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THE EFFECTS OF AGE ON TONE-BURST EVOKED OTO-ACOUSTIC EMISSIONS

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Willard Charles Hooks, Jr.

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of the requirements for

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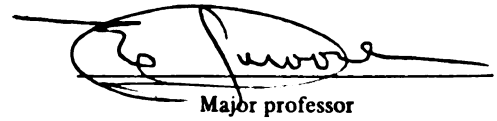

Major professor

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THE EFFECTS OF AGE ON TONE-BURST EVOKED OTO-ACOUSTIC EMISSIONS

By

Willard Charles Hooks, Jr.

A THESIS

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ABSTRACT

THE EFFECTS OF AGE ON TONE-BURST EVOKED OTO-ACOUSTIC EMISSIONS

By

Willard Charles Hooks, Jr.

This study was designed to determine if the age of human subjects affects the tone-burst oto-acoustic emissions. In addition, the operating characteristics of an experimental ear probe assembly were tested through the analysis of emissions.

This present study employed an experimental probe assembly which was placed in the outer two-thirds of the ear canal. Two groups of women were divided by age. The control group consisted of subjects from 18-25 years of age. The experimental group consisted of women ≥40 years of age. Statistical analyses (ANOVA) were used to test three hypotheses based on differences between emission amplitude and latency. Short tone-bursts were used. The frequency composition of emissions was tested for two frequencies of 800 and 1500 Hz. Three stimulus amplitudes of 55 dB, 45 dB and 35 dB p.e. dB SPL were presented as repeated measures. Statistical significance was not achieved, however, a few salient trends were noted: (1) The emissions decreased in proportion to the stimulus amplitude for both groups (2) Emissions in the younger groups were evoked at a lower presentation level.

To my parents

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TABLE OF CONTENTS

APPENDICES

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

In the context of engineering and material science, acoustic emission (AE) may be defined as the tensional or compressive stress N/m^2 generated during dynamic processes in materials. By convention, the term "acoustic emission" has been used to describe low level sounds or pressure waves in materials. Under most circumstances, the acoustic emission is not audible to the unaided ear; however, in less conventional forms (timber subjected to loads near structural failure) these pressure waves (described as clicks, sounds, micro seismic activity, and elastic shock) can be detected above the threshold of normal hearing Re: 0 dB HL @ 1.0 kHz (Liptai, Harris, & Tatro, 1972). In practice, acoustic emission analysis is a highly sensitive technique used to detect active microscopic structural events in materials. These events are characterized by transient elastic waves that generate localized regions of energy (Matthew & Hay, 1983; Schofield, 1975).

The source of AE activity e.g., in geologic materials and/or mediums, is rather nebulous; however, it appears that the origin may be related to dynamic processes of deformation and failure, accompanied by a sudden release of strain energy (Hardy, 1981). Strain is a result of a change in the volumetric dimensions or shape of a solid due to applied forces. These forces may change the body concerning volume and length per unit length. This is the kind of process that perhaps occurs within the cochlear partition of most mammals.

Recently, AE technology has been used to describe the use of a medium subjected to load factors. The practical use of emission analysis has advanced with the use of dedicated software and hardware such as averaging programs, microcomputers, filters, and transducers sensitive enough to detect dislocation phenomena in metals and other

materials (Liptai et al., 1972). This marked the first use of AE analysis in modern day acoustic emission technology (Kaiser, 1953). Such technology has offered a viable method for evaluating the basic structural integrity of many engineering structures (Schofield, 1975). The technique has developed rapidly over the last two decades as a nondestructive evaluation technique and as a tool for materials research (Matthews, & Hay, 1983; Schofield, 1983), particularly as an indicator of imminent structural failure (Williams, 1980).

Statement of Problem

Considering auditory (micro-mechanics) the incidence of evoked oto-acoustic emissions (EOAEs) in older adults is not well documented. As such, a logical sequel to probable diminished mechanical activity within the cochlea may be a corollary to diminutive emissions. Therefore, to make an appropriate analysis of OAEs, it is essential that the nature of damage be understood. In like manner, the relation between assumed vibratory activity, i.e., traveling wave and the effects of aging, may become requisite for accurate OAE analysis. However, biological aging itself is not clearly understood and the implications for aging sensory systems may be more obscure. To be sure, there is a growing need to address these problems of geriatric research in audiology.

Conceptual Framework

Applying the basics of AE analysis to biomechanics, Kemp (1978) discovered narrowband 'oto-acousitic emissions' evoked by transient pulses from the ear canal. He postulated that there was an active process within the cochlea that provided feedback to modulate the traveling wave (von Békésy, 1960) in respect to the vibratory motion of the basilar membrane. This basic premise of oto-acoustic emissions was proposed over forty years ago by Gold (1948). The exact 'active zone' of OAEs has not been established,

however, it has been generally postulated by one theory that OAEs are of a mechanical origin (Anderson et al., 1979; Kemp & Brown, 1986, Wilson, 1980; Zerlin, 1969;). In this context it was surmised that emissions originated from various active regions within the cochlea.

In contrast, another theoretical construct of emissions suggests that local impedance discontinuities produce isolated emissions fixed or restricted to a place along the cochlear partition. These emissions arise from the cochlea as sharp peaks in normal subjects (Wit and Ritsma, 1980; Ruggero, Rich, and Freyman, 1983).

Emissions from the ear canal suggest a normal cochlea composed of sensory cells that oscillate in response to sound at low levels (Anderson et al., 1979; Kemp, 1978, 1986). In practice, the technique is very sensitive to cochlear pathology, i.e., hair cell damage and insults to the stria vascularis (Kemp et al., 1986). In contrast to material science, the detection of emissions from the ear canal is a benign indicator of structural integrity (Kemp, 1978). It turns out that oto-acoustic emissions are distinct responses that may indicate cochlear pathology (Kemp, 1978, 1986, Kemp et al., 1986).

Taken together, oto-acoustic emissions like other electrophysiological responses, may be affected by several independent variables (Wit and Ritsma, 1980; Kemp et al., 1986) including several stimulus parameters (Moore, 1983, 1984). In this context several investigators have observed and recorded oto-acoustic emissions under varying conditions: such as intensity (Kemp, 1978; Zwicker, 1986; Probst, Coats, Martin, & Lonsbury-Martin, 1986; Neely, Norton, Gorga, & Jesteadt, 1986), intense noise, (Kemp, 1986; Norton, Chaplin & Mott, 1986; Brown, 1988), frequency variations (Kemp, 1978, 1986; Zwicker and Schloth, 1984; Strickland, Burns, & Tubis, 1985), temperature (Whitehead, Wilson & Baker, 1986), gender (Strickland et al., 1985; Probst et al., 1986) ototoxicity (Rutten, 1980; Kemp et al., 1986; Tanaka, 1988; Tanaka, O-uchi, Shimada, Koseki, 1988), posture (Schloth et al., 1983), psychological effects (Horst, Wit, & Ritsma, 1983), and age (Strickland et al., 1985; Bonfils, Uziel, & Pujol, 1988a).

Theoretical Assumptions

Clearly, the theoretical construct of aging has been identified and defined by several investigators, through observations from predictive behavior and/or processes. In early investigations, aging processes were described as factors that rendered individuals more susceptible, as they grew older, to various intrinsic or extrinsic factors. Progressively, the interaction of these components may cause death (Comfort, 1960).

It has been noted that aging is a naturally developing biological process that limits the adaptive possibilities of an organism. The inability to adapt increases the likelihood of death or reduces the life span and produces age-related pathology (Froklis, 1982).

Senescence, on the other hand, has referred to the overall considerations of age-related changes , the majority of which occur during the period in the latter years life (Comfort, 1982). The condition of senescence refers to degenerative changes that usually mark the ending of life (Moment, 1982). Aging and senescence are universal and intimate properties of most living organisms.

The foundation of gerontology research itself has certainly been built upon various definitions of aging. A basic component to any definition has been the theory that supports the propositions that serve to explain biological aging. In view of several definitions of aging, there have been several biological theories postulated with little agreement among investigators. In practical terms, theories have been of little use if they have not enabled us to make predictions and to control various phenomena. In this context, the inherent synthetic framework of gerontology has made it difficult to develop a composite perspective of aging. Other reasons for dispute and contradiction reside in the complex interaction of systems and subsystems from several species. However, a large number of aging theories has offered plausible explanations derived from predictable commonalties among species and the laws governing known biological function(s). The theories of aging have led to a dichotomy between programmed and

predetermined processes or stochastic and random processes (Arking, 1991). A classification of the basic organization from several constructs is shown in Table I-1. Aging is not the result of a diminishing cell count, nor is aging the result of cell wear beyond repair. However, complex processes associated with aging may pertain to more than one cause.

Table I-1 A classification of aging theories

Level at Which Effect of Change Is Executed	Origin of the Change	
	Stochastic	Programmed
Intracellular	Altered proteins Somatic mutations DNA damage and repair Error catastrophe Dysdifferentiation Free radical Waste accumulation	Metabolic theories Genetic theories Selective Death
Intercellular	Cross-linkage Wear and tear	Neuroendocrine Immunological

Note. From Biology of Aging: observations and principles by R. Arking 1991 Englewood Cliffs: Prentice Hall.

Population Trends

Almost invariably, there have been similar trends in population projection outside the United States which have addressed the implications of aging for public health purposes. In Switzerland, the average percentage 65+ population has been forecast to be 75% and 85% for males and females respectively. In like manner, the number of people of 80+ will have a projected increase to approximately 6% in 2015 from 2% in 1980 (Roos, 1990). Reports of demographic gerontology have shown a characteristic shift toward 65+ in Great Britain and in many regions of the former Soviet Union, in particular Soviet

Georgia (Duckett, 1991; Evans, 1984; Medvedev, 1984). In contrast, however, the 65+ population has not risen above 11% from recent estimates from South America. Figures from much of Asia show similar results (Bureau of Census, 1987). The unknown health effects of aging among undeveloped and developing nations cannot be indexed. As such, the possible underlying causes of aging, in certain cultures, will probably remain unclear without culturally appropriate health care (Olshansky, Carnes, and Cassel, 1993).

Implications of Aging in the United States

As the year 2000 A.D. approaches, it is becoming clearer that the largest segment of the United States population is approaching the status of being also the oldest segment. During the period between 1950 and 1980 the population age 65+ doubled (Taeuber, 1983). There was also an increase in the percentage of the total population. Present data suggest that there is a dip in aging trends. The decline is due to low Depression era birth rates (Birren, Vercruyssen, and Fisher, 1990; Verbruggee, 1989; National Research Council, 1988). However, there will be an apparent shift in the U. S. population toward age 65 and older with significant implications for the nation's social, economic, and public health institutions (see Table I-2) To keep pace with the growing phenomenon of an aging population, it is essential that we meet the needs of the elderly today. An incomplete understanding of fundamental aging, senescence, and their antecedents justifies the need for future research.

Table I-2 Actual and Projected Growth of the Aged United States Population (numbers in millions)

Year	Total Population	65 - 74 Years No.	65 - 74 Years %	75 - 84 Years No.	75 - 84 Years %	85 - Years + No.	85 - Years + %	65 - Years + No.	65 - Years + %
1980	226.5	15.0	66	7.7	3.4	2.2	1.0	25.5	11.3
1990	250.0	18.0	7.2	10.3	4.1	3.5	1.4	31.8	12.7
2000	268.0	17.7	6.6	12.2	4.6	5.1	1.9	35.0	13.1
2010	283.0	20.3	7.2	12.2	4.3	6.8	2.4	39.3	13.9
2020	296.3	29.8	10.2	14.3	4.8	7.3	2.5	51.4	17.3
2030	304.3	34.4	11.3	21.1	6.9	8.8	2.9	64.3	21.1
2040	308.0	29.2	9.5	24.5	8.0	12.9	4.2	66.6	21.6
2050	308.9	30.0	9.7	21.0	6.8	16.1	5.2	67.1	21.7

Note. From America in transition: an aging society. by Taeuber, C. (1983). Current Population Reports, Special Studies Series P-23, No. 128. Bureau of the Census. Washington, D. C.: U.S. Department of Commerce.

The table above emphasizes also a growing need for health professionals to be trained in the area of gerontology and related area of aging research and treatment. However, the current support for research in the biomedical aspects of aging and geriatrics is less evident.

To appreciate the characteristics of aging in human and other mammalian species, insects have been useful as models for testing the theories of aging. Generally, not all insects are suitable for experimentation in the area of aging. Anatomically and biochemically the functional requirements for all organisms meet in several ways. However, the fruit fly *Drosophila melanogaster* shows age-dependent deterioative changes which suggest that senescence accompanies time-dependent changes. These transformations in the cells are rather common to all multicellular organisms (Lamb, 1984).

At present, gerontology is a field of inquiry the focus of which is not a formal study of medicine as pediatrics, psychiatry, or internal medicine. Gerontology does not have its own fundamental constructs but rather crosses several disciplines. Audiology

emerged as another field that combined several areas in social and biological science. As such, researchers and practitioners gerontologists share an interest in aging from a variety of backgrounds. Demographics, federal training programs, and integrative analysis of several areas provide conditions for emerging gerontology questions (Verbruggee, 1989). The specific questions vary but follow a few common themes: (1) What is the cause of the deterioative changes that occur during the life span of certain species? (2) Why do closely related species (mouse and human) have very different life spans? (3) Can deterioative /degradative changes be postponed or reversed? (4) Is it possible to prolong life and is it worth it? (Arking, 1991)

Definition of Aging

To appreciate the attributes of aging, Arking (1991) made some distinctions between aging changes and normal developmental changes. The following observations will clarify general considerations to the study of gerontology: (1) Not all time-dependent changes are fundamental aging changes. (2) Aging changes usually occur at maturity (the genesis may occur earlier). (3) Aging processes are deleterious, progressive, and cumulative.

Aging processes may involve some basic changes. However, Strehler (1982) suggested the following conditions constituted true aging processes:

(1) deleterious - reduced function

(2) progressive - occur gradually

(3) intrinsic - not due to modifiable environmental agent

(4) universal - all members of a species should show such gradual deficits with advancing age.

Taken together, a strict working definition for aging has been proposed, the words of which lead to an acronym CUPID: cumulative, universal, progressive, intrinsic, and

deleterious. These changes that arise with the arrival of reproductive maturity and consequently lead to death (Arking, 1991, p. 9). As such, CUPID's heuristic function helps researchers to organize experiments and aids interpretation. Thus, a powerful framework for explaining various phenomena can be supported by a stringent operational definition.

A Prospectus of An Aging Auditory System

Almost invariably, the likelihood of illness increases with advancing age. In many instances, poor health ushers in diseases, concomitant debilitating psychological and physiological conditions. However, using the strict CUPID criteria, an aging auditory system is unaffected by environmental or extrinsic influences. Intrinsic processes affect aging. The incidence of a hearing disorder begins to emerge with older persons. Approximately 25% of those among 65 and 74 years of age possess some form of diminished hearing, part of which is nearly twice that of persons among 45-64 years of age (NCHS, 1981, 1982). If current trends continue, the incidence of hearing impairment, in the 65-74 age range, will continue to grow linearly possibly to the year 2050 (Fein, 1983). We are just beginning to understand some of the biological processes coterminous with advancing hearing loss.

Significant differences in the susceptibility to acoustic trauma emerge with extrinsic or environmental influences during the aging process. Nevertheless, it is difficult to make determinations of the effects of aging. In an industrialized society, the effect of aging on the auditory system is an immediate concern for those who make decisions in a rather litigious environment within this society. The judicial system is replete with numerous court cases fueled by employer liability. At best, we can only make estimates of hearing loss from group data as a function of age (Lebo and Redell, 1972). Conservative interpretation sometimes follows (in a strict understanding) the theoretical constructs of aging process(es) within the auditory system.

The normal aging processes are diverse. As such, various forms of deficiency emanate. Generally, aging has less of an effect on the air and bone conduction pathways than on the neural and sensory components of the auditory system. Secondary effects of the peripheral ear, i.e., growth of longer and thicker hair, accompanying dryness, and thinning shadow senescence. However, hearing loss related to age complicates pre-existing conditions with increasing severity. The progression often advances from the periphery to the central auditory system.

The term presbycusis implies a hearing loss caused by the degenerative changes of aging. The age and onset rate may vary widely. Logically, the durability of the auditory mechanism, as other body systems, changes significantly. Fatigue and physical stress subject force (strain, shear) to the ear (Schuknecht, 1974). Clearly, presbycusis is a part of a constellation of symptoms that revolve around diminished bodily functions. As such, cochlear hearing loss invokes a litany of diseases including Alport's disease, vestibular schwannoma, Refsum's syndrome, Paget's disease, otosclerosis, dominant photomyoclonus, and diabetes (Armbrecht, 1985; Corso, 1981; Goodhill and Harris 1979; Schuknecht, 1974).

Specifically, there may be four types of presbycusis: (1) Sensory which is associated with epithelial atrophy and degeneration of hair cells and the cells that support them in the cochlear basal coil. (2) Neural involves a loss of the neuronal population of the auditory pathways. (3) Strial combines deficiencies of the scala media with the biochemical and ionic properties of endolymphatic fluid. There may be an atrophy of the stria vascularis the function of which maintains a metabolic balance (Koerner, 1970). The constituents of the endolymphatic fluids may change through anabolic processes that damage epithelial tissue. (4) Conductive is the result of a generalized stiffening of the structures associated with cochlear partition vibration. Very seldom, if ever, does a categorical pure form of presbycusis occur alone.

The incidence of the four types of presbycusis is described in Table I-3 as documented from post-mortem temporal bone analysis of 160 ears (100 subjects). Strial presbycusis may be the most prevalent of all the types (Schuknecht, Watanuki, Takahashi, Belal, Kimura, Jones, & Ota, 1974).

Table I-3 Incidence of Four Types of Presbycusis

Type of Presbycusis	Ears	Individuals	Percent
Sensory	21	12	11.9
Neural	51	31	30.7
Strial	52	35	34.6
Inner ear conductance	36	23	22.8
Total	160	101	100.0

Source: Schuknecht, Watanuki, Takahashi, Belal, Kimura, Jones, & Ota, 1974. Atrophy of the stria vascularis, a common cause of hearing loss. Laryngoscope, 84, 1777-1821.

Obvious departures from normal middle ear characteristics include the accumulation of fluid within the middle ear cleft, usually associated with young children but not uncommon among the elderly population. Fluid within the middle ear space leads to conductive hearing losses and, in chronic cases, renders the tympanic membrane flaccid and less elastic. Arthritic transformations have an apparent link with advancing age.

Cartilage loses its translucency; while at the same time fewer cells are present in this rather amorphous tissue. As the rate of protein synthesis decreases, calcification of the matrix accelerates toward the formation of new bone. The most regressive component of cartilaginous aging may become evident within the fourth decade of life. The cartilage

matrix then becomes inefficient in the diffusion of waste products and cells die as an indirect result (Arking, 1991; Goodhill, 1979).

Specifically, calcification of the articular cartilage decreases the mechanical efficiency of the ossicular chain. A closing of the space between the incudomalleal and incudostapedial joints often leads to an obliteration of the articular spaces (Etholm & Belal, 1974; Honrubia & Goodhill, 1979). In like manner, the synovial membranes become less flexible with an overall stiffening of joints, manifested audiometrically in significant air-bone gaps (Goodhill). The loss in resiliency compounds degeneration and atrophies of ossicular muscles and ligaments. In severe instances, the pathologic condition of otosclerosis forms new bone in the cochlear capsule, immobilizing the footplate of the stapes in the oval window (Schuknecht et al., 1974).

