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To the Graduate Council:

I am submitting herewith a thesis written by Robert Joseph Dunlop entitled "The Effect of External Ear Canal Air Pressure Variation on Bone Conduction Sensitivity in Sensorineural Hearing Loss and Normals." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Audiology.

Samuel B. Burchfield, Major Professor

We have read this thesis
and recommend its acceptance:

Accepted for the Council:

Vice Chancellor
Graduate Studies and Research

THE EFFECT OF EXTERNAL EAR CANAL AIR PRESSURE VARIATION ON BONE CONDUCTION SENSITIVITY IN SENSORINEURAL HEARING LOSS AND NORMALS

A Thesis

Presented for the

Master of Arts

Degree

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Robert Joseph Dunlop

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ABSTRACT

The effect of external ear canal air pressure variation on bone conduction sensitivity, the Gelle technique, was investigated in normal hearing, high frequency sensorineural hearing loss, and low frequency sensorineural hearing loss subjects. Group comparisons were made in order to explore the feasibility of this technique as a diagnostic indicator of Meniere's disease.

Twenty-seven subjects were examined with this procedure, measuring the threshold shift of a bone conduction signal as influenced by + 400 mm H_2O and - 400 mm H_2O of external ear canal air pressure at the test frequencies 250, 500, 750 and 1,000 Hz. The mean threshold shifts for the normal hearing and high frequency hearing loss groups were statistically compared by an analysis of variance. Threshold shifts for the low frequency hearing loss subjects were descriptively analyzed, since only three subjects were found who met the selection criteria for this group.

Previous studies have shown that air pressure variations in the external auditory canal would lower bone conduction thresholds of individuals without conductive hearing impairments, whereas no bone conduction threshold shift was observed in individuals with conductive impairments.

Therefore, it was anticipated that normal hearing and high frequency sensorineural hearing loss subjects would exhibit threshold shifts for variations of ear canal pressure since a conductive lesion

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was not suspected for these groups. However, owing to a mechanical impairment factor assumed to be operative in Meniere's disease, the low frequency hearing loss subjects were not expected to show as much threshold shift for the bone conduction tone as the other experimental groups.

As expected, the results indicated that there were no significant differences between the normal hearing and high frequency sensorineural hearing loss groups. The low frequency sensorineural hearing loss subjects, however, exhibited less threshold shift than the other experimental subjects for the + 400 mm H_2O pressure conditions at 750 and 1,000 Hz.

The results of this study have indicated that the normal hearing and high frequency sensorineural hearing loss subjects reacted essentially as a homogeneous group, revealing equivalent threshold shifts under similar air pressure variations in the external ear canal. The responses of these groups place them in the category of non-conductive involvement of the hearing apparatus.

The low frequency sensorineural hearing loss group presented slightly reduced threshold shifts at 750 and 1,000 Hz under the positive pressure condition, suggesting the presence of an associated cochlear conductive component to the hearing loss.

The trends observed in the low frequency hearing loss group should not be considered significant as the sample size was restricted. Additionally, the variability of the threshold shifts for all subjects overlapped, precluding diagnosis based on the degree of shift observed. Due to these factors, a definitive statement concerning the efficiency

of the Gelle technique as a diagnostic indicator of Meniere's disease must await further refinement of the measurement technique and additional test results.

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CHAPTER I

INTRODUCTION

In 1881, Gelle devised a measure of stapes mobility (Huizing, 1960). Employing the phenomenon discovered by Wheatstone in 1827, Gelle observed thresholds of normal hearing individuals decrease for air and bone conduction measures by increasing the pressure in the external ear canal. Further, he was able to differentiate between normal middle ear functioning and stapedial ankylosis. This same procedure applied to individuals with otosclerosis, resulted in a threshold shift for air conduction as with normals; however, bone conduction thresholds remained unaffected.

Other investigators (Huizing, 1960; Rahm, Strother and Crump, 1965; Dankbaar, 1970), utilizing the Gelle technique, also reported similar results. They further noted that this effect occurred primarily in the low frequencies (below 1,000 Hz) and was evident with both positive and negative pressures.

To understand the effect of pressure in the ear canal on hearing sensitivity, one must consider the impedance formula (Johansen, 1948; Huizing, 1960):

$$|Z| = \sqrt{r^2 + (2\pi F \cdot M - \frac{S}{2\pi F})^2} .$$

As pressure in the ear canal is increased or decreased, the stiffness (S) of the system increases. Impedance audiometry utilizes this principle for clinically measuring the acoustic impedance of the middle ear.

This increased or decreased pressure alters the resonant frequency and the impedance (Z) of the system. It was noted earlier that as the stiffness of the ear was increased the resonant frequency was raised to a higher frequency (Johansen, 1948). If the test frequency (F) is lower than the original resonant frequency of the ear, the increased stiffness (S) will raise the impedance (Z) of the ear thereby decreasing hearing sensitivity. However, if the test frequency (F) is higher than the original resonant frequency of the ear the increased stiffness will first only slightly increase the impedance, and then for even higher frequencies decrease the impedance and enhance hearing sensitivity.

In general, if the effect of air pressure on hearing sensitivity is investigated at frequencies below the resonant frequency of the human ear (2,000 - 2,500 Hz) maximally reduced hearing sensitivity should be observed. Conversely, if this effect is investigated at frequencies higher than the resonant frequency of the ear, then hearing sensitivity may only be slightly decreased or even increased at certain higher frequencies. Therefore, the maximum negative effects of pressure on hearing sensitivity can be viewed in the frequency region below 2,000 Hz.

It can be seen from the present discussion that effects of air pressure on hearing sensitivity are maximal below the resonant frequency of the ear. Also these effects are greatest in the normal functioning ear, as the conduction apparatus is forced out of its normal position by a change of pressure, resulting in decreased hearing sensitivity. However, in ears that have already undergone alterations that increase stiffness to their mechanical systems the effects of the further

addition of stiffness would be much smaller than in normal ears, resulting in threshold shifts much less than in normals. Thereby, the differences in the magnitudes of threshold shifts between subjects at similar frequencies may be due to the mechanical efficiency of the system, indicating differential effects of pressure between pathologies, which may be diagnostically valuable.

All previous studies conducted with the Gelle procedure were limited to populations with middle ear anomalies or normals, the systematic investigation of this effect in sensorineural hearing loss has not been previously attempted.

In general, sensorineural hearing loss subjects, such as those with high frequency sensorineural hearing loss as caused by noise trauma, should have functionally normal middle ear conductive systems. Thus, they should exhibit bone conduction threshold shifts under air pressure variations similar to normal hearing subjects. Conversely, subjects with low frequency sensorineural hearing loss as in Meniere's disease, should respond differentially to the effect of air pressure variations. This contention is better understood upon further consideration of the literature concerning the hearing loss in Meniere's disease.

I. HEARING LOSS IN MENIERE'S DISEASE

Meniere's disease, or endolymphatic hydrops, is an inner ear disorder caused by increased fluid volume in the membraneous labyrinth and characterized by sensorineural hearing loss, tinnitus and vertigo (Alford, 1972). Typically, the first symptom is hearing loss that fluctuates during the incipient stage and becomes progressively worse

(Schuknecht, Benitez, and Beekhuis, 1962). The audiometric configuration of this disease, during the incipient stage, is predominantly seen as a low frequency, sensorineural loss (House, 1968), which is confined almost exclusively to Meniere's patients (Dayal, Kane, and Mendelsohn, 1970). However, due to the fluctuating nature of this disorder, it is difficult to consider a single measure as a reliable indicator of the disease. Hearing sensitivity during early audiometric tests may be well within normal limits while subsequent results may show severe or even profound sensorineural hearing loss. This hiatus prompted the present study which investigated the effect of external ear canal air pressure variation on bone conduction sensitivity as an alternative to traditional audiometric tests for the identification of Meniere's disease. To accomplish this it was necessary to consider the physiological mechanisms responsible for producing the hearing loss seen in Meniere's disease.

II. PRECIPITATING FACTORS

Three physiological causes have been considered to produce the hearing loss observed in Meniere's patients: (1) sensory cell damage, (2) biochemical change, or (3) mechanical impairment of sound transmission.

