A CASE STUDY OF SALICYLATE INTOXICATION AND ITS EFFECT ON THE FUNCTION OF THE AUDITORY SYSTEM

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ABSTRACT

A CASE STUDY OF SALICYLATE INTOXICATION

AND

ITS EFFECT ON THE FUNCTION OF THE AUDITORY MECHANISM

By

Diane M. Giraudi

Aspirin is a commonly used non-prescription drug. It can, with large doses, induce transient hearing loss, tinnitus, and vertigo. This study examined the reaction of the auditory system to high levels of aspirin, i.e., salicylate, in a 39 year-old female patient whose auditory history was otherwise unremarkable.

Peripheral and central auditory tests examined the reaction of the auditory system to salicylate intoxication. The test battery consisted of pure tone audiometry, speech audiometry, tone decay testing, the Short Increment Sensitivity Index (SISI), impedance audiometry, otoadmittance audiometry, brief tone audiometry, the Staggered Spondaic Word test (Katz, Basil, and Smith, 1963), dichotic listening task, filtered speech, time compressed speech, and pitch match tasks.

The patient's absolute hearing thresholds were found to be at the lower limits of the normal range with middle ear mobility and pressure within normal limits. The acoustic

reflex was present bilaterally.

Results of the Short Increment Sensitivity Index at high and low sensation levels demonstrated a normally functioning peripheral auditory system. The only direct indicator of cochlear pathology were the results of brief tone audiometry. However, this may be true because the other tests of cochlear disorder will, at times, fail to detect a subclinical or slight cochlear lesion. The results of the pitch match tasks were at the upper limits of the conductive hearing loss category (Graham and Newby, 1962; Nodar and Graham, 1965).

Central auditory testing consisted of time compressed monosyllables of the Northwestern University Auditory Test Number Six, low pass filtered speech, Staggered Spondaic Words, and dichotic consonant-vowels. Temporal lobe lesion due to salicylate intoxication was not indicated by these tests. Performance on the time compressed speech task did not markedly decrease across the levels 0 %, 30 % and 60 % time compression. Filtered speech results were comparable to the normative data established by this study. The Staggered Spondaic Word test results demonstrated less than 1 % error. Simultaneously presented dichotic syllables did not indicate cerebral dominance.

The findings of this case study appear to demonstrate a cochlear site of lesion since the integration function for

acoustic energy was less than that seen in individuals without cochlear disorder. Tests examining retrocochlear function did not show abnormal adaptation or increased sensitivity to small changes in intensity levels. Auditory function at the cortical level was not impaired due to salicylate intoxication.

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And, to Gene, who asked the now famous question, "Has anyone ever done a pitch match with salicylates?". You gave the push that got me into this and--I learned. Thank you.

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CHAPTER I

INTRODUCTION

There has been a major interest in the phenomenon of transient hearing loss induced by high levels of salicylate in the blood stream because aspirin is a commonly used non-prescription drug. When high salicylate levels are present, intoxication and subsequent transient hearing loss are said to occur. Investigators have explored the fluctuant hearing loss phenomenon with the goal of discovering the auditory mechanisms involved, whether the hearing loss is temporary or permanent, the serum levels for intoxication and subsequent hearing loss, the focus of the action of the aspirin, and the physiologic reactions to salicylates.

The purpose of this study was to investigate the effects of salicylate intoxication on the perception of pitch and on cochlear, retrocochlear, and central processes. Since the determination of the area or areas affected via pitch match tasks would only indicate sensorineural or conductive hearing loss, examination of the learing loss with special auditory

tests was warranted to differentiate among cochlear, retrocochlear, and central sites of lesion.

SALICYLATE OTOTOXICITY

Early Research

In 1903, Wittmaak (as cited in DeMoura and Hayden, 1968) reported on animal research concerning the influence of salicylates and quinine on the auditory system, specifically the cochlea, of the test animals. He noted the disappearance of Nissl bodies and changes in the nuclei of the spiral ganglion cells due to quinine. Because he suspected that salicylates acted on the inner ear in a similar manner, Wittmaak concluded that these changes occurred with salicylate poisoning. However, in 1913, Lindt's experimentation with salicylates (as cited in DeMoura and Hayden, 1968) did not verify Wittmaak's findings. Experimenting with animals, he did not find consistent changes due to aspirin in the cochlear structures of the test animals. The animals used in the previous studies were not specified by DeMoura and Hayden (1968).

In an effort to discover additional information about the action of salicylates and quinine upon the mechanisms of the ear, Covell (1936) administered sodium salicylate and quinine bisulfate to guinea pigs, white rats, and rabbits. He found similar reactions upon the cochlea from both drugs, with quinine

being the more pervasive reactant. The temporal bone sections of the experimental group of animals revealed mitochondrial changes in the cochlear duct and a loss of the ground substance in the spiral ganglion, especially in the lower modiolus. Increased protein levels were also observed. Covell also noted pronounced changes in the outer hair cells of the Organ of Corti. The mitochondria, which indicate presynaptic areas, were shapeless and the hair cells themselves were swollen. He attributed mitochondrial abnormalities to direct protoplasmic poisoning.

Further research (Mosher, 1938) found that hemorrhage occurred in the cochlea of guinea pigs as the result of salicylates, but this has been attributed to the temporal bone removal technique then employed (as cited in DeMoura and Hayden, 1968). Fable-Hansen in 1941 (as cited in Waltner, 1955) reported that Reissner's membrane was depressed into the cochlear duct more often after salicylates or quinine injection. He suggested that the cochlear changes were hypotonic in nature, i.e., lower osmotic pressure of the cochlear fluids. But Fable-Hansen noted these findings cautiously because of the inconsistency of the disruptions in the discovered abnormalities.

Waltner (1955) speculated as to the possible reaction of the cochlear structure to the salicylates. He hypothesized

that the reaction of salicylates on hearing may be due to direct protoplasmic poisoning, to interference with the normal blood supply to the inner ear, and/or to increased intra-labyrinthine pressure. He indicated that the latter hypothesis was the most probable result of salicylate intoxication.

Recent Investigations

Recent research has not demonstrated permanent structural damage to occur to the stria vascularis, sensory epithelium, spiral ganglion, or cochlear nerve as the result of salicylate intoxication. However, deviations do occur that effect the efficiency of the auditory system.

Bernstein and Weiss (1967) investigated the effects of salicylate intoxication on rheumatoid arthritics. In the pre-toxic state, hearing for pure tones and speech was within normal limits bilaterally. After administration of the aspirin, mild bilateral symmetrical sensorineural hearing loss was noted. Temporal bone analyses of two patients with rheumatoid arthritis who had ingested large doses of aspirin for long periods of time showed no abnormalities in the hair cells, Organ of Corti, stria vascularis, or spiral ganglion.

Silverstein, Bernstein, and Davis (1967) reported the results of an experiment with cochlear microphonics and action potentials in cats injected with sodium salicylate. They found

an elevation in the threshold of the eighth nerve action potential and a decrease in the amplitude and dynamic characteristics of the cochlear microphonics due to high levels of aspirin in the blood stream. Latency of the cochlear microphonics did not appear to be affected.

Myers and Bernstein (1965) compared audiograms before and during salicylate intoxication of individuals with normal and abnormal hearing. To avoid confusion, hearing threshold levels of this study and the studies following will be discussed re: ANSI 1969. Subjects with normal pre-toxic audiograms exhibited mild bilateral flat sensorineural hearing losses during salicylate intoxication. The fourteen subjects with abnormal pre-toxic audiograms were divided into four subgroups: unilateral sensorineural hearing loss, acoustic trauma, high frequency sensorineural hearing loss, and flat presbycusic hearing loss. During salicylate intoxication, subjects with high frequency sensorineural hearing loss and those with acoustic trauma in the pre-toxic state showed an elevation in threshold primarily in the low frequencies at levels of approximately 40 dB HTL re: ANSI 1969 across all frequencies due to aspirin. The subjects with unilateral sensorineural hearing loss were found to have an elevation in threshold in the affected ear and in the better ear across frequencies on the order of 40 dB. The presbycusic subgroup exhibited a symmetrical, flat, bilateral hearing

threshold of about 40 dB HTL re: ANSI 1969, an increase of approximately 15 dB from the pre-toxic audiogram.

The above studies agreed that salicylates, when given a serum level of about 30 mg./100 ml., induced inhibitory effects on groups of cellular enzymes. This action produced an uncoupling of oxidative phosphorylation which caused an alteration in the biochemical or bioelectric properties within the cochlear duct. This in turn could effect the stria vascularis and the Organ of Corti by decreasing metabolic activity.

Characteristics of Salicylate Induced Hearing Loss

Although there is much disagreement among researchers as to the form of interference and the loci of involvement within the auditory system, most investigators have acknowledged a variety of characteristics typically associated with salicylate intoxication. Tinnitus, hearing loss, and vertigo may all be present in the individual. Once salicylates are withdrawn, rapid recovery of hearing, within 24-72 hours, and disappearance of tinnitus and vertigo are demonstrated in most individuals. With few exceptions (Kapur, 1965; Jarvis, 1965), hearing returns to pre-toxic levels. The hearing loss, in general, is bilateral, sensorineural, symmetric, and equivalent over all frequencies for those with normal hearing in the pre-salicylate state. The hearing loss rarely exceeds

40-50 dB HTL, and discrimination scores are depressed. Recruitment has been reported, but the method of measurement was not indicated. Cochlear microphonics are depressed and the eighth nerve action potentials are elevated.

Attempting to establish whether or not serum levels and amount of hearing loss were related, Myers, et al. (1965), Silverstein, et al. (1967), and Sheffield and Turner (1971) examined the levels of salicylate in the blood stream which preclude involvement of the hearing mechanisms and subsequent hearing loss. Myers, et al., demonstrated that as the plasma salicylate level reached 30 mg./100 ml., a hearing loss of approximately 30-40 dB HTL re: ANSI 1969 was observed. Further increase in serum levels increased the degree of hearing loss and related symptoms. So that as serum levels increased, elevation in threshold increased but not beyond 50 dB HTL re: ANSI 1969. There no longer was a direct correlation between plasma salicylate levels and the amount of hearing loss. Thus, it appears that additional salicylate taken into the blood stream does not continue to elevate hearing thresholds. Investigations conducted by Silverstein, et al. and Sheffield and Turner concurred with these findings.

Therefore, hearing loss incurred appears to be dosage dependent, transient, and may be accompanied by tinnitus and/or vertigo.

Histopathological Studies

Histopathological studies have increased the body of knowledge available on salicylates and their effects on the auditory system. To illustrate the course these studies have taken over the years, the investigations highlighting the research in this area shall be discussed according to research methodology.