At both the cellular and the tissue level, destructive or catabolic processes appear to affect the processing of sensory information associated with sensorineural hearing loss and speech discrimination deficits (Faingold, Gehlbach, & Caspary, 1991; Goodhill et al., 1979). In certain definite regions overt changes of cochlear structures, including the stria vascularis, sensory nerves, spiral ligament, the cochlear fluids, predominate.

According to Wright and Schuknecht (1972), the spiral ligament exhibits atrophy, that is consistent with advancing age. The severity of the atrophy is localized to the middle and the apical turns of the cochlea and consists of cystic acellular portions. These findings rely heavily on transmission and scanning electron microscopy and tissue mechanics. A few post mortem investigations of human cochlea, revealed vast discrepancies in the outer hair cell population. Among documented presbycusis patients such studies cannot distinguish between effects as a result of autolysis and those from aging (Gleeson and Felix, 1987). Cochlea investigation *in vivo* presents complications to data collection in relation to experimental artefact.

The classifications of presbycusis are open to interpretation, e.g., caution should be exercised with the sensory classification. Soucek, Michaels and Mason (1983) note that

the outer hair cells are most susceptible to damage. As such, progressive hearing loss associated with aging is sensory with the locus in the outer hair cells (Willot, 1991). The progression of damage in humans appears to course from the base to the apex of the cochlea (Johnsson & Hawkins, 1972) and substantiated by Schuknecht (1974). These findings have been supported among phylogenetically diverse models including Sprague-Dawley rats, an animal model used for many studies of aging.

Specifically, Feldman (1984) reported a complete atrophy of the organ of Corti and changes in the myelin in Sprague-Dawley rats (45-48 months). Bhattaharyya & Dayal (1989) found statistically significant differences between outer and inner hair cell damage from the rabbit cochlea. Interestingly, the damage was both in the apical and basal regions of the cochlear partition in rabbits ≥4 years which is not very old for a rabbit.

Several ethical issues arise should an investigation involve direct observation of the human cochlea *in vivo*. For this reason, alternative methods must be employed to characterize the auditory periphery. In particular, aging presents definite adverse changes that obscure our understanding. Thus, specific questions should be addressed.

Research Hypothesis

It may be far from obvious whether age is a significant factor that can affect oto-acoustic emission detection. There is a paucity of research that can argue specifically the effects of age, particularly with advancing age (40s, 50s, and 60s decades of human life). In most recent years, a few investigators have characterized a few emerging trends in emissions analysis related to aging, i.e., evoked oto-acoustic emission thresholds as a function of age (Collet, Gartner, Moulin, and Morgon, 1990) and emissions related to cochlear pathology (Probst et al., 1987). In practice, OAEs are evident among infants and young children (Kemp, 1978) and among individuals 21-42 years of age (Johnsen and Elberling, 1982; Probst et al., 1987; Norton et al., 1986). For the purpose of clarity, the CUPID criteria will be used (Arking, 1991).

An exhaustive review of the literature by the investigator revealed that there has been no specific investigation conducted on the effects of aging on evoked oto-acoustic emission from a human population in the 40s therefore, this investigation was designed to answer the following null hypotheses about evoked oto-acoustic emissions:

(i) The amplitude of the EOAE does not differ significantly with advancing age.

(ii) The frequency composition of the EOAE does not differ significantly as a function of advancing age.

(iii) The latency of the EOAE does not differ significantly with advancing age.

CHAPTER II

REVIEW OF LITERATURE

The cochlea is indeed the end organ of hearing, but hardly at the end of the auditory chain that links vibratory energy with the auditory sensation we term "hearing." Serially, the auditory periphery matches aerial vibrations and transduces mechanical energy into bioelectrical energy. Acoustic energy with frequency, intensity, and time information determines partially the pattern of displacement along the cochlear partition. The cochlea links a chain of mechanical and bioelectric events. Ultimately, the sensation of hearing emerges at the auditory cortex. For this reason, it is essential that one possess an understanding of the anatomy, physiology, and biophysics of the cochlea.

Anatomy and Physiology

A vast array of acoustic signals comprises the matrix of everyday life. Functionally, hearing is the first defense in survival and aids in the exchange of thoughts and ideas. To this extent, an understanding of hearing including the structures, systems, and processes germane to the auditory system is essential (Whitborne, 1985).

The external auditory meatus (EAM) extends from the epithelium of the tympanic membrane progressing laterally to the pinna. Cartilage overlaid by a thin layer of skin comprises the auditory periphery, pinna or auricle. The pinna of an elephant, for example, provides cooling. In lower animals, dogs, bats, and cats use the pinna for localization of sounds. In humans the pinnae fall short in localization under 4.0 kHz, However, above 4.0 kHz the pinnae enhance localization in the vertical plane (Durrant and Lovrinic, 1984). Through the EAM partial inelastic collisions of air molecules propagate energy to stimulate the cochlea. Regions of high and low particle density,

termed compressions or rarefactions, characterize the acoustic energy impinging onto the tympanic membrane (TM). Energy transmitted through the auditory ossicles (malleus, incus, stapes) follows the aerial motion in the EAM. The manubruium attachment to the TM guarantees phase unity with the cochlea. The anterior ligament of the malleus and the posterior ligament of the incus partially determine ossicular vibratory motion. Ligaments suspending the ossicles produce an axis of rotation is in the middle ear attic (Pickles, 1982).

The TM, coupled to muscular and ligamental attachments, produces mechanical impedance. The fluids of the cochlea represent a spring. As it turns out, both resistance and reactance vectorially combine to interact with the frequency of an acoustic stimulus the fluid against the footplate of the stapes. The mass and stiffness combine in a direct relationship to reactance (Wilbur, 1976).

The ossicles form a Class II lever action resulting from an applied force at the malleus. The ossicular arrangement optimizes energy transmission from air to fluid. Impedance in air yields 41.5 Ω and 5.6 kΩ for cochlear fluid constituents. An increase of applied force at the malleus offsets displacement at the footplate of the stapes. The lever system produces an excursion of the stapes toward the oval window, in a shorter distance than the malleus. The two ossicular motions occur in the same unit of time. Consequently, the volume velocity of the middle ear decreases with an increase of stapedial pressure (Wilbur, 1976; Glattke, 1973). Theoretically, pressure increases because of 17:1 ratio between the TM and the stapes footplate. The TM effective area spans 55 mm^2 to that of the 3.2 mm^2 stapes footplate (von Békésy, 1960).

Overall, there are three axiomatic factors influencing total impedance and impedance matching in respect to human hearing mechanism: (1) pressure increases as the impedance increases,(2) velocity increases as the impedance decreases, (3) a greater force at the malleus is offset by displacement at the stapes footplate (Durrant et al., 1984; Glattke, 1973, Goodhill and Honrubia, 1979; von Békésy, 1960).

Cochlear Mechanics

Our knowledge of the perplexities of cochlear structure and function steadily increased within the last 20 years. With the few, introduction of the traveling wave theory (von Békésy, 1960), prominent investigations stressed mechanical cochlea properties such as cochlear nonlinearity (Rhode, 1971, 1986; Dallos, 1973, 1981; DeBoer, 1980a, 1980b, Kemp and Brown, 1984), cochlear echoes (Kemp, 1978; Kemp and Brown, 1983; Kemp and Chum, 1980; Tonndorf, 1986), basilar membrane measurement (Yates and Johnstone, 1979; Selick, Patuzzi, and Johnstone, 1982; Khanna, 1986; 1984 Khanna and Leonard, 1986; Johnstone, Patuzzi & Yates 1986; LePage, 1987), outer hair cell motility (Flock, 1983, 1986; Strelioff and Flock, 1984; Ashmore, 1986; Brownell, 1986; Geisler, 1986; Lim, 1986; Zenner and Drenckhaln, 1986), and stereocilia-tectorial membrane interaction (Osborne, Comis, Chamberlain, & Sleplecky, 1986).

With the publication of these several investigators, it would appear that our understanding of cochlear micromechanics would be quite lucid. However, the vibratory pattern of the cochlea partition (to be addressed in detail later) has been, at best, an obscure problem of audition (von Békésy, 1960). In the context of problem solving, progress has been slow in determining the pattern(s) of cochlear response to various acoustic stimuli. Essentially, the mechanics of the cochlea *in vivo* limits our knowledge of auditory biophysics. Models of the cochlea support physical and/or quantitative constructs. It turns out that our knowledge of the cochlea is incomplete because of a paucity of research that uphold various theoretical assumptions (Dallos, 1973; Geisler, 1986; von Békésy, 1960; Johnstone et al., 1986; Kemp, 1980, 1986; Viergever, 1986; Zwislocki, 1986).

Invariably, cochleae from animals that had already died distinguished the experiments of von Békésy (1960). Typically, the postmortem procedures reconstructed

'near physiologic' conditions *in vivo*. The results of his experiments described the general vibratory activity of the basilar membrane or the cochlear partition. In simplification, the cochlea (a coiled structure) sometimes represents a straight canal encompassing an elastic sheet or a thin bar clamped at one end. On the contrary, neither the 'straight canal' nor the 'thin clamped bar' completely demonstrate this archetype (von Békésy, 1960; Dallos, 1973; Yost et al., 1985).

Empirical evidence shows that the stapes footplate moves into the oval window, thus producing a pressure wave or disturbance in the perilymph-filled vestibule of the scala vestibuli. Consequently, the wave meets a resistance due to the impedance of Reissner's membrane and the scalae. Disagreement remains among investigators concerning mass, frictional, and elastic characteristics of scalae and the membranes separating them (von Békésy, 1960; Dallos, 1973; Goodhill, 1979; Møller, 1983). Fibrous connective tissue comprises the basilar and Reissner's membrane. These two membranes line the wall of the cochlea, opposite the spiral lamina. The spiral ligament continues with the spiral lamina. An aggregate of supporting cells (simple cuboidal and pseudocuboidal), the tectorial membrane, and the hair cells comprise the receptor organ of hearing, namely, the organ of Corti that rests upon the basilar membrane (Glattke, 1973; Møller, 1983; Durrant et al., 1984; Lim, 1986; Rhode, 1971; Yost et al., 1985;).

The time-dependent fluids surrounding the basilar membrane generate internal friction. A component of traveling wave damping occurs because of fluid viscosity. Another partial of vibratory suppression results from the gradual variation of mass and stiffness along the basilar membrane. The predominant stiffness gradient along the basilar membrane, to some extent, generates tonotopic transformation. Specific localized regions along the cochlear partition separate duration, amplitude and phase information. As such, frequency specificity of the basilar membrane shifts with displacement at the apical region for low frequency and toward the basal region for high frequency

stimulation. The points of maximum stimulation change by approximately 4-5 mm for a unit change of an octave (von Békésy, 1960).

The variation in the width of the basilar membrane, from base to apex, causes an approximate 100:1 unit change in stiffness. The elasticity of the membrane itself does not change significantly along the continuum. The basilar membrane is stiffest and widest in the apical region. A peculiarity of the basilar membrane is that the stiffness changes are indirectly related to the cross-sectional area of the cochlea (Durrant et al., 1984; Pickles, 1982; Yates, 1979). The traveling wave progresses initially from the basal end of the scala media and then traverses the length of the cochlea to the apex (von Békésy, 1960). A paradoxical quality of the traveling wave is that the direction of the propagation will not change if the stapes was attached to the apical aspect of the cochlea. A wave phenomenon that progresses from the apex in the same manner as it would from the base is termed the traveling wave paradox (Yost & Nielsen, 1985). The pressure wave in the scala vestibuli causes a bulge of Reissner's membrane or vestibular membrane into the cochlear partition. Consequently the endolymph of this scala displaces in a direction away from the stapes. The energy transfer in the cochlear partition occurs about the surrounding scalae fluids (von Békésy, 1960). Employing cochlear models, von Békésy demonstrated that the mechanical properties of the cochlear partition determined exclusively characteristic vibratory patterns. The experiment simplified the calculation of hydrodynamic variables, i. e., viscosity. Within modeled media eddies commanded the basilar membrane vibratory pattern. Removal of fluid medium from canals of the human cochlea produced the minimum displacement possible. The movement that occurs along the partition changes proportionally varying frequency. In other words, the change in frequency (0.02-10 kHz) did not alter the vibratory pattern because the whole medium was vibrating in phase (von Békésy, 1960). A pure mathematical model of the cochlea involves time-variant information, i.e., phase and duration.

It should be noted that the stapes displacement is extremely small (about nanometers $\approx 10^{-9}$ m). The displacement decreases as a direct function of frequency when the sound pressure level (SPL) is held constant. Indeed, these exceedingly small displacements approach quantum mechanics. For these reasons measurement techniques are highly sensitive to vibratory cochlear activity that approaches Brownian movement.

The perilymphatic fluids mediate the transmission of mechano-acoustic energy from the stapes footplate. Fluid turbulence impedes some of the transmission. Amplitude-dependent eddies are often initiated rather abruptly. Stapedial force to the scala vestibuli overcomes fluid friction and changes the resting equilibrium (Tonndorf, 1986). Kiang (1986) postulated that removal of the coupling that presumably exists between the outer hair cell (OHC) stereocilia and the tectorial membrane and the reticular lamina would, most likely, allow a greater difference between the tectorial membrane and the reticular lamina. From these observations, it was suggested that greater stimulation of the inner hair cell (IHC) stereocilia might occur.

Almost invariably, cochlear partition displacement corresponds to an excursion of the stapes into the vestibule. One-dimensional fluid density rises instantaneously with the stapes forward progression (Dallos, 1973). As such, it may be tempting to liken this abrupt surge to a shock wave traversing the cochlear partition, however, true shock waves originate in the vacinity of sharp points or obstacles concerning a compressible fluid medium. At high velocities, the local compressions and rarefactions are not instantly reversible and are propagated out into the fluid medium as sound (in air). Essentially, the transport of momentum is converted subsequently into kinetic energy and heat. Fluid aerodynamics presents an indirect analog to hydrodynamics, e.g., high-velocity aerodynamic surfaces of approaching the speed of sound. To elaborate, the speed of sound in a hydra-fluid medium is nearly four times that of gaseous media. However, shock waves do not occur in the cochlear fluids. The transfer of energy within the cochlea is the same as the external auditory meatus; i.e., the partial inelastic collision of

air molecules. It would be more precise to consider the cochlea reacting to a shock stimulus rather than propagating a shock wave. The manipulation of two independent physical variables of the basilar membrane (those being stiffness and the coupling of adjacent parts) alters or produces various patterns. The stiffness of the stereocilia have been shown to provide damping in the guinea pig (Stelioff and Flock, 1984). The stiffness was shown to be inversely correlated with the length of the guinea pig steocilia bundles. These fundamental observations form the basis for modeling and support theoretical constructs, e.g., the traveling wave of hearing. The salient features of this theory make it desirable to show interrelationships with other theories or hypothetical constructs (von Békésy, 1960). Resonance underlies an alternate theory (reported by von Békésy) that assumes that the transverse fibers of the basilar membrane function as miniature resonators. According to theory, each resonator, vibrates to a discrete frequency. As a result, neural excitation occurs in the vacinity these resonators during basilar membrane deflection.

A major assumption of the resonance theory arises from individual resonators, embedded in fibrous connective tissue. Viscous fluids surround the resonators. However, the cellular matricies of these structures cannot be considered as true resonators. In addition, inherent damping characteristics prevent synchronous vibration within cochlear partition. The treatment of the cochlea as a single unit warrants a direct relationship to a common theme binding most theories of hearing. An assumption that mass, friction, and elasticity determine the vibratory pattern along the basilar membrane underpins the traveling wave theory. In another vein, the density of the basilar membrane would be difficult to extrapolate from the resistance of the scalae membrane, and the impedance, compliance, and cochlear fluid viscosity. Therefore, it is absolutely necessary, in some instances, to construct models that enable us to establish the nature of cochlear functional characteristics (von Békésy, 1960; Dallos, 1973, 1981; Sutton and Wilson, 1983; Zwislocki, 1986).

Several investigators of cochlear mechanics have not verified the importance of mass, friction, and elasticity (Dallos, 1973; DeBoer, 1980; Flock, 1983, 1986; Møller, 1983; Ashmore, 1986; Brownell, 1986; Geisler, 1986; LePage, 1987; Tonndorf, 1986; Viergever, 1986; von Békésy, 1960; Zenner et al., 1986). It may be far from obvious, but much of the disagreement has arisen from differences in experimental approaches and/or designs. Continuing to reason with a theme binding most theories of hearing, the methods of observing the cochlear vibratory activity (direct vs. indirect and invasive versus non invasive) have not been shared by all.

Direct Basilar Membrane Measurement

Basilar membrane vibratory patterns are almost infinitesimal in magnitude. The extremely small physical dimensions of the cochlea and its vibratory displacements truly approach quantum mechanics. As it turns out, the basilar membrane scala media, and the scala tympani are separated by approximately 100-200 μm. The basilar membrane is believed to perform the first significant tuning function of acoustic energy propagated from an aerial medium and transformed to a fluid medium. The amplitude of the basilar membrane vibratory motion is very small: (1.0 fm) at hearing threshold and almost (1.0 mm) at very intense levels (von Békésy, 1960; Dallos, 1973; Yates, 1979; Khanna, 1986; Rhode, 1986, 1971).

Laser Interferometry

The direct measurement of the basilar membrane can be a formidable undertaking, primarily because the technique must be sensitive to mechanical displacement above the noise of extraneous signals. In addition, the measurement must be physically small for localized measurements. In certain definite regions e.g., the basilar membrane, tectorial membrane the resolution of analysis should be for a wavelength matching the targeted structural motions. Difficult measurements arise in many instances. With these

considerations to measurement, our present technological capability is stretched to the limit. To be sure there are some ethical and practical reasons why most of what we know of cochlear mechanics has not been obtained from *in vivo* observations of human cochlea. That is, the techniques producing measurements influence potentially some trauma and generate artifacts (Dallos, 1973; Yates, 1979; Khanna, 1984, 1986; von Békésy, 1960). The available techniques of direct or invasive cochlea observation originated in the past research: (1) direct visualization (von Békésy, 1960), (2) Møssbauer technique (Johnstone and Boyle, 1967), (3) capacitive probe methods (LePage, 1973), (4) laser methods (Tonndorf, 1977).

In humans, the cochlea is encased within the petrosal portion of the temporal bone (the hardest bone in the body). Accessing the cochlea requires an arduous task of radical surgical intervention and indirect observation of the limits the gain in understanding. It is easy to see that various surgical approaches and or instruments heighten the probability of trauma to an extremely vulnerable cochlea. In practice, with rodents, this trauma may occur in many ways: (1) Drilling of the osseous wall of the cochlea produces injury to the organ of Corti, because of mechanical vibration, exposing the ear to acoustical noise and frictional heat generated by a surgical drill. Yet another reason for trauma arises from (2) forceps used in the opening of the bulla bone. As such, damage in this region can produce a loss in the cochlea action potential above 16 kHz. Fracture of the bulla bone appears to produce a lesser adverse effect than a fracture of the cochlear that houses the delicate organ of Corti. Notably, a transverse fracture of the temporal bone (in humans) involves the otic capsule and generally produces a profound sensorineural hearing loss (Goodhill, 1979). Specifically, the basal turns of the cochlea seem to be more susceptible to trauma than the apical turns. (3) The cooling of the cochlea produces a high-frequency hearing loss, primarily because of the prominence of the cochlear basal (first turn) aspect within the middle ear cleft, and (4) cauterization of blood

vessels in the round window membrane also produces a high frequency hearing loss (Yates, 1979; Khanna, 1984).