Sensory Cell Damage

The first physiological consideration, sensory cell damage, implies irreversible destruction of the hair cells of the cochlea. Bekesy (1932) established that a bone-conduction tone is cancelled by

an equal sensation level air-conduction tone adjusted 180° out-of-phase. He concluded that the net effects of the modes of stimulation are the same. Therefore, bone conduction thresholds can be interpreted as estimates of the integrity of the sensorineural system. This, however, has not been supported by histologic studies of Meniere's cases in which reduced bone conduction thresholds were observed. Hallpike and Cairns (1938) revealed that the major pathological finding in Meniere's patients was gross dialation of the endolymphatic system. They also noted degeneration of the sensorineural elements, but considered this due to post-mortem autolysis. Later research (Schuknecht et al., 1962) concluded that Meniere's patients had normal populations of hair and ganglion cells, with no morphological changes in structure. As sensory cell damage has been considered irreversible, the sensorineural hearing loss cannot be explained from this position. Further, the fluctuating nature of the disorder precludes cell damage. This implies that sensory cell damage cannot account for the reduced bone conduction thresholds exhibited by Meniere's patients during the incipient stage of the disorder, and that other factors must be operative. These histologic findings led Schuknecht et al. (1962), to suggest that the hearing loss may be explained as either a biochemical or a micro-structural change.

Biochemical Change

The second factor which may cause the hearing loss in Meniere's disease is biochemical change of the cochlear fluids. Normal perilymphatic fluid has a high concentration of sodium, whereas normal endolymphatic fluid has a high concentration of potassium. Lawrence

and McCabe (1959) reported that Bekesy observed the hair cells as oil droplets when looking at the organ of Corti through Reissner's membrane. Upon breaking this membrane it was noted that the cells took on an opaque appearance and the local electrical effects were reduced. It was suggested that the contamination of the endolymph may account for the hearing loss. Attempting to answer this question, Raush (1968) and Silverstein (1971) analyzed the electrolyte contents of endolymph and perilymph in normal and pathological ears. They found the sodium/potassium balance of both the perilymphatic and endolymphatic systems in Meniere's patients were within normal limits. These findings suggest that the biochemical interchange of fluid between the systems cannot be considered as a precipitating factor of hearing loss in early Meniere's disease.

Mechanical Impairment of Sound Transmission

Mechanical impairment, the third factor to be considered as a cause of the hearing loss in Meniere's disease, has been discussed in terms of the impedance formula. Utilizing this formula to interpret hearing loss configurations, Johansen (1948) and Campbell (1950) suggested that low frequency hearing losses are caused by stiffness in the sound conduction system. Although their discussion concerned air conduction, Sala's (1973) review of the literature suggests either intra-cochlear or extra-cochlear mechanical dysfunctions may also impair bone conduction sensitivity.

Intra-cochlear dysfunction. The low frequency sensorineural hearing loss, during incipient Meniere's disease, has been considered to

arise from mechanical stiffness wholly within the cochlea (Tonndorf, 1957). It was reported that, by increasing the endolymphatic fluid volume of a cochlear model, the natural stiffness gradient of the cochlear duct was reduced. The reduced stiffness gradient resulted in decreasing responsiveness of the model to low frequency stimulation. Tonndorf also noted that further increases or decreases of endolymph volume accounted for corresponding fluctuations in the responsiveness of the model. It was concluded that the low frequency, fluctuating hearing loss, observed in early Meniere's disease was a direct consequence of endolymphatic fluid volume.

A similar study was conducted with guinea pigs (McCabe and Wolsk, 1961). Cochlear AC potentials were measured as various pressures were applied in the scala media of the anesthetized animals. As reported in a previous study (Tonndorf, 1957) it was noted that responses decreased when increased pressure was applied, and the amount of decrease was directly related to the pressure applied. It was suggested (McCabe and Wolsk, 1961) that besides the change in tension of the basilar membrane implied by Tonndorf (1957), the shearing motion of the hair cells may also be decreased due to the compression effect of the encapsulated fluid.

Extra-cochlear dysfunction. Extra-cochlear mechanical factors also have been shown to decrease bone conduction sensitivity. This observation was made in experimental stapes fixation studies with animals (Smith, 1943) and also in clinical bone conduction measures with otosclerotic patients (Carhart, 1963). Both studies reported bone

conduction thresholds were reduced under fixation. However, this effect was found to differ between humans and animals due to the resonant frequency of the ear. It was observed that the cats (Smith, 1943) demonstrated reduced responses in the middle frequency region and the humans (Carhart, 1963) in the high frequency (2,000 Hz) region. A similar pathological mechanism has been hypothesized (Mygind, 1947) to be present in cases of early Meniere's disease. It was suggested that increased intralabyrinthine volume may damp the mobility of the stapes footplate, thereby reducing vibratory action during the inertial mode of bone conduction. Mygind (1947) further indicated this would account for the low frequency loss of bone conduction sensitivity, and implied that the hearing loss in early Meniere's disease was of a cochlear conductive nature.

Prior to Mygind's hypothesis, Bekesy (1942) investigated the effect of increased intralabyrinthine pressure on stapes mobility utilizing cadaver temporal bones and cochlear models. The procedure consisted of presenting a pure tone signal to the external canal while varying the intralabyrinthine pressure with a syringe. Alterations in the vibratory pattern of the stapes were noted at the round window as a change in displacement. It was concluded that intracochlear pressures did not appear to fixate the stapes. However, Bekesy's (1942) conclusions must be interpreted cautiously as they apply to the pathological mechanism of Meniere's disease. It should be noted that the physical passage of sound through the cochlea was measured and not the effect of increased pressures on sensitivity of hearing. Also, the pressures produced in the perilymphatic spaces of cadavers and cochlear models

may not be representative of the pressures produced by the endolymphatic system in Meniere's disease.

In summary, three physiological mechanisms have been considered as contributors to the hearing loss seen in Meniere's disease. Two of these mechanisms, sensory cell damage, and biochemical alterations of the cochlear fluids, cannot justify the hearing loss during the early stages of this disorder. This has been substantiated by previous studies revealing normal populations of hair cells and normal chemical balances within the cochlea of Meniere's patients.

The third mechanism, mechanical impairment of sound transmission, appears to be the predominant factor. Experimental production of hydrops-like conditions in cochlear models and guinea pigs has indicated that altered mechanical factors can reduce hearing sensitivity. However, the presence of a mechanical dysfunction accounting for the hearing loss in humans with Meniere's disease has never been systematically investigated. Gelle, in 1881, developed a procedure for assessing conductive pathology which is based upon hearing sensitivity assessment as a function of mechanical impedance variations. The Gelle technique has been used extensively in identifying middle ear conductive pathology, yet the diagnostic prowess of this procedure has never been explored with Meniere's patients. Gelle's technique may provide an alternate diagnostic tool, to the more traditional audiometric measures, capable of identifying Meniere's disease even during periods of remission.

III. PROBLEM

The differential diagnosis of Meniere's disease has been dependent upon three subjective symptoms and one objective measure: vertigo, tinnitus, a fullness sensation in the ear, and hearing loss. Simonton (1968), however, indicated the variations of these symptoms are so great that it would be impractical to consider a single pattern as an adequate description of Meniere's disease, thus making differential diagnosis difficult. As this disease is progressive in nature, resulting in a severe sensorineural hearing loss in many cases, there is an imperative need for early detection, treatment and/or prevention. The most objective measure for determining the presence of Meniere's disease is audiometry, yet at this time there is no association between the type and degree of hearing loss and the pathology. It is therefore suggested that audiometric measures need to be sensitive to the underlying physiological mechanism of the ear during the early stages of this disease.

IV. PURPOSE

The purpose of this study was to investigate the effect of external ear canal pressure variations on bone conduction sensitivity in normal hearing, high frequency sensorineural hearing loss and low frequency sensorineural hearing loss subjects, and to explore the feasability of this test as a diagnostic indicator of Meniere's disease. The normal hearing subjects were chosen as a control to compare the results of this study with the findings of previous studies. The high frequency sensorineural hearing loss subjects were

selected as representative of sensorineural hearing loss without concomitant mechanical dysfunctions. The low frequency sensorineural hearing loss subjects were selected as representative of sensorineural hearing loss with suspected mechanical dysfunctions.

It was anticipated that subjects with normal hearing and with high frequency sensorineural hearing losses would exhibit threshold shifts comparable to normal hearing subjects examined in previous studies. Further, it was suspected that if a cochlear conductive type hearing loss was present in the low frequency sensorineural hearing loss subjects, threshold shifts for this group would be less than the other experimental subjects and similar to those observed in subjects with middle ear conductive losses in previous studies.

Specifically, the following questions were asked:

1. What is the relationship between the mean Gelle influenced bone conduction threshold shift in high frequency sensorineural hearing loss, low frequency sensorineural hearing loss, and normal hearing subjects?