Recent histopathological studies using guinea pigs have not indicated structural abnormality of the Organ of Corti, stria vascularis, or spiral ganglion. Agreement in the assumption that salicylate ototoxicity results in uncoupling oxidative phosphorylation in the cochlea lends support to the contention of earlier researchers that the effect on the auditory structures is biochemical in nature. Krazanowski and Matschinsky (1971) reported data that revealed the locus of impairment to be in the energy metabolism mechanism of Reissner's membrane. However, Thalman, Miyoski, Kusakari, and Thalman (1973) pointed out that electrophysiological data, specifically the measurement of adenosine triphosphatase, have been limited. Adenosine triphosphatase (ATP) is an ester concerned with energy metabolism for muscular and other cellular activity. Measurement of ATP action at the cochlear nerve via ultramicrochemical techniques during salicylate intoxication led Thalman, et al., to conclude that although there appeared

to be an absence of detectable changes in the acoustic nerve and ganglion, the main action of salicylate induced hearing loss may be at the neural level. Support for this assumption can be found in an earlier study conducted by Silverstein, Bernstein, and Davis (1967). Because of a decrement in the cochlear microphonic and an elevation in the eighth nerve action potential during salicylate poisoning, they hypothesized that high concentrations of salicylate probably produced transitory effects which were inhibitory to the enzyme systems in the hair cells or at the synaptic nerve endings. Therefore, this may indicate a retrocochlear site of action.

In opposition to these investigations, McPherson and Miller (1974) determined the focus of the salicylic action to be the cochlea. These researchers assessed the reaction of the hearing structures to aspirin via measurement of cochlear potentials in guinea pigs. Elevated salicylate levels reduced the amplitude of the first negative peak (N_1) and decreased the amplitude, but not the latency, of the cochlear microphonics. They concluded that these findings were indicative of inner ear pathology.

Gold and Wilpizeski's (1966) results agreed with the conclusions reached by McPherson and Miller. When they implanted permanent electrodes into the ears of an experimental group of cats, Gold and Wilpizeski observed reduction in the N_1

amplitude accompanied by an elevation of 30 dB in N_1 threshold, and a depression of cochlear microphonics. These authors concluded that the site of action was intracochlear.

The inconclusive nature of the research thus far can be noted in the results of previous investigations. To date, determination of the focus of the negative effect of salicylic action has not been resolved.

Case Studies

Histopathological research has not allayed the confusion nor has it decided the controversy over the loci of involvement of salicylates. To further complicate this discussion, case study investigations have proved to be contradictory and without conclusive evidence as to loci of salicylate action. But discussion of case studies is imperative to understanding the research to date concerning salicylate ototoxicity.

In 1960, Kodman, Cull, and Lawson reported on a male patient who sustained a residual bilateral sensorineural hearing loss of a magnitude of approximately 80 dB HTL re: ANSI 1969 in the high frequencies. Generally, salicylate induced hearing loss rarely exceeds 50 dB HTL re: ANSI 1969. However, in addition to the salicylates, the patient had also been exposed to carbon monoxide. The treatment of exposure to carbon monoxide included chloroquin, a known ototoxic drug. Other

medications were pabalate H.C. and hydrocortizone. Withdrawal of salicylates resulted in improvement of hearing thresholds, leaving only residual bilateral high frequency impairment. The authors cautioned against attributing the sustained hearloss solely to aspirin because of the inability to disseminate the interactive effects of the aspirin, the other medications, and the carbon monoxide exposure. Kodman, et al. also noted an additional finding: clinical evidence strongly pointed to both a cochlear and a retrocochlear lesion which were at least partially reversible. Bone conduction thresholds and tinnitus they interpreted as indicative of cochlear pathology. However, the scores for speech discrimination did not decrease with increased presentation level as would be expected with a cochlear lesion.

Jarvis (1965) wrote of a case of severe unilateral sensorineural hearing loss possibly related to ingestion of 2-3 tablets every two hours for a period of three days for pain following a tooth extraction. This resulted in a hearing loss ipsilateral to the side of the extraction. Since the subject had not been taking another form of medication previously, salicylates were assumed to be the cause of the hearing loss. But, since the hearing loss occurred on the same side as the extraction, hyperaemia and pain could have produced reflex neural or circulatory disturbances that in turn modified inner ear

blood supply ipsilaterally, thereby resulting in increased susceptability of the hearing structures to the action of the salicylate. Idiosyncratic hypersensitivity to the larger-than-recommended dosage may also have induced the hearing loss.

Such an idiosyncratic sensitivity to aspirin was noted by Kapur (1965). Following a febrile illness during which three tablets of the usual strength were ingested, a severe bilateral hearing loss and dizziness occurred in a thirteen year-old patient. Although the origin of the child's hearing loss may be viral due to the fever, case history information, onset, symptoms, absence of upper respiratory symptoms, audiologic findings and vestibular test results indicated idiosyncratic hypersensitivity to acetysalicylic acid. The patient sustained a moderate to severe bilateral sensorineural hearing loss.

Salicylate induced hearing loss due to topical application was reported by Perlman (1966). Bilateral symmetrical sensorineural hearing loss and tinnitus were evidenced in two female patients from skin application of 24-35 mg./100 ml. of salicylate for treatment of psoriasis. Termination of salicylate treatments resulted in a return to pre-toxic hearing thresholds.

Investigations with Salicylate Ototoxicity using Rheumatoid Arthritics

Research employing rheumatoid arthritic subjects has yielded more information on the possible hearing loss caused by salicylate intoxication. Initially, investigations centered around rheumatoid arthritis as a possible cause of conductive hearing impairment due to involvement of the ossicular chain. Although the occurrance of rheumatoid nodules in the middle ear is a possibility, Goodwill, Lord, and Knill Jones (1972) did not find rheumatoid nodules or erosive changes in the ossicular joints in their analyses of temporal bone sections. Djupesland, Grønas, and Saxegaard (1973) performed pure tone air and bone conduction and impedance measurements (tympanograms and acoustic reflex) on rheumatoid arthritics with elevated hearing thresholds. Test results revealed middle ear pressure and compliance of the tympanic membranes in general to be within normal limits. The stapedial reflex was present. These results indicated a normally functioning middle ear. These authors concluded that although conductive pathology is possible in rheumatoid arthritics, their subjects did not demonstrate middle ear involvement.

Bernstein and Weiss (1967) examined auditory sensitivity in individuals taking salicylates for treatment of rheumatoid arthritis. They found central summation to be intact and a

depression of caloric function, as ascertained by the Hallpike bicaloric technique. They concluded that the action was peripheral and not central in nature.

The previous review has demonstrated the inconclusive and contradictory nature of the research in this area. Histological, audiologic and biochemical studies have yielded some information, but knowledge is still lacking concerning the loci of involvement. Covell (1936) stated, "the effects of various toxic agents such as drugs, poisons, and toxins on the mechanisms of hearing are more readily studied by tests of hearing than by examination of sections of the cochlea for pathological changes" (p. 633). Clinical tests of suprathreshold hearing function, examination of the acoustic characteristics of tinnitus, and tests of central auditory function may prove to be more sensitive indicators of salicylate intoxication, and may provide information as to the specific areas of the auditory system involved.

TINNITUS AND PITCH MATCH

Tinnitus has long been recognized as an indicator or symptom of otologic dysfunction (Graham and Newby, 1962). Tinnitus is a common occurrance with salicylate intoxication (Kapur, 1965). In many cases it was the first symptom experienced by the patient (Jarvis, 1965). Various authors

have discussed the subjective appraisal of the tinnitus experienced. Some have described the tinnitus as being high pitched and others have depicted the tinnitus to be a "buzz" or a "roar" or musical notes. Determination of these descriptions or terms was not given by the investigators. Individual patient subjective classification most probably accounted for the variety in the reports.

Graham and Newby (1962) investigated the acoustic characteristics of tinnitus in an attempt to establish a basic method for analyzing non-vibratory tinnitus and to determine whether differences among pathological populations existed. By employing subjective reports, initial and final pitch matches, and loudness judgments, these authors found a difference among pathological states. The results of this study indicated that individuals with sensorineural hearing losses with tinnitus tend to match their tinnitus to high frequency tones, whereas those with conductive pathology did not match their tinnitus to frequencies greater than 1400 Hz. Graham and Newby concluded that the tinnitus experienced with a conductive hearing loss may be the product of a different physiological mechanism than that associated with sensorineural pathology.

Nodar and Graham (1965) continued exploration into the possibilities set forth by Graham and Newby. They noted that with Meniere's disease, a cochlear pathology, a "buzz" or "roar" was the discriptive classification of the tinnitus perceived. Replicating Graham and Newby's experimental design for a pitch match, their findings confirmed Graham and Newby's results. Conductively hearing impaired subjects matched their tinnitus to the frequency range of 90 to 1450 Hz, with a median frequency of 490 Hz. The subjects in the sensorineural group, excluding those with Meniere's disease, compared their tinnitus to the frequencies 545-7500 Hz, with a mediam frequency of 3900 Hz. However, those with Meniere's disease associated their tinnitus to the frequency range of 90 to 900 Hz exclusively. The median frequency for this group was 320 Hz.

Thus, subjects with Meniere's and those with conductive impairment appear to experience a similar representation of tinnitus although the etiologies are not alike. Nodar and Graham speculated that similar physiologic mechanisms cause the tinnitus that accompanies these hearing disorders.

Therefore, via a tinnitus pitch match, information is available which allows differentiation between hearing losses which are conductive in nature from those that are sensorineural. But, when patients with Meniere's disease are considered, results of a pitch match are not commensurate with

those generally found in sensorineural populations. This may also be true with those individuals experiencing tinnitus as the result of salicylate intoxication. The enzyme actions and/or intralabyrinthine pressure may be affected in a manner which inhibits the normal conduction of the cochlear fluids.

A comparison of the tinnitus reported by the subject with high serum salicylate levels and the data presented by Graham and Newby could yield additional information as to the action salicylates have upon the auditory system. A low frequency pitch match may be indicative of an inner ear conductive action, such as that hypothesized by Nodar and Graham to occur in patients with Meniere's disease. Since biochemical and histopathological studies have been inconclusive in determination of site of salicylic action, a pitch match may yield more complete data and understanding of transient hearing loss that occurs during salicylate intoxication. As Covell (1936) explained, tests of hearing may prove to be the preferred method of examining the effects of drugs upon the auditory system. Therefore, he felt that clinical tests investigating hearing function may be more sensitive measures of salicylate toxicity.

The literature in the area of audiologic exploration of salicylate ototoxicity is limited. From the preceding review of the research it may be assumed that more study is warranted.

Covell (1936) and McPherson and Miller (1974) called for investigations using audiological assessment, yet the bulk of the research conducted in the intervening 38 years following Covell's suggestion has been filled with an abundance of biochemical and histopathological studies. All of these experiments have left us without definitive answers concerning salicylic action on auditory functioning.

STATEMENT OF THE PROBLEM

Additional investigation of the effect of salicylates on the auditory system is necessary and the mode of exploration should be with audiologic tests. A pitch match and sophisticated audiometric tests will assist in better understanding of the reaction of the auditory system to elevated salicylate levels.

Although the literature employing special audiometric test procedures is limited, some researchers have given consideration to audiologic testing.

Pedersen (1974) limited his experiment to brief tone audiometry on fourteen individuals with salicylate intoxication. His results were indicative of cochlear pathology. Unfortunately, other special auditory test were not performed.

A pitch match may also furnish data concerning site of salicylate action in the auditory system and the form the

action takes on this system. The results of this analysis can then be employed in the area of speculation as to which of the following hypotheses is the force of action during salicylate intoxication: interference with intralabyrinthine fluid conduction or enzymatic inhibition at the neural level.