Speckle Stroboscopy

By stroboscopic illuminations, von Békésy (1960) visualized the cochlea directly. This crude approach incorporated an extensive surgery. The outcome of his experiments described the general vibratory patterns of the basilar membrane under various continuum positions (to be discussed later). Measurements were obtained at sound pressures (130-140 dB) well above normal physiologic levels. An intense yet cool stroboscopic light exposed microscopic vibrating cochlear structures. Placement of highly reflective specks (silver particles) enhanced the targeted area. The signal triggered once per cycle synchronized a stroboscopic beam. Illumination of the silver particles continued for a brief duration. The independent variable was the time delay of the triggering acoustic stimuli. From what subsequent researchers have found, the use of laser increases the accuracy of measurement. However, the available technology at the time of experimentation restricted von Békésy (Dallos, 1973; Yates, 1979; Khanna, 1984, 1986). Laser interferometry for basilar membrane measurements is in an emerging stage for cochlear research. The technique utilizes both light and sound stimulation, yet it is not essentially a laser technique. It provides the benefits of a spectrally pure, low-noise source. Interferometry yields accurate measurement of length, vibratory amplitude and selected physical properties of fluid mediums such as gases and liquids. A phase modulation technique varies wave fronts along a path between two points. A light source (reference) and detectors (object) are initiated by the basilar membrane *de facto* under observation.

The extreme sensitivity of the device detects extraneous vibrations (noise) from the motion of respiration and other physiologic activity. The procedure entails the placement of microscopic mirrors on the basilar membrane. The mirrors ($\approx 10^{-8}$ g) and an area of

($7x10^{-6}$ m^2) alter the vibratory response of the inner ear system through mass loading (Dallos, 1973; Yates, 1979; Khanna, 1984, 1986).

Møssbauer Technique

The Møssbauer technique is a form of a resonance spectroscopy that is active while the scala tympanic is filled with perilymph. It is a similar in idea to optical, electro-magnetic, or nuclear paramagnetic spectroscopies (Yates, 1979). Fundamental to the physics of characteristic particle acceleration is velocity, insofar that the reabsorbtion of radiation may be controlled by imposing a velocity difference between the source and the absorber. In practice, the nucleus of an atom that has been excited vibrates and radiates gamma rays, the nucleus loses energy, and the vibration amplitude decays. The duration is measured in half-life (the time in which the radioactivity associated with an isotope is reduced by half through decay). In the context to sympathetic and nuclear resonance, both the emitting and the absorbing nucleus must have the same energy levels. Shifts in wavelength through the Doppler effect coincide with changes in the gamma ray frequency (Dallos, 1973; Johnstone et al., 1986; Yates, 1979). Direct measurement and interpretation yield several potential problems. Specific obstacles accompanying the Møssbauer technique include (1) poor dynamic range, (2) lack of immediate feedback, (3) slow data acquisition, and (4) the handling of hazardous materials. The source may not be vibrating in sympathy with the basilar membrane. To this extent, the source may be mechanically affecting the vibratory medium and altering responses. Moreover, the radioactivity of the source causes damage in the vacinity of the hair cells. Thus, it would appear that trauma and artifacts are coupled with this measurement technique (Dallos, 1973; Johnstone et al., 1986; Sellick, Patuzzi, and Johnstone, 1982; Khanna, 1984; Yates, 1979).

As with other measuring techniques, artefacts and trauma may affect the interpretation of the results. There are two salient problems with the Møssbauer technique: (1) draining of perilymph produces sensitivity losses and frequency selectivity

relative to cochlear neurons in the sharply tuned tip region; and (2) under typical operating conditions, a strong radio frequency field applied to the organ of Corti may damage the tissue in proximity to the probe. If the fluid level rises to a point of contact with the probe, damage would be extensive due to an increase of current (Khanna, 1984).

From these observations clearly data cannot be retrieved from precise physiologic conditions. However, the imprecision does not eclipse the simplicity and the exact analog of the probe's sinusoidal output. The basilar membrane vibration output can be filtered and displayed directly on an oscilloscope (Dallos, 1973; Yates, 1979; Khanna, 1984, 1986).

Capacitive Probe Method

The capacitive probe method measures the electrical capacitance between the vibrating object (membrane or an earthed conductor) and the tip of a small probe conductor, usually 50-100 μm in diameter (Wilson, 1973). Near infintesimal variations of sensitivity can be detected through the Ohm meter frequency modulated circuitry. A small metal plate is positioned in the proximity of a vibrating object forming a capacitor. The capacitance changes with the varying distance of the capacitor. The inverse square law applies as the sensitivity decreases with an increase in distance. The oscillating frequency changes with capacitance (Dallos, 1973; Khanna, 1984; Yates, 1979). An interference-induced modulation from phase differences between the two wave fronts produces a resultant variable wave. This technique inherently produces cochlear damage.

Optical Heterodyne Spectroscopy

Optical heterodyne spectroscopy is the same as laser interferometry, yet it allows for an attenuation of the beam strength to match the poorer reflection from the vibrating target. Still another variation of laser interferometry is time-averaged and real-time holography that relies on a coherent laser wave front for operation. This technique results in an image on photographic film which reconstructs an image of a vibrating object with bands of light and dark. The tints and shades represent isometric contours of constant

amplitude of vibration. The approach is not capable of producing a direct analog representation of sinusoidal motion and is limited to a magnitude of greater than one wavelength. Generally, the laser group of measurement offers good sensitivity and precision, yet contingent of the depth of the perilymph. On the negative side, (1) it has not been determined whether mirrors will vibrate in sympathy with the basilar membrane; (2) the hardware to support the reflectors is cumbersome and heavy; (3) the procedure will not operate in perilymph because of the refractive characteristics of the fluid medium; (4) the surgery involved is quite extensive (Khanna, 1984, 1986; Yates, 1979).

Filtering Characteristics Of The Cochlea

The research of several investigators supports various theoretical constructs that suggest that the basilar membrane is the most critical component in filtering and sensory transduction (Dallos, 1973, 1981; Kemp, 1978; DeBoer, 1980; Møller, 1983; Johnstone, Patuzzi, & Yates, 1986; Kemp et al., 1984; Khanna, 1984; Khanna et al., 1986; Zwislocki, 1986). The basilar membrane is compared to a tunable resonator, inasmuch that the resonance frequency is different in specific locations along the basilar membrane. The membrane does not appear to have absolute spectral selectivity (Møller, 1983). However, knowledge of the exact values of stiffness, mass, and friction at certain loci is sufficient to predict the type of vibration a sound will initiate along the cochlear partition. By convention, the basilar membrane is considered sharply tuned and vulnerable to several injurious conditions (von Békésy, 1960; Dallos, 1973; Kim, 1980; Møller, 1983; Durrant et al., 1984; Kemp et al., 1984; Khanna, 1984; Johnstone et al., 1986; Kiang, Liberman, Newell, & Guinan, 1986).

Cochlear Linearity v. Nonlinearity

When sinusoidals, not present at the input are added to the output, a nonlinear system is produced. In other words, a nonlinear system alters or filters an input signal, changing it from a simple to complex waveform. Unlike a linear assemblage, a nonlinear system changes the input signal relative to the frequency and time domain. Specifically, time-dependent events in a nonlinear system produce distortion (Dallos, 1973; Durrant et al., 1984; Yost et al., 1985). An argument for linearity in the cochlea was based on observations that did not show a unit change in the vibratory pattern of the cochlear partition, even with a stimulus that was depressed to half its amplitude (von Békésy, 1960). However, Rhode (1971) discovered that the nonlinearity of the cochlear diminishes during anoxia or after death (Pickles, 1982). Clearly, post-mortem observations led to the rather "linear" conclusions of von Békésy.

There are many nonlinearities occurring within the cochlea that produce harmonic and intermodulation distortion. Albeit, there are two main types of nonlinearity in the auditory periphery: (1) one that causes nonlinear distortion and (2) another that is slow to produce nonlinear distortion products. A nonlinearity exists when the output is not proportional to the input (Møller, 1983).

Hydrodynamic nonlinearities are associated with: (1) Amplitude-dependent events such as eddies that produce harmonic distortion, and (2) Amplitude-dependent events that produce asymmetrical basilar membrane displacements. The latter of the two involves a subsequent envelope detection in the form of beats (Tonndorf, 1986). It turns out that a significant number of these nonlinearities are frequency dependent. A common characteristic of a nonlinear system, when driven by a sinusoidal force, is the capacity to generate new frequency components. The change in the resultant wave form is a function of nonlinearity (hydrodynamic, mechanical) properties within the cochlea (Dallos, 1973).

The interaction of a nonlinear system and the wave forms consists of one frequency f1 and a complex wave form consisting of two frequencies f1 and f2 thus, if f1 is an input to a nonlinear device, then the output contains f1 and is associated higher harmonics: 2f1, 3f1, 4f1, etc. (Yost et al., 1985).

There are instances when both f1 and f2 are inputs to the ear. The dual input of f1 and f1 and f2 leads to a phenomena termed "distortion product," or "combination tones" of several main types, e.g. (a) cubic difference tones, (b) harmonic tones, (c) difference tones, and (d) summation tones (Durrant et al., 1984; Pickles, 1982; Yost et al., 1985).

The terms 'distortion product' and 'cubic difference tone' are used interchangeably. There are two main problems with the exchange in respect to cubic difference tones: (1) 2f1-f2 can be generated by many different types of nonlinearity minus an odd order non-linearity; (2) a cubic distortion nonlinearity generates 2f1-f2 as well as 2f2-f1, the both of which may be nonspecific. Finally, a distortion product refers to a physical or physio-logical entity whereas a combination tone refers to a perceptual entity (Kim, 1980).

Distortion products appear to be 'cochleogenic' (Kemp, 1986). A cochlea origin for distortion products has been suggested by several investigators whereby a nondistorted stimulus was used to describe the nonlinear behavior of the auditory system. Fundamental observations of distortion non-specificity underscore the inherent difficulty that rises from separating distortion components within the auditory system (Brown and Kemp, 1984; Burns, Strickland, Tubis, Jones, 1984; DeBoer, 1980a; Kemp, 1986 1978; Kemp et al., 1983, 1984; Kim, 1980;).

In an investigation of the mechanical and/physiological correlates of nonlinearity in adults cats and chinchillas, Kim (1980) found that neither afferent nor efferent neural activity is required for the generation of 2f1-f2. Distortion products were vulnerable to anoxia and potassium cyanide KCN and disappeared with (N1 and N2) after perfusion. The physiologically vulnerable processes of the Organ of Corti probably affect the macromechanics of the cochlear partition and the middle ear (Kim, 1980).

As a result of overstimulation, the mechanical properties of the hair cells may produce changes that generate difference tones. The nonlinearity seems to be either hair cell-dependent or generated from definite structures, i.e., supporting cells, spiral ligament. The primaries f1-f2 are found in these regions (Møller, 1983). Experimental data derived from physical models did not support the premise that fluid eddies appear in real ears (Tonndorf, 1986). His models have shown that high friction produced within much narrower cochlear scalae ruled out angular perturbations as an explanation for hydro-nonlinearity. Thus, it appeared that a relatively higher sound pressure level is required to generate eddies in real ears than in enlarged models. In this sense, the displacement has been shown to vary at different sound pressure levels. Larger displacements occurred toward the scala tympani than toward those of the scala vestibuli.

Zwislocki (1986) designed experiments to determine whether the tectorial membrane mass, together with its elastic attachments to the spiral limbus and the organ of Corti, comprise a mechanical resonance system. The results of his investigation revealed the changes in cochlear frequency selectivity. In a similar manner to the resonance model, reported by von Békésy (1960), Tonndorf found that the incompressibility of the fluid dominated the transverse motion of the tectorial membrane. The reticular lamina was indirectly related to the basilar membrane motion. The hydromechanical coupling was enhanced by a minimal separation of the tectorial membrane and the reticular lamina of the organ of Corti. The transverse motion caused a resonating tectorial membrane to move orthogonally to the motion of the basilar membrane. Torque forces produced an energy transfer.

Up to this point, the basilar membrane has been discussed as a passive filter immersed in an incompressible fluid medium. The roughly triangular scala media has variable geometry with a conservation of volume and mass (Gold, 1948; Dallos, 1973, 1981; DeBoer, 1980; Møller, 1983; Khanna, 1984, Khanna et al., 1986; Durrant et al.,

1984; Johnstone et al., 1986; Kemp et al., 1984; Lim, 1986; Zwislocki, 1988; Zwislocki et al., 1986).

Cochlear Micromechanics

Micromechanical activity within the cochlear partition is at the level of the hair cell and the sensory cilia on the superior aspect of the hair cells (Dallos, 1973). The mammalian auditory sensory organs comprise a dual system (inner hair cells and outer hair cells) with different cellular organization and innervation patterns. In humans, there is a single row of approximately 3500 flask-shaped inner hair cells (IHCs). The IHCs array the entire length of the organ of Corti. On the average, there are 13,400 cylindrical outer hair cells (OHCs). Three-to-five rows of OHC parallel the IHC (Pickles, 1982; Durrant, 1984; Lim, 1986). The specific cellular mechanical process of sensory transduction is not clearly understood. However, it is generally agreed that the bending of the stereobundle (bundle of stereocilia) in an excitatory direction triggers the release of neurotransmitters at the afferent nerve ending region (Dallos, 1973; DeBoer, 1980b; Johnstone et al., 1986; Osborne et al., 1988; Kemp, 1978; Kemp et al., 1984; Nielsen and Slepecky, 1986; Lim, 1986).

The IHC are located along the osseous spiral lamina and have limited motion, in contrast to the strategic location of the OHCs which are situated on the basilar membrane. Generally, the OHCs are considered to be mechanically active and the IHCs are passive transducers. This dichotomy is open to argument. The inner and outer pillar cells, which border the tunnel of Corti provide a rigid support in a radial direction, in contrast to Dieter cells, the support of which is in a longitudinal direction (Ashmore, 1986; Brownell, 1986; Dallos, 1981; Durrant et al., 1984; Flock, 1983, 1986; Geisler, 1986; Lim, 1986; Zenner et al., 1986).

There has been a general consensus that the hair cells, particularly in the apical region, cannot be considered as passive transducers (Ashmore, 1986; Brownell, 1986;

Flock, 1986, 1983; Zenner et al., 1986). The contractile proteins of actin and myosin, within the crenulated hair cells, may be responsible for motile capability. The hair cells have been observed to elongate and shorten as a result of experiments, wherein pharmacological and electrical treatments (Mountain, 1986) have been used to induce irritability. Motility of isolated OHCs from intracellular ionic injections, transcellular, and extracellular stimulation has produced both rapid and slow responses (Brownell, 1986; Kemp and Souter, 1988).

There is significant evidence which directs emphasis to the OHCs as cellular elements involved in the active hearing correlates of nonlinearity in adult cats and chinchillas. Neither afferent nor efferent neural activity is required for the generation of 2f1-f2. However, Collet et al. (1990) implicated descending neuronal control for the active micromechanical properties of the cochlea. Distortion products were vulnerable to anoxia mediated by potassium cyanide (KCN) and disappeared with (N1 and N2) after profusion into the cochlear partition (Kim, 1980).

Motility has been observed with OHCs isolated from the organ of Corti (Geisler, 1986; Zenner et al., 1986). It is all very well to consider the motile properties of the OHCs *in vivo* as actively involved in cochlear mechanics; but that case is unlikely. It turns out that the forces generated must be sufficiently large and generated at a high rate to control macroscopic mechanics. These changes would produce impedances along the basilar membrane (Ashmore, 1986; Tonndorf, 1986; Zwislocki, et al., 1988).

The laminated cisternal system is unique to the cochlear hair cells found along the length of the OHCs. Motility of the OHCs is evidenced through pharmacological manipulation that modifies the cisternae, possibly affecting oto-acoustic emissions (to be addressed later). In some instances electromiosis may be active. Electromiosis is a thermodynamic phenomenon, similar to streaming potentials. To this extent, an electrical field induced by movement of fluid adjacent to a charged surface results in an interaction of electrophoretic counterions. These ions are held near or next to the charge of the

surface. The electrical field is parallel to the plane membrane rather than a perpendicular orientation. This is comparable to a field potential with inherent repulsive and attractive field effects (Ashmore, 1986).

Several features of the OHC electro-mechanical response are difficult to resolve with the data that are available on the contractile mechanisms. The viscosity of the cisternal fluid (the substance of which is not known) may be much too great to allow the rapid transport that would be essential to causing changes toward the stapes. The primary limiting factor would be a boundary layer condition with frictional characteristics, probably 200 times larger than the actual viscosity of the fluid (Ashmore, 1986; Brownell, 1986; Lim, 1986).

Active Cochlear Feedback

Gold (1948) postulated that the filtering action of the cochlea depends on a biochemical feedback process within the cochlea, an activity that exists in a cochlear microphonic electrical field. The theory suggests a targeted field that contains sufficient energy (more than a passive filter) to reconvert energy into efficiency approximating unity. In addition, the principle function as a dynamo, is an active mechanism where an applied signal releases a chain of events involving an additional supply of energy.

The regeneration hypothesis (Gold, 1948) offers an explanation to possible electromechanical activity. Regeneration occurs as a result of the cochlear microphonic effect and the audibility of electrical signals. An assumption proposes that a feedback channel exists between oscillating fibers of the basilar membrane producing an electrical field.

Several hair cell mechanisms may be capable of macromechanical changes (in the ear canal) related to electro-mechanical transduction: (1) ionic mechanisms, which appear to be similar to those of the photoreceptor in the eye, sensitive to mechanical strain (Lim, 1986, Osborne et al., 1988). Strain produces angular deformation without a change in the

volume of a given structure. In this instance, the applied force leads to deformation in certain definite regions that gives rise to hyperpolarization and depolarization. (2) Mechanical transduction results from the linkage of specific hair cell components (stereocilia) and their attachment to the actin filaments. The transduction channels are within the region of the stereocilia (Flock, 1983, 1986; Nielsen & Slepecky, 1986; Zenner et al., 1986). (3) Synaptic mechanisms act at the base of the cell to govern the release of transmitter substances at the afferent synapse. Excitation of efferent fibers may interfere with or modulate the mechanics of the hair cell system. These hair cell mechanisms may be the dominant active processes for receptors with motor capacity (Flock, 1983, 1986).

The effect of the OHC motility on cochlear vibration depends on a hypothesis that the coupling of hair cells may account for the discrete frequency-selectivity of the basilar membrane. Constructing a model, Geisler (1986) utilized OHC deformation forces to possibly justify a variety of observed phenomena ranging from improved frequency selectivity of the cochlear partition to electrically induced sounds in the external ear canal. In an effort to monitor cochlear condition during interferometric methods, Khanna and Leonard (1986) found that the basilar membrane response relates to the condition of OHCs through the rigid coupling to the basilar membrane. A strong correlation exists between OHC condition and the basilar membrane response This association suggests that the mechanics of the OHCs may have an active role in determining the mechanical response of the basilar membrane.

The essence of mechanical wave motion is an oscillation or a disturbance traveling through a medium (solids, gases) without transporting matter with the energy. When simple mechanical or hydromechanical systems are joined, a disturbance in one component causes a change in other components. Here again, the treatment of the cochlear partition as one unit is warranted based on observations from the experiments of von Békésy (1960). It appears that the basilar and Reissner's membranes vibrate in phase

at frequencies below 3.0 kHz (von Békésy, 1960). Within such a frequency range, the cochlear partition could be treated as single elastic layer (Dallos, 1973; Møller, 1983).

