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Assessment of Hearing Sensitivity by Use of the Acoustic Reflex in the Geriatric Population

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AN ABSTRACT OF THE THESIS OF Donna Elvira Gilham for the Master of Science in Speech Communication with major emphasis in Audiology and Speech Pathology presented May 14, 1976.

Title: Assessment of Hearing Sensitivity by Use of the Acoustic Reflex in the Geriatric Population.

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The purpose of this study was to investigate the use of the SPAR test (Sensitivity Prediction by the Acoustic Reflex) on a sample of older adults who professed negative otologic histories. Twenty-seven subjects, ranging in age from sixty-four to eighty-three years of age (54 ears) were ultimately chosen as subjects. A young adult group, ranging

in age from eighteen to thirty years of age, with normal audiometric hearing levels served as controls. Reflex threshold levels were obtained using 500 Hz., 1000 Hz., and 2000 Hz. pure tones and noise stimuli. Assessment of hearing loss was calculated on the difference between measures to ascertain hearing loss category and slope of loss according to a formula developed by Jerger (1974).

The following conclusions were made on the basis of data collected in this investigation:

- 1) The SPAR test was significant in assessing hearing sensitivity levels among the sample of geriatric subjects.
- 2) The SPAR test was not statistically significant in assessing slope of loss in this sample of geriatric subjects.
- 3) Reflex thresholds were consistently higher for aging than for a sample of young adults with normal hearing. This was true even among elderly subjects with average pure tone thresholds in the normal range.
- 4) A significant portion of this carefully screened older adult sample displayed abnormal middle-ear function (as measured by tympanometry) in the absence of conventional audiometric indications of this.
- 5) A significant portion of this older adult sample provided audiometric evidence of middle-ear pathology.

ASSESSMENT OF HEARING SENSITIVITY
BY USE OF THE ACOUSTIC REFLEX
IN THE GERIATRIC POPULATION

by

DONNA E. GILHAM

A thesis submitted in partial fulfillment of the
requirements for the degree of

MASTER OF SCIENCE
in
SPEECH COMMUNICATION
with emphasis in Audiology
and Speech Pathology

Portland State University
1976

TO THE OFFICE OF GRADUATE STUDIES AND RESEARCH:

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ACKNOWLEDGMENTS

I wish to express thanks and appreciation to:

Dr. James Maurer, my academic advisor, for his generosity and assistance not only in time spent on this study, but also in the use of his office and equipment, without which my study would have been impossible.

Mrs. Mary Gordon and Dr. Steve Brannan for serving on my thesis committee.

Mr. Michael Fairchild for his expertise and assistance with the statistics necessary for this project.

Dr. Stephen Fausti for his confidence and support in all of my professional activities.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

The invention of the impedance bridge and the perfection of tympanometry has opened an entirely new field in audiological assessment of hearing sensitivity. Impedance audiometry has two clinical utilities: 1) to measure the compliance of the middle ear, and 2) to monitor the intraural reflex. The reflex is monitored as a change in the physics of the middle ear, i.e., a change in impedance and admittance values. This usually happens in response to contralateral stimulation of 70 to 90 dB above hearing threshold level. Among the uses of monitoring the acoustic reflex has been its diagnostic value for disorders of the eighth nerve and lower brainstem as it is elicited bilaterally in normal subjects. In order for it to be elicited contralaterally, both afferent and efferent pathways of the VIII nerve must be intact as well as those of the VII nerve prior to the stapedial branch. The stapedial tendon must be intact and attached to the head of the stapes, and there must be sufficient mobility in the ossicular chain to allow contraction (Feldman, 1963). Since the contralateral reflex threshold of normal subjects occurs at levels of 70 to 90 dB above hearing threshold level, many cases may

be studied which would not be possible with standard audiological procedures.

Recently, the acoustic reflex has been used as a means of assessing hearing loss and slope of loss in a wholly new approach to assessment of hearing sensitivity (Jerger, 1974). A basic assumption of this method is that different sounds have different degrees of perceived loudness, and, therefore, different threshold values. The relationship between different types of stimuli has been the basis for a new test of assessment of hearing sensitivity.

Review of the literature has shown that no research has been conducted using this method of hearing assessment on a purely geriatric sample of the population. Since additional variables such as conductive loss from stiffening of ligaments, muscles, and connective tissues, enlarged ear canals, and recruitment of loudness enter into the testing of the older person, a study of the reliability of this method should prove useful for clinical and research purposes.

Statement of Purpose

The purpose of this study was to determine if the method of assessing hearing loss and slope of loss is statistically valid when applied to a sample of the geriatric population with no known concomitant otologic problems other than presbycusis.

The principle questions posed in this study were:

- 1) Will the determination of acoustic reflex thresholds be statistically valid in assessing magnitude of hearing loss in this sample of persons over sixty-five years of age?
- 2) Will acoustic reflex thresholds be statistically valid in assessing slope of loss among members of this sample?
- 3) Will reflex thresholds of the older population group be statistically different from those of the young adult control group?

CHAPTER II

REVIEW OF THE LITERATURE

Incidence and Prevalence of Hearing Loss Among Older Persons

According to data obtained by the National Health Survey which was conducted by the United States Department of Health, Education, and Welfare during the period from October, 1960 to December, 1962, the age group of sixty-five years and older is the segment of the population which is most severely afflicted with problems of hearing impairment.

Data obtained from this survey further showed that 28.2 percent of adults with a hearing loss of +16 dB or greater (ASA 1951 standards) were in the age range from sixty-five to seventy-four and 48 percent were in the range from seventy-five to seventy-nine years; whereas, .8 percent were in the age range from eighteen to twenty-four years. These statistics clearly demonstrate the area of greatest hearing difficulty exists within the geriatric population and are more extensively illustrated in Table I. The United States Department of Health, Education, and Welfare projects (based on these statistics) that approximately twenty-four million persons over sixty-five years of age will suffer from impaired hearing by 1980.

TABLE I

INCIDENCE OF HEARING LOSS

Sex and age	Hearing level re audiometric zero					
	-5 dB or lower	-4 to +15 dB	+16 dB or higher	-5 dB or lower	-4 to +15 dB	+16 dB or higher
<u>Both sexes</u>	Number per 100 adults			Number of adults in thousands		
All ages, 18-79 years----	46.8	45.9	7.3	51,983	50,984	8,120
18-24 years-----	74.1	25.1	0.8	11,535	3,914	120
25-34 years-----	67.3	31.4	1.3	14,524	6,771	277
35-44 years-----	54.5	42.6	2.9	12,929	10,089	680
45-54 years-----	40.5	55.2	4.3	8,328	11,352	896
55-64 years-----	22.5	67.2	10.3	3,518	10,501	1,618
65-74 years-----	9.9	61.9	28.2	1,103	6,911	3,150
75-79 years-----	1.6	50.4	48.0	46	1,446	1,379
<u>Men</u>						
All ages, 18-79 years----	43.3	49.1	7.6	22,845	25,845	4,054
18-24 years-----	68.3	30.5	1.2	4,873	2,180	86
25-34 years-----	63.0	35.6	1.4	6,479	3,663	139
35-44 years-----	50.9	45.4	3.7	5,786	5,172	415
45-54 years-----	35.0	60.9	4.1	3,508	6,121	405
55-64 years-----	23.2	66.2	10.6	1,742	4,979	796

TABLE I--Continued

Sex and age	Hearing level re audiometric zero					
	-5 dB or lower	-4 to +15 dB	+16 dB or higher	-5 dB or lower	-4 to +15 dB	+16 dB or higher
	Number per 100 adults			Number of adults in thousands		
<u>Men</u>						
65-74 years-----	8.5	61.0	30.5	421	3,034	1,517
75-79 years-----	2.5	48.8	48.7	36	696	696
<u>Women</u>						
All ages, 18-79 years----	49.9	43.0	7.1	29,138	25,139	4,066
18-24 years-----	79.0	20.6	0.4	6,662	1,734	34
25-34 years-----	71.3	27.4	1.3	8,045	3,108	138
35-44 years-----	58.0	39.8	2.2	7,143	4,917	265
45-54 years-----	45.7	49.7	4.6	4,820	5,231	491
55-64 years-----	21.9	68.0	10.1	1,776	5,522	822
65-74 years-----	11.0	62.8	26.2	682	3,877	1,633
75-79 years-----	0.7	51.9	47.4	10	750	683

National Health Survey, Series 11, Number 11.

Rupp (1970) stated that almost nineteen million (10 percent of the total population of the United States) are sixty-five years of age or over. More than a million are over eighty-five, and some six million are between the ages of seventy-five and eighty-five. According to these senior citizen's own opinion poll, vision and hearing are ranked first in importance for a healthy old age. At the present time, this is not possible for a majority of older persons; 90 percent of those over sixty-five years of age need glasses and 66 percent have serious hearing problems by the age of eighty (Rupp, 1970).

Acoustics and Anatomy of Hearing

In order to understand the role of the middle ear reflexes in hearing, it is necessary to review certain aspects of the anatomy and functioning of the ear. The process begins as sound waves enter the pinna which serves as a funnel to direct sound waves through the external auditory meatus to the tympanic membrane. The external auditory meatus is a curved, irregularly-shaped tube which conducts sound waves to the tympanic membrane and serves as a frequency filtering device (Zemlin, 1968). Wever and Lawrence (1954) estimated the speed of sound in the meatus to be 350 meters per second. Fleming (1939) found the resonant frequency of the meatus to be 3800 Hz. This has been confirmed by later investigation (Zemlin, 1968).

