

AN ABSTRACT OF THE THESIS OF

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Tinnitus is usually accompanied by a hearing loss; however, many patients with tinnitus present with normal hearing. The hypothesis was proposed that individuals with tinnitus who do not have an accompanying peripheral hearing loss, have tinnitus as a result of a disorder in the Central Auditory Nervous System. In order to test this hypothesis clinically, a central auditory test battery was administered to ten normal hearing tinnitus patients.

Results revealed that three subjects performed normally on the entire central test battery, thus the cause of their tinnitus is unknown. The remaining seven subjects performed abnormally on at least one of the central auditory tests, with results indicating a possible brain stem disorder for five of these subjects and a diffuse lesion involving the brain stem and temporal lobe for one subject. Results were questionable for the one remaining subject.

A number of conclusions were drawn regarding the results of this study, with the final conclusion being the acceptance of the hypothesis.

A STUDY OF TINNITUS PATIENTS
WITH NORMAL PERIPHERAL AUDITORY SYSTEMS

by

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A STUDY OF TINNITUS PATIENTS WITH NORMAL PERIPHERAL AUDITORY SYSTEMS

CHAPTER I

INTRODUCTION

The word tinnitus is derived from the Latin "tinnire" meaning a ringing. In dealing with cases of hearing impairment the audiologist may note a majority of patients whose chief complaint is a "roaring", "buzzing" or "ringing" in the ears. These head noises, called tinnitus, are a common symptom in otologic practice. They are often said to be more disabling to a patient than hearing loss itself.

The subjective location of tinnitus is known to vary among individuals. When localized at the level of the ears, either one or both, it is referred to as tinnitus aurium and can be specifically described as ringing, whistling, steam escaping, etc. Tinnitus cerebri refers to noises heard at various places in the head. It is typified by poor localization, a generalized nonspecific description, and is thought to originate in the cerebral cortex (Nodar, 1972). Tinnitus may be further defined as a sensation of sound for which there is no external source or vibration. Fowler (1941) distinguished two types of tinnitus aurium. One type can be heard by an observer and is called either vibratory or objective tinnitus. The other type is perceived only by the patient and is referred to as either non-vibratory or subjective tinnitus. In the present study, when the term tinnitus is utilized, it will be in

reference to subjective tinnitus.

STATEMENT OF PROBLEM

Tinnitus is a very common symptom whose nature and cause has puzzled physicians throughout history and continues to perplex otologists today. Countless efforts have been made to determine the causes of tinnitus, but these attempts only seem to reveal the diversity and variety of symptoms among tinnitus patients. As Vernon (1977) remarked, the specific cause(s) of tinnitus are not known, however, when tinnitus occurs in a person having a hearing problem of some type, the cause of the ear disorder is also assumed to be causing the tinnitus.

Although tinnitus is primarily associated with an accompanying hearing loss, it is also present in individuals with essentially normal hearing. It is among these individuals that the presence of tinnitus is especially puzzling since the present theories linking the cause of tinnitus to the auditory system do not seem to apply. Therefore, it is evident that further investigation is necessary to define the mechanisms which cause it and the present study is an attempt to investigate the condition of the central auditory nervous system in patients experiencing tinnitus in the presence of a normal peripheral auditory system.

PURPOSE

Tinnitus has been found to accompany all types of diseases affecting the ears from the most trivial and transient to the more serious and intractable. This includes diseases affecting all different parts of the mechanism, extending from the external meatus through the middle

ear, the cochlear and vestibular portions of the inner ear, the auditory nerve to the central connections of the auditory nerve in the brain (Harper, 1951). In Vernon's (1977) opinion, however, there is little known about the causes of tinnitus, with even less knowledge of how any of these diseases produce head noises.

Past research attempts to identify the mechanism or mechanisms responsible for tinnitus have been questioned. Goodhill (1950) remarked that if it were a disease entity, one would approach its study by looking for a single set of circumstances appearing in the histories of all who suffer from it. However, when considering tinnitus as a symptom, the important factor lies in finding a common mechanism by which several pathologies underlying tinnitus act upon the neural pathways to cause the perception of sound by the individual. The author is essentially in agreement with Goodhill's position; however, it seems reasonable that before a common mechanism can be established it is necessary to determine the diseases which accompany tinnitus, as well as the similarities and differences among those with the symptom. Tinnitus is frequently found in conjunction with a hearing impairment. Estimates of the occurrence of this complaint in otologic practice have been reported to be as high as 85%, but may also be common to all individuals to some degree (Fowler, 1944). It seems logical to assume then that tinnitus is a disturbance of the auditory system since only the auditory neural pathway appears able to transmit an impulse that will be perceived at the cortex as sound (Graham, 1958). A review of the literature, however, also indicated that tinnitus is observed among individuals with normal peripheral hearing (Venters, 1953, Fowler, 1913, Seltzer,

