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City University of New York PH.D. 1985

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THE EFFECT OF STIMULUS CENTER FREQUENCY AND BANDWIDTH ON THE RESPONSE-TIME OF THE HUMAN ACOUSTIC REFLEX

by

MINDY WERTHEIMER SIRLIN

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

1985

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March 15, 1985
date

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March 15, 1985
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Abstract

THE EFFECT OF STIMULUS CENTER FREQUENCY AND BANDWIDTH ON THE RESPONSE-TIME OF THE HUMAN ACOUSTIC REFLEX

by

Mindy Wertheimer Sirlin

Adviser: Professor Harry Levitt

Acoustic reflex response-time, defined as the time (in msec) between stimulus onset and 10% of the maximum impedance change caused by the acoustic reflex, was measured in ten normal young adult subjects. Five of the subjects exhibited a biphasic response and five exhibited a uniphasic response.

Prior to the main experiment, a new procedure for simulating the phase and amplitude changes caused in the ear by the acoustic reflex was used to determine whether and to what extent the measuring equipment was adding to the observed response-times. The equipment used in this study had virtually no delay in response and had a rise time of approximately 12 msec to an electrically simulated reflex. These findings indicated that the instrumentation was accurately tracking the impedance change caused by the reflex.

In the main experiment, stimuli consisted of five

bandwidths (1/6, 1/3, 1, 2 and 4 octaves) centered at three frequencies (500, 1000 and 4000 Hz) presented both at ART and at 10 dB above ART.

The data indicated that acoustic reflex response time is significantly faster for 500 Hz stimulation than it is for 4000 Hz stimulation. There were no significant differences between biphasic and uniphasic response times. Changes in bandwidth did not result in significant changes in response-time at 500 or 1000 Hz. At 4000 Hz, for signals wider than an octave, acoustic reflex response-time was significantly shorter than it was for signals narrower than an octave. The small initial negative peak of the biphasic reflex behaved differently than the major observed portion of the response.

There were important differences between ART and reflex response-time. The effect of frequency on response-time was opposite that of the effect of frequency on ART. While there is a critical band effect for acoustic reflex threshold, there is not a critical band for acoustic reflex response time.

DEDICATION

I dedicate this dissertation, with love and gratitude,

to the memory of my father, Joel Wertheimer.

ACKNOWLEDGEMENTS

Whereas I haven't taken the longest time in recorded history to finish the requirements for this degree, I haven't set any speed records, either. It should not be surprising, then, that I feel I have incurred a number of debts along the way.

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and helpful comments. Stanley Gelfand, the external examiner, contributed immensely to the final copy with many important suggestions.

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Judy Rubin-Spitz guided me through the maze of computer-assisted data analysis. She helped to make the data analysis (one of the aspects of the study that I dreaded the most) a pleasant experience, and thus, earned my sincerest gratitude.

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forget all that they did to help me to achieve this goal.

Beverly Wertheimer, my mother, listened for years to my hopes, my fears and aspirations in relation to this degree. She is right to think that she earned it too, for in many ways, she did.

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These last thanks are for the two people dearest to my heart, and who, more than anyone else, lived this degree with me. My husband, Ed, and daughter, Joanna, provided the glue that kept me together throughout the course of this project. Ed was absolutely unwavering in his support and in his belief in my ability to finish the degree, even when I had grave doubts about it myself. He gave selflessly and willingly in more ways than I can count. Joanna helped by just being her beautiful little self, with her smile that warms me like sunshine!

My thanks to each and every one of you.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

Over the past fifteen years, the measurement of the acoustic reflex has become a standard part of the audiologic evaluation. The widespread commercial availablility of the electroacoustic impedance bridge (now called the electroacoustic immittance device), and the results of a number of clinical and research studies identifying the theoretical and diagnostic implications of the reflex contributed to the incorporation of reflex testing into the clinical routine.

There are several reasons for the importance of the acoustic reflex. First, it is a completely objective measure, requiring no subjective judgements or voluntary responses on the part of the subject. Second, it is a bilateral response to unilateral stimulation. Third, the reflex arc goes no higher than the superior olivary complex. Thus, the contralateral acoustic reflex response depends upon (and yields information about) the integrity of three systems: the afferent portion of the reflex arc in the stimulus ear, the efferent portion of the reflex arc in the response ear, and the middle ear systems of both ears. By measuring the acoustic reflex, the clinician or researcher rapidly and objectively obtains a wealth of information regarding the peripheral ear and lower order neurons of the auditory system.

Most of the early research into the clinical usefulness and theoretical implications of the acoustic reflex was focused upon the acoustic reflex threshold. Specifically, the effects of various stimulus parameters such as frequency (Jepsen, 1963; Deutsch, 1968; Peterson and Liden, 1972; Margolis and Popelka, 1975), bandwidth (Flottorp, et al., 1971; Djupesland and Zwislocki, 1973; Popelka, et al., 1976; Margolis, et al., 1980) and duration (Metz, 1951; Jerger, et al., 1977; Richards and Goodman, 1977) have been studied in both normal and impaired ears.

As a result of these and other studies, we know that the normal acoustic threshold reflex occurs in response to signals about 70 - 90 dB above the threshold of hearing, depending on the frequency and bandwidth of the signal. We also know that there is a critical band for acoustic reflex threshold, as well as temporal summation at both acoustic reflex threshold and suprathreshold levels. Thus, the presence, absence, level, and differential response of the acoustic reflex threshold to noise and tones has attained diagnostic significance. In addition, a good deal of work has been done on predicting hearing levels from the acoustic reflex (Niemeyer and Sesterhenn, 1974; Jerger, et al., 1974; Silman and Gelfand, 1979), and on determining the effect of age and hearing loss on acoustic reflex threshold (Jepsen, 1963; Jerger, et al., 1974, 1978; Silman, 1979).

The early acoustic reflex literature notes larger variation in responses near threshold than in those above

threshold (Moller, 1962, 1974; Borg, 1972). While one recent publication (Popelka, 1981) contends that the variability at acoustic reflex threshold is related to procedural variables, others (Silman and Gelfand, 1981; Gelfand, Piper and Silman, 1983; Gelfand and Piper, 1984) indicate that the intersubject variablility of acoustic reflex thresholds in normal and cochlear impaired ears is high. Given the questions raised in the literature regarding the stability of acoustic reflex threshold measurements, it seems important to clarify the issue of the variability of the response at threshold.

Recently, it has been observed that changes in the time course of the acoustic reflex may reflect significant pathological changes in the ear (Anderson, et al., 1969; Norris, et al., 1974; Clemis and Sarno, 1980, 1980A; Olsen, Stach and Kurdziel, 1981; Stach and Jerger, 1984). In addition, the use of computer averaging techniques to record the acoustic reflex response has rendered temporal changes in the reflex-response trace easier to observe (Stach and Jerger, 1984). Thus, aspects of the acoustic reflex other than acoustic reflex threshold have been recently studied.

Specifically, temporal characteristics such as acoustic reflex latency (Moller, 1958; Dallos, 1964; Norris, et al., 1974; Ruth and Niswander, 1976; Sieminski, et al., 1977; McPherson and Thompson, 1977; Clemis and Sarno, 1980, 1980A; Church and Cudahy, 1984; Stach and Jerger, 1984), rise time (Gorga and Stelmachowicz, 1983) and decay characteristics

(Norris, et al., 1974) have been examined. Latency measures have been of particular interest because of their apparent sensitivity to retrocochlear disorder (Clemis and Sarno, 1980, 1980A). However, there is a paucity of data regarding many aspects of the initial response time of the reflex in the normal ear, and regarding the manner in which it is affected by that of the measuring system.

While the effect of intensity on normal acoustic reflex latency has been clearly established (Metz, 1951; Moller, 1958; Dallos, 1964), very little research has been done to determine the effects of frequency and bandwidth on the response time of the acoustic reflex. Thus, while we know that there are differences in acoustic reflex threshold sensitivity to different frequencies and that there is a critical band for acoustic reflex threshold, we do not know the effects of frequency and bandwidth on the response time of the acoustic reflex in normal ears. Clearly, information regarding the normal temporal behavior of the acoustic reflex is necessary, especially since the temporal morphology of the reflex appears to have diagnostic significance (Clemis and Sarno, 1980, 1980A; Stach and Jerger, 1984; Jerger, et al., 1984).

One aspect of the normal acoustic reflex response that complicates latency measures is the biphasic acoustic reflex. While the majority of acoustic reflex reponses are uniphasic, a biphasic response has been noted in a percentage of subjects in a number of studies (Neergard and

Rasmussen, 1966; Hung and Dallos, 1972; Ruth and Niswander, 1976; McPherson and Thompson, 1977; Love and Stream, 1978; Clemis and Sarno, 1980; Zito and Roberto, 1980). However, the temporal characteristics of the biphasic reflex have not yet been explored. There was no attempt in any of the studies cited to determine the similarities and differences between the biphasic and uniphasic reflex response time. The small, initial negative peak of the biphasic reflex is sometimes ignored, and sometimes defined as reflex onset, depending upon the author of the study.

Previous studies of temporal aspects of the acoustic reflex have been complicated by methodological problems. Unaveraged reflex-response traces are noisy, making it difficult to pinpoint temporal changes with certainty. The response delays and filter time constants of the measuring equipment were either not measured or not taken into account in specifying measured reflex latencies. A standard measure of acoustic reflex latency has not yet been defined, and the usefulness of traditional latency measures is being questioned (Stach and Jerger, 1984; Jerger, et al., 1984).

After taking steps to measure and account for those sources of error, this study was designed to answer the following research questions:

1. Do changes in stimulus center frequency affect the response time of the acoustic reflex?

2. Do changes in stimulus bandwidth affect the response time of the acoustic reflex?

3. Is acoustic reflex response time a stable measure when measured in the same subjects on different days?

4. How do the affects of frequency, bandwidth and test replication on acoustic reflex threshold compare with the effects of the same variance components on acoustic reflex response time?

5. How does the response time of each peak of the biphasic acoustic reflex compare with the response time of the uniphasic reflex?[1]

---

[1] In the early stages of data collection it became apparent that some of the subjects were exhibiting a biphasic acoustic reflex. Thus, an additional research question was added.

CHAPTER 2

REVIEW OF THE LITERATURE

THE MIDDLE EAR MUSCLES AND THE ACOUSTIC REFLEX

It has long been known that the stapedius and tensor tympani muscles of the middle ear exhibit reflex contractions. When the eliciting stimulus is an acoustic signal, the reflex contraction is called an "acoustic reflex" (Jepsen,1963). Non acoustic stimuli, such as an air jet to the eye or touching the skin of the external auditory meatus, will also cause the tympanic muscles to contract. Djupesland (1976) notes that the middle ear muscles contract during certain movements of the head and neck, such as chewing, swallowing and vocalization. Borg and Zakrisson (1975) found that stapedius muscle activity could be detected (in humans undergoing ear surgery) 100 msec before vocalization. While the measurement of non-acoustic reflexes has been suggested as a differential diagnostic tool for middle ear lesions (Jerger, 1975; Djupesland, 1976), the acoustic reflex is the subject of this dissertation.

ANATOMY OF THE MIDDLE EAR MUSCLES

Anatomically, the stapedius and tensor tympani muscles are similar in that they both have many relatively short, parallel fibers. This arrangement, according to Wever and Lawrence (1954), allows for great tension with little displacement of the muscles themselves. The stapedius

measures about 6 mm in length, and is the smallest muscle in the body, but, according to Lilly (1981), has the longest tendon in the body compared to the length of the muscle. The tensor tympani is about 25 mm in length (Wever & Lawrence, 1954).

Both muscles lie within bony sheaths. The tensor tympani lies within the semicanal parallel and superior to the eustachian tube, and the stapedius lies within the pyramidal eminence on the posterior wall of the middle ear. Only their tendons enter the middle ear cavity. Jepsen (1963) reports that according to Von Bekesy, the arrangement of the middle ear muscles within bony casings serves to insulate the ear from muscular vibrations during the transmission of sound. The tensor tympani tendon attaches to the manubrium of the malleus at a right angle to the primary direction of the muscle, and the stapedius tendon attaches to the neck of the stapes (Yost and Nielson, 1977).

The muscles have different innervation and reflex arcs. The tensor tympani is innervated by a branch of the fifth cranial (trigemminal) nerve, and the stapedius is innervated by a branch of the seventh cranial (facial) nerve. According to Moller (1983), the details of the pathway for the tensor tympani reflex arc are unknown, although it is believed to be controlled via the medial superior olive or lateral lemniscus. The neural pathway of the stapedial reflex arc is complex, involving many subcortical nucleii. However, it is generally agreed that the reflex arc goes no higher than

the pons at the level of the superior olivary complex. Simply stated, the four major meurons of the stapedius reflex arc are the:

1) primary afferents in the spiral ganglion;
2) ventral nucleii of the cochlear nerve;
3) neurons of the ipsilateral and contralateral medial superior olivary complex; and
4) motoneurons in the nucleus of the seventh (facial) nerve (Moller,1974).

EFFECTS OF MIDDLE EAR MUSCLE CONTRACTION

Contraction of the tensor tympani results in a medial (inward) pull on the malleus (Wever and Lawrence, 1954), which in turn pulls the entire ossicular chain inward and forward (Love and Stream, 1978). While it is generally agreed that the function of the tensor tympani is to tense the tympanic membrane, Wever and Lawrence (1954) add that it "exerts a tension nearly at right angles to the course of the ossicular chain as a whole" (p.179). Stapedial contraction exerts lateral tension on the ossicular chain, pulling the stapes posteriorly. It causes only very small movement of the eardrum because its pull on the stapes is almost perpendicular to the outward - inward movement of the stapes footplate (Moller, 1974). According to Kobrak (1959), stapedial contraction produces a pull on the stapes that pivots the stapes footplate, displacing the anterior

portion of the footplate laterally by .01 cm from the oval window. This effectively dampens the motion of the entire ossicular chain.

Thus, the two muscles pull the ossicular chain laterally, but in opposite directions: the tensor pulls medially and forward while the stapedius pulls laterally and posterially. Be that as it may, as stated by Yost and Nielson (1977), "contraction of the middle ear muscles reduces the transmission of pressure through the ossicular chain" (p.39). Moller (1983) adds that stapedial contraction reduces the energy reaching the inner ear.

Specifically, the acoustic reflex reduces the transmission of intense, low frequency sound to the cochlea because it results in increased stiffness of the middle ear. Borg (1968) reports that at frequencies below the middle ear's natural resonance frequency (about 1000 Hz), a 1 dB increase in sound pressure at the tympanic membrane results in only a 0.6 to 0.7 dB increase in the sound reaching the cochlea. His subjects had temporary unilateral paralysis of the stapedius muscle due to Bell's palsy, and he used the change in impedance in the affected ear during reflex elicitation pre and post recovery as a measure of excitation at the cochlea. At frequencies above the ear's natural resonance, only strong ("near maximal") contraction reduces sound transmission to the cochlea (Moller,1974). This indicates that the reflex does more than simply increase the stiffness of the ossicular chain. The experiments of Kobrak

(1959), Borg (1968) and Moller suggest that stapedial contraction changes the mode of vibration of the stapes within the oval window in such a way that its motion is less efficient in transmitting vibration to the fluid within the cochlea. Thus, the major effect of decreased low frequency transmission during acoustic reflex contraction is the result of increased stiffness, while the lesser effect of reduction of high frequency energy is the result of a change in the vibratory mode of the stapes.

Based on these experimental results and on similar observations in earlier studies in experimental animals (see Jepsen, 1963) it is clear that the effect of acoustic reflex contraction is different for different frequencies.

FUNCTIONS OF THE ACOUSTIC REFLEX

One recent theory regarding the function of the acoustic reflex that takes its differential frequency effect into account is proposed by Moller (1983). He proposes that the reflex is a feedback system, regulating low frequency transmission to the cochlea to a greater extent than it regulates high frequency transmission to the cochlea. He found additional evidence (Moller, 1962) to support this theory in the reflex traces to 500 Hz and 1400 Hz stimulation. At 500 Hz, there is a faster onset and damped oscillation at reflex onset because of the greater influence on middle ear transmission at low frequencies. At 1450 Hz, because of minimal regulation, the response is slower and

does not exhibit oscillation. It should be noted that others (Peterson and Liden, 1972; Hung and Dallos, 1972; Borg, 1976) also report a rhythmic reflex onset to low frequency stimuli. Peterson and Liden (1972) hypothesize that its cause is the blood pulse rate or breathing rate of the subjects. Hung and Dallos (1972) and Wilbur (1976), like Moller (1962, 1983) attribute the oscillation at reflex onset to the frequency dependent attenuation of sound in the middle ear that is caused by the activation of the acoustic reflex. Borg (1976) refers to the stapedial reflex as a "control system" (p.250).

Moller's theory is in agreement with the earlier Protection Theory (also called Intensity Control Theory) described by Wever and Lawrence (1954) and Jepsen (1963). According to these views, the acoustic reflex protects or safeguards the inner ear against overstimulation by strong sounds. Clearly, observations that the middle ear muscles contract during certain head and neck movements and during vocalization (Djupesland, 1976) also suggest a protective function. Indeed, most of the experimental evidence in the literature is in firm support of this theory. The limitations of the theory with regard to latency and muscular adaptation or fatigue will be discussed later.

According to Protection Theory, the acoustic reflex protects or safeguards the inner ear against overstimulation by strong sounds. Data on noise induced TTS and fatigue also support this theory, indicating that exposure to broad

spectrum noise causes maximum TTS at about 4000 Hz (Ward, 1963).

Dallos (1973) points out that most "self-noises," such as as chewing and vocalization, are low frequency noises that create a great deal of internal noise. The fact that such noises elicit the middle ear muscle reflex, which attenuates low frequencies, suggests that the protective function also includes some enhancement of the signal-to-noise ratio of environmental sounds. The finding that reflex activity precedes vocalization by 100 msec (Borg and Zakrisson, 1975) also suggests that the middle ear muscles play a role in preventing the low frequency component of one's own speech from masking other high frequency sounds (Moller, 1983). Other theories of tympanic muscle function receive less support from experimental data. According to Wever and Lawrence (1954) and Jepsen (1963), the Frequency Selection or Accomodation Theory was proposed as early as 1683 and was again described in 1863. It is an analogy with the process of visual selection, and asserts that the ear fixes and follows tones just as the eye does. According to this theory, the muscle contractions vary, allowing them to pick out particular components in a complex sound by adjusting the resonance frequency of the ear to them. Considering that there is virtually no experimental evidence to support this line of reasoning, and that the muscular action is a simple reflex, not under voluntary control, the theory is no longer considered viable.

Another early theory, proposed by Valsalva in 1710 is the Fixation Theory. This theory states that the tympanic muscles help to maintain the ossicular chain in its proper position. In addition, their contractions prevent excessive lateral motion of the ossicles. This theory, according to Wever and Vernon (1954) received some support from Helmholtz. Jepsen (1963) considers Von Bekesy's (1936) view that the muscles function to prevent the generation of clicking sounds between the ossicles at high frequencies to be support for this theory as well. Unlike the Accomodation Theory, this theory is well supported. The fragility of the ossicular chain following the severance of both the tensor and stapedial tendons is evidence for their contribution to its strength and rigidity.

The Labyrinthine Pressure Theory states that the acoustic reflex raises the pressure of the fluid within the labyrinth of the inner ear, changing its mechanical performance. Wever and Lawrence (1954) report that early animal and cadaver experiments show that while the muscular contractions do change the pressure in the labyrinthine fluid, these changes do not modify transmission within the fluid.

In summary, the most current theory regarding the function of the acoustic reflex, as proposed by Moller (1983), is that the reflex is a feedback system, affecting the transmission of low frequencies more than high frequencies within the middle ear by increasing the

stiffness of the ossicular chain and changing the mode of vibration of the stapes within the oval window. This theory relates to and agrees with the earlier Protection Theory which also receives good experimental validation in the literature. The Accomodation and Labyrinthine Pressure Theories are not verified by experimental data. The Fixation Theory is supported by the observation that the ossicular chain is fragile when the muscular tendons are severed. Thus, two major functions of the tympanic muscles and their reflex emerge. They are 1) fixation of the ossicular chain and 2) feedback - intensity control.

BIPHASIC ACOUSTIC REFLEX

A phenomenon that occurs in a percentage of subjects studied, and which must be examined in order to understand the physiological underpinnings of the acoustic reflex, is the biphasic acoustic reflex.

The first report of a biphasic acoustic refles response was made by Neergaard and Rasmussen (1966) who studied air pressure changes in the external auditory meatus during contraction of the stapedius muscle. Most tracings began with negative volume displacements. Hung and Dallos (1972) were the first to document a biphasic impedance change at the onset of the acoustic reflex response. It is described as "an initial small `negative change´ or `dip´ in the impedance change" (p.1174), and is noted to have occurred consistently in two subjects, regardless of the type of stimuli used to elicit the reflex. The researchers

attribute the observed biphasic change to a brief period of stapedial relaxation prior to contraction. They attach the label "latency relaxation (LR)" to the brief negaive impedance change, and cite LR as a well known physiological mechanism. Recently, however, the latency relaxation explanation of the biphasic reflex has been disputed (Mangham, et.al., 1983). Another early reference to the biphasic response appears in an article about acoustic reflexes, stapedial fixation and otosclerosis. Terkildsen, et.al. (1973) note that many normal ears "exhibit a small initial negativity at the onset of a reflex..." (p.153).

Although later studies (Ruth and Niswander, 1976; McPherson and Thompson, 1977; Love and Stream, 1978; Clemis and Sarno, 1980; Zito and Roberto, 1980) also report a biphasic reflex response, Love and Stream (1978) assert that the biphasic reflex is probably underreported in the normal population. Two studies (Zito and Roberto, 1980; Clemis and Sarno, 1980) found biphasic responses in both normal and impaired ears. Furthermore, Zito and Roberto (1980) contend that the use of an averaging computer to track the reflex response increases the accuracy of the response trace. Now that the averaging computer is being used more often in acoustic reflex studies, it seems reasonable to assume that biphasic responses will be more widely reported in the future.

Table 1 presents a summary of studies that report a biphasic response. It is important to note that the

biphasic reflex is not unanimously present in all of the subjects of these studies, but rather, usually occurs in a percentage of the subjects studied.

Table 1
Summary of Studies Reporting Biphasic Reflexes

YEAR	AUTHORS	N	NATURE OF RESPONSE
1966	Neergard & Rasmussen	"most of 28 Ss"	"most tracings begin with negative volume displacements"
1972	Hung & Dallos	2 of 4 Ss	"initial small negative change in impedance"
1976	Ruth & Niswander	5 of 6 Ss	"diphasic reponse"
1977	McPherson & Thompson	11 Ss	initial negative change in impedance
1978	Love & Stream	5 of 12 normal ears	"initial transitory negative component."
1980	Clemis & Sarno	47 normal 12 Meniere's 12 SNHL 16 retro-cochlear	biphasic response seen in the "majority" of Ss
1980	Zito & Roberto	10 normal Ss 10 SNHL Ss	At intensities higher than 100 dB HL, biphasic response; for stimuli lower than 100 dB HL, monophasic waveform.

There have been a number of mechanisms proposed to explain the occurrence of the biphasic acoustic reflex. One such mechanism includes the possibility of tensor tympani involvement in the acoustic reflex response. The possibility that the tensor tympani initiates, but that the stapedius is responsible for the mojor observed portion of the response may be dismissed for two reasons: 1) the tensor tympani is not acoustically activated in humans (Moller, 1974) and 2) animal studies indicate that tensor tympani latency is greater than stapedius latency (Galambos and Rupert, 1959; Mendelson, 1961; Djupesland, 1965; Love and Stream, 1978).

Two recent studies, Love and Stream (1978) and Mangham, et al. (1983), investigated the opposite possibility, that the stapedius initiates, but the tensor typmani is responsible for the major observed portion of the response. The two studies used different techniques and found different results. Love and Stream (1978) hypothesized that the stapedius muscle initiates the response, causing the negative portion of the biphasic reflex, whereas the tensor tympani (whose contraction they hypothesized to be triggered by stapedial tension) is responsible for the major, positive portion of the response. To test this hypothesis, they connected an impedance bridge to fresh cadaver ears, replaced the stapedius and tensor tympani muscles with wires, and observed the compliance changes that

resulted from normal directional pulling on the wires that were connected to the tensor tympani tendon and the stapes. Because a minimal pull on the tensor caused a large positive deflection and a pull on the stapedius caused a small negative deflection, they concluded that the biphasic acoustic reflex results from the independent action of the two muscles, with the stapedius initiating the response. They proposed the existence of a "proprioceptive feedback loop" located between the stapedius and the tensor, wherein tension on the stapedial tendon triggered contraction of the tensor tympani.

Mangham, et. al. (1983), tested the same hypothesis in monkeys, measuring acoustic reflexes before and after surgical interruption of stapedial and/or tensor tympani muscle function. The stapedius to tensor feedback loop described by Love and Stream (1978) was also surgically disrupted in some monkeys. Tensor tenotomy in three animals "had little effect on the general form of the acoustic reflex" (p.111), and both monophasic and biphasic reflexes occurred with the stapedius and/or tensor tympani intact. Based on these results, the authors conclude that the initial negativity of the biphasic reflex is not attributable to a single muscle, nor does the biphasic reflex require two muscles acting together. This study offers convincing new evidence to support contentions in the earlier literature (based on a number of human and animal experiments) that, except for startle responses, only the stapedius muscle

responds to sound (Wever and Lawrence, 1954).

If the tensor tympani is not the cause of the biphasic reflex, then what is? Four other mechanisms have been proposed. As described earlier, Hung and Dallos (1972) proposed that the stapedius muscle briefly relaxes prior to its contraction ("latency relaxation"), causing the initial negative dip in impedance during acoustic reflex contraction. Mangham, et.al. (1983) point out that the physiological mechanism to which Hung and Dallos (1972) attributed latency relaxation is a twitch response of isolated muscle which is too small "to account for any but the smallest decrease in impedance in a biphasic acoustic reflex" (p.106).

Another mechanism prosposed as an explanation for the biphasic impedance change at reflex onset is a change in the volume of the external auditory meatus caused by the reflex's inward or outward pull on the eardrum. However, Mangham, et.al. (1983) observed the biphasic decrease in impedance in a human subject with a prominent biphasic reflex and found it to be too large to be accounted for by a change in the volume of the external canal.

The third possible explanation of the biphasic reflex hypothesizes that the small increase in muscle tension at reflex onset may briefly reduce the impedance of the middle ear cavity, increasing sound transmission to the cochlea. Two observations conflict this hypothesis. First, increases in middle ear tension would increase the transmission of

high frequency tones, not the low frequency tones in the range used to measure acoustic impedance (Moller, 1983). Second, Borg (1972) found that biphasic impedance changes are not accompanied by biphasic changes in cochlear microphonics. This confirms the lack of improvement in sound transmission to the cochlea at reflex onset.

The current explanation of the biphasic reflex, and the one supported by the data supplied by the Mangham, et.al. (1983) study on live monkeys is called "cochlear uncoupling." In cochlear uncoupling, there is a transient disengagement of the cochlea from the middle ear at reflex onset, causing the brief decrease in impedance and in sound transmission manifested in the negative portion of the biphasic reflex. Uncoupling may occur at the incudostapedial joint or incudo-malleal joint (Mangham, et.al., 1983). Miller and Keith (1979) found variability between individuals in the action of the incudostapedial joint. This variability may account for the fact that only a percentage of subjects exhibit a biphasic reflex.

In summary, studies of the acoustic reflex using the impedance method usually report a percentage of subjects exhibiting a biphasic impedance change. Available data indicate that this biphasic response is not caused by the indiduval or synergistic action of the tympanic muscles, nor is it caused by transient stapedial relaxation, volume changes in the external auditory meatus or impedance mismatch of the middle and inner ear. The most plausible

explanation for the biphasic response is cochlear uncoupling, a transient uncoupling of the cochlea from the middle ear that occurs at reflex onset. Individual variability in the incudostapedial joint accounts for the lack of unanimity of response waveform in all subjects studied.

TECHNIQUES FOR OBSERVING THE ACOUSTIC REFLEX

DIRECT OBSERVATION

Direct observation of the activity of the tympanic muscles is difficult because of their small size and location within bony sheaths. According to Love and Stream (1978), direct visualization of the tympanic muscles requires significant alteration of the normal middle ear anatomy. Direct observation of the tendons of the two muscles is possible, however. In fact, the earliest observation of middle ear muscle activity were direct visualizations, both in experimental animals and in humans.

According to Wever and Lawrence (1954), the first observation of an acoustically initiated middle ear muscle response was made by Hensen in 1878. He opened the middle ear cavities of experimental dogs, inserted needles in the tendon of the tensor tympani and was able to see the needle move when sounds were delivered to the ear. Wever and Lawrence (1954) also reported on the early work of Kato (1913) who similarly observed sound initiated movements of the stapedial tendon in experimental cats and rabbits. The first reported observations of stapedial movement in humans

were made through a microscope in two ears with large perforations of the tympanic membrane. Jepsen (1963) credited Luscher (1929) with this observation. Perlman and Case (1939) also reported that Luscher demonstrated that reflex contractions could be elicited by acoustic stimulation of both the contralateral and ipsilateral ear. In 1933, according to Perlman and Case (1939), Lorente de No described the reflex arc of the stapedius in rabbits. In 1948, Kobrak used a technique called "optical perforation" to observe contractions of the stapedius in ears with perforations and normal ears as well (Jepsen, 1963).

ELECTROMYOGRAPHY

Most studies recording electromyographic (EMG) action potentials from electrodes placed in or near the middle ear muscles have been done in experimental animals. The earliest reported EMG studies with animals were done in the 1950s (Jepsen, 1963). While these early studies do give quantitative information about response latency, they do not yield quantitative information about the strength of contraction (Moller, 1974). More recently, Borg (1972), using EMG recordings in rabbits, has been able to make quantitative determinations regarding contractile strength.

Since EMG recordings require surgery to expose the muscles, data about muscle action potentials from the normal human ear are not available. However, EMG data has been recorded during ear operations. Perlman and Case (1939) had four surgical patients in whom the tendon of the stapedius

and the stapes were fully exposed for various reasons (chronic otitis media, radical mastoidectomy and perforation of the tympanic membrane). They were successful in detecting muscle action potentials from these patients, and were able to make quantitative statements regarding the latency of the human acoustic reflex response. According to Moller (1974), Salomon and Starr (1963) have obtained recordings from both the tensor tympani and the stapedius in patients before, during and after surgery.