Sound waves traveling in a medium of a definite elasticity and density will not pass easily into another medium of different elasticity and density; a portion will be reflected away. In any system which has efficient transmission of energy, the impedance of the receiving medium must match that of the transmitting system. In the ear, the impedance of the waves in air must somehow be made to match that of the liquids of the inner ear. In order to do this, the middle ear must increase the pressure at the oval window by a factor of sixty-three, or by about 28 dB. Wever and Lawrence (1954) found the ratio between the area of the tympanic membrane and stapes foot-plate to be 21:1. When combined with the ossicular lever ratio, this affords a mechanical advantage of 18:3, or an increase in SPL of about 25.5 dB (Zemlin, 1968). As a result of resonance in the auditory canal, the sound pressure at the eardrum is higher than at the entrance of the canal with a 10 dB peak around 3500 Hz. (Moller, 1972).

In 1561, Fallopius was the first person to describe the middle ear and give it the name "tympanum," or "army drum." The lateral wall of the middle ear is formed by the tympanic membrane, which is concave, smooth, and pearl gray. It consists of three layers of tissue: 1) a thin, outer, cutaneous layer; 2) a fibrous, middle layer; and 3) an internal layer of mucous membrane. A small portion of sound waves impinging upon the tympanic membrane is absorbed;

a major portion is reflected back into the external auditory meatus. The reflected portion is measured in tympanometry (Zemlin, 1968).

The middle ear, or tympanic cavity, is an irregular space within the petrous portion of the temporal bone. It consists of two parts: the tympanic cavity proper, and the epitympanic recess, which extends above the level of the tympanic membrane (Zemlin, 1968). The largest part of the tympanic cavity is filled by the ossicles (the malleus, incus, and stapes), the three smallest bones of the human body, which transmit sound waves to the inner ear (see Figure 1). The head of the malleus and the bulk of the

Figure 1. Schematic of middle ear cavity, frontal view (Zemlin, 1968).

incus extend up into the epitympanic recess. The footplate of the stapes fits into the oval window, where it is fixed into place by the annular ligament. The oval window and the round window are two openings in the bony medial wall of the middle ear cavity; the round window is closed by a thin membrane. As the pressure waves travel through the middle ear by way of vibrations of the ossicular chain, the footplate of the stapes is rocked back and forth, thus transmitting pressure waves to the liquid-filled cochlea of the inner ear.

The ossicles are suspended by a series of ligaments; the malleus is suspended by three ligaments, the incus by one. Due to the method of suspension, the inertia of the system is very small and its rotational axis is near the center of gravity. This causes abrupt damping of the sound transmission once vibrations have ceased (Zemlin, 1968).

In addition to the ligaments which suspend the ossicular chain, the malleus and stapes bones are attached by tendons to the two intra-ear muscles, the tensor tympani (25 mm. in length) and the stapedius (6 mm. in length), which are the two smallest striated muscles in the body. The tendons of these two muscles differ from other tendons because of the large amount of elastic tissue. Jepsen (1963) felt this elastic property served to dampen vibrations of the ossicles and to cause less sudden onset of muscle traction. The tympanic muscles have two unique

features: their short fibers are arranged to give great tension with very little linear displacement, and they are completely encased in bony canals with only their tendons entering the tympanic cavity. Bekesy (1936) felt this arrangement reduces vibration which may interfere with sound transmission and also reduces the mass of the ossicular chain. Contraction of the stapedius pulls the stapes downward and outward at right angles to the direction of movement of the ossicular chain. This greatly reduces its force upon the liquids of the cochlea (Jepsen, 1963). The tensor tympani muscle originates in a bony canal nearly parallel to the canal of the Eustachian tube. The distal part of the muscle lies on a bony shelf, the cochleoform process, which acts as a pulley over which the tendon of the tensor tympani lies before bending and attaching to the upper part of the manubrium of the malleus. Contraction of this muscle draws the malleus at almost right angles to the direction of rotation of the ossicular chain and increases tension of the tympanic membrane (Jepsen, 1963). Recent research has suggested this action may not be triggered by loud sounds, but rather, as a general startle reflex (Klockhoff and Anderson, 1960).

The innervation patterns of the two muscles reflect their origins. Since the stapedius is developed from the second branchial arch, it is supplied by the nerve to that arch, the facial nerve. The tensor tympani, which develops

from the first branchial arch, is supplied by the third branch of the trigeminal nerve. The middle ear is innervated by three cranial nerves: the acoustic or VIII nerve, the facial or VII nerve, and the trigeminal or V nerve. The facial nerve has both a motor root and a sensory root. The motor root originates in the lower part of the pons. The fibers emerge from the brain at the level of the superior olive, where they join the sensory branch which originated in the geniculate ganglion. The combined roots of the facial nerve join the acoustic nerve and enter the internal acoustic meatus. At the bottom of the meatus, it separates and enters the portion of the facial canal which lies between the vestibule and the cochlea. As the facial canal reaches the medial wall of the middle ear, it changes directions and forms a "knee" which is known as the geniculate ganglion. Behind the oval window, the facial nerve canal becomes vertical. At this point, the pyramidal eminence, which is a bony protuberance containing the stapedial tendon, is attached to the facial canal. It also contains the stapedial branch of the facial nerve which supplies the stapedius muscle (Wolferman, 1970).

The trigeminal is the largest of the cranial nerves. Its mandibular branch contains both motor and sensory fibers. Its motor root branches off the interior maxillary nerve and sends small filaments to the otic ganglion and thus supplies the tensor tympani muscle with motor fibers (Zemlin, 1968; Jepsen, 1963).

The auditory nerve consists of both an afferent and an efferent branch. The afferent portion has its origin in the hair cells of the cochlea. Radial fibers innervate the inner hair cells while both radial and spiral fibers innervate the outer cells. Rasmussen (1953) estimated about 30,000 auditory nerve fibers are present in man; Schucknecht (1956) found about 51,000 in the cat. There are about 500 efferent nerve fibers which terminate on both inner and outer hair cells (Rasmussen, 1960). The auditory nerve fibers divide in a regular way to send branches to each dorsal and ventral cochlear nucleus, from where they ascend through a series of synapses to the auditory cortex (see Figure 2).

Figure 2. The ascending (afferent) neuronal chain from the cochlea to the cortex. Numbers indicate neuron levels (Gaecek, 1972).

The Acoustic Reflex

In 1878, Hensen was the first to observe acoustic middle ear muscle reflexes of the tensor tympani in dogs. In 1913, Kato demonstrated acoustic reflex contractions not only emanate from the tensor tympani, but also from the stapedius. Luscher's investigation later confirmed these observations. He was the first to show that acoustic reflex contractions of the stapedius were a constant phenomenon in man. He found that the upper and lower frequency limits for the elicitation of the stapedius reflex were 14,000 Hz. and 90 Hz., respectively, and demonstrated that the reflex threshold was lowest at 2000 Hz. He also found that the reflex could be elicited both from ipsilateral and contralateral ears. Lindsay (1936) found the threshold of the stapedius reflex to be 65 to 85 dB above the threshold of normal hearing (Jepsen, 1963). Northern (1974) estimates that, in the normal ear, a loudness of from 70 to 100 dB will elicit an acoustic reflex, with the median being 82.2 dB (approximately 85 dB on the audiometer).

Band-pass filtered noise is a more effective stimulus in eliciting the reflex than pure tones. Moller (1962) found that if the energy content of a tone and a noise band centered around the same frequency are compared, noise can elicit the reflex at about 5 dB below the level of the tone.

Jerger (1974) has found a 20 dB difference in the reflex thresholds of pure tones and broad band noise. Once threshold is passed, the strength of the muscle contraction increases roughly in proportion to the magnitude of the stimulus. It is active over a relatively narrow dynamic range between its threshold and level of saturation. The ability of the muscles to perform finely graded contractions is determined by the innervation ratio (ratio of efferent to motor fibers). The ratio in the tensor tympani is approximately 1:7; the ratio in the stapedius is from 1:3.5 to 1:5.6. These are unusually high ratios compared to other body muscles. The possibility of two kinds of motor units in middle ear muscles--tonic (characterized by continuous tension) and phasic (tension fluctuating) has been hypothesized by Okamoto et al. (1954).

On the basis of animal studies (Wersall, 1958; Kirikae, 1960; Wever and Vernon, 1956; Galambos and Rupert, 1959), it has become fairly well established that contraction of the tympanic muscles results in a reduction in sensitivity to low frequency tones. Wever and Bray (1942) investigated the function of the stapedius muscle by observing the effects upon the electrical responses to the cochlea. They found that tension applied through a thread attached to the stapedius tendon causes a marked reduction in transmission for low tones, a smaller reduction for high tones, and improvement followed by decline for tones of the middle

range. Borg (1968) found that attenuation of the acoustic reflex was greater at frequencies below the resonant frequency of the middle ear than above it. A tone of 500 Hz. was attenuated from 12 to 15 dB; whereas, a tone of 1450 Hz. was attenuated only from 0 to 6 dB.