2. What is the relationship between the mean Gelle influenced bone conduction threshold shift and the external ear canal pressure conditions of + 400 mm H_2O and - 400 mm H_2O?

3. What is the relationship between the mean Gelle influenced bone conduction threshold shift among the test frequencies of 250, 500, 750, and 1,000 Hz?

CHAPTER II

METHODS AND PROCEDURES

The effect of external ear canal air pressure variation on bone conduction sensitivity was investigated in three groups of subjects examined at four audiometric frequencies over three conditions of air pressure. The experimental groups consisted of subjects with (1) normal hearing, (2) high frequency sensorineural hearing loss, and (3) low frequency sensorineural hearing loss. The test frequencies were 250, 500, 750, and 1,000 Hz. Bone conduction thresholds were determined under the following conditions of air pressure in the external ear canal: (1) 0 mm H_2O, (2) + 400 mm H_2O, and (3) - 400 mm H_2O.

I. SUBJECTS

Twenty-seven subjects were selected from the Audiology Center case files of The University of Tennessee Hearing and Speech Center, and from local otolaryngology offices. Selection criteria were based on age and clinical manifestations. The effects of age differences were minimized by restricting the subject population of the study within the age range of 20 to 55 years. The experimental subjects were classified into three groups according to the following criteria for the test ear:

Group 1. Normal Hearing Subjects

A. History free of any significant ear pathology.

B. Air and bone conduction thresholds no worse than 15 dB HTL (ANSI, 1969) for the octave frequencies of 250 through 8,000 Hz.

C. Air-bone gaps no greater than 10 dB at any one frequency.

D. Speech discrimination scores of 90 percent or better.

E. Normal tympanogram tracings.

Group 2. High Frequency Sensorineural Hearing Loss Subjects

A. History free of significant ear pathology, with the exception of noise exposure.

B. Air and bone conduction thresholds no worse than 15 dB HTL for the frequency range 250 to 1,000 Hz, with a sloping configuration no better than 30 dB at 4,000 Hz.

C. Air-bone gaps no greater than 10 dB at any one test frequency.

D. Speech discrimination scores of 80 percent or better.

E. Normal tympanogram tracings.

F. At least 14 hours shall have transpired since last exposure to hazardous noise (OSHA, 1974).

Group 3. Low Frequency Sensorineural Hearing Loss Subjects

A. History free of any significant ear pathology, with the exception of complaints of tinnitus, vertigo, or sensation of fullness in the ear (Alford, 1972).

B. Air and bone conduction thresholds within the envelope of 35-45 dB at 250 and 500 Hz, 20-40 dB at 1,000 Hz and no worse than 15 dB for the test frequencies of 2,000, 4,000, and 8,000 Hz.

C. Air-bone gaps no greater than 10 dB at any one test frequency.

D. Speech discrimination scores of 90 percent or better.

E. Normal tympanogram tracings.

Twelve subjects were accepted as meeting the selection criteria for the high frequency sensorineural hearing loss group and twelve for the normal hearing group. Three subjects were acquired who fit the selection criteria for the low frequency sensorineural hearing loss group. The mean hearing losses for each of these groups are displayed in Figure 1.

The relatively small number of subjects comprising the low frequency hearing loss group was due to two factors. First, their accessability was restricted as Meniere's disease has been seldom seen in the pre-clinical/incipient stage, and secondly, the stringent selection criteria for this study further reduced those who were available.

Screening

Each subject was audiometrically screened prior to the experimental procedure. The testing environment (Figure 2) consisted of the subject seated in an audiometric test booth (IAC, model 1203A-CT-L) with a noise floor of 27 dBA. Pure tone thresholds (re: ANSI, 1969) were obtained using a wide range audiometer (Grason-Stadler, model

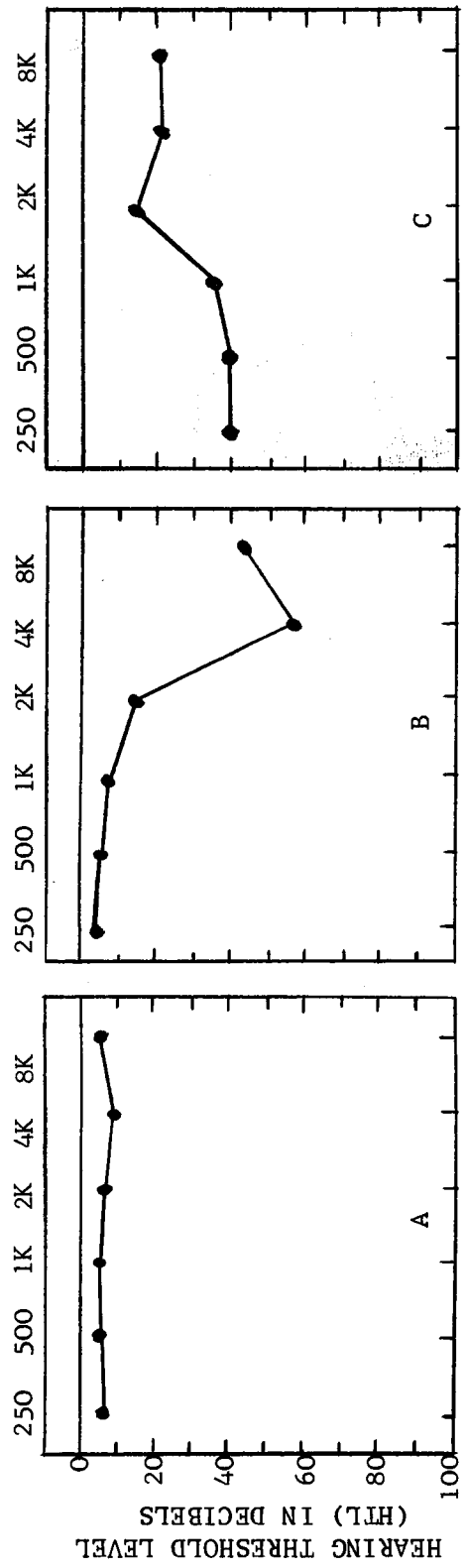

FIGURE 1. Mean hearing losses for (A) normal hearing subjects, (B) high frequency sensorineural hearing loss subjects, and (C) low frequency sensorineural hearing loss subjects.

