these data into reciprocal values. The general finding of previous studies of a slower rate of change in ABR latencies compared to both their amplitude and loudness functions is evident in our results.

From their investigation of the MLR, Madell and Goldstein (1972) obtained a mean loudness exponent of 0.28 from their normal-hearing subjects' magnitude estimates to clicks. Their MLR amplitude exponents of 0.11 and 0.09 for the MLR Po-Na and Na-Pa respectively, are also in fairly good agreement with the results of the present study, which gave an exponent of 0.13 for Na-Pa amplitudes for non-masked brief tones. Their Figure 2 shows a shallower function for the latency decrease of MLR waves Po, Na, Pa, and Nb compared to the amplitude results, but their analyses did not include exponents for latency measures. The rate of decrease in wave Pa latency found in the present study for non-masked tones yielded a mean exponent value of 0.08, which is the same as that obtained for wave V of the ABR. This is consistent with the results of other studies that used a simultaneous recording technique and reported a similar rate of latency decrease

with increasing intensity for the ABR and MLR to non-masked clicks (e.g., Picton et. al., 1974) and tones (e.g., Wu & Stapells, submitted).

The finding that the characteristics of the auditory evoked potentials change at a slower rate than do measures of loudness growth is now extended to include tone-evoked ABR/MLRs recorded using acquisition parameters that optimize these responses. This result may be due to the inherent difference between these measures' response output (i.e., voltage versus cognitive numerical judgement), to the choice of methods used to compare the two measures, or possibly to some other methodological issue that has yet to be appropriately applied/determined. The transient auditory evoked potentials reflect synchronous neural activity of cells that are geometrically organized so that their dipole fields summate: synchronous activity is greatest at the onset of a sound (Picton, 1990). Neuronal activity that is processed at different rates and/or from cells that generate fields oriented in multiple directions is not recorded by the electrode at the scalp (Picton, 1990). These electrical responses might, therefore, reflect activity of processes that are necessary for evaluation (or transformation) of incoming sensory information, and may not include the activity of the cerebral processes involved in their analysis (Picton, Woods & Proulx, 1978; Zwislocki, 1969).

Because the amplitudes of the auditory evoked potentials are related to stimulus intensity, however, they are also related to the perceived loudness of acoustic stimuli, and many findings support such a relationship (e.g., Davis, Bowers & Hirsh, 1968; Davis, Mast, Yoshie & Zerlin, 1966; Davis & Zerlin, 1966; Knight & Beagley, 1969; Uziel & Seneclause, 1978). For example, in his studies in electrocochleography, Eggermont

(1977) examined the whole nerve action potential (AP) in normal and hearing-impaired ears using tones, and found steeper AP intensity-amplitude functions in the sensorineurally-impaired ears compared to the results from normal-hearing and conductively-impaired listeners, similar to the steeper loudness function obtained in persons with sensorineural hearing loss (e.g., Hellman & Meiselman, 1990). More recently, the assumption that ABR amplitudes are related to loudness (Kiessling, 1982, 1983) was tested by Davidson, Wall and Goodman (1990) who recorded the click-evoked ABR from normal-hearing subjects and reported a strong relationship between ABR amplitudes and subjective magnitude estimates.

The problem of relating a quantity (such as an exponent) of the evoked potential amplitude growth to a psychoacoustic correlate has also to do with the choice of parameters used to obtain these measures. Many methodological factors that bear on the acquisition of these responses can affect the outcome of both measures, and some may affect only one. For example, the electrophysiologic test is influenced by filtering, type of stimuli, rate of presentation, number of responses averaged, and biological as well as external noise sources among others (e.g., Picton et al., 1981; also see review by Picton, 1990). Behavioral loudness measures are influenced by the psychoacoustic method, the listening period, the type of stimuli, the rate of presentation, order of stimulus presentation, and the subject's ability to match an internal numerical representation of stimulus level, among others (e.g., Stevens, 1957; Hellman & Zwislocki, 1961; Marks, 1991). As a case in point, both the *average* ABR/MLR amplitude and behavioral loudness growth functions show a fairly linear decrease as intensity is decreased to low-

sensation levels (Figure 5). Some individuals' AEP amplitudes at low-sensation levels, however, revealed a leveling off, or flattening of their responses at and near threshold (Figures 3 and 4), which is probably a "floor effect" due to the AEP amplitude dropping to the level of the residual background noise in the AEP waveform. As well, the same low-sensation-level plateau is also observed in some of the individual near-threshold AME measures obtained in the present study, and by others (e.g., Buus, Müsch & Florentine, 1998). It may be that biological noise effects, a common parameter bearing on both responses, are responsible for low-sensation-level plateaus of some individual subjects' ABR/MLR and/or loudness results. Or it may be that other concomitant factors, such as a subject's bias toward a particular numerical representation at near-threshold levels in the case of loudness growth, or the number of sweeps averaged in the case of amplitude growth, may be responsible for this finding in some individual results, but which are not reflected in the mean group data (present study; Buus et al., 1998; Elberling & Don, 1987).

A second common parameter that affects the outcome of both measures, rate of stimulus presentation, bears closer scrutiny. The transient evoked potentials are rate sensitive, such that increasing stimulus presentation rate decreases AEP amplitudes and increases latency (Picton et al., 1981; Keidel & Spreng, 1965; Davis & Zerlin, 1966). More relevant to the present discussion is the finding that increasing repetition rate results in a slower rate of AEP amplitude growth (Darling & Price, 1990; Keidel & Spreng, 1965; Picton et al., 1978b; Rau, 1968). The opposite effects are seen for behavioral loudness measures, such that the loudness of brief tones or clicks increases for repetition

rates greater than 5/s (Garner, 1948; Darling & Price, 1989, 1990; Zwicker & Fastl, 1999), and the slope of the loudness function steepens with increasing repetition rate (Darling & Price, 1990; Zwicker & Fastl, 1999). Because rate has an opposite effect on the electrical and behavioral measures to increasing intensity, it may be that repetition rates of 3/s or 5/s would yield ABR/MLR amplitude and loudness growth exponents that are equal. However, employing a very slow rate (i.e., 1-3/s) for ABR/MLR acquisition in order to achieve numerical equivalency in the behavioral and electrical exponents compromises efficiency of data collection, especially from a clinical standpoint. Rather, it may be for future studies to determine standards of comparative measures using established test parameters that may then be used for research and clinical purposes. The lack of numerical agreement in exponent size between the neural responses and the function that describes loudness perception does not preclude establishing such a functional relationship (Eggermont, 1982b; Jacobson & Hyde, 1985).

A final point to consider regarding the exponent difference between the neural and behavioral responses is that their relationship may be better evaluated by some other method(s). Such is the focus of the present studies by which we sought to establish whether or not these AEPs reflect part of the neural processes that contribute to the pschoacoustic loudness percept by comparing the relative changes in the masked AEP exponent with behavioral measures of loudness growth for masked tones. It is well established that the loudness function for partially-masked tones becomes steeper with increasing threshold shift (Cefaretti & Zwislocki, 1994; Hellman & Zwislocki, 1964; Schlauch, Digiovanni & Ries, 1998; Stevens & Guirao, 1967; Zwicker & Fastl, 1999). If

the ABR and MLR reflect the neural activity that subserves loudness perception, then the changes observed in the slopes of the masked loudness function should also be reflected in the characteristics of the masked electrical responses.

Effects of masking. The results of the present study show that broadband masking noise effectively shifted thresholds for detection of 1000-Hz brief tones to 40 and 60 dB nHL levels (Chapter 5, present volume), and caused an increase in the rate of change in the amplitudes and latencies of the ABR and MLR with increasing intensity and loudness. Masked ABR and MLR intensity-amplitude functions steepen at about the same rate within each condition, and their slopes increase with increasing threshold shift. The mean ABR intensity-latency functions for masked tones also steepen with masking. This pattern of ABR/MLR amplitude growth/latency decrease for masked tones is similar to that seen for loudness. Subjective magnitude estimates of these partially-masked brief tones also yield steeper slopes than the response for tones alone. Although the slope increase for masked tones is greater for subjective loudness compared to the change in slope for masked ABR/MLR amplitudes and latencies, the relative amount of increase is not significantly different between the electrophysiologic and behavioral measures. These results indicate that the ABR/MLR reflect, at least in part, the neural processes that subserve loudness perception.

This is the first study to examine masked tone-evoked ABR/MLR intensity-amplitude/latency functions in normal-hearing subjects and their relation to subjects' masked behavioral measures of loudness growth. An earlier investigation of the slow cortical N1 showed similar results (Davis, Bowers & Hirsh, 1968). Davis et al. (1968)

obtained cortical auditory evoked potential recordings to 250, 1000, and 4000 Hz tones in normal-hearing subjects. Tones were presented in the quiet and in narrow-band noise centered about each tone frequency. The masked N1 intensity-amplitude functions were steeper than the non-masked response, and the function steepened with increasing threshold shift, similar to the ABR/MLR findings obtained in the present study. The consistency of the findings of increasing rate of amplitude growth with increased threshold shift seen in the masked electrophysiologic responses reflecting neural activity from successive levels of the auditory system pathway [i.e., brainstem, early cortical (MLR), and slow cortical (N1) responses] strongly supports the notion that AEP amplitudes are, in part, related to loudness.

Loudness growth for these brief 1000-Hz tones also increases with increasing threshold shift, in agreement with the general findings of previous investigations that reported the effects of a noise masker on the loudness growth of long-duration (Cefaretti & Zwislocki, 1994; Gleiss & Zwicker, 1964; Hellman & Zwislocki, 1964; Lochner & Berger, 1961; Schlauch et al., 1995, 1998; Stevens & Guirao, 1967) and short-duration tones (Florentine, Buus & Robinson, 1998; Richards, 1977; Schlauch et al., 1998). For example, Schlauch et al. (1998) obtained masked loudness growth functions for 4000-Hz tones of 250 ms and 2 ms duration from normal-hearing subjects using a loudness balance procedure, and found that the slope of the loudness function was steeper for the high-level noise condition compared to that obtained in a low-level noise condition for both duration stimuli. Their exponents of 2.17 and 5.43 for the 2-ms tones presented in 62 and 83 dBSPL broadband noise, respectively, are larger than the exponents of 0.77 and 1.54

obtained in the present study for 5-ms 1000-Hz tones presented in comparable noise levels (67 and 82 dBSPL), but the effect of an exponent increase with increased threshold shift is seen in both sets of data.

Masking causes similar effects of increased rate in response growth for both the neural and behavioral responses, indicating that common underlying physiologic mechanisms likely are responsible for the "recruitment" seen in the masked normal listeners' response measures. These include both peripheral and central mechanisms (e.g., Arle & Kim, 1991; Burkard & Palmer, 1997; Costalupes et al., 1984; Delgutte, 1990, 1996; Dolan & Nuttall, 1989; Gerken, 1991; Oxenham & Plack, 1997; Pang & Guinan, 1997; Phillips, 1987, 1990; Phillips & Hall, 1986; Ruggero, Robles & Rich, 1992; Stevens & Guirao, 1957; Zwicker & Fastl, 1999). At the cochlear level, the broadband noise stimulates the whole basilar membrane, obscuring the response to the tones, and changes the mechanical-to-electrical transduction of the hair cells (e.g., Dolan & Nuttall, 1989; Geisler, Yates, Patuzzi & Johnstone, 1990). Simultaneous presentation of a tone signal at levels just above the masker level cause mechanical suppression, which reduces the gain of the cochlear amplifier resulting in reduced nonlinearity of basilar membrane motion (Ruggero et al., 1992). As the intensity of the tones increases to moderate and high levels, the traveling wave motion of the basilar membrane becomes more passive, and its frequency tuning curves are broadened (Evans, 1977), incorporating activity from a larger area of the cochlear partition. These effects of the noise that cause an increase in threshold, reduction in the nonlinearity of basilar membrane motion, and spectral summation of activity from a larger response area of the basilar membrane are thought to

contribute to the more linear (i.e., steeper) masked intensity growth function (e.g., Dolan & Nuttall, 1989; Pang & Guinan, 1997; Oxenham & Plack, 1997; Ruggero et. al., 1992; Schlauch et. al., 1998; Yates et al., 1990; Zwicker & Fastl, 1999).

The effects of a noise masker on central neural responses include excitatory and inhibitory processes, saturation by the noise background, and possibly convergence of inputs at more central loci that may resist saturation on many central neurons, thereby allowing for enhancement of the signal in noise (e.g., Arles & Kim, 1991; Gerken, 1991; Phillips, 1987; Phillips & Hall, 1986). For example, a continuous noise background has been shown to impose little or no change in the discharge rate of some cortical neurons, leaving the full range of those cells available for encoding supra threshold tones presented in that noise (Gumnit & Grossman, 1961; Phillips & Hall, 1986). In addition, tone intensity functions of some cortical cells are steepened by noise maskers when there is significant sensitivity or threshold shifts (Phillips and Hall, 1986), and enhancement of the intensity function to the tone may therefore be due to excitation of a larger population of cortical neurons than that to non-masked tones of the same intensity (Phillips, 1987).

These effects of tone and noise interactions are seen in both the masked evoked potential and behavioral responses in the present study. For example, the normal listeners gave an average loudness estimate of 5.6 for 70 dB nHL non-masked tones. For this same signal level in the 40 dBEM condition, the average estimate of 3.6 indicates a reduction in loudness for these partially-masked tones. Similarly, the mean voltage values of 0.48 μV and 0.55 μV obtained from recordings to masked 70 dB nHL tones for the ABR and MLR, respectively, show a reduction in the magnitude of the electrical

responses for this level signal compared to the non-masked response (0.78 μV for the ABR and 0.90 μV for the MLR). When signal level increased to 90 dB nHL, however, the magnitude of the masked responses increased to values that were about equal to or slightly greater than those obtained to 90 dB nHL non-masked tones. That is, the loudness estimate of 10.3 given for 90 dB nHL tones in 40 dBEM noise is nearly equal to the estimate of 9.7 given for the non-masked tones, and the same pattern is seen in the neural responses. The masked ABR and MLR voltage values of 1.03 μV and 1.32 μV, respectively, are also about the same as the magnitude of the non-masked response for this high-intensity level signal (ABR = 1.02 μV and MLR = 1.21 μV). Thus, the broadband noise produces masked loudness and amplitude functions that are steeper than the non-masked response, indicating that these behavioral and evoked potential measures are related.

The rate of change in the latencies of the masked ABR and MLR shows effects of the noise masker that are somewhat comparable to those observed in the loudness and amplitude results. That is, the mean masked ABR and MLR intensity-latency functions are steeper than the non-masked responses. There are, however, differences between these evoked potential response measures and the masked amplitude and behavioral results. Although, masked ABR intensity-latency functions showed a similar pattern of change in their slopes as that seen for loudness, their exponent increase relative to the non-masked response is significantly smaller than the AME results, and their relative amount of increase for the higher-level noise condition is also smaller than the loudness exponent increase. These results suggest that ABR latency may not reflect all of loudness

growth or, possibly, that the rate of change in the timing of the ABR, although level dependent, instead reflects other aspects of signal processing such as rise/fall time (Heil, 1997a,b; Phillips & Burkard, 1999), the effects of which are determined in large part by peripheral mechanisms (Heil & Irvine, 1997).

Masked MLR latencies show an average increase in slope compared to the non-masked response, but the increase for the 40 dBEM condition is not significantly different than the non-masked response, and the change in the amount of relative increase between the 40 and 60 dBEM conditions is very small and highly variable. Indeed, masked MLR intensity-latency functions are the most variable among all measures; about half of the noise-masked normal-hearing subjects showed essentially no change or a decrease in their masked MLR intensity-latency slopes. This contrasts with the masked loudness, ABR/MLR amplitude, and ABR latency results, all of which showed increases in their slopes with increasing masker level. The smaller effect of the noise masker on the MLR latencies possibly reflects central mechanisms of masking that enhance the response to signal at this more rostral level of the auditory pathway (e.g., Gerken et al., 1991), including convergence of activity at the early cortical level from which this response emanates (Hashimoto et al., 1995; Heil, 1997a,b; Liégeois-Chauvel et al., 1994). The variability seen among subjects in the changes in timing of their masked MLR may be due to the fact that this evoked potential contains only low-frequency energy (Suzuki et al., 1983a, 1984b) resulting in more latency jitter, and thus more variable latency measures compared to the ABR, which contains both low- and high-frequency energy, and therefore more precise latency resolution (Kodera et al., 1978, 1979; Yamada et al.,

1977). The fact that masked ABR and MLR intensity-latency functions behave differently, in contrast to their amplitude results, indicates that they reflect different aspects of sound intensity (e.g., Heil & Irvine, 1997; Phillips & Burkard, 1999), and that this parameter of the evoked potential recordings likely is not a useful index of loudness growth. Although masked ABR/MLR intensity-amplitude functions also show variability in the size of the individual exponents, the noise produced a consistent effect of amplitude slope increase across subjects which is also seen in the subjects' behavioral results.

Effects of sensorineural hearing loss. Loudness recruitment seen in the behavioral results of the sensorineurally-impaired subjects is also reflected in their ABR and MLR amplitude functions. That is, the subjects with primarily cochlear hearing loss showed a faster rate of ABR and MLR amplitude growth relative to the normal response, consistent with their increased rate of loudness growth for 1000-Hz short-duration tones. In addition, the slope of the ABR and MLR amplitude functions steepened with increasing hearing loss, similar to the increase in the masked ABR and MLR intensity-amplitude functions of the normal-hearing subjects. These results thus extend the masked findings obtained from the normal-hearing listeners, providing further support for the notion that AEP amplitudes do reflect, at least in part, the neural sources underlying loudness perception. The ABR intensity-latency functions of the SNHL subjects also are steeper than the normal non-masked response, but this was not the case for their MLR latencies, which showed no consistent change in slope with increasing hearing loss, and thus do not appear to reflect subjective loudness.