There have been many theories advanced as to the primary function of the acoustic reflex. Among these are the accommodation to optimum sounds and the fixation theory, which proposes that the reflex provides needed rigidity for the ossicular chain and prevents changes in articulation between ossicles during high acceleration. Simmons (1964) warned that caution should be exercised in assigning a purely protective role to the acoustic reflex. He demonstrated that muscle reflexes can become active at much lower levels than assumed by the protection theory. As part of an animal's common orienting response to unusual environmental sound, the middle ear contractions take place along with obvious head and body movements. They serve to attenuate inborne sounds and help "tune" in on external stimuli. The reflex can take place with head movements, vocalization, chewing, and swallowing. Fletcher and Riopelle (1960) demonstrated the protective effect of the acoustic reflex in an experiment in which they triggered the reflex by a 1000 Hz. tone at 98 dB SPL before exposing subjects to gunfire.

Of great practical interest is the latency period before onset of the acoustic reflex. When a tone is turned

on rapidly, there is a characteristic delay interval between onset of the stimulus and onset of response. Perlman and Case (1939) recorded this in human beings at about ten milliseconds. Wever and Lawrence (1954) cited the mean value of .06 seconds for the stapedius and .15 seconds for the tensor tympani. The magnitude of the latent period is found to be inversely proportional to the strength of the acoustic stimulus. A range of from 40 to 160 milliseconds has been observed in human subjects when stimulated with a wide-band noise of various intensities. The existence of the latent period has important practical and theoretical consequences in relation to the functioning and effectiveness of the acoustic reflex. Due to the latency, the reflex is unable to perform its postulated protective role when elicited by a very brief stimulus.

Bekesy (1936) showed another defense mechanism of the middle ear whereby the latency period is eliminated. At moderate sound pressures, the stapes rotates around a vertical axis, but when the sound pressure is greatly increased, the vibration axis is changed, so that the stapes rotates around a longitudinal axis through its base. The first movement causes an in and out displacement of cochlear fluid; the second causes the fluid to surge back and forth without any effect on the cochlea (Zemlin, 1968).

Most information pertaining to the quantitative features of reflex dynamics were gathered by recording the input

impedance to the ear. Assymetry is the most conspicuous feature of the on-off response. At every stimulus level, the on response is considerably faster than the off response (Dallos, 1964). This implies that the muscle contraction process is much more rapid than the relaxation. Dallos (1964) concludes the response dynamics depend upon both stimulus intensity and frequency. At any given frequency, there is apparently a given feedback gain that determines the basic nature of the response.

Moller (1962) found that reflex thresholds are approximately 5 to 10 dB higher with contralateral, as opposed to ipsilateral, stimulation. At any intensity above reflex threshold, the impedance change is less in the contralateral ear (Dallos, 1973).

Recent studies have led researchers to conclude the stapedius plays a more dominant role than the tensor tympani in altering transmission through the middle ear. Despite its smaller size and strength, it may be the more important of the two muscles in this respect. Contraction of the tensor tympani is believed to be associated with a general startle reflex rather than a reaction to loud sounds (Jepsen, 1963). Djupesland (1962) observed contraction of both tympanic muscles upon contraction of the periorbital muscles (eye muscles), and Klockhoff (1960) observed this after air-jet stimulation. The relations to frequency are somewhat different for the two muscles.

The tensor tympani reduces transmission and shifts the sensitivity curve upward along the frequency scale. For the stapedius muscle, there is no noticeable shift of the region of maximal sensitivity, which under tension remains about 3000 Hz. There is, however, a significantly greater impairment of tones below this region than of tones above it; within the region itself, there is a slight attunement (Dallos, 1973).

Borg's Research to Establish the Reflex Arc

Several investigators have studied the reflex arc of the tympanic muscles. Among these are Hammerschlag, 1899; Forbes and Sherrington, 1914; Lorente de No, 1933; Tsukamoto, 1934; and Rasmussen, 1946; but previous to Borg's (1973) colossal research project on the neuronal organization of the acoustic middle ear reflex, the pathways of this reflex had not been studied in detail. The acoustic reflexes and the olivo-cochlear efferent tract both influence the response of the cochlea to sound, but there seems to be important differences between these two systems at various frequencies and intensities. Because of the work of Rasmussen, the path of the olivo-cochlear bundle has become well known, but the path of the acoustic reflexes has remained vague. The acoustic reflex appears to be the most regular, stable, and least complexly organized motor or anatomical response to sound. Since the acoustic stapedial reflex has become a

widely used tool for diagnosis of hearing impairment, it becomes very important to understand the physiology and anatomy of the central routes of reflex activation. Prior to Borg's investigation, information was incomplete regarding the parts of the ascending pathway which were involved in middle ear reflexes.

Borg employed a combination of physiological and anatomical methods on rabbits with chronic brain lesions. His studies were based on retrograde reactions to neuron lesions of the pathways. From these studies, he came to the following conclusions regarding the neuronal organization of the middle ear reflex pathways:

The first synapse of both the stapedius and tensor tympani reflex pathways is in the ventral cochlear nucleus. The dorsal cochlear nucleus is not involved, at least with pure tone stimulation. It is believed that the thin fibers of the ventral cochlear nucleus are the ones involved in motor responses since they are the same size as the thin fibers of the trapezoid body and have bilateral connections to the medial superior olive body.

Borg did not conclusively prove the location of the second synapse of the stapedius reflex, but he believed that all evidence points to the medial superior olive as having both crossed and uncrossed connections to the ventral cochlear nuclei and to the ipsilateral seventh nerve. Rasmussen (1964) noted this earlier when he observed crossed

connections from the medial superior olive to the contralateral seventh nerve in the cat. The pathway of the crossed stapedial reflex has a synapse either in the ipsilateral or contralateral medial superior olive. Results of the study indicated uncrossed and crossed connections from the medial superior olive and a direct path from the ventral cochlear nucleus to the contralateral seventh nerve.

The tensor tympani reflex first-order and second-order neurons follow the same course as for the stapedius reflex to the level of the trapezoid body. It is thought to proceed to the medial superior and possibly to the ventral nucleus of the lateral lemniscus.

The third synapse of the reflex arc is the connection with motor neurons; this is the VII or facial nerve for the stapedius and the V or trigeminal for the tensor tympani. The majority of studies indicate that motor neurons for the stapedius are situated in the medial group of the VII nerve together with the motoneurons of the external ear muscles. The location of the neurons which connect the tensor tympani with the trigeminal nerve are not well understood. At the present, no direct connections from second-order neurons to the cranial nerve V have been established (Borg, 1973). The motor neurons follow the facial nerve to the stapedius in the middle ear and the mandibular branch of the trigeminal nerve to the tensor tympani.

There are also indirect pathways which lie parallel

to the direct paths; these indirect pathways are slower. Researchers have speculated on the involvement of the extra-pyramidal system in the slow activity since Massion (1949) found that the cells of the red nucleus of the cat and rabbit respond to sound. Lorente de No (1933) pointed out that connections to the motor nuclei from the nuclei of the lateral lemniscus, which descend from the inferior colliculus, suggest alternative pathways. Further evidence implies the motor nuclei of the middle ear muscles are exposed to reflex activity from the brain-stem reticular formation and from certain sensory areas of the skin and face (Moller, 1962).

The work of Borg (1973) thus revealed the presence of two ipsilateral and two contralateral direct pathways of the stapedial acoustic reflex. It also revealed one ipsilateral and one contralateral path for the tensor tympani reflex. Borg's work suggests the two peripheral control systems, the middle ear reflexes and the olivo-cochlear efferent bundle, seem to be organized in two separate neuroanatomic systems. The two systems are not completely independent, however, as the complete interruption of the crossed olivo-cochlear bundle lowers the threshold of the middle ear reflexes for sound above 2000 Hz. Borg concluded that this influence is probably at the cochlear level. Figure 3 illustrates the paths of the ipsilateral and contralateral acoustic reflexes.

The Efferent System

Due largely to the work of Rasmussen (1946, 1953, 1960, 1967) the presence of a system of descending, centrifugal fibers, linking the cortex with the organ of Corti, has been conclusively demonstrated. Two descending chains of neurons, both arising in the auditory cortical areas, and both traveling the same pathways as the ascending system, have been determined (see Figure 4).

The olivo-cochlear neurons are the final link in the descending chain. The entire bundle contains about 500 fibers--both ipsilateral and contralateral. It travels in

Figure 4. The descending (efferent) neuron chains. Nerve fibers terminating in cochlea are those of the olivo-cochlear bundle (Gaecek, 1972).

the vestibular branch of the VIII nerve and emerges from the brainstem between two divisions of the nerve. The cochlear efferents leave the vestibular nerve via the vestibucochlear anastomosis (Oort) and enter the cochlea in the basal region to form the intraganglionic spiral bundle within Rosenthal's canal. This bundle runs apicalward with fibers feeding off regularly. About 3000 efferent fibers enter the organ of Corti as a result of arborization. They course longitudinally under inner hair cells in the inner spiral bundle. About 800 fibers cross the tunnel of Corti and terminate in approximately 40,000 contacts with cell bodies of the outer hair cells (Dallos, 1973).

It is not fully understood how inhibition functions. The first physiological study of crossed centrifugal fibers was made by Galambos, who used macroelectrodes to show that electrical stimulation suppresses the action potential at the round window of the cochlea. Another possible hypothesis is that efferent stimulation may cause changes in impedance in the organ of Corti. A working hypothesis presented is that the large efferent synapses cause post-synaptic hyperpolarization of the base of the external hair cells, which, in turn, counteracts the mechanism which releases transmitter substances for interaction of afferent impulses (Kimura and Wersall, 1955).