DISPLACEMENT OF THE EARDRUM

There have been some attempts to record middle ear muscle activity by measuring the displacement of the eardrum or by measuring air pressure changes in the sealed external auditory meatus. Neergaard and Rasmussen (1966), for example, used electromanometry to monitor air pressure changes in the external auditory canal caused by the contraction of the stapedius. However, since the stapedius gives rise to only very small movement of the eardrum (Moller,1974), and the tensor tympani is not acoustically activated in humans (Wever and Lawrence, 1954), this is not a very efficient method to use for recording the action of the middle ear muscles.

COCHLEAR MICROPHONICS

The recording of changes in the electrical potentials of the cochlea, assumed to reflect pressure variations in the labyrinthine fluid caused by the muscle contraction (Wever and Lawrence, 1954) is another widely used method in animal

experiments. In 1937, Wiggers, experimenting on guinea pigs, found a drop in cochlear potentials at low frequencies during reflex contractions. In 1938, Wever and Bray, experimenting on cats, found a reduction in coclear potentials at 500 Hz and lower frequencies during reflex contraction. In 1942, Galambos, experimenting on bats found cochlear microphonic activity up to 55K Hz while the reflex was activated. After death, or under anesthesia, the cochlear microphonic response was increased, indicating that the reflex reduces transmission and that the relaxation of the reflex increases transmission (Moller, 1958).

ACOUSTIC IMPEDANCE: BASIC CONCEPTS

Metz's observation in 1946 that stapedial contraction changes the acoustic impedance measured at the tympanic membrane is generally considered to mark the beginning of an era of extensive research on the acoustic impedance of the ear. The impedance technique clearly dominates the literature since 1946, and its convenience for use in human subjects has added significantly to the clinical diagnosis auditory disorders. Quantification of the parameters of acoustic reflex latency has the potential for contributing to the diagnosis of retrocochlear abnormalities (Clemis and Sarno, 1981; Stach and Jerger, 1984; Stach, et al., 1984A) and providing information about the function of the middle ear muscles.

Acoustic Impedance (Z) is comprised of the complex interaction of mass reactance, stiffness reactance, and

resistance in the middle ear. Impedance is a concept that was introduced by engineers and is analogous to Resistance. Resistance refers to the opposition to the flow of current in an electrical ciruit. By analogy, acoustic resistance refers to the opposition to the flow of volume velocity in an acoustic medium.

Resistance in an electrical circuit is defined by the ratio of voltage over current, ie,

$$R = V/I$$

where R is Resistance, V is Voltage and I is Current. Clearly, the larger the resistance, the smaller the current for a given voltage. A remarkable aspect of almost all materials is that the resistance is virually a constant over a very large voltage and current range. This invariant property of resistance is often referred to as Ohm's Law (Levitt, 1985).

By analogy, if there is a reactive component in a circuit, eg, a capacitive reactance (stiffness reactance in an acoustic system), or an inductive reactance (corresponding to mass reactance in an acoustic system), this reactance will oppose the flow of current in much the same way as a resistance except that phase must be taken into account. Specifically, when an alternating current (AC) flows through a resistance, both the current and resistance are in the same phase. However, when an AC flows through an inductive reactance, the voltage leads the current by 90°. When an AC flows through a capacitive

reactance, the voltage lags by 90^{o}. In general, this relationship is represented by the formula

$$\tilde{Z} = \tilde{V}/\tilde{I}$$

where $\tilde{Z}$ is impedance. Note that the equation for impedance is analogous in form to that for resistance. The important difference is that vector quantities are used in impedance. $\tilde{Z}$, $\tilde{V}$ and $\tilde{I}$ are complex quantities and hence have both amplitude and phase.

The magnitude of the acoustic impedance is of particular interest and is given by the amplitude of $\tilde{Z}$, ie,

$$/ Z / = \sqrt{R^2 + \left(2\pi fM - \frac{K}{2\pi f}\right)^2}$$

where / Z / represents the amplitude of the impedance, R represents resistance, f respresents frequency, M respresents mass, $2\pi fM$ stands for mass reactance, K represents the coefficient of elasticity, and $\frac{K}{2\pi f}$ stands for elastic reactance. When mass reactance and elastic reactance are combined, the formula reduces to:

$$/ Z / = \sqrt{R^2 + X^2}$$

where X is the total reactance in the system and is equal to the sum of the mass and elastic reactances.

It may be noted that $\tilde{Z}$ is the vector sum of $\tilde{R}$ and $\tilde{X}$ and that both the amplitude and phase of Z can be obtained geometrically (Levitt, 1985).

IMPEDANCE AND THE ACOUSTIC REFLEX

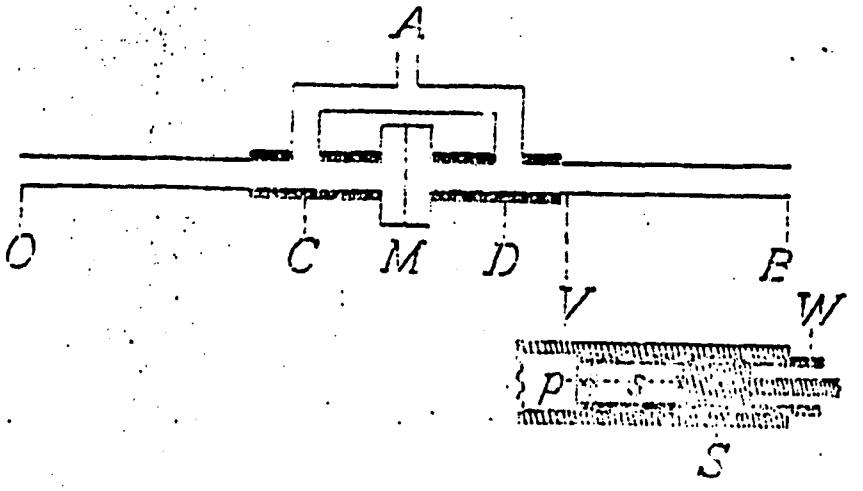
The effects of the acoustic reflex on the physiology of the middle ear and on the transmission of sound in the middle ear were discussed earlier in this chapter. In this section, the way in which the acoustic reflex affects the impedance of the middle ear will be explored.

As stated earlier, acoustic reflex contraction causes an increase in stiffness of the middle ear because of the change in tension on the ossicular chain. Metz (1946) was the first to observe that stapedial contraction changes the impedance measured at the tympanic membrane. Dallos (1964), Moller (1958, 1962 and 1964), and Feldman and Zwislocki (1965) further studied the effect of the reflex on the impedance of the ear. Their findings indicate that the major effect of stapedial contraction is an increase in reactance (compliance). Dallos (1964) found that resistance changes during contraction were not always in the same direction for all subjects, possibly because of a biphasic reflex in two of his four subjects. However, since in monitoring the impedance of the ear to detect acoustic reflex contraction an examiner is concerned only with a sudden change in impedance regardless of direction, this variability in the resistance component of impedance is not of major importance.

THE MECHANICAL IMPEDANCE BRIDGE

The diagram below appeared in Metz's 1951 article entitled, "Studies on the contraction of the tympanic

muscles as indicated by changes in the impedance of the ear." It illustrates the mechanical acoustic impedance bridge.



It consists of a tube (O,B), the ends of which are closed by the impedances to be compared. At the left side of the tube (O) is the subject's ear. At the right side of the tube (B), a variable acoustic impedance is shown on an enlarged scale. A membrane (T) separates tube (O,B) into two tubes of equeal diameter and length. The two tubes are connected through a Y tube that bridges the membrane. The top of the Y tube (A) goes to the examiner's ear (or may be attached to a microphone). When the impedances at O and B are equal, there will be a pressure node (null) at A because sound waves reaching it are of equal amplitude and are 180 degrees out of phase. Impedance measurements are made by reading the iariable impedance standard after the null is achieved.

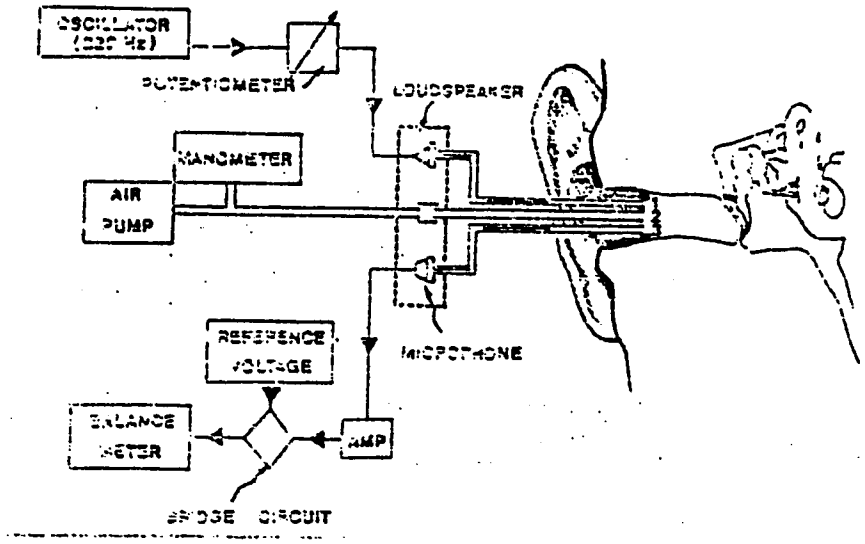
A change in tension on the tympanic membrane, such as that caused by the acoustic reflex, will upset the balance of the system and the tone will be heard again at A. Metz (1951) explains that by using this method, the examiner will "hear" the onset of contraction just as previous investigators saw it (p.399).

There were shortcomings in the original instrumentation. It was large and clumsy, did not allow independent resistance and reactance measurements, and did not exclude the effect of the variable volume of air in the ear canal (Zwislocki, 1982). Modifications by Zwislocki (1963) corrected for these deficiencies. For example, with

the Zwislocki bridge, the volume of the ear canal is determined by injecting alcohol into the ear canal between the speculum of the impedance bridge and the tympanic membrane. An equal volume is then added to the volume of tube B, allowing for the direct measurement of impedance and requiring no further calculations (Zwislocki, 1982). Independent compliance and resistance controls were also built into the Zwislocki bridge, allowing independent readings of those values. However, according to Zwislocki (1982), there were many flaws in the commercial version of the bridge, and alcohol injection into the ear canal was found to be objectionable. Therefore, the mechanical impedance bridge has limited use.

ELECTROACOUSTIC IMPEDANCE BRIDGE

The system now in routine clinical use, and the one used in this study is the electroacoustic impedance bridge. The probe tube set up is shown in the schematic drawing below.



It consists of two parallel narrow tubes, one delivering the test sound (probe tone) to the ear canal and the other leading the reflected tone out of the ear canal to a microphone. According to Zwislocki (1982) "when the acoustic impedances of the tubes alone or in combination with the acoustic impedances of the electroacoustic transducers attached to them are much higher than the acoustic impedance of the ear, the sound pressure generated in the ear canal is directly proportional to the acoustic impedance of the ear" p.6). Thus, the electroacoustic bridge doesn't provide direct information about the acoustic impedance at the eardrum, but measures a combination of that impedance and the impedance of the air enclosed in the ear canal.

For detection of the acoustic reflex, we are not interested in the absolute impedance of the ear but rather in the relative impedance change that occurs with reflex contraction. Activation of the reflex changes the impedance of the system by causing an unbalancing of the bridge,which is visually detectable to the examiner through the deflection of a meter needle.

It should be noted that while the electroacoustic impedance bridge supplies adaquate information regarding the presence or absence of a reflex contraction, there are concerns regarding the timing of the bridge's response for the measurement of acoustic reflex latency. These concerns will be addressed in a later section of this chapter.

THE USE OF COMPUTER AVERAGING

In measuring temporal aspects of the acoustic reflex response, it is important that a baseline of pre-reflex activity be established and that any and all changes from that baseline be clearly observable. One problem encountered in accomplishing such an observation is the poor signal-to-noise ratio of available impedance bridges. In a single reflex trace, particularly near acoustic reflex threshold, the response (signal) can be obscured by background noise. In this case, noise does not only mean acoustic noise, but also refers to any undesired interference from electrical or mechanical disturbances. Recently, the use of computer averaging techniques has contributed greatly to accurate response observations because of the improvement in the signal-to-noise ratio that such averagers afford. Silman and Gelfand (1982) and others (Norris, et.al., 1974; Ruth and Niswander, 1976; Clemis and Sarno, 1980; Mangham, et.al., 1980; Stach and Jerger, 1984) suggest that averaging eight to sixteen responses is sufficient to achieve a clear response trace. The major steps in computer averaging are reviewed next.

According to Berlin (1978) and Berlin and Dobie (1979), the most important principle of computer averaging is that stimulus onset and computer onset be time locked. The input to the computer averager is both target signal (in this case the signal from the impedance bridge recorder output) and noise. We must assume that the noise is random, and that

its amplitude varies with time in a Gaussian distribution, while the signal is not random, and that its amplitude varies in a regular fashion. Each subsequent sweep of the computer adds linearly to the one that precedes it. Thus, the signal value will increase in the computer's memory while the noise value will approach zero. According to Berlin (1978), the improvement in signal-to-noise ratio is equal to the square root of the number of sweeps. Thus, averaging sixteen responses yields a four to one improvement in signal-to-noise ratio.

A computer averaged waveform is outlined as follows: A computer sweep or scan through a set of bins or addresses is initiated by a trigger that coincides with the onset of the stimulus. The scanner dwells at each address for a specified period of time. While dwelling at each address, the scanner digitizes any voltage at that address and, according to Berlin (1978), "translates those voltages into a vertical height proportional to the voltage coming from the source" (p. 118). Knowing how long the scanner dwells at each address allows the conversion of dwell time into elapsed time from the onset of the stimulus. For example, if the dwell time of a scanner is one msec and if there is a change in the waveform at the twentieth address, we know that it has taken twenty msec for the change to occur. Thus, address number x dwell time = elapsed time (Berlin, 1978).

In summary, computer averaging is a relatively recent

addition to the instrumentation used to quantify the morphological and temporal aspects of the acoustic reflex. Its major contribution is the achievement of improved signal-to-noise ratios that render the reflex waveform clearer and easier to visualize. This allows the observer to decide where in the waveform significant changes occur with greater confidence.

CONCERNS REGARDING PRECISION OF MEASUREMENT

TIME DELAY AND TIME CONSTANT OF IMMITTANCE DEVICES

Earlier in this chapter, the principles of impedance, immittance and the electroacoustic impedance bridge were described. Concerns regarding the timing of the reflex measuring device's response for the measurement of acoustic reflex latency were mentioned; in this section, the time delay and time constant of immittance devices as possible sources of artifact in the reflex waveform will be explored.

In the earliest investigations regarding acoustic reflex latency (Moller, 1951, 1958, 1961; Dallos, 1964;), the possibility that artifact from the measuring device might contaminate the reflex waveform was not even mentioned. One of the first published discussions of the effect of the temporal characteristics of electroacoustic impedance bridges on observed reflex measures appears as an appendix in Ruth and Niswander's (1976) study. They refer to an earlier investigation (Sundby, Flottorp and Djupesland, 1971) whose technique was the same as theirs, but whose

results were not widely disseminated. Prior to that, as stated by Lilly (1984), "many clinicians and investigators...failed to consider adequately the effects of instrument characteristics of acoustic reflex measurements" (p. 3).

Ruth and Niswander (1976) describe two pertinent temporal aspects of immittance measuring devices: 1) equipment latency and 2) filter time constant. Equipment latency is defined as the time delay between an acoustic event and the first registration of that event by the measuring device. The filter time constant is a function of the filter used to remove noise from the reflected probe tone, and, according to Ruth and Niswander (1976), is calculated by taking the reciprocal of the filter bandwidth. Lilly (1984) defines the leading edge time constant as "the time from the onset of the test pulse to the point at which the leading edge of the waveform reaches 63.2% of its maximum value" (p. 13).

The effect of these temporal aspects is as follows: if the reflex change occurs during the period of time that corresponds to equipment latency, then the change in immittance will not be registered without a time delay. Thus equipment latency should be subtracted from all latency determinations. The filter time constant governs the most rapid reflex change that the bridge can track. If the reflex occurs faster than the time constant, the bridge will not track the change accurately. However, if reflex

rise time is longer than the time constant of the bridge, the change will be tracked accurately (Ruth and Niswander, 1976).

Beginning with Sundby, Flottorp and Djupesland (1971), a number of investigators have used a variety of techniques to determine the time delay of commercially available immittance devices. Lilly (1984) provides a comprehensive description of the available data and also extrapolates the time constant of many bridges from the studies he reviews.

According to Ruth and Niswander (1976), Sundby, et al. (1971) investigated the temporal characteristics of the Madsen ZO-70 impedance bridge by setting up an acoustic interaction in a sealed cavity and noting how quickly the bridge registered the effects of the interaction. They found that the latency of their impedance bridge was 20 msec. Ruth and Niswander (1976) used a similar techniques. They sealed the probe of a Madsen ZO-70 into a rigid 2-3 cc plastic cylinder which was sealed at one end by a thin rubber membrane. An earphone was fastened to the membrane, and electrical pulses delivered to the earphone were registered as "impedance-like" deflections by the impedance bridge. They also found a delay of 20 msec.

Morgan, Gilman and Dirks (1977) also evaluated the response time of the Madsen ZO-70. They designed a special calibration cylinder with a volume of 0.83 cubic centimeters. There is a rubber diaphragm at one end. An electromagnetic acuator plunger, capable of activation by a

range of electrical signals, was put in contact with the diaphragm. Electrical pulses to the plunger caused acoustical pulses in the the cylinder. The electrical and acoustical pulses were found to occur within 2 msec of each other, and were also found to have identical risetimes. Using this technique, when the probe of the bridge was sealed in the calibration cylinder, a 70 msec delay in the response of the Madsen ZO-70 was found.

Margolis and Gilman (1977) evaluated the response delay of the Grason-Stadler 1720B electroacoustic immittance meter by effecting changes in the amplitude of the probe tone while the probe tip was sealed in a 2 cc cavity. By wiring a variable resistor into the probe tone delivery circuit, they were able to produce a rapid reduction in the amplitude of the probe tone within the calibration cavity. The microphone in the probe tip picked up the SPL change in the cavity, and the immittance meter responded as if an acoustic reflex contraction had occurred. In their 1977 article, Margolis and Gilman report that the change in the immittance meter's output voltage in response to the change in the probe SPL "began essentially instantaneously..." However, Lilly (1984) reports personal communication from Margolis that indicated that measurements on the original oscilloscope photograph showed a 14 msec response delay for the Grason-Stadler 1720B. In addition, Margolis told him of an initial overshoot of approximately 30%; a stable output was not achieved until after approximately 320 msec.

Popelka and Dubno (1978) devised a technique for temporal measurements of immittance devices which does not require an external signal, access to the circuitry of the instrument being measured, or a special calibration cavity. In addition, the procedure uses equipment that is widely available in many clinics and laboratories, and is normally used to measure hearing aid characteristics.

The probe unit of the immittance device is connected to a standard 2 cc coupler and microphone. The microphone output is fed through a sound level meter to an electronic switch, an amplifier and a hearing aid receiver. The output of the hearing aid receiver is fed back to the 2 cc coupler via the tubing on the probe unit normally used to vary the air pressure during tympanometry. Thus, by activating the electronic switch, the probe tone generated by the immittance device is amplified and fed back to the coupler, simulating a change in the impedance within the coupler.

Using this procedure, the temporal characteristics of both the Madsen ZO-70 and the Grason-Stadler 1720B were measured. For the Madsen ZO-70, a 20 msec latency was found, in agreement with Sundby, et al., 1971 and Ruth and Niswander, 1976. The Grason-Stadler 1720B exhibited an onset with "essentially no delay," but also exhibited "substantial overshoot," requiring almost 400 msec to track accurately. These findings are in agreement with the published findings of Margolis and Gilman (1977).

Wilson, Shanks, Jones and Danielson (1982) designed a

calibration cavity to evaluate the Grason-Stadler 1723 and found a delay of approximately 34 msec.

It is obvious that there is not yet a standard method of measuring temporal aspects of immittance devices. A recommended procedure for measuring the temporal characteristics of aural acoustic immittance instruments is currently being developed by the American National Standards Institute (ANSI, working group S3-60). The instrumentation designed by Popelka and Dubno (1978) is being considered for this standard. The studies reviewed next have either used this instrumentation or developed similar instrumentation based on the same principles.

Popelka (1979) evaluated the temporal characteristics of the Grason-Stadler 1723. He found a time delay of approximately 100 msec. This value does not agree with the findings of Wilson, et al. (1982) for the same device using a different technique. Silman and Gelfand (1982) found that the Madsen ZO-73 had a delay of 29 msec. Lilly (1984) evaluated the new Amplaid 702 and found a 16 msec delay for the "scope" output (which was designed to measure acoustic reflex latency) and a 29 msec delay for the "recorder" output.

Table 2 summarizes the initial time delays and filter time constants of the many immittance measuring devices and studies reviewed in this section. In all cases, the time delays come from the results published in each study; were available, the filter time constant has been calculated by

Lilly (1984) either through direct examination of the data or personal communication with the author.

Table 2
Summary of Data re: Response Time and Time Constants of Available Immittance Devices

MANUFACTURER AND MODEL	TIME DELAY	TIME CONSTANT	METHOD	SOURCE
Madsen ZO-70	20 msec		acoustic interaction in a sealed cavity	Sundby, et al., 1971
Madsen ZO-70	20 msec		acoustic interaction in a sealed cavity	Ruth & Niswander 1976
Madsen ZO-70	70 msec	54 msec	special calibration cavity	Morgan, et al., 1977
Madsen ZO-70	20 msec		acoustic interaction in a sealed cavity	Popelka & Dubno, 1978
Grason-Stadler 1720B	"essentially no	35 msec	acoustic interaction in a sealed cavity	Margolis & Gilman
Grason-Stadler 1720B	"essentially no delay"		acoustic interaction in a sealed cavity	Popelka & Dubno, 1978
Grason-Stadler 1723	100 msec	90 msec	acoustic interaction in a sealed cavity	Popelka, 1979
Grason-Stadler 1723	34 msec	85 msec	special calibration cylinder	Wilson, et al., 1982
Madsen ZO-73	22 msec	26 msec	acoustic interaction in a sealed cavity with an added external signal	Silman & Gelfand 1982
Amplaid 702 "Scope"	16 mse c	33 msec	acoustic interaction in a sealed cavity	Lilly, 1984
Amplaid 702 "Recorder"	29 msec	78 msec	acoustic interaction in a sealed cavity	Lilly, 1984

Examination of Table 2 reveals that different procedures can yield different results, even for the same instrument. For example, the Madsen ZO-70 was evaluated in four separate studies, three of which found similar results. The fourth study used a different technique, and found a longer time delay than the other three. Bosth studies of the GS 1720B found "essentially no delay in response even though they used different techniques. The two studies of the GS 1723 found different values using different techniques. Clearly, until a standard equipment array and procedure for determining the temporal characteristics of aural acoustic immittance measuring devices is established, it is incumbent upon every laboratory and clinic to determine the equipment latency and filter time constant of the immittance devices in use.

In summary, the temporal characteristics of immittance measuring devices are of interest when the equipment is used to evaluate the temporal characteristics of the acoustic reflex. If artifact from the measuring device is found, it should be either corrected or accounted for. At present, there is no one standard procedure for determining the response characteristics of immittance devices, but a number of studies have found that commercially available instruments vary in their response time from "essentially no delay" to 100 msec. The instrumentation and procedure used to determine the response time and filter time constant of the immittance device used in this research is described in

Chapter 2 of this dissertation.

ACOUSTIC REFLEX THRESHOLD

Acoustic reflex threshold (ART) may be defined as the minimal sound intensity which causes a reflex contraction of the stapedius muscle. It has been asserted (Wever & Lawrence, 1954) that we can observe reflex contractions in our own ears because "a sudden sharp, unexpected sound produces kinesthetic and pressure sensations referred to the ears..."(p. 180). However, most observers (and all researchers) rely on some form of indirect, objective measurement. The presence of reflex contraction is determined by the technique of measurement. For example, using the immittance technique, a reflex contraction causes an immittance change which may be visually detected as a deflection of a meter needle (a typical clinical technique) or as a change on a strip chart recording or computer averaged oscilloscopic trace (a typical research technique). In the early animal and surgical experiments, exposure of the middle ear cavity for direct observation of the stapedial tendon allowed for direct visual observation of stapedial contraction and its effects on the ossicular chain. In EMG studies, stapedial contraction is noted in the recording of action potentials from electrodes placed in or near the middle ear muscles. In studies using cochlear microphonics, ART is defined as the lowest stimulus level that causes a change in the electrical potentials recorded from the cochlea.

The lowest level of stimulus that results in a measurable contraction (by whichever measurement technique is used) is the level of the acoustic reflex threshold. The bulk of the research uses the impedance or immittance method for detecting a reflex contraction. As noted earlier in this chapter, the immittance technique is both noninvasive and convenient for use in humans with normal and abnormal hearing. It is currently the most widely used clinical and research method of measuring the status of the middle ear, along with comparisons of air conduction and bone conduction thresholds.

The level of the ART is typically expressed in one of three scales: the dB SPL scale (re: .0002 dynes/cm), the dB HL scale (re: ANSI, 1969), or the dB SL scale (re: the individual's threshold of hearing). An example, adapted from Silman and Gelfand (1982) will illustrate how this works. If a 1000 Hz stimulus causes a reflex contraction at 97 dB SPL, the ART is 97 dB SPL. The same ART may also be expressed as 90 dB HL because the ANSI, 1969 correction factor for audiometric zero is 7 dB. If the person being tested has a 10 dB HL hearing threshold at 1000 Hz, then the same threshold may be expressed as 80 dB HL.

Among the many studies of ART and the factors affecting it, there does not seem to be a strong consensus regarding a preferred scale for ART levels (SPL, HL or SL). Rather, each study specifies the scale used. Comparison between studies therefore sometimes requires the subtraction of the ANSI,

1969 correction for average normal hearing.

THE EFFECT OF FREQUENCY ON ACOUSTIC REFLEX THRESHOLD

In an early study Jepsen (1951) used the impedance technique to measure the acoustic reflex thresholds of 98 normal ears. The results indicated that the average ART is about 80 dB SL, with no significant differences between frequencies for stimuli form 250 to 4000 Hz. Metz (1952), using the impedance method, found that thresholds varied between 70 and 90 dB SL. Jepsen (1963) later found that ARTs did vary with frequency, with the best sensitivity at 1000 and 2000 Hz (ARTs at approximately 78 dB SL). At 250, 500 and 4000 Hz, the average ART was 85 dB SL. Deutsch (1968) used the Zwislocki mechanical impedance bridge to determine the ART for 250 Hz, 2000 Hz, 4000 Hz, 2000 Hz narrowband noise, 4000 Hz narrowband noise and white noise. The precise bandwidths of the narrowband noise is not specified. Out of thirty subjects, only four had acoustic reflex responses at 250 Hz and only 15 had responses at 4000 Hz. All subjects responded to the 2000 Hz and noise stimuli. The average ART at 2000 Hz was 82 dB SL. The responses to the noise stimuli were at the lower sensation level of 62 dB. Peterson & Liden (1972) found that the average ART for pure tones (averaged across frequencies was between 90 and 90 dB HTL; noise stimuli were approximately 15 dB more efficient in eliciting the reflex, with the average ART for noise at 75 dB SPL. Margolis and Popelka (1975) found the following mean ARTs in dB SPL:

Table 3
Mean ARTs for Normal Ss (from Margolis and Popelka, 1975)

SIGNAL	ART in dB SPL
250 Hz	99.7
500 Hz	97.65
1000 Hz	91.05
2000 Hz	90.25
4000 Hz	91.50
WBN	77.20
NBN	97.20

These data also indicate that the greatest sensitivity is a 1000 and 2000 Hz, with wide band noise about 13 dB more efficient in eliciting the reflex.

In summary, the human acoustic reflex appears to be most sensitive to the frequencies to which average normal hearing is the most sensitive (1000 and 2000 Hz). At those frequencies, the reflex occurs at about 80 dB above the threshold of hearing. At higher and lower frequencies (500 and 4000 Hz), the reflex requires a sound intensity at about 85 dB above the threshold of hearing to be activated. Broadband noise is even more efficient that pure tones in eliciting the reflex; the relationship between signal bandwidth and ART will be discussed later in this chapter.

THE EFFECT OF INTENSITY ON ACOUSTIC REFLEX THRESHOLD

From the data reported in the previous section, it appears that the reflex is activated at any given frequency once the intensity reaching the cochlea reaches a certain level. Acording to Dallos (1973), once the level reaching the cochlea exceeds threshold level, the strength of contraction increases "roughly in proportion with the

magnitude of the stimulus" (p. 489). The response increases over a range of about 30 dB and then levels off. Thus, as stated by Dallos (1973), "the acoustic reflex is active over a relatively narrow dynamic range between its threshold and level of saturation" (p. 479).

CONTRALATERAL, IPSILATERAL AND BILATERAL SENSITIVITY

As noted earlier, Luscher (1929) is credited with the first observation that bilateralreflex contractions could be elicited by acoustic stimulation of either ear. Moller (1962) found that in humans, ipsilateral sensitivity is somewhat greater than contralateral sensitivity, the ipsilateral ART being about 5 dB lower than the contralaterl ART at 500 Hz. In addition, the impedance change for contralateral stimulation is smaller than for ipsilateral stimulation, varying from person to person from 2 to 14 dB, and is different for different frequencies. A more recent study (Laukli and Mair, 1980), however, found no significant differences between the contralateral and ipsilateral reflexes of forty subjects. Bilateral stimulation yielded ARTs that were about 3 dB more sensitive than ipsilateral stimulation (Moller, 1962).