Although the cochlear partition may be associated with a single stable mechanical system, the salient feature of the cochlear dynamics is a combination of mechanics and hydrodynamics. As such, the effect of fluid disturbance by virtue of the stapes is twofold: (1) There is an alteration in the resting equilibrium pressure of the fluid by exerting a force in one direction, a situation which causes (2) a change in the equilibrium density of the fluid medium. As a result, no mass of fluid can be lost and no mass can be generated, i.e., mass is conserved. A mechanical wave propagates energy from the base to the apex through a medium bound to an equilibrium position by mass, elasticity and a viscous fluid. The inherent variable geometry of the surrounding scalae appears to be a negligible factor because of allowable mechanical motion (Dallos, 1973; Yost et al., 1985).

Oto-Acoustic Emissions

In 1978, D. T. Kemp bridged a thirty-year span of auditory research when he linked his discovery of narrowband emissions with the work of Gold (1948), who postulated that there was an active process which provided feedback to modulate the traveling wave (von Békésy, 1960). Sealing both a subminiature loudspeaker and a microphone into the external auditory meatus, Kemp (1978) first reported the existence of evoked and spontaneous emissions in the ear canal after a delay of 5-15 ms.

The source of sensory excitation appears to be on the basilar membrane and extends over a region of mechanical impedance gradients. In the context of human cochlear mechanics, impedance discontinuities arise possibly because of a mechanical response of the transduction mechanism. Stimulation leads to a traveling wave reflection (Kemp, 1978). It would appear that OAEs from modeling represent a leakage of energy from a functional forward traveling wave due to some mechanical perturbation (Kemp, 1978,

1986; Rutten, 1980). The source of the OAE may be closely associated with the mechanism responsible for cochlear frequency selectivity (Kemp & Chum, 1980). The source may emanate from within specific frequency bandwidths (Neely et al., 1988; Sutton & Wilson, 1983). Long latencies appear to be related to discontinuities along the vibratory medium.

The existence of some spontaneous oto-acoustic emissions (SOAEs) appear to emerge from active nonlinear feedback forces within the cochlea. These forces appear to be influenced by physiological vulnerability of emissions to ototoxic drugs and frequency locking of SOAEs by the statistical properties of the ear canal. In the context of quantum theory, the statistical mechanics of the auditory periphery may contribute to a sum total of individual molecular energies. As it turns out, these may be energies of vibration such as rotation and translation. In addition, influences arise form incremental pressure in a frequency band containing a prominent SOAE and by external tones (Jones, Tubis, Long, Burns, and Strickland, 1985; Kruger, 1986). The results of suppression tuning properties of the OAE distortion product 2f1-f2, measured in the ear canal of gerbil and man, have shown frequency-dependent characteristics (Brown et al., 1984; DeBoer, 1980b, Kim, 1980; Rabinowitz and Widin, 1984; Schloth, 1983; Zurek, 1985).

These pressure waves, resolved through computer averaging techniques, have been referred to as evoked cochlear mechanical responses (EMCR) (Kemp, 1981), evoked oto-acoustic emissions (EOAE) (Kemp, 1978), Kemp echoes, (DeBoer, 1980a), or cochlear echoes (Kemp, 1978). Emissions, believed to be the result of distortion within the cochlea (distortion products), have also been observed (Moore et al., 1984).

Several investigators subsequent to the initial research have also suggested that a retrograde, cochleogenic traveling wave may be capable of producing macromechanic changes in the middle ear, producing a brief wave of pressure in the ear canal. The research in this area has encompassed a wide variety of areas: effects of aspirin (Anderson and Kemp, 1979), recordings from normal adults (Elberling and Johnsen,

1985), relationship to audiometric fine structure (Horst, Wit, & Ritsma, 1983; Sutton and Wilson, 1983), recordings from neonates using auditory brainstem response to assess EOAE., (Webb, Hutchingson, Connell, Smith, and Buffin, 1990), EOAE alone (Johnsen and Elberling, 1982), in relationship to cochlear damage (Kemp et al., 1980), latency of emissions (Norton, Chaplin, & Mott, 1986), and the electrical correlates or distortion products (Kemp et al., 1984; Mountain, 1986).

Cochlear Modeling (as related to oto-acoustic emissions)

Modeling can quantify physically and account for many observable features of stimulus-frequency acoustic emissions in the ear canal. The occurrences include multiple echoes and resonance (Kemp et al., (1980). As stated in the section on basilar membrane filtering, specific regions of sharp tuning explain partially variations of nonlinear feedback. These regions originate from physiological events related to cochlear macro-mechanics (DeBoer, 1980a, 1980b; Kemp, 1986; Kemp et al., 1980). A reverse traveling wave, resulting from impedance discontinuities along the basilar membrane (Kemp, 1978), is derived from one of two models that explain the generation of cochlear echoes. The other major model (Wilson, 1980) suggests that a synchronous swelling and shrinking of hair cells, possibly trailing a second filter with increased travel time and limited maximum amplitude, may be the generator. These observations are based on irregularities seen in frequency mapping of the cochlea. The purpose of mapping serves to determine the summed electrical activity of cochlear mechanics. A dichotomy exists relative to the origin of a feedback process and the specific form of its interaction with cochlear mechanics. The comparison may be related to (1) a compressional wave of infinite speed or propagation and thus giving rise to the same sound pressure throughout the medium and/or (2) a traveling wave with propagation amplitudes related to resonance cutoff phenomena (DeBoer, 1980b). In this case, the second wave is stimulated by the first. Feedback coincides with OHC motility or to an electro-physiologic active process. The interaction with pressure or changes in the volume velocity of the middle ear

transmits as disturbances in the fluid constituents of the cochlea (Wilbur, 1976). A resultant reverse traveling wave, at the termination of the cochlear partition, may be a primary emission source disturbance (DeBoer, 1980a; Kemp et al., 1980; Kemp, 1978).

The transmission of the compressional wave transpires virtually as an instantaneous event. In this circumstance, one could observe a reflected wave (echo) with a post-onset time equal to the total traveling time of the primary wave. This assumption, however, contradicts the observations of Kemp (1978,1980). Recall that a compressional wave generates equivalent levels of sound pressure throughout an elastic medium (DeBoer, 1980b). Amplification occurs, conceivably, in a retrograde transmission of total traveling wave energy, increasing as it progresses over any region (DeBoer, 1980; Kemp et al., 1980; Kemp, 1978). The OAE response is, arguably, an elusive response and may related to an anatomical defect(s) yielding abnormal propagation characteristics. Logically, such abnormalities concur with prolongation of traveling wave excursion time.

Incidence

All emissions produced in normal ears are limited to small levels and very rarely exceed 20 dB SPL. The sound pressure of EOAEs grows proportionately to the stimulating sound pressure amplitude as long as the stimulus does not exceed approximately 20 dB SPL. Evidence points to the cochlea in respect to the mechanical response of the EOAE. The EOAE is not found in ears with hearing loses exceeding a mild severity (>30 dB HL). Kemp (1978) reported emissions in all 37 of the subjects that he used in the initial oto-acoustic emission study. However, subsequent investigations have fallen short of the original study and have not been able to match the perfect success of Kemp: 92%-100% among adults and children (Kemp, Bray, and Brown, 1986; Stevens, 1987), 75% in 8 ears (Horst, Wit, and Ritsma, 1983), inconclusive evidence between gender and age (Strickland et al., 1985), ≤ 95% for emissions in aging subjects (Collet, Gartner, Moulin, and Morgon, 1990), not readily observed in individuals with sensorineural hearing loss (Rutten, 1980; Tanaka, 1988), with minimal intraaural differences of ≤10 dB (Tanaka,

O-Uchi, Shimada, and Koseki, 1988). As it turns out, Spontaneous oto-acoustic emissions are very common in about 33% of all people tested although the emissions are not usually noticed subjectively, (Kemp, 1978) ≤60% among the hearing impaired, (Bonfils & Uziel, 1989). They are continuous as long as external sounds do not suppress them, with most found between 1 and 2 kHz and as high as 8.0 kHz (Brown, 1988; Brown et al., 1984; Kemp, 1978; Rabinowitz & Widen, 1984). Their levels are ≤20 dB SPL within an occluded ear canal. Kemp et al. (1986) reported that a hermetic seal is essential for recordings ≤400 Hz. There are instances when suprathreshold measurements above 30 dB HL lead to a lack of response for low levels (Kemp, 1978; Zurek, 1985; Zwicker, 1986; Zwicker, et al., 1984).

As it turns out, outer hair cell motility has been implicated as a modulator of emissions thus, affecting the frequency of occurrence (Bonfils et al., 1988). In an alternative procedure, distortion product emissions in humans with thresholds of ≤20 dB have been recorded (Lonsbury-Martin, Harris, Hawkins, Stagner, and Martin, 1990).

Overall, Willot (1991) concludes that oto-acoustic emissions and the behavioral audiometric thresholds co-vary with cochlear damage. Whether outer hair cell damage and subsequent dysfunction will adversely affect the emissions is disputed (Bonfils et al., 1988b; Collet et al., 1990). Taken together, the oto-acoustic emission is probably not a suitable procedure to include in the basic test battery except for those measures near threshold. Interactions between the fluid, tissues, and structures cannot be attributed to the variations in oto-acoustic emissions.

Factors Affecting OAEs

Empirical evidence indicates that OAEs vary (in some instances) accompanying noise exposure (Kemp et al., 1986; Norton et al., 1986), ototoxic drugs (Anderson et al., 1979), temperature (Whitehead et al., 1986), cochlear hearing loss (Probst and Hauser, 1990; Tanaka, 1988; Tanaka et al., 1988b), and posture (Schloth et al., 1983). Acoustic trauma

cochlear dysfunction and presbycusis were categorically analyzed in young adults. The results suggested that the EOAE was not frequency-specific.

Continuing to reason in terms of cochlear trauma, various ototoxic agents specifically kanamycin, remove OHC and their stereocilia, but olivocochlear bundle stimulation only changes some of the OHC mechanical properties. Intravenous furosemide injections produce a decrease in the endocohlear potential (Kiang, 1986). Similar changes of the cochlear microphonic are seen with KCN profusion (Kim, 1980). It is assumed that aspirin consumption weakens the biomechanical cochlear feedback amplification the effect of which may be responsible for the sharpening of the cochlear mechanical and neural responses. The OAE is obliterated by agents such as aspirin (Tubis et al., 1987) but is not affected by posture (Wilson, 1980) or temperature (Whitehead, Wilson, and Baker, 1986).

In recent years, the effects of age have emanated from the research (Strickland et al., 1985; Probst, 1987; Bonfils et al., 1988a), in gerbils after birth (Norton, Bargones, and Rubel, 1991). The incidence of SOAE s has been investigated with the use of some adult subjects (Burns et al., 1984; Johnsen et al., 1982; Kemp, 1981). Burns et al. (1985) postulated that if SOAEs are associated with OHC damage, the incidence might be expected to correlate with age. By convention, it is believed that degeneration of OHCs may occur soon after birth, with a continual progression throughout the aging process. A logical supposition was that if SOAEs were associated with normal localized degeneration of hair cells, their incidence would decrease with age. Strickland et al. (1985) found no significant difference in the number of emissions per subject between infant, children, and adult populations samples. Overwhelmingly, there was an incompatibility between the incidence of SOAEs and age.

Cochlear Fluid Viscosity

Ionic changes may affect the viscosity of the fluid constituents of the cochlea (von Békésy, 1960; Dallos, 1973; Salt & Konishi, 1986). An inverse relationship existing between viscosity and stiffness with advancing age may mathematically cancel each other relative to their effect on cochlear vibratory motion. Specifically, the micromechanical level is unknown. In like manner, the viscosity of the fluid within the subcisternae is not well understood.

Tinnitus

Tinnitus emerges as one of the most salient symptoms associated with an adult sensorineural hearing loss (Berlin, 1981; Goodhill, 1979; Hazell, 1981). Episodes of tinnitus may be an indication of a number of disorders, that makes it baffling to both the sufferer and the health professional (usually a physician) providing the treatment. Research points to an origin of tinnitus in the cochlea, principally because of an emerging understanding of oto-acoustic emissions (Kemp, 1978, 1981; Kemp et al., 1980; Wilson, 1980; Wilson et al., 1981, Ashmore, 1986; Brownell, 1986). There are two popular theories, that attempt to explain the origin of tinnitus: (1) Abnormal electro-chemical conditions (possibly yielding field potentials the force of which may affect mechanical properties) of the actin filaments within individual hair cells leading to an abnormal spontaneous firing of the associated auditory nerve. The 'misfiring' may arise at the level of the transduction site. (2) The organ of Corti contains elements (motile OHCs) which are believed to have the capacity to generate an audible frequency of vibration. In a similar manner, other micromechanical forces from within the cochlea may be relevant to tinnitus. However, more research needs to be done before we can consider that the OAE is synonymous with tinnitus (Kemp, 1978, 1981; Flock, 1983, 1986, Mountain, 1986).

CHAPTER III

INSTRUMENTATION AND PROCEDURE

The evoked oto-acoustic emission (EOAE) instrumentation system comprises three groups of components: (1) stimulus generation, (2) electrophysiological recording, and (3) stimulus-response monitoring. Figure III-1 presents a schematic representation of the EOAE system. Primarily, the instrumentation is part of a microcomputer-based system for auditory evoked potentials. The system is adaptable for the generation of various stimuli in addition to the recording of neuro-audiologic and neurophysiological data. Specifically, oto-acoustic emissions, compound action potentials, and auditory brainstem response data can be recorded, displayed and analyzed (Moore, Rakerd, Robb, Semela, and Hooks, 1989). In particular, the assemblage of components was re-configured slightly for this investigation shown in Table III-1, III-2, and III-3.

Table III-1. Individual components of signal generation

Component	Trade Name
Function generator	(Mi^2 208)
Dual attenuator	(Mi^2 108)
DC amplifier	(Technics® SE-9060)
Data timer	(Mi^2 214)
Subminiature receiver	(Knowles Electronics B6295-K1592)

Table III-2. Individual components of signal recording

Component	Trade Name
Data Amplifier	(Data Inc. 2124 Model 2)
Analog/Digital Converter	(Mi^2 202)
Memory buffer	(Mi^2 210)
Input/Output	(Mi^2 100)
Subminiature microphone	Knowles Electronics Model 1954

Table III-3. Individual components of signal monitoring

Component	Trade Name
Oscilloscope	(Tektronix D15)
Frequency counter	(Hewlett Packard 5314K)
Acoustic monitor (2)	
(a) stimulus	(Sentry 100A studio monitor)
(b) response	(Ampex AA620)
Multimeter	(Hewlett Packard 3466A)
Spectrum analyzer	(Hewlett Packard 3582A)
Computer	(IBM PC AT)
Printer	(IBM Proprinter II)
Plotter	(IBM 6180)

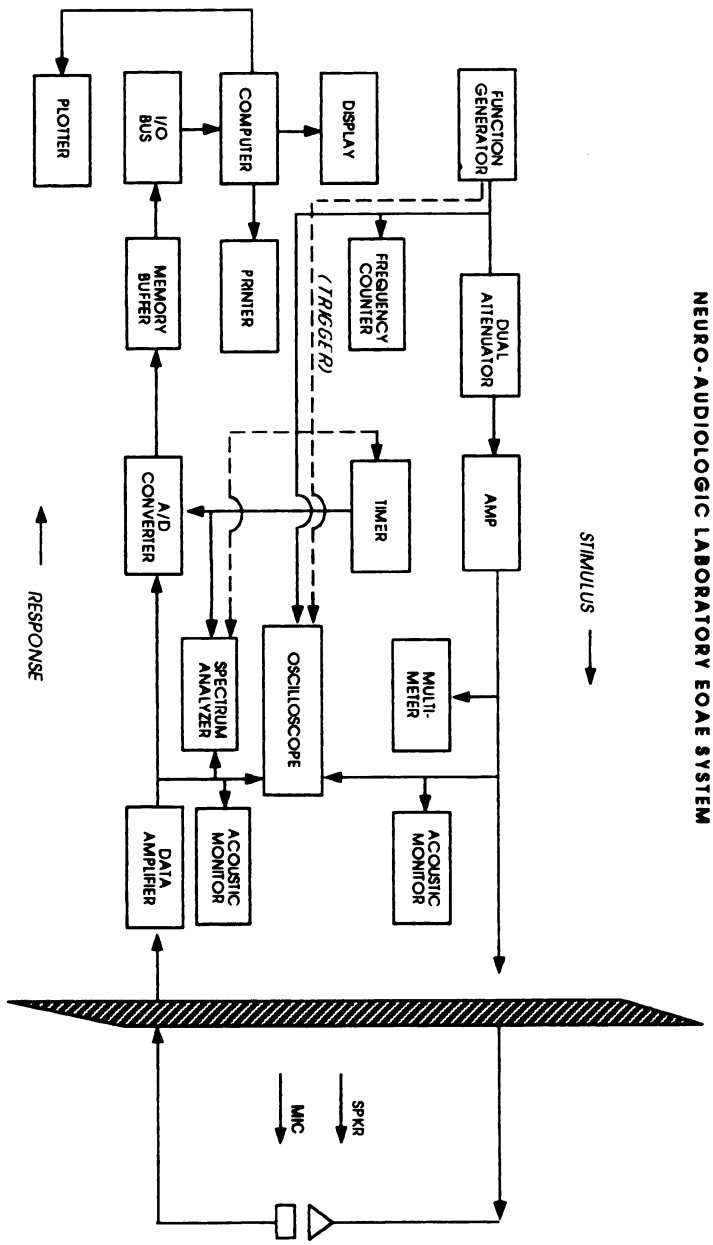


Figure II-1. Instrumentation

Emission Probe Assembly

The emission probe assembly (EPA) used in this study was derived from several previous prototypes developed in the Neuro-audiologic Lab of Michigan State University. The exemplar of the emission probe assembly (EPA-1) was devised with two sub-miniature transducers (Knowles BJ-159 microphone and a 1955 receiver), both of which were housed in a modified tympanic ear canal probe (Madsen, 2250). This kind of arrangement required probe tubing to the microphone to permit the retrieval of the emissions. The receiver delivered a filtered click to the ear via the cylindrical inlet to the ear canal.

The next two designs of the sensory device incorporated a Brüel & Kjaer (B& K 4179) low-noise microphone coupled to PEtubing via a 2 cc adapter. A section of PE tubing directed the stimulating signal, from a subminiature speaker to the ear. A separate section of tubing routed a signal from a microphone to the ear. Both tubes fit a tympanometric ear tip for an occlusive but not hermetic seal. The tubing design was similar to the instrumentation of Lonsbury-Martin et al., (1990). The configuration of the microphone introduced proximity effects (to be discussed later), the outcome of which led to the development of the subsequent EPA devices.