The ratio of efferent to afferent fibers is about 1:100. The efferents may have a means of detecting small

changes in frequency and thus take part in some sort of frequency discrimination. It is estimated that the maximum inhibitory action of crossed efferent fibers in the ear corresponds to about 5 to 15 dB. Fex (1967) concluded the centrifugal cochlear systems form part of a complex feedback mechanism, but there is no secure knowledge of how central connections are activated (Dallos, 1973).

The two inhibitory systems appear to operate in completely different ways. The acoustic reflex might be compared to a digital computer (it has an all or none reaction), while the efferent system resembles more closely an analog computer (it is effective on a continuum of sound intensity). The acoustic reflexes represent a lower brain-stem function, while the efferent system represents neural action from the level of the auditory cortex down the entire pathway. The acoustic reflex lowers sound transmission by altering the physical properties of the middle ear; the efferent system appears to lower sound transmission by altering transmission qualities of the neural components of the inner ear.

Critical Band Concept

Studies of human observers to bands of noise and other complex sounds have led to a measure of what appears to be a basic unit of hearing, the critical band. The concept of the critical band is a very important factor in measuring reflex threshold. Jerger (1974) has defined the critical

band as a certain width on the basilar membrane that is essentially independent in terms of its loudness contribution from its neighbors. Jerger (1974) employed the concept of the critical band to explain the difference in reflex thresholds elicited by white noise and by pure tones. He found the basis for this phenomenon seems to be that the acoustic reflex (the stapedius contraction) is based upon the apparent loudness of a sound rather than on its actual physical intensity. Decibel for decibel, a broad band noise contains energy in many contiguous critical bands.

In the human cochlea, the estimated critical band at 1000 Hz. covers from one to two millimeters of the basilar membrane. The fact that this loudness function bears a close resemblance to several functions of frequency such as difference limen for frequency and the mel scale for pitch (Bekesy and Rosenblith, 1951) makes it appear that all functions of loudness and frequency correspond to constant distances on the basilar membrane. The critical band concept corresponds to constant distances on the basilar membrane. If there are assumed to be 24 critical bands on a 32 millimeter long membrane, each critical band represents about 1.3 millimeters of topography on the basilar membrane. The following table (Table II) by Scharf (1970) demonstrates the number and width of the critical bands as well as the upper and lower cutoff frequencies. It is apparent the width of the critical bands increases with each increase in frequency

TABLE II

EXAMPLES OF CRITICAL BANDWIDTH

Number of Cr. Band	Center (Hz.) frequency	Critical (Hz.) bandwidth	Lower cutoff frequency (Hz.)	Upper cutoff frequency (Hz.)
1	50	--	--	100
2	150	100	100	200
3	250	100	200	300
4	350	100	300	400
5	450	110	400	510
6	570	120	510	630
7	700	140	630	770
8	840	150	770	920
9	1,000	160	920	1,080
10	1,170	190	1,080	1,270
11	1,370	210	1,270	1,480
12	1,600	240	1,480	1,720
13	1,850	280	1,720	2,000
14	2,150	320	2,000	2,320
15	2,500	380	2,320	2,700
16	2,900	450	2,700	3,150
17	3,400	550	3,150	3,700
18	4,000	700	3,700	4,400
19	4,800	900	4,400	5,300
20	5,800	1,100	5,300	6,400
21	7,000	1,300	6,400	7,700
22	8,500	1,800	7,700	9,500
23	10,500	2,500	9,500	12,000
24	13,500	3,500	12,000	15,500
25				

Scharf (1970).

and the area above 1000 Hz. contains many more critical bands than the area below it.

Fletcher (1940) hypothesized the existence of a critical band for masking. He suggested that when a white noise masks a tone, only a relatively narrow band of frequencies surrounding the tone does the masking. Energy outside this area contributes relatively little or nothing.

Gassler (1954) made careful measurements of the phenomenon of the threshold of complex sounds. His results indicated that total energy necessary for a sound to be heard remains constant as long as the energy is contained within a limiting bandwidth.

Zwicker (1958) advanced the theory that the loudness of a pure tone is a composite loudness because the displacement of the basilar membrane is spread over many critical bands. He assumed the "loudness" of these critical bands summates to give total loudness to a tone. Following this assumption, the loudness of a noise is thought to equal the sum of the individual loudnesses of the component critical bands.

Many researchers have studied the critical band mechanism in persons with sensori-neural hearing losses. Among the first investigators were Lightfoot, Carhart, and Gaeth (1953). They plotted masked threshold shifts against masker level over a wide range of effective masking. They found that persons with sensori-neural loss showed less masked threshold shifts at low frequencies and more at high fre-

quencies than normal hearing persons.

De Boer (1960, 1964) was the first to report direct measures of the critical band in a pathological ear. He found strong evidence of an enlarged critical band.

Jerger, Tillman, and Peterson (1960) used masking by octave bands of noise in tests with patients with impaired hearing and discovered that the hypacusic ear has a greater spread of masking rather than an altered signal to noise ratio. This difference was noted for remote (downward) and upward masking, but not at frequencies within the masker band. At an effective masking level of 30 dB, hypacusic patients exhibited 5 to 15 dB more remote and upward masking (especially upward) than either normal hearing persons or those with conductive losses. Ritmanic (1962) found that sensori-neural patients and normal hearing persons exhibited the same masking effect at the center frequencies of the masking bands, but the sensori-neural subjects exhibited from 10 to 20 dB more remote and upward masking at all frequencies from 250 to 4000 Hz.

Scharf and Hellman (1966) found that in ears with cochlear impairments and an average hearing loss of 65 dB, the critical band was much wider than normal, because loudness did not change with changes in frequency. This was true with an increase of six or seven normal-sized critical bands.

Among the most recent researchers is Martin (1974), who

used a loudness balancing technique to compare the relative loudness of a pair of tones as compared with a 2000 Hz. single tone on both normal hearing subjects and those with sensori-neural hearing loss. The subjects with sensori-neural hearing loss showed a reduced loudness effect which might suggest a widening of the critical band mechanism.

Jerger (1974) feels that the abnormal widening of the critical bands, coupled with the loss in high frequency sensitivity so characteristic of sensori-neural hearing loss would have a substantially greater effect on the total loudness of a broad band noise than on the loudness of individual sinusoids within that band. Jerger (1974) defines loudness as that neural activity which bears a one-to-one correspondence with a human listener's loudness experience. "We assume that the reflex mechanism operates on that parameter of neural activity to which human listeners typically assign the psychological construct 'loudness'" (Jerger, 1974). The result would be a reduction in the loudness advantage enjoyed by broad band noise in the normal ear.

The critical band mechanism may be compared to a set of band-pass filters with a variable center frequency, which seems to discriminate between sound energy within the band and that outside it. Several experiments have led to the theory that the locus of the critical band mechanism is perhaps in the cochlea (Niese, 1960; Bekesy, 1960). The largest support for the theory of the locus of the critical

band mechanism being peripheral came from Bekesy's experiments (1960) of inhibition in the eye and on the skin of units of sensation and inhibition. Bekesy named these "neural inhibitory units." Zwislocki (1965) suggested these neural units may correspond to the critical band in the ear with each critical band corresponding to about 1300 neurons. He related these neural units to lateral inhibition in the receptor organ; in the ear, this meant the neural network of the cochlea. Scharf (1970) felt that all data leads to the conclusion that the critical band may be just one stage of a multi-stage filtering process which includes mechanical filtering on the basilar membrane, neural filtering in the cochlea, and additional neural filtering at higher stages in the auditory system.

The procedure employed in this new method of testing (SPAR Test) consists of eliciting the acoustic reflex by pure tones and by broad band white noise. A pure tone causes loudness sensation only within its critical band; a series of critical bands sums the contributions to form a total loudness. Since broad band noise excites all critical bands, less intensity is required to elicit a reflex by its use in normal ears than is required by use of a pure tone stimulus.

In the case of sensori-neural hearing loss, the critical bands are believed to have widened, and, consequently, decreased in numbers. More energy is required to elicit a reflex by broad band noise than in normal ears. The rela-

tionship between the two thresholds is the basis for prediction of hearing sensitivity.

Presbycusis

Hearing loss that develops as a function of advancing age has been termed presbycusis. In spite of the fact that it represents the most common cause of loss of hearing sensitivity among sensori-neural disorders in adults, it has been long neglected. Formerly, the popular theory was that presbycusis was due to atrophy of the organ of Corti and the spiral ganglion cells. More recent evidence (Hinchcliffe, 1962a) has suggested it also includes changes in the external and middle ear mechanisms as well as deterioration of tissues in the central auditory pathways and auditory cortex. Glorig and Davis (1961) have reported data which suggest a conductive component in presbycusis. Their results revealed an air-bone gap that increased progressively with higher frequencies and advancing age. This report served as primary motivation for an investigation by Sataloff, Vassalo, and Menduke (1965); their research failed to find any evidence of an air-bone gap in presbycusis. Goodhill (1969) feels presbycusis should not be restricted to sensori-neural disorders, as the aging process occurs in ligaments, muscles, and connective tissue. Mechanical lesions occur in the middle ear involving the tympanic membrane and the ossicular chain. Among these, it is quite likely that primary

malleal fixation is a significant type.