Moller (1983) noted that since the natural stimulation of the ears is bilateral, testing the acoustic reflex via contralateral stimulation is "unfortunate." The development of immittance devices (the Madsen ZO-72) and American Electromedics 83) that can deliver ipsilateral as well as contralateral stimulation has led to more frequent use of

ipsilateral ART measurements specifically for the diagnosis of retrocochlear pathology (Greisen and Rasmussen, 1970; Jerger and Jerger, 1975). However, while the measurement of the ipsilateral reflex has great potential in the area of differential diagnosis, technical problems with the instrumentation (Wilbur, 1976), and the large variablility between people, between frequencies and between studies requires that caution be used in generalizing the results of ipsilateral measures. Moreover, ipsilateral latency measures have not yet been recorded, probably because the need to filter the ingoing signal out from the reflected probe signal in the ipsilateral ear would add to the time constant of the immittance device.

THE EFFECT OF SIGNAL BANDWIDTH ON ON ACOUSTIC REFLEX THRESHOLD

As previously discussed, early studies of the acoustic reflex threshold noted that broad band noise was a more efficient eliciter of the reflex response than pure tones. This section reviews subsequent research into the details of this noise - tone difference. As will be described, the findings indicate that there is a relationship between signal bandwidth and acoustic reflex threshold. Specifically, there is a critical band for acoustic reflex threshold, beyond which, ARTs decrease in level.

The critical band concept emerged from a number of psychophysical studies (Sharf, 1959, 1961) and is considered a measure of the frequency selectivity of the ear (Zwicker,

1975). The critical band is that bandwidth at which subjective responses (such as loudness judgements) abruptly begin to change as the band of noise or the spacing of complex tones increases, while the overall SPL of the stimulus remains constant (Flottorp, Djupesland and Winther, 1971). That is, the loudness of a band of noise will remain constant as the bandwidth increases up to a critical point: as the bandwidth increases beyond that critical point, the perception of loudness increases. The critical band for masking is that bandwidth that contains sound power equal to that of a pure tone in the center of (and just masked by) the band of noise (Yost and Nielson, 1977). That is, a tone in the center of a critical band of noise whose overall SPL is equal to the SPL of the noise will be inaudible (or just masked). As the bandwidth of the noise increases, the tone becomes audible.

The psychophysical critical bandwidth varies with frequency (Sharf, 1966). At low frequencies it is constant, but above 500 Hz it increases in bandwidth (Djupesland and Zwislocki, 1973).

According to Djupesland (1976), the location of the critical band mechanism within the auditory system has not been clarified. Nor do we yet fully understand how the ear discriminates between sound energy inside and outside the critical band. However, Djupesland (1976) reported that the results of several experiments indicate a peripheral location, perhaps within the cochlea, for the critical band

mechanism. Certainly, if a critical band exists for the acoustic reflex, the mechanism must exist at a level lower than the pons, for this is the highest point in the reflex arc.

Over the last decade, there has been a good deal of clinical interest in the difference in reflex sensitivity to pure tones and noise. A number of studies have demonstrated that in subjects with sensorineural hearing loss, the difference between ARTs for noise and pure tones is much smaller than in subjects with normal hearing, and that the amount of the difference is related to hearing loss. Furthermore, a number of strategies have been developed to predict and/or detect hearing loss based on the noise - tone relationship in ARTs (Niemeyer and Sesterhenn, 1974; Jerger, Burney, Mauldin and Crump,1974; Popelka, Margolis, and Wiley, 1976; Margolis and Fox, 1977; Silman and Gelfand, 1979).

Two important relationships have emerged from these studies: 1) ARTs for tones remain constant for mild hearing losses and increase for losses above about 50 dB and 2) the noise - tone difference is large for normally hearing subjects and also for patients with hearing losses worse than about 80 dB. For patients with hearing losses averaging between about 40 and 75 dB, the noise - tone difference in ART is relatively small. For a more complete review of the relationship between hearing loss, the ART and the bandwidth of reflex activating signals, see Popelka (1981).

STUDIES OF ART AND BANDWIDTH IN NORMAL EARS

Using band-limited random noise and two cascaded filters, Flottorp, et al. (1971) found that reflex thresholds in 16 normal subjects were constant for bandwidths less than a specific value which they called the "critical bandwidth." Then, as bandwidth was increased, the reflex threshold decreased by about 3 - 6 dB per octave. The difference between reflex thresholds for the tonal signal and the very wide bandwidth signal was 28 dB. Djupesland and Zwislocki (1973) investigated the reflex critical band using two tone complexes of variable frequency separation. Their results confirmed the earlier findings of Flottorp, et al. (1971), but with different threshold differences. They found that outside the critical band (whose width equalled the center frequency of the complex in this study), the acoustic reflex threshold was 10 dB lower than it was to signals within the critical band. They attribute this 10 dB difference to the relationship between the stapedius response and loudness; they report that the same results were found in psychoacoustic experiments on equality of loudness.

Popelka, et al. (1976) suspected that the discrepant tone - noise differences (10 dB vs. 28 dB) found by Djupesland and Zwislocki (1973) and Flottorp, et al. (1971) were attributable to the different spectral densities of the activating signals (two-tone complexes vs. filtered noise). They varied the spectral density of the activating signal by

increasing the number of components in their reflex activating signal until reflex thresholds were similar to those obtained using bandpass filtered noise. They found that "tonal complexes with five to nine components equally spaced in log frequency produced reflex thresholds as low as filtered noise at 1000 Hz with a 3062 Hz bandwidth" (p.154). A second experiment reported in same study was conducted using 61 component tonal complexes whose highest and lowest frequency components were consecutively removed to determine the effect of bandwidth on acoustic reflex threshold. The results indicated a decrease in acoustic reflex threshold as the bandwidth of the signal increased. The width of the critical band was in "reasonable agreement" with the previously reported critical bandwidths but the decrease was found to be a curvilinear rather than a straight line function because of the accumulation of more data points around the value of the critical band.

The most recent study of the effect of bandwidth on acoustic reflex threshold (Margolis, et al., 1980) used low pass (fixed lower cutoff = 100 Hz) and high pass (fixed upper cutoff = 6000 Hz computer generated noise. As the upper cutoff of the low pass noise was reduced from 6000 to 707 Hz, reflex thresholds increased at a rate of about 4 dB per octave. This is in good agreement with the findings of Flottorp, et al. (1971). As the lower cutoff of the high pass noise was increase from 100 to 707 Hz, no change in acoustic reflex threshold was observed. Increases above 707

Hz again resulted in the 4 dB per octave increase in ART. These data indicate that the energy in the region below 707 Hz of a wide band noise does not significantly contribute to the reflex whereas the energy between 707 and 6000 Hz contributes equally.

In summary, it has been clearly established that the acoustic reflex is sensitive to differences in the bandwidth of activating signals. While this suggests a critical band for the acoustic reflex beyond which ARTs decrease, the critical bands suggested are substantially wider that psychoacoustic critical bands. The effect of bandwidth on the latency of the response at threshold and suprathreshold levels has not yet been established.

THE EFFECT OF STIMULUS DURATION ON ACOUSTIC REFLEX THRESHOLD

Moller (1983) noted that the sensitivity of the acoustic reflex is affected by stimulus duration the in following manner: as stimulus duration decreases below approximately 200 msec, sensitivity decreases; for sounds longer than 200 msec, reflex sensitivity is independent of duration. Jerger, Mauldin and Lewis (1977) report that the change in ART for a tenfold change in stimulus duration is up to 20 dB. In addition, when elicited by short duration stimuli, the maximal, or saturation, value of the reflex response is lower than it is when the eliciting stimulus is longer than

200 msec in duration (Moller, 1983).

This phenomenon is known as "temporal summation" and is similar to psychoacoustic temporal summation at threshold or of loudness, although greater intensity changes are needed for ARTs than for loudness judgements. Richards and Goodman (1977) found a frequency dependency in the function describing temporal summation at ART with great summation at the higher frequencies. These researchers, as well as Silman and Gelfand (1982) note that there is large inter- and intra-subject variability in temporal summation for the ART, both within and between studies. Silman and Gelfand (1982) therefore advise "extreme caution in the clinical application of temporal summation of the acoustic reflex" (p. 116).

For long duration signals (>10 seconds) the magnitude of the acoustic reflex (as measured by a change in impedance) generally decreases as stimulus duration increases. This phenomenon is known as acoustic reflex adaptation (or decay). Metz (1951) noted that the slow and gradual relaxation of the muscles during long duration stimulation was not attributable to muscle fatigue since it was possible to increase contraction to the original strength by stimulating with a different frequency whil the first one was still on. Rather, the relaxation noted was attributed to adaptation in the afferent portion of the reflex arc.

In the normal ear, when the stimulus is at levels at

least 10 dB above ART, there is no decay in the magnitude of the reflex response at 500 and 1000 Hz, even after three minutes of stimulation; at 2000 Hz, there is some decay after about 14 seconds and at 4000 Hz, there is some decay after about 9 seconds (Habener and Snyder, 1974, Anderson, et al., 1969). Patients with retrocochlear pathology exhibit acoustic reflex amplitude decay of 50% or more within 10 seconds for 500 Hz and/or 1000 Hz stimuli (Anderson, et al., 1969). Since patients with Meniere's disease will also exhibit some decay within 10 seconds for 500 Hz and 1000 Hz stimuli, Olsen, Stach and Kurdziel (1981) recommended restricting the test duration to 5 seconds, since patients with retrocochlear pathology exhibit decay within 3 seconds of signal onset.

In summary, when stimulated by signals with durations shorter than 200 msec, the ART exhibits temporal summation, with each ten fold decrease in duration increasing the ART by up to 20 dB. When stimulated by signals with long durations, there is some adaptation of acoustic reflex magnitude in the normal ear at high frequencies only, while ears with retrocochlear pathology tend to exhibit rapid adaptation both at high and low frequencies. This phenomenon of acoustic reflex decay is clinically useful for the differential diagnosis of retrocochlear pathology.

PRECISION & RELIABILITY OF ACOUSTIC REFLEX THRESHOLD

Moller (1962) noted that acoustic reflex responses near reflex threshold have a larger variation than those above

threshold, a finding confirmed in animal experiments. He concluded that the actual threshold could therefore not be determined with certainty and suggested that the intensity level needed to produce a 10% impedance change was much more reproducible than the intensity needed to produce a just detectable impedance change and should be defined as "threshold." This recommendation was reinforced by Borg (1972) and Djupesland (1980). Moller (1974, 1983) also stated that a threshold curve for the acoustic reflex is difficult and impractical to obtain because of the great variablility of reflex responses to threshold level stimuli. Nevertheless, the measurement of the ART defined as the minimum level necessary to produce a detectable immittance change persists, both in the laboratory and in the clinic.

Popelka (1981) contended that the variablility in ARTs reported in Moller's early studies are related to "procedural variables" (p.11). He demonstrated that two such variables, stimulus increment size and the sensitivity of the response measurement system cause variablility in the ART which is unrelated to the reflex itself. He also compared the results of more recent studies regarding the ART in normal subjects (Flottorp, et al., 1971; Peterson and Liden, 1972; Margolis and Popelka, 1976; and others) and reported good agreement across studies and low variablility within each study. His presumption is that these more recent studies employed small increment size and highly sensitive measurement systems, and that this accounts for the degree

of consistency not found in the earlier studies.

Other recent studies (Gelfand and Piper, 1984; Gelfand, Piper and Silman, 1983; Silman and Gelfand, 1981) however, indicate that the intersubject variability of ARTS in normal and cochlear impaired ears is high. Specifically, even though the relationship between ART and hearing loss is specifiable in the form of regression equations, these equations account for less than 50% of the variance in ART levels in normal and cochlear impaired ears. Gelfand and Piper (1984) suggest that audiologists use upper and lower acceptable criteria levels (such as 90th and 10th percentiles) based on the distributions of ARTs as a function of hearing loss in order to form diagnostic impressions.

In summary, there is some disagreement in the literature regarding the reliability of acoustic reflex threshold, both in normal and impaired ears. Some studies indicate high variability while others indicate low variability. Until the question is settled, it seems to be incumbent upon each investigator to replicate the measurements of ARTs in separate sessions within each experiment and to compare those measurements to determine if statistically significant differences exist.

SUBJECT VARIABLES AFFECTING ART

AGE

Jepsen (1963) and Jerger, et al. (1974, 1978) and many other have studied the way in which ART varies with age.

Jerger, et al. (1974) found that ARTs to tonal stimuli decline from about 90 dB HL in the 0 to 9 year age group to about 84 dB in the 20 to 29 year age group. There are only small differences between the 20 to 29 year age group and the older groups, but the oldest group studied was only 50 to 59 year. There was no age effect when the reflex eliciting signal is broad band noise. Margolis and Popelka (1975) found that the pure tone ARTs for infants from birth to six weeks were abot 4 to 7 dB higher than those of normal hearing adults; for noise, the ARTs of the two groups were the same. Jerger, et al. (1974) found a systematic increase in the number of children exhibiting the acoustic reflex as age increase from 0 to six years.

With regard to ARTs in subject older than 50 years, Silman (1979) reported reflex thresholds for two groups of normally hearing subjects, one aged 21 to 36 years and the other aged 60 to 76 years. Normal hearing was defined as hearing at 20 dB HL or less from 250 to 8000 Hz. This controlled for the appearance of any age related changes that were really reflections of changes in hearing sensitivity. ARTs for 500, 1000 and 2000 Hz were similar in both groups, but the elderly group had higher thresholds for broad band noise. Handler and Margolis (1977) had reported similar findings in eight normal hearing subjects aged 57 to 66 years. Their subjects had slightly higher ARTs for 4000 Hz and high-pass noise as well as for broad band noise.

In summary, while ARTs in newborns are higher than

those in adults, within the first year of life, they approach adult levels. In normal hearing young adults and normal hearing elderly subjects, the changes in ARTs for 500 Hz to 2000 Hz that occur with age are apparently small, if present at all. ARTs for 4000 Hz and noise stimuli do, however, increase in elderly subjects (Popelka, 1981; Silman, 1979).

DRUGS AND ALCOHOL

Moller (1983) reported that drugs such as ethanol and barbituates decrease the sensitivity of the acoustic reflex by an amount proportional to the level of the substance in the blood. In addition, responses to the same sound intensity is smaller after alcohol ingestion. Specifically, a blood alcohol level of .05% increases the ART by 2 dB while a blood alcohol level of .14% increases the threshold by 14 dB. These results were repeated in animal experiments.

Recently, Shen (1983) reported two cases of alcoholic neuropathy associated with the eighth cranial nerve. Since the eighth nerve is part of the afferent portion of the reflex arc, the finding of reduced acoustic reflex sensitivity as a result of alcohol injestion is not surprising.

HEARING LOSS

The measurement of the acoustic reflex threshold using electroacoustic immittance devices is a measurement that is well ensconced in the routine clinical audiologic evaluation

Most patients referred for audiological evaluation receive at least an acoustic reflex threshold test as a feature of the immittance protocol.

Aside from the noninvasive nature of the test and its ease of administration, the measurement of the acoustic reflex threshold is a powerful differential diagnostic tool because the results will be different depending on whether the ear under test is normal, or has a conductive, sensorineural or retrocochlear lesion. The purpose of this section is to briefly review the different results typically expected in ears with each type of pathology.

In discussing the clinical interpretation of acoustic reflex testing, it is necessary to specify whether the lesion is present in the "stimulus (or activator) ear" or the "monitored ear." In contralateral testing, the stimulus ear is the ear receiving the reflex activating signal and the monitored ear is the ear with the probe assemble, the one in which the acoustic reflex contraction is monitored. In ipsilateral testing, the stimulus ear and the monitored ear are the same. In clinical evaluation of the ART, we are always testing the ART of the activator ear.

Conductive lesions affect the acoustic reflex in two ways. A conductive lesion in the monitored ear may result in an absent acoustic reflex even if the stimulus ear is normal because the conductive lesion (fluid, ossicular chain fixation or discontinuity) will interfere with the detection of the immittance change caused by the acoustic reflex. A

conductive lesion in the stimulus ear will result in an ART which is elevated by the amount of the conductive hearing loss, provided it is possible to deliver an adequate activator intensity to the cochlea. If the conductive impairment is bilateral, acoustic reflexes will be absent in both ears.

Recall that in normal ears the reflex is usually elicited at sensation levels of 70 to 95 dB, depending upon the frequency and bandwidth of the eliciting signal. Metz (1952) found that in ears with cochlear lesions the reflex may be elicited by tonal signals at SLs less than 60 dB. Recall also that the same is not true for noise; ARTs for noise are not present at reduced SLs in sensorineural ear. Furthermore, the bandwidth effect noted for normal ears is absent in sensorineural ears (Popelka, 1981).

While Metz (1952) attributed this phenomenon to loudness recruitment, it has since been demonstrated (Margolis and Popelka, 1975) that the acoustic reflex is not related to loudness. Nevertheless, the presence of the acoustic reflex for tonal stimuli in sensorineural ears at reduced sensation levels means that the SPL level of the ART in normal and cochlear impaired ears is similar, at least in patients with mild to moderate sensorineural hearing loss. In patients with severe to profound hearing loss, acoustic reflexes are often absent (Popelka, 1981).

To summarize, the relationship between ART and sensorineural hearing loss is as follows: for tones, the

ART is present at reduced SLs (normal SPLs) for mild to moderate losses. Severe to profound sensorineural losses may exhibit elevated or absent reflexes. The noise - tone difference in ARTs is large for normally hearing subjects and patients with hearing losses worse than 80 dB. For hearing losses between 40 and 75 dB, the noise - tone difference is small (Popelka, 1981). Another finding worth mentioning is that patients with sensorineural hearing loss sometimes have no observable reflex at 4000 Hz, possibly due to increased hearing loss at this frequency. Wilbur (1976) notes that 10% of patients with sensorineural loss in her clinic have no reflex at 4000 Hz.

As previously described patients with retrocochlear pathology exhibit acoustic reflexes that are either absent or present with abnormally rapid decay (Olsen, et al., 1975, 1981). It should be noted that since the absent acoustic reflex in the presence of normal or mildly impaired hearing is potentially such an important sign, it is necessary to rule out conductive pathology in both ears. This is relatively easy to do by considering abnormal findings in pure tone audiometry (such as an air-bone gap) or tympanometry (Wilbur, 1976).

Finally, in lesions higher than the reflex arc, such as midbrain or cortical lesions, acoustic reflexes are usually present since the lesion does not interfere with the acoustic reflex.

RESPONSE TIME (LATENCY) OF THE ACOUSTIC REFLEX

While there is a substantial amount of normative data available regarding acoustic reflex threshold, there is relatively little data available regarding the parameters of normal acoustic reflex response time (or latency). Indeed, there is a lack of uniformity in the definition of latency from laboratory to laboratory; and as described above the possibility of equipment artifacts contaminating the measurement of temporal changes within the reflex response has not been adaquately controlled for, and is not consistently adressed within and between studies.

Response latency may be defined as "the time, in seconds, it takes a biological system to respond to an appropriate stimuli" (McPherson and Thompson, 1977). Hecker and Kryter (1965) defined latency as the time period between stimulus onset and the start of muscle contraction. As noted by Stach and Jerger (1984) and Silman and Gelfand (1982), the traditional definition of acoustic reflex latency using the immittance method is the time period between signal onset and the first detectable immittance change. However, it is difficult to establish a criterion point representing the first detectable change, particularly when the reflex onset is gradual and the reflex is of small amplitude (as it is at and near acoustic reflex threshold). In addition, as previously described, currently available immittance bridges have poor signal-to-noise ratios. While these signal-to-noise ratios may be improved by the use of computer averaging, the point of onset remains difficult to

establish in traces with gradual onset and low amplitude.

Some researchers have operationally defined acoustic reflex latency as the time period between signal onset and a point where reflex magnitude reaches a predetermined percentage (10%, 50%, 90%) of its maximum amplitude. While this approach circumvents the difficulty in choosing a single point of onset, there has been a lack of uniformity in the percentage chosen, causing differences in the reflex latency values found in different studies (Silman and Gelfand, 1982). For example, Norris, et al. (1974) chose several points along the response curve to measure latencies (stimulus onset to reflex onset, stimulus onset to 95% of maximum amplitude, etc.). Colletti (1974) defined latency as the period from signal onset to 5% of maximum impedance change. Stach and Jerger (1984) measured the time from signal onset to the time where the reflex has attained 20% of its maximum amplitude.

Borg (1972), Moller (1974) and others defined latency as the time period from stimulus onset to the time when the reflex has attained 10% of its maximum amplitude. Working group S3-60 of the American National Standards Institute has been working since 1975 to establish a standard for Aural Acoustic-Immittance Instruments. The following definition of initial latency is taken from Appendix B in draft 1982-A of the proposed standard, and appears in Lilly (1984):

"Initial latency is defined as the time (in

> seconds) from the beginning of an instantaneous immittance change to 10% of the measured steady-state immittance change" (Lilly, 1984, p.14).

Once a standard is established, intitial response time (or latency) there will be greater uniformity between laboratories, a situation which is crucial if response time measures are to be used clinically.

The latency of the human acoustic reflex has also been measured by cochlear microphonic recordings (Hallpike, 1934), electromyography (Perlman and Case, 1939), electromanometry (Neergaard and Rasmussen, 1966), and observation of acoustic immittance change (Metz, 1951; Moller, 1958; Dallos, 1964; Norris, et al., 1974; Ruth and Niswander, 1976; Sieminski, et al., 1977; McPherson and Thompson, 1977; Clemis and Sarno, 1980, 1980A; Jerger and Hayes, 1983; Church and Cudahy, 1984; Stach and Jerger, 1984).

Table 4 presents a summary of the latency values reported by the various researchers and techniques mentioned above. It is immediately apparent that there is considerable variablility between studies, and that latency values vary according to the technique of measurement used and the time period measured. The shortest latencies were found using EMG and CM recordings, but these techniques are impractical for routine clinical use. EMG requires direct surgical access to the middle ear and was done by Perlman and Case (1939) on four patients in whom the stapedial tendon was fully exposed during surgery or because of large

perforations of the tympanic membrane. Hallpike (1934) noted the time when a change in the magnitude of the cochlear microphonic occurred following stimulation with a "loud" signal and inferred that the change represented a contraction of the middle ear muscles. The most practical, non-invasive and widely used method of measurement is the immittance method. The observed latencies are longer than the more direct EMG technique because they represent the time period between stimulation and the recording of theimmittance change caused by contraction. However, the technique does not require sedation, nor does it require an exposed stapedial tendon. The measurement is quick, painless, and the measuring equipment is widely available.

Table 4
Summary of Response Time Values Reported in the Literature

AUTHORS	TECHNIQUE	LATENCY	STIMULUS
Perlman & Case (1939)	EMG - direct observation	10.5 msec	1000 Hz at 100 dB
Hallpike (1934)	cochlear microphonic	10.0 msec	
Neergaard & Rasmussen(1966)	electroman-ometry	15-17 msec	1000 Hz at 100 dB SPL
Metz (1951)	mechanical impedance	35 msec 150 msec	1000 Hz at 110 dB 1000 Hz at 80 dB SPL
Moller (1958)	electro-acoustic impedance	25 msec 130 msec	500 Hz at 115 dB 500 Hz at 90 dB
Dallos (1964)	mechanical impedance	40 msec 160 msec	WN at "high intensity" WN at "low intensity"
Norris, et al. (1974)	electro-impedance	129.1 msec	1000 Hz at 10 dB above reflex threshold
Ruth & Niswander (1976)	electro-acoustic	90 msec 100 msec 170 msc	WN at 100 dB SPL 3000 Hz at 100 dB SPL 500 Hz at 100 dB SPL
Sieminski et al. (1977)	electro-acoustic	150 msec 300 msec	2000 Hz at 16 dB above reflex threshold 2000 Hz at ART
McPherson & Thompson (1977)	electro-acoustic impedance	96 msec	2000 Hz at 10 dB above reflex threshold
Clemis & Sarno (1980)	electro-acoustic impedance	93 msec 105 msec	2000 Hz at 10 dB above 1000 Hz at 10 dB above reflex threshold
Church & Cudahy (1984)	immittance	101 msec 111 msec	500 Hz at 10 dB above 2000 Hz at 10 dB above reflex threshold
Stach & Jerger (1984)	immittance	125 msec	2000 Hz at 110 dB SPL

INTENSITY, BANDWIDTH AND FREQUENCY EFFECTS ON ACOUSTIC REFLEX RESPONSE TIME

Table 4 shows that using the impedance (now called the immittance) method, latency at acoustic reflex threshold varies between 100 and 300 msec when the eliciting stimuli are tones. It is well documented that as stimulus intensity increases, latency decreases. Dallos (1973) noted that acoustic reflex response latency is inversely proportional to the strength of the stimulus. The shortest supra-reflex threshold latency reported is 25 msec at 115 dB (Moller, 1958) and the longest is 150 msec at 16 dB SL re: acoustic reflex threshold (Sieminski, et al., 1977). It is also widely noted that while the intra and inter-subject variability of latency measures is high when measured at acoustic reflex threshold, the variability decreases as stimulus intensity increases.

White noise was used as an eliciting stimulus in only two studies (Dallos, 1964; Ruth and Niswander, 1976). The latencies that Dallos (1964) found using white noise stimuli are longer than the latencies to tones reported in other studies. However, Ruth and Niswander (1976) found shorter latencies to white noise than to tones. There were no studies in which band limited noise was used as an eliciting stimulus. Thus, the effect of noise signals on the time course of the acoustic reflex is unestablished because there is a paucity of such data in the literature and the two studies that used white noise found opposite effects. The effect of stimulus frequency on the time course

of the acoustic reflex has also not been widely studied. As is the case with noise signals, the available studies show opposite frequency effects. Ruth and Niswander (1976) found longer latencies at low frequencies than at high, as did Clemis and Sarno (1980). But, Moller (1974), Church and Cudahy (1984) and Stach and Jerger (1984) report faster response times at low frequencies than at high.

Thus, while the effect of stimulus intensity on acoustic reflex response time and its associated variability seems to be clearly established, there are limited and conflicting data regarding the effects of frequency and bandwidth on the response time of the acoustic reflex. Certainly, the wealth of data available on the effects of frequency and bandwidth on acoustic reflex threshold invites comparison of response time data using the same eliciting stimulus parameters.

A systematic investigation of the effects of frequency, bandwidth and intensity on the response time of the acoustic reflex in young, normal adult listeners was the goal of this study.

CHAPTER 3

METHODOLOGY

In any study of the acoustic reflex, there are two instrumentation systems to consider: 1) the reflex activating system, which delivers the stimulus to the ear and 2) the reflex measurement system, which records the reflex response in the ear either contralateral or ipsilateral to the stimulated ear. In this chapter, the two instrumentation systems are described separately. The subjects, experimental design and data analysis are described in subsequent sections of the chapter.

REFLEX ACTIVATION SYSTEM - EXPERIMENTAL STIMULI

Experimental stimuli consisted of 1/6, 1/3, 1, 2 and 4 octave bands of noise centered at 500, 1000 and 4000 Hz, respectively. The signals were presented at two intensity levels: 1) acoustic reflex threshold and 2) 10 dB above acoustic reflex threshold. Stimulus duration was 500 msec, with a 2 second interstimulus interval.

The noise bands were produced by filtering wide-band, flat spectrum noise. The bandwidths were chosen to facilitate comparison to the findings reported in the literature regarding the effect of bandwidth on acoustic reflex threshold; they are similar to the signals used by Flottorp, et al. (1971) and Popelka, et al. (1976). Filtered noise, rather than tonal complexes of varying frequency separation, was used because the acoustic reflex is known

to be more sensitive to noise than it is to tones. Noise stimuli were used to control for noise-tone sensitivity differences, and to insure that the observed effects were attributable to signal bandwidth rather than spectral peaks in the signal.

The noise signals were produced using a random noise generator (Grason Stadler 901 B). The output of the noise generator was shaped by two cascaded filters (Allison AL-2AB). Cascading the filters produced a 60 dB per octave roll off at band limits. The signals were amplified by a solid-state stereo amplifier (Realistic SA-500) and controlled in level with a calibrated attenuator (0.1 dB steps). The signal was then passed through an impedance matching transformer (Grason Stadler model E10589A) and fed to a TDH-49 earphone in an MX-41/AR cushion.

Experimental stimuli were calibrated using the equipment shown in Figure 1. A sine wave which was continually variable in frequency was produced by a random sine generator (Bruel & Kjaer model 1024). This signal was fed through the cascaded filters, amplifier and attenuator to a sound level meter and associated filter set (Bruel & Kjaer type 2203, type 1616) the output of which was fed to a vacuum tube voltmeter (Hewlett Packard 400 D) and electronic counter (Hewlett Packard 522 B). For each bandwidth, the upper and lower frequency cutoffs of the cascaded filters were set to the desired nominal cutoffs. The frequency of the sine wave produced by the random sine generator was

continually varied until the meter of the voltmeter indicated that the intensity of the signal had decreased by 3 dB. At that point, the actual frequency cutoff of the filter was noted on the frequency counter. Because there is some discrepancy between the actual frequency cutoffs and those registered on the filter dials, the Allison filter cutoffs were then adjusted until the measured upper and lower cutoffs were as close as possible to the nominal upper and lower frequency cutoffs of each noise band.

Once the upper and lower band limits for each signal were set, the random sine generator was replaced with the random noise generator to be used in the experiment (Grason Stadler 901 B). The maximum output of the signal generating system (in dB SPL) was then measured for each noise band.

Table 5 shows the nominal and measured upper and lower cutoffs for each noise band. Table 6 shows the maximum output of the system in dB SPL as a function of signal center frequency and bandwidth.

Figure 2 shows the frequency response of the TDH-49 earphone. Note that it does not effectively deliver signals much higher in frequency than approximately 6000 Hz. Thus, while the two and four octave bands of noise centered at 4000 Hz should have upper cutoffs of 8000 Hz and 16000 Hz respectively, the actual upper cutoffs of these experimental stimuli were determined by the response curve of the headphone. As will be seen in Chapter 6, this affects the interpretation of response time data for these two noise

bands.