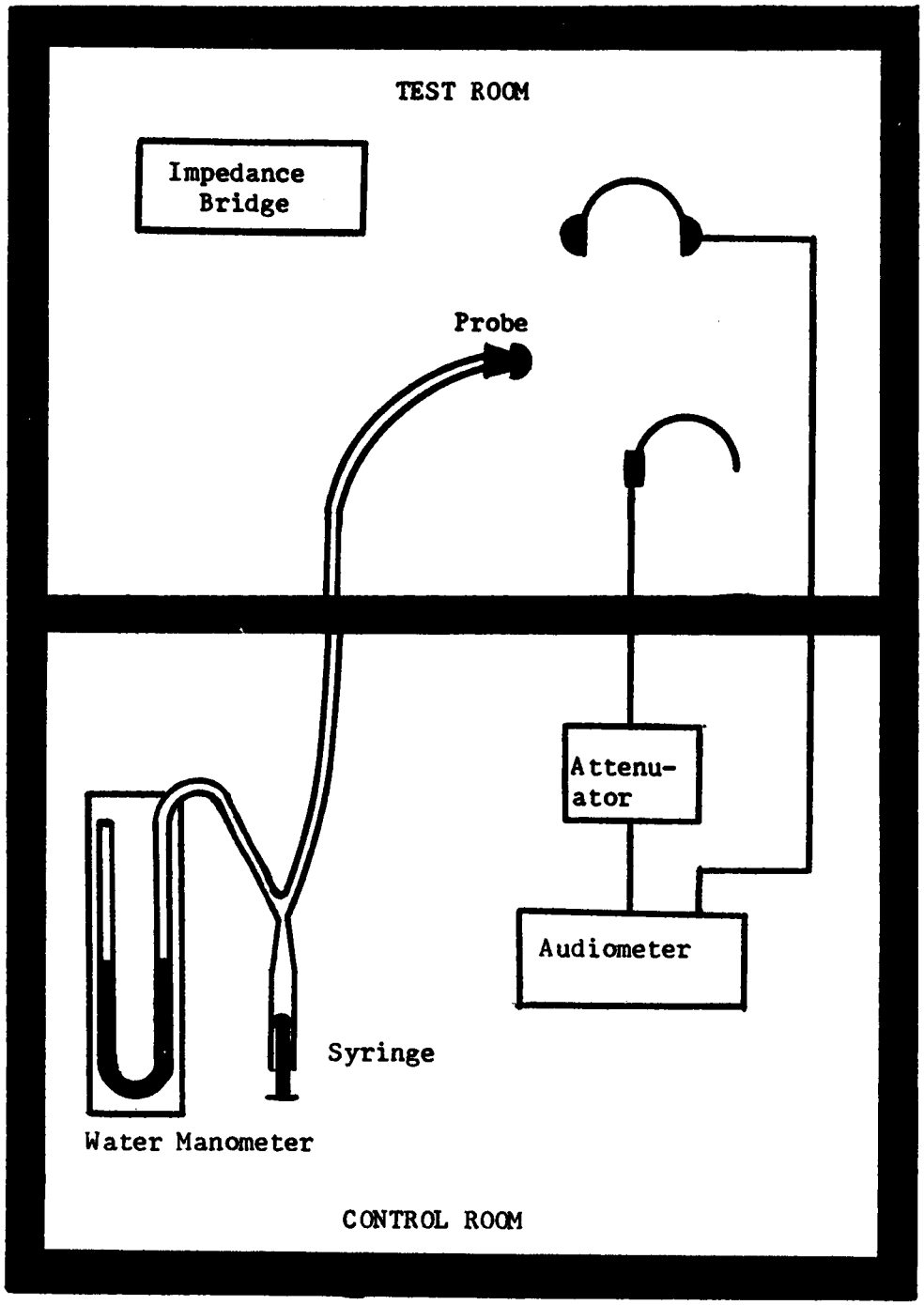

Figure 2. Audiometric testing environment.

1704), TDH 50 headphones mounted in MX 41/AR cushions and a Radioear B-70-A bone conduction vibrator. The Hughson-Westlake technique (Carhart and Jerger, 1959) was utilized for determining thresholds.

The Central Institute for the Deaf (CID) auditory tests W-2 and W-22 provided stimuli to determine speech reception thresholds and speech discrimination scores (Hirsh et al., 1952). Both measures were made according to the "Monitored Live Voice" procedures recommended by Berger (1971).

Masking of the non-test ear was conducted for all bone conduction measures and whenever the air conduction stimulus to the test ear equaled or exceeded the bone conduction threshold of the opposite ear by 40 dB (Sanders and Rintleman, 1964). Narrow band masking stimuli was provided by the audiometer. Mashing procedures followed those recommended by Hood (1960).

Huizing (1960) indicated that the Gelle effect may be decreased in individuals with middle ear impairments. Therefore, tympanometry was performed on all subjects to preclude middle ear anomalies not detected through pure tone audiometry. Utilizing an impedance bridge (American Electromedics, model 83) tympanograms were plotted, indicating the impedance of the middle ear transmission system as a function of air pressure variation at intervals of 50 mm H_2O from + 200 mm H_2O to - 300 mm H_2O.

II. EXPERIMENTAL DESIGN

Apparatus

Measurement of the Gelle effect for bone conduction stimuli was made with the subject seated in the audiometric booth. Pure tone stimuli were presented from the audiometer via the bone vibrator. Pilot data indicated that the occlusion effect forced thresholds below the lower limits of the audiometer (-10dB) at the lowest test frequencies. This was corrected by adding a 20 dB attenuator to the circuit, allowing threshold measurement to - 30 dB HTL. Masking stimuli were presented to the non-test ear through the TDH 50 earphone in an MX 41/AR cushion. The air pressure variations were applied in the external ear canal of the test ear with the ear occluded by a probe. An eight foot piece of polyetheline tubing was attached to the probe and led through the wall of the test room, into the control room. At this point the tubing was connected in parallel with a water manometer, scaled from + 500 mm H_2O to - 500 mm H_2O.

Calibration

Audiometer calibration was conducted with the Bruel and Kjaer precision sound level meter (model 2203), octave filter set (model 1613), condensor microphone (model 4132), artificial ear (model 4152), and artificial mastoid (model 4930), in the isolated test booth. Measures which did not conform to ANSI (1969) standards for air conduction or HAIC norms (dB re: 1 dyne) for bone conduction, were corrected. A precise linearity check of the bone conduction circuitry was conducted at 2 dB increments for the test frequencies of 250, 500, 750,

and 1,000 Hz. Increments were found to be within $\pm$ .3 dB accuracy.

Calibration of the manometer was determined by placing a second manometer (scaled in mm H_2O) in the test room, at the probe end of the pressure system. Increased and decreased pressure changes were equal for both manometers in their excursion with no observable delay.

Procedure

Threshold shift of the bone conduction tone was measured as a function of increased and decreased pressure in the external ear canal. The bone vibrator was positioned over the mastoid of the test ear, with the non-test ear under the masking earphone. A probe was selected, so as to provide a firm but comfortable seal of the test ear canal, and attached over the nozzle of the pressure assembly. This was in turn firmly inserted into the ear canal of the test ear.

The subject was instructed to ignore the masking signal to the non-test ear and the changes of pressure in the ear canal of the test ear. He was also encouraged to listen carefully for the pure tone stimulus, and to indicate when he first heard it by depressing the button on the hand switch.

Prior to testing, the pressure in the ear canal was varied between the positive and negative pressure peaks to ensure that a leak-proof seal had been obtained. If a leak was evident, the probe tip was either reinserted or replaced.

Testing was conducted in three steps. First, masking stimuli were presented to the non-test ear at a level of 30 to 50 dB HTL, depending upon the threshold of the non-test ear. To prevent any

possibility of crossover, masking was continued through the test and increased whenever crossover was suspected. Secondly, the effects of order of presentation were counter-acted by presenting the frequency and pressure conditions following a calculated random order (Appendix, Table 2). Finally thresholds were determined for the test tone. A study by Dankbaar (1970) indicated that the mean threshold shift in normals was on the order of 10 to 15 dB. In order to measure the small effects of the Gelle test, threshold for this study was determined using 2 dB increments. Once threshold was established, the pressure condition was readjusted following the randomized order indicated. This procedure was followed until thresholds were recorded for each pressure condition at each test frequency.

III. ANALYSIS

Threshold shift was determined as the difference between the relative threshold at 0 mm H_2O and the threshold at + 400 mm H_2O for positive effects, and between 0 mm H_2O and threshold at - 400 mm H_2O for negative effects. Individual threshold shifts for positive and negative pressures for all groups were obtained and compared to determine if the pathological conditions caused a differential response to this criterion measure.

The normal hearing and high frequency sensorineural hearing loss groups were statistically compared in a three-factor mixed design with repeated measures on two factors (Bruning and Kintz, 1968). Performance of the low frequency sensorineural hearing loss subjects was

descriptively compared as only three subjects were found who fit the selection criteria for this group.

Reliability of the Gelle influenced bone conduction threshold shifts was established by re-examining three subjects from the normal hearing and high frequency loss groups and two subjects from the low frequency loss group, approximately one week following the initial test. A Pearson Product Moment correlation was computed between the test and retest scores for each of the groups. The results of this analysis revealed a significant correlation ($r = +.6862$; $r_{.001} = +.6524$; $df = 20$) for the high frequency hearing loss group, whereas the test-retest reliability for the normal ($r = +.3325$) and low frequency hearing loss ($r = +.2429$) groups approached significance at the .5 alpha level. The relatively low correlation for the normal and low frequency groups was not unexpected, as thresholds are known to vary clinically as much as 5 dB from test to retest.

CHAPTER III

RESULTS AND DISCUSSION

Threshold shifts for the subjects examined in this study are presented in the Appendix under Table 3 (Normal hearing subjects), Table 4 (High frequency sensorineural hearing loss subjects), and Table 5 (Low frequency sensorineural hearing loss subjects). In order to answer the three experimental questions, the data were statistically analyzed in a treatment by treatment by subjects design with a chosen alpha level of significance of .05. Table 1 summarizes the analysis which considered two of the experimental groups (normal hearing and high frequency hearing loss), the external ear canal air pressure variations and the test frequencies. The low frequency sensorineural hearing loss group was descriptively compared to the other groups.

It should be noted that throughout this discussion, references to threshold shift, unless otherwise specified, indicated that the hearing threshold under either positive or negative pressure conditions was worse than the threshold for ambient pressure.

I. GROUP EFFECTS

The first experimental question considered the three experimental groups:

(1) What is the relationship between the mean Gelle influenced bone conduction threshold shift in high frequency sensorineural hearing loss, low frequency sensorineural hearing loss and normal hearing subjects?

TABLE 1. Summary of analysis of variance for mean Gelle influenced bone conduction threshold shift for groups (high frequency sensorineural hearing loss and normal hearing), pressures (+400 mm H_2O and -400 mm H_2O), and frequencies (250, 500, 750, and 1,000 Hz).