Schuknecht (1964) has identified four types of presbycusis: sensory, neural, metabolic, and mechanical. Sensory or epithelial involves atrophy of the organ of Corti and the auditory nerve at the base of the cochlea. The primary site of degeneration in the cochlea is probably the supporting cells. Neural presbycusis is the result of loss of neurons in the auditory pathways and cochlea. This is the cause of a condition known as phonemic regression described by Gaeth (1948). This is a condition often encountered in the older person in which speech discrimination difficulties are greater than might be expected from the pure tone thresholds exists.

Metabolic presbycusis is thought to be caused by defects in the physical and chemical production of energy used by the sense organs. It is believed to be due to atrophy of the stria vascularis which is responsible for maintenance of bioelectric and biochemical properties of the endolymph. Alterations of these properties affect the entire scala media, resulting in a flat audiogram.

Mechanical presbycusis is believed to be due to a disorder of the mechanics of the cochlear duct which could result in loss of elasticity of the basilar membrane. The resulting audiogram shows the greatest loss in the high frequencies.

Welford and Birren (1965) discussed the aging process

in the neural system which causes a difference in the rate of conduction of nerve fibers; this, in turn, causes the neural message to be spread out in time as it reaches cortical centers and produces a "blurring." They attribute poor discrimination to increased "neural noise" and a lowered resistance to noise interference which they believe to be caused by loss of central cells and random neural activity. They also believe that a "blurring" effect may be due to longer aftereffects of neural activity which could cause distortion in a fast, continuously varying stimulus such as speech.

Gerard (1967) relates deficits in the aged with a decreased number of functionally available neurons and a prolonged time of fixation of an experience or memory. He says that neurons may become unavailable either by anatomical loss or by irreversible entry into a fixed neuron assembly. He feels that summation of neurons at synapses and ease of irradiation of activity through neuron nets diminishes with age.

Konnigsmark (1969) classified cell changes due to aging into six categories and arrived at the conclusion that each classification of cells has its own life span and that the aging process appears to vary from cell to cell and organ to organ. He felt all of these processes are likely to be found in the auditory system.

A number of recent investigations have revealed

histological evidence of aging in the auditory system. Kirikae (1964) discovered considerable evidence that signs of degeneration due to age have been identified in the central auditory nervous system. Changes in the major nuclei of the auditory pathways and auditory cortex were noted. Among these changes were a decrease in number of cells, atrophy and shrinkage of cells, accumulation of pigment, and a ghostlike, indistinct appearance of cells.

Makishima (1967) found that arteriosclerotic narrowing of the internal auditory artery correlated positively with atrophy of the spiral ganglion and with degree of impairment. Johnson and Hawkins (1972) found that aging is characterized by a disappearance of capillaries in the auditory system.

Krmpotic-Nemanic (1969) has found increased deposits of bone in the internal auditory meatus in the region of the spiral tract in the area corresponding to the basal turn of the cochlea. She feels the increasing pressure from these bone deposits may lead to tinnitus, to over sensitivity to loud sounds, and to eventual degeneration of spiral ganglion cells. Krmpotic-Nemanic stated:

In aging, a progressive apposition of dense connective tissue, osteoid, and bone, respectively occurs in the bottom of the internal auditory meatus. It can be seen microscopically as closure and disappearance of the holes for the nerve bundles in the region of the basal coil and progresses towards the modiolar region. This new-formed tissue compresses the nerve fibers and possibly also the arteries passing through the spiral tract. The result is atrophy first of the corresponding nerve fibers on the periphery of the nerve bundles and later also of the ganglionic cells. It is possible that the compression of the arteries causes, in some cases, atrophy of the stria vascularis.

Many methods have been used and studies conducted to explain the aging process. As a result of these studies, the following factors have emerged as being contributors: 1) reduction in cell production, 2) extracellular deposition of materials, 3) aging of elastic tissue, cartilage, and bone, 4) overgrowth of cells, 5) accumulation of substances in neurons, muscle cells, liver cells, and the adrenal cortex, 6) intracellular changes of uncertain nature. It is likely that all of these types of processes occur in the auditory system (Naunton, 1973).

According to the hydrodynamic theory of Bekesy, each sound which stimulates the cochlea also stimulates the area below the maximal amplitude of vibration. In the light of this concept, presbycusis is seen as a type of degenerative disease whose effects start early in noise-laden civilized environments (Rosen et al., 1964). Glorig and Nixon (1962) conducted an investigation of persons who had lived and worked in a quiet environment and had never suffered acoustic trauma. Their finding confirmed the fact that presbycusis and noise-induced hearing loss are two separate entities. All of these investigations highlight the fact that biological aging and chronological aging (which is the basis for hearing surveys) are two separate processes which do not necessarily run in parallel courses.

There is a growing need for better understanding of presbycusis and other geriatric symptoms. The processes of

social disengagement or dependence can be reduced by maximizing the geriatric patient's auditory abilities. Consonant with this goal, new methodologies in measuring the consequences of age on audition should be evolved. Investigations have been conducted using the method of comparing thresholds of acoustic reflex to predict hearing loss on populations ranging in age from 3 to 92 years of age (Jerger, 1974). No study has yet concentrated upon the effectiveness of this testing method on an exclusively geriatric population. Many members of the geriatric population prove to be very difficult to test by conventional audiological methods. An objective method of predicting hearing loss would prove to be a valuable clinical tool in testing these people. It would also serve as a confirmation of results obtained by conventional methods.

Measurement of Acoustic Impedance

The new clinical technique of acoustic impedance measurement at the tympanic membrane involves new concepts, terminology, and mathematical procedures which traditionally have not been emphasized in audiology training programs (Lily, 1973). Acoustic impedance represents the opposition by a surface to the flow of acoustic energy through that surface (Fulton and Lloyd, 1969). The acoustic impedance of the ear is dependent primarily upon the mobility of the middle ear system, which is dictated by the effects of three

relative components: stiffness, mass, and resistance (Zwislocki, 1961). Stiffness is introduced by the eardrum, the ligaments and muscles of the middle ear, the volume of air in the middle ear, and the two cochlear windows. Mass is determined primarily by the weight of the ossicular chain. This is reduced to a minimum in normal ears by the balance of the ossicular chain around its axis of rotation. Resistance is contributed by the input impedance of the cochlea and the frictional movement of the ossicles (Zwislocki, 1963). Figure 5 demonstrates these sources of impedance.

Figure 5. Peripheral auditory system and block diagram of air cavities and middle-ear structures that contribute to acoustic impedance at plane of measurement in external auditory meatus (Lily, 1973).

In a normal middle ear, contractions of either the stapedius or tensor tympani muscle produce a measurable change in acoustic impedance at the lateral surface of the tympanic membrane. This relation may be modified by middle ear infections, cochlear disease, and lesions that affect the trigeminal facial, or auditory nerves. The relation between threshold of audibility and threshold of acoustic reflex has been used as an objective measure of hearing sensitivity for young children (Jerger, 1970). It has also been used to provide an objective measure of recruitment for loudness. Contraction of the middle ear muscles will produce maximum impedance change at the tympanic membrane when the air pressure within the external auditory meatus is identical to the air pressure within the middle ear cavity (Jepsen, 1963).

Clinical acoustic impedance measurements may be classified as either static or dynamic. Static or absolute acoustic impedance measurements reflect directly the transmission characteristics of the middle ear system and are reported in absolute physical units. Dynamic acoustic-impedance measurements may be reported in relative terms and measure dynamic changes in impedance caused by the acoustic reflex or the opening of the Eustachian tube (Lily, 1973). This study will be interested in dynamic measurements.

The study of impedance involves the analysis of the "opposition" offered by a system to the "flow" of energy.

The term impedance may be defined as the complex ratio of a force-like quantity to a velocity-like quantity. Thus, the concept of impedance involves vector quantities and requires two numbers to specify it completely (Lily, 1973). The manufacturers of one of the latest electroacoustic meters note that the vectors of resistance and reactance, running at right angles to each other, combine into impedance precisely as the two legs of a right triangle "combine" in the hypotenuse; namely, by the Pythagorean relationship ($Z = \sqrt{R^2 + Y^2}$, where Z = impedance, R = resistance, and Y = reactance). The vector of reactance consists of two storage components, stiffness and inertia. Stiffness is identified as negative reactance and inertia as positive reactance. These two are 180 degrees out of phase so their net result is the algebraic sum of the two.

Berlin and Cullen (1975) gave the following formula for the total impedance of any mechanical system: $Z = \sqrt{R^2 + (2\pi fM - \frac{k}{2\pi f})^2}$ where M = the mass of a system, k = the stiffness, and R = the resistance factor. The above formula demonstrates the frequency-dependent relationships between impedance, stiffness, and mass; mass reactance is greatest in the high frequencies, while stiffness reactance (elasticity) is greatest in the lows.

Conventional pure-tone audiometry has relied upon a comparison of pure-tone thresholds obtained by measurement of the entire auditory system by air conduction with

measurement of the sensori-neural capacity by bone-conduction. As Feldman (1963) pointed out, this method only indirectly measures the function of the middle ear. The traditional air-bone gap frequently has been an inadequate method of measuring middle ear function and diagnosing impairment. Neither otoscopic nor audiometric examinations by this method have been entirely reliable in differentiating among a variety of middle-ear pathologies. Otosopic examination depends upon the subjectivity of the examiner while audiometric examination relies upon the subjectivity of the patient (Zwislocki, 1963). It became increasingly apparent to clinicians in the field of audiological testing that a more objective, definitive method of measurement was necessary.