Table 5
Nominal and Measured Upper and Lower Band Limits
(Frequency in Hz) for Each Experimental Stimulus

	LOWER CUTOFF (-3 dB)		UPPER CUTOFF (-3 dB)	
	NOMINAL	MEASURED	NOMINAL	MEASURED
CF 500 Hz				
1/6 OCTAVE	472	470	530	530
1/3 OCTAVE	445	445	561	562
1 OCTAVE	354	353	707	704
2 OCTAVES	250	230	1000	1000
4 OCTAVES	125	125	2000	1997
CF 1000 Hz				
1/6 OCTAVE	944	944	1060	1058
1/3 OCTAVE	891	891	1122	1129
1 OCTAVE	707	707	1414	1414
2 OCTAVES	500	500	2000	2000
4 OCTAVES	250	249	4000	4007
CF 4000 Hz				
1/6 OCTAVE	3776	3773	4220	4222
1/3 OCTAVE	3564	3578	4490	4494
1 OCTAVE	2828	2829	5657	5663
2 OCTAVES	2000	2005	8000	6288
4 OCTAVES	1000	1009	16000	6407

Table 6
Maximum Output (in dB SPL) as a Function of Center Frequency and Bandwidth.

	OCTAVES				
	1/6	1/3	1	2	4
500	110	115	118	118	118
1000	112	118	118	118	118
4000	119	119	118	118	118

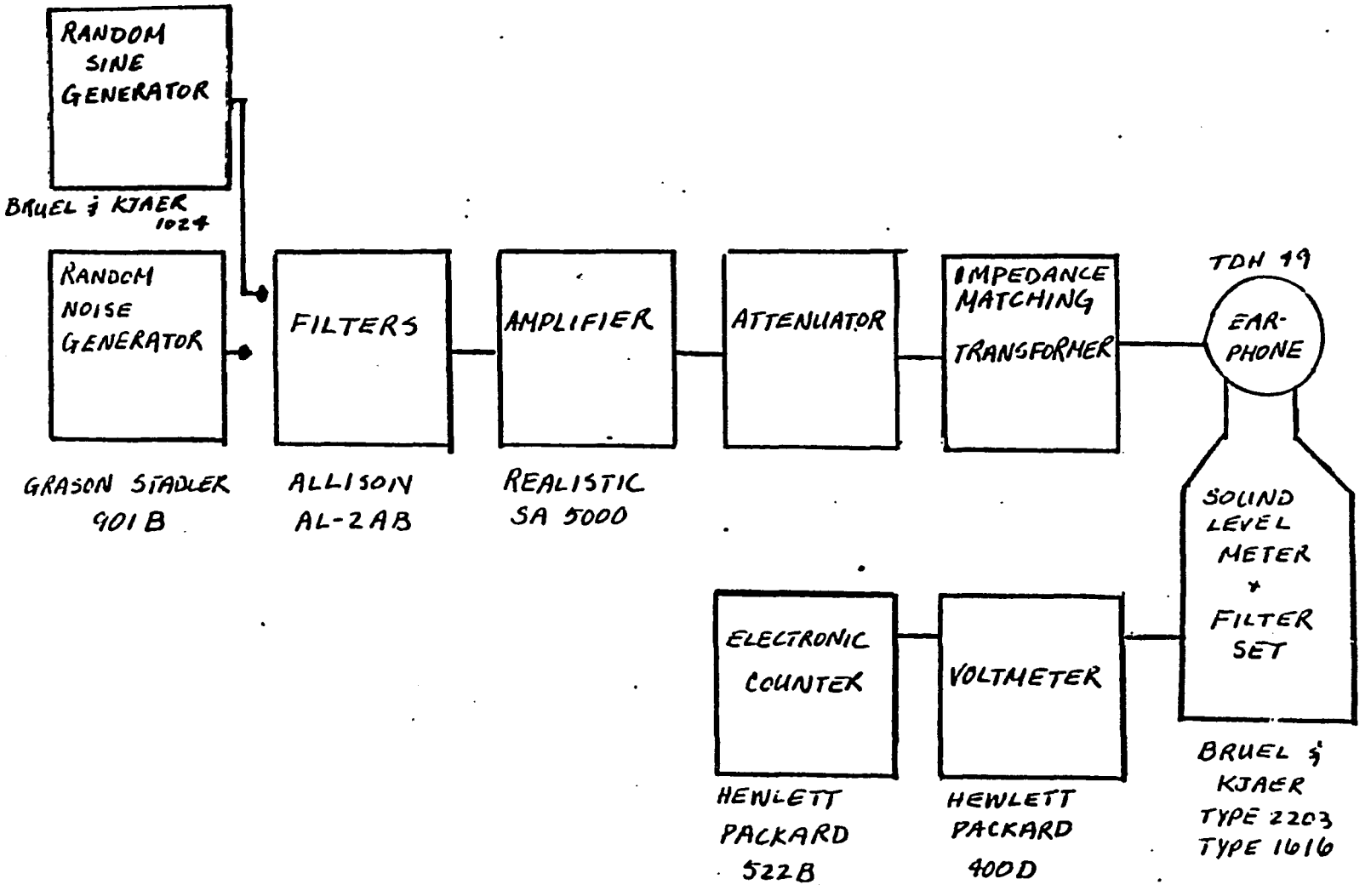


Figure 1. Instrumentation used to produce and calibrate the experimental stimuli.

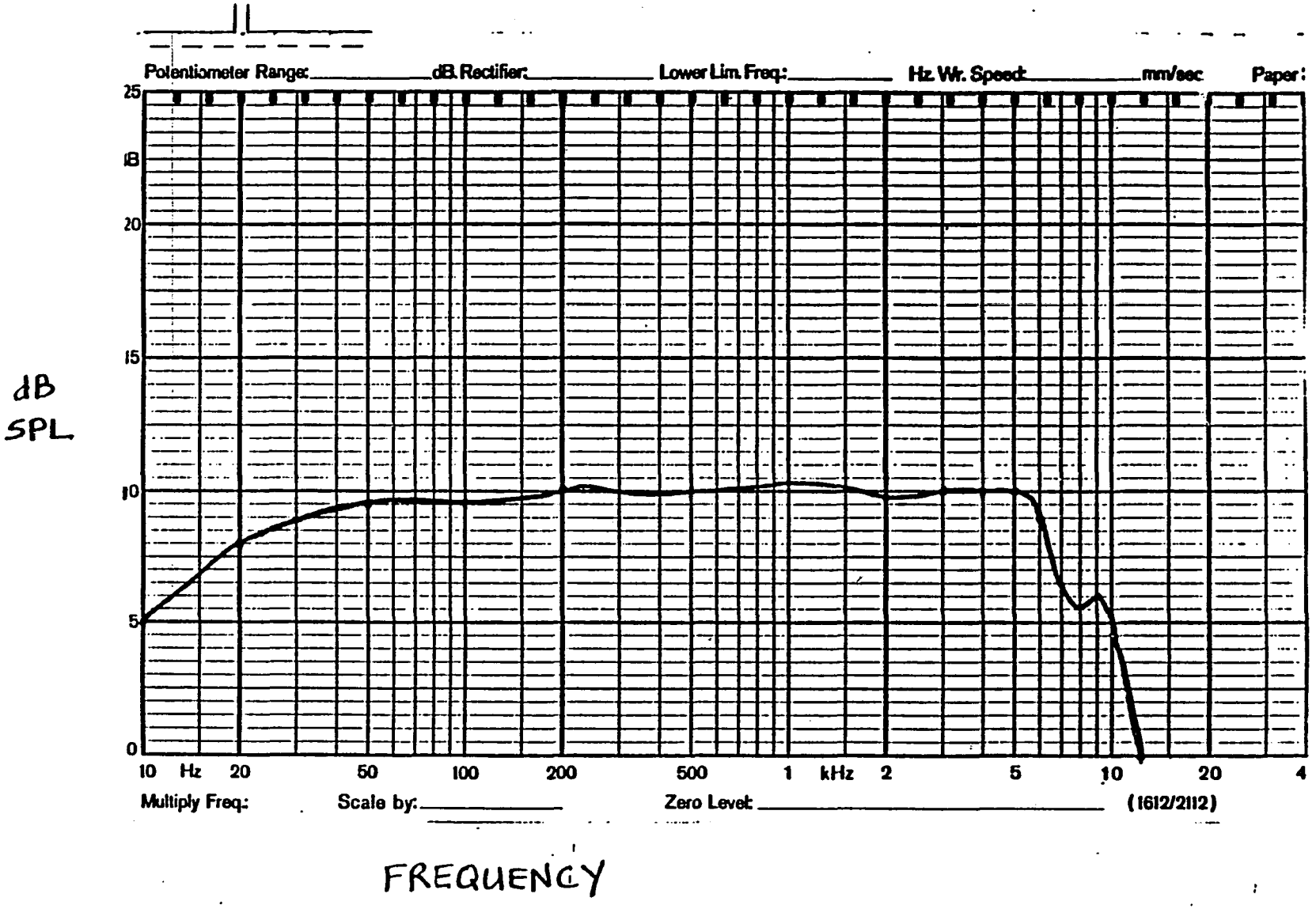


Figure 2. Frequency response of the earphone used in the experiment as measured in a 6 cc coupler.

TIME SEQUENCE OF EXPERIMENTAL STIMULI

Figure 3 shows the time sequence of the experimental stimuli. The duration of each stimulus was 500 msec. This duration was chosen because it is longer than that which causes rapid temporal summation at reflex threshold (Moller, 1983) and shorter than that which induces adaptation of the acoustic reflex (Metz, 1951). The interstimulus interval was 2 seconds to insure independence of stimulus effects.

The timing of the signals was controlled by Grason Stadler 1200 series logic modules. The rise time of the switch was virtually zero ("fast" setting). A 100 msec prestimulus period was introduced to establish a baseline against which to compare impedance change as a function of time.

REFLEX MEASUREMENT SYSTEM

The impedance change caused by the acoustic reflex was measured using the susceptance (B) channel of a Grason Stadler 1720B Otoadmittance meter, using the 220 Hz probe frequency. The voltage at the "recorder" output of the B meter is proportional to the change in acoustic impedance. This output became the signal input to a Digital Signal Averager (Nicolet model 1010). The Grason Stadler 1200 series timer triggered the averager to begin averaging 100 msec prior to the onset of the reflex activating stimulus. This 100 msec prestimulus period provided a baseline against which to compare impedance change as a function of time, as described above.

As shown in Figure 3, the window of the averager was 1000 msec. Each averaged response included a 100 msec prestimulus period, 500 msec of impedance change as a function of time during reflex activation, and 400 msec of post-stimulus recovery. The signal was off for an additional 1600 msec prior to the next signal.

Sixteen repetitions of the reflex response at each intensity level and bandwidth were averaged by the response averager. A hard copy of the averaged response was obtained by recording the output from the response averager with an X-Y plotter (Hewlett-Packard, model 7035B).

Figure 4 is a block diagram of the instrumentation used in this study. Note that the reflex activating system consists of the instruments shown to the left of the face while the reflex measurement system consists of the instruments shown to the right of the face.

The reflex activators were the noise stimuli described earlier. The output of the random noise generator (Grason Stadler 901B) was shaped by cascaded filters (Allison AL-2B), amplified (Realistic SA-500), attenuated by a calibrated attenuator (0.1 dB steps) and impedance matched (Grason Stadler E10589A) prior to delivery to the ear by a TDH-49 earphone in an MX 41/AR cushion. Their timing was controlled by the Grason Stadler 1200 series modules.

The reflex measurement system consisted of an electroacoustic admittance device (Grason Stadler 1720B), digital signal averager (Nicolet 1010), oscilloscope

(Tektonix Type RM 503), and X-Y Plotter (Hewlett Packard 7035B). As previously described, the voltage at the output of the B meter of the otoadmittance device is proportional to the change in acoustic susceptance and was the input to the signal averager. The Grason Stadler 1200 series produced a trigger pulse that caused the averager to begin averaging 100 msec prior to the onset of the reflex activator, providing a baseline against which to compare impedance change as a function of time.

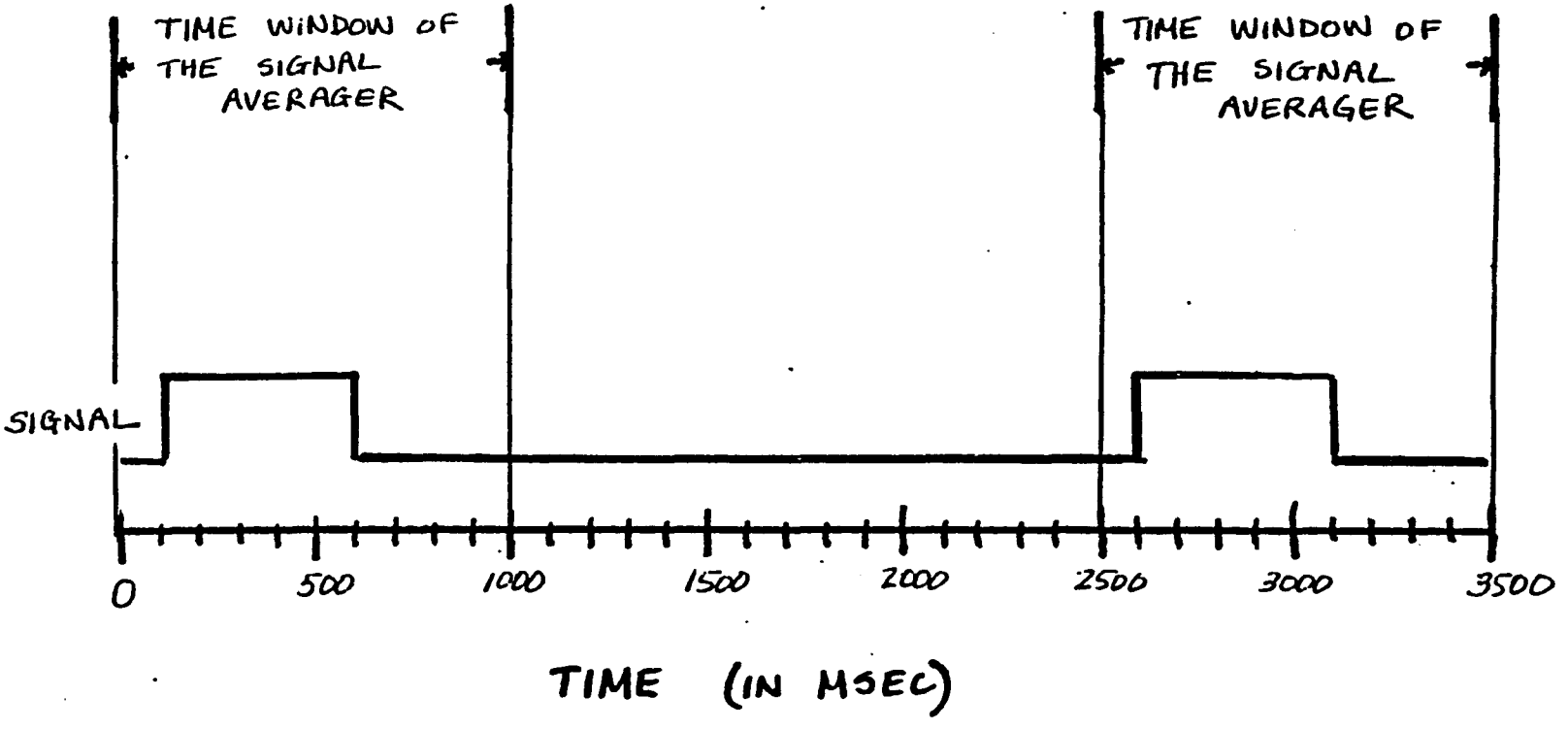


Figure 3. Time sequence of the experimental stimuli.

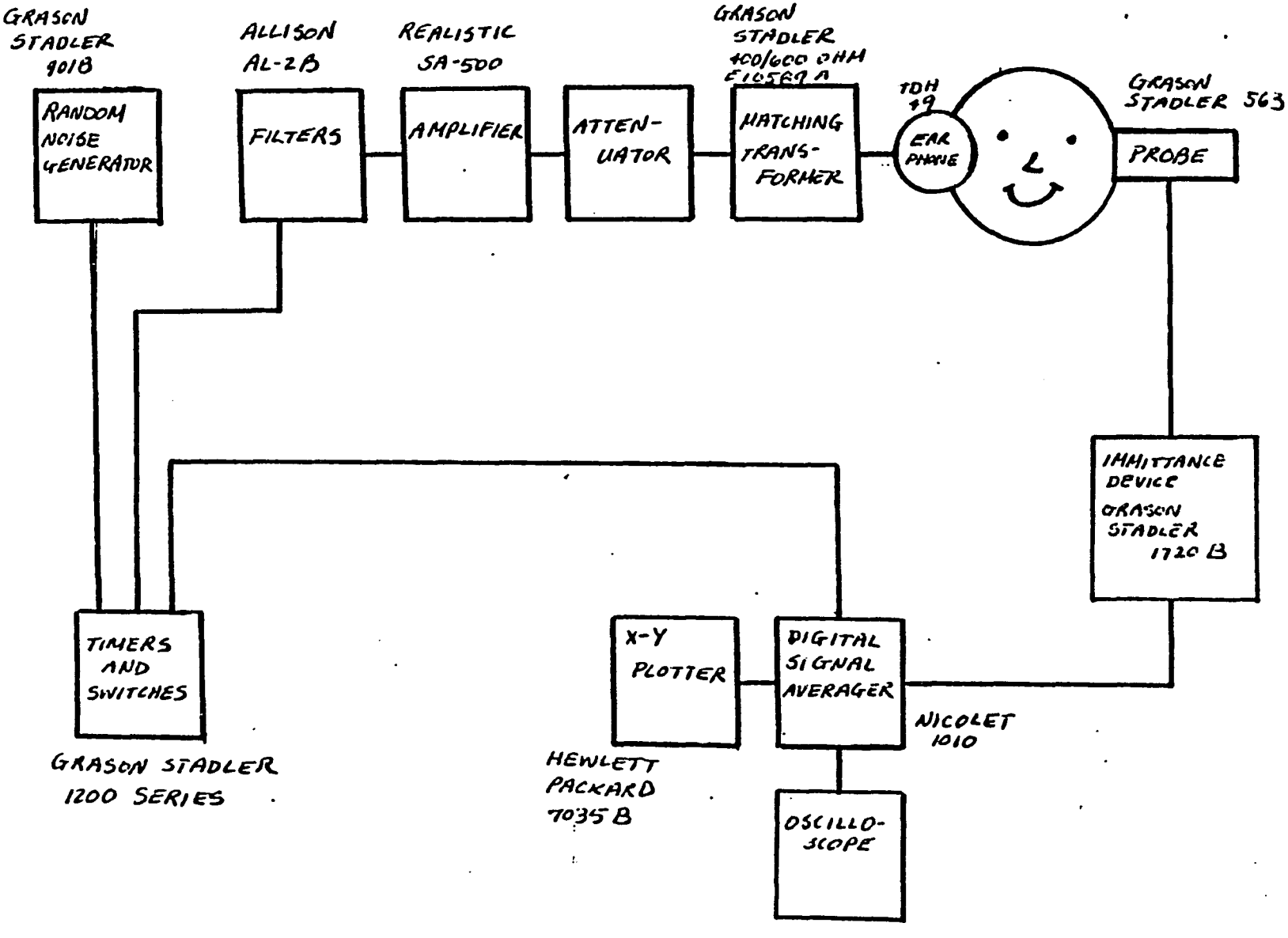


Figure 4. Instumentation used for the experiment.

SUBJECTS

Ten normal hearing young adults, five males and five females, aged 23 to 31 years were subjects for this study. All subjects had normal hearing (thresholds no poorer than 15 dB HL re: ANSI, 1969 by both air and bone conduction) and negative history of otological disease. Prior to the beginning of the experiment, otoscopy and tympanometry were performed. All subjects had tympanogram susceptance peaks between +100 and -100 mm H 0 bilaterally, and acoustic reflexes were present at levels within normal limits in both ears.

In order to comply with the 1971 Department of Health, Education and Welfare Policy on Protection of Human Subjects described by B. H. Gray (1975), each subject indicated informed consent by reading and signing a consent form prior to participation in the study (see Appendix A).

EXPERIMENTAL DESIGN

The dependent variable in this study was the initial response time of the acoustic reflex. The independent variables were: stimulus center frequency, stimulus bandwidth, stimulus level and test replication.

All testing was done inside a double walled IAC sound treated room which met the ANSI standard for audiometric environments (ANSI, 1960). The calibration of the reflex activating signals was checked daily for frequency, duration and level.

Testing was done in six 45 minute sessions, three sessions to complete the first replication (test), three sessions to complete the second replication (retest). Session length was limited to no more than 45 minutes because the subject would become uncomfortable if the probe assembly was left in the ear for a longer period of time. Each subject participated in no more than one session per day.

One frequency was tested per test session in random order. Within each session, bandwidths were tested in fixed order, from narrow to wide. For each bandwidth and frequency tested, 16 responses were obtained and averaged. It has been suggested (Stach and Jerger, 1984) that averaging 8 responses yields a sufficiently precise reflex-response trace. In this study, averaging 16 responses was found to be even more precise, and the extra time needed to obtain 16 responses was minimal.

Subjects were instructed to remain as quiet and still as possible during acoustic reflex measurement. See Appendix B for a copy of the instructions to the subjects. Subjects were allowed to read during the data collection. Prior to acoustic reflex activation, the subject's subjective threshold of audition in dB SPL for each noise band was determined.

An adaquate seal between the ear and the probe assembly was obtained and monitored throughout the data collection via observation of the manometer of the immittance

instrument. Tympanometry confirmed normal middle ear function in the recording ear. Air pressure was set to the point of maximum middle ear compliance.

Acoustic reflex threshold was defined as the lowest activating stimulus intensity producing a visually detectable, timelocked change in impedance. Threshold measurements were obtained by increasing the activating stimuli in ten dB steps until an impedance change was observed on the B meter. The activating stimulus was then decreased in one dB steps until a change was no longer observed. Intensity was again increased, this time in 1 dB steps, until an impedance change was again observed. At the lowest intensity level producing a just detectable change in impedance (acoustic reflex threshold), sixteen responses were averaged and the reflex response curve was plotted.

After sixteen responses at acoustic reflex threshold were averaged and plotted, the intensity of the reflex activator was increased by 10 dB. Sixteen responses at this level were then averaged and plotted. This procedure was repeated for all five octave bandwidths for each of the three center frequencies. The averaged reflex responses were used as the data for each condition.

DATA ANALYSIS

Figure 5 shows typical acoustic reflex responses. Note that there are two typical patterns of response: uniphasic and biphasic. In this study, five of the ten subjects exhibited a uniphasic response and five exhibited a biphasic response. Thus, while the data were combined for the analyses of variance, the grouping variable of "response type" was used to compare the biphasic and uniphasic data.

Initial response time is a combination of the latency and rise time of the instrumentation and the latency and rise time of the reflex. To determine if the equipment latency and rise time were adding to the measured response time of the reflex, an additional experiment was performed prior to the collection of reflex response data. The purpose of the additional experiment was to determine the response delay and filter time constant of the Grason Stadler 1720B otoadmittance device that was used in this study. The additional experiment is described in Chapter 4.

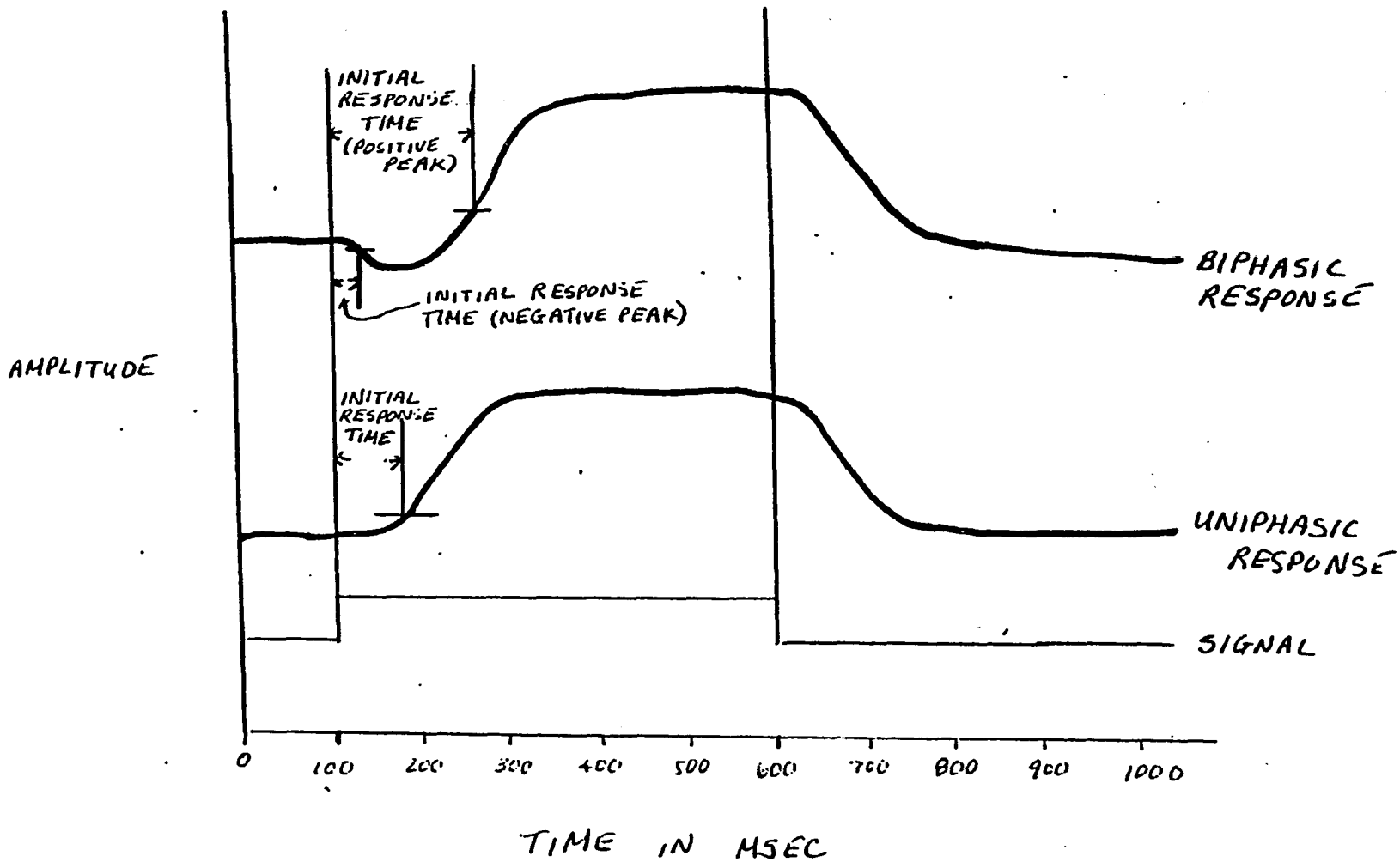


Figure 5. Typical acoustic reflex responses.

Initial response time of the acoustic reflex is defined in this study as it has been defined by Borg (1972) and Moller (1974) and Lilly (1984). The initial response time of the acoustic reflex is the time period between the onset of stimulation and the point on the reflex curve that represents 10% of the maximum reflex amplitude.

The label "latency" traditionally refers to the time period between signal onset and initial impedance change, a point which is difficult to find in responses with gradual onset and low amplitude. The diagnostic value of that measurement is currently being questioned in the literature (Jerger and Hayes, 1983; Stach and Jerger, 1984; Stach, Jerger and Oliver, 1984), and the findings of these studies suggest that the information in the initial portion of the curve is diagnostically more significant than the time period between signal onset and initial impedance change. In addition, the measurement to the point corresponding to 10% of the maximum reflex amplitude is currently under consideration as a standard (Lilly, 1984). For those reasons, both the term "latency" and the traditional measurement of the period between signal onset and impedance change onset have been abandoned in this study..

Another reason for measuring to a point corresponding to 10% of maximum reflex amplitude to avoid the uncertainty inherent in visual monitoring of the susceptance meter. Meter movements in the area below the 10% point are difficult to observe and are subject to many sources of variability.

These uncertainties are either eliminated or reduced considerably by using a reference point of at least 10% of the maximum.

For all subjects in this study, both biphasic and uniphasic, the time period between the onset of the stimulus and the point along the averaged reflex-response curve that corresponds to 10% of the maximum positive reflex amplitude (in mm) was measured. In addition, for the five subjects with a biphasic response, the time period between signal onset and the point corresponding to 10% of the maximum negative amplitude attained by the reflex was also measured.

Separate analyses of variance of the threshold data and the data obtained at 10 dB above threshold were done because the response time at acoustic reflex threshold was much more variable than the response time at the supra-threshold level.

Analysis of variance techniques were used to determine the significance of the effects of response type (uniphasic vs. biphasic), test (first measure vs. second measure), frequency, bandwidth and their interactions for all subjects' responses to both ART level stimuli and stimuli presented at a level 10 dB above the ART. Post facto analyses were used to determine the significance of differences between bandwidths and frequencies (Winer, 1971).

CHAPTER 4

CALIBRATION EXPERIMENT TO DETERMINE THE RESPONSE DELAY AND FILTER TIME CONSTANT OF THE MEASURING EQUIPMENT

This chapter describes an experiment that was devised to determine the response delay and filter time constant of the Grason Stadler 1720B otoadmittance device used in this study. The term "response delay" refers to the time period between the onset of impedance change and the response of the device to that change. The term "filter time constant" refers to the time period from the onset of the impedance change to the point at which the response curve obtains 63.2% of its amplitude (Lilly, 1984). The purpose of this experiment was to determine whether and to what extent the equipment latency and time constant might add to the measured response time of the acoustic reflex.

A simple procedure for simulating the impedance of the ear in a coupler was used. The procedure uses the 220 Hz probe tone generated by the otoadmittance meter. It involves the measurement of the phase and amplitude of the 220 Hz probe tone as it is reflected from the tympanic membrane and walls of the external auditory meatus. The observed measurements were then duplicated with a modified 220 Hz probe tone inserted into and reflected from a coupler rather than an ear. While there are many applications for such a simulation, the objective in this study was to track the otoadmittance meter's response to the simulated impedance change in a coupler.

The procedure differs from previous simulations in that

the simulated change in impedance in the coupler exactly duplicates the change that occurs in the ear during an acoustic reflex. It is a simple and straightforward technique, but the result is applicable at only a single frequency, in this case the frequency of the probe tone generated by the otoadmittance meter.