The fourth in a series (EPA-4) of assemblies utilized two transducers similar to the insert microphones, the design of which has been used by Etymotic® Research, Inc. The microphones were designed to be inserted comfortably into the ear canal, thus eliminating the need for tubing. The EPA-4 comprised two subminiature transducers (Knowles EA 1954 electret microphone and a Knowles BG295K 1952 receiver). In the context of experimental precision, the microphone sensitivity was the more critical of the two transducers. The retrieval of emissions from the ear canal is optimal near the threshold of

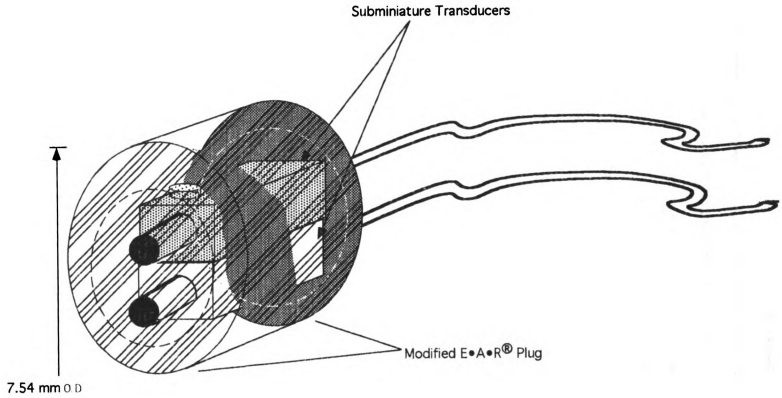


Figure III-2. EMISSION PROBE ASSEMBLY-4

hearing sensitivity. For this reason, microphone noise may have been a concern since extraneous energy can contaminate EOAE data.

As it turns out, the microphone component generates energy with a frequency response spectrum that is essentially flat to 6 kHz with a nominal output impedance of 3.5 kOhm. It has also a low sensitivity to vibration, a necessary characteristic for emission analysis. The microphone produced a noise floor of -20 dB SPL at 1.0 kHz and -26 dB at 5.0 kHz. It was noted that modification of the microphone casing has been shown to increase the sensitivity by 2-3 dB. The modification entailed a port (vent) perpendicular to the longitudinal plane of the transducer (see Figure III.2.). Specifically, the volumetric dimensions behind the diaphragm have been increased in some cases to gain acoustic efficiency, an observation supported by Knowles Electronics technical data (1985). Alternatively, they suggested that summing four independent microphones in conjunction with an algorithmic method increased the signal-to-noise ratio through an increase in the averaging or analysis of the data.

A modified Cabot Corporation E•A•R® plug housed both transducers (Model PL101) as shown in Figure. III-2. Modification of the plug embodied a bore centered through the longitudinal plane. The consequent volumetric dimensions were as follows: (a) internal diameter (I.D.) = 7.54 mm; (b) outside diameter (O.D.) = 14 mm.

The I.D. of the plug was an optimal design for several reasons: (1) securing the juxaposition microphone/receiver arrangement, thus diminishing the potential for dynamic friction; (2) providing comfort to the subject(s) during the experimental sequence and data collection; and (3) yielding an occlusive seal the function of which was similar to the configuration described in earlier EOAE studies (Kemp, 1978; Kemp and Brown, 1983). In particular, EPA-4 negated the coupling of P E tubing to the transducers. Empirically, the use of such tubing has produced damping factors and "proximity effects," the outcome of which has been shown to suppress SPL when coupled to hearing aids (Burkhard and Sachs, 1977).

Signal Generation

Type of Stimuli

A tone burst was used to elicit the OAE response from the cochlea. The signal can be characterized as a sine tone, amplitude modulated by another sine tone. Mathematically, the signal is a variation of a cosine window represented below:

$$A_0 sin\left(\pi \frac{\tau^0}{\tau_0}\right) sin\left(\chi 2\pi \frac{\tau^0}{\tau_0}\right)$$

Where A_0 = 4096 *points from the zero crossing*

χ = *the number of cycles in the window*

$\tau = \{0, \tau_0\}$ τ_0 = *duration of window*

A 1000 Hz sine tone produced from a function generator (Hewlett Packard 3310A) was visually matched to a 1.0 kHz, 6-cycle tone burst to determine the peak equivalent (P.E.) decibel values. A sound level meter (Larson-Davis Model 800B) coupled to a condenser microphone (Brüel & Kjaer) quantified amplitude measurements. A dynamic range from 116-23 dB SPL characterized an in-house software-driven attenuator program.

Under typical experimental conditions, the tone burst was held to a duration ≤ 5 ms since the first response of the EOAE occurs 6-8 ms after the stimulus onset (Kemp, 1978, 1986). The repetition rate was held constant at 11.1/ s with phase-locking maintained by the function generator Mi^2 system (Moore et al., 1984).

Calibration

Typically, calibration was performed under two conditions: (1) open system and (2) closed system. The configuration of the open system was sequenced with the microphone in a rectilinear distance from the headset of a TDH-49 earphone (Teledyne) seated in a foam rubber cushion (MX/AR 41). The headset served as a signal source.

The orientation of the open system was used to determine (exclusively) proximity effects of receiver-to-microphone distance. All measurements were recorded from an azimuth of 0°.

The closed system was comprised of a critically damped hard-walled cavity, to simulate an ear canal volume (2 cc) coupled to the EPA-4. Specifically, a plastic cylindrical cavity was employed to determine the proximity effects of (d_2-d_1) over a range of 1-20 mm. Selected probe calibration recordings of frequency dispersion were altered as a function of distance (d_2-d_1). The time history of the tone burst stimulus was recorded and averaged using analog-to-digital components (A/D) components.

Tone bursts were digitally generated using the digital control unit (Mi^2 system) and controlled by a calibrated software-driven attenuator. The tone burst provides specificity of stimulation near threshold (Davis, Hirsh, Popelka, and Formby, 1984; Eggermont and Odenthal, 1976). For this reason, the tone burst was used to derive responses near threshold. The tone burst stimuli were similar to previous acoustic emission research (Neely, Norton, Gorga and Jesteadt, 1988). A tone burst window estimated the theoretical travel time of a signal within the cochlea. The total duration of the tone burst varies with the number of cycles within the signal indicated in Table III-4. All stimuli were routed to a subminiature earspeaker (Knowles Electronics BJ- series), housed in the EPA-4 device. A pre-amplifier was used to increase the EOAE response signal prior to delivery to the data amplifier. The signal was amplified and usually monitored by an oscilloscope to maintain an optimal signal-to-noise ratio.

Equipment adjustments to the data amplifier (Data Inc. 2124 Model 2) were made to compensate for individual subject differences in ear canal volume and insertion gain. Specifically, as the stimulus amplitude was decreased an amplification correction factor was applied to enhance the EOAE detectability.

Table III-4 Tone burst duration and frequency composition

Frequency (Hz)	Time (ms)	Cycles
800	5.1	4.0
1000	4.0	4.0
1500	4.0	6.0
2000	4.0	8.0

Signal Processing

The emissions were obtained with the subject seated in a reclining position. All experimentation was performed with the subject inside an acoustically-controlled environment Industrial Acoustics Company (IAC). A (micro-computer IBM AT) was used to average 512 samples of the signal retrieved from the microphone. A signal was routed through the circuitry of the data amplifier at a bandpass of 0.3 to 3 kHz, re: -3 dB points. The sampling frequency was set at 100 kHz, matched to a 0 ms stimulus delay, with an analysis time of 20 ms. An artefact rejection routine was employed in the range of +4 to -4 volts, which corresponds to ±80%

Subjects

The right ears of 22 audiometrically-normal adults were be examined. The subjects will comprised of two groups in respect to age: a younger group (average 21.86 years) and an older (average 46.27 years). Audiometrically normal subjects were determined by a hearing loss no greater than 25 dB HL from 0.25 -8 kHz in each ear (ANSI-69). A case history provided screening information to determine occurrences of conditions which loss

may have been shown to interfere with the OAEs, e.g. aspirin (Anderson et al., 1979). To this extent, potential subjects taking high dosages of aspirin were rejected.

A reclining chair within an acoustically shielded enclosure served as a platform for data collection. Seating the subjects in a reclining position reduced the physiologic noise that can interfere with the recording of evoked oto-acoustic emissions (see Figure III-3.). The subjects were encouraged to keep swallowing to a minimum because eustachian tube changes can alter the EOAE response. The emission probe assembly was mounted to a modified head band. The EPA-4 fitted within the outer third of the ear canal. The right ear was selected for all subjects.

An OAE threshold measurement preceded all data collection. A threshold measurement involved a behavioral response to a 1000 Hz tone burst. The test signal repetition rate was 11.1/sec. The stimuli for data collection consisted of two tone bursts (800 Hz and 1500 Hz). On separate sequences the respective signals were presented as two descending series of 55, 45, and 35 dB nHL. The stimulus amplitude values had a correction factor of -23 dB. In other words 55 dB corresponded to $\approx$ 3 dB HL. The responses were obtained through the Mi^2 modular system, and these data were temporarily stored in the computer. The analysis of these data points was determined with the Mi^2 signal processing program.

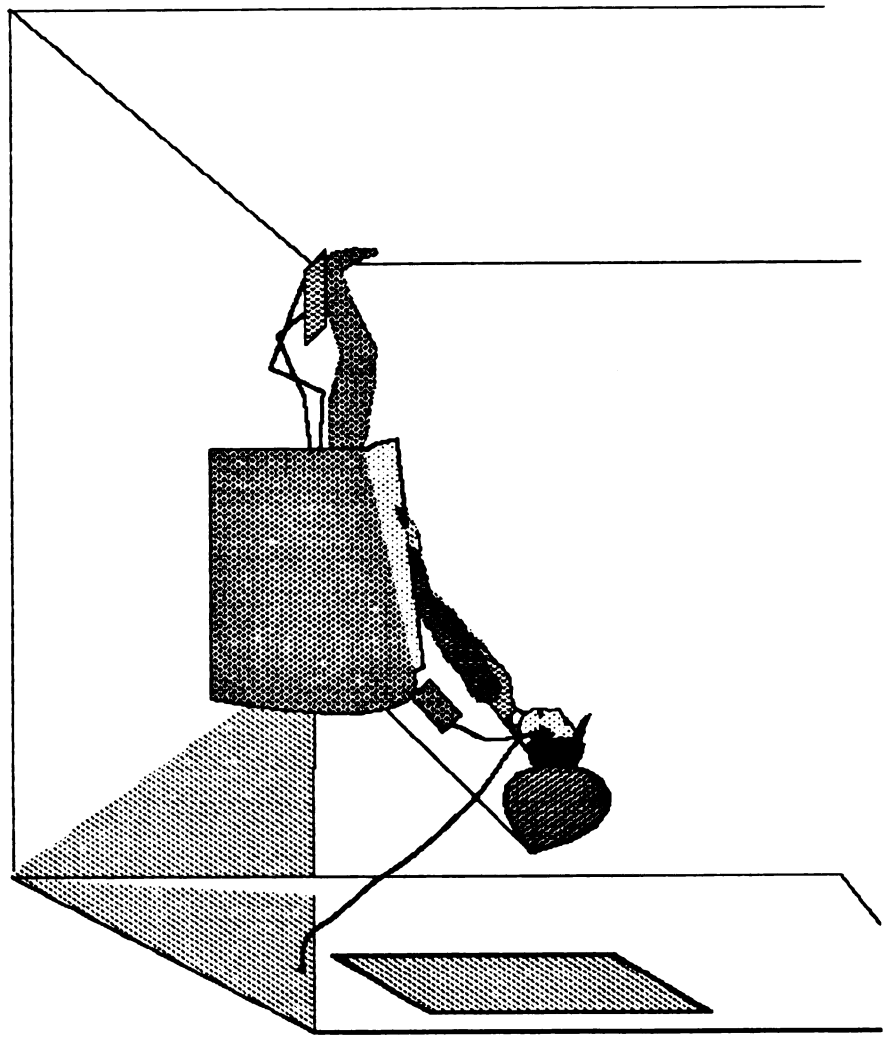


Figure III-3. Experimental Environment

Signal Analysis

Two 'observation intervals' determined when the expected EOAE response would occur. Based on the theoretical cochlear travel time pioneering research(Eggermont et al., 1976; Zerlin, 1969) and more recent studies (Avan, Bonfils, Loth, Narcy and Trotoux, 1991; Collet, Gartner Moulin and Morgon, 1990; Kemp, 1978, 1986), the first observation interval (I) occurs 6-8 milliseconds after the stimulus onset or post stimulus time (pst). The second observation interval (II) emerges 12-14 ms pst. The travel time for a disturbance within the cochlear partition (from base to apex) approximates 5 ms (Zerlin, 1969). Theoretically, the EOAE response could not occur before 5-6 ms, and the EOAE response threshold increases with age (Bonfils, Bertrand, and Uziel, 1988b; Collet et al., 1990; Probst, Lonsbury-Martin, Martin, and Coats, 1987).

Each observation interval corresponded to an amplitude measurement in SPL peak equivalent decibel values (re 20 μPa. Stimulus amplitude and frequency were the independent variables with EOAE response amplitude and latency as the outcome variables. The best of two trials represented these data sets. Statistical analysis of the mean data resolved whether a significant difference existed between age groups, among amplitudes, and between frequencies as modeled in Fig. III-4.. A total of 22 cases comprised the data set for both younger and older subjects (eleven per group). All subjects were of the feminine gender. Women were selected over men based on the prevailing understanding of sociocusis and the heterogeneity of hearing loss among men (Lebo et al., 1972).

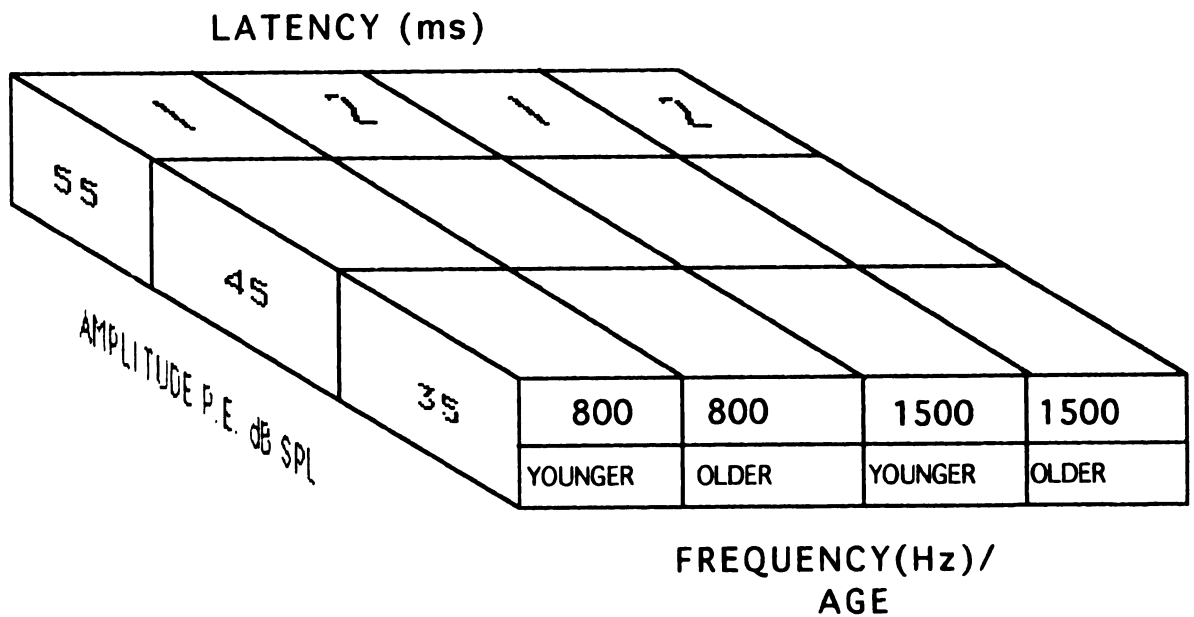


Figure III-4. Statistical Model

CHAPTER IV

RESULTS

This study sought to investigate the evoked oto-acoustic emission in two groups of subjects. One group was composed of audiometrically normal younger subjects. The experimental group consisted of audiometrically normal older subjects. The stimulus parameters of frequency (800 and 1500 Hz) and intensity (35, 45, 55 dB SPL P.E.) served as the independent variables. The EOAE latency, in two defined observation intervals and amplitude of these intervals, served as the dependent variables. The data were analyzed using the Statistical Package for Social Science (SPSS) and SYSTAT® for the Macintosh. Three hypotheses were posited:

(i) The amplitude of the EOAE does not differ significantly with advancing age.

(ii) The frequency composition of the EOAE does not differ significantly as a function of advancing age.

(iii) The latency of the EOAE does not differ significantly with advancing age.

Certain assumptions were made on the results of the previous literature and theoretical constructs. For example, it was expected that the most robust response would occur at the lowest stimulus intensity (35 dB SPL P.E.), while saturation of the response was expected at the highest intensity (55 dB SPL P.E.) (Stevens, 1988; Stevens, Webb, Hutchingson, Connell, smith and Buffin, 1990). These investigations accounted for the nonlinear response of the cochlea. The latency of the EOAE was expected to increase with a lower frequency (800 Hz) (Moore and Davis, 1984). It was expected that the

EOAEs of older subjects should show greater latency and smaller response magnitude to each set of independent variables (Bonfils et al., 1988a; Collet et al., 1990).

The predictable results were also based upon the findings of Bonfils et al., 1988b Kemp, 1978; The most robust response should have occurred at the lowest stimulus amplitude, whereas saturation of the response was expected at the highest stimulus presentation level (Stevens, 1988; Stevens, Webb, Hutchingson, Connell, Smith and Buffin 1990). The latency was expected to increase with a lower frequency. As it turns out, recent research suggests that the responses for the older subjects should be affected by age (Bonfils et al., 1988a Collet et al., 1990).

Audiometric air conduction pure tone threshold and middle ear immittance values determined the eligibility of candidates. Case history information verified factors that could potentially confound these data. Specific conditions that alter or obliterate the EOAE include: high dosages of aspirin, sensorineural hearing loss, middle ear pathology, acoustic trauma, and ototoxic agents. To this extent, potential subjects were eliminated that evidenced conditions that could compromise the cochlear response.

Tone bursts of 800 Hz and 1500 Hz (shown in Table III-4) were routed through the emission probe assembly (EPA-4) with the following constants:

(1) Stimulus polarity	Condensation
(2) Repetition rate	11.1/sec
(3) Samples	512
(4) Test ear	Right
(5) Time window	20 ms (0 s delay).
(6) Sample frequency	100 kHz

The EOAE was amplified within a range (1.2×10^3 -to 1.2×10^5) before data storage. Response amplitudes varied among subjects. Generally, as the stimulus amplitude decreased, the amplification factor of the response was manually increased. The recorded data represent the best of two trials for frequency, duration and amplitude.

Latency was analyzed as a categorical variable (observation intervals I and II) at two frequencies (800 Hz and 1000 Hz) and two age groups (younger and older). The continuous quantitative variables comprised amplitude (all negative decibel values) and latency values in milliseconds.

The data were analyzed applying a one-way ANOVA by comparing the amplitudes at three stimulus levels (55, 45, and 35 dB SPL P.E.). The six stimulus amplitudes for each frequency were matched about age (younger and older). All amplitude values corresponded to negative decibels (SPL P.E.). The negative logarithmic units were fixed to a reference (2×10^{-5} N/m^2). Therefore, less negative units represented an increase in sound pressure level. Low level (35 dB) tone-burst stimuli elicited the lowest EOAE amplitudes. Typically, emissions have emerged near an individual's audiometric threshold. This was observation noted by previous researchers (Bonfils et al., 1988a, 1988b; Kemp, 1978, Kemp et al., 1986; Wit et al., 1980; Zurek, 1985). The Appendix A registers the tabulated values for mean amplitude for both 800 Hz and 1500 Hz.

Hypothesis 1. The amplitude of the response does not differ significantly with advancing age.