Source	SS	df	ms	F	P
Total	6488.48	191	--	--	--
Between Subjects	1741.98	23	--	--	--
Between Groups	150.52	1	150.52	2.08	--
Error $_b$	1591.46	22	72.34	--	--
Within Subjects	4746.50	168	--	--	--
Pressure	42.18	1	42.18	1.48	--
Frequency	61.39	3	20.46	.54	--
Group X Pressure	96.37	1	96.37	3.37	--
Group X Frequency	255.42	3	85.14	2.24	--
Pressure X Frequency	114.05	3	38.02	2.54	--
G X P X F	48.88	3	16.29	1.09	--
Error $_1$	629.01	22	28.59	--	--
Error $_2$	2511.69	66	38.06	--	--
Error $_3$	987.51	66	14.96	--	--

It was anticipated that normal hearing subjects and those with high frequency sensorineural hearing losses would exhibit threshold shifts comparable to normal hearing subjects examined in previous studies. The middle ear mechanism in these subjects was suspected to be functionally normal, therefore threshold shifts should be in the range of 5 to 15 dB (Stephens and Arnold, 1968; Dankbaar, 1970). Inspection of Table 1 reveals that the subjects with high frequency sensorineural hearing loss and those with normal hearing did not differ significantly in their response to the Gelle bone conduction test ($F = 2.08$; $F_{.05} = 4.30$; $df = 1/22$). The mean threshold shifts for the experimental groups were -13.02 for the high frequency hearing loss group and -11.25 for the normal hearing group. Since these results agree with previous findings (Stephens and Arnold, 1968; Dankbaar, 1970) for normal hearing subjects, the original contention that these two groups would be homogeneous is supported.

It was anticipated that if a cochlear conductive pathology was present in Meniere's disease, threshold shifts for this group would be similar to those observed in subjects with middle ear conductive losses in previous studies. Earlier studies (Rasmussen, 1948; Huizing, 1960; Dankbaar, 1970) have indicated that subjects with conductive hearing losses would show a reduced threshold shift for the Gelle procedure at all frequencies. Usually these shifts were in the range of 0 to -2 dB (Dankbaar, 1970). The mean threshold shift observed for the low frequency hearing loss subjects in this study was -10.17. Contrary to original expectations, this also fell within the range of observed shifts for normal hearing subjects. Since the mean threshold shifts

for the low frequency group were greater than the shifts reported for conductive pathologies, the physiological mechanisms responsible for these hearing losses do not appear to produce equal effects in the middle ear. However, it was noted that the mean threshold shift for this low frequency hearing loss group was slightly less than the other groups examined. This trend was best illustrated in the results for the pressure and frequency conditions.

II. DIFFERENTIAL EFFECTS OF PRESSURE

The second experimental question concerns the effects of the positive and negative external ear canal pressure variations:

(2) What is the relationship between the mean Gelle influenced bone conduction threshold shift and the external ear canal pressure conditions of + 400 mm H_2O and - 400 mm H_2O?

The effects of positive and negative air pressure variations for the normal hearing and high frequency hearing loss subjects were also considered by the analysis of variance. Table 1, page 23, reveals no significant differences ($F = 1.48$; $F_{.05} = 4.30$; $df = 1/22$) between the pressure conditions for these groups. Previous studies have conflicted in their observations concerning the differential effects of positive versus negative pressures. Arnold and Schindler (1963) and Stephens and Arnold (1968) indicated that greater threshold shifts were observed for negative pressures than for positive pressures. Huizing (1960) however, has indicated a greater effect for the positive pressures. These differences are not significant, as reported advantages of either positive or negative pressures rarely exceed 2 or 3 dB.

The mean threshold shifts for the high frequency hearing loss, low frequency hearing loss and normal hearing groups are displayed in Figure 3. This figure reveals that the low frequency hearing loss group had approximately 4 dB less threshold shift than the other groups for positive pressures. This mean shift was also 4 dB less than that produced by negative pressures in the same group. Although this finding was suggestive of a differential effect of pressure, it was not altogether surprising. Earlier studies (Rasmussen, 1948; Dankbaar, 1970) indicated that no pressure differences were noted for subjects with ossicular fixation. However, in this study we are probably dealing with a dampened system, not one rigidly fixed. If we consider the increased volume in the cochlea to express itself outward against the oval and round windows, then an opposing force such as positive pressure would have less of an effect than a complimentary force such as negative pressure. If this is a dampened system, as opposed to a fixated one, then a differential effect may be also noted for the test frequencies.

III. FREQUENCY EFFECTS

The final experimental question to be considered in this study concerns the effect of the test frequencies:

(3) What is the relationship between the mean Gelle influenced bone conduction threshold shift among the test frequencies of 250, 500, 750, and 1,000 Hz?

Previous studies (Huizing, 1960; Rahm et al., 1965) have shown the threshold shift for the Gelle technique to be frequency specific. A greater mean shift was noted to occur in the lower frequency range

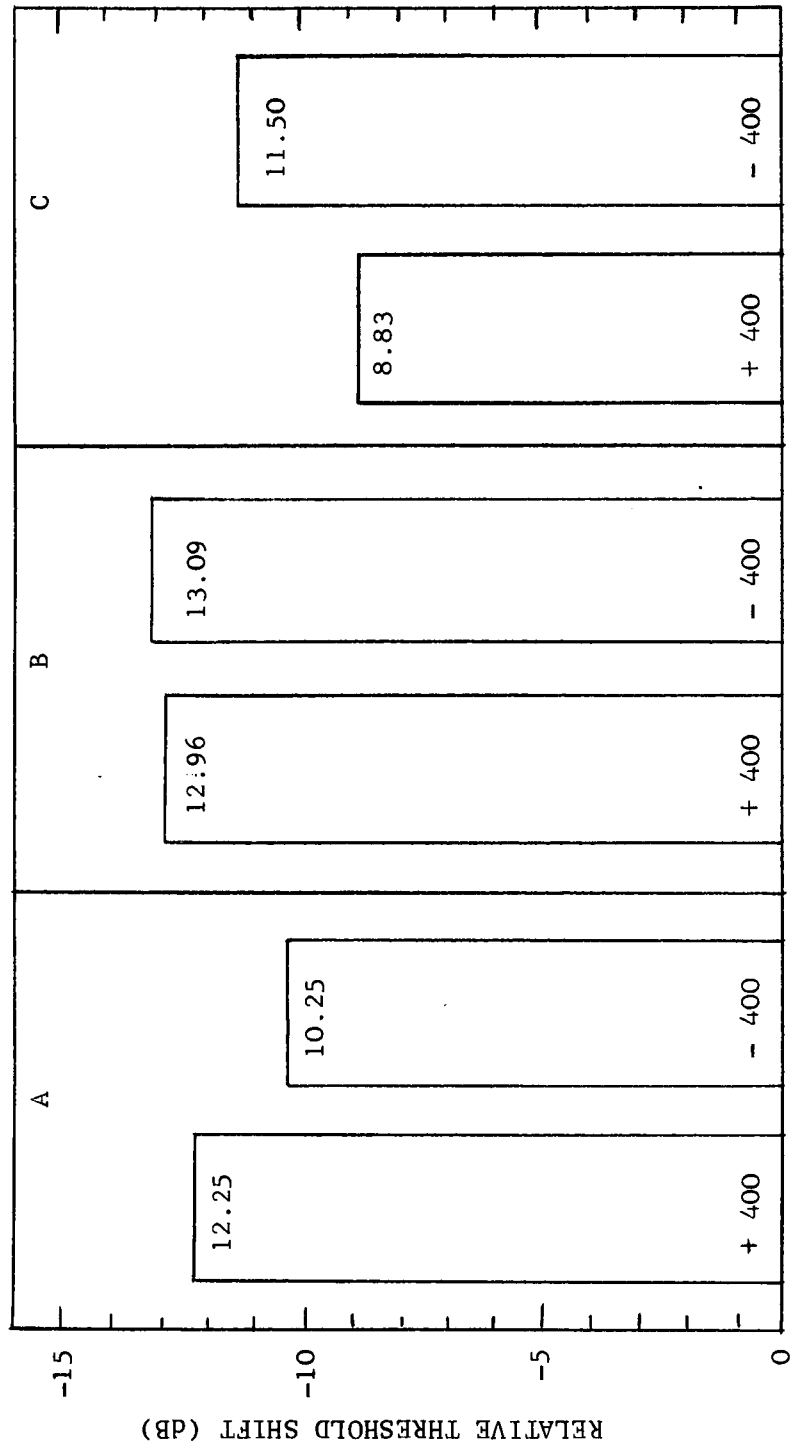

FIGURE 3. Mean Gelle influenced threshold shifts for (A) normal hearing subjects, (B) high frequency sensorineural hearing loss subjects, and (C) low frequency sensorineural hearing loss subjects for positive (+ 400 mm H_2O) and negative (- 400 mm H_2O) external ear canal air pressure variations.

between 500 and 800 Hz, with the effect decreasing on either side of this range. The shifts observed in this study did not support these findings. Statistical analysis (Table 1, page 23) between the high frequency hearing loss and normal hearing groups indicated no significant differences ($F = .54$; $F_{.05} = 2.76$; $df = 3/66$) in the mean threshold shifts for the various test frequencies. The mean threshold shifts for each of these groups at each of the test frequencies was displayed in Figure 4. The observed threshold shifts for this study were found to be in accord with the conclusions of Stephens and Arnold (1968), who also noted the absence of frequency specific effects. This they attributed to the fact that masking was not consistently used in the previous studies.