Webster (1919) introduced the concept of acoustic impedance, but it was directed towards musical instruments rather than clinical audiology. The work of Tröger (1930) is regarded as the first systematic study of acoustic impedance at the tympanic membrane. The first practical acoustic impedance bridge was built by Steward in 1926; this was followed by the acoustic bridge of Schuster in 1934. Metz (1946) demonstrated, using Schuster's bridge, that a variety of middle ear pathologies caused a change in impedance at the tympanic membrane. He was able also to detect contractions of the stapedius muscle (Zwislocki, 1963).

The Metz bridge did not prove adequate for clinical practice; it was a large instrument which required complex adjustment and computation. It did not compensate for individual differences in ear canal size, so gave variable results. Zwislocki (1961) modified the earlier bridge of Metz. The two most significant improvements were compensation for individual ear canal volume and a stable matching impedance which provided the examiner with a direct reading of compliance and resistance at the eardrum. Now, measurement of acoustic impedance at the tympanic membrane was "not just another audiological test," but constituted a "whole new field of investigation with an inherent new methodology" (Zwislocki, 1965).

An electronic counterpart of the Metz bridge was offered by Terkildsen and Nielsen in 1960. It provided a source of air pressure which was continuously variable from a negative value through zero, to a positive value. This allowed the recording of tympanograms. All measurements were measured and calibrated in absolute figures, and a measure of complex impedance was obtained.

At the present time, two versions of the electro-acoustic impedance meter are in use clinically. The first model has greater diagnostic value than the Terkildsen-Nielsen model. It can be used for relative impedance measurement, absolute impedance measurement, and tympanometry. It utilizes the principle that the volume of

a hard-walled cavity can be determined by measuring the sound pressure level in the cavity when a pure tone is applied (Lamb, 1969). This machine measures acoustic impedance and its two components--resistance and reactance; it differs from the mechanical bridge in that, by employing only a single 220 Hz. probe tone, it measures only the stiffness characteristics of a system. Relative impedance measurements may also be made. When the intra-tympanic muscles contract, compliance of the eardrum is altered, causing a disturbance of the balance meter (Lamb, 1969).

A second version of the electroacoustic impedance meter employs two probe tone frequencies (220 Hz. and 660 Hz.) and measures admittance of energy flow and its two components, conductance and susceptance. By employing two probe tones, it measures the mass component of reactance as well as the stiffness component.

CHAPTER III

METHOD

Forty-three persons initially were selected to participate in this study as experimental subjects; however, sixteen were eliminated. In this group, an airtight seal could not be obtained on four subjects, and one subject decided not to participate. Seven persons were eliminated because of abnormal tympanograms, although conventional audiometric testing did not reveal an air-bone gap at either ear. Another four people yielded pure tone testing results indicative of conductive pathology. Ultimately, a total of 27 subjects were utilized for the study.

This clinical sample of 27 persons who were selected to serve as subjects for this study was drawn from a variety of sources, including the files of Project ARM (Auditory Rehabilitation Mobile) of Portland State University, Portland City Housing Projects for Senior Citizens, the Volunteer Department of the United States Veteran's Hospital at Portland, Oregon, and private nursing homes for the elderly. In order to participate in the study, a subject was required to meet the following criteria: 1) sixty-five years of age or older, 2) a negative history of middle ear pathology, 3) a normal tympanogram at each ear when tested on the

electroacoustic impedance audiometer.

The ages of the subjects ranged from 64 to 83 years, with a median age of 73.2 years. Eleven of the subjects (26.8 percent) were males; thirty (73.2 percent) were females. See Appendix A for screening forms used in interviewing subjects for determining a negative history of middle ear pathology.

A group of ten young adults ranging in age from 18 to 30 years with a median age of 22.7 years and zero hearing thresholds were selected to serve as controls for the study. This group also displayed normal middle ear function as determined by impedance audiometry.

Instrumentation

All impedance testing was conducted at Emanuel Medical Center in Portland, Oregon. An Electromedic Impedance Audiometer was employed for the study. The Electromedic Bridge is built in two sections, the impedance section (Model #81) and the stimulus generator section (Model #83). The headband assembly provides an impedance measurement ear-probe on the side of the probe ear, and a TDH39 earphone provides stimulation on the contralateral side. Pure tone testing was conducted in a Suttle acoustic suite, using a Maico dual-channel audiometer, Model MA-24.

There are three major functional parts of the impedance system: 1) a probe-tone generator which emits a 220 Hz.

probe-tone, 2) a simple air pump, and an electromanometer for accurately measuring individual outer canal pressure, and 3) a sound pressure level and compliance sensing measurement system which monitors the sound pressure level of the residual probe tone in the outer canal. "Residual" in this sense refers to the energy which does not flow through the tympanic membrane into the middle ear (see Figure 6).

Air pressure and sound pressure are carefully controlled and induced in the outer canal and any changes are accurately measured. When 200 millimeters H_2O pressure is introduced into the outer canal, the tympanic membrane and middle ear become rigid; the outer canal approximates a "hard-walled" cavity, and compliance is at a minimum. When pressure on both sides of the tympanic membrane is equal, a condition of maximum compliance and sound conduction and minimum impedance is obtained. A tympanogram, or graph of middle ear function, is obtained by continuously varying the air pressure in the outer canal from +200 millimeters H_2O to -200 millimeters H_2O and observing changes in the compliance. At both extremes, minimum compliance is noted. With stapedial muscle contraction, the tympanic membrane is pushed laterally and compliance is reduced, causing a recordable needle deflection of the Compliance Change Meter. The averages for the acoustic reflex have been found to be 82.2 dB for pure tones and 65.0 dB for white noise (Jepsen, 1963; Metz, 1952).

Figure 6. Diagram of impedance measurement ear-probe and related measuring devices.

The stimulus section consists of a pure-tone generator of frequencies from 250 Hz., 500 Hz., 1000 Hz., 2000 Hz., and 4000 Hz. (250 Hz. and 4000 Hz. were not used in this study); a broad-band noise generator with an attenuation range of 40 dB to 125 dB SPL; a low-pass noise generator with a cut-off frequency of 2600 Hz.; and a high-pass noise generator with a cut-off frequency of 2600 Hz. All noise generators have an attenuation range of 40 dB to 125 dB SPL and an attenuation of 24 dB per octave (personal communication from Irwin Klar, Vice President American Electromedics Corporation, July, 1975). A stimulus interrupter switch initiates presentation of the selected stimulus and may be adjusted to provide a six second delay between action and stimulus presentation for reflex latency measure.

A physical calibration of the intensities of the various reflex eliciting signals is virtually impossible because of many variables. Among these are differences in the way different sound level meters measure broad band noise, calibration accuracy of pure tones, and frequency response variations in individual earphones (Jerger, 1974; Klar, 1975). Because of the many variables, a biological calibration was performed as follows: ten subjects (ages eighteen to thirty years) with normal hearing were tested both by pure tone audiometry and by impedance reflex measurements as described in the methods section of this report. The average of these scores were used as norms

for the equipment. A physical calibration of the broad-band noise was regularly performed, using a Bruel and Kjaer sound level meter, Model 1613.

Test Procedure

Prior to the individual audiological evaluation, each subject was given an otoscopic evaluation to determine the condition of the tympanic membrane and the presence or absence of cerumen. If excessive cerumen was present, the subject was instructed to have his ear cleaned before any further testing procedures were administered.

Impedance testing was conducted in a relatively quiet examining room with the subject seated in a chair facing the examiner. Prior to each evaluation, the procedure was explained to the subject, using an illustration of the inner, middle, and outer ear, and explaining how the probe was to be placed to secure a seal in the meatus. The subject was told to sit very quietly, to avoid swallowing, talking, or coughing, if possible. A headband was placed on the subject's head; an earphone was secured over the ear contralateral to testing, and a seal was obtained with the use of a soft rubber cuff in the test ear. Static compliance measurements were obtained at both ears. This was done by measuring the volume of air between the probe tip and the tympanic membrane when the membrane was clamped by a +200 millimeters/H₂O air pressure and subtracting the net value

from that obtained at the point of maximum compliance of the tympanic membrane.

The second measurement obtained was tympanometry. A hermetic seal was effected in the ear canal; air pressure was varied from +200 millimeters/H₂O to -200 millimeters/H₂O. The procedure produces a tympanogram, which depicts compliance changes in relation to air pressure variation in the ear canal. After tympanometry, the air pressure in the ear canal was reduced to the point of maximum compliance of the tympanic membrane, and reflex thresholds were obtained. Pure tones of 500 Hz., 1000 Hz., and 2000 Hz. were presented to the test ear. Presentation was administered in 5 dB steps until the lowest point that a 10 millimeter/H₂O deflection of the needle was noted. Following these measures, unfiltered white noise, low-pass white noise (below 2600 Hz.), and high-pass white noise (above 2600 Hz.) were presented in turn. Reflex thresholds of these different stimulus presentations were noted.

A third measurement obtained was pure-tone air and bone conduction thresholds. As previously indicated, none of the 27 experimental subjects or the 10 control subjects displayed an air-bone gap.

The results of conventional pure-tone audiometric data for each individual were then compared with results of the SPAR test (Sensitivity Prediction by the Acoustic Reflex). In cases of doubtful responses to pure tone

testing, a speech reception threshold was obtained on the individual and used as a basis for comparison of results.