The findings in the present study relating the growth in ABR/MLR amplitudes and loudness are in agreement with the results obtained from studies that investigated the relation between the rate of amplitude growth of the slow cortical response (N1) and loudness measures from subjects with sensorineural hearing loss (Clayton & Rose, 1970; Knight & Beagley, 1969; Uziel & Seneclaus, 1978). For example, Knight and Beagley (1969) reported good agreement between the steeper loudness growth function obtained from unilaterally-impaired subjects using the Alternate Binaural Loudness Balance procedure (ABLB; Fowler, 1936), and the growth of the N1 recorded to 20-ms 1000-Hz tones. The slope of the N1 intensity-amplitude function in sensorineurally-impaired subjects increases also with increased threshold shift (e.g., Uziel & Seneclaus, 1978), in agreement with the ABR and MLR results for the subjects with mild-to-moderate sensorineural hearing impairment who participated in the present investigation. In further support of this relationship, AME and ABR/MLR amplitude functions are significantly correlated, although the amount of predicted variance is low, due probably to a restriction of range of hearing loss included in the present study.

The relation between the increase in tone-evoked ABR intensity-latency and loudness functions seen in the present study is consistent, also, with the click-evoked ABR findings reported by Serpanos et al. (1997). These researchers also reported steeper-than-normal click-evoked ABR intensity-latency functions obtained from their subjects with flat sensorineural hearing loss, but did not find a significant correlation between the exponents of the wave V intensity-latency functions and the AME functions they obtained from magnitude estimate and production procedures. Low, non-significant correlations

were also obtained between the AME and ABR/MLR intensity-latency functions in the present study. The effects of sensorineural hearing loss on the behavior of ABR and MLR amplitudes and latencies found in the present study are in agreement, also, with the results of previous studies that reported increases in the rate of amplitude growth (ABR: Hecox, 1983; Kiessling, 1982, 1983; MLR: McFarland et al., 1977) and latency decrease (ABR: Arslan et al., 1988; Galambos & Hecox, 1978; Suter & Brewer, 1983; Yamada et al., 1979) in the click-evoked ABR and tone-evoked MLR recordings obtained from subjects with sensorineural hearing loss.

The effects of sensorineural hearing loss on the neural and behavioral measures in the present study are in agreement with the reduction in the dynamic range of hearing associated with loudness recruitment (Hallpike, 1976). Several different cochlear and neurophysiological mechanisms have been proposed to account for this abnormally rapid growth of loudness and amplitude increase. Recruitment is attributed primarily to peripheral effects of the loss or damage of hair cells in the cochlea due to disease or trauma (Ruggero, 1992; Salvi, Henderson & Hamernik, 1983; Saunders et al., 1991; Zhang & Zwislocki, 1995), which results in loss of the cochlear amplifier, loss of sensitivity at the tip of the basilar membrane tuning curves, broadened frequency tuning curves, and reduction in the nonlinearity of the basilar membrane (Geisler, 1998, pp 301; Salvi et al., 1983; Saunders et al., 1991). The more linear response of the basilar membrane to increasing sound pressure level due to partial or complete elimination of the active feedback mechanism of damaged or diseased outer hair cells is thought to result in a shift in threshold and steeper, or more linear, intensity-growth functions (Oxenham &

Plack, 1997; Patuzzi, 1996; Salvi et al., 1983; Zhang & Zwislocki, 1995). As well, auditory neurons that innervate pathologic cochleae have raised absolute threshold, steeper-rate intensity functions (e.g., Schmiedt & Zwislocki, 1980), and their tuning curves become broader and, therefore, more overlapping (Evans, 1978). This being so, it has been hypothesized that these physiologic mechanism of VIIIth nerve function in subjects with impaired cochleae also play a basic role in loudness recruitment (Evans, 1978; Phillips, 1987).

Studies that have recorded evoked potential measurements from multiple central sites, including the inferior colliculus and cortex, have shown that these electrical responses to tones in cochlear-impaired animals increase rapidly with intensity and are larger than normal (i.e., amplitude enhancement) despite the reduced cochlear output to low- and moderate-intensity tones (e.g., Gerken, 1991; Gerken, Solecki & Boettcher, 1991; Henderson & Salvi, 1998; Qui, Salvi, Ding & Burkard, 2000; Salvi, Saunders, Gratton, Arehole, & Powers 1990; Salvi, Wang & Ding, 2000; Szczepaniak & Moller, 1996; Syka, Rybalko & Popelar, 1994; Wang, Salvi & Powers, 1996). The increase in these evoked potential amplitudes is thought to be due to selective loss of inhibition at central loci, and to decreased temporal integration resulting from cochlear damage (Gerken et al., 1991). Henderson & Salvi (1998) suggest that this enhanced neural activity recorded from rostral sites may be related to loudness recruitment seen in sensorineurally-impaired individuals. These effects are seen in the neural and behavioral responses of the impaired listeners in the present study (see also Chapter 5, this volume). The average increase in the slopes of their ABR amplitude/latency functions is slightly greater than their results for the MLR,

probably due to the effects of peripheral damage and the loss of cochlear nonlinearities which are reflected to a greater degree in the brainstem recordings that include activity from cochlear, VIII[th] nerve, and lower brainstem sites (e.g., Legatt et al., 1988; Møller, 1994). However, the absolute amplitudes of the SNHL subjects' MLRs are the same as or larger than those of the normal-hearing subjects (Chapter 5, present volume) and, relative to the non-masked normal response, the MLR amplitude exponent increase for the sensorineurally-impaired subjects is larger than that seen in their ABR results, consistent with response enhancement at this more rostral level (Henderson & Salvi, 1998; Salvi et al., 2000).

The slope of the MLR intensity-latency functions for this group of hearing-impaired subjects with mild-to-moderate hearing loss showed no consistent effect of cochlear pathology on the timing of the early cortical response. These results indicate reduced effects of peripheral pathology at this more rostral level and/or possibly central enhancement of signal at this early cortical level (e.g., Gerken, 1991; Syka & Rybalko, 2000).

Despite the small number of subjects included in the present study, the moderate correlation seen between the behavioral loudness measures and degree of hearing loss is in fairly good agreement with previous studies that reported results of larger groups of hearing-impaired subjects, and showed that the slope of the loudness growth function depends on the severity of hearing loss (Hellman, 1999; Hellman & Meiselman, 1990). The variability in the behavioral results is consistent also with earlier studies that have

shown intersubject variability in AME measures obtained from normal (Viemeister & Bacon, 1991; Hellman 1981), and impaired listeners (e.g., Hellman & Meiselman, 1990).

ABR and MLR amplitude exponents for the sensorineurally-impaired subjects are strongly correlated with hearing threshold, suggesting that this parameter of the electrical response may be a useful index of recruitment in hearing-impaired listeners (e.g., Eggermont, 1982b). Their ABR latencies, on the other hand, show less correspondence with increasing threshold shift compared to that seen for the amplitude results, and their MLR latency exponents do not correlate at all with hearing threshold, reinforcing the notion that this parameter of evoked potential recordings is likely not a metric of the loudness percept.

Effects of noise masking vs sensorineural hearing loss. Overall, broadband masking and sensorineural hearing loss caused similar effects of increasing rate of change in ABR/MLR amplitudes and latencies, and loudness growth. ABR/MLR amplitude and loudness growth functions are steeper for both the noise-masked normal-hearing (NMNH) and SNHL subjects, and their exponents increase with the level of threshold shift, indicating that these electrophysiologic and behavioral responses are related. Their ABR intensity-latency functions are steeper also, but the ratio of exponent increase is smaller than that seen in their amplitude results. The similarity in the pattern of ABR latency results between these two groups of impaired listeners is likely due to the fact that the configuration as well as the degree of hearing loss were similar for both groups of subjects. MLR latency exponents for both the NMNH and SNHL subjects show essentially no consistent effect of increasing threshold shift.

The results of the electrophysiologic and behavioral responses to increasing intensity obtained in the present study also show some differences between the cochlear-impaired and noise-masked listeners' responses. The biggest difference is seen in the behavioral loudness growth measures. The slopes of the loudness functions for partially-masked 1000-Hz tones are considerably steeper and more variable than those obtained from the sensorineurally-impaired subjects with similar thresholds, indicating a greater effect of the noise masker on these behavioral responses compared to the effects of cochlear pathology. This contrasts with the findings reported by Hellman (1988), which show that broadband noise and cochlear hearing loss result in nearly identical loudness functions for long-duration tones. The results of the present study indicate that this is not true for short-duration stimuli, and this may be due, in part, to the fact that higher noise levels are required to shift thresholds of brief tones in normal listeners than that required for > 200-ms tones, yielding steeper loudness growth functions (Schlauch et al., 1998). The difference in the behavioral results between groups may also be related to changes in temporal integration (e.g., Florentine, Fastl & Buus, 1988), which is thought to relate to loudness (Buus, Florentine & Poulsen, 1999; Florentine et al., 1998). It is known, for example, that temporal integration for threshold detection is reduced in cochlear-impaired subjects, and that the reduction is positively correlated with the amount of hearing loss (Buus et al., 1999), whereas normal listeners show the same amount of temporal integration for non-masked and partially-masked tones (Florentine et al., 1988, 1998). These effects were seen in the threshold results reported in Chapter 5 (present volume). Temporal integration varies for supra threshold tones among subjects, but the results for

cochlear-impaired listeners generally show less temporal integration than NMNH subjects for moderate- and high-signal levels (Buus et al., 1999; Florentine et al., 1998).

The mean ABR amplitude exponent for the cochlear-impaired group is larger than their average MLR exponent, whereas the ABR and MLR amplitude exponents of the noise-masked subjects are the same within each noise condition. However, the ratio of increase in ABR/MLR amplitudes relative to the non-masked normal response shows a similar pattern for both groups, such that the overall MLR amplitude exponent increase is larger than that seen for the brainstem response. ABR latency exponents are larger for both the SNHL and NMNH subjects, compared to the non-masked normal response, probably reflecting the shift in threshold which is mainly due to peripheral mechanisms of masking/cochlear pathology. On the other hand, there is no consistent change in the slope of the MLR intensity-latency function for the cochlear-impaired group, relative to the normal response, whereas MLR intensity-latency functions increased for most of the noise-masked normal listeners, reflecting differences in the way signal intensity is processed at the cortical level for masked normal listeners vs SNHL listeners.

The similarities in the ABR/MLR findings between these two groups of impaired listeners extend the findings of both physiological (e.g., Gerken, 1993) and psychoacoustic (e.g. Schlauch et al., 1998; Stevens & Guirao, 1967) research that showed similar results of intensity coding/loudness growth in NMNH and cochlear-impaired subjects, and suggest that some common underlying neurophysiological mechanism(s) is/are responsible for the slope increase. The differences between these sensorineurally-impaired and NMNH subjects are not surprising given the fact that etiology of hearing

loss differs between groups, and the ABR/MLR results in the present study provide new information about where and how signal intensity processing occurs within the cochlear-impaired and masked-normal auditory system pathways in humans, and confirm that the loudness recruitment seen in these two groups of impaired listeners is also a result of different physiologic mechanisms (Phillips, 1987).

Implications

The ABR and MLR and behavioral measures provide a window into the ways in which the brain processes sound intensity in the normal and impaired auditory systems. The magnitude of change in the ABR and MLR amplitudes seen in the present study, in conjunction with the amplitude results of the slow cortical auditory evoked potential, show that at each stage of processing, these evoked potentials reflect, at least in part, the neural sources that contribute to behavioral output measures of intensity increase.

These findings have a number of implications for the study of normal-hearing processes and patient populations. ABR and/or MLR amplitude growth may be investigated in the pediatric population (i.e., <5 years of age) in order to assess developmental issues of intensity processing in these young listeners for whom loudness data are unavailable (Launer, 1998). Although ABR and possibly MLR amplitudes have, in the past, been considered too variable to establish normative data (e.g., Hecox & Burkard, 1982; Kraus et al., 1994), the results in the present investigation which were derived from a small group of adults showed fairly consistent amplitude exponents across subjects and conditions. This implies that normative ABR and MLR intensity-amplitude growth rates may be obtained from groups of infants and young children of different ages.

which may then be applied in both research and clinical endeavors. For example, evoked potential amplitude growth determined from normal-hearing children might be used to address the notion that immaturity of intensity processing is a possible contributing factor to the immaturity of many psychophysical results observed in infants, such as temporal resolution (Werner, 1996). They may also be incorporated in the audiologist's repertoire as a reference of normal hearing status.

Probably the most important application of the finding that ABR/MLR amplitudes relate to loudness is in the study and treatment of infants and older patients with hearing loss for whom behavioral data are unavailable. The results of the present study show that ABR/MLR intensity-amplitude functions might index loudness recruitment in subjects with mild-to-moderate cochlear loss of hearing sensitivity, and thus provide both diagnostic and therapeutic information. The current trend toward universal hearing screening of newborns will result in an increasing number of infants identified with hearing impairment who will subsequently be fitted with amplification by age 6 months or earlier (Gravel, 2000; The Pediatric Working Group, 2000). Presently, the ABR serves as a primary tool for estimating behavioral thresholds in infants aged 0-36 months, and as the results of the present study suggest, it may be possible to use the ABR to estimate loudness growth in this young population. Evoked potential measures of intensity processing in infants with peripheral hearing loss can serve to differentiate cochlear from neural involvement, which would improve our understanding of genetic hearing loss and its consequences (e.g., Harrison, Stanton, Ibrahim, Nagasawa & Mount, 1993; Willot & Turner, 2000). At the individual level, such information may also lead to effective

intervention strategies (The Pediatric Working Group, 2000). For example, ABR/MLR estimates of loudness growth can direct the clinician to select appropriate hearing aid parameters (Schum, 2000), and may also be used to verify and monitor aided performance (e.g., Brown, Klein & Snydee, 1999). In addition, ABR/MLR estimates of loudness growth will bear on the acquisition of later evoked potentials, such as the MMN and acoustic change complex (ACC), that have been used successfully to assess auditory discrimination capacity in normal-hearing and hearing-impaired adults using supra threshold stimuli (e.g., Whiting, Martin & Stapells, 1998; Martin & Boothroyd, 2000; Martin, in preparation). The research and clinical application of these AEPs in other groups would require some knowledge of the patient's loudness experience in order to reliably and comfortably assess these important aspects of speech sound processing in those infants, children, and adults who are unable to report their experience of sounds.

In the present studies, ABR/MLRs to supra threshold tones were obtained at several intensities. However, acquiring recordings at three or four test levels above threshold would likely be sufficient to derive valid intensity-amplitude results for loudness estimation, and such a test protocol would remain within acceptable time constraints for data collection. Examination of the relationship between these electrophysiologic and behavioral measures with intensity in hearing-impaired subjects with different types (e.g., conductive, neural) and configuration of hearing loss awaits future studies.

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Table 1. Mean and median ABR, MLR, and AME exponents from the normal-hearing subjects to 1000-Hz tones presented in three levels of broadband masking noise. N = 11

	Masking Condition					
	0 dBEM		40 dBEM		60 dBEM	
	Amp	Lat	Amp	Lat	Amp	Lat
Measure						
ABR						
Mean	0.18	0.08	0.28	0.10	0.48	0.16
sd	0.04	0.01	0.11	0.01	0.16	0.04
Median	0.19	0.07	0.26	0.10	0.46	0.15
MLR						
Mean	0.13	0.08	0.26	0.11	0.44	0.17
sd	0.03	0.05	0.08	0.06	0.15	0.12
Median	0.12	0.07	0.25	0.11	0.43	0.15
AME						
Mean	0.29		0.77		1.54	
sd	0.06		0.30		1.29	
Median	0.27		0.74		1.10	

dBEM = Effective Masker level

Table 2. Summary of the proportional increase in slopes (exponents) with increasing masker level for 10 subjects. Each subject's exponent change from Quiet to 60 dBEM was divided by their exponent change from Quiet to 40 dBEM (i.e., 60/Q - 40/Q). (See text).

	ABR		MLR		AME
	Amp	Lat	Amp	Lat	
Mean	1.98	1.50	1.88	1.67	1.65
sd	0.83	0.37	0.68	1.13	0.82
Median	1.73	1.43	1.86	1.26	1.43

Table 3. Mean and median ABR, MLR, and AME exponents from the normal-hearing (N = 11) and hearing-impaired subjects (N = 12) to 1000-Hz tones.

	Normal-Hearing		Hearing-Impaired	
	Amp	Lat	Amp	Lat
Measure				
ABR				
Mean	0.18	0.08	0.38	0.11
sd	0.04	0.01	0.16	0.04
Median	0.19	0.07	0.31	0.11
MLR				
Mean	0.13	0.08	0.29	0.08
sd	0.03	0.05	0.13	0.04
Median	0.12	0.07	0.27	0.02
AME				
Mean	0.29		0.46	
sd	0.06		0.14	
Median	0.27		0.40	

Figure Captions

Figure 1. Representative ABR/MLR recordings to 1000-Hz tones from one normal-hearing subject (Subject # 21) are displayed. Tones were presented alone (0 dBEM, left column), or simultaneously with 40 dBEM (center column) or 60 dBEM (right column) broadband noise. Each waveform is the average of 4000 (≥70 dB nHL) or 8000 (<70 dB nHL) trials. Each column includes data obtained over a range of intensities, indicated to the left of the waveforms, with the subject's corresponding AME scores. The ABR wave V and MLR wave Pa are labeled for a high intensity response in the three conditions.

Figure 2. Representative ABR/MLR recordings to 1000-Hz tones from one normal-hearing subject (Subject # 29) are displayed. Tones were presented alone (0 dBEM, left column), or simultaneously with 40 dBEM (center column) or 60 dBEM (right column) broadband noise. Each waveform is the average of 4000 (≥70 dB nHL) or 8000 (<70 dB nHL) trials. Each column includes data obtained over a range of intensities, indicated to the left of the waveforms, with the subject's corresponding AME scores. ABR wave V and MLR wave Pa are labeled for a high intensity response in the three conditions.

Figures 3. Amplitude, latency, and loudness data from the subject in Figure 1 plotted on log-log coordinates. The individual peak-to-peak amplitudes of the ABR V-V" and MLR Na-Pa (upper panels) are plotted as a function of intensity for the 0 dBEM = Δ, 40 dBEM = O, and 60 dBEM = □ conditions (symbols denote the same condition in each

graph). In the lower panels, the latency reciprocal function is plotted for ABR wave V and MLR wave Pa. In the right panel are the loudness growth functions obtained using the Absolute Magnitude Estimation procedure. Regression analysis (solid line) performed on the values obtained for the ABR, MLR, and AME in each condition yielded exponent values for each function, and this subject's exponents are indicated to the right of the symbol representing each of his functions.