1947, Heller & Bergman, 1953, and Graham & Newby, 1962). Normal hearing is generally defined in the literature as a hearing loss no greater than 20dB HL across frequencies 250-8000 Hz, no difference between air and bone conduction responses, normal tympanograms and acoustic reflex thresholds and a speech discrimination score of 90% or better. These measures are the result of a basic audiological test battery designed to test the peripheral auditory system. In the present study, a peripheral loss refers to a hearing impairment due to a disorder prior to the decussation at the level of the cochlear nuclei. The central auditory system, on the other hand, refers to the central pathways above the cochlear nuclei. In a similar manner, the symptom of tinnitus is also thought to be generated in both the central and peripheral systems. As stated previously, tinnitus aurium (heard at the ears) is thought to be peripheral, whereas tinnitus cerebri (heard in the head) is thought to be a central phenomenon.

Tinnitus is a common auditory symptom which is primarily associated with an accompanying hearing loss. However, many individuals with normal hearing have also presented themselves with this complaint. It is with these individuals who have no apparent lesion within the auditory system to cause a hearing loss that the cause of tinnitus is especially questionable.

In a similar manner, Jerger and Jerger (1975), who have done extensive research in the field of auditory disorders, reported that most patients with disorders of the auditory portions of the brain stem and temporal lobe also have normal peripheral hearing; i.e., pure-tone sensitivity, acoustic reflex contractions and PBmax (speech discrimi-

nation) scores within normal limits. The lesions of these individuals were located within the central auditory nervous system, and therefore, normal results on peripheral auditory tests are not surprising.

In addition to the battery of tests designed to assess peripheral auditory function, a separate group of tests are available for testing the central auditory system. These tests are referred to as a central auditory testing battery with speech audiometry being the primary stimulus. Research shows that certain individuals with confirmed lesions at some point in the central auditory pathways are unable to perform as well as would be expected on these tests. The problems these individuals have in processing the more complex speech stimuli appear to be a result of disorders located within the central neural portions of the auditory system. It is reasonable to assume that these disorders of the central nervous system could readily be responsible for initiating the neural activity that produces tinnitus. Therefore, the purpose of this study was to investigate the condition of the central auditory nervous system in subjects experiencing tinnitus in the presence of a normal peripheral auditory system. This was accomplished by obtaining an evaluation of the patient's hearing ability by measuring the peripheral auditory mechanism with a standard audiometric testing battery, by measuring the central auditory mechanism with a central auditory testing battery, and by measuring the subject's tinnitus. The population for this experiment consisted of ten patients experiencing tinnitus who displayed normal hearing.

The hypothesis which guided this study states that those patients with tinnitus who do not have an accompanying peripheral hearing loss,

have tinnitus as a result of a disorder in the central auditory nervous system. The equipment, the methodology and the subjects utilized for this experiment are discussed in Chapter III following a short historical review.

CHAPTER II

HISTORY

Introduction

"Whatever the reason for tinnitus, it has worried physicians for many centuries." (Myers, 1975, p. 64). The truth in this statement is evident in the vast amount of literature on this puzzling and distressing symptom. Attempts to unravel the mysteries of tinnitus are represented in the great volume of journal articles published in this century alone. Researchers have utilized many approaches in an attempt to explain the clinical causes, mechanisms and treatments of tinnitus. These studies afford one an opportunity to have a greater understanding of the prevalence, etiologies and characteristics of tinnitus. However, efforts have proven to be less encouraging in providing information concerning the mechanism and treatment of this symptom.

As stated previously, the purpose of this study is to gain further insight into the mechanism of tinnitus as related to central auditory pathways. Therefore, the following review of the literature includes a discussion of the pertinent information in the areas of tinnitus and central auditory dysfunction.