Further, examination of Figure 4 suggested that the low frequency hearing loss group behaved in a frequency specific manner. The greatest difference was noted at 1,000 Hz, where the low frequency group shifted 4.63 dB less than the other groups. This trend was emphasized further in Figure 5, where the effect was most marked for positive pressures. Here the low frequency hearing loss group had 5 to 8 dB less threshold shift than either the normal hearing or the high frequency hearing loss groups at 750 Hz, and 4 to 9 dB less threshold shift at 1,000 Hz.

These results were suggestive of an increased mechanical impairment for the low frequency hearing loss group. Two considerations lend support to this contention. First, as indicated by Johansen (1948) mechanical impedance has been known to be frequency specific, and secondly, the nature of the Gelle technique has been to reveal

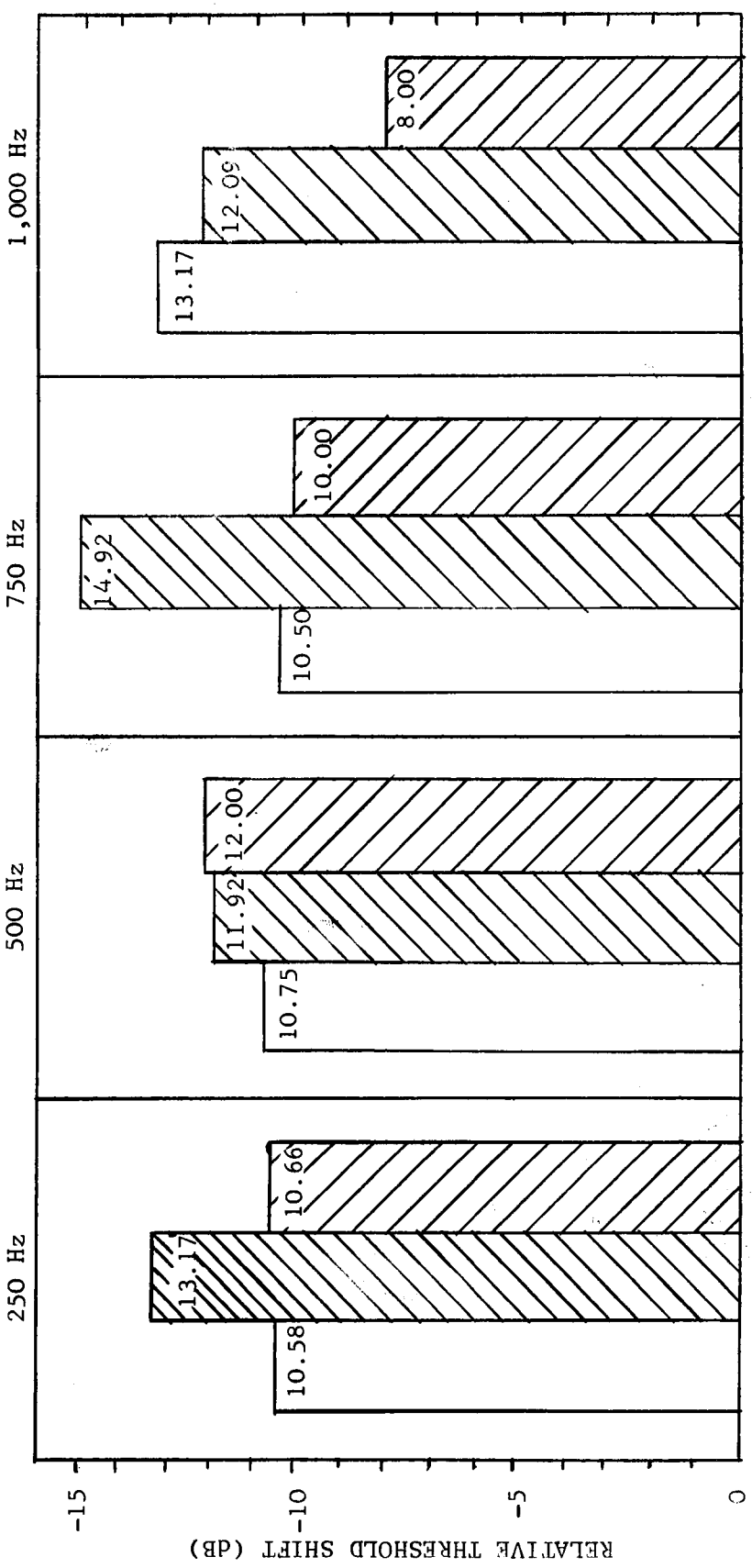

FIGURE 4. Mean Gelle influenced threshold shifts for (□) normal hearing subjects, (▨) high frequency sensorineural hearing loss subjects, and (▧) low frequency sensorineural hearing loss subjects, for the test frequencies 250, 500, 750, and 1,000 Hz.

FIGURE 5. Mean Gelle influenced threshold shifts for (□) Normal hearing subjects, (▨)High frequency sensorineural hearing loss subjects, and (▧) Low frequency sensorineural hearing loss subjects for (A) positive and (B) negative pressures at the test frequencies 250, 500, 750, and 1,000 Hz.

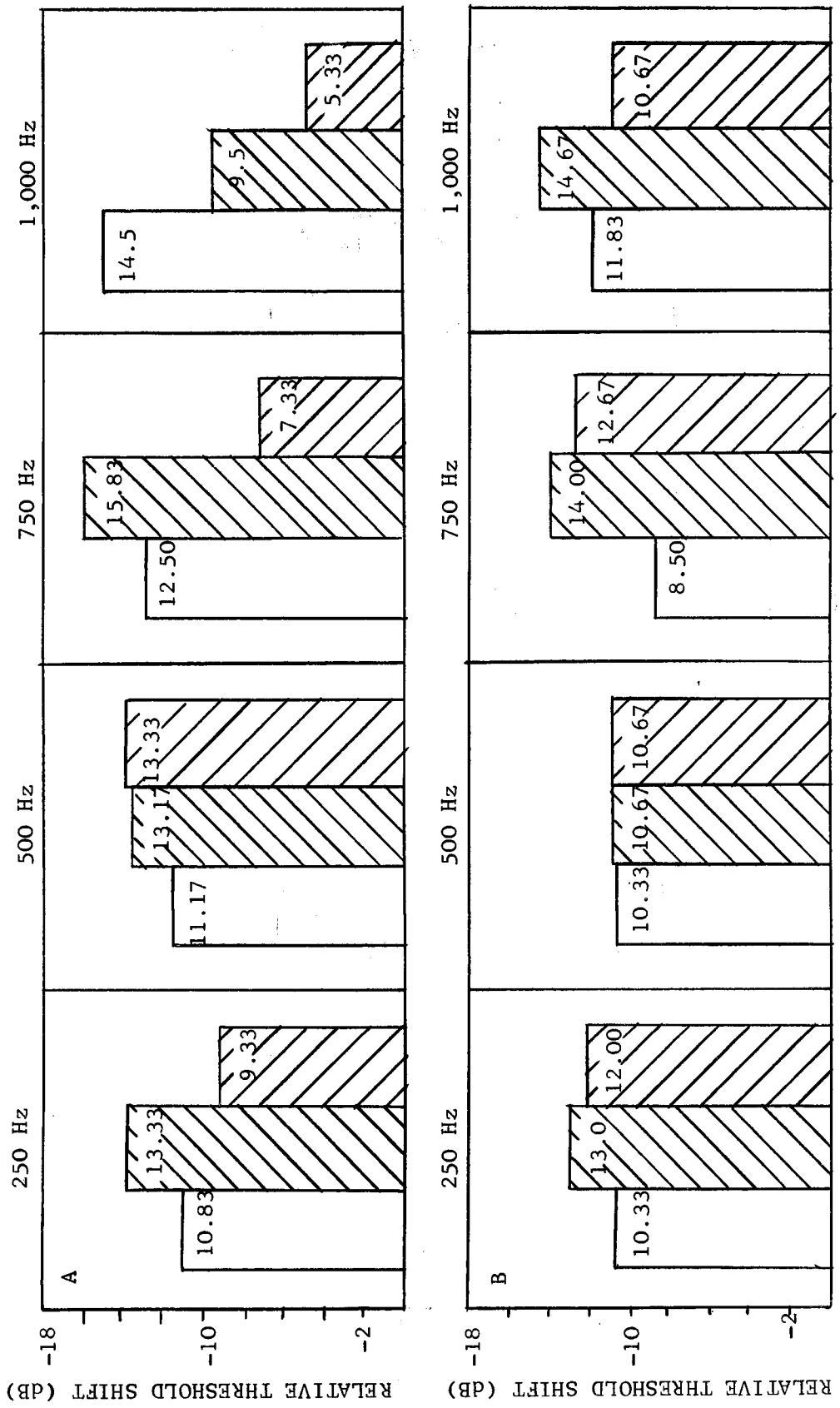
250 Hz
500 Hz
750 Hz
1,000 Hz
A
B
13.33
10.83
9.33
13.33
13.17
11.17
15.83
12.50
7.33
14.5
9.5
5.33
13.0
10.33
12.00
10.33
10.67
10.67
14.00
8.50
12.67
14.67
11.83
10.67
-18
-10
-2
RELATIVE THRESHOLD SHIFT (dB)