Analog traces of a representative older subject at 800 Hz (35, 45, and 55 dB SPL P. E.) are shown in Figure IV-2. In a manner like the responses from the younger subject, the oscillations immediately following the stimulus decreased as the stimulus intensity decreased. It was noted, however, that frequency dispersion was quite evident for the older subject. Asymmetry of the EOAE for the older subject was apparent in the older subject.

Quantification of the 800 Hz analog data for the younger and older subjects at observation 1 is shown in Figure IV-3. It was evident in the first observation interval, that the older subjects yielded greater response amplitudes (less negative) than the younger subjects at all three stimulus intensity levels. For both groups, there was a progression of greater amplitude as the stimulus was increased at each stimulus level,

using the ±1 standard deviation. However, the older subject variability, at the highest stimulus level, is greater than that of the younger subjects. On the other hand, the positive accelerating slope for the younger subjects is more gradual than that for older subjects.

The quantified amplitude data for younger and older subjects at 800 Hz, stimulus levels of 35, 45, and 55 dB at observation 2 are shown in Figure IV-4. A contrast of the analogous stimulus levels revealed EOAE amplitude differences. We saw that the amplitude was higher for the older subjects than the younger subjects when comparing intensity levels of the stimulus. In distinction, there was a progression of greater amplitude for observation interval 2 as the stimulus intensity was increased.

The 1500 Hz analog data for a representative younger subject at stimulus levels of 35, 45, and 55 dB are displayed in Figure IV-5. Frequency dispersion was observed immediately following the tone-burst envelope, an event which was consistent with the traces in Figure IV-2. In consideration of the correction factor, the EOAE amplitude increased with an increase in stimulus amplitude.

In like manner, 1500 Hz analog traces for a representative older subject are shown in Figure IV-6. The most salient characteristics were minimal response amplitude and asymmetry. In this instance, there was more intrasubject variability for the younger group. Frequency dispersion was not as obvious in these traces.

The overall mean amplitudes for both 800 Hz and 1500 Hz followed a similar pattern, i.e., the amplitude of the emissions decreased as the stimulus amplitude decreased. However, the scale or probable slope was different. The variances within the older group were closer than the younger group.

On the average, the EOAE amplitude for younger subjects was lower than the older subjects. Thus, a one-way ANOVA was calculated to determine whether a significant difference existed between the mean amplitude values for both the younger and older groups. As it turns out, the within subject variation for both groups was not homo-

geneous; this was substantiated by the variance ratio test (Bartlett-Box F-test) on all 22 cases.

In all but three instances, the heterogeneity of variance did not support one of the main assumptions for ANOVA validity. However, a significant variance ratio (F-test) was noted for 800 Hz (55 dB, observation 1) and 1500 Hz (55 dB, observation 2). Specifically, the respective F values for 800 Hz and 1500 Hz were (F 5.894 ▪ p= .051) and (F 11.81 ▪ p< .001) and suggest a "probable difference." Notwithstanding, the first hypothesis could not be rejected with any validity using a one-way ANOVA procedure as shown in Table IV-1 and IV-2. The values were not valid because of unequal variances. The repeated measures upon each subject made multiple t-tests inappropriate because the independence assumption would have been violated. The Appendix B presents the univariate homogeneity of variance tests for amplitude at 800 and 1500 Hz. To determine if the amplitude measures were free of error variance, reliability coefficients were calculated on 22 cases (both groups). A reliability coefficient resolves the degree to which a test is free of error variance or measurement error. The range, based on the Cronbach α is between the values of .00, with 1.00 indicating complete reliability (Borg and Gall, 1983) as the reliability coefficient represents the cumulative effect of differences due to chance between all subjects. The reliability coefficients for this investigation were statistically significant for all amplitude measurements at 800 Hz and 1500 Hz (see Table IV-1). Specifically, Cronbach α values were: 0.816 (observation interval 1) and 0.843 (observation interval 2).

Table IV-1 Reliability Coefficients for Amplitude 800 Hz and 1500 Hz (22 cases)

Observation Interval	Reliability Coefficient	Number of Amplitudes
1	.816 *	6
2	.843 *	6

Cronbach *$\underline{\alpha}$ >.40.

Figure IV-1 Analog traces for EOAEs at 35, 45, & 55 dB SPL P.E. at 800 Hz for a typical younger subject

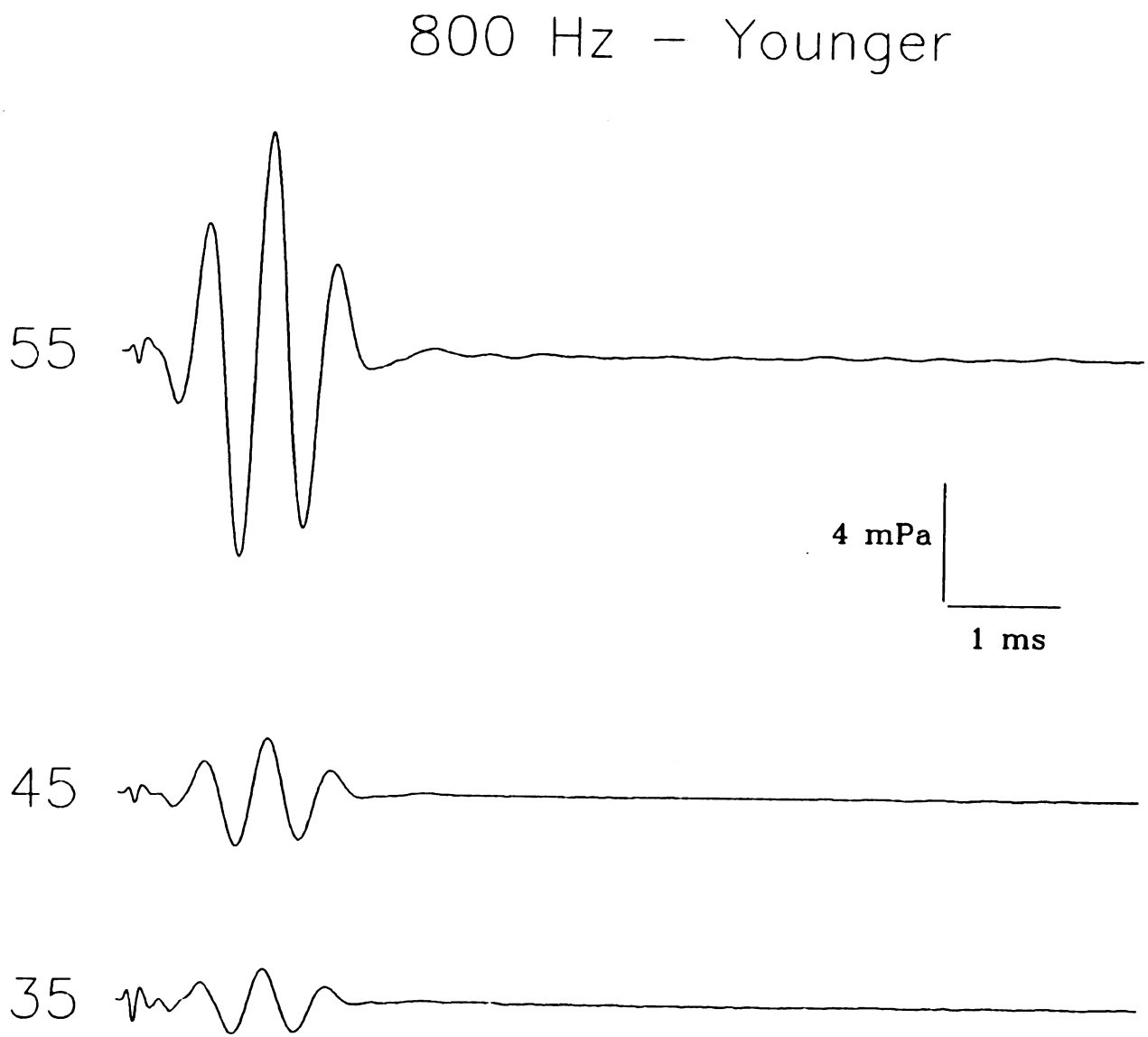

800 Hz – Younger
55
4 mPa
1 ms
45
35

Figure IV-2 Analog traces for EOAEs at 35, 45, & 55 dB SPL P.E. at 800 Hz for a typical older subject

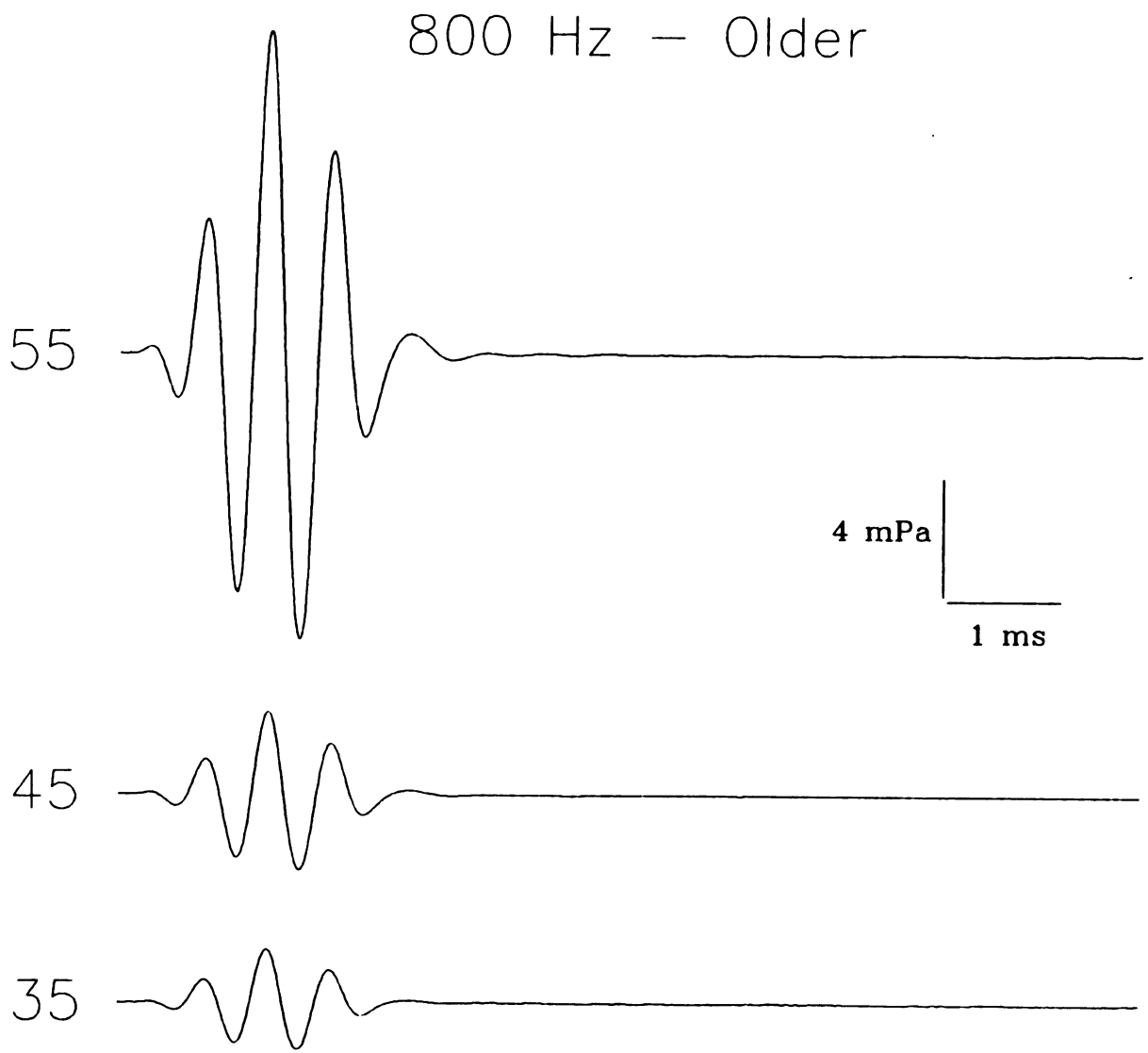
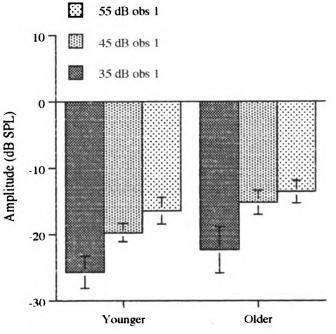
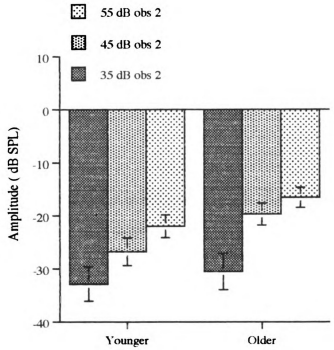

800 Hz – Older
55
4 mPa
1 ms
45
35

Figure IV-3 Mean 800 Hz EOAE amplitude as a function of stimulus intensity for younger and older subjects for observation interval 1.



Amplitude 800 Hz Observation 1

Figure IV-4 Mean 800 Hz EOAE amplitude as a function of stimulus intensity for younger and older subjects for observation interval 2.



800 Hz Amplitude Observation 2

Figure IV-5 Analog traces for EOAEs at 35, 45, & 55 dB SPL P.E. at 1500 Hz for a typical younger subject

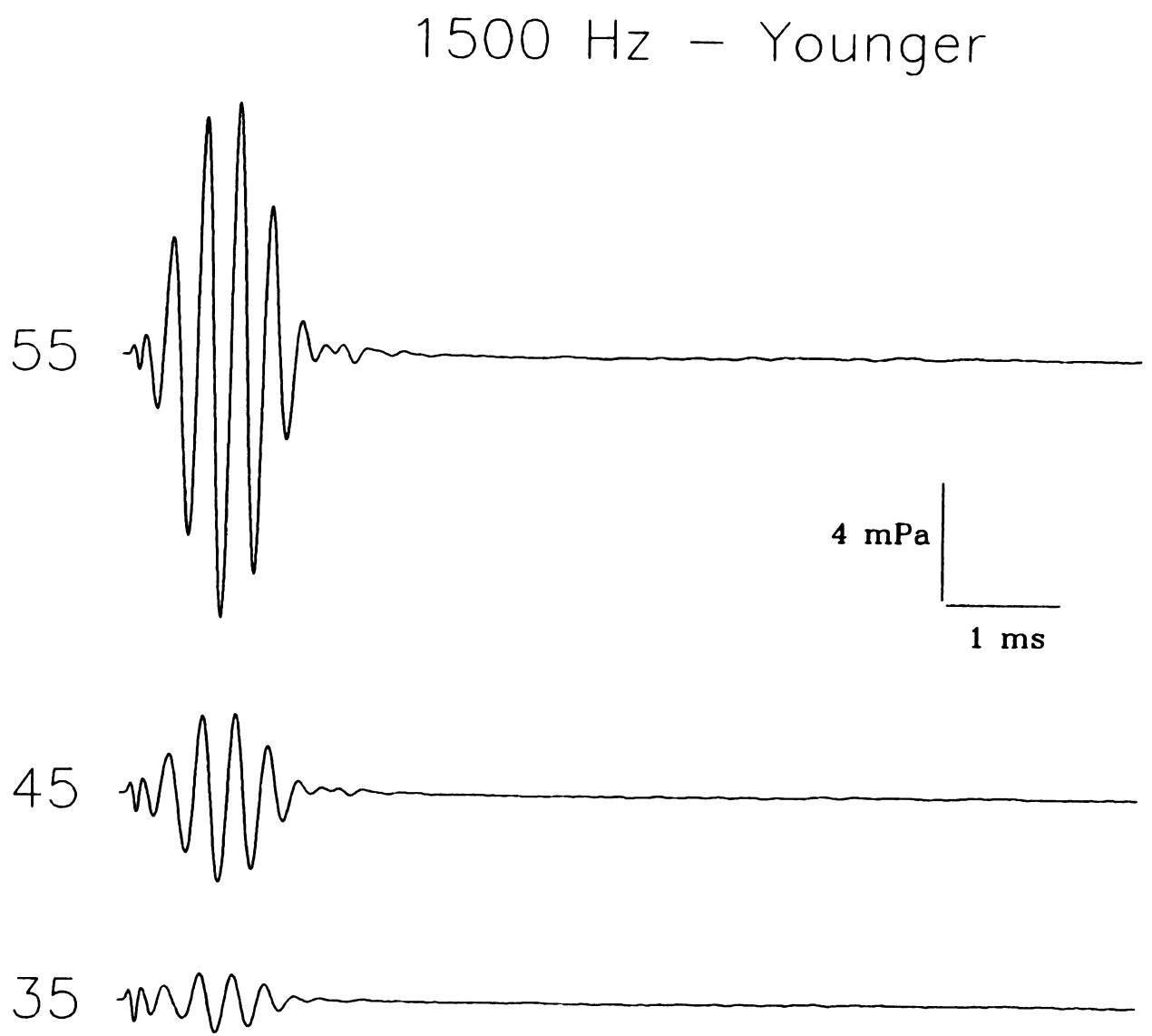

1500 Hz – Younger
55
4 mPa
1 ms
45
35

Figure IV-6 Analog traces for EOAEs at 35, 45, & 55 dB SPL P.E. at 1500 Hz for a typical older subject