The first step in the procedure was to measure the reflected 220 Hz probe tone before and during acoustic reflex activation. In this way, the effects of the reflex on the phase and amplitude of the reflected probe tone were revealed. The effects of the reflex were then simulated in a coupler by substituting two simple capacitance-resistance networks (one to simulate the "no reflex" condition, one to simulate the "reflex" condition) for the ear.

The capacitance-resistance networks were designed to modify the ingoing probe tone in such a way that the tone reflected from the walls of the coupler had the same phase and amplitude as the tone reflected from the walls of the external auditory meatus and eardrum before and during reflex activation. By switching from one capacitance-resistance network to the other, the effect of the reflex on the probe tone within the external auditory meatus was simulated within a coupler. Computer averaging the recorder output of the otoadmittance meter as it responded to the simulated impedance change allowed for the identification and quantification of artifact in the time course of the otoadmittance meter's response to the reflex.

The procedure required two stages. Stage I identified the amplitude and phase of the reflected probe tone (B) using a null method. The objective was to equalize and cancel the reflected probe tone (B) using a modified probe tone (A) generated by the otoadmittance meter. The null method was used because very small changes in amplitude and phase were detectable. In addition, it provided a means of specifying a reference signal representing the reflected tone (B) from the real ear.

As can be seen in Figure 6, two leads were taken from the otoadmittance meter: 1) tone A, the ingoing 220 Hz probe and 2) tone B, the reflected 220 Hz tone. Since tone B was relatively low level, it needed to be amplified (amplifier B). A narrow band filter centered on the tone frequency was used to remove background noise that would have reduced the sensitivity of the null technique. Since the reflected tone (B) had been amplified and filtered, its phase and amplitude were altered as represented by B′ in Figure 6. Note that the amplitude and phase difference between B and B′ was kept constant throughout all subsequent measurements. B′ was now used as the reference for the reflected tone.

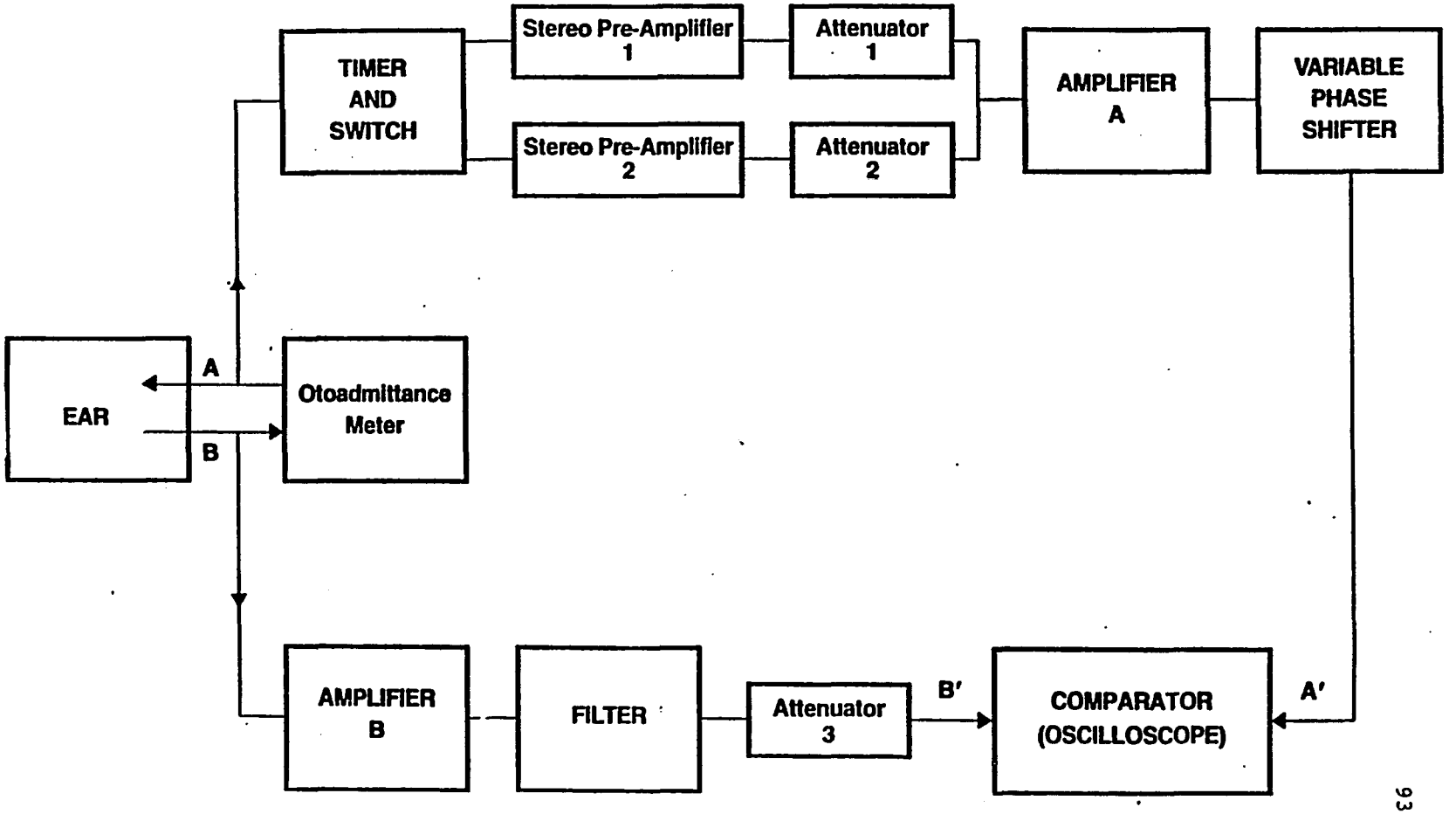


Figure 6. Instrumentation used in the Stage I equalization and cancellation.

The purpose of the upper pathway in Stage I was to modify tone A so that it was identical in both amplitude and phase to tone B′ at the comparator. The comparator was a conventional oscilloscope which provided a comparison between two simultaneous inputs (A′-B′). Note that any small difference between A′ and B′ showed up as a deflection on the oscilloscope, hence the sensitivity of the technique. Tone A′ was adjusted to be identical to B′ in both amplitude and phase using the attenuator(s) and the variable phase shifter shown.

Note that the upper pathway included a timer and switch and a dual pathway involving a separate pair of preamplifiers and attenuators. This equipment may seem extraneous to the actual equalization and cancellation for which the system was designed. However, it was essential that all of the equipment to be used at a later stage (Stage 2) be placed in line for the equalization and cancellation so that the influence of each piece of equipment on the phase of tone A′ was taken into account.

Two equalizations and cancellations were required: one for the ear without a reflex and one with the reflex activated. The intent here was to find that relative phase shift and attenuation such that A′ was identical to the reflected tone (B′) for the real ear with and without the reflex.

Based upon the results of the measurements obtained in Stage I, two simple capacitance-resistance networks were

built to provide the relative phase shift to make the phase of A′ identical to that of B′. Attenuators were used to make the amplitude of A′ equal to that of B′. Pathway 1 was used to equate A′ and B′ for the no-reflex condition, pathway 2 was used to equate A′ and B′ for the reflex condition.

The purpose of Stage II was the simulate the changes measured in Stage I. As can be seen in Figure 7, capacitance-resistance network 1 (or CR-1) was placed in pathway 1 and capacitance-resistance network 2 (or CR-2) was placed in pathway 2 of the upper pathway. The accuracy of the cancellation using the CR-1 and CR-2 networks was checked by obtaining a null with the real ear and these two networks in circuit with the appropriate attenuator settings.

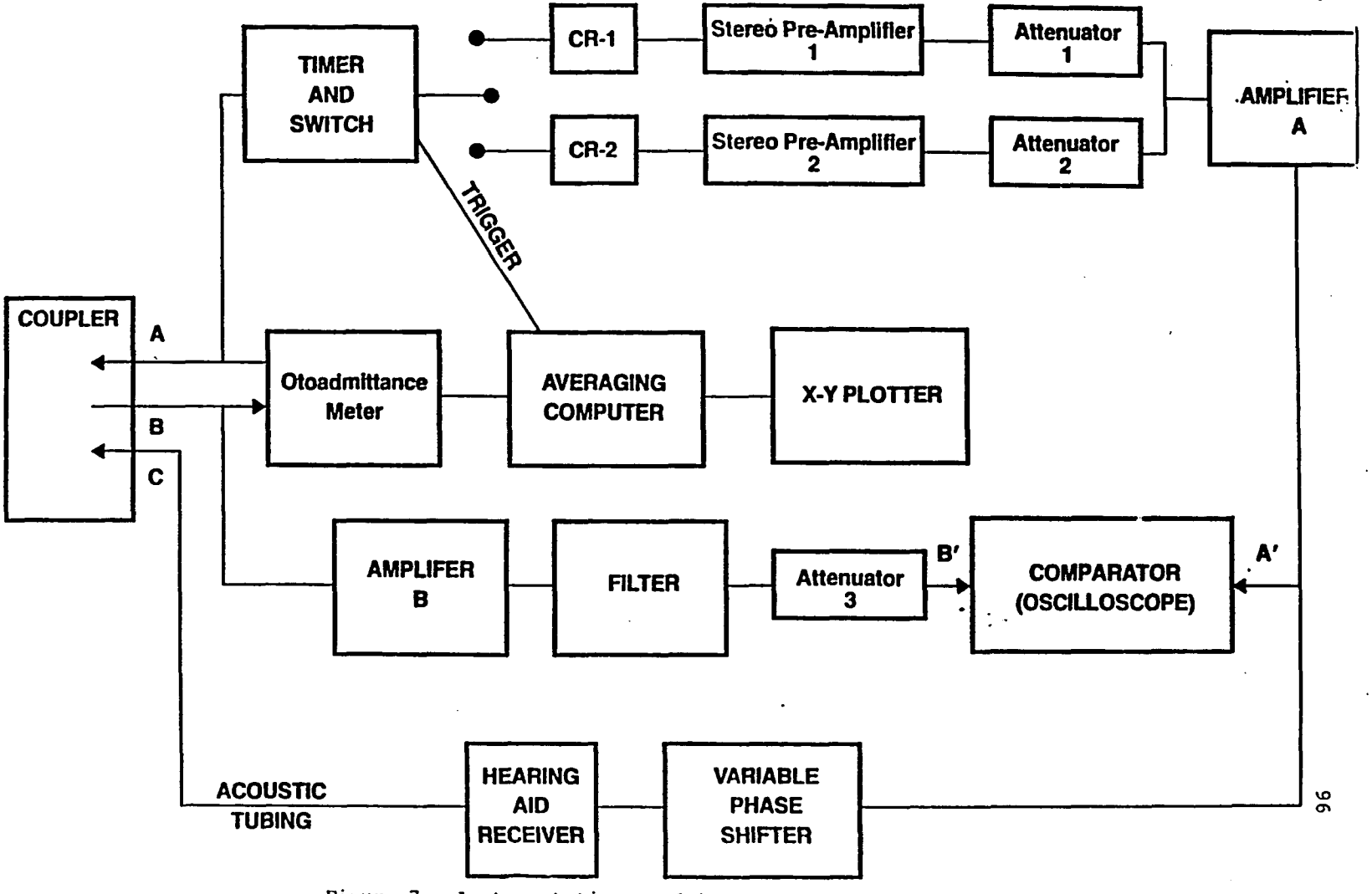


Figure 7. Instumentation used in Stage II to measure the response time and filter time constant of the otoadmittance meter.

The purpose of Stage II was to replace the real ear with a coupler such that the reflected tone (B′) was unchanged. This was done by inserting a tone (C) into the coupler (using the tube normally used to increase air pressure) so that the reflected tone (B′) at the comparator was once again equal to tone A′ for both the no-reflex and reflex conditions. The variable phase shifter and attenuator 3 were used for this purpose. They also compensate for any phase shift and amplitude change produced by the hearing aid receiver and acoustic tubing.

If C was much larger than A, then B′ would change automatically by the correct amount when the circuit switched from CR-1 to CR-2. If A was not negligibly small compared to C, then A should have been eliminated by removing the tube for A from the probe. Since C was much larger than A, the change in B′ was automatic.

Using the procedure described, Table 7 shows the phase shifts that were required to cancel tone B′ in Stage I using the Grason Stadler 1720B Otoadmittance meter. The amplitude under the no reflex condition was used as a reference.

The reflex was elicited by a 1000 Hz tone presented at 10 dB above the acoustic reflex threshold of the contralateral ear of a subject with normal hearing.

Table 7
Phase and Amplitude Changes Caused in the Ear by the Acoustic Reflex.

REAL EAR - NO REFLEX		REAL EAR - REFLEX		EFFECT OF REFLEX	
amplitude change	phase shift	amplitude change	phase shift	amplitude change	phase shift
6.1 dB*	70° *	6.5 dB	80°	0.4 dB	10°

*The amplitude under the no reflex condition was used as a reference

Using Stage II of the procedure, the response characteristics of the Grason Stadler 1720B Otoadmittance Meter were measured. The duration of the probe signal in CR-1 or CR-2 was 500 msec for each condition. The switching rise time was varied from FAST to 5, 10, 25, and 50 msec. The change in susceptance noted on the B meter of the otoadmittance meter was approximately 2 millimhos. The recorder output of the GS 1720B was averaged over 16 responses (N=16) and plotted to determine its response latency and rise time during the change from the no-reflex to the reflex condition. The results in each switching condition are plotted in Figure 8.

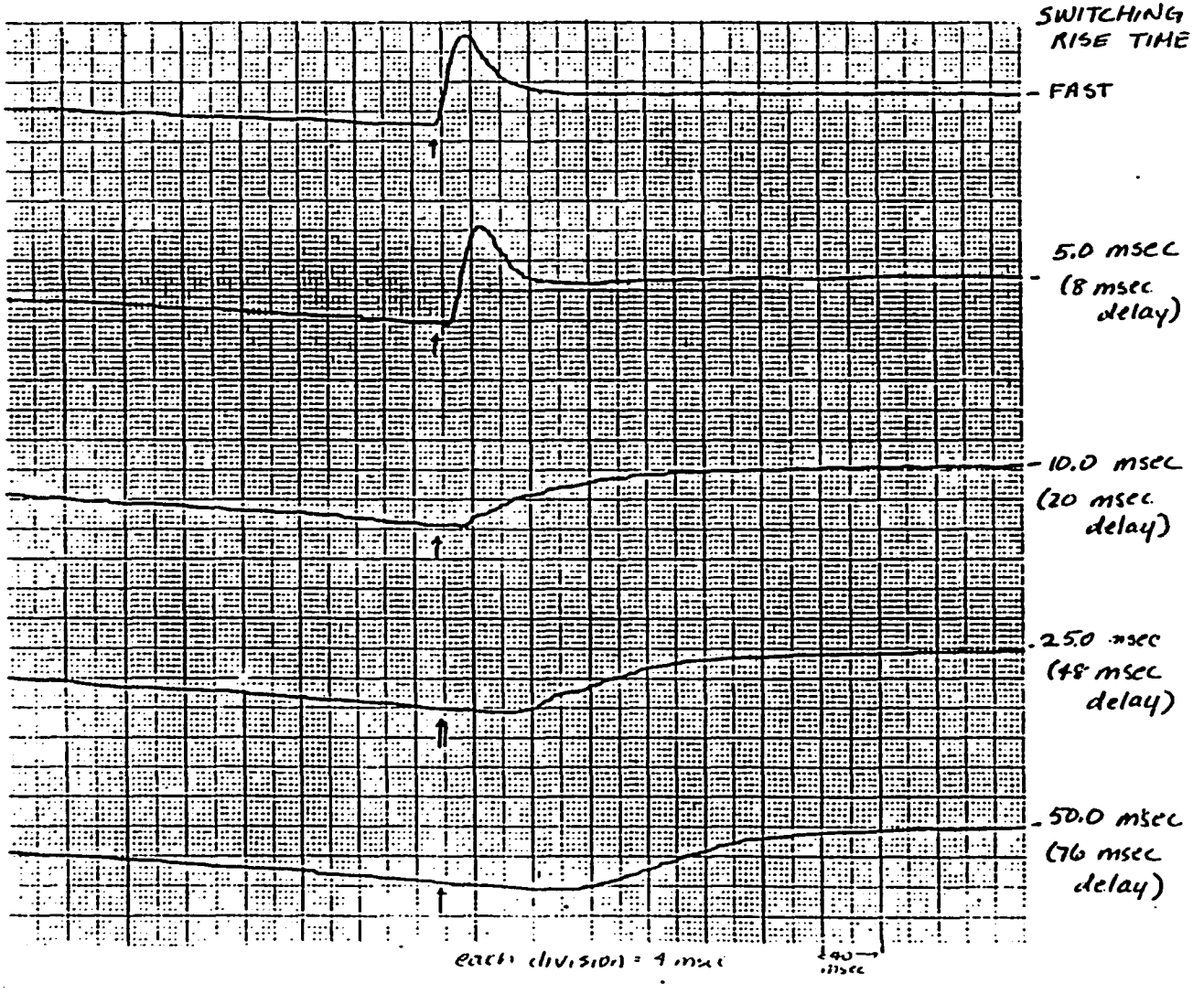


Figure 8. Response characteristics of the Grason Stadler 1720B Otoadmittance Meter.

The results indicate that this meter had essentially no delay in response to an instantaneous change in impedance but that there was substantial amplitude overshoot to the FAST and 5.0 msec switching rise times. This finding is in good agreement with the tracking of a 0.5 acoustic mmhos change in impedance in a coupler reported by Popelka & Dubno (1978) and with the rapid reduction in probe amplitude tracked by Margolis and Gilman (1977). As switching rise time increased, the overshoot disappeared, but a delay in the response of the otoadmittance meter was introduced. The rise time of the instrument, measured by determining the time period from the onset of the instantaneous immittance change to the point at which the response curve obtained 63.2% of its amplitude (Lilly, 1984) was 12 msec.

In summary, since we were interested in looking at acoustic reflex response time, it was essential that we first examine the response characteristics of the meter to be used, to insure that the measuring device was not adding artifact to the reflex trace. We chose to devise a new technique that simulated the phase and amplitude changes observed in the normal ear during acoustic reflex activation. The technique was based on the null method, which was not only simple to use, but was also sensitive to very small changes in phase and amplitude. We then constructed capacitance-resistance networks that electrically simulated the changes that were observed using the real ear, and observed how the otoadmittance meter

responded to the change from the "no reflex" condition to the "reflex" condition. The results indicated that the Grason-Stadler 1720B otoadmittance meter had essentially no delay in response time to an instantaneous change in phase and amplitude on the order of that which occurs in the ear during an acoustic reflex.

CHAPTER 5

RESULTS

ACOUSTIC REFLEX RESPONSE TIME TO 10% OF MAXIMUM POSITIVE RESPONSE AMPLITUDE

Two analyses of variance were done: one on the data obtained at acoustic reflex threshold and one on the data obtained 10 dB above acoustic reflex threshold. The effects of response type (uniphasic vs. biphasic), time order (first test vs. second test) frequency, bandwidth and their interactions on the initial response time of the acoustic reflex were examined in each analysis.

RESPONSE TIME AT ACOUSTIC REFLEX THRESHOLD

Table 8 shows the results of the analysis of variance of the data obtained at acoustic reflex threshold and the mnemonics used for the variance components. The differences between reflex response times for different response types (uniphasic vs. biphasic) was significant at the .05 level. The main effects of Frequency, Bandwidth and Time Order did not reach significance. There was a significant interaction was between Time Order and Frequency ($p < 0.0042$). It should be noted that because each frequency was measured on a different day, frequency differences, if any, were confounded with time-order effects. The ANOVA, however, shows neither Frequency nor Time-Order to be statistically significant. The TF interaction is indicative of significant variability between days.

In general, acoustic reflex response time at acoustic reflex threshold was not significantly affected by frequency

or bandwidth and was not significantly different during the second test than it was during the first test.

Table 8
Analysis of Variance with Repeated Measures.
Response Time to 10% of Maximum Positive Amplitude.
N = 10. Stimulus Level = ART.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Response Type	R	1	605521.6	6.99	<.05
Time Order	T	1	2385.7	0.49	
	TR	1	7008.3	1.45	
Frequency	F	2	4776.5	0.84	
	FR	2	5138.1	0.90	
	TF	2	22654.1	7.85	<.05
	TFR	2	228.9	0.08	
Bandwidth	B	4	4512.9	2.17	
	BR	4	1834.4	0.88	
	TB	4	1458.7	0.81	
	TBR	4	2128.9	1.18	
	FB	8	2985.1	1.63	
	FBR	8	3305.7	1.81	
	TFB	8	2206.8	1.84	
	TFBR	8	2063.4	1.72	

The significant difference in response time at acoustic reflex threshold between the two response types is apparent when the mean response times are considered. The mean response time at ART for the uniphasic response was 228.27 msec and the mean response time ART for the biphasic response was 318.12 msec.

Figure 9 shows the interaction between Time Order and Frequency at acoustic reflex threshold. The response time at ART decreased between measures at 500 Hz, remained the same between measures at 1000 Hz and increased between measures at 4000 Hz.

Post facto examination of the difference between frequencies using Student-Newman-Keuls (Winer, 1971) indicates that the mean response times for 500 Hz and 4000 Hz were significantly different from each other in the second (but not in the first) measurement.

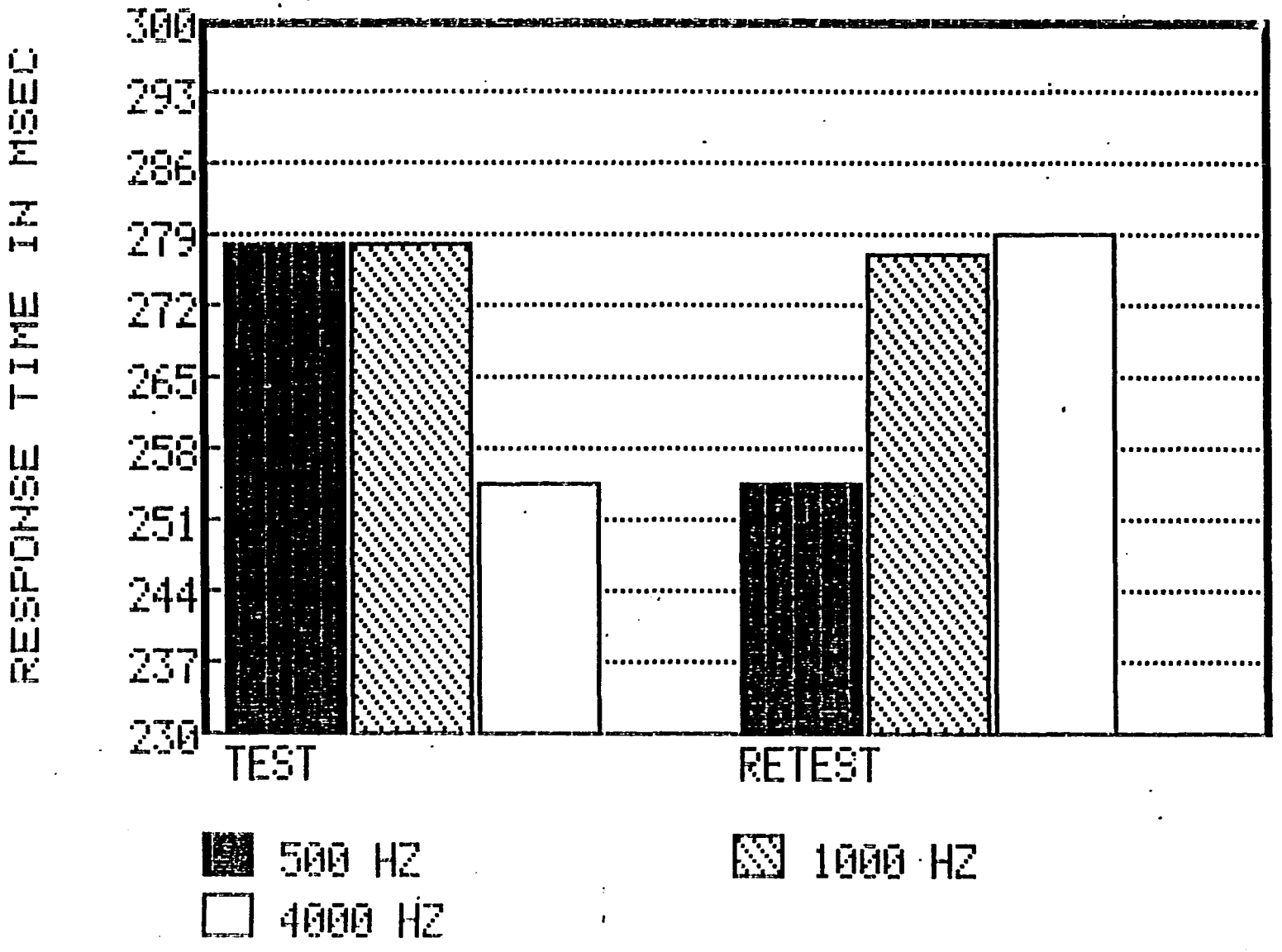


Figure 9. Mean response time at ART for each center frequency, test vs. retest measurement. N = 10.

RESPONSE TIME AT 10 dB ABOVE ACOUSTIC REFLEX THRESHOLD

A second analysis of variance with repeated measures was done to determine the significance of the variables for all subjects when the reflex was elicited by stimuli at a level 10 dB above acoustic reflex threshold. Table 9 shows the results of this analysis of variance.

The main effect of Frequency at 10 dB above acoustic reflex threshold was highly significant ($F = 19.79$; $p < 0.00001$), as was the interaction between Frequency and Bandwidth ($F = 4.96$, $p < 0.0001$). The main effects of Response Type, Time Order and Bandwidth did not reach significance, although the Bandwidth effect approached significance ($F = 2.47$, $p < 0.0646$).

As noted previously, frequency differences were confounded with time-order differences. However, in this case, no significant time order effect was found, and the effect of frequency was highly significant.

Figure 10 shows the mean response time at 10 dB above acoustic reflex threshold as a function of stimulus center frequency. Note that the fastest mean reflex response time at 10 dB above ART occurs with a center frequency of 500 Hz (145.54 msec). The mean reflex response time increases to 156.07 msec at 1000 Hz, and to 177.40 msec at 4000 Hz. Thus, the rate of increase in acoustic reflex response time for signals presented 10 dB above the ART is approximately 10 msec per octave. There is a 10.53 msec increase in response

time from 500 to 1000 Hz, and a 21.33 msec increase in the two octaves from 1000 to 4000 Hz.

Table 9
Analysis of Variance with Repeated Measures.
Response Time to 10% of Maximum Positive Amplitude.
N=10. Stimulus Level=10 dB Above ART.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Response Type	R	1	46725.1	1.79	
Time Order	T	1	2397.0	3.20	
	TR	1	308.0	0.41	
Frequency	F	2	26309.4	19.79	<.0001
	FR	2	497.1	0.37	
	TF	2	34.9	0.07	
	TFR	2	118.2	0.25	
Bandwidth	B	4	936.8	2.46	
	BR	4	544.1	1.43	
	TB	4	107.3	0.40	
	TBR	4	142.4	0.52	
	FB	8	2202.4	4.96	<.001
	FBR	8	617.3	1.39	
	TFB	8	128.4	0.68	
	TFBR	8	122.4	0.65	

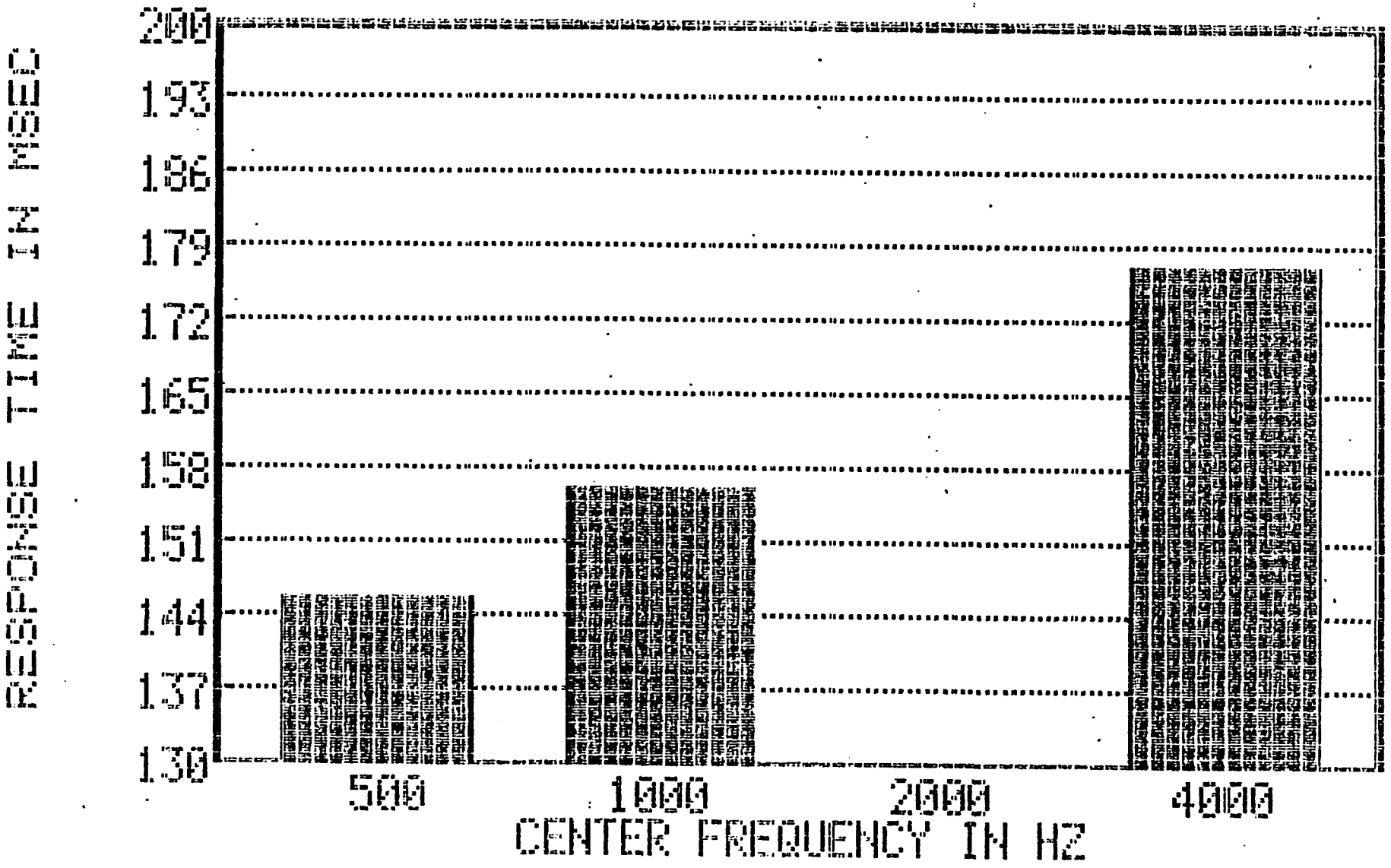


Figure 10. Mean acoustic reflex response time as a function of stimulus center frequency. N = 10. Stimulus level = 10 dB above ART.