Additional study is called for. Perhaps new information regarding salicylates and hearing will not surface. But an increase in knowledge, understanding, and awareness of cochlear, retrocochlear, and central processes will certainly be realized. Expanded examination of this subject is therefore imperative.

The question under consideration in this study is: to what frequency does the individual undergoing salicylate intoxication match the tinnitus he perceives and what information will special test procedures yield regarding cochlear, retrocochlear, and central auditory function?

Bernstein and Weiss (1967) used Bekesy audiometry in audiologic examination of patients with high serum salicylate levels. Their subjects did not exhibit abnormal tone decay as measured by this procedure, that is a separation greater than 20 dB (Jerger, 1960). Sanders, Josey, and Glascock (1974) have demonstrated that Bekesy audiometry is not the best indicator of abnormal adaptation to acoustic stimuli because the stimulus is constantly changing over time. For this reason, they

explained that measurement of abnormal tone decay via Bekesy does not stress the auditory system as do other tests for tone decay, e.g. Carhart Threshold Tone Decay test (Carhart, 1957). In fact, they found that in a great number of cases erroneous conclusions were made on the basis of Bekesy tracings alone. An earlier study by Rosenberg (1972) also stressed the necessity of obtaining samples of auditory behavior that were correlated. He explained that the diagnosis of auditory disorder cannot be made on the findings of a single test. Instead, a test battery approach is preferred and advocated.

With this in mind, the present knowledge of special tests and procedures can and must be used with individuals undergoing salicylate intoxication. Applying a test battery consisting of peripheral and central auditory tests, and an analysis of the perceived tinnitus to the population of rheumatoid arthritics undergoing salicylate intoxication will increase our knowledge of salicylic effect on the auditory system--peripheral and central aspects.

Pitch matches may also furnish data concerning site of salicylate action in the auditory system and the form the action takes on this system. The results of this analysis can then be employed to speculate as to which of the following hypotheses is the force of action during salicylate intoxication; interference with intralabyrinthine fluid

conduction or enzymatic inhibition.

The questions under consideration in this study were:

1) to what frequency does the individual undergoing salicylate intoxication match the tinnitus she perceives?

2) what information will special test procedures yield regarding cochlear, retrocochlear, and central auditory function?

CHAPTER II

EXPERIMENTAL PROCEDURES

Subject

The subject taking part in this study was selected on the basis of experiencing tinnitus while using salicylates. To achieve an optimum effective anti-inflammatory level, serum salicylate levels of at least 15 to 30 mg./100 ml. must be reached (Mongan, Kelly, Nies, Porter, and Paulus, 1973). Since tinnitus and hearing loss can be seen at levels of 30 mg./100 ml. on the average, this subject was chosen. Hearing loss due to salicylates was not a requisite for inclusion in this study.

The subject was a 39 year-old white female. Case history information was unremarkable with the exception of daily ingestion of 19-20 aspirin tablets for treatment of rheumatoid arthritis. There was no history of middle ear infection or other otologic disorder (See Appendix I).

Ten normal hearing young adults ages 17-23 years of age were evaluated to establish normative data for the filtered speech used in this study. Hearing was screened at 15 dB

HTL re: ANSI 1969 for the frequencies 250-8000 Hz. Speech reception thresholds and word discrimination scores were established prior to testing with filtered speech. Different forms of the Northwestern University Auditory Test Number Six were administered in the filtered and unfiltered conditions.

Instrumentation

Audiometric equipment consisted of a Grayson-Stadler 1701 clinical audiometer for routine and special testing and the initial pitch match; a Grayson-Stadler 1720-B Otoadmittance meter and Madsen ZO-70 Impedance bridge (coupled to a Beltone 10-C portable audiometer) for tympanometry, stapedial reflex measurement, and reflex decay; a Grayson-Stadler 1701 X-Y recorder for graphic representation of brief tone thresholds; a Hewlett-Packard 3310-A function generator for continuous frequency adjustments during the final pitch match; a Beckman 6148 frequency counter for frequency assessment during the final pitch match; a Grayson-Stadler 1208 power supply, switch and timer for variable stimulus duration for brief tone audiometry; a Sony TC 366 two-channel tape recorder for routine speech audiometry and tests of monotic and dichotic listening ability; and a matched set of TDH-49 earphones in MX 41/AR cushions (See figure 1).

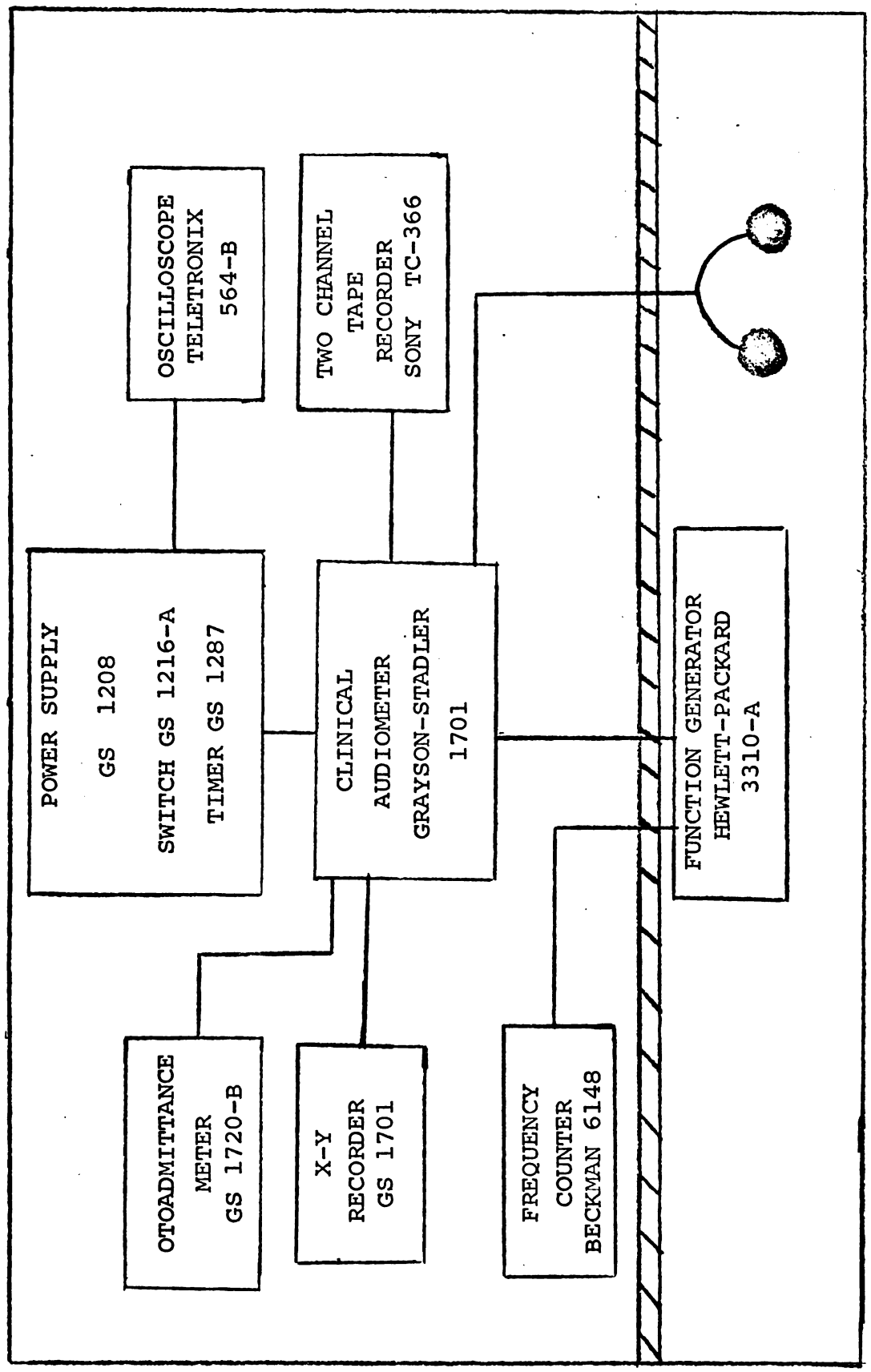

Figure 1: Block diagram of instrumentation used for auditory testing.

Calibration

Additional equipment was used for general calibration purposes and for calibration of the instrumentation used to filter a taped reproduction of the Northwestern Auditory Test Number Six (NU#6) Forms A and B (Tillman and Carhart, 1966).

Calibration consisted of Random Sine Generator (Bruel and Kjaer type 1024); a Frequency Spectrometer (Bruel and Kjaer type 2112); a graphic level recorder (Bruel and Kjaer type 2305); an Oscilloscope (Teletronix 564-B); and a Spectrum Analyzer (Teletronix 3L5).

A tape of filtered speech was made by reproducing the NU#6 Forms A and B on an Ampex AG 402 two-channel tape recorder and then feeding the output through a General Radio 1925 Multifilter One-third Octave and Band Filter with related switch assembly. The rejection rate was 32 dB in the first octave with no measureable output at 1000 hertz (Hz) and the cut-off frequency was 500 Hz (Lynn and Gilroy, 1972). The filtered speech was recorded on Scotch AV 176 low noise oxide recording tape with the Ampex AG 440 two-channel recorder.

Signal intensity of the Grayson-Stadler 1701 clinical audiometer for routine and special testing and the pitch matches and the Beltone 10-C portable audiometer for acoustic reflex and reflex decay testing was in accordance with the

specifications of the American National Standards Institue (ANSI S3.6 1969).

Intensity calibration was checked with an input of 70 dB HTL at the eleven frequencies. Appropriate corrections were made where necessary. Deviation of the sound pressure level reading from the hearing threshold level dial by more than 3 dB at the frequencies of 250 to 3000 Hz inclusive, by more than 4 dB at 4000 Hz, or by more than 5 dB at the frequencies above or below this range indicated that a correction was to be made.

Linearity was measured at 1000 Hz by starting at the limits of the audiometer (110 dB HTL) and descending in 5 dB steps. Linearity did not differ from the dial reading by three-tenths of the dial setting or 1 dB, whichever was larger (ANSI, 1969). These measurements were made for both earphones with a Bruel and Kjaer type 4152 artificial ear using a Bruel and Kjaer type 4144 pressure microphone attached to a Bruel and Kjaer type 2204 sound level meter.

Frequency output on the Grayson-Stadler 1701 and the Belton 10-C audiometers was checked with the Beckman Frequency Counter (6148). Frequency output for 125-8000 Hz was found to be within $\pm$ 3% of the given frequency.

Speech was calibrated with each tape by the 1000 Hz calibration tone on the individual tapes. Complete calibration for speech was made prior to testing by directing

the speech signal through the test earphones to an artificial ear (Bruel and Kjaer type 4152) with pressure microphone (Bruel and Kjaer type 4144) coupled to an Audio-Frequency spectrometer (Bruel and Kjaer type 2112).

Rise-decay time and on-time duration were checked for the SISI. The rise-decay time was found to be 50 msec, and the duration of the tonal stimulus was 200 msec. These are in accordance with Jerger's (1960) recommendations.