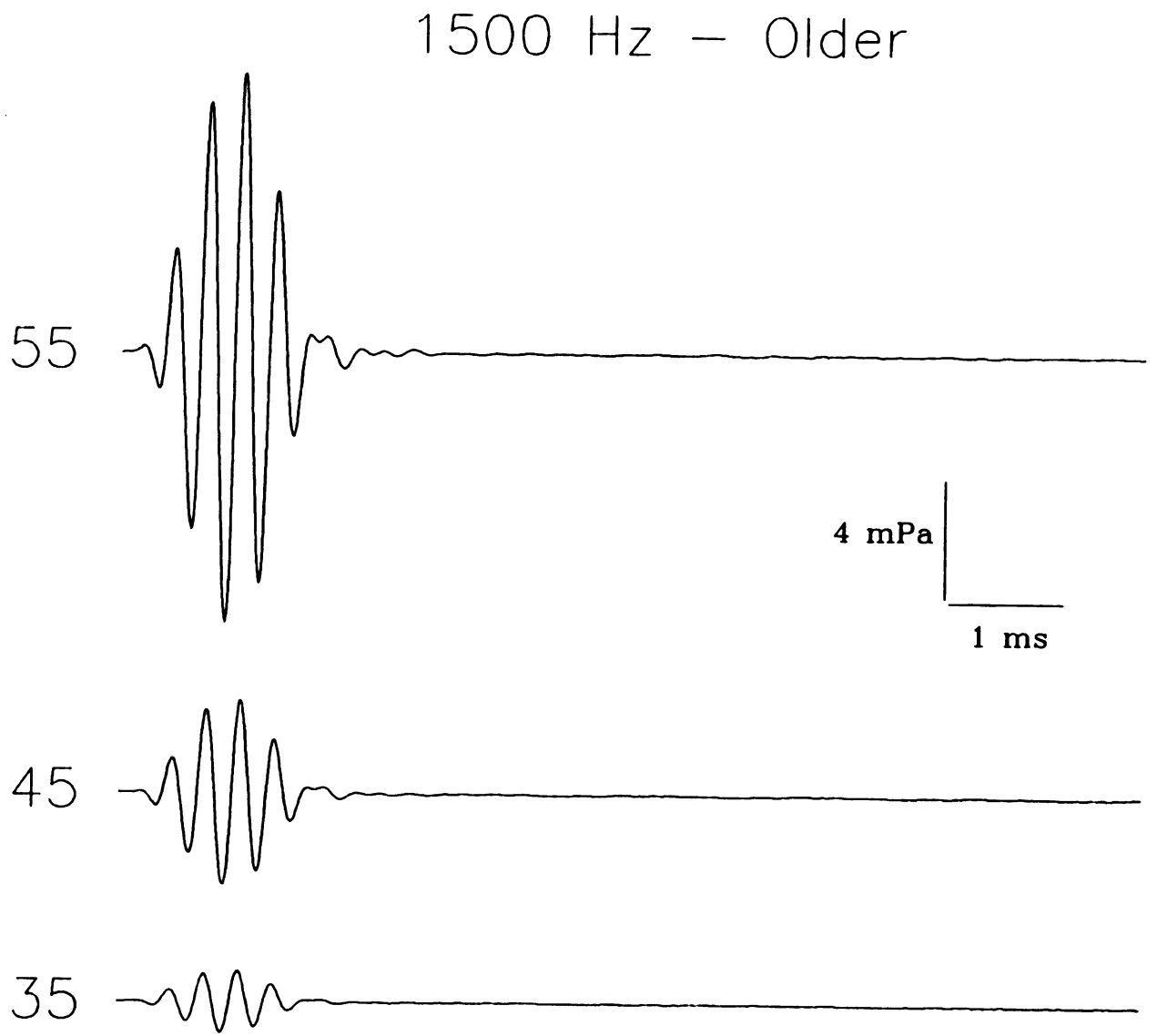
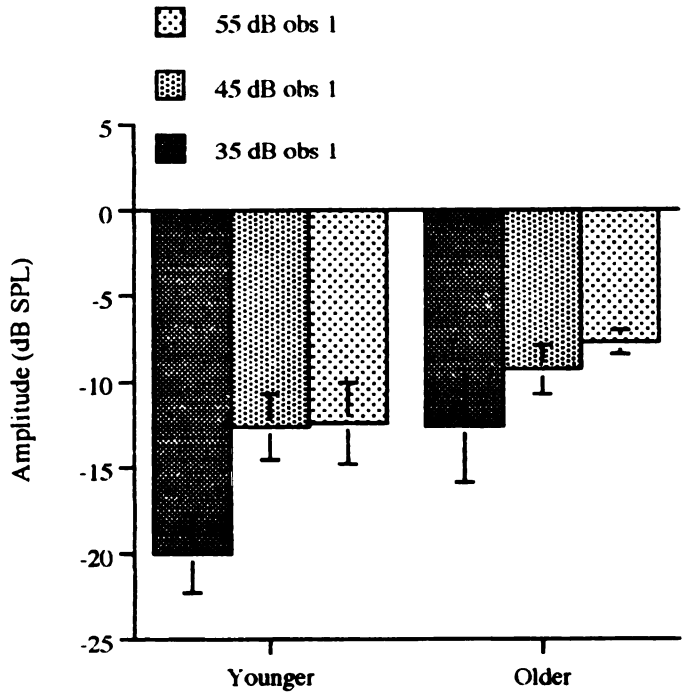
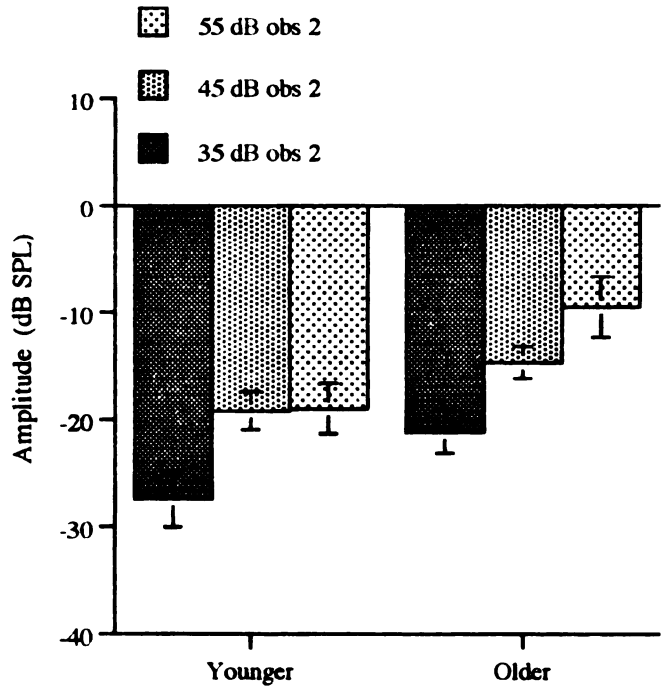

1500 Hz – Older
55
4 mPa
1 ms
45
35

Figure IV-7 Mean 1500 Hz EOAE amplitude as a function of stimulus intensity for younger and older subjects for observation interval 1



Amplitude 1500 Hz Observation 1

Figure IV-8 Mean 1500 Hz EOAE amplitude as a function of stimulus intensity for younger and older subjects for observation interval 2



Amplitude 1500 Hz Observation 2

Hypothesis 2. The frequency composition does not differ significantly as a function of advancing age. The one-way ANOVA design for hypothesis 1 was used also to examine the difference between frequency by the mean response amplitude. The null hypothesis was not rejected. However, (55 dB, observation 2) corresponding variance ratio values were recorded for within subject effects (F 5.894 ≈ p= .051) and (F 11.81 ≈ p< .001) (See Tables IV-3 and IV-4). The significant interpretation of this finding will be discussed in more detail in Chapter V. The amplitude of 35 dB was the focus of this hypothesis because of the greater frequency specificity of the tone burst at low levels (Eggermont, Spoor and Odenthal, 1976; Norton and Neely, 1987). In addition, the emissions have been shown to become saturated or obliterated at higher stimulus amplitudes, i.e., 55 dB and 45 dB.

The data represented in Figure IV-3 and Figure IV-4 indicated that the amplitudes between both groups were not significantly different for 800 Hz. A specific instance in observation interval 1 emphasizes the statistically significant measurement error. A difference was expected at 35 dB. Observation interval 2 at 800 Hz yielded a discernible trend of descending amplitudes and with less variability within the groups. The mean tabulated data reflects the equivalence between the 800 Hz latency values for both observation intervals.

A contrast of the 1500 Hz stimulus levels revealed EOAE amplitude differences. The Figure IV-7 & Figure IV-8 show greater differences between the 35 dB latencies for younger and older subjects. The differences, however, were offset by the measurement error. Inconsistent patterns were noted with hypothesis 1. Similar discrepancies existed with these data points.

Table IV-2. Tests of Between-Subjects Effects (Amplitude 1 x Age)

Source	SS	DF	MS	F	p
Within Cells	4003.46	20	159.18		
Constant	54155.79	1	54155.79	270.54	.000***
Age	558.51	1	558.51	2.79	.110

***p < .001.

*Analysis was based on log transformed variables.

Table IV-3. Tests of Between-Subjects Effects (Amplitude 2 x Age)

Source	SS	DF	MS	F	p
Within Cells	3183.60	20	159.18		
Constant	27915.93	1	27915.93	175.37	.000***
Age	166.46	1	166.46	1.05	.319

***p < .001.

*Analysis was based on log transformed variables.

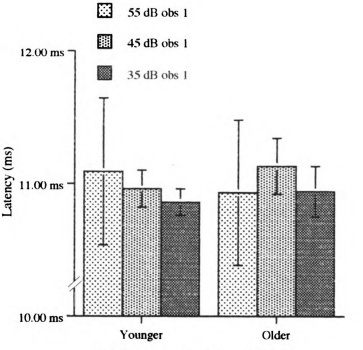
Hypothesis 3 The latency of the EOAE does not change with advancing age.

The quantified latency data for younger and older subjects at 800 Hz, stimulus levels of 35, 45, and 55 dB at observation 1 are shown in Figure IV-9. A contrast of the stimulus levels revealed inconstant EOAE amplitude differences. The quantified data for observation 2 indicated the latency increasing as the stimulus amplitude decreased (see Figure IV-10).

In a manner similar to 800 Hz, it was difficult to determine a trend for 1500 Hz in the quantified latency data for younger and older subjects at observation interval 1 (see Figure IV-11). As it turns out, both the younger and group followed an inconsistent pattern that relative to the stimulus amplitude. The latency for observation interval 2

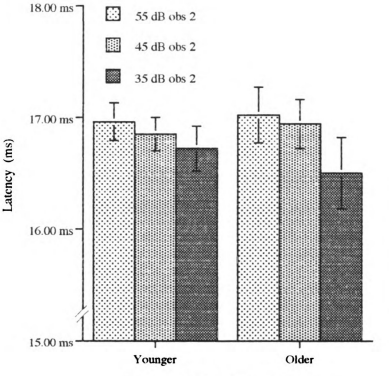
presented comparable trends in the mean values and the error variance. Again consistent measurement error accompanied the mean values for latency between both groups (see Figure IV-12).

Figure IV-9 Mean 800 Hz EOAE latency as a function of stimulus amplitude for younger and older subjects (observation interval 1)



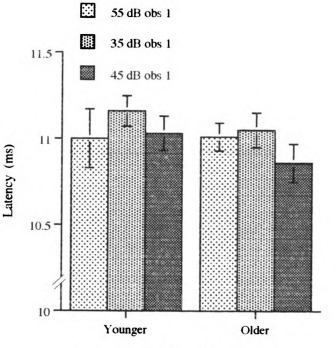
800 Hz Latency Observation 1

Figure IV-10 Mean 800 Hz EOAE latency as a function of stimulus amplitude for younger and older subjects (observation interval 2)



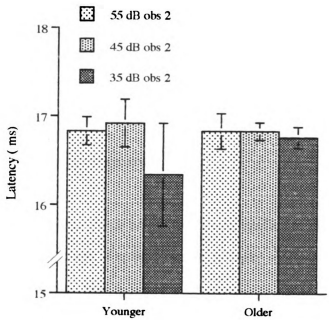
Latency 800 Hz Observation 2

Figure IV-11 Mean 1500 Hz EOAE latency as a function of stimulus amplitude for younger and older subjects (observation interval 1)



Latency 1500 Hz Observation 1

Figure IV-12 Mean 1500 Hz EOAE latency as a function of stimulus amplitude for younger and older subjects (observation interval 2)



Latency 1500 Hz Observation 2

On the surface the latency values for observation interval 2 appear curvilinear. However, the data is spurious and does not follow a trend based on a curvilinear function. The reliability coefficients for the latency data are shown in Table IV-7. On 22 cases, there were consistent and marginally significant error. The Cronbach α value was borderline for observation interval 2.

Table IV-4. Reliability Coefficients for Latency 800 Hz and 1500 Hz (22 cases)

Observation Interval	Reliability Coefficient	Number of Latencies
1	.739*	6
2	.488*	6

Cronbach *$\underline{\alpha}$ >.40.

Analysis was based on raw variables.

In order to test this hypothesis an ANOVA was applied to determine the differences between mean latency by age. On the average, unequal variances existed within the younger and older groups. The

Appendices A 1 & A.2 register the 800 Hz tabulated values for mean latency. In the first observation interval, significant variance ratio was recorded for 55 dB for 800 Hz and 1500 Hz. A significant variance ratio was noted for 800 Hz (55 and 45 dB, observation 2) and 1500 Hz (45 and 35 dB, observation 2). The Appendix Bshows the univariate homogeneity of variance tests for 800 and 1500. The ANOVA was not valid based on the heterogeneity of variance within group effect. These data suggest that there is no significant difference in frequency as a function of age. Thus, the third hypothesis was not rejected.

Table IV-5. Tests of Between-Subjects Effects (Latency 1 x Age)

Source	SS	DF	MS	F	p
Within Cells	11.91	20	.60		
Constant	15976.62	1	15976.62	26823.91	.000***
Latency	.03	1	.03	.06	.816

***p < .001.

*Analysis was based on log transformed variables.

Table IV-6 . Tests of Between-Subjects Effects (Latency 2 x Age)

Source	SS	DF	MS	F	p
Within Cells	47.34	20	2.37		
Constant	36892.68	1	36892.68	15585.49	.000***
Latency	1.27	1	1.27	.54	.472

***p < .001.

*Analysis was based on log transformed variables.

The investigator of this study identified the uncertainty associated with the use of parametric statistics in this study. The small sample size reduced the potential statistical power generated from a larger sample size. Nevertheless, there were fundamental reasons for not applying a nonparametric approach:

1. Nonpararametric statistics do not make assumptions about the distribution of the outcome. The data represented in this study were continuous and quantatative which usually follow a normal distribution.
2. Nonparametric statistics rank order the outcomes so that the precision employed during data retrieval is lost.

3. Small sample sizes <10 are suitable for nonparametric tests. The sample size for this study was borderline.
4. The assumption of a normal distribution may have been violated in several instances. Appropriately, the data can be transformed if the homogeneity of variance assumption is broken using parametric procedures. Known non-normal distributions cannot be transformed (Steel and Torrie, 1980).

Limitations

- The investigator recognized several limitations inherent in this study by using a prototype.
- Primarily, the design was based on, often sketchy, details from previous research papers. However, the final model provided the best results similarly to previous examples.
- The instrumentation and experimental protocol had not been used with any previous emission research from the Neuro-audiologic Laboratory at Michigan State. Therefore, these data contribute to a pool of normative data for this system and those to follow.
- In retrospect, the sample size was small for the inferences that could be made.
- There was no attempt to apply the results of this study to the general population.
- The failure to achieve equal variances limited the options for statistical analysis.
- Multiple t-tests would have violated the independence assumption for repeated measures. Regression analysis would have been inappropriate for this data set because of related measures.

CHAPTER V

SUMMARY AND DISCUSSION

This chapter will be presented in four parts: the summary if research, discussion of the major findings, conclusion of the study, and recommendations and implications for future research.

Summary of the Study

The specific aim of this study was to test experimentally, the relationship between two age groups and evoked oto-acoustic emissions (EOAE). The selected age groups were divided between 'younger' (average age 21.86) and 'older' (average age 46.27). The outcome of age differences was evaluated on the EOAE response amplitude (dB SPL P.E.) as a function of frequency (800 Hz and 1500 Hz) and three stimulus amplitudes (55, 45, and 35 dB HL). Responses were scored at two observation intervals the duration of which is based on the cochlear partition travel time (Eggermont et al., 1976; Zerlin, 1969). An additional purpose of the study was the development of a prototypical device and an integrated instrumentation retrieval system. The EOAE system and emission probe assembly-four prototype (EPA-4) were extensions from previous electro-physiologic research using a microcomputer and dedicated hardware and software (Moore et al., 1989). The EOAE system arose from a research model that could evaluate the auditory system from the periphery to central regions. Exploratory research and development led to a determination of several operating characteristics and limitations.

Findings of this study were based on the research and development of the EOAE system and data collection in the period of 1988 to 1991. A few hypotheses were based

on theoretical and empirical grounds. Emissions of a mechanical origin formed the conceptual framework for this study. Parametric statistics were used to test the model proposed in this project.

Discussion of Major Findings

The discussion regarding the outcome of this study should be considered with circumspection in the context of the limitations (see p.90) The present study investigated a few basic relationships: (a) the relationship between the age and EOAE amplitude, (b) the relationship between age and EOAE latency, (c) the effect of several stimulus parameters, i.e., stimulus amplitude and frequency, on the EOAE response. Technically, all acoustic emission analysis is performed at very low SPL values (Matthews & Hay, 1983; Hardy, 1981) the findings of which have been consistent with specific studies of oto-acoustic emission analysis (Avan et al., 1991; Johnsen, 1982; Kemp, 1986, 1978).

On the average, this investigator found that the most robust responses were not recorded at low stimulus amplitudes for both 800 and 1500 Hz, a result that contradicted the findings of others (Collet et al., 1990; Probst et al., 1987; Bonfils et al., 1988b; Elberling et al., 1985). However, this set of data did not show a consistent trend within the groups themselves (see Figures IV-3 & IV-4, IV-7 & IV-8). The response is saturated at high stimulus amplitudes. The mean threshold for the EOAE response was lower for the group of younger subjects . The within subject variability was statistically significant. Homogeneity of variance tests were performed to determine the basis of the ANOVA assumptions or other parametric tests. In contrast, the between subject variability was not significant (see tables IV-1 and IV-2).

The data were log transformed to perform the ANOVA. As a result there was not a significant difference in amplitude as a function of age. This result contradicts the conclusions of Bonfils et al., 1988a. Prior research has indicated that the EOAE

threshold elevates with advancing age (Avan et al., 1991; Probst & Hauser, 1990; Probst et al., 1987). Presbycusis has been shown to elevate the threshold of subjects (Bonfils et al., 1988a; Tanaka, O-uchi, Shimada, and Koseki, 1988). All of the subjects for this study were tested audiometrically normal, i.e., air conduction pure tone thresholds <20 dB HL and normal middle ear functioning, a model performed by Kemp (1986). The current study presents data that are consistent with the direct relationship that EOAE threshold increases nonsignificantly with advancing age. Avan et al. (1991) reported significant correlations between EOAE correlations and pure tone audiometric thresholds.

The tone burst stimulus has been shown to provide frequency specificity near threshold (Davis et al., 1984; Eggermont et al., 1976). The most sensitive recordings for the EOAE were near threshold 35 dB nHL (indicated). On the other hand, the stimulus at 55 dB corresponds to a saturated response. The ANOVA for hypothesis 2 (see page 77) was not rejected based on the findings from the first hypothesis. The differences between the age groups by frequency were insignificant. Again the within subject variability was significant. These within subject effects precluded an analysis based on regression. Analysis of 22 cases, the frequency differences were not significant. Previous research notes that emissions are seldom seen between 750 Hz and 1500 Hz (Bonfils et al., 1988a; Kemp, 1978, 1986) and seldom above 2000 Hz (Avan et al., 1991). In a similar manner, emissions were recorded at 800 Hz and 1500 Hz. Consistently, emissions have been recorded near 1000 Hz (Probst et al., 1987; Zwicker and Schloth, 1984; Kemp, 1978) In distinction, Kemp et al. (1986) suggested that a hermetic seal be used for emissions ≤400 Hz.

These data also represent an analysis that targeted the linear cochlear response. Previous studies employed a subtraction of the nonlinear cochlear response from a linear response. The resulting emission was then recorded within the time window of 6-8 ms pst (Bonfils et al., 1988a;Kemp, 1978; Norton et al., 1987) The first occurrences of emissions were recorded 11-13 ms pst. The latency of the response was not consistent

with pioneering and subsequent emission research, i.e., the first emissions occur approximately, 6-8 ms pst (Kemp, 1978) and 5 ms pst (Bonfils et al, 1988b; Neely et al., 1987).

The data, of the present study were analyzed within a 20 ms time window (0 s delay). In other words, the signal was analyzed with the response. The stimulus signal (tone burst) was restricted to ≤5 ms. Representation of exemplary analog data shows the relative amplitude of the emissions (see Figures IV-1 & IV-2, IV-5 & IV-6). In another context, the responses emerged 6-8 ms after stimulus cessation. It is not clear why the delay was much longer (pst) for the latencies of this study.

The latency data were transformed and an ANOVA was calculated on observation interval 1 and 2. The null hypothesis was retained for differences in latency as a function of age (see Tables IV-3 and IV-4). The data reflect wide intragroup variability (see Figures IV- 9 through IV-12). There has not been any specific research addressing EOAE latency as a function of age. It has been confirmed through the traveling wave theory that the latency of a disturbance decreases with higher frequency (Hubbard, 1986; Dallos, 1981,1973; Sutton et al., 1983; von Békésy, 1960; Gold, 1948; Gold & Pumphery, 1948). A mechanistic explanation of emissions was the conceptual framework for this study (see pages. 2, 9).