Post facto examination of the difference between frequencies using Student-Newman-Keuls procedure at $p < 0.05$ indicates that the mean response time at 10 dB above acoustic reflex threshold for a 4000 Hz center frequency is significantly longer than the mean response times at the same level for 500 and 1000 Hz center frequencies.

Figure 11 displays the interaction between Frequency and Bandwidth ($F = 4.96$, $p < 0.0001$). Mean response time is plotted as a function of bandwidth for all three center frequencies. The figure illustrates that the effect of stimulus bandwidth is different for the three center frequencies. It can be seen that a difference of approximately 10 msec in mean response time is maintained between the 500 Hz and 1000 Hz center frequencies at all bandwidths except 1/6 octave. Furthermore, the response time remains relatively constant for those two center frequencies as a function of bandwidth. This observation is supported by post facto examinations of the data. For both the 500 Hz and 1000 Hz center frequencies, the Student-Newman-Keuls procedure at the 0.05 level indicates that mean initial response times at all five bandwidths are not significatnly different from each other.

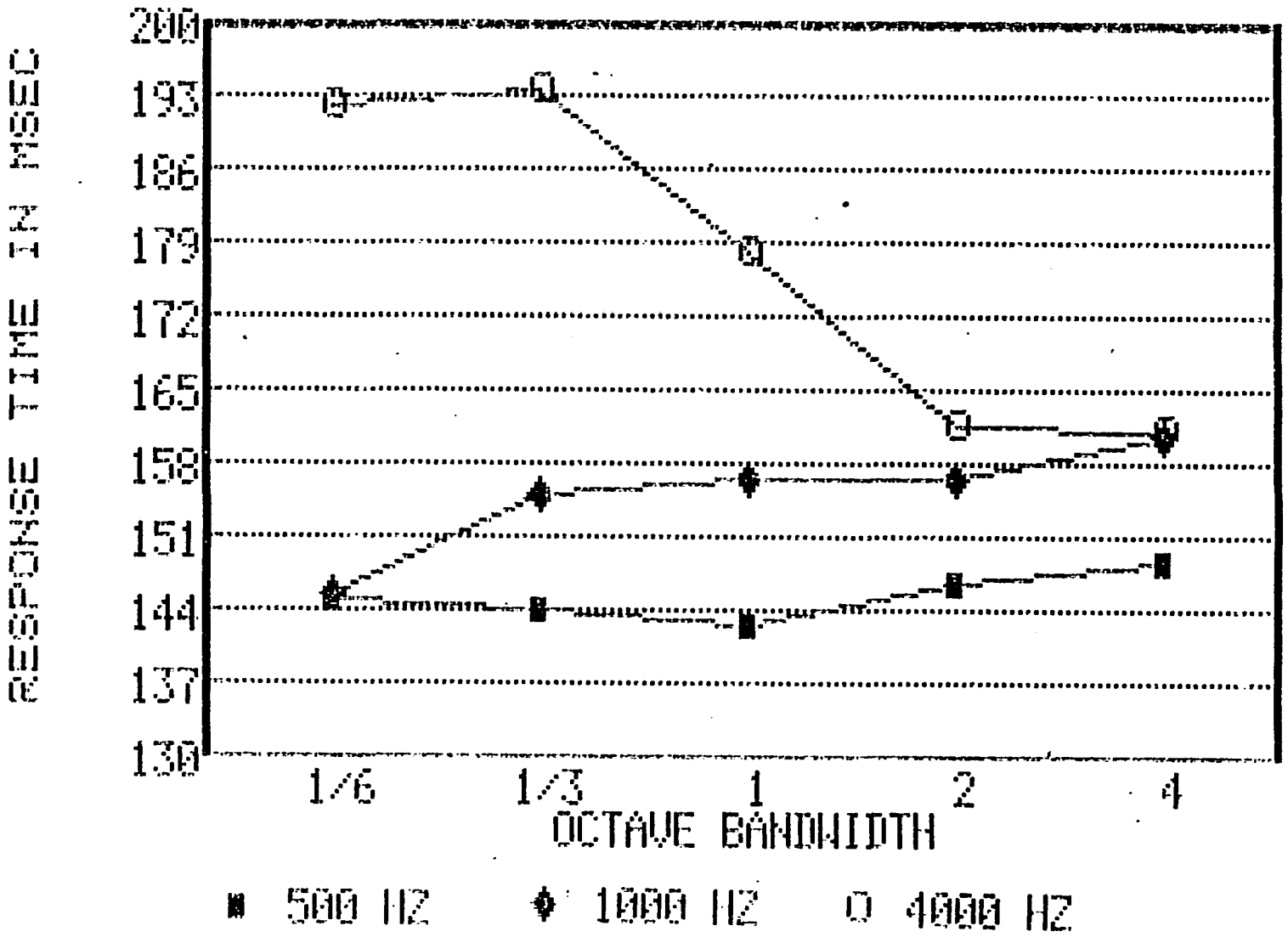


Figure 11. Mean acoustic reflex response time as a function of bandwidth for three center frequencies (500, 1000 and 4000 Hz). N = 10. Stimulus level = 10 dB above ART.

Stated differently, with a 500 Hz or 1000 Hz center frequency, changes in bandwidth did not cause significant differences in the initial response time of the acoustic reflex when the reflex at 10 dB above acoustic reflex threshold.

For the 4000 Hz center frequency, the situation is quite different. As shown in Figure 11, for the two widest bandwidths centered at 4000 Hz, the upper frequency cutoff of the noise bands is determined by the response characteristics of the earphone that delivers the signals. For the TDH-49 earphone, the highest frequency transduced is about 6400 Hz. However, even with this limitation in frequency transduction, the wider bandwidth stimuli do result in significantly shorter response times. The Student-Newman-Keuls procedure for the 0.05 level indicated that the mean response times at the two widest bandwidths (2 and 4 octaves) were significantly different from the mean response times at the 1/6, 1/3, and 1 octave wide bands.

SUMMARY OF RESULTS RE: RESPONSE TIME OF THE ACOUSTIC REFLEX AS MEASURED TO THE POINT CORRESPONDING TO 10% OF MAXIMUM REFLEX AMPLITUDE

At acoustic reflex threshold, the main effect of Response Type (uniphasic vs. biphasic) and the Time Order by Frequency interaction significantly affected the initial response time of the acoustic reflex.

Subjects with a biphasic acoustic reflex had significantly longer initial reflex response times than subjects with uniphasic reflexes. The effect of frequency differed significantly from measure to measure, probably because of the variability of the response at threshold.

Changes in bandwidth, time order and center frequency were not found to significantly change the reflex response time to stimuli presented at acoustic reflex threshold level.

At 10 dB above acoustic reflex threshold, the main effect of frequency and the Frequency by Bandwidth interaction significantly affected the initial response time of the acoustic reflex. Stimuli centered at 500 and 1000 Hz resulted in significantly faster response times than stimuli centered at 4000 Hz. The effect of bandwidth differs with center frequency. Wideband signals centered at 4000 Hz result in significantly shorter response times than narrowband signals centered at 4000 Hz. For 500 Hz and 1000 Hz, changes in signal bandwidth do not significantly alter acoustic reflex response time at 10 dB above acoustic reflex threshold.

At 10 dB above acoustic reflex threshold, there are no significant differences between uniphasic and biphasic response times measured to 10% of maximum positive reflex amplitude. In general, changes in bandwidth do not significantly alter initial response time and no significant differences are noted in the data collected on different days.

RESPONSE TIME OF THE BIPHASIC REFLEX

In order to further analyze the biphasic reflex at acoustic reflex threshold, a third analysis of variance was performed. The results of the analysis of variance of the main effects of Time Order, Frequency, Bandwidth and their interactions on the mean acoustic reflex response time (measured to 10% of the maximum positive reflex amplitude) at acoustic reflex threshold for subjects with biphasic responses is shown in Table 10.

As can be seen in Table 10, the only effect that approached significance was the Time Order by Frequency interaction ($F = 4.18$, $p < 0.0571$). This interaction was significant at $p < .0042$ in the first analysis of all subjects' acoustic reflex threshold level data.

Here again, because each frequency was measured on a different day, frequency differences were confounded with time-order differences. The ANOVA shows no significant time order effect, and the TF interaction, whic is indicative of between day variability only approached significance.

Since the TF interaction approaches significance, and for the sake of comparison to Figure 10, the mean response time at ART for each frequency (first measure vs. second measure) for the subjects with biphasic responses is plotted in Figure 12.

Table 10
Analysis of Variance with Repeated Measures.
Response Time to 10% Maximum Positive Amplitude. N = 5
Subjects with Biphasic responses. Stimulus Level = ART.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Time Order	T	1	608.0	0.18	
Frequency	F	2	7999.7	1.49	
	TF	2	11528.1	4.18	.0571
Bandwidth	B	4	2969.4	2.02	
	TB	4	1786.5	0.99	
	FB	8	2881.4	1.68	
	TFB	8	1285.7	0.98	

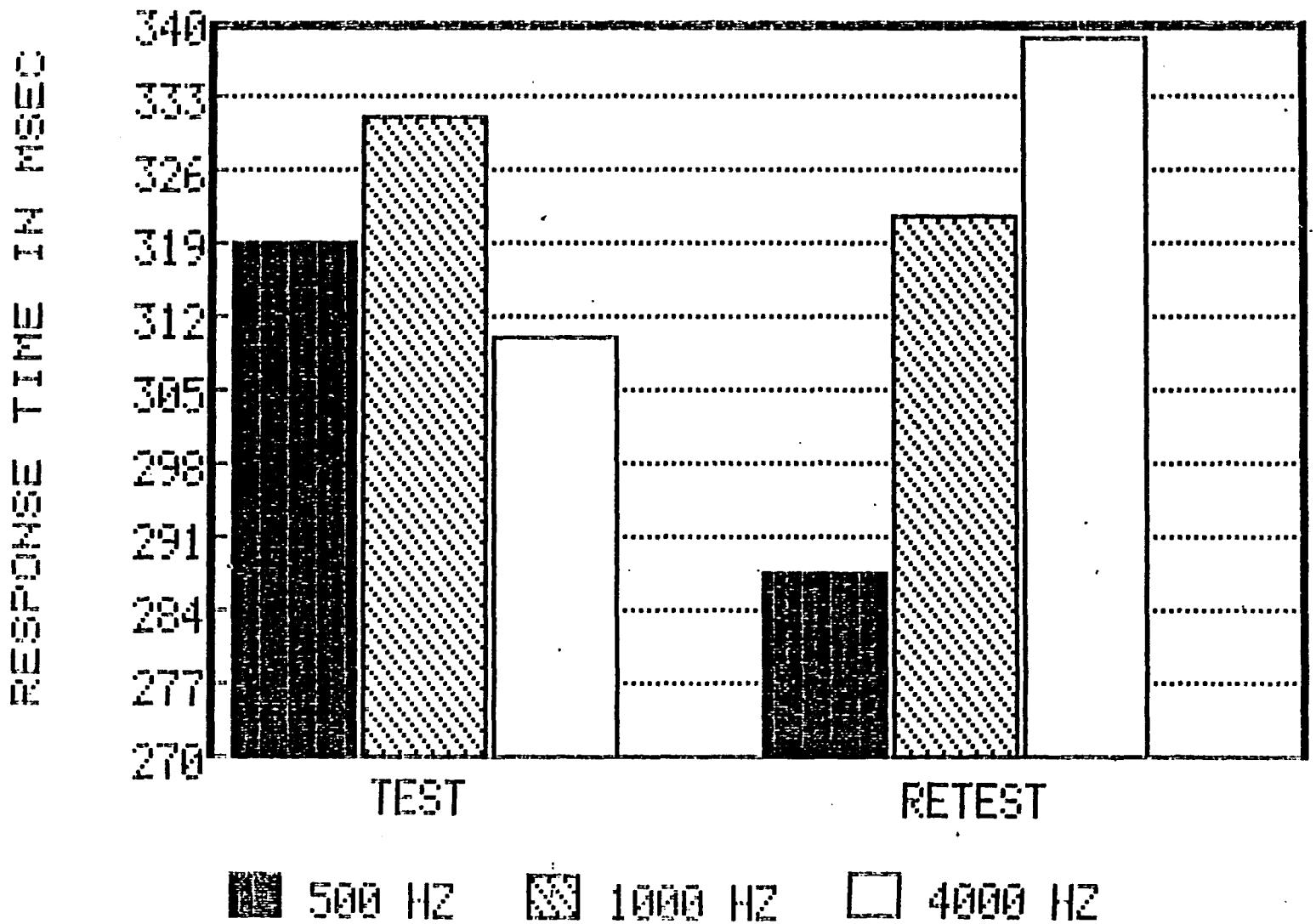


Figure 12. Mean response time at ART' for each frequency, first measurement vs. second measurement. N = 5 biphasic subjects.

RESPONSE TIME TO THE NEGATIVE PEAK OF THE BIPHASIC RESPONSE

Two additional analyses of variance were done to examine the effects of the variables on the response time measured to 10% of the maximum amplitude of the initial negative peak of the biphasic response. One analysis was done on the data obtained at acoustic reflex threshold, and the other analysis was done on the data obtained 10 dB above acoustic reflex threshold.

Table 11 shows the results of the analysis of variance for the data obtained at acoustic reflex threshold. Frequency and Bandwidth significantly influenced the mean response time of the initial negative peak of the biphasic response at acoustic reflex threshold. Response time did not significantly change from test to retest, nor were there any significant interactions of the variables in this analysis.

Table 11
Analysis of Variance with Repeated Measures.
Mean Response Time to 10% of Maximum Negative
Amplitude of the Biphasic Response.
N = 5 Subjects with Biphasic Responses.
Stimulus Level= ART.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Time Order	T	1	3119.0	3.16	
Frequency	F	2	8282.9	7.20	<.05
	TF	2	217.6	0.26	
Bandwidth	B	4	4661.5	5.07	<.01
	TB	4	1028.0	1.42	
	FB	8	695.2	0.39	
	TFB	8	1177.2	1.77	

The effect of Frequency on the response time of the initial negative peak of the biphasic acoustic reflex at acoustic reflex threshold is illustrated in Figure 13. Note that response time decreases as frequency increases. Post facto examination of the difference between frequencies using Student-Newman-Keuls procedure at $p < 0.05$ indicates that response times (measured to 10% of maximum negative amplitude) to stimuli centered at 500 Hz were significantly different from response times to stimuli centered at 1000 and 4000 Hz.

The effect of Bandwidth on the response time of the initial negative peak of the biphasic acoustic reflex at acoustic reflex threshold is illustrated in Figure 14, which plots the mean response time of the negative peak as a function of bandwidth. Post facto examination of the difference between bandwidths using Student-Newman-Keuls procedure at $p < 0.05$ indicates that response times to stimuli with the two narrowest bandwidths (1/6 and 1/3 octave) were significantly longer than response times to stimuli with the widest bandwidth (4 octaves). Response times to 1 and 2 octave wide signals were not significantly different from each other. Nor were they significantly different from the response times to any other bandwidth used in eliciting the acoustic reflex at acoustic reflex threshold.

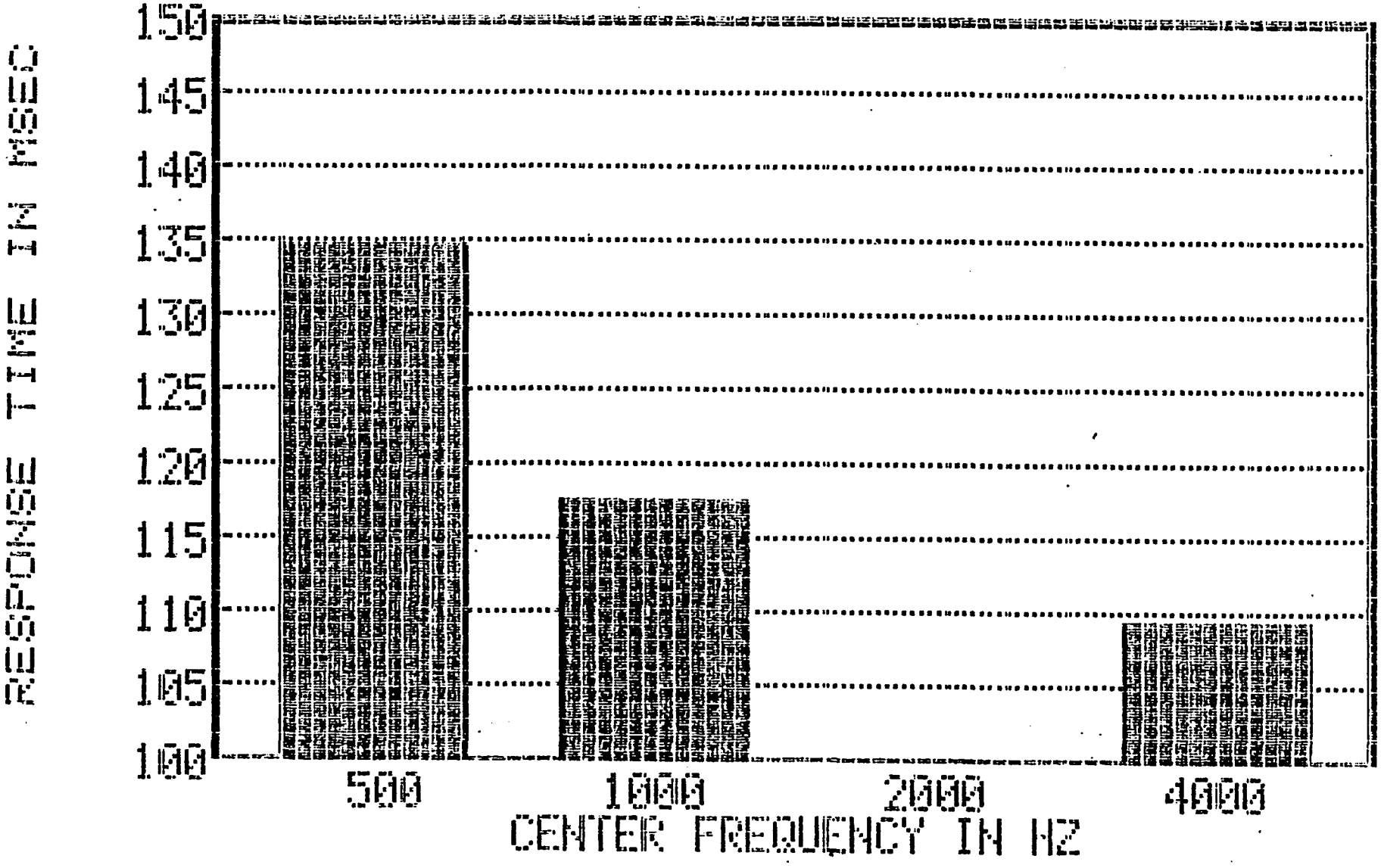


Figure 13. Mean response time to 10% of maximum negative amplitude as a function of frequency. N = 5 subjects with biphasic responses. Stimulus level = ART.

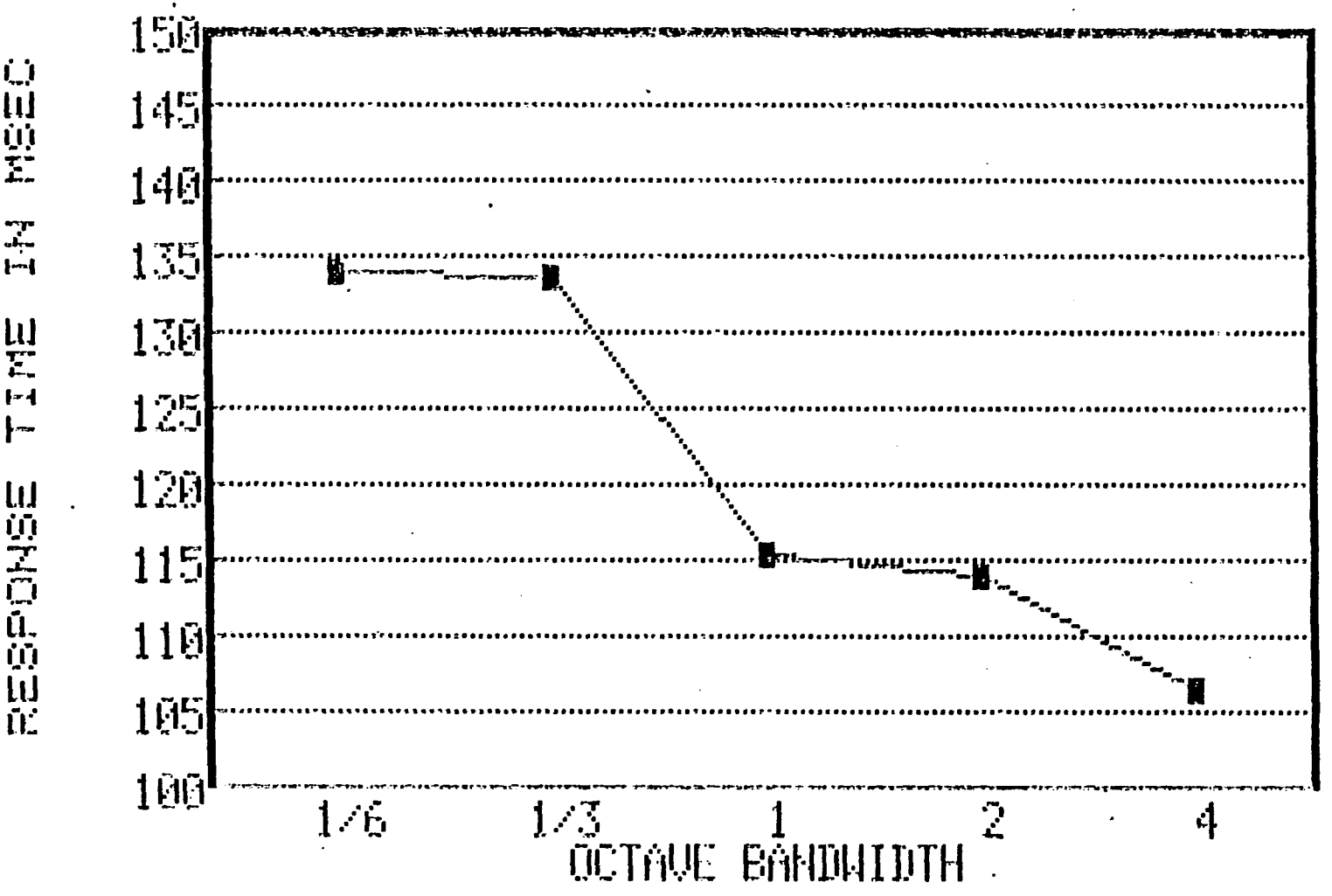


Figure 14. Mean response time to 10% of maximum negative amplitude as a function of bandwidth. N = 5 subjects with biphasic responses. Stimulus level = ART.

Table 12 shows the analysis of variance for the data obtained 10 dB above acoustic reflex threshold from the five subjects with biphasic responses. Bandwidth was the only variable that had a significant effect on the response time of the initial negative peak of the response. Time Order Replication, Frequency, and their interactions did not have a significant effect the response time of the negative portion of the biphasic response when the reflex was elicited by signals presented 10 dB above acoustic reflex threshold.

The effect of Bandwidth on the response time of the negative peak at 10 dB above acoustic reflex threshold is shown in Figure 15, which plots response time as a function of bandwidth for the five biphasic subjects. The response time for the widest bandwidth was 9.0 msec faster than the response time for the narrowest bandwidth.

Post facto analysis of the data using the Student-Newman-Keuls procedure for the 0.05 level indicated that the three narrowest bandwidths (1/6, 1/3 and 1 octave) produced significantly shorter response times than the two widest bandwidths (2 and 4 octaves). Stated differently, once the bandwidth of the stimulus was wider than 1 octave around a 500, 1000 or 4000 Hz signal, the response time of the initial negative peak of a biphasic response was significantly shorter than it was for stimuli narrower than 1 octave wide.

Table 12
Analysis of Variance with Repeated Measures.
Mean Response Time to 10% of Maximum Negative Amplitude of the Biphasic Response. N = 5 Subjects with Biphasic Responses. Stimulus Level = 10 dB above ART.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Time Order	T	1	665.7	1.32	
Frequency	F	2	606.5	0.62	
	TF	2	190.5	1.37	
Bandwidth	B	4	544.8	5.59	<.01
	TB	4	49.5	0.74	
	FB	8	135.2	0.82	
	TFB	8	65.7	0.84	

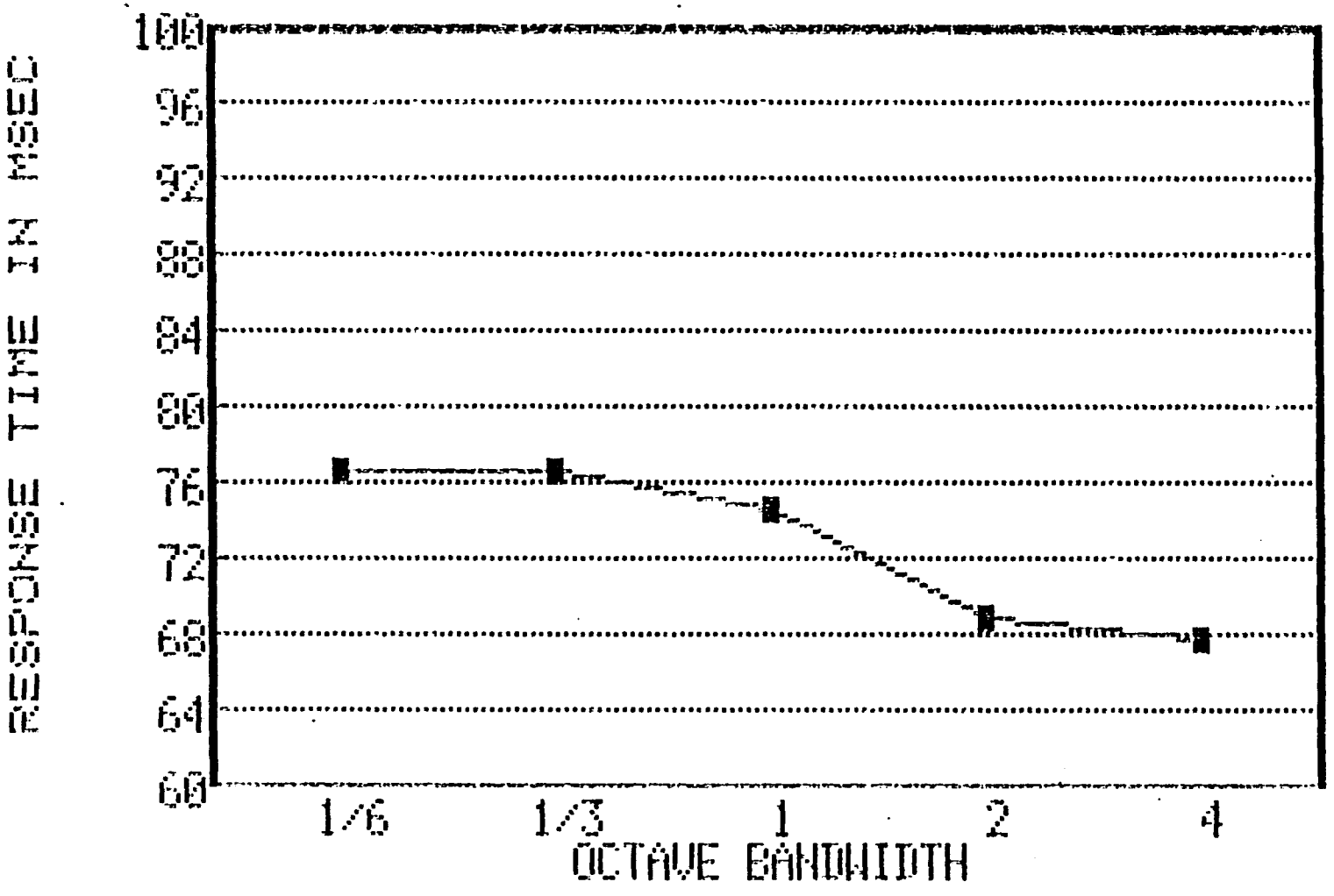


Figure 15. Mean response time to 10% maximum negative amplitude as a function of bandwidth. N = 5 subjects with biphasic response. Stimulus level = 10 dB above ART.

In summary, the results of the analyses of the response time of the biphasic acoustic reflex were as follows:

1. When response time was measured to the point on the response curve corresponding to 10% of maximum positive reflex amplitude, the biphasic response time to threshold level stimuli was significantly longer than the uniphasic response time. However, when the stimulus level was raised to 10 dB above acoustic reflex threshold, there were no significant differences between the biphasic and uniphasic reflex response times.

2. When response time was measured to the point corresponding to 10% of maximum negative reflex amplitude, changes in center frequency and bandwidth resulted in significant changes in acoustic reflex response time for signals presented at acoustic reflex threshold. Signals centered at 500 Hz yielded significantly longer response times than signals centered at 1000 or 4000 Hz. Narrow band signals (1/6 and 1/3 octave wide) yielded significantly longer response times than 4 octave bandwidth signals at acoustic reflex threshold.