The possibility of spread of energy to frequencies other than the test frequency during brief tone audiometry was assessed with the Teletronix 3L5 Spectrum Analyzer coupled to the Teletronix 564 Oscilloscope. Energy was 44 dB or more below the fundamental frequency for the second and third harmonics at the frequencies 500, 1000, and 2000 Hz at 500, 200, and 20 msec.

Procedure

The subject was seen on two occasions, During the first session, pure tone air conduction, speech reception testing (SRT), word discrimination, SISI (at high and low sensation levels), brief tone audiometry, otoadmittance, impedance, tone decay, and the pitch matching tasks were performed. In the second session, pure tone air conduction, SRT, word discrimination, SISI (at high and low sensation levels), tone decay

and impedance were readministered. At this time central testing of hearing at the cortical level was also administered.

Hearing sensitivity of the ten normal hearing subjects was screened at 15 dB HTL re: ANSI, 1969 for the frequencies 250 to 8000 Hz. Speech reception threshold, unfiltered and filtered word discrimination ability were assessed.

Three tests were routinely used to assess hearing for both ears: pure tone air conduction testing, speech reception threshold determination, and word discrimination testing.

Special peripheral test procedures were employed to evaluate the site of lesion for both ears. They were the Carhart threshold tone decay test, the Short Increment Sensitivity Index (SISI) at low and high sensation levels, brief tone audiometry, otoadmittance and impedance audiometry.

Two pitch matching tasks were made for both ears. The first match was performed with the audiometer to establish an estimate of the range of frequencies which approximated the tinnitus experienced by the subject. A second pitch match was made with the Hewlett Packard 3310-A function generator which allowed for a continuous change in frequency over the entire audible frequency range. This provided a finer estimate of the frequency analogous to the tinnitus.

The test battery for central auditory function consisted of time compressed Northwestern University Auditory Test Number Six (NU#6), Staggered Spondaic Words (Katz, Basil, and Smith, 1963), low-pass filtered speech (Lynn and Gilroy, 1972), and simultaneous dichotic Consonant-Vowel (CV)

The instructions were:

> You will be hearing some words. The words are: airplane, armchair, baseball, birthday, cowboy, daybreak, doormat, drawbridge, duckpond, eardrum, farewell, grandson, greyhound, hardware, headlight, horseshoe, hotdog, hothouse, iceberg, inkwell, mousetrap, mushroom, northwest, oatmeal, padlock, pancake, playground, railroad, schoolboy, sidewalk, stairway, and whitewash. Repeat the words after the gentleman on the tape, no matter how soft the words may become.

Discrimination for speech under earphones was assessed monaurally for both ears at 40 dB sensation level (SL) re: SRT. The speech stimuli were taped lists of the N.U. #6 form D (Carhart and Tillman, 1966).

The subject was told:

> You will be hearing some sentences. These will not decrease in loudness. Please repeat the last word in each sentence after the gentleman on the tape. For example, if he says, You will say, 'black', all you must repeat is the word "black". Do you have any questions?

Tone decay was measured via the Carhart Threshold Tone Decay test (Carhart, 1957). The tone was presented at 500 and 2000 Hz at pure tone threshold. The subject was instructed:

> Once again you will be hearing a tone. This time I want you to press the button down when you hear the tone and keep the button depressed as long as you hear the tone. Should the tone disappear, be prepared, it may become audible again.

The SISI (Jerger, Shedd, and Harford, 1959) was assessed at 20 dB SL the pure tone threshold at 500 and 2000 Hz. The subject was required to respond when an increment in the test signal was heard. The test procedure was then repeated at a higher sensation level of 75-80 dB to evaluate possible retrocochlear involvement. Thompson (1963) reported that when the SISI was presented at high sensation levels those with normal hearing evidenced high or positive SISI scores whereas individuals with retrocochlear pathology continued to show low or negative SISI scores. The instructions were:

> You will be hearing a tone continuously. This tone will "jump" in loudness at various intervals. Each time you hear the "jump" in loudness, please depress the button. Are there any questions?

Brief tone audiometry was administered at 500, 1000, and 2000 Hz. Research indicated that normal ears integrate acoustic energy in a near linear function at these frequencies (Wright, 1968). Thresholds were obtained at these frequencies using durations of 500, 200 and 20 msec with a rise-decay time of 10 msec. Bekesy tracings were obtained and the subject was told:

> You will be hearing pulsed tones. Depress the button as long as you hear the tones and release the button when you can no longer hear the tones.

At the end of testing with brief tones a tracing with a continuous tone was made to yield additional information. The subject was reinstructed with the standard directions; however, instead of a pulsed tone the subject was informed that she would be hearing a continuous tone. The same frequencies were evaluated.

Tympanograms for both ears were obtained via otoadmittance and impedance audiometry. Reactance and susceptance measures were made using 220 and 660 Hz tones with the otoadmittance meter. The threshold for the acoustic reflex and reflex decay were evaluated on the impedance bridge. Reflex decay was tested at 500 and 1000 Hz because considerable decay had been found in normals at 2000 and 4000 Hz (Anderson, Barr, and Wedenberg, 1970). The subject was told:

> For this test, all you must do is sit quietly. Please do not talk or swallow. What you will be feeling is air being put into your ear canal and then withdrawn.

The initial pitch match was administered with a clinical audiometer. Using the eleven available frequencies, each frequency was presented twice at 10 dB SL re: pure tone thresholds.

At the end of testing with brief tones a tracing with a continuous tone was made to yield additional information. The subject was reinstructed with the standard directions; however, instead of a pulsed tone the subject was informed that she would be hearing a continuous tone. The same frequencies were evaluated.

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> For this test, all you must do is sit quietly. Please do not talk or swallow. What you will be feeling is air being put into your ear canal and then withdrawn.

The initial pitch match was administered with a clinical audiometer. Using the eleven available frequencies, each frequency was presented twice at 10 dB SL re: pure tone thresholds.

The subject was instructed:

> You will be hearing some tones. I would like you to listen to the tones. They will be presented twice. After the second presentation, please mark the sheet of paper in front of you as to whether the tone was higher, lower, or equal to the noises in your ears.

The preceding results were analyzed following a five minute rest period. This yielded a frequency range which the subject judged as equal to the perceived tinnitus. The final pitch match was then made with the function generator at 10 dB SL re: the average threshold for this frequency range. The subject was told:

> With the white dial in front of you, please vary the pitch by turning the dial. First make the tone noticeably higher in pitch, then lower in pitch than the noises in your ears. Finally, I want you to make the tone equal to the noises in your ears.

Three trials were given for each ear and each was evaluated monaurally. The average of the three trials was taken as the frequency match of the tinnitus.

Time compressed Northwestern University Auditory Test Number Six (Beasley, Schwimmer, and Rintelmann, 1972) were administered monaurally at 0%, 30%, and 60% compression. The word lists were presented at 40 dB SL the patient's monaural SRT. The subject was instructed in the same manner as for word discrimination testing for this and filtered speech.

Discrimination of low pass filtered speech (Lynn and Gilroy, 1972) was evaluated at 60 dB SL monaural SRT. The cut-off frequency was 500 Hz with a rejection rate of 32 dB in the first octave.

Different forms of the Northwestern University Auditory Test Number Six were used for the time compressed monosyllables and the filtered monosyllables. Word discrimination was assessed with form D. Form A was used for the low pass filtered speech test. Finally, form B was used in testing discrimination for speech under various conditions of time compression.

The Staggered Spondaic Word test (Katz, Basil, and Smith, 1963) was presented dichotically at 50 dB SL re: SRT. The subject was instructed:

> You will be hearing some two syllable words. Please repeat the words after the gentleman on the tape.

Dichotic consonant-vowel (CV) syllables (Berlin, Lowe, Thompson, and Cullen, 1968) were administered simultaneously with no lag time between ears. The presentation level was 50 dB HTL. The subject was instructed in this manner:

> You will be hearing some single syllables. I want you to repeat the syllables you hear after the gentleman on the tape.

Analysis of Data

The results of this study were analyzed quantitatively. Data obtained from the pitch match was compared to the categories established by Graham and Newby (1962) and Nodar and Graham (1965). On the basis of the hypotheses that the hearing loss and tinnitus caused by salicylates may be due to increased intralabyrinthine pressure (Waltner, 1955) or may be due to enzyme inhibition (Myers and Bernstein, 1965), the subject undergoing salicylate intoxication was expected to match her tinnitus to the same frequency range as those with conductive pathology or Meniere's disease (Nodar and Graham, 1965).

The peripheral and central test results were interpreted with respect to site of lesion. The results were examined in order to obtain a better understanding of the cochlear, retrocochlear, and central processes of the ear during salicylate intoxication.

Data obtained for the ten normal hearing subjects for low pass filtered speech was analyzed by measures of central tendency. The mean, median, mode, and range were calculated.

CHAPTER III

RESULTS

Peripheral Test Results

Results of puretone air conduction audiometry using pulsed tones of 500 msec with a 50% duty cycle indicated hearing to be at the lower limits of the normal range bilaterally (Davis and Kranz, 1964). Speech reception testing (SRT) was eight decibels (dB) better than these results. This inconsistency may have been the result of the tinnitus interfering with the pure tone thresholds but not with the speech stimuli. Speech discrimination under earphones at 40 dB sensation level (SL) re: SRT was excellent for both ears. (See Table 1)

Table 1: Pure tone air conduction thresholds tested at 250-8000 Hz bilaterally. Pure tone average (PTA), speech reception threshold (SRT), percentage correct (%) for Northwestern University Auditory Test Number Six (NU # 6) monosyllables at 40 dB sensation level re: SRT indicated hearing at the lower limits of normal. Results were essentially the same for both test sessions.

EAR	250	500	1000	2000	4000	8000	PTA	SRT	% LIST
R	25	20	20	15	20	30	18	10	100 NU#6
L	25	20	20	15	30	30	18	10	100 NU#6

Although hearing was within normal limits bilaterally, the thresholds were at the lower limits of normal. A battery of sophistocated audiometric tests were employed to further evaluate the effects of aspirin on the auditory system.

The Carhart Threshold Tone Decay test was not indicative of abnormal adaptation for either ear. In the left ear, 5 dB or less of tone decay was seen for the frequencies 500 and 2000 Hz. The right ear did not evidence any tone decay for the two test frequencies. (See Table 2).

The Short Increment Sensitivity Index (SISI) was administered at 20 dB SL and 80 dB SL re: pure tone thresholds for the frequencies 500 and 2000 Hz at low and high sensation levels. At low sensation levels, the SISI was negative, i.e., not indicative of cochlear pathology, bilaterally. Inadvertently, testing at 80 dB SL was initially conducted without masking. Since the subject returned for additional evaluation, the SISI was readministered with masking contralateral to the test ear. The test results with and without masking revealed positive SISI scores for both ears. These results (see Table 2) were not consistent with retrocochlear pathology (Thompson, 1963).

Table 2: Results of Carhart Threshold Tone Decay test and masked and unmasked Short Increment Sensitivity Index (SISI)

			RIGHT EAR		LEFT EAR	
CARHART TONE DECAY			500 Hz	0 dB	500 Hz	0 dB
			2000 Hz	0 dB	2000 Hz	5 dB
SISI		LOW	500 Hz	5%	500 Hz	15%
	unmasked		2000 Hz	0%	2000 Hz	5%
		HIGH	500 Hz	100%	500 Hz	100%
			2000 Hz	100%	2000 Hz	100%
	masked	HIGH	500 Hz	100%	500 Hz	80%
			2000 Hz	100%	2000 Hz	100%

The preceding test results do not demonstrate cochlear or retrocochlear lesion. In fact, examination of these findings portrayed the auditory system as normal since there was no signigicant adaptation and no increased sensitivity to small changes in intensity.