TINNITUS AURIUM

Prevalence of Tinnitus

The symptom of tinnitus has been a recognized problem for centuries, recorded as early as Hippocrates, ca. 400 B.C. (Engstrom & Graf, 1952). It is experienced by a significant portion of the population and is found in both the presence and the absence of hearing loss. A review of the literature indicated differing opinions on the prevalence of the problem. In reviewing 2000 clinical cases of all kinds of ear diseases, Fowler (1912) reported that 66% of all patients experienced tinnitus. Similar findings were reported by Jones and Knudsen (1928). Venters (1953) placed the incidence of tinnitus relative to aural disease at only 16.5% with the incidence among 1466 cases of deafness being 16%. This drop was related to the progress made in the practice of otology in the past forty years.

Wegel's (1931) impression was that people entirely without tinnitus are extremely rare, and he doubted if such cases existed at all. The explanation offered was that people are not usually aware of it until the noise becomes so loud that it interferes with the perception of weaker sounds. Seltzer (1947), Heller and Bergman (1953) and Riddell (1956) agreed that almost everyone had head noises of some sort, probably unnoticed by normal hearing persons due to the masking effects of environmental noise. These authors have shown that approximately 94% of normal hearing adults experienced tinnitus when placed in a sound proof testing situation.

Other investigators have reported somewhat different results. Graham and Newby (1962) found that 40% of 25 normal hearing subjects

had tinnitus. Patients in their study were audiometrically determined to have normal hearing in contrast with other studies which accepted the subjective opinions of the patients. Graham and Newby allowed no room for error in the test population which they felt explained the discrepancy in the findings between studies. Vernon (1978) reported that tinnitus patients who have normal hearing are rare with only 8 to 10% of 513 patients having normal hearing with tinnitus.

Hatton (1965) reported that no age group is immune to tinnitus, but it is found more frequently in people over 50 years of age. Of his subjects, the 51-70 year age group supplied 49% of the total, 10% were between 11-30 years, and 1% were in the first decade. Men and women were found to be equally affected and no race or color seems to escape it. Other studies were generally in agreement with these findings (Fowler, 1948, Venters, 1953, and Vernon, 1978).

The lack of agreement found in the many research efforts indicates that there is no clear idea of exactly how many people do suffer from tinnitus. The U.S. Department of Health, Education and Welfare (1968) in an attempt to determine the prevalence of tinnitus in the U.S. population, released statistics that provided a better estimate of the scope of the problem. They reported that 36 million adults in the U.S. experienced the problem and 7.2 million (20%) complain of severe tinnitus. This means that approximately 20% of the population suffers from some type of tinnitus with 4% being of a severe, debilitating nature.

Etiology of Tinnitus

The possible causes of tinnitus have not been confined to any specific area of the ear (Alpiner, 1968). This leaves an infinite

number of possibilities open to the specialist in the process of differential diagnosis. Due to the number of pathologies associated with tinnitus, Goodhill (1950) described it as a symptom, not a disease, and stated that it must be treated as such. By including tinnitus as one of the common characteristics of otologic disturbances (others being otorrhea, otalgia, vertigo, and deafness) it has influenced the trend toward an etiological diagnosis. Furthermore, due to the complexity of the anatomic pathway involved in auditory physiology, the assessment of tinnitus as a symptom is significant in the differential diagnosis of otologic disturbances. According to Atkinson (1947), the importance of tinnitus as a symptom is two-fold in that it implies an active lesion, and also involves the threat of impending or increasing deafness.

Morgan (1877) presented a paper on tinnitus aurium and outlined theories of the various conditions which produced tinnitus and its treatments. Comparatively, these theories are the same as those presented by researchers today. Exhaustive lists outlining the associated pathologies are provided, suggesting that the etiological diagnosis must be based upon anatomic localization and pathologic physiology. Goodhill (1952) remarked that tinnitus may arise from any location within the auditory pathway from the external ear to the auditory cortex, and that any histopathologic abnormality in the auditory pathway may be responsible for the genesis of the symptom. Table 1 suggests the multiplicity of anatomic and pathologic factors important in tinnitus etiology, and in general is representative of the lists generated by other researchers.