Conclusions

The evoked oto-acoustic emission has emerged from the technology and study of material science. In the context of engineering, the cochlea is a mechanical phenomenon that rivals any device of human hands. When Kemp (1978) presented his pioneering findings, health professions were equipped with additional knowledge for auditory assessment. New instrumentation was required to examine indirectly a mechanical aspect of the inner ear. Several prototypes have arisen from research and development. As a

consequence, several studies have attempted to refine the retrieval of EOAEs and to interpret the effect of several parameters, i.e., stimulus amplitude, frequency, and various signal configurations.

The present study involved the development of an emission probe assembly. The data were subjected through the fourth prototype Emission Probe Assembly-4 (EPA-4). As such, the present study represents the first use of the instrumentation to answer questions that implicate normal auditory functioning. The EPA-4 was integrated into a system of instrumentation that was configured to optimize signal generation and data analysis. The performance of the probe assembly was monitored by the data analysis.

Philosophically, most studies can be divided between those that propose a specific region of EOAE origin and the supposition that emissions originate from a broader region. Localization of the active zone for emissions may be in a specific region (Wit et al., 1980; Ruggero et al., 1983), or it may emanate from a broader combination of regions (Kemp, 1978). The loci of the emissions may be a modulated mechanism related to tinnitus. This study was exploratory with an inclination toward the findings of Kemp (Brown & Kemp 1984; 1986, Kemp et al., 1986, 1978).

The present study was designed to determine whether the EPA-4 delivers a signal and to determine if it could retrieve a response. In addition, three hypotheses were tested:

(i) The amplitude of the response does not change with advancing age.

(ii) The frequency composition does not differ significantly as a function of advancing age.

(iii) The latency of the EOAE does not change with advancing age.

There were 22 female subjects selected for this study. All subjects represented normal pure tone audiometric thresholds. The stimuli for data collection consisted of two

tone bursts (800 Hz and 1500 Hz). Each tone burst was restricted to ≤ 5 ms in duration. On separate sequences the respective signals were presented as two descending series of 55, 45, and 35 dB nHL. The stimulus amplitude values had a correction factor of -23 dB. In other words, 55 dB corresponded to ≈ 03 dB HL.

No attempt was made to generalize the results to the general population. Parametric statistical procedures (ANOVA) did not produce any statistical differences by age. The statistical power of the design could have been increased with a larger sample. Nonparametric procedures would have rank ordered the precise recorded values. Even though the specific hypothesis testing was not significant, there were some lessons learned. Foremost, each new EPA design presented potential incompatibilities with selected instrumentation. Second, reconfiguration of the system can be adversely affected by human subject factors, i.e., fatigue, comfort, and hearing sensitivity.

At the time of this writing no one has produced any definitive evidence for the cause of presbycusis. Empirical evidence supports the supposition that aging has an adverse effect on the peripheral auditory system (see pages 9-10). The incidence for sensory and conductive hearing loss associated with age approximates 60% of all presbycusis (Schuknecht et al., 1974; Gleeson et al., 1987) and Anniko and Bagger-Sjöbäck (1985) have specific reports (post mortem) that implicate outer hair loss in the basal region of the cochlear partition as a major contributor to progressive hearing loss. It is reasonable to suggest that if the cochlear partition becomes stiffer, the amplitude of the emissions would be diminished and the latencies prolonged. To be sure, if any theory of hearing offers a benefit it should be able to explain the process with clarity. In like manner, a theory of aging should be concise to explain specific events during the process with predictions. Caution must be exercised in the interpretation of presbycusis for there are many underlying causes (Willot, 1991).

Taken together, aging processes appear to be governed by both programmed and random occurrences. However, the trend toward low, stable death rates has been

observed for a significant segment of the world population. Should the current decline continue medical ethics will be challenged to pursue and provide bold technologies and therapeutic interventions (Olshansky, Carnes, and Cassel, 1993). Clearly, we need to refine an operational definition of aging, i.e., a cumulative, universal, progressive, intrinsic, and degradative process (CUPID), as we can identify specific conditions of aging. Age-related hearing loss will be ever prevalent in the post World War II 'baby boom' generation. We must channel our energy to understand not only specific theories of hearing but also those of aging.

Surgical intervention to the cochlea in humans raises several ethical issues on the quality of life and the benefit of radical medical procedures. Therefore, collaborative efforts in the areas of Sensory Physiology, Chemical Engineering, Physics, Biomechanics, Molecular Biology, Biochemistry, Pharmacology and several Allied Health Professions (Audiology, Speech Pathology, Radiology) may unlock the mystery of age-related hearing loss. It is reasonable to suggest that we may be able to pharmacologically alter the fluid constituents of the cochlear partition and rendering it less stiff. The complex characteristics of aging provide us with several theoretical constructs many of which are disputed. Clearly, it is well established that the effects of aging cannot be controlled simply through 'healthy lifestyles' or strict dietary therapy (Olshansky et al., 1993). At current predictions and actual observations limited resources send a stern warning that the delivery of services to the aged is growing urgent.

The present possibilities may be limited, but the foundation for more hearing research is extensive. Some of the most recent research in the auditory periphery suggests that the tissue mechanics may hold some of the secrets concerning cochlear function using patch clamp techniques to explore outer hair cell motility (Ashmore, 1986; Brownell, 1986; Flock, 1983). In addition, molecular biology may provide some clues into hair cell regeneration, associated signals for gene expression, and cell fate. To this extent, Rubel (1993) reported that rudimentary hair cell proliferation was observed in avian species

(chick). It is reasonable to suggest that the recovery of sensory epithelium within the cochlea is on the leading edge of study for aging sensory systems. Ultimately, many paths may lead us to the truth.

Implications for Future Research

The purpose of this study was exploratory in nature. Its fundamental purpose was to identify the effect of age on the oto-acoustic emission response amplitude and latency. In addition, the study involved the research and development of an emission probe assembly. Serendipitously, a tone-burst of 4.1/s was used one younger subject not included in this study. In retrospect, the emissions were quite remarkable with discernible peaks at 1500 Hz. As such, the following suggestions for future research may advance our understanding:

(1) A larger sample should be used on a study of multiple age groups to increase the statistical power.
(2) An EOAE investigation of age differences using 750 Hz, 1000 Hz, and 1500 Hz to correlate the audiogram with recorded data should be considered.
(3) A complex tone-burst signal with a 750 Hz fundamental with components at 1000 Hz and 1500 Hz may provide information relative to auditory frequency selectivity.
(4) Studies of emissions with infants so that results can be compared with existing findings is recommended.
(5) A stimulus presentation rate of 4.1/s should be explored to determine if emissions can be resolved at that rate.
(6) Subjects with known episodes of tinnitus or other head noises may be suitable subjects for future emission research.

APPENDICES

APPENDIX A

Appendix A1. Younger amplitude values at 800 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Amplitude of EOAE (SPL) for younger subjects at 0.8 kHz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AC	23.67	-15.20	-23.68	-19.64	-23.43	-18.15	-21.33
CC	20.50	-11.02	-18.62	-15.45	-23.29	-22.88	-31.65
GD	24.16	-18.74	-24.84	-21.41	-28.77	-31.58	-36.86
HA	21.75	-34.11	-38.75	-30.98	-48.18	-26.38	-50.75
MK	23.83	-20.30	-25.56	-21.17	-28.61	-28.40	-38.10
SK	21.83	-10.51	-16.10	-18.27	-32.45	-14.83	-17.98
VA	23.41	-12.45	-17.53	-17.81	-23.10	-27.40	-31.28
YJ	23.50	-16.82	-25.38	-19.25	-23.26	-33.39	-39.25
BH	18.33	-10.67	-16.02	-16.65	-22.11	-18.24	-24.78
RJ	19.16	-15.14	-13.09	-13.73	-12.78	-19.58	-22.70
SR	20.33	-16.34	-22.33	-23.09	-28.86	-42.32	-47.03
TOTAL	240.47	-181.30	-241.90	-217.45	-294.84	-283.15	-361.71
MEAN	21.80	-16.50	-22.00	-19.70	-26.80	-25.70	-32.90
S.D.	2.10	6.70	7.05	4.60	8.70	8.10	10.60

Appendix A2. Older amplitude values at 800 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Amplitude of EOAE (SPL) for older subjects at 800 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AD	45.50	-8.90	-10.90	-4.01	-10.91	-6.30	-32.47
CL	41.41	-12.98	-21.63	-18.73	-24.15	-18.19	-24.96
HR	43.00	-11.88	-14.45	-17.01	-20.00	-14.42	-25.41
SK	38.00	-14.65	-17.35	-21.71	-33.10	-30.66	-38.41
WA	44.83	-9.80	-11.98	-10.90	-13.11	-38.78	-49.76
RD	50.83	-7.36	-12.13	-11.65	-15.66	-17.24	-24.82
BM	54.50	-18.47	-17.58	-19.95	-16.52	-30.13	-33.25
TL	40.10	-27.43	-32.52	-18.47	-23.77	-17.47	-19.48
GL	42.50	-14.88	-3.79	-8.50	-10.70	-36.95	-15.80
BR	46.67	-12.30	-19.24	-8.72	-15.64	-20.61	-27.97
PE	61.67	-9.61	-11.10	-13.20	-15.20	-9.68	-11.70
TOTAL	509.01	-148.26	-172.67	-152.85	-198.76	-240.43	-304.03
MEAN	46.27	-13.48	-15.70	-13.90	-18.07	-21.86	-27.64
S.D.	6.95	5.60	7.42	5.66	6.72	10.78	10.68

Appendix A3. Younger latency values at 800 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Latency of EOAE (ms) for younger subjects at 800 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AC	23.67	11.12	17.58	11.12	17.11	11.12	17.08
CC	20.50	11.08	17.46	11.08	17.46	11.03	17.46
GD	24.16	11.03	17.46	11.97	17.46	11.03	17.46
HA	21.75	10.96	16.06	10.89	16.54	10.88	15.99
MK	23.83	11.83	16.83	11.00	16.09	10.60	16.83
SK	21.83	11.08	17.46	10.70	17.05	10.72	17.05
VA	23.41	10.96	16.08	10.96	16.05	10.87	15.99
YJ	23.50	11.29	17.24	10.89	17.24	11.29	17.24
BH	18.33	11.20	16.96	10.56	16.96	10.56	16.96
RJ	19.16	10.17	17.00	10.14	17.00	10.13	15.43
SR	20.33	11.25	16.40	11.25	16.40	11.25	16.40
TOTAL	240.47	121.97	186.53	120.56	185.36	119.48	183.89
MEAN	21.85	11.09	16.95	10.96	16.85	10.86	16.72
S.D.	2.03	0.39	0.56	0.45	0.51	0.34	0.67

Appendix A.4 Older latency values at 800 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Latency of EOAE (ms) for older subjects at 800 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	45 II
AD	45.50	11.31	17.65	11.95	17.65	11.02	15.69
CL	41.41	12.09	18.11	11.12	17.35	11.12	17.58
HR	43.00	11.08	15.98	11.55	17.33	10.23	15.99
SK	38.00	10.03	17.21	11.91	17.34	11.29	17.09
WA	44.83	11.03	17.92	11.03	17.69	10.68	17.46
RD	50.83	12.09	17.11	12.06	17.11	12.06	17.11
BM	54.50	10.10	15.74	10.22	16.12	10.22	14.52
TL	40.10	10.20	17.34	10.20	15.72	10.64	17.00
GL	42.50	10.30	15.83	10.38	15.76	10.38	14.90
BR	46.67	11.25	17.17	11.25	17.15	11.91	17.00
PE	61.67	10.75	17.15	10.75	17.15	10.75	17.11
TOTAL	509.01	120.23	187.21	122.42	186.37	120.30	181.45
MEAN	46.27	10.93	17.02	11.13	16.94	10.94	16.50
S.D.	6.95	0.74	0.82	0.69	0.72	0.62	1.05

Appendix A5. Younger amplitude values at 1500 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Amplitude of EOAE (SPL) for younger subjects at 1500 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AC	23.67	-8.60	-15.65	-8.18	-15.65	-17.52	-24.83
CC	20.50	-4.01	-10.90	-6.16	-14.92	-14.92	-22.28
GD	24.16	-24.97	-31.55	-16.47	-27.28	-24.32	-35.72
HA	21.75	-22.41	-29.93	-24.15	-30.35	-33.70	-41.15
MK	23.83	-6.16	-12.95	-9.92	-16.77	-18.64	-28.43
SK	21.83	-4.04	-9.91	-8.78	-13.39	-16.42	-21.35
VA	23.41	-5.76	-13.35	-5.68	-13.09	-13.80	-19.88
YJ	23.50	-14.62	-23.10	-15.48	-22.48	-33.85	-43.22
BH	18.33	-23.19	-26.72	-22.90	-23.70	-18.06	-23.80
RJ	19.16	-11.51	-16.42	-12.20	-18.41	-12.48	-17.20
SR	20.33	-11.31	-18.37	-8.97	-15.25	-16.39	-23.80
TOTAL	240.47	-136.58	-208.85	-138.89	-195.64	-220.1	-301.66
MEAN	21.85	-12.45	-19.00	-12.63	-19.21	-20.00	-27.45
S.D.	2.04	7.87	7.63	6.38	5.87	7.46	8.75

Appendix A6. Older amplitude values at 1500 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Amplitude of EOAE (SPL) for older subjects at 1500 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AD	45.50	-8.07	-14.99	-11.80	-18.86	-12.07	-19.57
CL	41.41	-7.40	-13.35	-7.86	-13.43	-11.62	-17.62
HR	43.00	-6.02	-13.98	-16.48	-26.02	-26.02	-33.00
SK	38.00	-12.04	-18.79	-11.70	-18.49	12.23	-18.91
WA	44.83	-5.19	-11.08	-6.22	-13.98	-18.47	-26.20
RD	50.83	-5.21	-12.58	-9.43	-15.39	-15.43	-23.43
BM	54.50	-10.51	-15.17	-5.93	-12.23	-26.29	-30.68
TL	40.10	-8.86	-13.47	-4.96	-9.67	-9.16	-13.74
GL	42.50	-7.63	-13.99	-8.24	-12.44	-7.04	-11.48
BR	46.67	-5.05	-10.47	-4.23	-9.73	-14.17	-18.42
PE	61.67	-9.16	-12.20	-5.46	-11.50	-14.20	-20.02
TOTAL	509.01	-85.14	-104.29	-92.31	-161.74	-138.35	-233.07
MEAN	46.27	-7.74	-9.49	-8.39	-14.73	-12.57	-21.19
S.D.	6.95	2.30	9.38	3.71	4.84	10.83	6.63

Appendix A7. Younger latency values at 1500 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Latency of EOAE (ms) for younger subjects at 1500 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AC	23.67	11.16	16.92	11.15	16.91	11.13	16.42
CC	20.50	11.13	16.25	11.13	17.51	11.28	16.87
GD	24.16	11.75	16.92	11.15	18.22	11.14	18.19
HA	21.75	10.00	17.05	11.28	17.05	11.29	17.04
MK	23.83	11.13	16.25	11.13	16.24	11.12	11.52
SK	21.83	11.13	16.93	11.16	16.93	10.52	16.92
VA	23.41	11.13	17.51	11.12	17.51	11.28	16.87
YJ	23.50	11.01	17.50	11.10	16.86	11.09	18.72
BH	18.33	10.20	17.00	10.65	14.87	10.22	15.34
RJ	19.16	11.88	17.00	11.87	17.64	11.22	16.99
SR	20.33	10.44	15.76	11.08	16.39	11.07	14.92
TOTAL	240.47	120.96	185.09	122.82	186.13	121.36	179.80
MEAN	21.85	11.00	16.83	11.16	16.92	11.04	16.35
S.D.	2.04	0.58	0.53	0.28	0.88	0.34	1.97

Appendix A8. Older latency values at 1500 Hz for observation intervals 1 and 2, stimulated at 55, 45, & 35 dB SPL P.E.

Latency of EOAE (ms) for older subjects at 1500 Hz from 55-35 dB

	AGE	55 I	55 II	45 I	45 II	35 I	35 II
AD	45.50	11.13	16.85	11.13	16.90	11.13	16.89
CL	41.41	11.18	16.95	11.65	16.95	11.16	17.25
HR	43.00	11.16	17.57	11.16	17.25	11.23	16.52
SK	38.00	11.09	16.85	11.09	16.40	11.09	16.84
WA	44.83	11.16	16.94	11.16	17.25	11.15	16.92
RD	50.83	11.20	17.62	11.20	16.95	10.56	16.96
BM	54.50	10.56	16.96	10.55	16.95	10.56	16.96
TL	40.10	10.61	17.01	10.61	17.01	10.17	17.01
GL	42.50	10.64	15.10	10.64	16.38	10.63	15.78
BR	46.67	11.19	16.96	11.18	16.96	10.55	16.95
PE	61.67	11.20	16.32	11.20	16.32	11.19	16.31
TOTAL	509.01	121.12	185.13	121.57	185.32	119.42	184.39
MEAN	46.27	11.01	16.83	11.05	16.85	10.86	16.76
S.D.	6.95	0.26	0.67	0.33	0.33	0.37	0.41

APPENDIX B

Appendix B1 Univariate homogeneity of variance tests for amplitude at 800 Hz and 1500 Hz at observation interval 1 (22 cases)

800 Hz Univariate Homogeneity of Variance Test Results

Amplitude	F	p
55 I	3.609	.058
45 I	0.0205	.886
35 I	0.9053	.342

* p <.05.

1500 Hz Univariate Homogeneity of Variance Test Results

Amplitude	F	p
55 I	5.894	.015 *
45 I	.2240	.636
35 I	.2550	.614

* p <.05.

Appendix B2 Univariate homogeneity of variance tests for amplitude at 800 Hz and 1500 Hz at observation interval 2 (22 cases)

800 Hz Univariate Homogeneity of Variance Test Results

Amplitude	F	p
55 II	.0157	.900
45 II	.0254	.873
35 II	.0163	.898

* p <.05.

1500 Hz Univariate Homogeneity of Variance Test Results

Amplitude	F	p
55 II	11.81	.001 ***
45 II	1.506	.220
35 II	1.171	.279

*** p <.001.

Appendix B3 Univariate homogeneity of variance tests for latency at 1500 Hz and 1500 Hz at observation interval 1 (22 cases)

800 Hz Univariate Homogeneity of Variance Test Results

Latency	F	p
55 I	5.975	.015 *
45 I	1.848	.174
35 I	6.726	.010 **

*p <.05. **p <.01.

1500 Hz Univariate Homogeneity of Variance Test Results

Latency	F	p
55 I	4.717	.030*
45 I	0.000	.989
35 I	0.236	.627

*p <.05.

Appendix B4 Univariate homogeneity of variance tests for latency at 1500 Hz and 1500 Hz at observation interval 2 (22 cases)

800 Hz Univariate Homogeneity of Variance Test Results

Latency	F	p
55 II	12.19	0.000*
45 II	0.512	0.474
35 II	4.404	0.036*

*p <.05. ***p <.001.

1500 Hz Univariate Homogeneity of Variance Test Results

Latency	F	p
55 II	0.492	.483
45 II	7.595	.006*
35I I	17.36	.000*

**p <.01. ***p <.001.

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