FIGURE 5.

conductive impairments as observed by reduced bone conduction threshold shifts under ear canal air pressure variations. Since the pathological lesion in Meniere's disease has been considered to lie wholly within the cochlear capsule, it is indicated that the vibratory action of the middle ear may have been influenced by the endolymphatic fluid volume increase. Thus the consideration of an inner ear conductive hearing loss should not be untenable.

IV. DISCUSSION

The results of this study have noted slight differences between the low frequency sensorineural hearing loss group and the other groups examined. The magnitudes of the threshold shifts for the normal hearing and high frequency sensorineural hearing loss subjects were similar to normals in previous studies. Thus, suggesting the presence of essentially normal functioning conductive systems. Conversely, the low frequency sensorineural hearing loss subjects revealed slightly reduced threshold shifts at 750 and 1,000 Hz for the positive pressure condition. This suggested the presence of a functionally different mechanical system than the normal hearing or high frequency hearing loss subjects. However, their responses were not as noteably reduced as those observed in subjects with middle ear anomalies associated with conductive hearing losses.

These differences were believed due to the variations in the mechanical factors related to the hearing loss. In normal hearing and high frequency sensorineural hearing loss subjects the conductive mechanism was essentially normal, therefore threshold shifts were in

the ranges expected. However, conductive hearing losses in which the middle ear mechanisms were directly affected by anomalies associated with the hearing loss, a great reduction in the threshold shifts were observed. Considering the primary pathological mechanism in Meniere's disease has been located in the cochlear capsule, then only secondary effects (the influence of increased endolymphatic fluid volume on the middle ear) will alter the mechanical properties of the conductive mechanism. Thus, this secondary effect may yield only a minor alteration of the mechanical functioning of the conductive system as observed in minimal reduction of threshold shifts.

Although trends were noted, several shortcomings in this study limit the usefulness of the data. In reference to the low frequency sensorineural hearing loss group, the sample population for this study was restricted to three subjects, therefore considering the observed trends as representative of the Meniere's population at large would be highly premature. Also, the subjective nature of this technique led to a wide range of threshold shifts as noted by the standard deviation scores (Appendix, Tables 3, 4, and 5). This would cause considerable difficulty in clinical diagnosis as scores for different pathological conditions would greatly overlap. Considering these difficulties, the use of the Gelle technique as a diagnostic indicator for Meniere's disease would not be warranted at this time, however, with refinement of this procedure and/or the use of more objective measures, the underlying considerations may be diagnostically useful.

Such objective measures might include impedance audiometry or electro-cochleography. In view of the nature of these tests, the

variability observed with the more subjective measures may be reduced revealing the basic effects of the pressure variations on hearing sensitivity.

With these considerations in mind, the results of this study may open new avenues for the differential diagnosis of the Meniere's population.

CHAPTER IV

SUMMARY AND CONCLUSIONS

The effect of external ear canal air pressure variation on bone conduction sensitivity, the Gelle technique, was investigated in normal hearing, high frequency sensorineural hearing loss, and low frequency sensorineural hearing loss subjects. Group comparisons were made in order to explore the feasibility of this technique as a diagnostic indicator of Meniere's disease.

Twenty-seven subjects were examined with this procedure, measuring the threshold shift of a bone conduction signal as influenced by + 400 mm H_2O and - 400 mm H_2O of external ear canal air pressure at the test frequencies 250, 500, 750, and 1,000 Hz. The mean threshold shifts for the normal hearing and high frequency hearing loss groups were statistically compared by an analysis of variance. Threshold shifts for the low frequency hearing loss subjects were descriptively analyzed, since only three subjects were found who met the selection criteria for this group.

Previous studies have shown that air pressure variations in the external auditory canal would lower bone conduction thresholds of individuals without conductive hearing impairments, whereas no bone conduction threshold shift was observed in individuals with conductive impairments.

Therefore, it was anticipated that normal hearing and high frequency sensorineural hearing loss subjects would exhibit threshold

shifts for variations of ear canal pressure since a conductive lesion was not suspected for these groups. However, owing to a mechanical impairment factor assumed to be operative in Meniere's disease, the low frequency hearing loss subjects were not expected to show as much threshold shift for the bone conduction tone as the other experimental groups.

As expected, the results indicated that there were no significant differences between the normal hearing and high frequency sensorineural hearing loss groups. These subjects reacted essentially as a homogeneous group, revealing equivalent threshold shifts under similar air pressure variations in the external ear canal. The magnitude of their responses place them in the category of non-conductive involvement of the hearing apparatus.

The low frequency sensorineural hearing loss subjects, however, exhibited less threshold shift than the other experimental subjects for the + 400 mm H_2O pressure condition at 750 and 1,000 Hz. These results suggested the presence of an associated cochlear conductive component to the hearing loss.

The trends observed in the low frequency hearing loss group should not be considered significant as the sample size was restricted. Additionally, the variability of the threshold shifts for all subjects overlapped, precluding diagnosis based on the degree of shift observed. Due to these factors, a definitive statement concerning the efficiency of the Gelle technique as a diagnostic indicator of Meniere's disease must await further refinement of the measurement technique and additional test results.

BIBLIOGRAPHY

BIBLIOGRAPHY

Alford, B. R. Committee on hearing and equilibrium; report of subcommittee on equilibrium and it's measurement; Meniere's disease: criteria for diagnosis and evaluation of therapy for reporting. Trans. Amer. Acad. Opth. & Otol., 76, 1462-1464 (1972).

American National Standards Institute. American National Standard Specifications for Audiometers. (S3.6-1969) New York: ANSI, 1970.

Arnold, G. E., and Schindler, P. Gelle test with Bekesy audiometry. Acta. Otolaryng., 56, 33-50 (1963).

Bekesy, G. von. Zur theorie des horens bei der schallaufnahme durch knochenleitung. Ann. Physik., 13, 111-136 (1932).

Bekesy, G. von. Uber die schwingungen der schneckentrennwand beim praparat und ohrenmodell. Akustische Zeitschrift, 173-186 (1942).

Berger, K. W. Speech audiometry. In D. A. Rose (Ed.), Audiological Assessment. Englewood Cliffs: Prentice-Hall, Inc., 207-240 (1971).

Bruning, J. L., and Kintz, B. L. Computational Handbook of Statistics. Glenview: Scott, Foresman and Co. (1968).

Campbell, P. A. The importance of the impedance formula in the interpretation of audiograms. Trans. Amer. Acad. Ophth. & Otol., 245-251 (Jan.-Feb., 1950).

Carhart, R. Effects of stapes fixation on bone-conduction response. In H. F. Schuknecht (Ed.), International Symposium on Otosclerosis. Boston: Little, Brown and Co., 175-197 (1962).