The slope of the tympanograms for impedance and oto-admittance were within normal limits for both ears. Eardrum compliance was 0.4 cc in the right ear and 0.39 cc in the left ear. These results demonstrated normal eardrum mobility bilaterally, The acoustic reflex was present at all frequencies tested for both ears and elicitation levels were not elevated.

These results showed that middle ear integrity was intact bilaterally. The stapedial reflex was sustained at 500 and 1000 Hz in both ears for 10 seconds. This was not consistent with retrocochlear pathology (Anderson, Barr, and Wedenberg, 1970). (See Table 3)

Table 3: Thresholds for the acoustic reflex for frequencies 500, 1000, 2000, and 4000 Hz. The reflex thresholds are in hearing threshod levels (HTL) and sensation levels (SL) re: pure tone thresholds. Reflex decay values are the time in seconds (sec) the reflex was maintained.

PROBE TONE EAR	TEST TONE EAR		500 Hz	1000 Hz	2000 Hz	4000 Hz
		HTL	90 dB	85 dB	85 dB	105 dB
L	L	SL	70	65	70	85
		HTL	85	85	90	110
R	L	SL	65	65	75	70
	R	sec	10	10		
REFLEX DECAY	L	sec	10	10		

Graphic representation of impedance and otoadmittance results can be found on figures 2 and 3 (A and B) and show normal middle ear pressure and eardrum mobility.

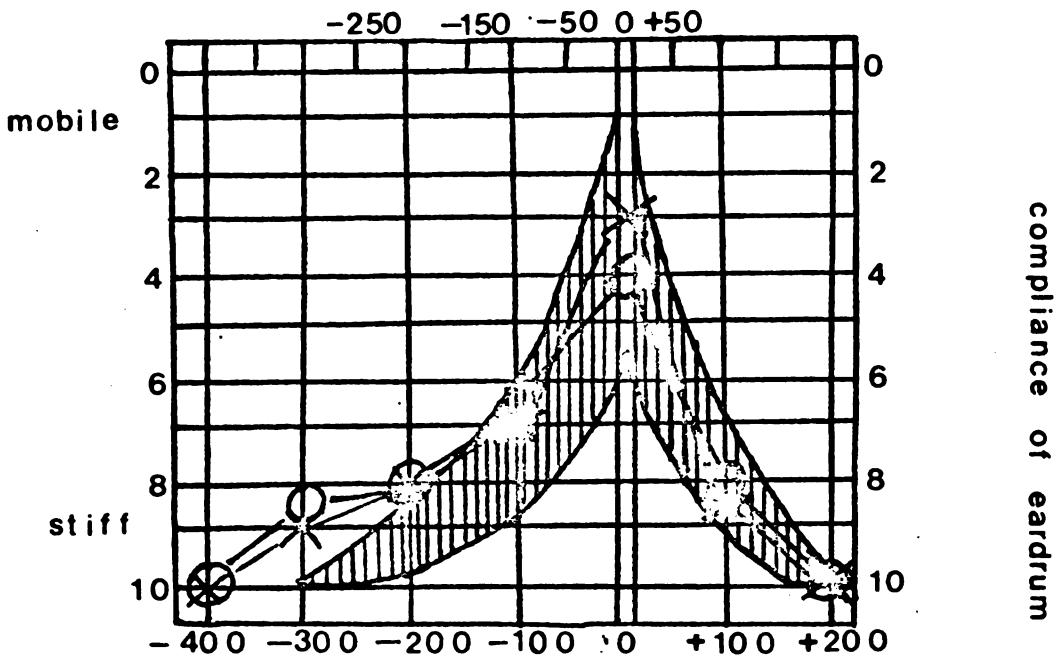


Figure 2: Tympanogram shows normal findings of middle ear compliance as air pressure is increased and then decreased in the ear canal.

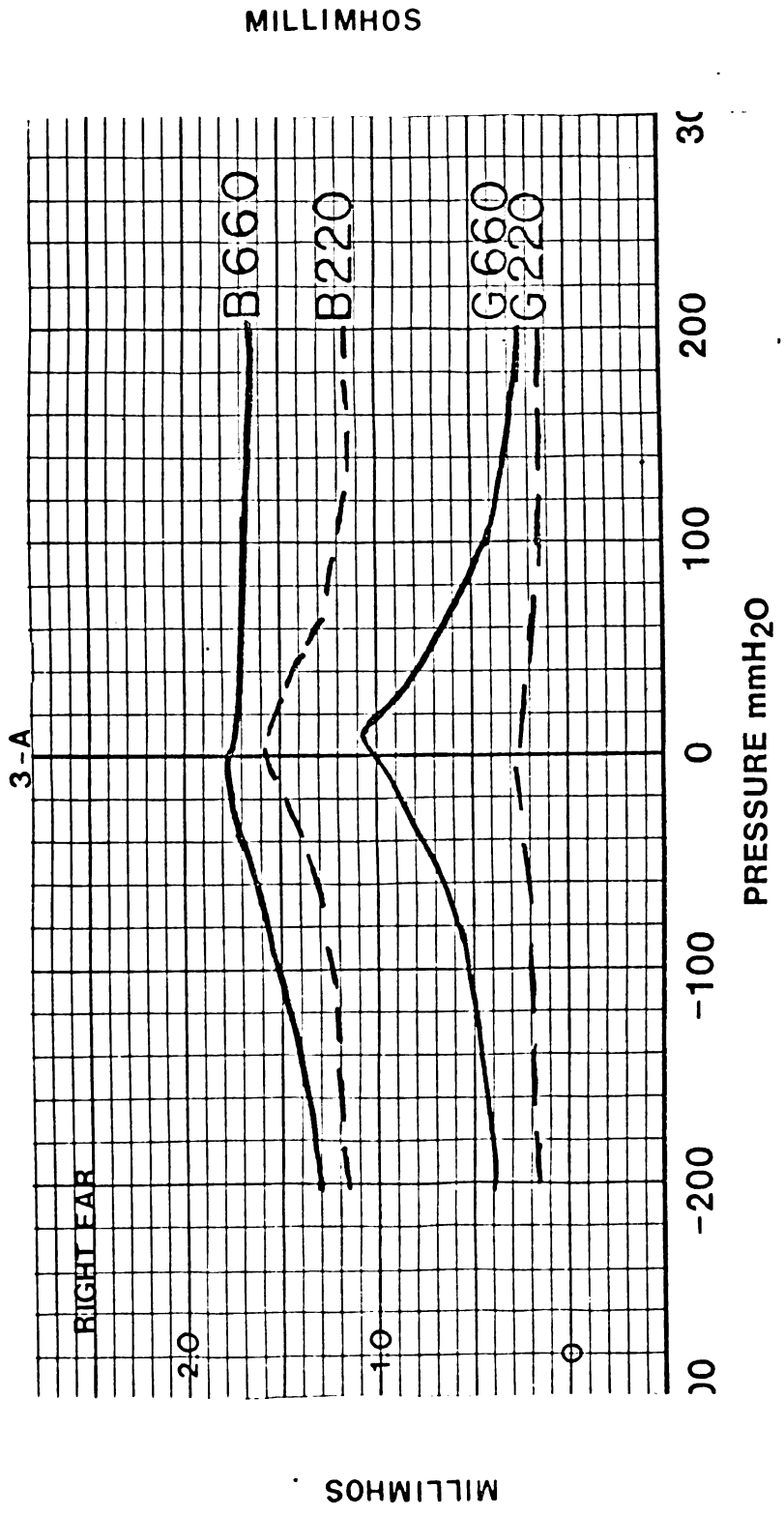


Figure 3-A: Normal otoadmittance results for the subject's right ear.

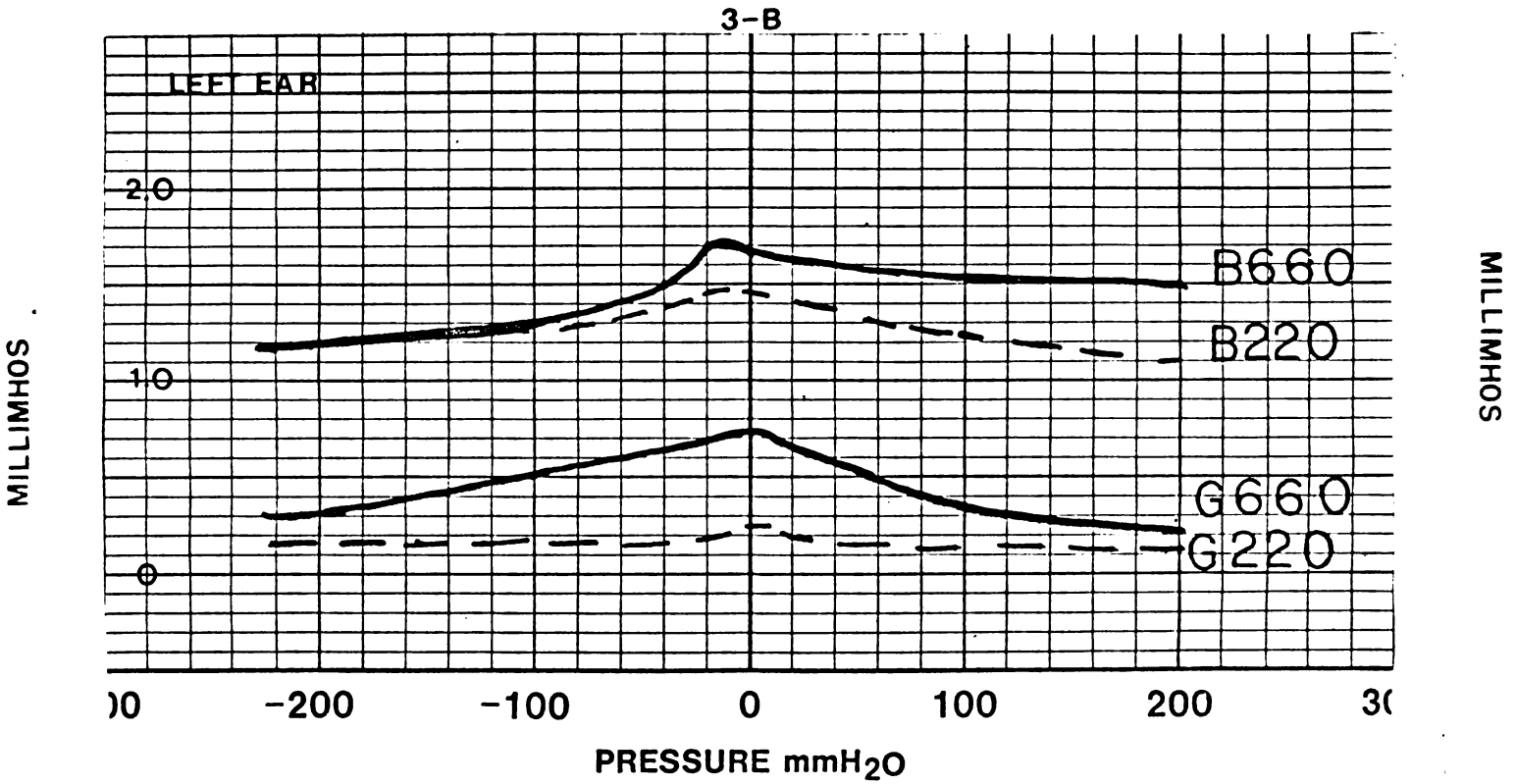


Figure 3-B: Normal otoadmittance findings for the subject's left ear.