3. When response time was measured to the initial negative peak of the biphasic reflex at 10 dB above acoustic reflex threshold, only changes in bandwidth resulted in significant changes in reflex response time. Once the bandwidth of the stimulus was wider than 1 octave, response time was significantly shorter than it was for stimuli narrower than 1 octave in width.

THE EFFECTS OF THE EXPERIMENTAL VARIABLES ON ACOUSTIC REFLEX THRESHOLD

An analysis of variance of the main effects of response type, frequency, time order, bandwidth and their interactions on the acoustic reflex thresholds (in dB SPL) of all subjects was done. This analysis allowed for comparison to the published literature. It was also of interest to compare the effects of frequency and bandwidth on ART with the effects of the same factors on acoustic reflex response time. Table 13 shows the results of this analysis.

Table 13 shows that the effects of Frequency and Bandwidth were significant at the 0.05 level. The effect of bandwidth on acoustic reflex threshold is highly significant ($F = 50.69$, $p < 0.00001$). Response Type (uniphasic vs. biphasic) and Time Order did not significantly affect the acoustic reflex threshold in these subjects.

The interactions of Time Order by Bandwidth and Frequency by Bandwidth were also highly significant (TB F value = 4.38, $p < 0.0062$; FB F value = 11.06, $p < 0.00001$).

As noted previously, because each frequency was measured on a different day, frequency differences were confounded with order differences. For these data, the ANOVA shows no significant Time-Order effect.

Table 13
Analysis of Variance with Repeated Measures.
Mean Acoustic Reflex Threshold in dB SPL. N = 10.

SOURCE OF VARIABILITY		DF	MEAN SQUARES	F VALUE	TAIL PROBABILITY
Response Type	R	1	607.7	0.51	
Time Order	T	1	102.0	0.75	
	TR	1	148.4	1.10	
Frequency	F	2	367.5	4.74	<.05
	FR	2	79.0	1.02	
	TF	2	29.7	1.36	
	TFR	2	1.8	0.08	
Bandwidth	B	4	413.4	50.69	<.0001
	BR	4	4.1	0.51	
	TB	4	12.8	4.38	<.01
	TBR	4	3.8	1.32	
	FB	8	54.6	11.06	<.0001
	FBR	8	5.1	1.05	
	TFB	8	1.5	0.52	
	TFBR	8	1.4	0.49	

Post facto examination of the acoustic reflex threshold differences between frequencies using the Student-Newman-Keuls procedure at 0.05 indicated that ARTs for 1000 Hz were significantly different from ARTs for 4000 Hz.

Figure 16 shows ART as a function of frequency. Note that the acoustic reflex was most sensitive to signals centered at 1000 Hz, and was less sensitive to lower and higher frequency signals.

Post facto analysis of the differences in ARTs between bandwidths using the Student-Newman-Keuls procedure at the 0.05 level indicated that ARTs to 2 and 4 octave bandwidth signals (regardless of center frequency) were significantly different from each other and from ARTs to 1/6, 1/3, and 1 octave bandwidth signals. Stated differently, once signal bandwidth exceeded 1 octave, acoustic reflex threshold decreased significantly. The difference in sensitivity between the narrowest and widest bandwidth was 6.70 dB.

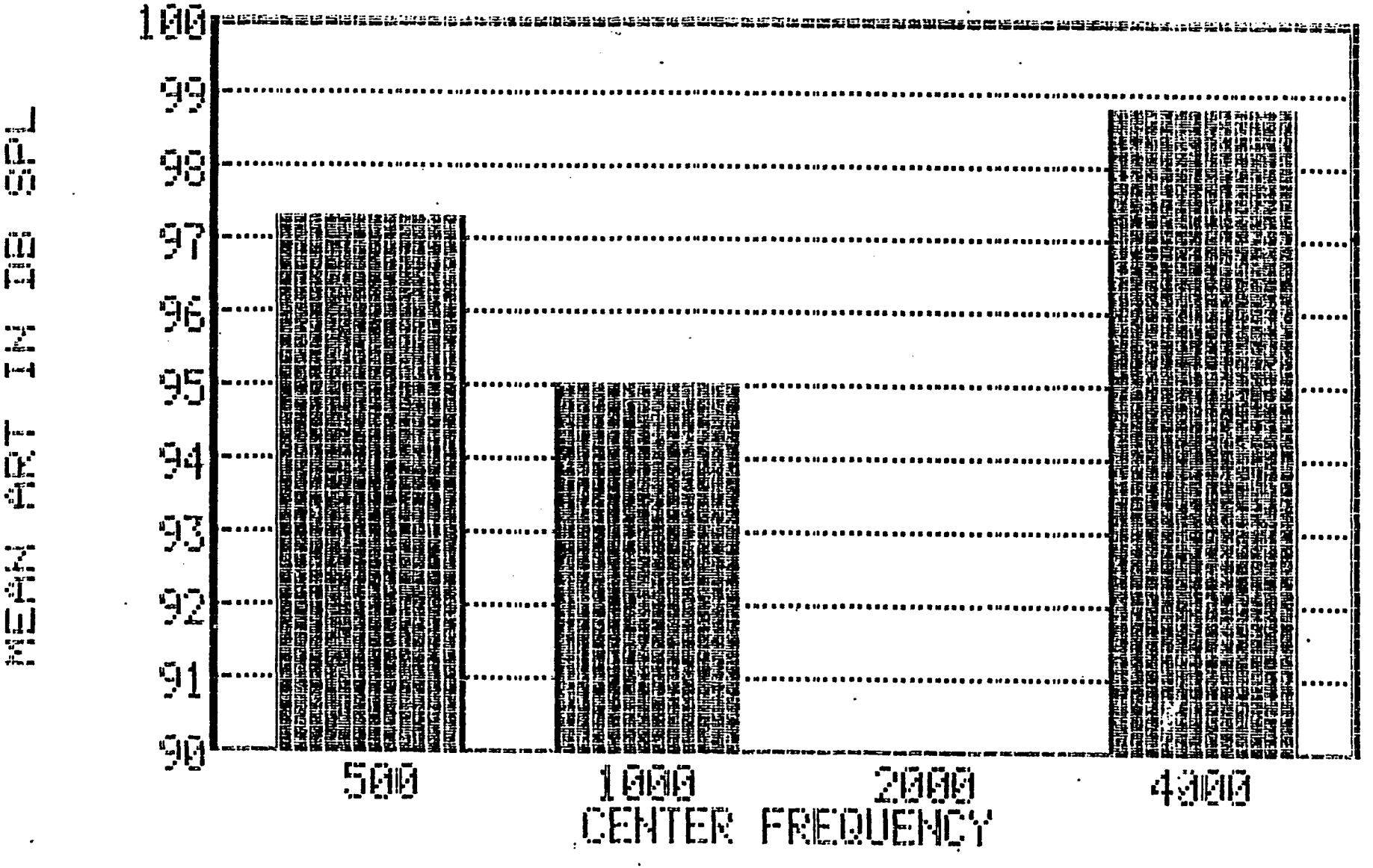


Figure 16. Mean acoustic reflex threshold in dB SPL as a function of center frequency. N = 10.

Figure 17 shows ART as a function of bandwidth. Note that the acoustic reflex was more sensitive to wider bandwidth signals than it was to narrower bandwidth signals.

Post facto examination of the FB interaction using the Student-Newman-Keuls preocedure at the 0.05 level indicated that for 500 Hz, the ART at the widest bandwidth (4 octaves) was significantly lower than it is at any other bandwidth. The difference in sensitivity between the ART to the widest bandwith and the ART for any other bandwidth was 5 dB. The same results were obtained at 1000 Hz. ARTs for stimuli from 1/6 octave to 2 octaves were essentially the same while at 4 octaves, ARTs decreased by 5 dB.

For 4000 Hz, the post facto examination of the data yielded a somewhat more complicated pattern. ARTs to the 1/6, 1/3, and 1 octave bandwidth signals did not significantly differ from each other, while ARTs at 2 and 4 octave bandwidths did differ significantly from each other and from the three narrower bandwidths. Stated differently, for signals centered at 4000 Hz, the acoustic reflex was more sensitive to signals with bandwidths wider than one octave, while at 500 and 1000 Hz, it was more sensitive to signals wider than two octaves.

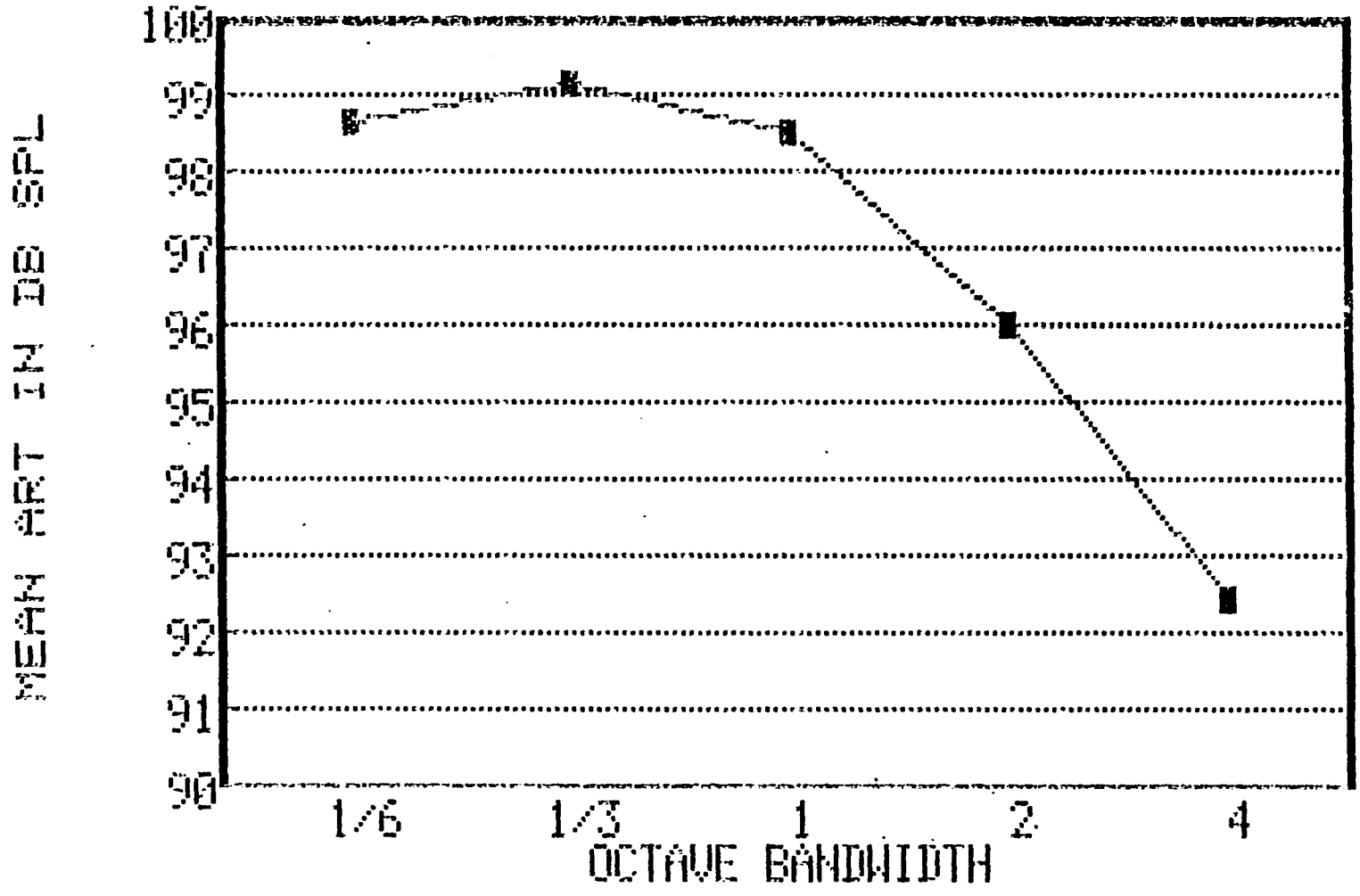


Figure 17. Mean acoustic reflex threshold in dB SPL as a function of bandwidth. N = 10.

Figure 18 shows ARTs as a function of signal bandwidth for the three center frequencies (500, 1000 and 4000 Hz). As described, at 500 and 1000 Hz, ARTs remain relatively constant for signals up to two octaves wide and were about 5 dB more sensitive at wider bandwidths. At 4000 Hz, the increase in sensitivity occured for signals wider than one octave. The rate of increase in sensitivity for 4000 Hz signals was also about 5 dB per octave.

Post facto examination of the interaction between time order and bandwidth using the Student-Newman-Keuls procedure for the 0.05 level yielded the following results. During the initial test, ARTs to 1/6, 1/3, and 1 octave bandwidth signals were not significantly different from each other while the ARTs to 2 and 4 octave bandwidth signals were significantly different from each other and from the three narrower bandwidths as well. That is, the acoustic reflex was more sensitive to signals wider than 1 octave during the initial test. The decrease in sensitivity for the wider band signals was about 4 dB per octave for the test measure.

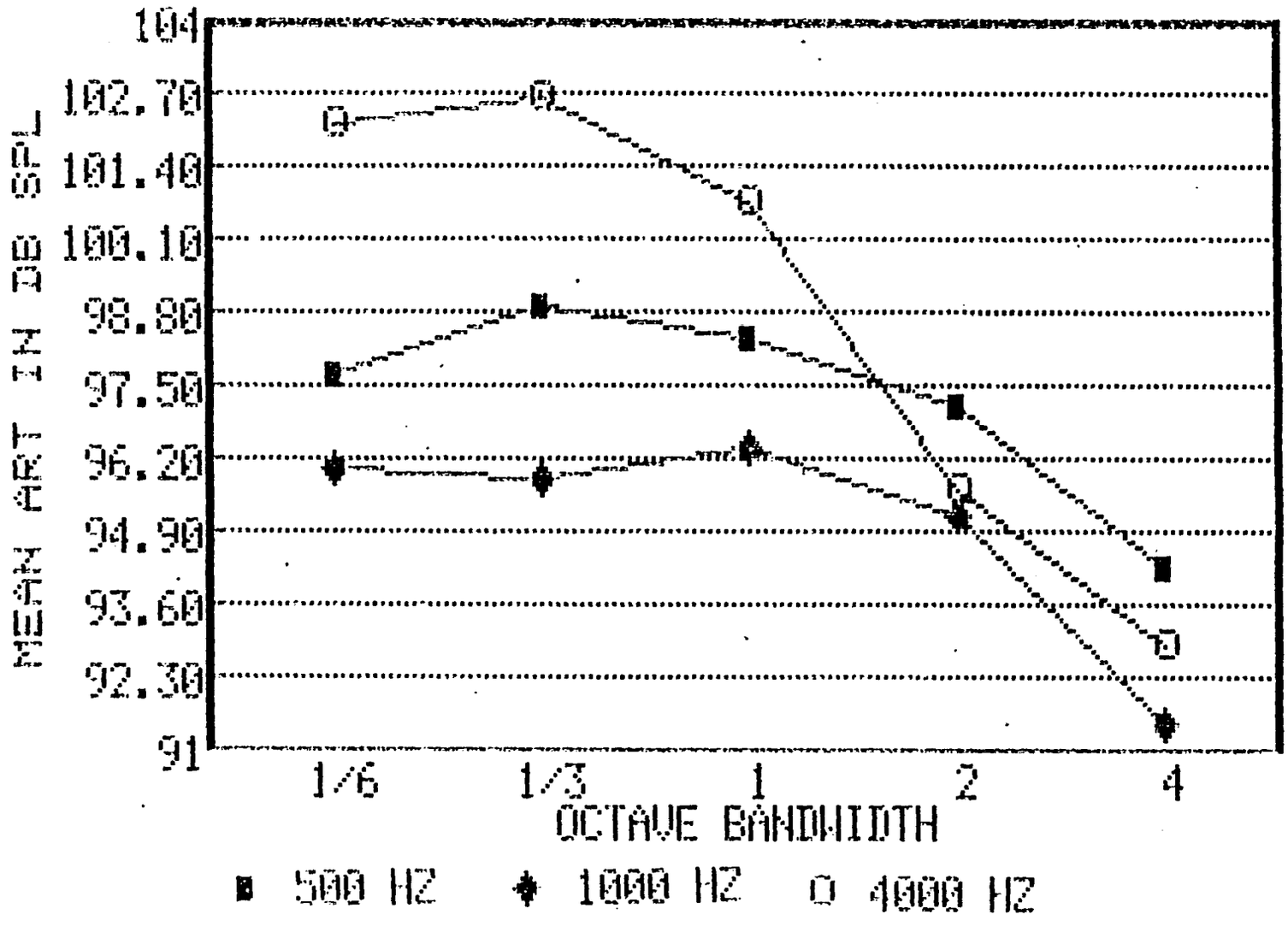


Figure 18. Mean acoustic reflex thresholds in dB SPL as a function of bandwidth for 500, 1000 and 4000 Hz center frequencies. N = 10.

During the retest, the pattern of results was the same, but the change in sensitivity was 3 dB per octave. The TB interaction appears to be due to the fact that during the initial test, the ART was highest for the 1/3 octave band signal while during the retest, the ART was highest for the 1 octave band signal.

The TB interaction is shown in Figure 19. As can be seen, for both test and retest, the increase in reflex sensitivity occured when signal bandwidth exceeded one octave, after which there was a 3 - 4 dB decrease in reflex sensitivity per octave.

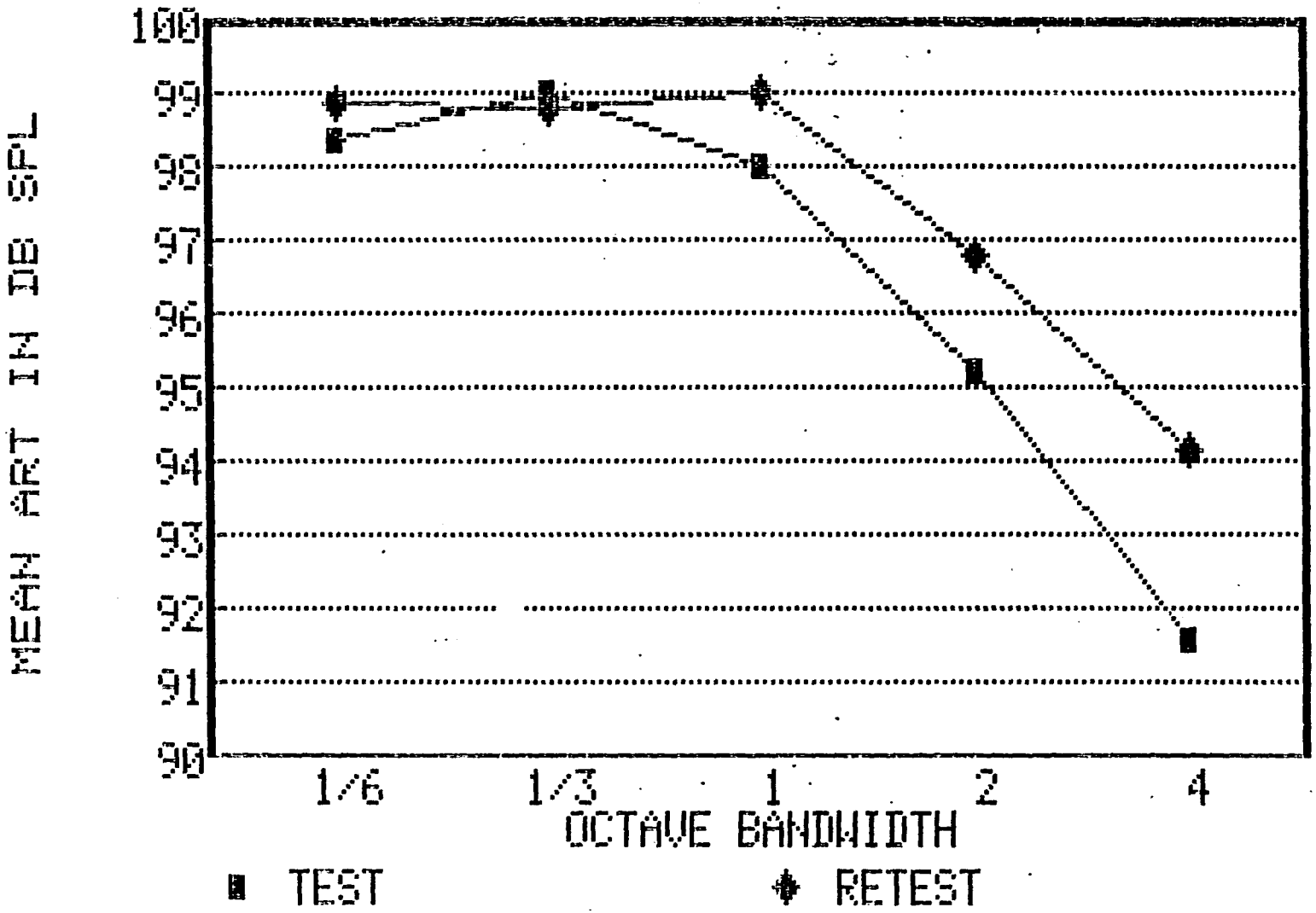


Figure 19. Mean acoustic reflex threshold as a function of bandwidth for two replications. N = 10

The results of the analysis of variance with repeated measures for the effects of response type, frequency, bandwidth, time order and their interactions on the acoustic reflex threshold may be summarized as follows:

1. The acoustic reflex was significantly more sensitive to signals centered at 1000 Hz than it was to signals centered at 4000 Hz.

2. There was a critical bandwidth for acoustic reflex threshold, and the width of that bandwidth varies with frequency. The rate of decrease in acoustic reflex threshold for signals wider than the critical band was 3 - 4 dB per octave.

CHAPTER 6

DISCUSSION

This study examined the effects of stimulus center frequency and bandwidth on acoustic reflex response time at acoustic reflex threshold (ART) and at 10 dB above ART in subjects with uniphasic and biphasic reflexes. Related issues were the reliability of response time measurements and the similarities and differences between acoustic reflex threshold and acoustic reflex response time.

There were important differences between the response time data obtained at ART and at 10 dB above ART. At ART, acoustic reflex response time was significantly more variable than it was at 10 dB above ART. At ART, stimulus center frequency did not significantly affect reflex response time while at 10 dB above ART it did. Likewise, stimulus bandwidth did not significantly affect response time at ART, but at 10 dB above ART there was a significant bandwidth effect that differed with center frequency.

There were also important differences between acoustic reflex threshold and acoustic reflex response time. The frequency that caused the fastest response was not the frequency to which the acoustic reflex was most sensitive. While there was a critical bandwidth for acoustic reflex threshold at all three frequencies tested, there was only a bandwidth effect for acoustic reflex response time at one of the three stimulus center frequencies.

There were no significant differences between the biphasic and uniphasic responses, both in terms of acoustic reflex threshold and acoustic reflex response time at 10 dB above ART. There were some interesting similarities between the response time of the initial negative portion of the biphasic response and the reflex threshold. The details and implications of the effects described above will now be discussed.

EFFECTS OF STIMULUS CENTER FREQUENCY ON ACOUSTIC REFLEX RESPONSE TIME

No systematic frequency effect was found at ART. However, there was a significant interaction between Time Order and Frequency, indicating that the effect of frequency on the response time at ART differed from one measurement to another, and that the frequency effect at ART was variable.

At 10 dB above ART, the effect of stimulus center frequency was highly significant. The interaction between Time Order and Frequency was not significant. This suggests that changes in stimulus center frequency significantly changed acoustic reflex response time at 10 dB above ART and that this effect was stable from one test session to another, unlike the variable frequency effect at acoustic reflex threshold.

Thus, for this effect, we noted the variability in the response time of the acoustic reflex at ART that is often reported in the literature pertaining to acoustic reflex latency and response time. At 10 dB above ART, however, the response time measurements were stable and repeatable.

Since the measurements at ART and at 10 dB above ART were done in the same session, these findings were not confounded by any time order effect. It should also be noted that the F values and tail probabilities at 10 dB above ART are well in excess of the corresponding F values and tail probabilities at ART.

The observed frequency effect warrants further discussion. The 500 Hz center frequency was found to result in a faster mean acoustic reflex response time (145.54 msec) than 1000 Hz (156.07 msec) and 4000 Hz (177.40 msec). These results are at variance with what would be expected if acoustic reflex response time were a reflection of the traveling wave within the cochlea, and with the effect of frequency on acoustic reflex latency reported by Ruth and Niswander (1976) and Clemis and Sarno (1980).

The contrast between the data in this study and the data in the two studies cited may be explained by differences in equipment and reflex response measurement. Both of the cited studies measured the acoustic reflex latency as the time period between stimulus onset and reflex onset, rather than to a point along the reflex curve. As described previously, the onset measurement was difficult to make from a practical standpoint because of the gradual onset and low amplitude of the reflex. As noted by Borg (1972), Moller (1974), Jerger and Hayes (1983), Lilly (1984) and Stach and Jerger (1984), measurements to a fixed percentage of maximal amplitude are preferable because they circumvent these practical problems.

The measurement used in this study, (time period between stimulus onset and 10% of maximum reflex amplitude) is currently being considered as a standard (Lilly, 1984). It is more practical than the onset measurement for the reasons mentioned above. In addition, it is more precise than the onset measurement. Precision increases as the slope of the curve increases, and the reflex-response curve is steeper at a percentage of maximal amplitude than it is at reflex onset.

Stach and Jerger (1984) state that "...the maximal value of temporal measurement lies not in the latency to onset but rather in the rise time characteristics of the reflex waveform" (p. 292). Measuring to a percentage of maximum amplitude takes the rise time characteristics of the reflex into account, and as noted by Moller (1962) and Borg (1972), the rise time of the reflex is faster for low frequencies than it is for high frequencies. Measurements to a fixed percentage apparently reflect the faster reflex rise time for the low frequencies.

The reason for the variance between the data in this study and the traveling wave phenomenon is found in the literature (Moller, 1983; Borg,1976) and has been discussed recently by Jerger, Stach and Oliver (1984A). It is well known that the acoustic reflex functions as a feedback system, regulating transmission of low frequencies to a greater extent than it regulates high frequency transmission to the cochlea. The faster onset at the low frequencies, as

well as the damped oscillation that occurs at reflex onset (Moller, 1962, Borg, 1972) is caused by the attenuation of the reflex eliciting signal by the reflex itself. Jerger, et al. (1984A) proposed that if the a 500 Hz signal were not attenuated by the reflex itself, then it would have a longer rise time and thus a longer response time. However, since the reflex does attenuate the low frequencies more than the high frequencies, it is not unexpected that reflex response time increases with increases in stimulus frequency. The same researchers noted that above 2000 Hz, there is not much difference in acoustic reflex response time; the maximal attenuation of the reflex is for frequencies below 2000 Hz.

The data reported in this study are in good agreement with the data of Church and Cudahy (1984) and Jerger, et al. (1984). Perhaps this is so because all three studies measured the reflex response time to a percentage of maximum amplitude. Although Church and Cudahy (1984) used a complicated formula to determine the slope function of the acoustic reflex and measured to a different point along the response curve than the one used in this study, they also found that the response at 500 Hz was 10 msec faster than the response at 2000 Hz. Jerger, et al. (1984A) also reported a 10 msec difference in the response times of the acoustic reflex at 500 and 2000 Hz. They measured to the point on the reflex response curve corresponding to 20% of maximum positive amplitude.

Thus, when the response time of the acoustic reflex was

measured to a fixed percentage of reflex amplitude, regardless of the percentage chosen, the effect of stimulus frequency was such that the response time was faster at 500 Hz than it was at higher stimulus frequencies.

In conclusion, the effect of stimulus center frequency on acoustic reflex response time was not observed at ART, but was observed at 10 dB above ART. Response time was faster for signals centered at 500 Hz than it was for signals centered at 1000 or 4000 Hz. This finding suggests that the response time of the reflex was not governed by the traveling wave within the cochlea but by the feedback characteristics of the reflex itself.

EFFECT OF BANDWIDTH ON THE RESPONSE TIME OF THE ACOUSTIC REFLEX

Changes in bandwidth did not significantly affect acoustic reflex response time at ART, but there was a bandwidth effect (that differed with center frequency) at 10 dB above ART.

Changes in mean response times caused by changes in bandwidth were only significant for the 4000 Hz center frequency. For both the 500 Hz and 1000 Hz center frequencies, response times at all five bandwidths were essentially the same. For the 4000 Hz center frequency, however, the mean response times at the two widest bandwidths (2 and 4 octaves) were significantly shorter than the mean response times at the 1/6, 1/3, and octave wide bands.

Consideration of the experimental stimuli sheds some light on the possible cause of the significant bandwidth effect at 4000 Hz. As noted previously, the upper cutoffs of the two widest bandwidths centered at 4000 Hz were determined by the frequency response characteristics of the earphone used in the experiment, and thus did not exceed approximately 6400 Hz. While the 1/6, 1/3 and 1 octave wide signals centered at 4000 Hz did not contain energy at frequencies lower than 2800 Hz, the 2 and 4 octave bands had lower cutoffs of 2005 Hz and 1007 Hz respectively. It is possible that the faster response times for the wide band signals centered at 4000 Hz result from the energy at and

below 2000 Hz contained in those bands, as well as from the lack of frequencies above 6400 Hz in those bands. Given the significant frequency effect found in this and previous experiments, with shorter response times for low frequencies than for high, it is not surprising that the noise bands containing no energy below 2800 Hz have longer response times than the two bands with reduced upper cutoffs and lower cutoffs at or below 2005 Hz.

Nor is it surprising that there was not a significiant bandwidth effect for the noise bands centered at 500 and 1000 Hz. For all but the widest band centered at 1000 Hz, none of the upper cutoffs exceed 2000 Hz. The low frequency energy present in each noise band may have accounted for the consistency of response times across bandwidths for those two center frequencies.

There are no previous studies available regarding the effect of bandwidth on acoustic reflex response time or latency to which to compare the data obtained in this study.

In conclusion, the bandwidth effect was subtle and was noted for only one frequency. While there was not a significant bandwidth effect for signals centered at 500 and 1000 Hz, significantly shorter response times were noted for the 2 and 4 octave wide signals centered at 4000 Hz. These were the only two 4000 Hz signals containing energy in the frequency region below 2000 Hz. Considering the frequency effect noted in the previous section, these findings suggest that the low frequency energy present in those two signals

accounts for the observed effect.