The following formula (Jerger, 1974) was employed to determine the difference between thresholds of pure tones and white noise:

$$\begin{aligned}
 a &= \text{reflex threshold SPL for 500 Hz.} \\
 b &= \text{reflex threshold SPL for 1000 Hz.} \\
 c &= \text{reflex threshold SPL for 2000 Hz.} \\
 d &= a + b + c / 3 \\
 e &= \text{lowest reflex threshold SPL among a, b, and c} \\
 f &= \text{reflex threshold SPL for BBN} \\
 l &= d - f \text{ (PTA - BBN)} \\
 m &= a - f \text{ (500 - BBN)} \\
 n &= e - f \text{ (PT - BBN)} \\
 D &= \frac{1 + m + n}{3}
 \end{aligned}$$

To predict slope of loss, the difference between thresholds for low-pass and high-pass filtered white noise was calculated. If the difference was zero or positive, a flat configuration was predicted. If the difference was in the range of -1 to -5, a gradual slope was predicted. If the difference exceeded -5, a steep slope was predicted (Jerger, 1974). Table III provides criteria for prediction of sensitivity loss.

The following criteria will be followed for categorizing losses:

Normal - Pure tone average less than 25 dB HL
 Mild-Moderate - Pure tone average 25 dB to 49 dB HL inclusive
 Severe - Pure tone average 50 dB to 84 dB HL inclusive
 Profound - Pure tone average 85 dB HL or more

The following criteria will be followed for categorizing slope of loss:

TABLE III
CRITERIA FOR ASSESSMENT OF HEARING SENSITIVITY

Difference in reflex thresholds pure tone and broad band noise	Threshold levels for broad band noise in SPL	Prediction of hearing sensitivity
20 or larger	anywhere	normal
15 - 19	80 dB or less	normal
15 - 19	81 dB or more	mild-moderate
10 - 14	anywhere	mild-moderate
less than 10	89 dB or less	mild-moderate
less than 10	90 dB or more	severe
reflexes not observed		profound

Jerger (1974).

Flat - Difference between 1k and 4K - less than 5 dB

Gradual - Difference between 1k and 4K - 6 dB to 40 dB

Steep - Difference between 1k and 4K - 40 dB+

Data Analysis

Data relative to the geriatric sample and the control sample of young adults were analyzed as follows: 1) distribution of errors of assessment according to category of loss, 2) distribution of errors of slope of loss according to category, 3) distribution of errors of combined severity of loss and slope of loss according to category, 4) correlation of pure-tone averages of thresholds of hearing with reflex thresholds of stimuli, 5) means, standard deviations, and ranges of reflex thresholds of geriatric adults and young adults.

A Z test for statistical probability was performed for assessment of severity of loss, slope of loss, and combined severity and slope of loss to evaluate the possibility of results happening by chance.

A one-tailed t test of related means was performed to ascertain if there was a statistically significant difference in means of reflex thresholds between young, normal-hearing adults and geriatric subjects.

Pearson Product-Moment Correlation Coefficients were calculated on the correlation between hearing threshold levels and reflex threshold levels for all types of stimuli used in this research project.

CHAPTER IV

PRESENTATION OF RESULTS

The first question under investigation at the beginning of this study was: Will the determination of acoustic reflex thresholds be statistically valid in assessing magnitude of hearing loss among persons over sixty-five years of age who have no known otologic problems other than presbycusis? Table IV compares SPAR assessment results with actual results of conventional audiology for fifty-four aging ears. Of the twenty-four ears assessed as having normal hearing sensitivity, fourteen had audiometric results within normal limits (25 dB pure tone averages, or less); ten had hearing within mild-moderate limits (25 dB to 49 dB PTA); none had severe or profound losses. Of the twenty-seven ears assessed to have mild-moderate losses, sixteen actually yielded audiograms in this category; nine showed normal hearing; and two had severe losses. Of the three ears which were assessed to have profound hearing losses (lack of reflexes), one was shown to be normal and two were shown to have mild-moderate losses.

Table V displays the distribution of assessment errors for loss of hearing sensitivity. Accuracy of assessment was highest for mild-moderate (59.26 percent) and normal (58.34

percent) categories, and the lowest for profound (0 percent). In only three ears were serious errors of assessment made, i.e., that of assessment of profound hearing losses. Of this group, none were profoundly deaf. A Z test of statistical probability indicated that results of severity of loss were significant at the .01 level of confidence (Mendenhall, 1967).

TABLE IV
ASSESSMENT OF SEVERITY OF LOSS

N=54		GERIATRICS			
Actual	Assessed				
	Normal	Mild-Moderate	Severe	Profound	
Normal	14	9	0	1	
Mild-Mod.	10	16	0	2	
Severe	0	2	0	0	
Profound	0	0	0	0	
Total	24	27	0	3	

TABLE V
DISTRIBUTION OF ERRORS
ASSESSMENT OF SEVERITY

N=54			
Type of errors	Number of ears	Percentage	
None	30	56%	
Moderate	21	39%	
Serious	3	5%	
Total	54	100%	

$$Z = 3.23$$

$$P = .0006$$

$$\alpha = .9994$$

Figure 7. Distribution of predictive errors according to category of prediction (N=54) (After Jerger, 1974).

The second question under investigation was: Will acoustic reflex thresholds be statistically valid in assessing slope of loss among members of this sample? The results for slope of loss were not successful for the geriatric subjects, as displayed in Table VI. Assessment of slope of loss was also unreliable among the sample of young adults tested. In spite of regular physical calibration of low-pass and high-pass noise stimuli, reflex thresholds for these stimuli varied widely. Low-pass thresholds ranged from 70 dB to 105 dB, and high-pass from 75 dB to 110 dB, even though all subjects displayed absolutely normal 0 dB hearing levels. It should be noted, however, that the largest percentage of errors (56 percent) were again moderate. Only 4 percent of errors of slope were serious. No loss configuration was assessed as flat or gradual among the steeply sloping losses (see Table VII).

TABLE VI
PREDICTION OF SLOPE OF LOSS

N=45		GERIATRICS		
Actual	Predicted			
	Flat	Gradual	Steep	
Flat	1	2	2	
Gradual	10	11	10	
Steep	0	3	6	
Total	11	16	18	

TABLE VII
DISTRIBUTION OF ERRORS
ASSESSMENT OF SLOPE

N=45		
Type of error	Number of ears	Percentage
None	18	40%
Moderate	25	56%
Serious	2	4%
Total	45	100%

$$Z = .3000$$

$$\sigma = .6368$$

$$P = .3632$$

Table VIII compares the actual pure tone hearing loss slope categories of 44 aging ears with the slope of loss predicted from the SPAR data. An expected drop in the accuracy of assessment of the test occurred because of the poor assessment ability of the slope of loss. Table IX shows the distribution of errors. The majority of errors were moderate (77 percent); only 2 percent were severe. Persons with severe and profound losses were necessarily eliminated from this test because of lack of reflexes with high-pass noise stimulus.

The third question under investigation in this study was: Will reflex thresholds of the older population prove

TABLE VIII
ASSESSMENT OF BOTH SLOPE OF LOSS AND SEVERITY

N=44				
GERIATRICS				
Actual	Predicted			
Hearing Loss Slope Category	Normal	Normal Sloping	Mild-Mod.	Mild-Mod. Sloping
Normal	0	2		2
Normal Sloping	8	2	4	2
Mild-Moderate	2			
Mild-Moderate Sloping		9	5	7
Severe			1	
Total	10	13	10	11

TABLE IX
DISTRIBUTION OF PREDICTIVE ERRORS
ASSESSMENT OF SLOPE AND SEVERITY

N=44		
Type of error	Number of ears	Percentage
None	9	21%
Moderate	34	77%
Severe	1	2%
Total	44	100%

$$\begin{aligned}
 Z &= .91 \\
 \alpha &= .8289 \\
 P &= .1711
 \end{aligned}$$

to be statistically different from those of the young adult control group? Table X presents means, standard deviations,

TABLE X
MEANS, STANDARD DEVIATIONS, RANGES
OF REFLEX THRESHOLDS (dB)

GERIATRICS							
	500 Hz.	1K Hz.	2K Hz.	PTA	Wht. N	LPN	HPN
$\bar{X}$	105.64	99.50	104.19	102.68	93.43	97.65	103.30
SD	11.02	8.23	10.86	4.12	11.02	11.37	23.25
R	81-126	76.5-131	88.5-133.5	82-123	75-115	75-125	85-115
YOUNG ADULTS							
	500 Hz.	1K Hz.	2K Hz.	PTA	Wht. N	LPN	HPN
$\bar{X}$	96.25	93.5	94.5	93.3	71.5	89.0	92.0
SD	6.17	4.97	7.0	3.59	5.36	14.15	14.59
R	91-116	86.5-101	83.5-103.5	81-103	70-90	70-105	75-110

and ranges of reflex thresholds for young, normal-hearing persons and geriatric subjects. Thresholds for the older population were from 6 dB to 22 dB greater than they were for young normals. The greatest difference in levels was noted for white noise, with significant differences also observed for low-pass and high-pass filtered noise.

Figures 8 and 9 demonstrate the differences in ranges and standard deviations between young adults and geriatrics. It is apparent that the ranges of reflex thresholds are far greater than in the young population.

Table XI displays results of a one-tailed t test of related means (Bruning, 1968) comparing reflex threshold

Figure 8. Reflex thresholds of normal young adults, depicting means, ranges and standard deviations.