Brief tone audiometry was conducted at 500, 1000, and 2000 Hz at 500, 200, and 20 msec durations (Wright, 1968). Integration functions were found using Bekesy tracings for each test frequency by comparing the midpoints of the excursions for each duration (See Table 4).

Table 4: Brief tone thresholds computed by averaging the midpoints of the excursions for the frequencies 500, 1000, and 2000 Hz.

RIGHT EAR								
msec	500 Hz	1000 Hz	2000 Hz	msec	500 Hz	1000 Hz	2000 Hz	
500	12 dB	6 dB	15 dB	500	18.7 dB	20.7 dB	24.37 dB	
200	15	9	12	200	21.1	22.5	25	
20	20.8	13	20	20	26.1	24.5	30	

The differences between the long and short durations were approximately 6 dB for the right ear and approximately 5 dB for the left ear. (See Table 5). An ear without cochlear lesion is expected to demonstrate a 10 dB increase in threshold as the stimulus duration is decreased by a factor of 10 (Pedersen, 1974). This pattern of energy integration has not been observed in individuals with cochlear lesion. Persons with inner ear disorder display an integration function of less than 10 dB when stimulus duration is decreased by a factor of 10 (Harris, Haines, and Myers, 1958).

Table 5: The differences between 500 msec and 20 msec and between 200 msec and 20 msec tones are shown.

RIGHT EAR				LEFT EAR			
msec	500 Hz	1000 Hz	2000 Hz	msec	500 Hz	1000 Hz	2000 Hz
500-20	7.2 dB	7 dB	5 dB	500-20	7.4 dB	3.8 dB	5.62 dB
200-20	4.2	4	8	200-20	5	2	5
AVERAGE	5.7	5.5	6.5	AVERAGE	6.2	2.9	5.31

The initial pitch match task resulted in a bracketing of the frequency range in which the final pitch match task could be conducted. The range for both ears was 1000 to 4000 Hz. The final match consisted of an averaging of three separate trials for each ear. Testing both ears in this manner served as a reliability check for each ear. For the right ear the three trial average was 1425 Hz. The trials for the left averaged 1365 Hz. These frequencies coincide with the same category as those with conductive hearing loss (Graham and Newby, 1962; Nodar and Graham, 1965). The right ear was, however, at the upper limits of the conductive range. The pitch match tasks for both ears did not coincide with the frequencies matched by the subjects with Meniere's disease.

Central Test Results

Time compressed Northwestern University Auditory Test Number Six, Form B (Tillman and Carhart, 1966) was administered at 0%, 30%, and 60% time compression. At 0% and 30% time

compression, discrimination scores were 100% bilaterally. In the 60% time compression condition, discrimination for speech was 86% for the right ear and 84% for the left ear. Poor discrimination under extreme levels of time compression was not observed. Therefore, these results were not indicative of central auditory lesion because individuals with lesion demonstrate very poor discrimination in the 60% time compressed condition.

The Northwestern University Auditory Test Number Six Form A was filtered to pass all frequencies below 500 Hz and to cut all those above 500 Hz. The rejection rate was 32 dB in the first octave (Lynn and Gilroy, 1972) with no measurable response at 1000 Hz. Speech discrimination in this condition was 28% and 18% for the right and left ears respectively. This resulted in a 10% difference between ears, and was considered a significant difference. Since the task was thought to be difficult for normal listeners, a group of normal hearing young adults was also tested. (See Table 6).

Table 6: Percentage correct for three conditions of time compression (0%, 30%, and 60%) and filtered speech.

PERCENTAGE TIME COMPRESSION	0%	30%	60%	FILTERED SPEECH	
RIGHT EAR	100%	100%	86%	RIGHT EAR	28%
LEFT EAR	100%	100%	84%	LEFT EAR	18%

Findings indicated that normal hearing subjects scored below the norms established by Lynn and Gilroy. All subjects scored well below the range of 40 to 72% correct. Twenty percent of the subjects exhibited a difference between ears of 8% or more. (See Table 7). Therefore, a normal hearing population found this task to be difficult. The efficacy of this test with 32% rejection rate for identification of central auditory disorders appears to be too difficult since individuals without temporal lobe lesion perform poorly on this test.

Table 7: Mean, median, mode, range, and differnces of scores for ten normal hearing young adults.

MEAN	MEDIAN	RANGE	DIFFERENCES	MODE
All ears: 17.3%	16%	8-32%	--------	2%--2 4%--3
Right ears: 16%	16%	8-26%	5.4%	6%--3 8%--1
Left ears: 18.6%	16%	8-32%	5%	12%--1

The Staggered Spondaic Word test (Katz, Basil, and Smith, 1963) was another test used to identify central auditory lesion. The subject showed less than 1% total SSW error (0.86%). This indicated that a temporal lobe disorder did not appear to exist in this subject as measured by the Staggered Spondaic Word test since the competing speech signal did not interfere with discrimination.

Dichotic listening ability was assessed with the Simultaneous Dichotic Consonant-Vowels (Berlin, Lowe, Thompson, and Cullen, 1968). Two randomizations were given to the subject. Hemispheric dominance was not demonstrated. An equal number of responses were obtained for each ear with both randomizations. (See Table 8).

Table 8: Response to two randomizations of dichotic consonant-vowel syllables. Correct responses are noted with an asterisk(*). Total number of correct responses is noted at the bottom of each column.

RANDOMIZATION	I	J	
RIGHT EAR	LEFT EAR	RIGHT EAR	LEFT EAR
*pa	ba	ga	*ka
*ka	pa	*ta	*pa
*ga	ta	ta	*ga
*da	pa	*da	ga
*ka	da	da	pa
ba	ta	pa	*ba
ga	pa	ba	*ta
da	*ga	*ba	*ga
ba	*ka	pa	ba
pa	*da	*ka	*ba
*ba	ga	*ba	pa
*pa	ga	ga	pa
ta	ba	ga	*ba
ka	*ga	ga	ta
*ka	ta	da	*ba
ta	*da	*ga	da
ga	*ka	*ka	ta
*ta	pa	*ba	*da
ba	pa	ba	*ka
da	*ka	*ka	da
ta	ga	da	*ka
ga	ba	*ka	pa
da	ta	pa	ga
ka	*ba	*ta	*ba

Table 8: (continued)

RANDOMIZATION	I		J	
	RIGHT EAR	LEFT EAR	RIGHT EAR	LEFT EAR
	*ga	da	*ta	da
	*da	*ba	*ta	ka
	pa	ka	*ka	*ga
	ta	*ka	*pa	*da
	*ba	*da	*pa	*ta
	pa	*ta	da	ta
TOTAL	12	12	16	16

CHAPTER IV

DISCUSSION

Peripheral Testing

The subject exhibited peripheral hearing that was at the lower limits of normal hearing (Davis and Kranz, 1964). The subject reported noticing a decrement in acuity following treatment for rheumatoid arthritis which consisted of a daily intake of 19-20 aspirins. Although the routine test battery consisting of puretone air conduction threshold determination, speech reception threshold determination, and word discrimination testing indicated hearing to be within the normal range, the subject did report difficulty with her hearing and complained of continuous tinnitus.

With the exception of brief tone audiometry, the peripheral auditory test battery did not indicate cochlear or retrocochlear involvement. Tone decay and SISI testing, at high and low sensation levels, suggested a normally functioning inner ear and retrocochlear processes. Normal middle ear integrity was confirmed by impedance and otoadmittance measures. Therefore, even with a complete test battery as complete as this, no of site of lesion could be determined in this individual with borderline normal hearing. However,

brief tone audiometry either appeared to be a more sensitive measure for an individual with hearing at the lower limits of normal hearing or was the unusual finding since it was the sole indicator of cochlear site of lesion.

The ear integrates energy below critical duration in logarithmic manner in the subject without cochlear pathology. Decrement of stimulus duration by a factor or 10 results in an elevation in threshold of 10 dB. But, persons with cochlear lesions integrate energy by a smaller factor (Garner, 1947). In this subject, decrement in stimulus duration resulted in an increment in threshold by approximately 5-6 dB, an increase similar to that seen in patients with cochlear disorder. These results are consistent with those found by Pedersen(1974) in a population of rheumatoid arthritics undergoing salicylate intoxication. Thus, even though other test procedures did not reveal a site of lesion, brief tone audiometric results did suggest cochlear site of action.

Information regarding salicylic action on the auditory system was not definitive as the result of the pitch match task findings. This subject matched the tinnitus perceived to the frequencies 1365 and 1425 Hz. Comparison of these findings with those of Graham and Newby (1962) and Nodar and Graham (1965) shows that this subject appears to be experiencing a representation of tinnitus similar to patients with conductive

pathology. Although the subject's matches fall into the category of those with conductive pathology, the results for the right ear were 25 Hz from the upper limit of the category and those for the left ear were 100 Hz from the upper limits. Since this frequency range also overlaps with the frequencies of the sensorineural category, site of salicylic action cannot be drawn from pitch match results.

Central Testing

Central auditory test results did not appear to demonstrate lesion to the central auditory mechanism by salicylates. Under the extreme condition of 60% time compression , the subject showed performance consistent with normative data (Beasley, Schwimmer, and Rintelmann, 1972). The reduction in scores was minimal and within normal limits.

Filtered speech results of this subject compared to the normative data collected in this study did not evidence involvement of the central auditory system. Although the findings obtained here were not commesurate with those found by Lynn and Gilroy (1972), Williford (1967) noted that filter characteristics can make a large difference in the results obtained. He cautioned that if the clinician chooses to use filtered speech as a part of the test battery, collection of normative data for the individual tape of filtered speech must be made

before application in a test situation. Williford also reported that when speech (Northwestern University Auditory Test Number Six) was filtered using a cut-off frequency of 510 Hz and a rejection rate of 30 dB per octave at approximately 45 dB SL SRT, normal hearing individuals without temporal lobe disorder score 18.9% correct. His results are consistent with those found in this investigation.

The Staggered Spondaic Word (SSW) test and the Simultaneous Dichotic Consonant-Vowel test were also indicative of normal auditory functioning at the cortical level. The total SSW error was minimal. And, though the subject did not exhibit any tendency for cortical dominance, this is not uncommon.

The results of the central test battery suggest that auditory pathology on the cortical level due to salicylate intoxication did not exist in this subject. However, these results must be interpreted cautiously. Other tests of central auditory functioning may yield additional information regarding the effects of salicylates on the higher centers of the auditory system. The tests in this battery evaluated temporal lobe function. Additional tests evaluating brain stem function may indicate some interaction at that level.