Figure 4. Amplitude, latency, and loudness data from the subject in Figure 2 plotted on log-log coordinates. The individual peak-to-peak amplitudes of the ABR V-V' and MLR Na-Pa (upper panels) are plotted as a function of intensity for the 0 dBEM = Δ, 40 dBEM = ○, and 60 dBEM = □ conditions (symbols denote the same condition in each graph). In the lower panels, the latency reciprocal function is plotted for ABR wave V and MLR wave Pa. In the right panel are the loudness growth functions obtained using the Absolute Magnitude Estimation procedure. Regression analysis (solid line) performed on the values obtained for the ABR, MLR, and AME in each condition yielded exponent values for each function, and this subject's exponents are indicated to the right of the symbol representing each of her functions.

Figure 5. Mean amplitude, latency, and loudness data for 11 normal-hearing subjects are plotted on log-log coordinates. The mean peak-to-peak amplitudes of the ABR wave V-V' and MLR Na-Pa (right panels), wave V and Pa latencies (left panels), and loudness measures (AME, lower panel) are plotted as a function of intensity for the 0 dBEM = Δ, 40 dBEM = ○, and 60 dBEM = □ conditions (symbols denote the same condition in each graph). The solid line denotes a simple linear regression of these values.

Figure 6. Individual values of the amount of exponent increase for the 40 and 60 dBEM conditions relative to the results in the quiet (0 dBEM) for 11 normal-hearing subjects. Each subject's exponents in the 40 and 60 dBEM conditions were normalized by dividing them by their exponent for the 0 dBEM (Quiet) condition. The line indicates the level of "no change" in each subject's slope, relative to their results in the non-masked condition. ■ denotes results of Subject #19, whose results for MLR amplitudes and AME lie well outside those of the group results, and are not included in the mean.

Figure 7. Representative ABR/MLR waveform series from one normal-hearing subject (left panel) and two hearing-impaired subjects (#46 and #50) to1000-Hz tones. Tones were presented to each subject at 8 - to -10 intensities within the each individual subject's dynamic range. Waves V and Pa are indicated for a high-level tone presentation for each subject, and the subjects' loudness estimates are listed to the left of the same intensities as those used to obtain the ABR/MLR.

Figure 8. The data from the three subjects in Figure 7 plotted on log-log coordinates. The individual peak-to-peak amplitudes of the ABR V-V' and MLR Na-Pa (upper panels), wave V and Pa latency reciprocals (lower panels), and behavioral magnitude estimates (AME, right panel) are plotted as a function of intensity. The solid line represents simple linear regression. The individual subject's exponents for each function are indicated to the right of the symbol representing that subject's data.

Figure 9. Individual exponents for 11 normal-hearing and 12 hearing-impaired subjects' loudness (left panel), ABR/MLR amplitudes (upper panels), and latencies (lower panels), plotted as a function of their pure-tone behavioral threshold for 1000-Hz tones.

Figure 10. Mean amplitude, latency, and loudness data for the two subgroups of hearing-impaired subjects (solid symbols) plotted on log-log coordinates for ABR/MLR amplitudes (right panels), latency reciprocals (left panels), and for loudness growth (AME, lower panel). The normal-hearing subjects' results in the non-masked condition are also shown (open symbols).

Figure 11. Individual values of the amount of exponent increase for the sensorineurally-impaired subjects relative to the mean non-masked results of the normal listeners. Each impaired subject's exponent was divided by the non-masked normal-hearing subjects' mean exponent for the 0 dBEM (Quiet) condition for each measure. The line indicates

the level of "no change" in the impaired subject's slope, relative to the normal non-masked listeners' response.