Carhart, R., and Jerger, J. F. Preferred method for clinical determination of pure-tone thresholds. J. Speech Hearing Dis., 24, 330-345 (1959).

Dankbaar, W. A. The diagnostic value of Gelle's test. Acta. Otolaryng, 69, 266-272 (1970).

Dayal, V. S., Kane, N., and Mendelsohn, M. Patterns of pure tone hearing loss. Acta Otolaryng., 69, 329-332 (1970).

Hallpike, C. S., and Cairns, H. Observations on the pathology of Meniere's disease. J. Laryng. Otol., 53, 625-655 (1938).

Hirsh, I. J., Davis, H., Silverman, S. R., Reynolds, Elizabeth G., Eldert, Elizabeth, and Benson, R. W. Development of materials for speech audiometry. J. Speech Hearing Dis., 17, 321-337 (1952).

Hood, J. D. The principles and practice of bone-conduction audiometry. Laryngoscope, 70, 1211-1228 (1960).

House, W. F. A theory of the production of symptoms of Meniere's disease. In J. L. Pulec (Ed.), Otolaryngologic Clinics of North America; Meniere's Disease. Philadelphia: W. B. Saunders Co., 441-446 (1968).

Huizing, E. H. Bone-conduction; the influence of the middle-ear. Acta. Otolaryng., Supp. 155, 18-34 (1960).

Johansen, H. Relation of audiograms to the impedance formula. Acta. Otolaryng., Supp. 74, 65-75 (1948).

Lawrence, M., and McCabe, B. F. Inner-ear mechanics and deafness. J. Amer. Med. Ass., 14, 119-124 (1959).

McCabe, B. F., and Wolsk, D. Experimental inner ear pressure changes. Ann. Otol. Rhin. & Laryng., 70, 541-555 (1961).

Mygind, S. H. Experimental histological studies on the labyrinth: correlation of our histological and clinical experiments and observations; conclusive review. Acta. Otolaryng., 35, 108-129 (1947).

Occupational Safety and Health Administration. Occupational noise exposure; proposed requirements and procedures. Federal Register, 39, 207, 37773-37778 (1974).

Rahm, W. E., Strother, W. F., and Crump, J. F. The effects of pressure in the external auditory meatus. Ann. Otol., 65, 656-665 (1965).

Rasmussen, H. Studies on the effect on the air conduction and bone conduction from changes in the meatal pressure in normal subjects and otosclerotic patients. Acta. Otolaryng., Supp. 74, 54-64 (1948).

Raush, S. Biochemical aspects of the pathogenesis of Meniere's disease. In J. L. Pulec (Ed.), Otolaryngologic Clinics of North America: Meniere's Disease. Philadelphia: W. B. Saunders Co., 369-374 (1968).

Sala, O. Clinical significance and diagnostic value of auditory bone threshold. Acta. Otolaryng., 75, 152-155 (1973).

Sanders, J. W., and Rintleman, W. F. Masking in audiometry. Arch. Otolaryng., 80, 541-556 (1964).

Schuknecht, H. F., Benitez, J. T., and Beekhuis, J. Further observations on the pathology of Meniere's disease. Ann. Oto. Rhino. & Laryng., 71, 1039-1053 (1962).

Silverstein, H. Inner-ear fluid proteins in acoustic neuroma, Meniere's disease, and Otosclerosis. Ann. Otol. Rhino. & Laryng., 80, 27-35 (1971).

Simonton, K. M. Menieres disease: natural history. In J. L. Pulec (Ed.), Otolaryngologic Clinics of North America: Meniere's Disease. Philadelphia: W. B. Saunders Co., 423-432 (1968).

Smith, K. R. Bone conduction during experimental fixation of the stapes. J. Exp. Psychol., 33, 96-107 (1943).

Stephens, Charlene B., and Arnold, G. E. Audiometric Gelle test with a new transducer. J. Speech Hearing Res., 11, 411-427 (1968).

Tonndorf, J. The mechanism of hearing loss in early cases of endolymphatic hydrops. Ann. Otol. Rhinol. & Laryng., 66, 766-784 (1957).

APPENDIX

TABLE 2. Calculated random order of presentation of frequency and pressure conditions.

	Presentation 1	Presentation 2	Presentation 3	Presentation 4
#1.	250 - A +	500 A + -	750 + - A	1,000 - A +
#2.	500 A + -	750 + - A	1,000 - A +	250 A + -
#3.	750 + - A	1,000 - A +	250 A + -	500 + - A
#4.	1,000 - A +	250 A + -	500 + - A	750 - A +
#5.	250 A + -	500 + - A	750 - A +	1,000 A + -
#6.	500 + - A	750 - A +	1,000 A + -	250 + - A
#7.	750 - A +	1,000 A + -	250 + - A	500 - A +
#8.	1,000 A + -	250 + - A	500 - A +	750 A + -
#9.	250 + - A	500 - A +	750 A + -	1,000 + - A
#10.	500 - A +	750 A + -	1,000 + - A	250 - A +
#11.	750 A + -	1,000 + - A	250 - A +	500 A + -
#12.	1,000 + - A	250 - A +	500 A + -	750 + - A

TABLE 3. Computed individual threshold shifts for normals at + 400 mm H_2O and - 400 mm H_2O.*

	250		500		750		1,000	
	+	-	+	-	+	-	+	-
S_1	12	12	8	6	20	10	28	22
S_2	8	14	10	12	24	10	22	4
S_3	6	6	2	6	6	8	2	6
S_4	10	10	14	10	10	10	16	16
S_5	10	12	16	14	0	0	4	14
S_6	10	14	12	14	10	8	10	16
S_7	6	14	14	16	20	20	18	10
S_8	4	2	12	10	12	10	16	8
S_9	22	10	20	14	14	4	24	16
S_{10}	16	2	10	4	6	0	8	6
S_{11}	14	16	10	8	16	8	12	12
S_{12}	12	12	6	10	12	14	14	12
$\bar{X}$	10.83	10.33	11.17	10.33	12.50	8.50	14.50	11.83
SD	4.93	4.66	4.71	3.80	6.83	5.54	7.87	5.29

*Threshold shift was computed by subtracting threshold at 0 mm H_2O from + 400 mm H_2O for positive effects and from - 400 mm H_2O for negative effects.

TABLE 4. Computed individual threshold shifts for high frequency sensorineural hearing losses at + 400 mm H_2O and - 400 mm H_2O.*

	250		500		750		1,000	
	+	-	+	-	+	-	+	-
S_{13}	14	12	12	8	10	10	4	10
S_{14}	10	6	10	8	16	16	6	18
S_{15}	16	8	14	18	22	24	10	22
S_{16}	8	12	8	8	18	20	16	14
S_{17}	4	12	16	16	20	16	8	12
S_{18}	16	14	22	12	18	16	22	24
S_{19}	22	14	14	10	14	14	2	18
S_{20}	16	18	6	12	10	18	+ 6	12
S_{21}	14	14	14	6	22	14	2	+ 2
S_{22}	16	18	20	16	12	12	8	16
S_{23}	12	14	6	0	8	0	22	14
S_{24}	12	14	16	14	20	8	20	18
$\bar{X}$	13.33	13.00	13.17	10.67	15.83	14.00	9.50	14.67
SD	4.62	3.46	5.08	5.07	4.93	6.15	8.87	6.68

*Threshold shift was computed by subtracting threshold at 0 mm H_2O from + 400 mm H_2O for positive effects and from - 400 mm H_2O for negative effects.

TABLE 5. Computed individual threshold shifts for low frequency sensorineural hearing losses at + 400 mm H_2O and - 400 mm H_2O.*

	250 Hz		500 Hz		750 Hz		1,000 Hz	
	+	-	+	-	+	-	+	-
S_{25}	10	22	12	18	4	14	6	10
S_{26}	16	12	10	10	14	20	6	10
S_{27}	2	2	18	14	4	4	4	12
$\bar{X}$	9.33	12.00	13.33	10.67	7.33	12.67	5.33	10.67
SD	7.02	10.00	4.16	4.00	5.77	8.08	1.16	1.16

*Threshold shift was computed by subtracting threshold at 0 mm H_2O from + 400 mm H_2O for positive effects and from - 400 mm H_2O for negative effects.

VITA

Robert Joseph Dunlop was born in Austin, Minnesota, on November 14, 1948. He attended elementary schools in that city and was graduated from Pacelli High School in 1966. The following September he entered Austin State Junior College, and in June, 1968 received the A.A. degree in Liberal Arts. He continued his education at Mankato State College and graduated with the B.A. degree in June, 1970, majoring in Speech Pathology.

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