CONCLUSIONS

Data analysis regarding the pitch match was quantitative in nature. The subject matched her tinnitus to a frequency range compatable with individuals with middle ear pathology. However, these frequencies were also reported by Graham and Newby (1962) for patients with sensorineural lesion. Since the findings of a single subject were to be considered, determination of conductive or sensorineural category could not be made on the basis of pitch match.

Brief tone audiometry suggested cochlear site of lesion. Tone decay and acoustic reflex decay results did not reveal abnormal decay of acoustic stimuli. The SISI tested at high sensation levels (Thompson, 1963) also was not indicative of retrocochlear site of lesion.

In summary, the results obtained from brief tone audiometry appear to confirm Waltner's (1955) hypothesis that the transient hearing loss in this subject may be the effect of an increase in pressure within the cochlea or the effect of enzyme inhibition (Myers and Bernstein, 1965). Therefore this may be considered to be a problem of inner ear conduction due to either enzymatic changes or pressure build up in the cochlea.

Histopathological studies have not demonstrated consistent changes in cochlear or retrocochlear structures. Audiometric findings suggest that there is pathology in the inner

ear which impedes the efficient transmission of acoustic stimuli. The tinnitus and transient hearing loss seem to be the result of breakdown in intralabyrinthine fluid transport.

The conclusions from this study must be interpreted with caution. This was a single case study, therefore application to a larger population may be erroneous. However, possible sensitivity of brief tone audiometry should be investigated further. Support for the aforementioned hypotheses can now be seen with audiometric data.

IMPLICATIONS FOR FUTURE RESEARCH

From this study, two implications for additional research can be made. These suggestions are:

1) further examination of the effects of salicylates on the auditory system should be conducted with the test battery used in this study and a larger population.

2) additional study on the effectiveness of brief tone audiometry as an effective determiner of subclinical and/or slight cochlear lesions.

APPENDICES

APPENDIX A

CASE HISTORY

AUDIOLOGY ADULT HISTORY

NAME NR BIRTHDATE 12/26/35 AGE 39
OCCUPATION Claims Worker

Do you suspect that you have a hearing loss? Yes
When did you notice the hearing loss? After return from the hospital for treatment for rheumatoid arthritis.
Did your spouse (family) notice the loss before that time? No
What do you think caused the hearing loss? Aspirin
Has the hearing loss changed since its onset? Yes
If so, how? As the aspirin was decreased the hearing loss lessened.
Have you ever worked in a noisy place? No. Did you have to shout to be heard by a person less than three feet away? No. How often did you wear ear protectors? ----
How many years did you work there? -----
Have you ever used guns? No. Snowmobiles? No. Noisy power equipment? No.
Describe the present hearing problem (without a hearing aid):
Which ear has the hearing loss? Both
Does the hearing loss fluctuate(change) from day to day? No.
Do you have difficulties hearing the telephone from another room? Yes. Which ear do you use with the telephone? Left. Have you always used this ear for the telephone? Left. Have you always used this ear for the telephone? Yes. When did you change and why? ------ .
Do you have to have the speaker talk loudly? No.
Do you have difficulties hearing the doorbell from another room? No bell. If in the same room? ----------.
Is the speech of your family clear to you? Yes.
Is the speech of others clear to you in a noisy room? No.
Does your hearing loss interfere with your work? No
Does your spouse (family) think you ignore or misunderstand them? No.
Describe the noises in your ear: Like a pond with crickets creaking at night but no frogs.
Are the noises constant or intermittent? Mostly there.
Which ear? Both. When are they most noticeable? First arising and after taking aspirin
What do you do in resonse to loud sounds? Jump.
Have you ever tried a hearing aid? No. What kind? ----- How long? ------ During what activites did you use the aid? --------- What problems did you have in using the aid? ----------

Do you watch people's lips in order to understand their speech? No. Did you ever take lipreading lessons? No

Have you ever had a hearing test before? Where? Yes, last year -- State Health Screening test.

Have you ever had a medical evaluation of the hearing problem? Where? When? By whom? No.

What other medical problems do you have? Rheumatoid arthritis. For how long? Since May, 1975.

Do you get dizzy? Yes. How long does it last? Seconds. Do you feel lightheaded? Or, do you feel that you or the room is spinning? Lightheaded. How often do you get dizzy? Not very often. Do you have some warning (aura) about an impending dizzy spell? No. What causes the dizziness? Do not know. What do you do to stop the dizzy spell? Nothing.

Do you have any numbness in your face? No. Which side? ------ At what age? ----- Do you have any difficulty chewing or swallowing? No

Has your voice changed in any way? No.

Do you take aspiring daily? Yes. How many do you take each day? 19 How often do you take aspirin each day? Four times.

What other medications do you take? Gold shots. How often? Once each week. For how long? ----------

Have you ever taken any of the following drugs:

Kanamycin No For how long? ----------
Quinine No For how long? ----------
Streptomycin No For how long? ----------
Dihydrostreptomycin No For how long? ----------

Have you ever been hit on the head and knocked out? No. Which side? ------- At what age? ------- What happened? ----

Did you notice any difficulties with your hearing after the measles? No. The mumps? No. Scarlet fever? No. Or, chicken pox? No

Who else in your family has a hearing problem? No one. What is the believed cause of the hearing loss? ----------

What serious illnesses have you had in the past? None

Do you have any difficulties in fine hand coordination? -----

Can you see well enough to read a newspaper? Yes.

Have you ever had earaches? Yes. At what age? 30's. Were they medically treated? ----- Did you ever have a tonsilloadenoidectomy? -Yes.

Have you every had surgery on your ears? No. When? ---- For what reason? ----

Who is your physician? Dr. R.S. (Rheumatoligist): Dr. G.P and Dr. D.L. (Family Doctors)

ADDITIONAL COMMENTS----------

I did not notice any hearing loss until I returned from the hospital from being treated for rheumatoid arthritis. I was taking 20 aspirin a day and the first thing I noticed was I could not hear the phone when I was in another room. When I was in the same room with the phone the ring had a funny tone. I now can hear the phone, buy my hearing is still affected. Sometimes I cannot hear a person that is 5' to 10' away.

APPENDIX B

PITCH MATCH I ANSWER SHEET

ANSWER SHEET

PRESENTATION	HIGHER	LOWER	EQUAL
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			

APPENDIX C, D, and E

NORTHWESTERN AUDITORY TEST NUMBER SIX

FORMS A, B, AND D

N.U. Auditory Test #6 FORM A File no._____

Patient_____ Audiometer_____ Audiologist_____ Date_____

Ear_____	Ear_____	Ear_____	Ear_____
Signal_____ dB HL	Signal_____ dB HL	Signal_____ dB HL	Signal_____ dB HL
Masking_____ dB HL	Masking_____ dB HL	Masking_____ dB HL	Masking_____ dB HL
LIST I	LIST II	LIST III	LIST IV
laud_____	pick_____	base_____	pass_____
boat_____	room_____	mess_____	doll_____
pool_____	nice_____	cause_____	back_____
nag_____	said_____	mop_____	red_____
limb_____	fail_____	good_____	wash_____
shout_____	south_____	luck_____	sour_____
sub_____	white_____	walk_____	bone_____
vine_____	keep_____	youth_____	get_____
dime_____	dead_____	pain_____	wheat_____
goose_____	loaf_____	date_____	thumb_____
whip_____	dab_____	pearl_____	sail_____
tough_____	numb_____	search_____	yearn_____
puff_____	juice_____	ditch_____	wife_____
keen_____	chief_____	talk_____	such_____
death_____	merge_____	ring_____	neat_____
sell_____	wag_____	germ_____	peg_____
take_____	rain_____	life_____	mob_____
fall_____	witch_____	team_____	gas_____
raise_____	soap_____	lid_____	check_____
third_____	young_____	pole_____	join_____
gap_____	ton_____	road_____	lease_____
fat_____	keg_____	shall_____	long_____
met_____	calm_____	late (light)_____	chain_____
jar_____	tool_____	check_____	kill_____
door_____	pike_____	beg_____	hole_____
love_____	mill_____	gun_____	lean_____
sure_____	hush_____	jug_____	tape_____
knock_____	shack_____	sheep_____	tire_____
choice_____	read (reed)_____	five_____	dip_____
hash_____	rot_____	rush_____	rose_____
lot_____	hate_____	rat_____	came_____
raid_____	live_____	void_____	fit_____
hurl_____	book_____	wire_____	make_____
moon_____	voice_____	half_____	vote_____
page_____	gaze_____	note_____	judge_____
yes_____	pad_____	when_____	food_____
reach_____	thought_____	name_____	ripe_____
king_____	bought_____	thin_____	have_____
home_____	turn_____	tell_____	rough_____
rag_____	chair_____	bar_____	kick_____
which_____	lore_____	mouse_____	lose_____
week_____	bite_____	hire_____	near_____
size_____	haze_____	cab_____	perch_____
mode_____	match_____	hit_____	shirt_____
bean_____	learn_____	chat_____	bath_____
tip_____	shawl_____	phone_____	time_____
chalk_____	deep_____	soup_____	hall_____
jail_____	gin_____	dodge_____	mood_____
burn_____	goal_____	seize_____	dog_____
kite_____	far_____	cool_____	should_____
_____% correct	_____% correct	_____% correct	_____% correct

N.U. Auditory Test #6 FORM B File no. _____

Patient _____ Audiometer _____ Audiologist _____ Date _____

Ear _____	Ear _____	Ear _____	Ear _____
Signal _____ dB HL	Signal _____ dB HL	Signal _____ dB HL	Signal _____ dB HL
Masking _____ dB HL	Masking _____ dB HL	Masking _____ dB HL	Masking _____ dB HL
LIST I	LIST II	LIST III	LIST IV
burn	live	sheep	rose
lot	voice	cause	dog
sub	ton	rat	time
home	learn	bar	such
dime	match	mouse	have
which	chair	talk	mob
keen	deep	hire	bone
yes	pike	search	sail
boat	room	luck	rough
sure	read (reed)	cab	dip
hurl	calm	rush	join
door	book	five	check
kite	dab	team	wheat
sell	loaf	pearl	thumb
nag	goal	soup	near
take	shack	half	lease
fall	far	chat	yearn
week	witch	road	kick
death	rot	pole	get
love	pick	phone	lose
tough	fail	life	kill
gap	said	pain	fit
moon	wag	base	judge
choice	haze	mop	should
king	white	mess	pass
size	hush	germ	back
pool	dead	thin	hall
vine	pad	name	bath
chalk	mill	ditch	tire
laud	merge	tell	peg
goose	juice	cool	perch
shout	keg	seize	chain
fat	gin	dodge	make
puff	nice	youth	long
jar	numb	hit	wash
reach	chief	late (light)	food
rag	gaze	jug	mood
mode	young	wire	neat
tip	keep	walk	tape
page	tool	date	ripe
raid	soap	when	hole
raise	hate	ring	gas
bean	turn	check	came
hash	rain	note	vote
limb	shawl	gun	lean
third	bought	beg	red
jail	thought	void	doll
knock	bite	shall	shirt
whip	lower (lore)	lid	sour
met	south	good	wife
_____% correct	_____% correct	_____% correct	_____% correct