Figure 2. Reflex thresholds of geriatric adults, depicting means, ranges, and standard deviations.

TABLE XI
t VALUES--NORMALS AND GERIATRICS
REFLEX THRESHOLD LEVELS

Stimulus	t Value	Level of Confidence
500 Hz.	3.85	.005
1K Hz.	3.09	.005
2K Hz.	3.73	.005
PTA	9.11	.005
White Noise	5.46	.005
Low-Pass Noise	2.73	.005
High-Pass Noise	2.07	.025

levels of young adults and geriatrics. Significant differences were revealed for all stimuli beyond the .005 level of confidence except for high-pass white noise.

Additional information regarding the results of this study are presented in Table XII which demonstrates the correlations calculated on the relationships between thresholds of hearing data and reflex thresholds for the various stimuli. Pearson Product-Moment Correlation Coefficients indicated the relationship between pure tone average of the speech frequencies and reflex thresholds were statistically significant, while those between reflex thresholds and thresholds of hearing at 4000 Hz. and 8000 Hz. were not. These results suggest that the acoustic reflex is a mechanism which is triggered by neural activity in the lower frequency area of the cochlea, and thus serves to attenuate low-frequency sounds to a greater extent than those of high-frequency.

TABLE XII
CORRELATIONS BETWEEN THRESHOLDS
AND REFLEX LEVELS

N		r*	Sig. Values	Significance
44	4K and High-Pass Wht. Noise	.02	.10=.2428	Not Sig.
35	8K and White Noise	.08	.10=.2573	Not Sig.
51	4K and White Noise	.17	.10=.2428	Not Sig.
30	8K and High-Pass Wht. Noise	.13	.10=.2573	Not Sig.
51	PTA and Reflex at 1K	.78	.001=.4433	Sig.
51	PTA and Low-Pass Wht. Noise	.65	.001=.4433	Sig.
45	PTA and High-Pass Wht. Noise	.44	.01=.3421	Sig.
51	PTA and White Noise	.37	.01=.3541	Sig.
51	PTA and Reflex at 500 Hz.	.75	.001=.4433	Sig.
50	PTA and Reflex at 2K	.35	.02=.3218	Sig.

*Pearson's Product-Moment Correlations.

Discussion of Results and Implications

Clinical Implications

The results of this research were gratifyingly satisfactory for assessment of loss of hearing sensitivity in the geriatric population. The percentage of correct assessments was 56 percent; this compares very favorably with Jerger's 60 percent. The percentage of moderate errors (one scale divergence from actual) was 39 percent as compared to 36 percent in Jerger's studies. The percentage of serious errors (two scale divergences from actual) was 5 percent; Jerger found 4 percent. These results were very significant in view of the many variables which enter into the hearing sensitivity of the presbycusis subject. Among these

variables are recruitment, high-frequency hearing loss, variations in ear canal size, and conductive components.

The error in prediction of profound losses may be due to the fact that, in any group of normal hearing persons, there appears to be a very small percentage from whom an acoustic reflex cannot be elicited. This is a probable weakness of the SPAR test for individual patients. A very encouraging result of this testing was that 39 percent of the errors of assessment were only moderate (one scale from actual hearing). As a result, no person with a severe loss was predicted to have normal hearing. This could be a very serious mistake in a very old or a very young population.

Static compliance measures were calculated for all subjects on which tympanometry was performed. They were calculated by subtracting the volume measurement taken at +200 millimeters/H₂O air pressure in the external meatus from that taken at the point of maximum compliance of the tympanic membrane. The process eliminates the external ear canal volume and gives a value representative of the amount of mobility of the middle ear in cubic centimeters. Normal static compliance values range between .30 cc. to 1.60 cc. Stiff systems may demonstrate compliance values of less than .30 cc. and flaccid systems greater than 1.60 cc. (Jerger, 1972). Of the 51 geriatric ears on which this measure was taken, ten exhibited static compliance not within these limits. Six suggested flaccid middle ears;

four gave results which indicated stiffness. This measure was used as a check on normal middle ear function in the study.

Four of the original forty-three subjects (9.2 percent) were discovered with abnormal tympanograms which were accompanied by significant air-bone gaps. These people were urged to seek medical assistance. One might question the extent to which long-neglected, medically-correctible, hearing problems may exist in the older population, especially in view of the fact that none of these individuals acknowledged prior otologic symptoms.

The SPAR test provides a wholly new, objective method of assessing hearing sensitivity, and on the basis of this study, should prove very valuable as a means of checking the validity of pure tone thresholds in aging subjects.

Tympanometry is now becoming a vital, routine practice in most audiology clinics. Occasionally an ear may be encountered which is difficult to seal, but this is a very small segment of the geriatric population. The entire process can be completed in twenty minutes; this proves to be a wise investment of time in terms of the additional information which it provides.

Theoretical Implications

In the geriatric group, eight persons were found to

have hearing sensitivity within normal range, based upon the average of pure tone thresholds at 500 Hz., 1000 Hz., and 2000 Hz. and no evidence of noise-induced hearing loss. All of the "normal" sixteen ears displayed higher reflex levels for both average pure tone thresholds ($X = 101.4$ dB) and white noise ($X = 90.4$ dB) than did the group of normal young adults ($X = 93.3$ dB PTA) and ($X = 71.5$ dB) white noise. While still within normal limits, the older group of normal hearing persons had pure tone averages of 9.9 dB higher than those of the young adults. This may explain the 10 dB difference in the mean PTA reflex thresholds, but not the 21 dB difference in those for white noise. A very small group (four people) exhibited normal hearing in the high frequency area, but more high-pass noise energy was required to elicit reflexes than for young normals with similar thresholds. It appears that neural changes occur within the aging auditory system even in the presence of normal peripheral hearing sensitivity. This tends to confirm the theory (Scharf and Hellman, 1966; Jerger, 1974) that the critical bands widen and decrease in numbers in the aging process.

An interesting group consisted of persons who displayed abnormal tympanograms in the absence of air-bone gaps. Five persons of the original forty-three (11.6 percent) of the group fell into this category and provided further confirmation of Goodhill's theory (1969) that presbycusis involves an aging process in ligaments, muscles, and

connective tissues, and may include mechanical lesions which involve the tympanic membrane and the ossicular chain movements.

One seventy-one year old female subject was found to have pure tone averages of 2 dB and 3 dB. This serves to emphasize that chronological aging and biological aging are two separate entities, and contradicts Glorig's and Nixon's hypothesis (1962) that progress of hearing loss as a function of age can be described and predicted quite accurately.

CHAPTER V

SUMMARY AND IMPLICATIONS

The purpose of this study was to investigate the use of the SPAR (Sensitivity Prediction by the Acoustic Reflex Test) on a sample of older adults who had negative otologic histories. Twenty-seven subjects ranging in age from sixty-four to eighty-three years of age (54 ears) were chosen as subjects. A normal-hearing, young adult group, ranging in age from eighteen years to thirty years of age, served as controls. Reflex threshold levels were obtained, using 500 Hz., 1000 Hz., and 2000 Hz. pure tone and noise stimuli. Assessment of hearing loss was calculated on the difference between measures to ascertain hearing loss category and slope of loss according to a formula developed by Jerger (1974).

The following conclusions were made on the basis of data collected in this investigation:

- 1) The SPAR test was significant in assessing hearing sensitivity levels among the sample of geriatric subjects.
- 2) The SPAR test was not statistically significant in assessing slope of loss in this sample of geriatric subjects.

- 3) Reflex thresholds were consistently higher for aging than for a sample of young adults with normal hearing. This was true even among elderly subjects with average pure tone thresholds in the normal range.
- 4) A significant portion of this carefully screened older adult sample displayed abnormal middle-ear function (as measured by tympanometry) in the absence of conventional audiometric indications of this.
- 5) A significant portion of this older adult sample provided audiometric evidence of middle-ear pathology.

Implications for Further Research

Very little research has been conducted in the area of tympanometry for the older, hearing-impaired patient. This study was limited to a relatively small number of subjects and a large percentage of female subjects. Further research possibilities suggest using this technique on larger numbers of subjects and on a sample of male geriatric subjects. More research is also needed to establish reliability of assessment of slope of loss on a young, sensorineural population.

An additional field of research lies in the investigation of the critical band patterns in the aging process and

the influence of this patterning on the older subject's perception of speech. A shift of the critical band patterns in the high-frequency area of the basilar membrane may tend to impair a vital link in the speech discrimination process and cause distortion of the speech signal. This is a field of research virtually untouched at the present time and one which offers exciting research possibilities for the future.

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APPENDIX A

QUESTIONNAIRE

1. Name: _____
2. Address: _____
3. Telephone: _____
4. Birthdate: _____
5. Did you notice any significant hearing loss before the age of 50?
6. Was your hearing loss of gradual or sudden onset?
7. Have you ever had middle ear infections?
8. Do you suffer from frequent earaches?
9. Have you ever had surgery on your ears?
10. Were you exposed to a noisy environment in your job?
11. Do you include among your hobbies hunting, motorcycle riding, or home machine shop?
12. Were you ever a member of an active military unit?
 - a) Did your military occupation involve intense noise exposure?
13. Do you consider your general health to be good?
14. How would you rate your hearing - good - fair - poor - excellent?

I give my consent to use the information gained from this evaluation for thesis and research purposes.

APPENDIX B

1977 DIFFERENTIAL LOUDNESS SUMMATION TEST

TYMPANOGRAM