Figure 1

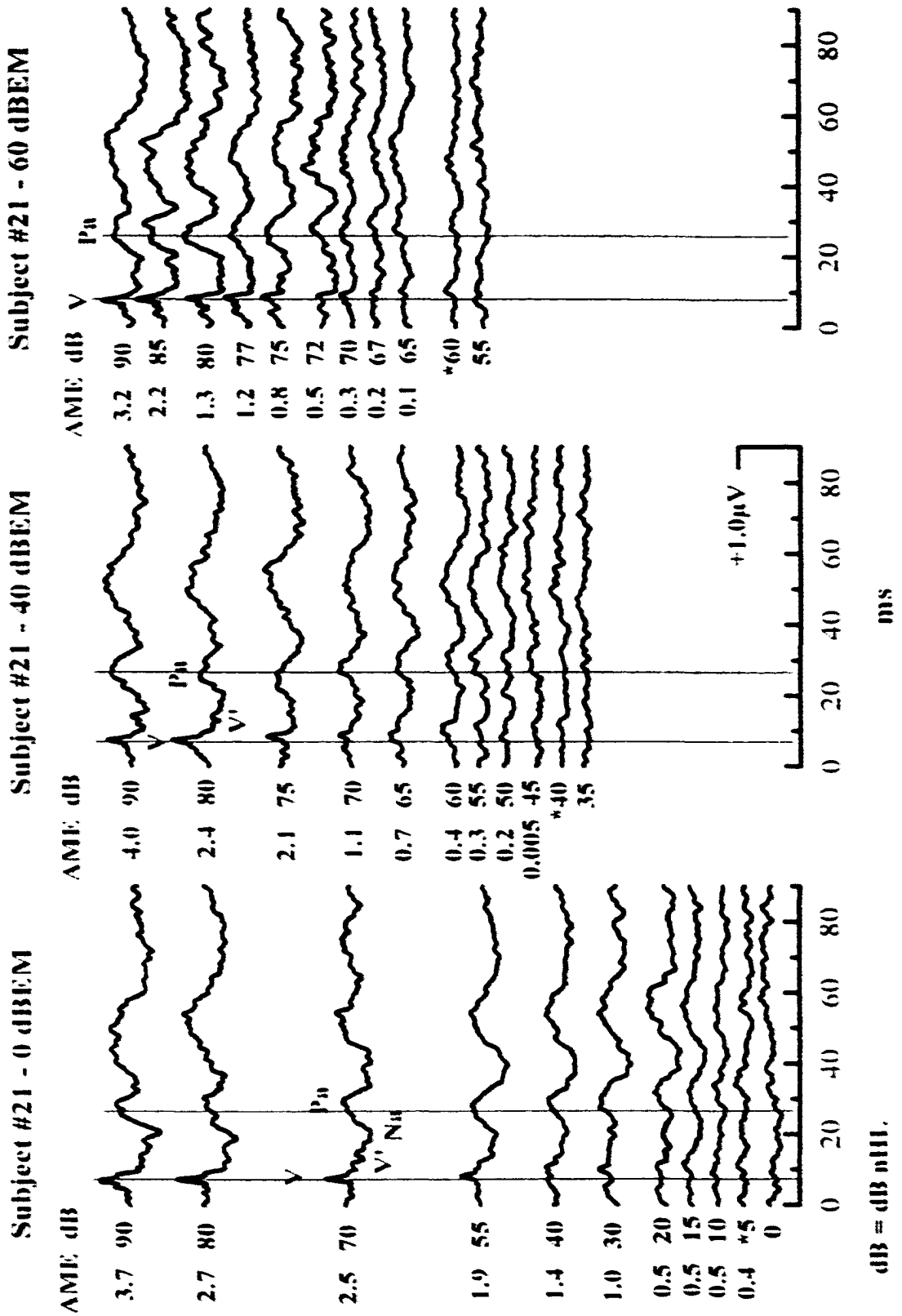


Figure 2

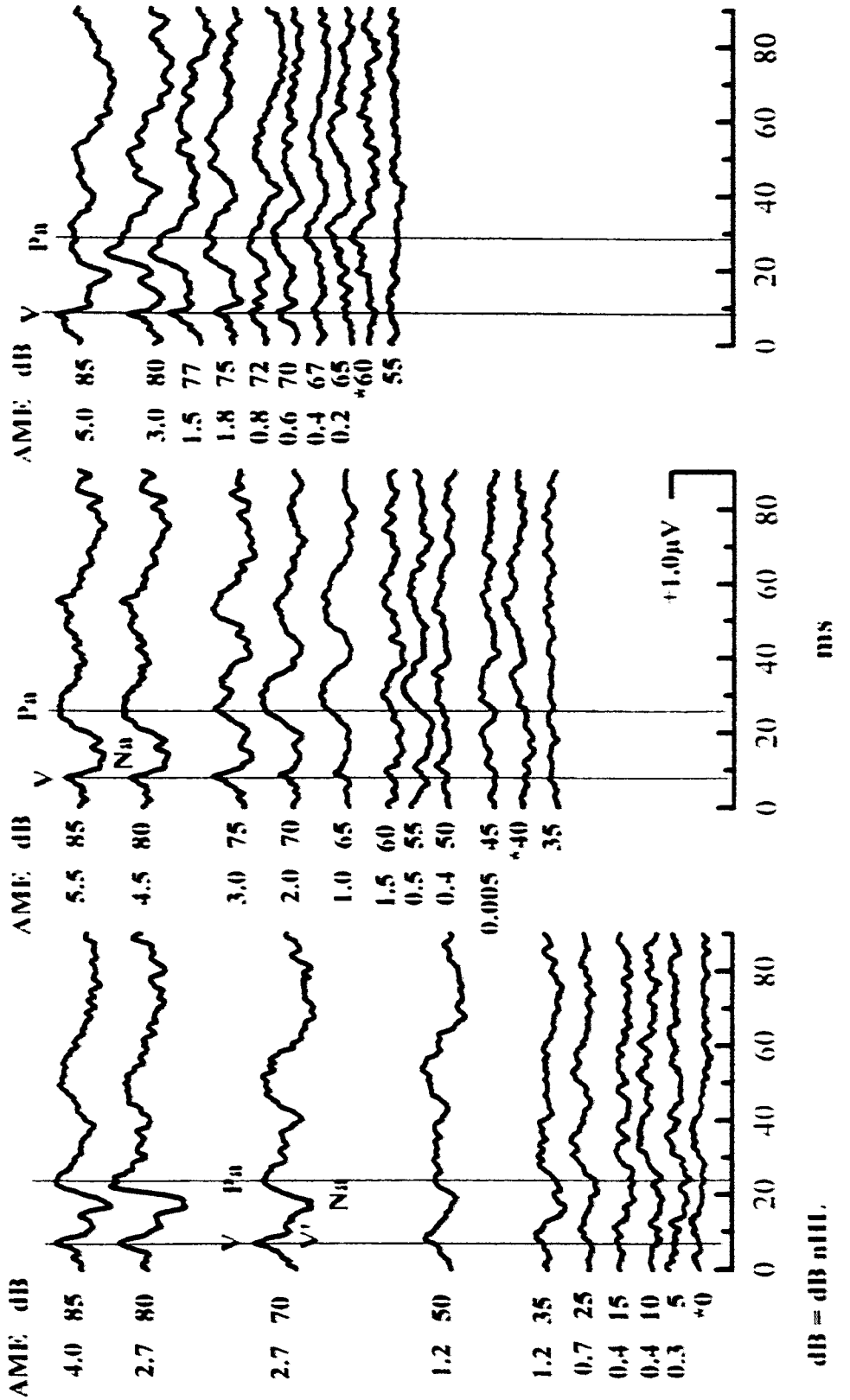


Figure 3

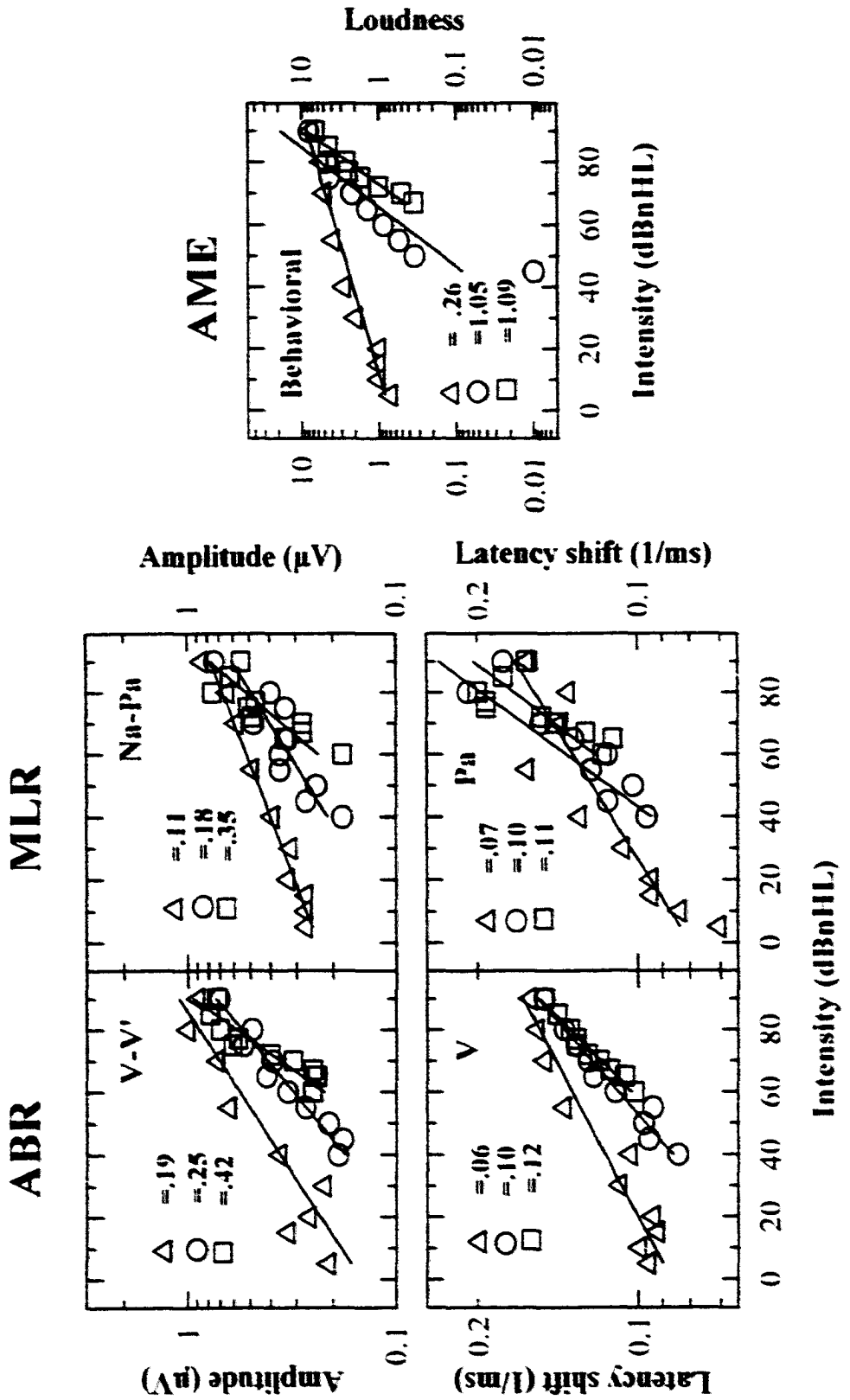


Figure 4

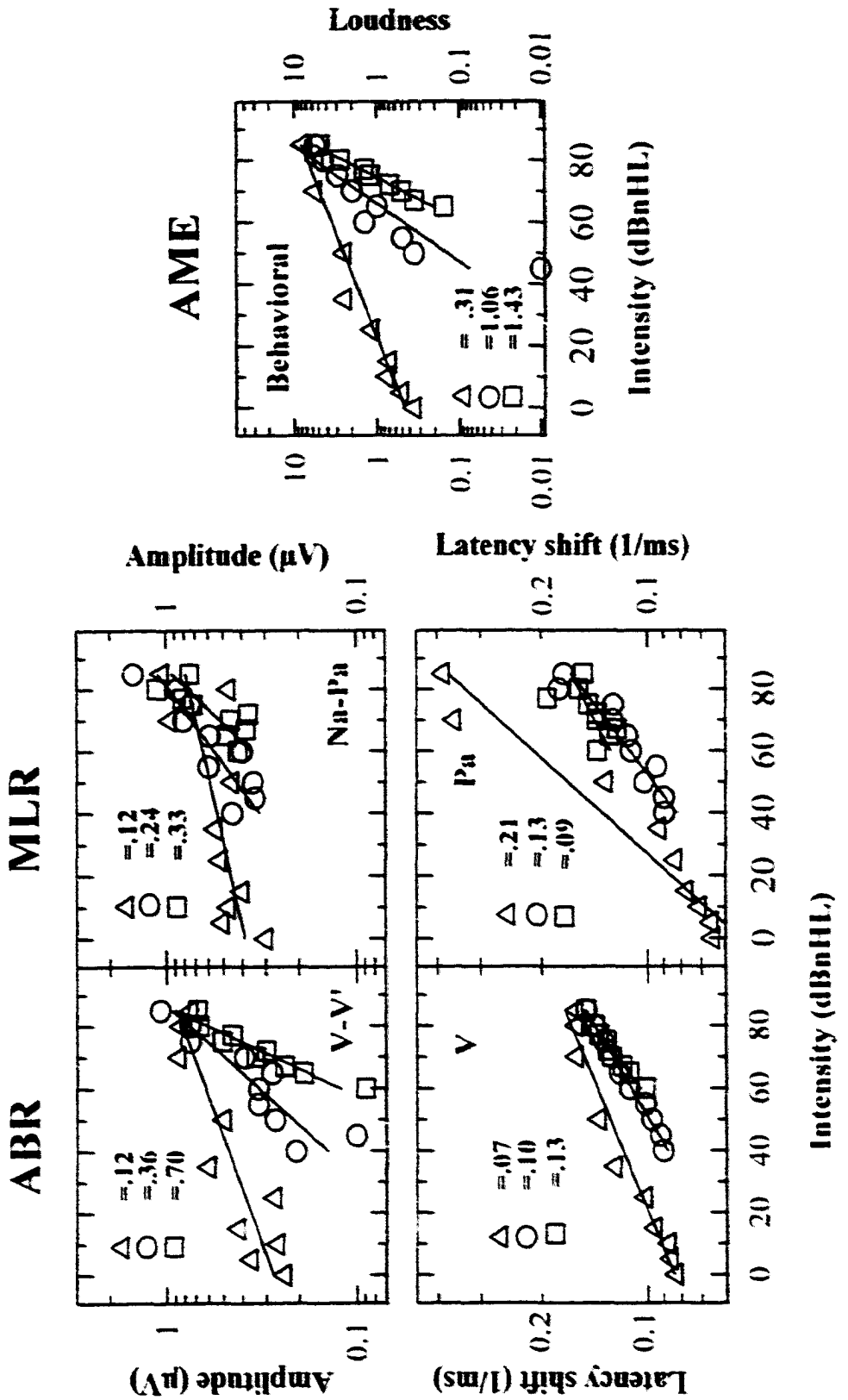
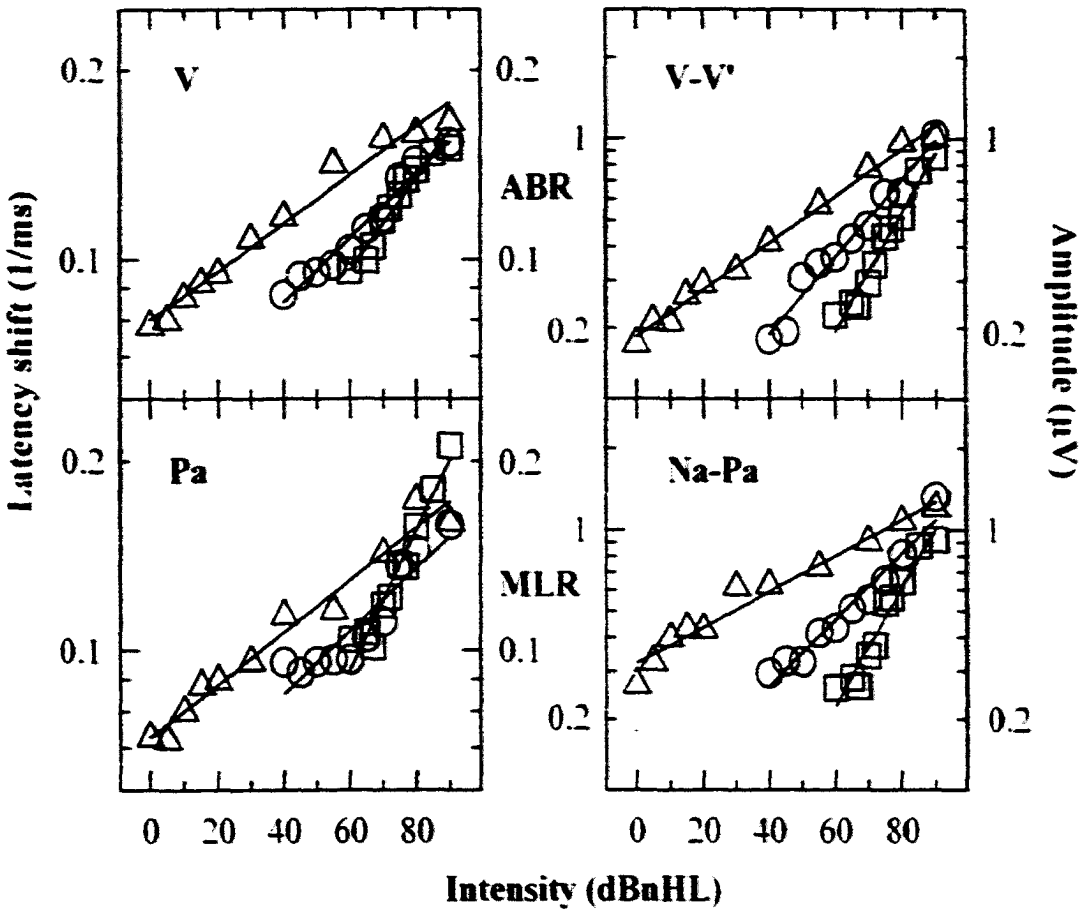