REPEATABILITY OF RESPONSE TIME MEASURES

The effect of frequency on reflex response time at ART varied significantly from measure to measure. However, at 10 dB above ART, there were no significant differences between mean response times measured on different days, nor was there a significant interaction between Time Order and Frequency.

It appears that acoustic reflex response time is a more stable measure when measured at supra-threshold levels than when measured at threshold level. The standard deviation of reflex response-time at ART was 65.75 msec while at 10 dB above ART it was 33.97 msec. Note that the response time at ART and at 10 dB above ART were tested in the same session so that these findings were not confounded with any time order effect. Perhaps the difference in variability is explained by the statistical nature of threshold and by the behavior of the reflex around threshold. By definition, a response at threshold will be present only 50% of the time. When present, the threshold response is small in amplitude and gradual in onset. Around threshold, a one or two dB difference in stimulus level may affect the very presence or absence of the response. However, when the stimulus level is raised to suprathreshold levels, a one or two dB difference in level will not dramatically influence the amplitude or

onset of the response, and the susceptibility of the measurement to error is reduced.

The greater variability in response time at ART than at supra-threshold levels has been reported in almost every study in the literature and has implications for the clinical use of acoustic reflex response time measurements. If the morphology of the reflex has diagnostic significance, as suggested by the work of Clemis and Sarno (1980, 1980A) and Stach and Jerger (1984), it should be recorded at least 10 dB above reflex threshold. In this way, the response is more repeatable and less susceptible to observer error.

In conclusion, acoustic reflex response time was variable at ART but was stable at 10 dB above ART. At threshold, a one or two dB difference may affect the presence of absence of the response, but above threshold, small differences in level probably do not affect the response shape or onset. The clinical implication of this finding is that during diagnostic testing, acoustic reflex response time should be measured at suprathreshold levels.

RESPONSE TIME VS. REFLEX THRESHOLD

The mean acoustic reflex thresholds (in dB SPL) obtained in this study were in excellent agreement with those reported in the literature, as were the measured effects of center frequency and bandwidth on the ART. As reported by Jepsen (1963), Peterson and Liden (1972), and Margolis and Popelka (1975), the sensitivity of the acoustic reflex was greatest (and ART was lowest) at the frequency to

which average normal hearing is the most sensitive (1000 Hz). Compared to 1000 Hz, 500 and 4000 Hz signals required an average of 2 - 4 dB more intensity to elicit the reflex response.

The present experiment replicated the bandwidth effect for acoustic reflex threshold previously reported in the literature (Flottorp, et al., 1971; Djupesland and Zwislocki, 1973; Popelka, et al., 1976; Margolis, et al., 1980). There seems to be a critical bandwidth for acoustic reflex threshold, and the width of the band varies with frequency. At 500 and 1000 Hz, the critical band was two octaves wide while at 4000 Hz it was one octave wide. Beyond the critical band, the rate of decrease in acoustic reflex threshold was 3 - 4 dB per octave, in good agreement with the data of Flottorp, et al. (1971), Popelka, et al. (1976), and Margolis, et al. (1980).

Given the good agreement between the threshold data obtained in this study and that in the literature, it seems reasonable to compare the effects of frequency and bandwidth on acoustic reflex threshold with the effects of frequency and bandwidth on acoustic reflex response time.

1. The frequency that caused the fastest response time (500 Hz) was not the frequency to which the acoustic reflex was most sensitive.

2. While there was an observable critical band for acoustic reflex threshold at 500 and 1000 Hz, there was not anobservable critical band for acoustic reflex response time

at those frequencies.

3. There was a significant bandwidth affect for acoustic reflex response time at 4000 Hz. Signals wider than an octave resulted in response times that were about 15 msec faster than narrower band signals. The critical band for acoustic reflex threshold for signals centered at 4000 Hz was also observed for the octave wide band, with a 3 - 4 dB per octave increase in reflex sensitivity for signals beyond this bandwidth.

The important implication of these findings is that the acoustic reflex threshold mechanism is not the same as the acoustic reflex timing mechanism, at least for the onset timeing of the reflex. As noted by Dallos (1973), threshold is crossed when the integrated energy reaching the cochlea exceeds a certain level. Just as with auditory thresholds, this level varies with frequency in such a way that sensitivity is greatest for signals in the mid-frequency range. However, the morphology of the response, with regard to timing apparently has less to do with the energy reaching the cochlea than it has to do with the attenuation characteristics of the reflex. The reflex attenuates the low frequencies more than the high frequencies. It seems plausible that a faster onset and rise time in response to low frequency stimulation is built into its feedback system.

The lack of a critical band for response time at 500 and 1000 Hz, even though there was a critical band for acoustic reflex threshold at those frequencies, may be

explained in the same way. Wider bandwidths caused greater integrated energy to reach the cochlea, causing the reflex to be activated at lower levels. The timing of the response remained the same across bandwidths because low frequencies were present in all noise bands centered at 500 and 1000 Hz. The bandwidth effect for response time at 4000 Hz may be attributed to the fact that bands narrower than an octave did not contain sufficient low frequency energy while the wider bandwidths did. Since response time was faster for the low frequencies, the reflex response was faster when those frequencies were present in the stimulus.

In conclusion, the signals to which the reflex threshold was most sensitive were not the ones which caused the fastest response. This finding suggests that the reflex threshold and the reflex timing mechanisms are different. Reflex threshold appears to depend on the energy reaching the cochlea while reflex timing appears to depend on the feedback and attenuation characteristics of the reflex.

THE BIPHASIC REFLEX VS. THE UNIPHASIC REFLEX

Response type did not significantly influence the timing of the major observed portion of the reflex response at 10 dB above ART. Nor did response type significantly affect acoustic reflex thresholds. However, at ART, subjects with biphasic responses had significantly longer response times than those with uniphasic responses.

The longer biphasic response time at ART is not surprising considering that the positive portion of the

reflex occurs later in time during the biphasic response than during the uniphasic response. However, when stimuli were raised to 10 dB above ART, the difference in response time between the two response types was not significant. It seems that at supra-threshold levels, the timing of the major observed portion of the response is the same for the two response types.

The small initial negative portion of the biphasic reflex did not behave in the same way as did the major positive portion of both the uniphasic and biphasic reflex. At acoustic reflex threshold, the response time to 10% of the maximum negative amplitude decreased with increasing frequency (in exact opposition to the increase in positive wave response time with increasing frequency). Perhaps the mechanism that underlies the initial negativity of the biphasic acoustic reflex, be it cochlear uncoupling, a tensor response, or a generalized startle that causes a tensor response, is a reflection of the traveling wave within the cochlea. In any case, the significant frequency effect on the negative portion of the biphasic reflex is not present when the stimulus level is raised to 10 dB above acoustic reflex threshold.

At acoustic reflex threshold and 10 dB above acoustic reflex threshold, the response time to 10% of the maximum negative amplitude was significantly shorter to wide bandwidth signals than it was to narrow bandwidth signals. The theoretical implication of these findings is that the

mechanism controlling the response time of the initial negative portion of the biphasic reflex is more like the threshold mechanism than it is like the timing mechanism described in the previous section. That is, it has more to do with the integrated energy reaching the cochlea and the movement of that energy within the cochlea than it does with the selective frequency attenuation of the reflex.

These findings also have important clinical implications. The clinician need not be concerned with the influence of the biphasic response on acoustic reflex threshold or acoustic reflex response time during diagnostic procedures, provided that response time is measured at least 10 dB above acoustic reflex threshold to a point along the reflex curve corresponding to 10% of maximum positive reflex amplitude. As a clinical tool, the response-time measurement is reliable despite differences between response types (uniphasic vs. biphasic). Note that there was only a 2.75 msec difference between the standard deviations of the five uniphasic subjects and the five biphasic subjects.

In conclusion, the type of response exhibited did not significantly affect acoustic reflex thresholds or the timing of the major observed portion of the response at 10 dB above ART. Thus, the effects of changes in stimulus frequency and bandwidth on the biphasic reflex are the same as their effects on the uniphasic reflex. The effects of changes in frequency and bandwidth on the initial negative

portion of the biphasic response are similar to the effects of those variables on ART. The theoretical implication of these findings is that the mechanism that controls the small initial negative portion of the biphasic response is more like the threshold mechanism than it is like the timing mechanism of the acoustic reflex.

There are two important clinical implications of these findings. First, the presence of a biphasic response does not seem to be a significant issue relative to ART or response time at 10 dB above ART. Second, the response time of the biphasic reflex should be measured in the same way that the response time of the uniphasic reflex is measured: to 10% of the maximum positive reflex amplitude at 10 dB above ART.

RECOMMENDATIONS FOR FUTURE RESEARCH

Using computer averaging and defining acoustic reflex response time to 10% of maximum positive reflex amplitude proved to be valuable methods of overcoming two of the major methodological problems encountered in measuring the acoustic reflex. These techniques should be considered for use in future studies of acoustic reflex morphology. The procedure devised for determining equipment response time and time constant was simple to use and extremely sensitive. It should also be considered for use in future studies to determine the response time and time constant of reflex measuring devices.

Further information should be obtained about the effect

of frequency on acoustic reflex response time at frequencies other than the three frequencies studied here. This would give a more complete picture of the effect of frequency on acoustic reflex response time. The use of more detailed changes in the bandwidths would also give a better picture of the response time as a function of the bandwidth.

Further information about the effect of bandwidth on acoustic reflex response time using tonal complexes of varying frequency separation would facilitate further comparison to the data regarding the effect of bandwidth and spectral density on acoustic reflex threshold.

Finally, the effect of subject variables such as age and hearing impairment (sensorineural and retrochlear) on the response time of the acoustic reflex needs to be further specified if acoustic reflex response time measurements are to be clinically useful and diagnostically significant.

CONCLUDING REMARKS

It is important to note that acoustic reflex response time, measured at 10 dB above acoustic reflex threshold, was the most precise, repeatable measurement in this study. It also revealed aspects of the auditory system that were different from those revealed by the ART. Specifically, ART and acoustic reflex response time are affected differently by stimulus frequency and bandwidth.

If the feedback hypothesis advanced by Borg (1976), Moller (1983) and Stach and Jerger (1984A) is correct, this measurement could be useful in testing the intactness of the

reflex feedback loop, and may provide important diagnostic information if added to the audiologic test battery. As noted above, the effect of sensorineural and retrocochlear hearing loss on the response time of the acoustic reflex needs to be further specified.

CHAPTER 7

SUMMARY AND CONCLUSIONS

While the effects of stimulus frequency and bandwidth on the threshold of the human acoustic reflex in normal and hearing impaired listeners has been examined in depth, very little research has been done to determine the effects of frequency and bandwidth on the response time of the acoustic reflex. Previous studies of acoustic reflex latency and response time were plagued by methodological problems. Poor signal-to-noise ratios contaminated reflex response curves. The response delays and filter time constants of the measuring equipment were not consistently determined or accounted for in response time measurements. Indeed, the very definition of latency was a problem.

Traditionally, the term latency referred to the time period between signal onset and reflex onset. However, the point of onset is difficult to find in a noisy curve with gradual onset and low amplitude, and it may be contaminated by equipment artifact.

In this study, three steps were taken to remedy the above noted methodological problems. First, computer averaging was used to improve the signal-to-noise ratio of the response curve. Second, a simple, yet very precise new procedure for determining the response time and the time constant of the measuring equipment was devised. Third,

response time was measured not to the point of reflex onset, but to the point along the reflex curve corresponding to 10% of the maximum reflex amplitude. This measurement is easier to make from a practical standpoint, and includes the morphologically important aspect of reflex rise time in the response time measurement.

The purpose of the present study was to determine the effects of stimulus center frequency and bandwidth on the response time of the acoustic reflex. Related issues were the repeatability of the response time measure, the similarities and differences between the uniphasic and the biphasic response and the comparison of frequency and bandwidth effects of acoustic reflex threshold with frequency and bandwidth effects on acoustic reflex response time.

Ten adults with normal hearing and negative otological history, five of whom exhibited a biphasic response and five of whom exhibited a uniphasic response, were the subjects in this experiment.

Experimental stimuli consisted of five signal bandwidths centered at three frequencies presented at acoustic reflex threshold and 10 dB above acoustic reflex threshold. Each stimulus was 500 msec in duration.

Testing was done in a sound treated room. Sixteen reflexes to each stimulus were computer averaged, and the averaged response served as the data. Each subject was tested two times on separate occasions under each stimulus

condition.

Data were subjected to analysis of variance and covariance techniques. The Duncan Multiple Range Test and Student-Newman-Keuls Test were used to determine significant differences between frequencies and bandwidths.

FINDINGS

The data obtained in this study indicated that:

1. When measured at acoustic reflex threshold, the effect of frequency on acoustic reflex response time is not repeatable. However, at 10 dB above acoustic reflex threshold, the response time of the acoustic reflex is stable between measures, and is significantly faster for 500 Hz stimulation than it is for 4000 Hz stimulation.

2. At acoustic reflex threshold, the response time of the biphasic reflex is significantly longer than the response time of the uniphasic reflex. However, at 10 dB above acoustic reflex threshold, there are no significant differences between the response times of the two response types.

3. At acoustic reflex threshold, changes in bandwidth do not result in significant changes in acoustic reflex response time. At 10 dB above acoustic reflex threshold, changes in bandwidth do not cause significant changes in response time at 500 or 1000 Hz. At 4000 Hz, for signals wider than an octave, acoustic reflex response time is significantly shorter than it is for signals narrower than

an octave.

4. The small initial negative peak of the biphasic acoustic reflex behaves differently than the major observed positive portion of the response. The response time to 10% of maximum negative amplitude is faster for 4000 Hz than it is for 1000 or 500 Hz, and the negative peak response time is faster for signals wider than an octave than it is for signals narrower than an octave.

5. Differences in acoustic reflex threshold caused by changes in center frequency and bandwidth are not the same as differences in response time caused by changes in center frequency and bandwidth. First, the effect of frequency on reflex response time is opposite that of the effect of frequency on acoustic reflex threshold. Second, while there is a critical band effect for acoustic reflex threshold, there is not a critical band for acoustic reflex response time.

CONCLUSIONS

The results of this study have theoretical and practical implications. From a practical standpoint, the results indicate that the clinical measurement of acoustic reflex response time should be done at 10 dB above acoustic reflex thresold because of the variablility of the response at ART. In the normal ear, there are no significant differences between biphasic and uniphasic responses provided that the response time is measured to 10% of maximum positive reflex amplitude. Whether this is the case with sensorineural or

retrocochlear impairments has yet to be determined.

From a theoretical standpoint, the findings implicate differences between the underlying mechanisms of acoustic reflex threshold and acoustic reflex response time. There is a critical band for ART but not for reflex response time. The effect of frequency on the response time of the reflex is opposite the effect of frequency on the threshold of the reflex. This indicates that while acoustic reflex threshold varies with the amount of integrated energy reaching the cochlea, and is most sensitive to the middle frequency range, acoustic reflex response time varies with the attenuation characteristics of the reflex and is fastest at the frequencies that are most attenuated by reflex activation.

APPENDIX A
INSTRUCTIONS TO SUBJECTS

INSTRUCTIONS TO SUBJECTS

This is a test to see how long it takes a tiny muscle in your ear to react to brief, loud tones and noises. In order to measure this response, it is necessary for one ear to have a small plug in it. You may think of this plug as a camera, recording the response of the muscle.

You will hear the tones and noises in the other ear. If the sounds are ever at an uncomfortable level, please let me know.

The best recordings occur when the person being tested sits very still. There will be many breaks during which you may yawn, chew, cough, etc., but please try to sit very still during the testing.

Any questions?

REMEMBER:
TRY TO SIT STILL
REPORT UNCOMFORTABLE LOUD SOUNDS

APPENDIX B
SUBJECT CONSENT FORM

CONSENT FORM
ACOUSTIC REFLEX RESPONSE TIME STUDY

Consent to Act as a Research Subject in a Dissertation Project at the Graduate Center of the City University of New York

The purpose of this study is to determine the effects of various brief noises and tones on the time it takes a tiny muscle in your ear to have a reflex reaction. It is hoped that the results will add to our understanding of normal hearing and aid in the diagnosis of hearing loss. There are no direct benefits to you arising from your participation.

In order to measure the reflex response, it is necessary to place a small probe (earplug) in one ear canal and present the brief noises or tones to the other ear through an earphone. Some of the brief noises will be loud, but never at a hazardous or uncomfortable level. There are no known adverse effects with this type of testing, which is used routinely in screening for hearing impairment.

The researcher is a licensed and certified Audiologist (New York State License #377, CCC-A, 1976). Prior to your participation in the study, she will test your hearing and visually inspect your ear canals for any abnormalities.

No personal information gathered in this study will be disclosed to anyone unless it is made anonymous. The data in any publication of this study will remain anonymous.

You are free to withdraw your consent and discontinue your participation in this study at any time, without penalty.

AUTHORIZATION: I have read the above and agree to participate in the project described above. Its general purposes, potential benefits, and possible hazards and inconveniences have been explained to my satisfaction.

SIGNATURE: DATE:

APPENDIX C

Individual acoustic reflex response-times to 10% of maximum positive reflex amplitude (values in msec).

SUBJECT 1, UNIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	188	268	220	300	220
ART +10dB	180	160	136	140	140
1000 Hz					
ART	224	240	248	300	272
ART +10dB	176	172	192	188	192
4000 Hz					
ART	200	212	236	180	260
ART +10dB	200	184	192	176	180

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	196	280	188	280	362
ART +10dB	182	180	172	212	244
1000 Hz					
ART	342	240	352	298	364
ART +10dB	176	180	196	220	198
4000 Hz					
ART	360	372	300	260	200
ART +10dB	268	208	200	140	192

SUBJECT 2 UNIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	360	152	160	260	152
ART +10dB	88	104	80	96	74
1000 Hz					
ART	272	176	180	200	136
ART +10dB	110	94	122	140	112
4000 Hz					
ART	228	200	208	168	124
ART +10dB	184	116	156	148	104

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	160	160	180	180	200
ART +10dB	80	130	88	80	68
1000 Hz					
ART	248	240	296	220	184
ART +10dB	118	140	140	140	122
4000 Hz					
ART	280	380	340	260	192
ART +10dB	184	180	168	140	144

SUBJECT 3, UNIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	316	260	190	212	260
ART +10dB	148	124	128	110	112
1000 Hz					
ART	180	180	180	180	180
ART +10dB	120	122	126	128	116
4000 Hz					
ART	180	160	140	140	134
ART +10dB	128	120	120	116	120

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	200	220	202	252	248
ART +10dB	130	120	128	126	112
1000 Hz					
ART	268	200	220	190	168
ART +10dB	120	124	120	120	112
4000 Hz					
ART	160	160	152	180	144
ART +10dB	120	124	124	122	112

SUBJECT 4, UNIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	176	192	140	200	200
ART +10dB	104	100	92	108	124
1000 Hz					
ART	188	190	160	156	168
ART +10dB	90	100	110	110	102
4000 Hz					
ART	184	188	176	168	180
ART +10dB	132	120	140	130	142

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	178	142	172	164	220
ART +10dB	110	112	116	120	120
1000 Hz					
ART	180	180	140	156	170
ART +10dB	118	108	120	112	120
4000 Hz					
ART	140	168	152	168	190
ART +10dB	144	128	132	136	144

SUBJECT 5, UNIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	320	296	340	260	280
ART +10dB	188	176	196	180	194
1000 Hz					
ART	316	400	276	340	300
ART +10dB	184	198	160	170	170
4000 Hz					
ART	328	260	252	236	240
ART +10dB	232	244	208	216	160

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	256	308	244	228	316
ART +10dB	172	172	176	172	168
1000 Hz					
ART	224	388	240	324	224
ART +10dB	160	180	172	180	170
4000 Hz					
ART	340	360	360	284	200
ART +10dB	232	240	212	208	172

SUBJECT 6, BIPHASIC POSITIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	200	208	202	212	210
ART +10dB	130	136	132	132	140
1000 Hz					
ART	218	192	228	244	214
ART +10dB	146	152	158	148	164
4000 Hz					
ART	288	204	226	220	200
ART +10dB	170	156	166	170	168

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	212	220	188	176	192
ART +10dB	138	148	136	152	152
1000 Hz					
ART	324	320	240	260	248
ART +10dB	180	176	172	160	180
4000 Hz					
ART	284	240	180	210	200
ART +10dB	192	196	160	148	172

SUBJECT 7, BIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	304	332	436	352	380
ART +10dB	154	132	140	134	142
1000 Hz					
ART	320	344	380	384	322
ART +10dB	144	138	140	138	142
4000 Hz					
ART	372	338	360	328	308
ART +10dB	188	176	186	154	164

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	264	260	440	288	248
ART +10dB	164	136	134	134	136
1000 Hz					
ART	272	300	332	304	320
ART +10dB	126	134	134	138	144
4000 Hz					
ART	392	360	280	400	364
ART +10dB	218	184	186	160	152

SUBJECT 8, BIPHASIC POSITIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	342	384	340	324	340
ART +10dB	166	178	176	178	184
1000 Hz					
ART	360	360	340	330	340
ART +10dB	190	200	212	186	228
4000 Hz					
ART	272	204	240	316	260
ART +10dB	180	224	224	190	172

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	272	342	322	272	256
ART +10dB	160	176	150	190	172
1000 Hz					
ART	324	300	330	244	248
ART +10dB	176	178	176	176	190
4000 Hz					
ART	300	352	272	304	304
ART +10dB	174	228	218	212	208

SUBJECT 9, BIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	324	328	280	332	328
ART +10dB	198	180	174	184	192
1000 Hz					
ART	364	360	452	316	274
ART +10dB	186	190	190	184	194
4000 Hz					
ART	336	388	342	328	324
ART +10dB	206	260	206	196	200

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	260	268	384	396	336
ART +10dB	172	176	176	200	196
1000 Hz					
ART	324	364	320	280	364
ART +10dB	200	198	192	194	208
4000 Hz					
ART	492	356	352	336	292
ART +10dB	212	260	216	196	192

SUBJECT 10, BIPHASIC POSITIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	320	340	340	360	360
ART +10dB	122	120	146	162	152
1000 Hz					
ART	376	384	340	420	420
ART +10dB	136	160	132	136	136
4000 Hz					
ART	340	396	368	364	428
ART +10dB	260	304	178	132	134

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	344	300	360	292	308
ART +10dB	122	122	180	124	156
1000 Hz					
ART	436	436	400	348	400
ART +10dB	176	156	168	168	212
4000 Hz					
ART	440	460	540	384	376
ART +10dB	224	220	176	140	192

APPENDIX D

Individual acoustic reflex response-times to 10% of maximum negative reflex amplitude (values in msec).

SUBJECT 6, BIPHASIC NEGATIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	92	100	72	76	76
ART +10dB	70	74	58	52	54
1000 Hz					
ART	64	74	64	68	50
ART +10dB	50	44	44	48	48
4000 Hz					
ART	60	58	60	58	60
ART +10dB	40	48	48	50	28

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	76	128	76	102	102
ART +10dB	74	100	88	80	80
1000 Hz					
ART	110	116	90	84	90
ART +10dB	68	84	70	64	76
4000 Hz					
ART	60	68	70	64	72
ART +10dB	52	50	52	40	36

SUBJECT 7, BIPHASIC NEGATIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	172	164	156	152	110
ART +10dB	88	72	76	66	72
1000 Hz					
ART	136	92	76	78	72
ART +10dB	72	72	56	58	58
4000 Hz					
ART	136	140	104	72	68
ART +10dB	80	74	68	72	64

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	148	120	132	120	100
ART +10dB	80	76	80	56	68
1000 Hz					
ART	126	132	92	108	82
ART +10dB	76	74	76	68	66
4000 Hz					
ART	144	112	104	90	64
ART +10dB	78	78	70	64	60

SUBJECT 8, BIPHASIC NEGATIVE RESPONSE TIME IN MSEC

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	184	128	142	104	104
ART +10dB	84	82	80	76	76
1000 Hz					
ART	128	124	100	100	100
ART +10dB	82	82	88	88	68
4000 Hz					
ART	100	88	100	120	116
ART +10dB	100	64	100	80	82

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	140	132	144	124	120
ART +10dB	88	92	82	82	74
1000 Hz					
ART	104	112	104	100	104
ART +10dB	80	92	84	88	72
4000 Hz					
ART	130	88	80	108	120
ART +10dB	84	84	64	94	80

SUBJECT 9, BIPHASIC NEGATIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	124	176	144	156	104
ART +10dB	104	88	96	86	82
1000 Hz					
ART	146	136	124	122	88
ART +10dB	74	72	66	76	64
4000 Hz					
ART	100	80	100	80	78
ART +10dB	76	80	78	62	66

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	112	194	128	148	104
ART +10dB	92	84	88	96	68
1000 Hz					
ART	154	128	126	124	164
ART +10dB	70	76	70	64	60
4000 Hz					
ART	280	84	72	80	52
ART +10dB	70	60	72	60	60

SUBJECT 10, BIPHASIC NEGATIVE RESPONSE TIME IN MSEC
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	166	108	184	100	204
ART +10dB	68	62	78	80	78
1000 Hz					
ART	100	220	208	140	164
ART +10dB	72	74	72	68	70
4000 Hz					
ART	140	204	152	168	104
ART +10dB	100	100	96	60	70

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	204	202	148	224	216
ART +10dB	68	64	88	60	76
1000 Hz					
ART	200	142	140	166	222
ART +10dB	72	66	72	72	102
4000 Hz					
ART	200	260	172	188	84
ART +10dB	128	132	80	64	76

APPENDIX E

Individual Acoustic Reflex Thresholds in dB SPL

SUBJECT 1

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	105	105	106	104	98
1000 Hz ART	102	104	104	100	97
4000 Hz ART	114	114	110	104	100

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	105	105	106	103	98
1000 Hz ART	102	102	103	102	98
4000 Hz ART	109	109	108	014	102

SUBJECT 2

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	100	105	105	97
1000 Hz ART	100	103	99	97	95
4000 Hz ART	99	104	103	93	93

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	105	108	108	99
1000 Hz ART	100	101	102	98	95
4000 Hz ART	105	105	102	99	98

SUBJECT 3

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	92	93	89	89	90
1000 Hz ART	91	92	93	91	89
4000 Hz ART	92	93	94	91	87

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	90	92	89	88	88
1000 Hz ART	87	88	88	89	88
4000 Hz ART	91	92	92	92	89

SUBJECT 4

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	93	98	96	88	83
1000 Hz ART	92	85	87	86	85
4000 Hz ART	96	94	91	88	84

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	88	87	85	85	84
1000 Hz ART	94	83	86	85	85
4000 Hz ART	102	100	100	90	88

SUBJECT 5

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	88	94	93	89	90
1000 Hz ART	94	94	94	91	85
4000 Hz ART	102	105	103	95	93

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	91	91	89	89	89
1000 Hz ART	92	92	92	88	86
4000 Hz ART	103	105	104	94	92

SUBJECT 6

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	92	91	89	88	83
1000 Hz ART	82	80	80	80	74
4000 Hz ART	89	89	85	74	72

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	102	103	101	98
1000 Hz ART	92	93	98	98	92
4000 Hz ART	94	95	92	93	88

SUBJECT 7

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	105	106	107	107	101
1000 Hz ART	102	103	103	102	98
4000 Hz ART	109	109	104	100	100

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	105	106	105	104	102
1000 Hz ART	102	103	103	102	98
4000 Hz ART	110	109	108	100	100

SUBJECT 8

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	99	99	99	98	96
1000 Hz ART	96	96	94	96	85
4000 Hz ART	99	99	97	91	86

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	100	100	98	98
1000 Hz ART	92	95	96	96	88
4000 Hz ART	99	99	97	91	86

SUBJECT 9

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	102	104	102	102	98
1000 Hz ART	101	101	102	101	92
4000 Hz ART	104	104	101	96	94

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	99	99	95	95	96
1000 Hz ART	92	93	95	93	93
4000 Hz ART	108	108	108	102	98

SUBJECT 10

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	100	99	100	97
1000 Hz ART	104	104	104	104	102
4000 Hz ART	108	109	108	108	104

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	100	102	103	103	100
1000 Hz ART	104	106	106	106	105
4000 Hz ART	111	111	108	108	104

APPENDIX F

Mean acoustic reflex response-times to 10% of maximum positive reflex amplitude (values in msec).

MEAN POSITIVE RESPONSE TIMES (N = 10)
FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	285	276	275	281	273
ART +10dB	148	141	140	142	145
1000 Hz					
ART	282	283	278	287	263
ART +10dB	148	153	154	153	156
4000 Hz					
ART	273	255	255	245	246
ART +10dB	188	190	178	163	154

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	234	250	268	253	269
ART +10dB	143	147	146	151	152
1000 Hz					
ART	294	297	287	262	269
ART +10dB	155	157	159	161	166
4000 Hz					
ART	319	321	293	279	246
ART +10dB	197	197	179	160	168

APPENDIX G

Mean acoustic reflex response-times to 10% of maximum negative reflex amplitude (values in msec).

MEAN NEGATIVE RESPONSE TIMES (N = 5)

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	148	135	140	118	120
ART +10dB	76	76	78	72	72
1000 Hz					
ART	115	129	114	102	95
ART +10dB	70	69	65	68	62
4000 Hz					
ART	107	134	103	100	85
ART +10dB	79	73	78	65	62

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz					
ART	136	155	126	144	129
ART +10dB	80	83	85	75	73
1000 Hz					
ART	139	126	110	116	132
ART +10dB	73	79	74	71	75
4000 Hz					
ART	161	122	100	106	78
ART +10dB	82	81	68	64	62

APPENDIX H

Mean Acoustic Reflex Thresholds in dB SPL

MEAN ARTs IN dB SPL

FIRST TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	97.6	99.0	98.5	97.0	93.3
1000 Hz ART	96.4	96.1	96.0	94.8	90.2
4000 Hz ART	101.2	102.0	99.6	94.0	91.3

SECOND TEST

	OCTAVE BANDWIDTH				
	1/6	1/3	1	2	4
500 Hz ART	97.8	98.9	98.3	97.4	95.2
1000 Hz ART	95.7	95.6	96.9	95.7	92.8
4000 Hz ART	103.2	103.3	101.9	97.3	94.5

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