N.U. Auditory Test #6 FORM D File No._____

Patient_____ Audiometer_____ Audiologist_____ Date_____

Ear_____	Ear_____	Ear_____	Ear_____
Signal_____dB HL	Signal_____dB HL	Signal_____dB HL	Signal_____dB HL
Masking_____dB HL	Masking_____dB HL	Masking_____dB HL	Masking_____dB HL
LIST I	LIST II	LIST III	LIST IV
laud_____	loaf_____	germ_____	food_____
knock_____	chair_____	talk_____	lease_____
page_____	goal_____	walk_____	hall_____
mode_____	shack_____	ditch_____	rose_____
week_____	far_____	mop_____	doll_____
hash_____	ton_____	hire_____	kill_____
yes_____	witch_____	youth_____	join_____
third_____	rot_____	pain_____	dip_____
whip_____	dab_____	gun_____	red_____
love_____	pick_____	dodge_____	came_____
lot_____	deep_____	sheep_____	hole_____
met_____	match_____	mouse_____	wash_____
limb_____	fail_____	check_____	sail_____
sell_____	said_____	bar_____	mob_____
door_____	wag_____	phone_____	ripe_____
goose_____	pike_____	beg_____	bone_____
sub_____	haze_____	mess_____	such_____
fat_____	white_____	life_____	check_____
kite_____	hush_____	five_____	wheat_____
boat_____	dead_____	seize_____	should_____
choice_____	gaze_____	soup_____	judge_____
chalk_____	live_____	pole_____	gas_____
hurl_____	keg_____	cab_____	bath_____
puff_____	calm_____	shall_____	yearn_____
reach_____	turn_____	chat_____	time_____
raid_____	lore_____	good_____	wife_____
nag_____	thought_____	note_____	thumb_____
pool_____	tool_____	hit_____	have_____
shout_____	rain_____	when_____	neat_____
tip_____	keep_____	luck_____	get_____
size_____	chief_____	tell_____	lose_____
keen_____	bite_____	jug_____	tape_____
jail_____	nice_____	pearl_____	make_____
sure_____	room_____	base_____	tire_____
tough_____	book_____	team_____	near_____
burn_____	numb_____	wire_____	chain_____
dime_____	pad_____	date_____	shirt_____
jar_____	bought_____	road_____	long_____
rag_____	south_____	cool_____	rough_____
death_____	juice_____	search_____	fit_____
bean_____	read (reed)_____	late (light)_____	kick_____
take_____	merge_____	void_____	mood_____
home_____	hate_____	rush_____	lean_____
raise_____	learn_____	rat_____	dog_____
vine_____	soap_____	thin_____	peg_____
which_____	young_____	half_____	sour_____
gap_____	voice_____	name_____	pass_____
king_____	mill_____	lid_____	vote_____
moon_____	gin_____	ring_____	perch_____
fall_____	shawl_____	cause_____	back_____
_____% correct	_____% correct	_____% correct	_____% correct

APPENDIX F

RAW NORMATIVE DATA

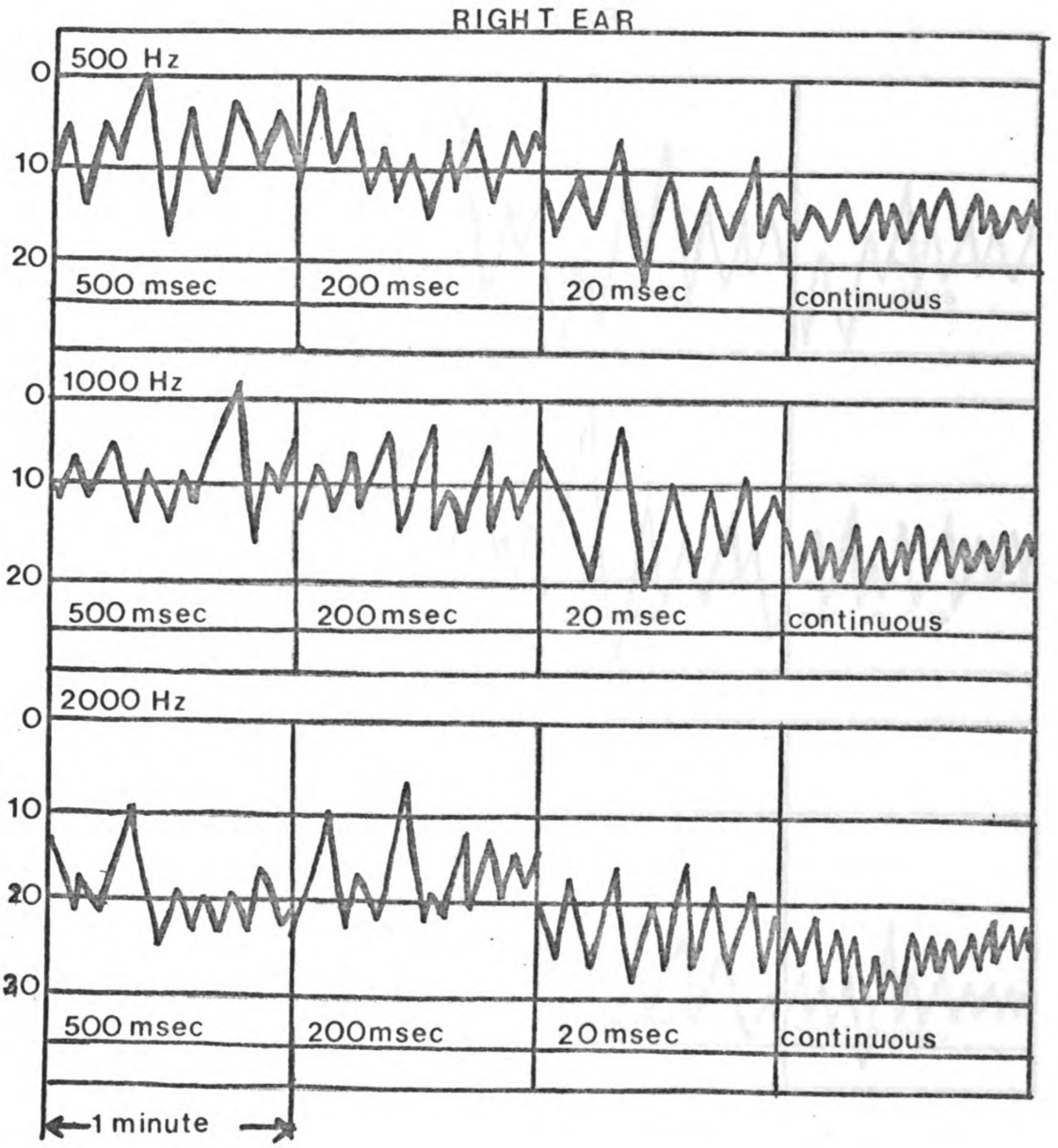
NORMATIVE DATA -- FILTERED SPEECH

FORM A				FORM B			
SUBJECT	RIGHT EAR	LEFT EAR	DIFF.	SUBJECT	RIGHT EAR	LEFT EAR	DIFF
1	18%	30%	12%	1	18%	16%	2%
2	26	32	6	2	12	16	4
3	18	22	4	3	16	20	4
4	14	20	6	4	8	16	8
5	20	14	6	5	10	12	2

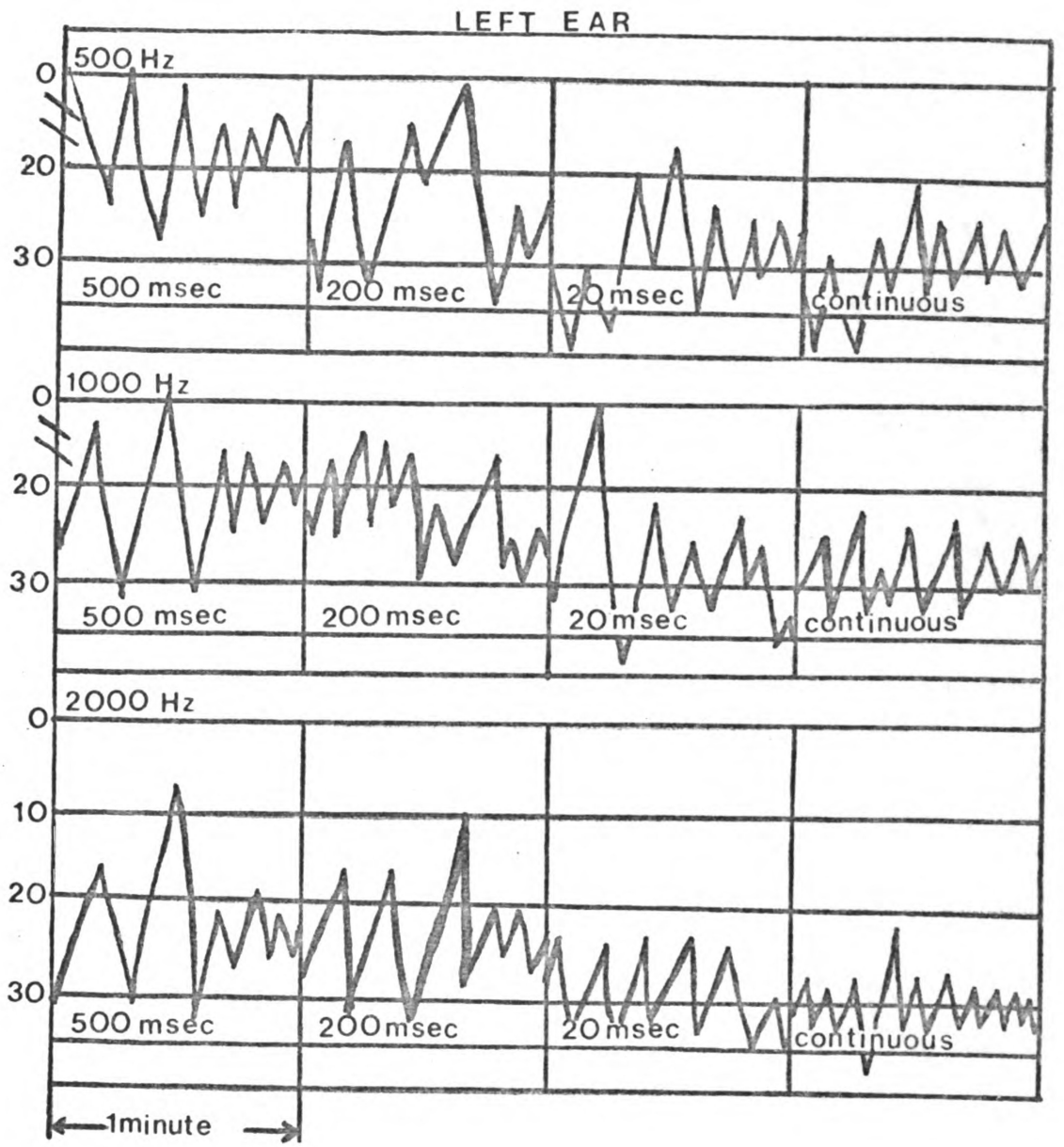
APPENDIX G

BRIEF TONE TRACINGS

HEARING THRESHOLD LEVEL IN DECIBELS: ISO 1964



HEARING THRESHOLD LEVEL IN DECIBELS: ISO 1964



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