Figure 5



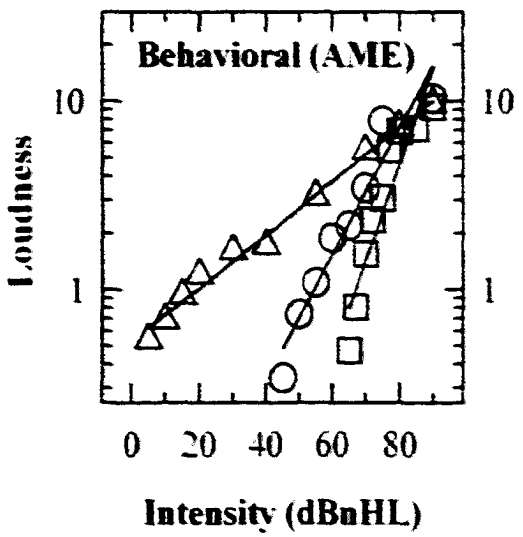


Figure 6

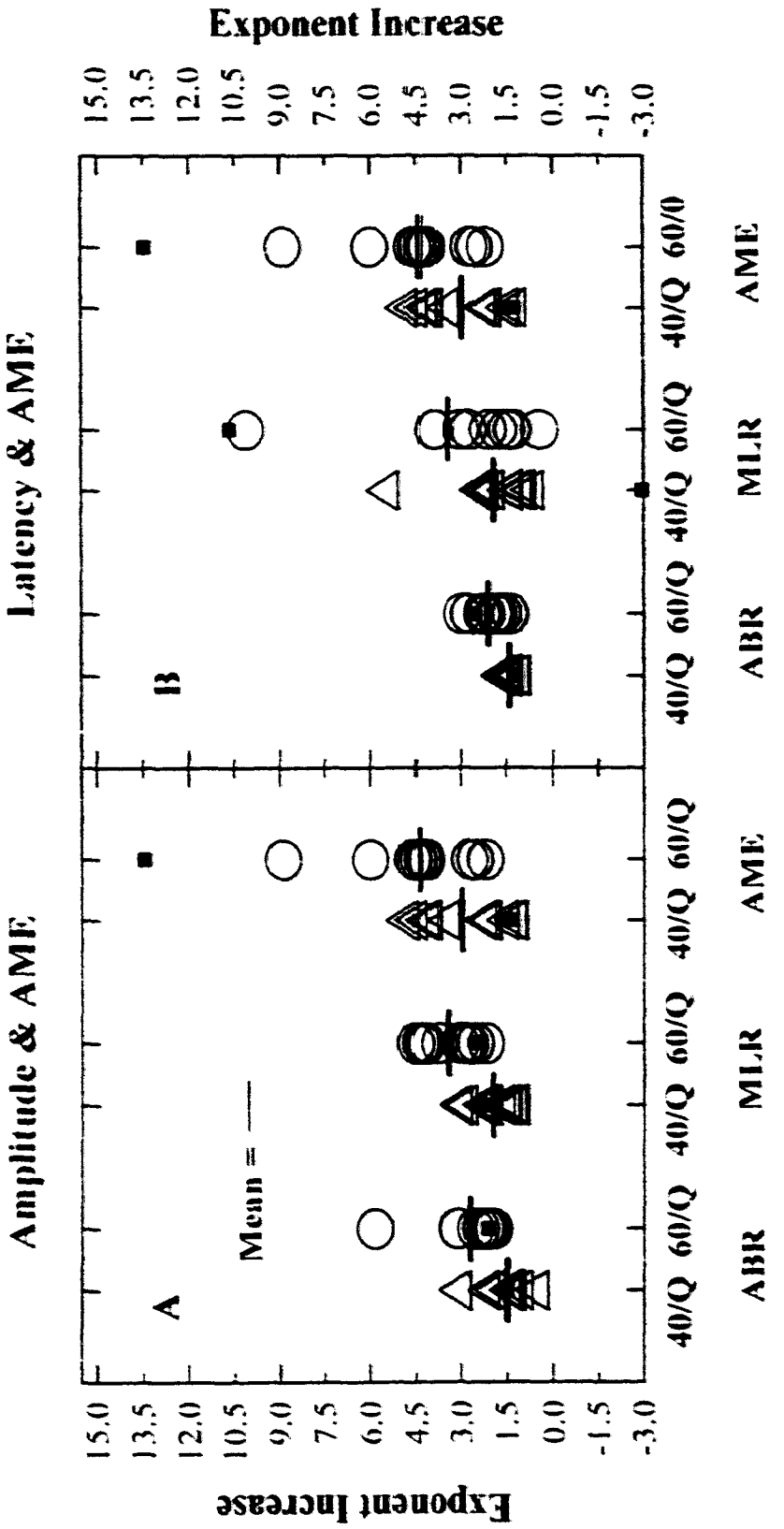


Figure 7

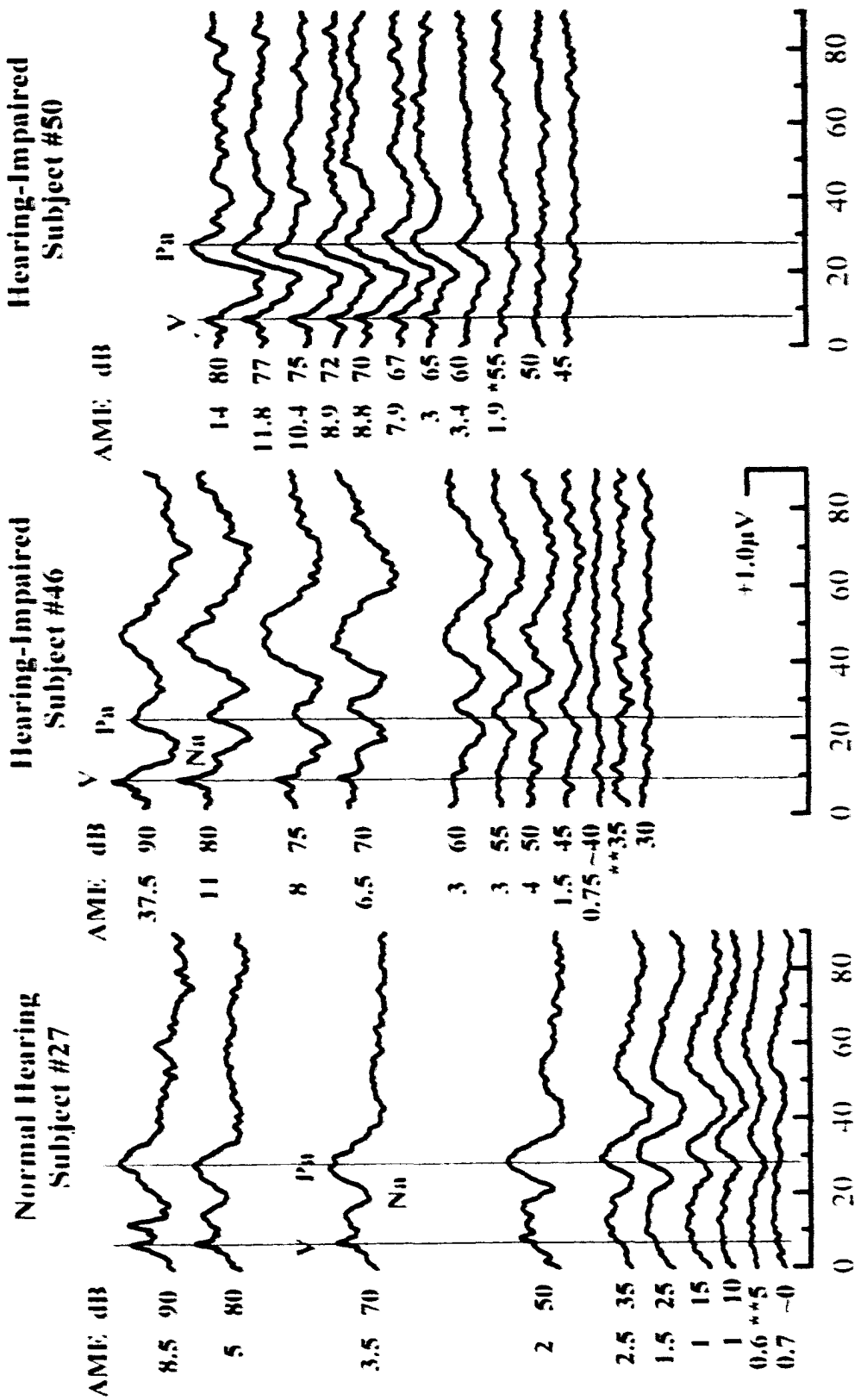


Figure 8

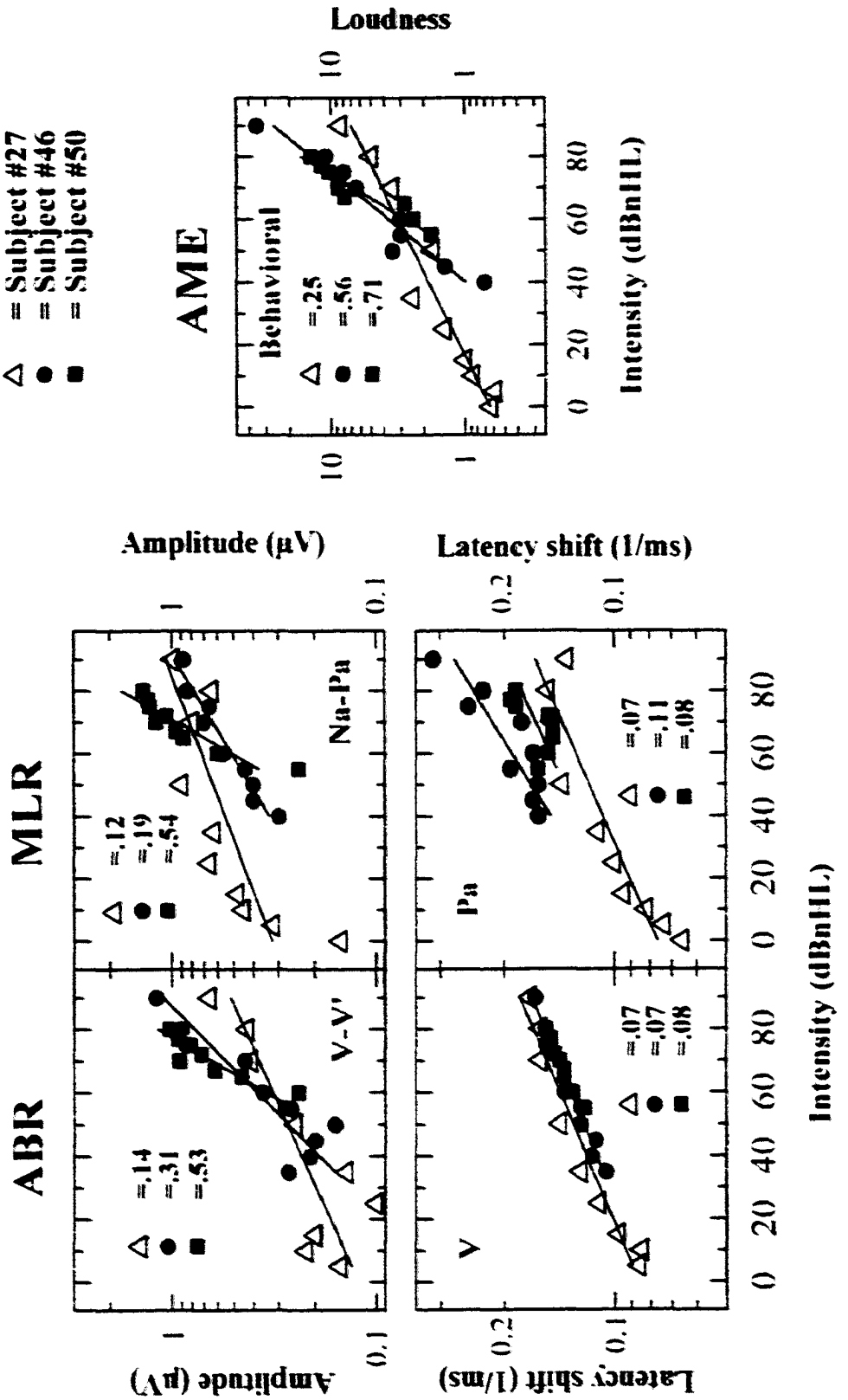


Figure 9

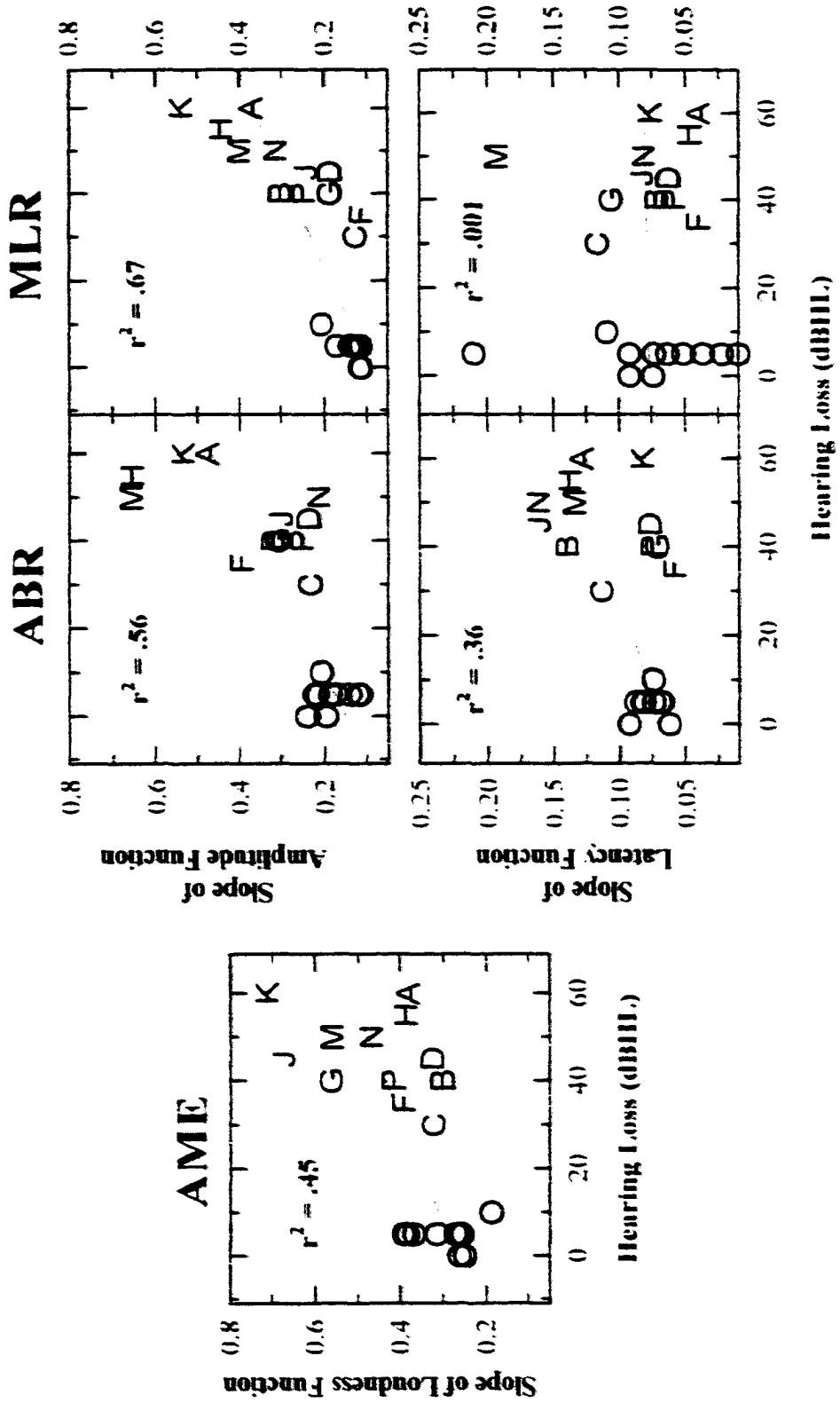


Figure 10

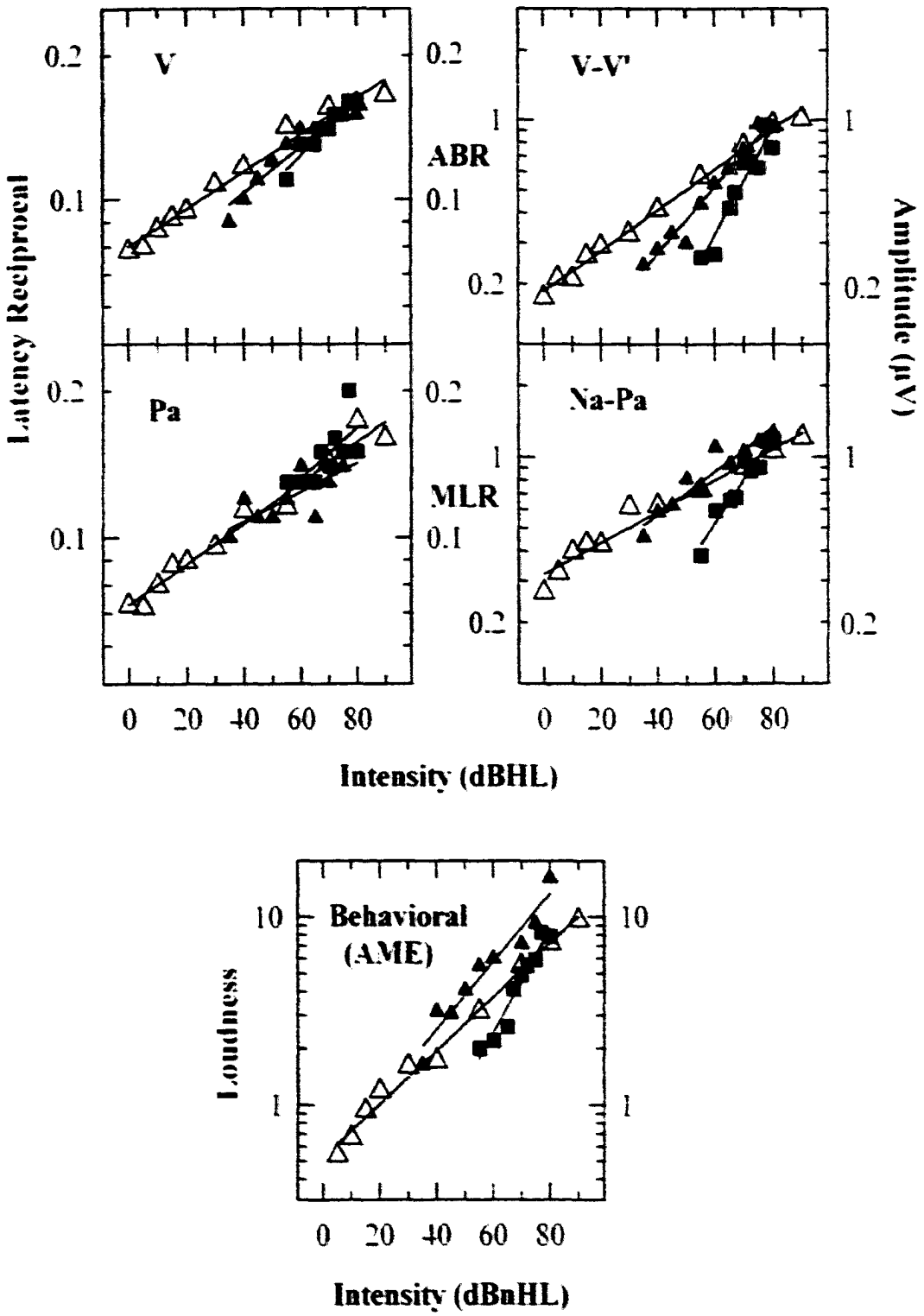
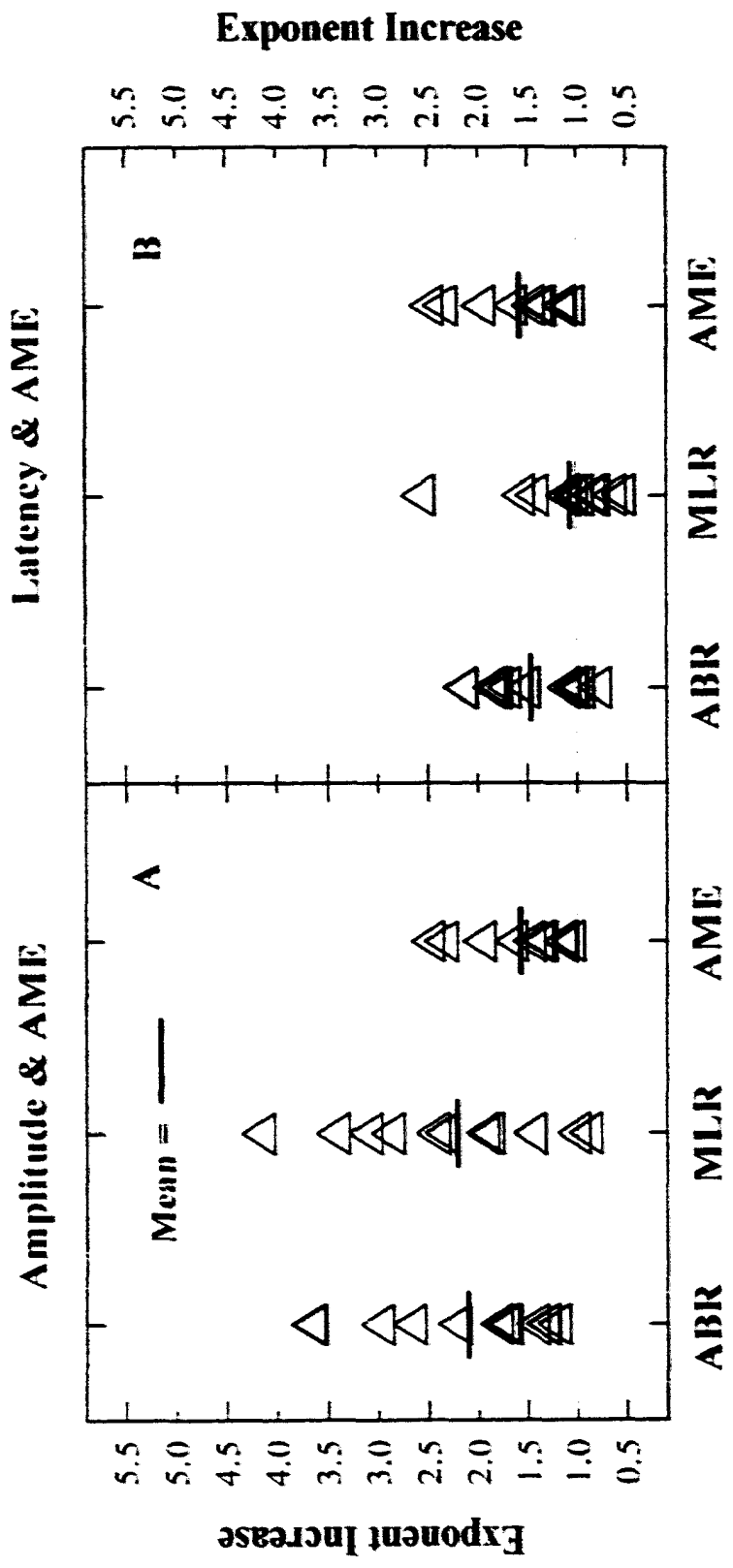


Figure 11



Footnote

1. This subject's AME exponents in the 0, 40 and 60 dBEM conditions were 0.90, 2.46, 2.42, thus his results overall are similar to those of the other subjects. However, because his AME exponent in the quiet condition was so large, it proved to be an outlier from the rest of the subjects' data for this condition. This subject's evoked potential results are consistent with the other subject's results, including the amplitudes and latencies in all three conditions. Although his measures are not included in the statistical analyses described in this paper, they are given along with the other individual data in the appendices.

Chapter 7

Summary and conclusions

Summary

ABR/MLR and behavioral measures of hearing sensitivity

The ABR and MLR to 1000-Hz tones are equally accurate in estimating behavioral hearing threshold in normal-hearing adults and in sensorineurally-impaired listeners with gradually sloping and flat configuration of hearing loss. In addition, behavioral threshold elevations of 40 and 60 dB imposed by a broadband masking noise in the normal listeners are also accurately reflected in their masked evoked potential responses. These results extend the findings of previous studies that reported good agreement between the tone-evoked ABR (e.g., Stapells, 2000b) and MLR (Xu et al., 1995) and behavioral threshold in normal-hearing and hearing-impaired subjects. Further, these results refute the notion that one or the other of these evoked potentials is superior in estimating behavioral threshold to low-frequency tonal stimuli (e.g., Laukli & Mair, 1988; McGee & Kraus, 1996; Musiek & Geurkink, 1981). At least for adults, both are equally good.

ABR/MLR and behavioral measures of loudness growth

Partially-masked tones yields steeper ABR and MLR amplitude and loudness growth functions, and their growth rate increases with increasing threshold shift. The main finding that the *relative* change in the masked electrical responses is not significantly different from that seen in the subjective loudness results indicates that the activity reflected in these evoked potentials derives, at least in part, from the same neuronal sources that subserve psychophysical experiences of loudness growth. ABR and MLR intensity-amplitude and loudness growth functions are steeper, also, for mild-to-moderately hearing-impaired listeners with primarily cochlear loss of hearing sensitivity,

augmenting the noise-masked normal listeners' findings. The overall consistent finding that the pattern of increased rate of growth in the magnitude of these electrophysiological responses in hearing-impaired subjects is similar to that observed in behavioral loudness measures confirms the view held by many researchers that the amplitude of the evoked potentials relates to the subjective percept of stimulus magnitude (Davis & Zerlin, 1966; Eggermont, 1977, 1982b; Davidson, Wall & Goodman, 1990; Gerken, 1991, 1992, 1993; Henderson & Salvi, 1998; Keidel, 1976; Keidel & Spreng, 1965; Kiessling, 1982, 1983; Knight & Beagley, 1969; Madell & Goldstein, 1972; Syka, Rybalko & Popelar, 1994). The correlations between the rate of ABR/MLR amplitude growth and hearing threshold for the sensorineurally-impaired listeners are moderately strong, despite the variability among this small group of subjects that was seen in both their electrophysiological and behavioral measures to increasing stimulus level. These results indicate that the ABR and MLR intensity-amplitude functions are an index of loudness recruitment, and thus might be used to estimate the loudness growth of hearing-impaired patients who are not capable of conveying reliable subjective judgements of loudness.

ABR and MLR intensity-latency functions of the impaired listeners were steeper, on average, compared to the non-masked normal response. ABR intensity-latency functions of the NMNH and SNHL subjects produced the most consistent exponent increase across subjects, and their exponents showed a moderate correlation with hearing threshold. Conversely, MLR intensity-latency functions were the most variable measure obtained from the impaired listeners and, interestingly, showed no correlation with hearing threshold. Indeed, although some of the hearing-impaired subjects had steeper MLR

latency slopes relative to the normal non-masked response, several subjects showed no change or a less- steep slope compared to the normal response. The steeper intensity-latency functions obtained in the impaired listeners are, in the main, likely due to peripheral effects of the noise masker/cochlear dysfunction and to configuration of hearing loss, and these effects are seen to a greater extent in the more caudal (ABR) response. At the early cortical level, MLR latencies show some peripheral effects of the masker/cochlear dysfunction, but the variability and/or lack of a consistent effect in these findings are likely due to central mechanisms of the effects of noise and cochlear dysfunction on signal level processing, including alteration and/or enhancement of the confluence of excitatory and inhibitory input at this higher loci of the auditory pathway (Bilak et al., 1997; Heil et al., 1994; Salvi et al., 2000; Willot et al., 1994). They also are attributed, in part, to the fact that the MLR reflects predominantly low-frequency post-synaptic activity resulting in less precision in peak timing. These differences between the ABR and MLR latency results suggest that this parameter of the evoked potentials is likely not an index of loudness (e.g., Eggermont, 1982b; Howe & Decker, 1984).

Implications

There are implications of the present studies in terms of brain processing of sound intensity as well as clinical application of these auditory evoked potentials.

Brain processing of sound intensity. Both the brainstem and early cortical auditory evoked potentials to tonal stimuli yield accurate and similar estimates of hearing sensitivity in normal-hearing and hearing-impaired adults. These findings indicate that threshold of audibility may be successfully predicted from the average of evoked potential

onset responses derived from generators in the peripheral and central auditory system pathways.

For all subjects in all conditions, ABR and MLR amplitudes increase and their latencies decrease as signal level increases. The changes in these evoked potential characteristics are the same for both the brainstem responses and early cortical MLRs in normal-hearing subjects. That is, as stimulus intensity increases from 0 to 90 dB nHL, both the ABR and MLR latencies decrease by about 7 ms, their amplitudes increase by about 1.0 μV, and the rate of change in these response characteristics with increasing signal level is about the same for these evoked potentials in normal listeners.

In response to supra threshold stimuli, the effects of the masker and cochlear loss of sensitivity on the ABR and MLR reveal some similarities as well as differences between (a) these two groups of impaired listeners, and (b) the characteristics of the ABR and MLR. The peripheral effects of the masking noise and cochlear pathology are evident in the ABR, with findings showing similar qualitative response measures for both groups of subjects. That is, ABR latencies are prolonged and amplitudes are reduced at and just above threshold relative to the normal non-masked response, reflecting a reduction in cochlear output as a result of the competing noise or cochlear damage. These effects are greater for the masked normal listeners than for the cochlear-impaired subjects. The near-normal amplitudes and latencies seen in the ABR recordings to high stimulus intensities in both groups of impaired subjects is consistent with the recruitment phenomenon, which was verified when the slopes of the ABR intensity-amplitude/latency functions were computed and found to be steeper than the normal listeners' response in the quiet, similar

to the psychophysical measures which also evidenced steeper loudness slopes for these impaired subjects.

With respect to intensity coding and recruitment, the evoked brainstem potential results, which reflect activity from the VIIIth nerve and brainstem structures up to the inferior colliculus (Møller, 1994), are consistent with hypotheses that suggest primarily peripheral (i.e., cochlear) genesis for intensity coding (e.g., Cheatham & Dallos, 2000; Viemeister, 1988; Yates et al., 1990) and loudness recruitment (Chatterjee & Zwislocki, 1997, 1998; Dolan & Nuttall, 1989; Doucet & Relkin, 1997; Phillips, 1987; Phillips & Carr, 1998; Relkin & Doucet, 1997; Zhang & Zwislocki, 1995).

Both peripheral and central effects of masking and cochlear pathology are evident in the MLR. Masked-MLR amplitudes are reduced relative to the non-masked normal response, and their rate of growth increases with increasing threshold shift, similar to the findings obtained for the masked ABR and loudness growth. The masked-MLR latencies are also prolonged, but to a lesser extent than that observed in the brainstem response, and the increase in these latency slopes are more variable than the ABR. These results likely reflect reduced effects of the masker at this higher level of the auditory system due to inhibitory and adaptive mechanisms that are not present in VIIIth nerve responses (Phillips,1987), providing a large number of activated neurons excited by supra threshold masked tones, as well as the confluence of source activity at this higher loci that is not available to primary auditory neurons (Heil, 1997a,b; Lockwood et al., 1999; Phillips & Burkard, 1999), and they are consistent with reports of response enhancement (or reduced effects of a noise masker) on the masked gross evoked potential at the level of the inferior

colliculus and auditory cortex in animals (e.g., Gerken, 1991; Gumnit & Grossman, 1961; Phillips, 1990). Response enhancement of signal intensity processing at these more rostral levels of the auditory pathway is seen also in the results of the cochlear-impaired listeners. These subjects' MLR amplitudes were the same or *larger* than the normal listeners' responses, non-masked or masked, and also their absolute latencies were the same or *shorter* than the normal listeners' results in the quiet and in noise, in agreement with single-unit and gross evoked potential studies which have also shown increases in unit activity and/or amplitude response at the level of the inferior colliculus and auditory cortex in animals with peripheral hair cell loss/damage (e.g., Gerken, 1991; Henderson & Salvi, 1998; Qui et al., 2000; Salvi et al., 1992, 2000; Syka & Rybalko, 2000; Wang et al., 1997), and with reports of decreased impulse response times and shorter cochlear response times in cochlear-impaired subjects (Don, Ponton, Eggermont & Kwong, 1998). As well, the slopes of the MLR intensity-amplitude functions steepened for both groups of impaired listeners, consistent with recruitment. To some extent, then, these results support the idea advanced by Gerken (1993) that the MLR in noise-masked normal-hearing and cochlear-impaired subjects likely reflects similar central mechanisms of signal enhancement at this higher level of the auditory system pathway.

Although it has been suggested that the underlying neurophysiologic mechanisms that give rise to recruitment seen in noise-masked normal listeners is a purely central phenomenon whereas recruitment in cochlear-impaired listeners derives solely from peripheral effects of cochlear disease or trauma (Phillips, 1987; Phillips & Carr, 1998), it appears from the composite ABR and MLR results of the present studies that both

peripheral and central mechanisms play a role in the more rapid rate of amplitude and loudness growth seen in both noise-masked normal-hearing and cochlear-impaired subjects (Henderson & Salvi, 1998; Komiya & Eggermont, 2000; Oxenham & Plack, 1997; Ruggero et al., 1992; Zhang & Zwislocki, 1995; Zwicker & Fastl, 1999).

Clinical implications. The results of these dissertation studies have a number of implications for both basic and applied purposes in the study and assessment of patient populations. First, simultaneous recording of the ABR and MLR to low-frequency tones yielded equally accurate predictions of hearing threshold in normal and impaired listeners, indicating that one or both of these evoked potentials may be successfully applied clinically for estimating hearing sensitivity in patients who are unable to respond behaviorally to pure-tone stimuli for audiological assessment. The finding that there is no significant difference between the ABR and MLR in their accuracy of behavioral threshold prediction for 1000-Hz tones extends the results of a small number of previous studies that used the same comparative recording technique and showed, also, that the tone-evoked ABR and MLR are both effective in yielding accurate estimates of hearing sensitivity in adult populations (Suzuki et al., 1981; Wu & Stapells, submitted). Technological advances now allow for simultaneous recording of the transient ABR/MLR which has been recommended for routine clinical use (Hood, 1995; Kraus & McGee, 1990), providing measures of auditory system integrity at both the brainstem and early cortical levels. As well, the recording parameters for obtaining frequency-specific threshold estimates are the same for the ABR and MLR with the exception of a longer analysis time required for the MLR.

Second, the ABR and MLR results with increasing intensity in these normal-hearing and hearing-impaired subjects provide useful information regarding differential diagnosis of hearing loss which may also be successfully applied clinically, although these preliminary findings suggest the need for further study. ABR and MLR amplitudes grow and their latencies decrease in a similar manner for normal-hearing listeners. Conversely, the recordings of the cochlear-impaired subjects revealed differences between their ABR and MLR amplitudes and latencies, relative to the normal response. That is, in contrast to the ABR which showed prolonged latencies and smaller amplitudes relative to the normal response, supra threshold MLR absolute latencies and amplitudes in mildly-to-moderately impaired adults with primarily cochlear dysfunction are the same as or *shorter and larger than* the normal response. Therefore, both the ABR/MLR latency decrease *and* amplitude increase with increasing stimulus level potentially provide a better indication of type of hearing loss than either characteristic alone These differences between the impaired subjects' brainstem and early cortical responses to supra threshold tones, relative to the normal response, lend further support to the recommended use of a simultaneous recording technique, such that the combined ABR and MLR results may serve the practical purpose of differentiating type of hearing loss. Further studies examining how the characteristics of the ABR and MLR are affected by conductive and mixed hearing loss of similar degree and/or configuration of hearing loss are needed before the ABR/MLR amplitude and latency changes to increasing signal level observed in the present studies may be meaningfully applied in the clinical setting.

The findings reported in Chapter 5 also showed that, in response to supra threshold tones, ABR and MLR absolute latencies were more prolonged and their amplitudes were more reduced for the noise-masked listeners than for the cochlear-impaired subjects relative to the normal response. These differences between the noise-masked and cochlear-impaired listeners' ABR and MLR results have important research and clinical implications. For example, recent studies of the late cortical responses in noise-masked normal-hearing listeners have reported increases in the latencies and decreases in the amplitudes of the N1, P2, and P3 with increasing masker level (Martin, 1997; Whiting et al., 1998). These authors suggest that their findings may be applicable to patient populations with similar degree and/or configuration of hearing loss for the study of speech perception in hearing-impaired populations (Whiting et al., 1998). The results from the present studies, however, indicate that this may only be true only for threshold measures. Future research of these later evoked potentials, which are obtained to stimuli presented at supra threshold levels (Martin, 1997), require that these waves be examined in groups of subjects with various types, degrees, and configurations of hearing loss. The MMN, for example, has been suggested as a potentially useful objective measure of speech perception/discriminatory processes in impaired listeners, including the effectiveness of initial hearing aid fitting as well as a means of monitoring changes in discrimination ability with amplification and therapy (Martin, 1997). The adult subjects in these dissertation studies presenting with mild-to-moderate sensorineural hearing loss showed normal or larger MLR amplitudes at lower sensation levels (re: threshold) than the normal listeners, and their rate of amplitude growth is consistent with behavioral

results indicating recruitment. This being so, investigators examining the later evoked potentials in this patient population will need to present stimuli at reduced sensation levels (i.e., within these subjects' reduced dynamic range) relative to that generally used for normal listeners (e.g., Nousak et al., 1996) in order to obtain estimates of their speech perception and discriminative abilities, and ensure that stimulus presentation levels do not exceed their loudness discomfort level. This latter point is especially so for measures obtained with amplification, including assessment of the electrical evoked potentials from infants and older subjects who are fitted with cochlear implants (e.g., Ponton & Don, 1995).

The main finding of the present dissertation studies that the ABR and MLR reflect, in part, neuronal activity involved in loudness perception also has a number of important basic and applied implications for study of loudness growth/intensity coding in normal-hearing and hearing-impaired populations. For example, ABR and/or MLR amplitude growth may be investigated in the pediatric population in order to assess developmental issues of intensity coding/loudness growth in these young listeners for whom behavioral loudness data are unavailable (Launer, 1998). Several researchers have successfully applied the ABR and MLR to the study of maturational issues in both normal-hearing (e.g., ABR: Eggermont, Ponton, Coupland & Winkelaar, 1991; Sininger & Abdala, 1996; Werner, Folsom & Mancl, 1993; MLR: Cone-Wesson et al., 1997; Kraus et al., 1985; Suzuki & Hirabayashi, 1987; Suzuki et al., 1984b; and see McGee & Kraus, 1996 for review) and hearing-impaired subjects (e.g., ABR: Ponton & Don, 1995), and these results augment known anatomical and physiological findings as well as psychophysical

results of auditory system development (Werner, 1996). Further, development of normative ABR/MLR intensity-amplitude data in both adult and pediatric listeners would provide valuable predictive measures of loudness growth that may then be applied clinically. Although considerable inter-subject variability was found in the present studies, there were strong correlations between the growth in ABR/MLR amplitudes and hearing loss. It is reasonable to suggest, then, that normative tone-evoked ABR/MLR intensity-amplitude/latency values may be derived from measures of larger groups of normal and impaired populations with various degrees and types of hearing loss for clinical purposes. This same objective has been met for behavioral measures of loudness growth (Killion, 1996; Schum, 2000), such that predictive loudness growth functions obtained from large groups of impaired listeners are presently used, both for developing algorithms for hearing aid circuitry (Schum, 2000), and also as a clinical tool for the initial selection of appropriate hearing aid parameters (Allen et al., 1990). Probably more important, however, is that ABR/MLR intensity-amplitude functions are an index of loudness growth in hearing-impaired subjects, and thus may be used in the selection and verification procedures employed clinically to ensure adequate and comfortable amplification to patient populations who are unable to participate in behavioral audiometric tasks.

Future studies

The present studies are the first to demonstrate that the neural sources contributing to loudness growth are reflected also in tone-evoked ABR and MLR recordings. As well, these dissertation studies provide useful information about intensity coding at the

brainstem and early cortical levels of the auditory system pathway in both normal and impaired listeners, including measures of hearing threshold and the electrophysiological response to increasing signal level. This work leads to potential investigations in the following areas: (1) Examination of supra threshold auditory capabilities in infants, including developmental changes, especially with regard to rate of growth in the characteristics of these evoked potentials in normal-hearing and hearing-impaired infants, children, and adults; (2) Study of the rate of growth in the neural responses to other frequency tones in subjects with various degrees, configuration, and types of hearing loss; (3) Further comparative examination of ABR/MLR/CAEP measures, especially as they may relate to recent findings re: re-organization of cortical activity in early onset hearing loss; and (4) Examination of neural changes produced by transient (e.g., otitis media) and progressive hearing loss.

Appendices

Appendix A

Sound Comfort

Imagine you are about to obtain a hearing aid. We need to do a test to determine where to set the amplifier on your hearing aid. We want to set it such that sounds do not become uncomfortable for you. If we set it too high, sounds could get uncomfortable, and you may not want to wear the hearing aid. You will hear some sounds through this earphone, and, after each one, I want you to tell me which of the loudness categories on this sheet best describes the sound to you. So after each sound, tell me if it was "comfortable", or "comfortable, but slightly loud" etc. I will be zeroing in on the uncomfortably loud category first because that is where we want the hearing aid to stop. Think of uncomfortably loud as where you would want the hearing aid to stop and not get any louder. I am not looking for a level of extreme discomfort or pain, but rather where the sound first reaches a level of discomfort. We want the haring aid to keep sounds in the comfortable regions and not let sounds get up into the uncomfortable regions. So after each sound, tell me which category best described it to you. Do you have any questions?

Loudness Categories

9. Uncomfortably loud
8. Very loud
7. Loud, but O. K.
6. Comfortable, but slightly loud
5. Comfortable
4. Comfortable, but slightly soft
3. Soft
2. Very soft
1. Nothing

(From Bentler & Pavlovic, 1990)

Appendix B

Absolute Magnitude Estimation

You are going to hear repeated tone bursts of different intensities in random order. Your task is tell me how loud they are by assigning numbers to them. You may use any positive numbers that appear appropriate to you – whole numbers, decimals, or fractions. Do not worry about running out of numbers, there will always be a smaller number than the smallest you use and a larger one than the largest you use. Concentrate on each burst sequence individually and don't be concerned with the numbers you use in the previous sequence. Simply try to match an appropriate number to each tone burst presentation regardless of what you may have called the previous stimulus. You may listen to the same tone burst sequence as often as you wish before deciding on your numerical estimate of its loudness. However, it is best to be as spontaneous and quick in your response as possible. After you have reached a decision, report your judgement to the experimenter through the intercom. Do you have any questions?

(from Hellman & Meiselman, 1988)

Appendix C - Chapter 5

Behavioral thresholds for long-duration (LD, dBHL) and short-duration (SD, dB nHL) 1000-Hz tones, and ABR/MLR thresholds (dB nHL) to 1000-Hz tones from the normal-hearing (N=12) and sensorineurally hearing-impaired (N=12) subjects.

Normal-hearing					Hearing-impaired				
S#	LD	SD	ABR	MLR	S#	LD	SD	ABR	MLR
11	5	5	0	5	41	60	55	60	65
12	0	0	0	0	42	40	30	35	30
13	5	5	15	15	43	30	25	30	25
14	5	0	0	0	44	45	30	30	35
16	5	0	10	10	45	35	35	45	45
17	-10	-10	-5	0	46	40	35	35	40
18	10	0	5	5	47	55	50	55	55
19	5	0	5	5	48	45	40	35	40
21	0	0	5	5	50	60	50	55	55
27	5	-5	5	0	51	50	50	55	55
28	5	0	0	5	52	50	40	50	50
29	5	-5	0	0	55	40	30	35	35
Mean	3.33	-0.83	3.33	4.17	Mean	45.83	39.17	43.33	44.17
sd	4.92	4.17	5.37	4.69	sd	9.49	9.96	11.15	12.03

Appendix D - Chapter 5

Behavioral thresholds for short-duration tones (SD, dB nHL) and ABR/MLR thresholds (dB nHL) from 12 normal-hearing subjects to 1000-Hz tones presented in two levels (40 and 60 dBEM) of broadband masking noise.

	40 dBEM				60 dBEM		
S#	SD	ABR	MLR	S#	SD	ABR	MLR
11	40	45	45	11	60	65	65
12	40	35	40	12	60	55	55
13	40	40	40	13	60	60	60
14	40	45	45	14	60	60	67
16	40	40	50	16	60	55	60
17	40	40	45	17	60	55	62
18	40	40	45	18	60	60	50
19	40	35	35	19	60	55	55
21	40	40	40	21	60	60	60
27	40	45	45	27	60	65	65
28	40	40	30	28	60	60	60
29	40	40	40	29	60	60	60
Mean	40	40.42	41.67	Mean	60	59.17	59.92
sd	0	3.34	5.37	sd	0	3.59	4.80

Appendix E - Chapter 5

ABR wave V latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones (0 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	5.9		5.9	5.9			6.3	6.7	6.3	5.9	5.9	
85		5.9			6.3							6.3
80	6.5	5.9	6.3	6.1	6.5	6.5	6.5	6.9	6.5	6.3	6.1	6.3
70	6.7	5.9	6.3	6.1	6.5	6.7	6.9	6.9	6.7	6.3	6.5	6.3
60			6.7									
55	7.1	6.7		6.7	6.9	7.3	7.3	7.5	7.3		7.1	
50										7.1		7.3
45			7.5			7.9						
40		7.9		7.9	7.9		8.5	8.5	9.7		10.3	
35	11.9		8.5							8.1		8.1
30		8.5		9.3	8.7	9.1	9.7	9.5	9.3		10.7	
25	9.7		9.3							9.1		9.9
20	12.1	9.1	9.9	10.1	9.7	10.5	11.1	11.1	10.7		12.3	
15	10.9	9.7	10.5	10.7	11.1		11.9	12.7	10.9	10.3	11.7	10.5
10	11.5	13.7		10.7	10.9	11.3	12.1	14.7	10.1	11.9	10.1	11.5
5		14.1		14.1		12.1	11.6	13.3	10.5	11.7	15.3	11.7
0	13.7	13.7		14.5		12.3					11.1	12.1
-5						12.3						

Appendix F - Chapter 5

ABR wave V latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones presented in 40 dB effective masking noise (40 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	6.3		5.9	6.5			7.1	7.1	6.7	6.5	6.5	
85		6.1			6.5							6.7
80	7.1	6.5	6.5	6.7	6.9	7.3	7.5	7.7	7.3	6.9	6.9	7.1
75	7.1	6.7	7.1	7.1	8.1	7.5	7.9	8.1	7.7	7.3	7.7	7.5
72						7.7						
70	7.7	7.3	7.5	7.7	11.1	8.1	10.9	10.9	8.1	8.1	10.9	7.9
67						8.3						
65	7.5	7.7	8.1	8.5	10.9	8.7	11.3	12.1	8.3	8.3	10.7	8.3
60	10.3	8.7	8.7	9.3	10.5	9.1	11.3	11.9	9.1	9.1	10.5	8.9
55	11.1	9.1	9.1	9.5	10.7	9.7	11.5	11.9	10.7	10.7	9.9	9.9
50	10.7	9.5	9.3	10.5	11.1	10.5	11.5	12.3	10.3	9.9	10.9	10.3
45	11.7	9.5	9.7	11.1	10.3	10.3	11.5	12.7	10.5	9.9		10.9
40		10.1	10.3		11.1	12.1	12.5	12.5	11.9		11.7	11.1
35		10.9						12.5				

Appendix G - Chapter 5

ABR wave V latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones presented in 60 dB effective masking noise (60 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	6.5		6.5	6.5			7.1	7.1	6.7	6.5	6.7	
85	6.3	6.1	6.9	6.5	7.1		7.3	7.5	7.1	6.7	6.7	6.7
80	6.7	6.5	7.3	6.9	7.5	7.3	7.9	8.5	7.5	7.1	7.1	7.1
77	7.1	6.9	7.3	7.3	7.9	7.3	7.9	8.7	7.7	7.5	7.3	7.3
75	7.5	7.1	7.9	7.3	8.5	7.7	8.3	12.1	7.7	7.5	7.7	7.7
72	7.5	7.3	8.5	7.5	8.7	8.1	11.3	12.9	8.1	7.7	7.5	7.7
70	8.3	7.7	7.5	7.9	9.7	8.1	11.7	12.3	8.5	11.5	6.9	7.9
67	10.7	7.9	9.1	8.5	9.1	8.5	12.1	12.7	8.9		12.1	8.5
65	10.7	8.1	9.3	8.5	10.3	8.7	11.7	13.3	9.5	11.9	13.1	8.9
62		8.9				9.1						
60		9.9	9.7	9.9	11.3	10.1	12.3	11.7	9.9		10.7	9.9
55		10.1			10.9	13.7		16.5				

Appendix H - Chapter 5

MLR wave Pa latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones (0 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	25.7		25.3	25.9			25.7	37.5	28.1	26.9	29.7	
85		25.9			29.3							23.3
80	29.1	25.3	28.3	25.1	29.5	26.7	25.7	37.7	27.3	28.1	30.7	22.9
70	31.7	27.1	28.9	27.5	30.5	29.3	26.7	38.3	27.5	27.9	29.9	23.5
60			28.9									
55	31.5	26.9		28.3	30.3	29.3	29.1	39.1		26.9	30.9	
50									27.9			28.3
45	33.1		28.7			30.3						
40		27.9		27.3	29.7		29.7	38.9		28.5	30.9	
35	33.3		28.7						29.7			31.5
30		28.9		30.3	30.9	31.1	30.9	39.5		30.1	32.1	
25	33.3		29.3						30.7			32.7
20	33.1	29.7	29.5	31.1	31.7	32.1	31.9	40.1		31.3	33.5	
15	32.5	30.7	30.5	30.9	31.7		32.3	39.5	31.3	31.3	32.5	33.7
10	31.7	32.9			30.7	33.7	33.9	39.3	32.9	32.7	32.7	34.9
5	33.7			32.9		33.9	35.1	37.5	34.3	34.9	34.3	35.9
0		32.5		33.1		35.7			35.9			36.1

Appendix I - Chapter 5

MLR wave Pa latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones presented in 40 dB effective masking noise (40 dBEM).

S# / dB nHL	11	12	13	14	16	17	18	19	21	27	28	29
90	28.7		24.7	27.7			24.5	34.5	29.1	26.3	29.5	
85		24.9			29.5							26.5
80	30.7	28.5	25.9	29.5	29.1	32.9	25.3	35.5	27.1	25.5	26.7	26.3
75	29.9	29.5	26.5	29.7	29.1	32.3	30.3	35.9	27.9	23.7	26.9	28.7
72						34.1						
70	30.5	30.5	27.3	29.7	31.3	33.7	30.7	35.3	29.9	27.3	27.9	28.7
67						32.3						
65	30.7	30.5	26.9	30.7	31.7	32.5	32.3	36.1	30.3	28.3	29.5	29.5
60	30.9	32.1	28.5	30.9	32.9	32.7	31.1	36.1	31.3	29.5	30.7	29.7
55	31.1	30.5	28.1	30.5	33.3	33.1	32.5	34.3	32.7	28.9	30.5	31.3
50	31.3	31.7	27.9	-0.9	33.3	33.1	31.7	33.5	31.1	30.5	30.7	30.5
45	33.5	32.7	28.3	32.3		33.1	32.1	33.3	31.9	29.5	31.5	31.9
40		30.7	28.7					34.3		31.1	31.9	31.9
35								33.1			31.5	

Appendix J - Chapter 5

MLR wave Pa latencies (in ms) from 12 normal-hearing subjects to 1000-Hz tones presented in 60 dB effective masking noise (60 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	28.3		24.9	27.5			22.9	31.3	28.7	26.9	23.7	
85	26.9	28.1	27.3	27.5	29.5		23.7	31.1	28.7	26.3	23.3	27.3
80	27.7	28.3	26.9	27.3	29.9	33.5	26.3	32.7	28.1	25.7	23.5	27.1
77	30.3	29.7	27.3	29.3	30.9	33.3	28.7	33.1	28.5	25.9	24.7	25.9
75	29.1	29.3	27.9	27.9	30.3	33.7	28.5	33.9	28.9	25.9	24.3	27.5
72	29.1	29.9	28.3	29.5	31.5	32.5	30.9	33.9	30.5	27.3	24.9	27.9
70	29.1	29.7	27.5	30.1	31.3	32.3	30.9	33.9	30.5	27.7	25.9	27.9
67	31.7	29.7	29.1	30.9	32.7	32.7	33.1	34.7	29.7	28.7	29.3	28.9
65	32.5	30.5	28.5		30.9	32.7	32.5	33.5	31.9	29.7	26.1	28.5
62		30.5				33.7						
60		31.5	28.5		31.7		32.5	35.1		29.3	29.9	27.9
55		32.1					31.5	35.3				

Appendix K - Chapter 5

ABR (V-V') amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones (0 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	1.14		1.77	1.1			0.85	0.99	0.89	0.66	0.72	
85		1.08			0.87							0.74
80	0.96	1.26	1.44	1.2	0.83	0.84	0.77	1.47	1	0.43	0.48	0.84
70	0.7	0.72	1.44	0.74	0.36	0.81	0.62	1.17	0.72	0.41	0.78	0.86
60			1.08									
55	0.44	0.68		0.81	0.45	0.48	0.32	0.54	0.63		0.81	
50										0.25		0.5
45			0.69			0.42						
40		0.78		0.38	0.42		0.18	0.46	0.36		0.33	
35	0.2		0.66							0.14		0.6
30		0.28		0.3	0.37	0.25	0.11	0.61	0.22		0.48	
25	0.14		0.51							0.1		0.27
20	0.16	0.4	0.38	0.36	0.27	0.27	0.15	0.34	0.26		0.32	
15	0.2	0.22	0.21	0.24	0.18		0.21	0.37	0.33	0.2	0.33	0.42
10	0.16	0.32		0.08	0.26	0.34	0.09	0.19	0.09	0.22	0.3	0.26
5		0.08		0.17		0.28	0.19	0.27	0.21	0.15	0.2	0.36
0	0.14	0.08		0.15		0.22					0.22	0.24
-5						0.16						

Appendix 1. - Chapter 5

ABR (V-V') amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones presented in 40 dB effective masking noise (40 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	1.53		1.98	0.91			1.17	1.22	0.7	0.3	0.45	
85		1.5			0.75							1.08
80	0.67	0.94	1.04	0.76	0.43	0.88	0.52	0.47	0.49	0.5	0.39	0.75
75	0.74	0.94	0.99	0.61	0.48	0.7	0.45	0.36	0.54	0.45	0.54	0.75
72						0.66						
70	0.43	0.5	0.84	0.4	0.33	0.76	0.27	0.39	0.39	0.28	0.75	0.39
67						0.56						
65	0.34	0.56	0.62	0.37	0.42	0.62	0.27	0.41	0.42	0.28	0.57	0.28
60	0.36	0.38	0.5	0.24	0.45	0.36	0.24	0.55	0.33	0.23	0.42	0.33
55	0.23	0.36	0.52	0.27	0.29	0.44	0.39	0.5	0.27	0.31	0.27	0.33
50	0.13	0.34	0.54	0.37	0.25	0.35	0.24	0.28	0.21	0.24	0.48	0.27
45	0.14	0.26	0.18	0.18	0.15	0.19	0.18	0.25	0.18	0.32		0.1
40		0.19	0.16		0.17	0.19	0.2	0.12	0.19		0.21	0.21
35		0.25						0.18				

Appendix M - Chapter 5

ABR (V-V') amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones presented in 60 dB effective masking noise (60 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	0.9		1.2	0.7			1.5	0.8	0.72	0.44	0.6	
85	0.88	0.7	0.86	0.77	0.71		0.98	0.69	0.78	0.42	0.9	0.69
80	1.05	0.6	0.52	0.66	0.47	0.58	0.46	0.42	0.7	0.39	0.36	0.67
77	0.57	0.5	0.52	0.48	0.32	0.54	0.43	0.43	0.57	0.41	0.39	0.45
75	0.63	0.36	0.36	0.4	0.32	0.63	0.28	0.27	0.61	0.39	0.72	0.51
72	0.45	0.32	0.36	0.3	0.25	0.45	0.27	0.35	0.4	0.25	0.48	0.3
70	0.38	0.2	0.31	0.2	0.24	0.36	0.16	0.33	0.31	0.27	0.36	0.34
67	0.06	0.16	0.21	0.22	0.21	0.29	0.19	0.43	0.25		0.39	0.24
65	0.27	0.2	0.23	0.21	0.28	0.27	0.27	0.43	0.24	0.18	0.21	0.19
62		0.34				0.25						
60		0.12	0.21	0.16	0.18	0.26	0.3	0.27	0.25		0.39	0.09
55		0.18			0.18	0.18	0.06	0.11				

Appendix N - Chapter 5

MLR (Na-Pa) amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones (0 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	2.02		1.06	0.86			0.92	1.4	0.85	0.99	1.59	
85		1.3			0.66							1.05
80	1.3	1.26	0.85	0.92	0.85	0.81	1.21	1.42	0.64	0.64	1.3	1.47
70	0.92	0.88	0.77	0.78	0.5	1.11	0.81	1.01	0.58	0.8	1.76	0.95
60			0.7									
55	0.94	0.72		0.48	0.33	0.81	0.69	0.7	0.49		1.39	
50										0.9		0.45
45	0.92		0.52			0.73						
40		0.78		0.3	0.48		0.65	0.7	0.39		1.07	
35	0.54		0.54							0.63		0.55
30		0.74		0.39	0.38	0.6	0.62	0.97	0.32		0.91	
25	0.54		0.51							0.66		0.52
20	0.34	0.5	0.31	0.32	0.33	0.46	0.39	0.56	0.33		0.74	
15	0.38	0.66	0.3	0.15	0.29		0.46	0.58	0.27	0.48	0.78	0.4
10	0.36	0.52			0.32	0.37	0.09	0.58	0.27	0.45	0.55	0.47
5	0.4			0.27		0.36	0.1	0.35	0.27	0.32	0.34	0.51
0	0.28	0.32		0.33		0.39				0.15		0.3

Appendix O - Chapter 5

M1,R (Na-Pa) amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones presented in 40 dB effective masking noise (40 dBEM).

S/N	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	2.07		1.28	0.8			2.12	1.54	0.74	0.6	1.44	
85		1.68			0.99							1.5
80	1.1	0.9	0.7	0.41	0.54	1.07	0.4	1.01	0.4	0.76	0.81	0.88
75	0.94	0.84	0.83	0.36	0.47	1.04	0.48	0.86	0.34	0.45	0.48	0.74
72						1.08						
70	0.54	0.82	0.44	0.35	0.31	0.72	0.4	0.81	0.48	0.45	0.51	0.81
67						0.66						
65	0.48	0.7	0.46	0.4	0.33	0.64	0.4	0.91	0.33	0.41	0.48	0.59
60	0.43	0.54	0.42	0.16	0.32	0.51	0.27	0.63	0.36	0.38	0.78	0.4
55	0.45	0.52	0.36	0.15	0.36	0.53	0.42	0.45	0.36	0.24	0.54	0.6
50	0.25	0.42	0.32		0.6	0.29	0.33	0.36	0.24	0.27	0.69	0.35
45	0.3	0.36	0.14	0.23		0.3	0.24	0.62	0.27	0.18	0.54	0.34
40		0.18	0.14					0.4	0.18		0.42	0.45
35		0.21						0.25			0.3	

Appendix P - Chapter 5

MLR (Na-Pa) amplitudes (in μV) from 12 normal-hearing subjects to 1000-Hz tones presented in 60 dB effective masking noise (60 dBEM).

S#	11	12	13	14	16	17	18	19	21	27	28	29
dB nHL												
90	1.5		1.04	0.68			1.08	1.13	0.55	0.54	0.96	
85	1.5	0.84	0.68	0.65	1.05		1.14	0.66	0.62	0.59	1.08	0.75
80	1.3	0.9	0.52	0.3	0.71	1.44	0.46	0.76	0.76	0.47	1.05	1.12
77	0.8	0.56	0.46	0.55	0.48	0.86	0.45	0.56	0.47	0.46	0.99	0.82
75	0.83	0.62	0.25	0.33	0.33	1.17	0.27	0.45	0.51	0.29	1.11	0.72
72	0.58	0.7	0.24	0.32	0.39	0.75	0.27	0.51	0.49	0.3	0.9	0.37
70	0.46	0.52	0.17	0.19	0.21	0.63	0.19	0.33	0.28	0.18	0.63	0.46
67	0.18	0.8	0.25	0.12	0.24	0.44	0.37	0.55	0.28	0.21	0.45	0.38
65	0.31	0.76	0.14		0.25	0.42	0.19	0.57	0.34	0.21	0.63	0.48
62		0.2				0.52						
60		0.58	0.17		0.3		0.25	0.39	0.18		0.42	0.42
55		0.3					0.17	0.25				

Appendix Q - Chapter 5

ABR wave V latencies (in ms) from 12 sensorineurally hearing-impaired subjects to 1000-Hz tones.

S#	41	42	43	44	45	46	47	48	50	51	52	55
dB nHL												
90	6.3				6.7	6.1						
85				5.9							6.7	
80	6.7	6.7	6.3	6.1	6.9	6.5	5.9	6.1	6.5		6.9	7.3
77							6.7		6.7	5.9		
75	7.3	6.9	6.7	6.5	6.9	6.5	6.7	6.3	6.7	6.1	7.1	7.3
72	7.7						6.7		6.9	6.3		
70	7.5	7.1	6.7	6.7	7.5	7.1	6.7	6.3	7.1	6.5	9.7	7.7
67	7.9						7.1		7.3	6.7		
65	7.9		6.9	6.7	7.9		6.7	6.7	7.3	6.7	9.5	7.9
62							6.7			7.1		
60	10.7	7.5	7.3	6.9		7.3	6.9	6.9	7.7	7.3	8.5	
55		8.1			8.1	8.1	11.5	7.1	8.3	8.7	8.5	8.3
50		8.5	8.3	7.5	8.3	8.1		7.5			16.3	8.5
45		9.7		7.9	9.1	8.9		8.7				9.7
40		10.3	9.3	8.7		8.7		15.3				10.1
35		17.1	9.5	9.7		9.5		13.3				10.7
30			14.1									

Appendix R - Chapter 5

MLR wave Pa latencies (in ms) from 12 sensorineurally hearing-impaired subjects to 1000-Hz tones.

	S#	41	42	43	44	45	46	47	48	50	51	52	55
dB nHL													
90		27.3				33.1	23.9						
85					25.7							33.3	
80		27.7	27.7	28.1	26.7	34.1	25.1	25.7	26.1	26.1		34.3	28.3
77								25.3		25.9	26.1		
75		27.9	28.5	27.9	27.7	36.1	24.7	25.9	27.5	26.1	27.3	34.5	29.9
72		27.3						25.9		27.3	28.7		
70		27.3	28.7	28.5	27.3	36.6	26.3	25.9	26.9	27.5	28.3	34.1	29.7
67		27.5						25.9		27.5	29.1		
65		29.1		29.7	27.9	36.9		25.9	27.5	27.5	28.3	34.1	29.7
62										-0.9	29.3		
60			28.7	28.9	27.5		26.7	26.1	29.1	27.3	29.3	34.1	
55			28.5			36.9	25.9	26.3	29.5	26.9	30.9	35.3	30.1
50			29.7	29.7	28.5	36.1	26.9		28.7			41.9	31.1
45			30.9		28.7	36.1	26.7		28.7				30.9
40			30.5	31.9	28.3		26.9		28.7				31.3
35			31.3	33.5	28.5								31.9
30			31.3	34.9									
25				35.5									

Appendix S - Chapter 5

ABR (V-V') amplitudes (in μV) from 12 sensorineurally hearing-impaired subjects to 1000-Hz tones.

S#	41	42	43	44	45	46	47	48	50	51	52	55
dB nHL												
90	1.26				1.18	1.2						
85				0.72							0.85	
80	0.68	1.44	0.68	0.99	0.81	0.9	0.94	0.8	1.04		0.38	0.86
77							0.96		0.94	0.96		
75	0.46	1.33	0.82	0.9	0.92	0.86	0.76	1.04	0.82	0.74	0.35	0.86
72	0.37						0.56		0.72	1		
70	0.51	0.76	0.9	0.57	0.81	0.44	0.66	0.78	0.92	0.9	0.3	0.94
67	0.3						0.56		0.62	0.48		
65	0.36		0.88	0.72	0.46		0.46	0.36	0.46	0.5	0.32	0.66
62										0.42		
60	0.22	0.5	0.5	0.63		0.36	0.14	0.68	0.24	0.36	0.38	
55		0.48			0.29	0.26	0.2	0.6	0.28	0.16	0.4	0.56
50			0.44	0.48	0.15	0.16		0.12			0.18	0.44
45		0.44		0.45	0.23	0.2		0.34				0.32
40		0.42	0.36	0.24		0.21		0.2				0.26
35		0.2	0.16	0.22		0.27		0.28				0.32
30			0.34	0.22								

Appendix T - Chapter 5

MLR (Na-Pa) amplitudes (in μV) from 12 sensorineurally hearing-impaired subjects to 1000-Hz tones.

	S#	41	42	43	44	45	46	47	48	50	51	52	55
dB nHL													
90		0.91				0.63	0.88						
85					1.89							1.35	
80		0.65	2.13	1.44	1.29	0.51	0.84	1.32	1.48	1.38		1.23	1.18
77								1.14		1.3	0.96	1.27	
75		0.39	1.93	1.36	0.96	0.71	0.66	1.12	1.4	1.28	0.82	0.9	1.14
72		0.31						1.24		1.06	0.88		
70		0.43	1.76	1.26	1.05	0.57	0.7	1.32	0.92	1.2	0.9	0.99	1.12
67		0.36						0.78		0.96	0.6		
65		0.32		1.22	0.87	0.59		0.92	1.1	0.88	0.5		0.86
62											0.54		
60			1.42	1.38	0.96		0.56	0.56	1.18	0.6	0.5	0.68	
55			0.96			0.47	0.44	0.34	1	0.24	0.34	0.57	0.68
50			1.16	1.18	0.9	0.43	0.4		0.82			0.34	0.78
45			1.02		0.81	0.32	0.4		0.64				0.56
40			0.66	1.26	0.6		0.3		0.36				0.34
35			0.28	0.82	0.4								0.34
30			0.48	0.68									
25				0.54									

Appendix U - Chapter 6

ABR (V-V') and MLR (Na-Pa) amplitude exponents from 12 normal-hearing subjects to 1000-Hz tones presented in three levels of broadband masking noise.

	ABR			MLR		
	0 dBEM	40 dBEM	60 dBEM	0 dBEM	40 dBEM	60 dBEM
S#						
11	0.22	0.46	0.69	0.17	0.38	0.70
12	0.24	0.32	0.46	0.11	0.33	0.28
13	0.21	0.40	0.54	0.13	0.37	0.59
14	0.22	0.29	0.52	0.14	0.28	0.59
16	0.19	0.26	0.37	0.12	0.14	0.50
17*	0.15	0.35	0.48	0.14	0.38	0.51
18	0.21	0.26	0.64	0.20	0.29	0.56
19	0.17	0.26	0.36	0.11	0.24	0.28
21	0.19	0.25	0.42	0.11	0.18	0.35
27	0.14	0.07	0.30	0.12	0.25	0.43
28	0.11	0.11	0.24	0.13	0.15	0.28
29	0.12	0.36	0.70	0.12	0.24	0.33
Mean	**0.18**	**0.28**	**0.48**	**0.13**	**0.27**	**0.45**
sd	**0.04**	**0.11**	**0.15**	**0.03**	**0.08**	**0.15**
Median	**0.19**	**0.28**	**0.47**	**0.12**	**0.26**	**0.47**

*Subject's exponents are not included in the ANOVA.

Appendix V - Chapter 6

ABR (V) and MLR (Pa) latency exponents from 12 normal-hearing subjects to 1000-Hz tones presented in three levels of broadband masking noise.

	ABR			MLR		
	0 dBEM	**40 dBEM**	**60 dBEM**	**0 dBEM**	**40 dBEM**	**60 dBEM**
S#						
11	0.07	0.12	0.17	0.06	0.06	0.16
12	0.08	0.09	0.13	0.08	0.12	0.11
13	0.06	0.09	0.11	0.05	0.10	0.14
14	0.08	0.10	0.11	0.08	0.08	0.15
16	0.06	0.09	0.13	0.02	0.11	0.08
17*	0.07	0.10	0.17	0.07	0.00	-0.01
18	0.07	0.09	0.18	0.10	0.19	0.38
19	0.07	0.09	0.19	0.01	-0.03	0.08
21	0.06	0.09	0.10	0.07	0.09	0.10
27	0.07	0.09	0.19	0.07	0.15	0.14
28	0.07	0.10	0.17	0.03	0.08	0.33
29	0.07	0.09	0.12	0.19	0.12	0.08
Mean	**0.07**	**0.09**	**0.15**	**0.07**	**0.09**	**0.15**
sd	**0.01**	**0.01**	**0.03**	**0.05**	**0.06**	**0.11**
Median	**0.07**	**0.09**	**0.15**	**0.07**	**0.09**	**0.12**

*Subject's exponents are not included the AVOVA

Appendix W - Chapter 6

Loudness (AME) exponents from 12 normal-hearing subjects to 1000-Hz tones presented in three levels of broadband masking noise.

	AME		
	0 dBEM	**40 dBEM**	**60 dBEM**
S#			
11	0.27	0.32	1.19
12	0.26	0.52	1.08
13	0.27	0.58	0.67
14	0.37	0.74	0.80
16	0.26	1.19	1.09
17*	0.90	2.46	2.42
18	0.19	0.74	1.11
19	0.39	0.53	5.25
21	0.25	1.18	2.19
27	0.26	1.05	1.09
28	0.38	0.56	1.03
29	0.31	1.06	1.43
Mean	**0.34**	**0.91**	**1.61**
sd	**0.19**	**0.57**	**1.26**
Median	**0.27**	**0.74**	**1.10**

*Subject's exponents are not included in the ANOVA

Appendix X - Chapter 6

ABR, MLR, and AME exponents from 12 sensorineurally hearing-impaired subjects to 1000-Hz tones.

	ABR		MLR		AME
S#	Amplitude	Latency	Amplitude	Latency	
41	0.48	0.13	0.37	0.04	0.38
42	0.32	0.14	0.30	0.07	0.30
43	0.23	0.11	0.13	0.12	0.32
44	0.23	0.08	0.19	0.06	0.32
45	0.39	0.06	0.11	0.04	0.39
46	0.31	0.07	0.19	0.11	0.56
47	0.65	0.14	0.44	0.05	0.38
48	0.30	0.16	0.24	0.08	0.67
50	0.53	0.08	0.54	0.08	0.71
51	0.66	0.13	0.40	0.19	0.56
52	0.21	0.16	0.31	0.08	0.47
55	0.25	0.07	0.25	0.06	0.41
Mean	**0.38**	**0.11**	**0.29**	**0.08**	**0.46**
sd	**0.16**	**0.04**	**0.13**	**0.04**	**0.14**
Median	**0.31**	**0.11**	**0.27**	**0.02**	**0.40**

Appendix Y - Chapter 5

Threshold differences - Normal-hearing subjects in three noise conditions
Wave (ABR, MLR) × Noise Condition (0, 40, 60 dBEM)

```
      DESIGN: 2 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-WAVES(2) x 2-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (thr3c99.sta) 1-WAVES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	16.0556	11	8.96465	1.790986	.207812
2	2*	160.8472*	22*	25.11995*	6.403167*	.006433*#
12	2	.4306	22	8.33965	.051628	.949797

Threshold differences - Normal-hearing and sensorineurally-impaired subjects
Wave (ABR, MLR) × Hearing (NH, SNHL)

```
      DESIGN: 2 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: 2-WAVES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (thrdif03.sta) 1-HEARING, 2-WAVES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	75.00000	22	64.39394	1.164706	.292182
2	1	8.33333	22	5.30303	1.571429	.223158
12	1	0.00000	22	5.30303	0.000000	1.000000

 # the p value in this and all subsequent ANOVAs does not reflect Huynh-Feldt Epsilon corrections.

Appendix Z - Chapter 5

Latency - NH subjects in three noise conditions

Intensity (80, 70, 60 dB nHL) × Wave (ABR, MLR) × Noise Cond. (0, 40, 60 dBEM)

```
      DESIGN: 3 - way ANOVA          , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN: 1-INTENSIT( 3):     1     2     3
     WITHIN: 2-WAVES(2) x 3-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lvpa012.sta) 1-INTENSIT, 2-WAVES, 3-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	119.01*	33*	12.89996*	9.226*	.000657*
2	1*	24985.16*	33*	6.74838*	3702.393*	0.000000*
3	2*	44.20*	66*	2.18976*	20.183*	.000000*
12	2	.41	33	6.74838	.061	.940959
13	4	4.51	66	2.18976	2.058	.096324
23	2*	12.54*	66*	2.48871*	5.040*	.009187*
123	4	.98	66	2.48871	.393	.813008

Amplitude - NH subjects in three noise conditions

Intensity (80, 70, 60 dB nHL) × Wave (ABR, MLR) × Noise Cond. (0, 40, 60 dBEM)

```
      DESIGN: 3 - way ANOVA          , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN: 1-INTENSIT( 3):     1     2     3
     WITHIN: 2-WAVES(2) x 3-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (avpa012x.sta) 1-INTENSIT, 2-WAVES, 3-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	2.327298*	33*	.127489*	18.2549*	.000005*
2	1*	.620174*	33*	.071365*	8.6902*	.005836*
3	2*	3.986526*	66*	.033888*	117.6389*	.000000*
12	2	.018799	33	.071365	.2634	.770018
13	4	.110499	66	.033888	3.2607	.016777
23	2	.010800	66	.024935	.4331	.650296
123	4	.030109	66	.024935	1.2075	.316012

Appendix AA - Chapter 5

Latency - Normal-hearing and SNHL subjects
Hearing (NH, SNHL) × Wave (ABR, MLR) × Intensity (70, 55, 40 dB nHL)

```
      DESIGN: 3 - way ANOVA          , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN: 1-HEARING ( 2):      1      2
     WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (anvlat03.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	10.10	16	23.64079	.4273	.522602
2	1*	13332.22*	16*	16.95239*	786.4510*	.000000*
3	2*	37.63*	32*	.92554*	40.6580*	.000000*
12	1	8.13	16	16.95239	.4795	.498567
13	2	2.34	32	.92554	2.5281	.095641
23	2*	6.14*	32*	.85333*	7.1939*	.002630*
123	2	.01	32	.85333	.0063	.993692

Amplitude - Normal-hearing and SNHL subjects
Hearing (NH, SNHL) × Wave (ABR, MLR) × Intensity (70, 55, 40 dB nHL)

```
      DESIGN: 3 - way ANOVA          , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN: 1-HEARING ( 2):      1      2
     WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (avpa03x.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.216366	16	.220405	.98168	.336534
2	1*	1.465337*	16*	.093061*	15.74593*	.001104*
3	2*	1.813414*	32*	.038321*	47.32124*	.000000*
12	1	.048981	16	.093061	.52634	.478630
13	2*	.204846*	32*	.038321*	5.34549*	.009932*
23	2	.009823	32	.018772	.52327	.597560
123	2	.019918	32	.018772	1.06100	.357971

Appendix AB - Chapter 5

Latency - Masked (40 dBEM) normal-hearing and SNHL subjects
Hearing (NMNH, SNHL) × Wave (ABR, MLR) × Intensity (80, 70, 60 dB nHL)

```
    DESIGN: 3 - way ANOVA        , fixed effects
 DEPENDENT: 1 variable (Repeated Measure)
   BETWEEN: 1-HEARING ( 2):     1     2
    WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lvpal3.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	65.75	22	15.59802	4.215	.052149
2	1*	16737.89*	22*	9.02732*	1854.138*	.000000*
3	2*	51.52*	44*	.62696*	82.166*	.000000*
12	1	.12	22	9.02732	.013	.910494
13	2*	6.29*	44*	.62696*	10.035*	.000257*
23	2	.00	44	.60084	.005	.995503
123	2	.10	44	.60084	.170	.844202

Amplitude - Masked (40 dBEM) normal-hearing and SNHL subjects
Hearing (NMNH, SNHL) × Wave (ABR, MLR) × Intensity (80, 70, 60 dB nHL)

```
    DESIGN: 3 - way ANOVA        , fixed effects
 DEPENDENT: 1 variable (Repeated Measure)
   BETWEEN: 1-HEARING ( 2):     1     2
    WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (avpal3.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1*	3.147372*	22*	.217742*	14.45462*	.000977*
2	1*	1.490638*	22*	.095476*	15.61262*	.000679*
3	2*	1.686078*	44*	.033059*	51.00280*	.000000*
12	1	.553908	22	.095476	5.80152	.024830
13	2	.089105	44	.033059	2.69536	.078661
23	2	.000928	44	.017477	.05312	.948330
123	2	.002730	44	.017477	.15621	.855850

Appendix AC - Chapter 5

Latency - Masked (60 dBEM) normal-hearing and SNHL subjects
Hearing (NMNH, SNHL) × Wave (ABR, MLR) × Intensity (80, 70, 60 dB nHL)

```
      DESIGN: 3 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lvpa23.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	67.38	22	14.84984	4.537	.044594
2	1*	16101.50*	22*	8.54694*	1883.891*	.000000*
3	2*	63.01*	44*	.84628*	74.455*	.000000*
12	1	7.98	22	8.54694	.934	.344396
13	2*	10.97*	44*	.84628*	12.965*	.000037*
23	2	.12	44	.49993	.244	.784277
123	2	.06	44	.49993	.125	.882428

Amplitude - Masked (60 dBEM) normal-hearing and SNHL subjects
Hearing (NMNH, SNHL) × Wave (ABR, MLR) × Intensity (80, 70, 60 dB nHL)

```
      DESIGN: 3 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: 2-WAVES(2) x 3-INTENSIT(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (avpa23x.sta) 1-HEARING, 2-WAVES, 3-INTENSIT					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1*	6.563417*	22*	.206040*	31.85511*	.000011*
2	1*	1.771783*	22*	.095677*	18.51835*	.000288*
3	2*	2.662889*	44*	.045112*	59.02809*	.000000*
12	1	.402062	22	.095677	4.20228	.052475
13	2	.123195	44	.045112	2.73085	.076215
23	2	.032794	44	.017951	1.82688	.172911
123	2	.042480	44	.017951	2.36648	.105647

Appendix AD - Chapter 6

Amplitude & AME exponents - NH subjects in three noise conditions
Measure (ABR, MLR, AME) × Noise Condition (0, 40, 60 dBEM)

```
      DESIGN: 2 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(3) x 2-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (azmeexp.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	3.600094*	20*	.230699*	15.60515*	.000083*
2	2*	3.215503*	20*	.174809*	18.39434*	.000029*
12	4*	.837113*	40*	.213994*	3.91185*	.008983*

Latency & AME exponents - NH subjects in three noise conditions
Measure (ABR, MLR, AME) × Noise Condition (0, 40, 60 dBEM)

```
      DESIGN: 2 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(3) x 2-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lameexp.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	6.200182*	20*	.210386*	29.47057*	.000001*
2	2*	1.923606*	20*	.203571*	9.44930*	.001291*
12	4*	1.249235*	40*	.195904*	6.37677*	.000456*

Appendix AE - Chapter 6

Amplitude exponents- NH subjects in three noise conditions
Measure (ABR, MLR) × Noise Condition (0, 40, 60 dBEM)

```
     DESIGN: 2 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(2) x 2-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (aameexp.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.018573	10	.006609	2.81039	.124590
2	2*	.514993*	20*	.008707*	59.14851*	.000000*
12	2	.001590	20	.004280	.37137	.694447

Latency exponents - NH subjects in three noise conditions
Measure (ABR, MLR) × Noise Condition (0, 40, 60 dBEM)

```
     DESIGN: 2 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(2) x 2-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lameexp.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.000322	10	.004619	.06976	.797057
2	2*	.048597*	20*	.003450*	14.08469*	.000152*
12	2	.000333	20	.002118	.15701	.855738

Appendix AF - Chapter 6

AME exponents - NH subjects in three noise conditions
Measure (AME) × Noise Condition (0, 40, 60 dBEM)

```
      DESIGN: 1 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-NOISECO(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (aameexp.sta) 1-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	4.373147	20	.589810	7.414496	.003898*#

 # p value does not reflect Huynh-Feldt Epsilon correction factors.

Appendix AG - Chapter 6

Amplitude & AME proportional exponent increase - NH subjects in three noise conditions

Measure (ABR, MLR, AME) × Noise Condition (40/0, 60/0 dBEM)

```
      DESIGN: 2 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(3) x 2-NOISECO(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (jjpr7amp.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	12.42537	18	2.488111	4.99390	.018825
2	1*	28.03351*	9*	.342427*	81.86702*	.000008*
12	2	.08194	18	.695262	.11786	.889502

Latency & AME proportional exponent increase - NH subjects in three noise conditions

Measure (ABR, MLR, AME) × Noise Condition (40/0, 60/0 dBEM)

```
      DESIGN: 2 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(3) x 2-NOISECO(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lameprp2.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	18.98371	18	3.745927	5.06783	.017953
2	1	17.54333	9	1.691897	10.36903	.010488
12	2	.69845	18	1.363693	.51217	.607665

Appendix AH - Chapter 6

ABR Latency & AME proportional exponent increase - NH subjects in three noise conditions

Measure (ABR, AME) × Noise Condition (40/0, 60/0 dBEM)

```
      DESIGN: 2 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(2) x 2-NOISECO(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lprvame01.sta) 1-MEASURES, 2-NOISECO					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1*	37.38014*	9*	2.005690*	18.63705*	.001941*
2	1*	11.07879*	9*	.535675*	20.68195*	.001390*
12	1	1.37184	9	.643928	2.13042	.178406

Appendix AI - Chapter 6

Amplitude & AME relative exponent increase - NH subjects in noise (60/0 ÷ 40/0)
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (jjpraa6-4.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	.286384	18	.750320	.381683	.688107

Latency & AME relative exponent increase - NH subjects in noise (60/0 ÷ 40/0)
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lsix-for7.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	.093771	18	.547928	.171137	.844061

Appendix AJ - Chapter 6

ABR Latency & AME relative exponent increase - NH subjects in noise (60/0 ÷ 40/0)
Measure (ABR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lsix-for7.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.123556	9	.429717	.287528	.604807

Appendix AK - Chapter 6

Amplitude exponents - SNHL subjects
Measure (ABR, MLR)

```
     DESIGN: 1 - way ANOVA        , fixed effects
 DEPENDENT: 1 variable (Repeated Measure)
   BETWEEN:          none
    WITHIN: 1-WAVES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (amps.sta) 1-WAVES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.050878	11	.006396	7.955165	.016653

Latency exponents - SNHL subjects
Measure (ABR, MLR)

```
     DESIGN: 1 - way ANOVA        , fixed effects
 DEPENDENT: 1 variable (Repeated Measure)
   BETWEEN:          none
    WITHIN: 1-WAVES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lats.sta) 1-WAVES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.005617	11	.001266	4.437346	.058933

Appendix AL - Chapter 6

Amplitude & AME exponents - SNHL subjects
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (ameamps.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	.085538*	22*	.011103*	7.703922*	.002911*

Latency & AME exponents - SNHL subjects
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (amelats.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	.524309*	22*	.006580*	79.68316*	.000000*

Appendix AM - Chapter 6

Amplitude exponents - NH and SNHL subjects
Hearing (NH, SNHL) × Measure (ABR, MLR)

```
      DESIGN: 2 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: 2-MEASURES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (ameamp.sta) 1-HEARING, 2-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1*	.355771*	21*	.020058*	17.73747*	.000392*
2	1*	.059107*	21*	.003810*	15.51443*	.000752*
12	1	.004742	21	.003810	1.24464	.277182

Latency exponents - NH and SNHL subjects
Hearing (NH, SNHL) × Measure (ABR, MLR)

```
      DESIGN: 2 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: 2-MEASURES(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (amelat.sta) 1-HEARING, 2-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.004589	21	.001737	2.699969	.115240
2	1	.002865	21	.001377	2.079755	.164013
12	1	.002514	21	.001377	1.824800	.191120

Appendix AN - Chapter 6

AME exponents - NH and SNHL subjects
Measure (AME) × Hearing (NH, SNHL)

```
      DESIGN: 1 - way ANOVA        , fixed effects
   DEPENDENT: 1 variable:     AME
     BETWEEN: 1-HEARING ( 2):     1     2
      WITHIN: none
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (ameamp.sta) 1-HEARING					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1*	.159419*	21*	.011994*	13.29165*	.001511*

Appendix AO - Chapter 6

Amplitude & AME proportional exponent increase - SNHL subjects (SNHL ÷ mean NH exponent in the 0 dBEM)
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (aprp3-0.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	1.427195	22	.357408	3.993177	.033150

Latency & AME proportional exponent increase - SNHL subjects (SNHL ÷ mean NH exponent in the 0 dBEM)
Measure (ABR, MLR, AME)

```
      DESIGN: 1 - way ANOVA        , fixed effects
  DEPENDENT: 1 variable (Repeated Measure)
    BETWEEN:          none
     WITHIN: 1-MEASURES(3)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lrprp3-0.sta) 1-MEASURES					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	.864212	22	.206713	4.180725	.028914

Appendix AP - Chapter 6

ABR Latency & AME proportional exponent increase - SNHL subjects (SNHL ÷ mean NH exponent in the 0 dBEM)
Measure (ABR, AME)

```
      DESIGN: 1 - way ANOVA         , fixed effects
   DEPENDENT: 1 variable (Repeated Measure)
     BETWEEN:          none
      WITHIN: 1-MEASURE(2)
```

STAT. GENERAL MANOVA	Summary of all Effects; design: (lrvameprp3-0.sta) 1-MEASURE					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.076954	11	.217522	.353776	.564018

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