ETIOLOGY OF TINNITUS

ANATOMIC LOCATIONS

External Auditory Canal

Tympanum

- a. Memb. Tymp.
- b. Tymp. Muscles
- c. Ossicles
- d. Tymp. Plexus
- e. Tymp. Vessels
- f. Eust. Tube
- g. Fenestrae Rotund and Ovale

Cochlea

- a. Perilymph
- b. Endolymph
- c. Organ of Corti

VIIIth Nerve

- a. Spiral Ganglia
- b. Trunk

Intracerebral

- a. Ventral and Dorsal Nuclei
- b. 2nd Order Neurons
- c. Med. Gen. Body
- d. 3rd Order Neurons
- e. Cortex

PATHOLOGIC LESIONS

Anemia

Hyperemia

Edema

Serous Exudate

Mucous Exudate

Purulent Exudate

Hemorrhage

Inflammation

Allergy

Neuronitis

Necrosis

Fibrosis

Tumor

Aneurysm

Capillary Fragility

Stasis

Sludging

Vascular Spasm

Vascular Sclerosis

Vasomotor Paralysis

Table I. Anatomic and Pathologic Factors Important in Differential Diagnosis of Tinnitus. (Goodhill, 1952, p. 780.)

In determining the etiology of tinnitus, hearing loss and its associated pathologies have commonly been considered as a possible cause. Deafness and tinnitus may be coincidental, but according to Fowler (1944) may be far apart etiologically. Venters (1953) supported this concept in a study of 150 cases which showed little relation between the degree of deafness, the severity of tinnitus, and whether it was intermittent or constant.

Goodhill (1950) described tinnitus as a double diagnostic problem and stated that any management of it which does not take into consideration the etiologic diagnosis along with the psychosomatic weighting factor will usually be ineffective. The psychosomatic diagnosis may be approached from two standpoints: 1) the emotional threshold or sensitivity level of the patient, and 2) specific psychodynamic problems. Goodhill believed that there is no correlation between the actual intensity of tinnitus as measured audiometrically and the amount of suffering of the patient. It is therefore important to assess the excitability level of the patient when determining their reaction to the tinnitus. Also helpful in diagnosis is the determination of a patient's phobias, anxiety states and conversion mechanisms. House (1977) also recognized the relationship between psychosomatic symptoms and tinnitus and has found success in reducing symptoms with biofeedback training. Many of his patients found that they did have emotional difficulties and were misplacing much of their anxiety and depression on their tinnitus. In contrast, Toglia et al (1969) remarked that it is a dangerous tendency to attribute complaints such as dizziness, tinnitus, etc. to psychodynamic and emotional etiologies, rather than to physio-

logic causes. Indications were that this could easily happen to the patient who has no hearing loss and no apparent physiological auditory disorder.

The list of etiologies and various conditions producing tinnitus are extremely impressive, but at the same time the task of diagnosis becomes increasingly difficult. There are many individuals with tinnitus who suffer from auditory disorders; however, some people appear to be relatively immune to any or all of these causes in relation to their tinnitus. Many persons have no tinnitus even under extreme provocations while others under little provocation suffer from marked tinnitus (Fowler, 1948). Therefore, it appears that there is pronounced idiosyncrasy or differing sensitivity in persons as to the degree of irritation at the time necessary to cause tinnitus. Tinnitus does accompany some pathological ear conditions, but according to Seltzer (1947), since they are not constantly present, ear pathology cannot be the determining factor. Wegel (1931) is under the impression, however, that the presence of tinnitus indicates an active or progressive lesion, and that cessation of it is an indication that progress of the degeneration or atrophy of tissue has been arrested.

Characteristics of Tinnitus

The characteristics of tinnitus have been determined subjectively with the patient giving a description of the sound and its severity, and objectively by measurement of the psychophysical parameters of frequency and intensity. In obtaining a description, it is necessary to remember that tinnitus is seldom uniform in character, and is dependent upon the situation, extent, severity, rapidity, degree of

remission, and the chronicity of the irritations (Fowler, 1912).

Subjective Description

The subjective noises of tinnitus have frequently been described as high-pitched tones, chirping, singing sounds, escaping steam, ringing, buzzing, pulsating sounds, crickets, crackling, hissing, humming, static, whirring, whistling, and blowing. Beethoven once described his affliction as the "roaring deafness" (House, 1977).

According to Fowler (1941), biochemical irritations producing tinnitus set up patterns quite different from the normal neural responses to sound vibrations. Tinnitus is not a real sound, but an illusion; thus, it seldom has exactly the same timbre of a pure-tone. This is one reason why patients experience difficulty in describing tinnitus subjectively.

Goodhill (1952) suggested that when a patient is asked to describe a subjective sensation such as tinnitus, the accuracy of the description will depend upon many factors including native intelligence, analytic subjective ability, and degree of semantic articulateness. Of specific importance was the patient's acquaintance with acoustic or musical terms. It was found that a patient with slight musical experience would have difficulty in pitch discrimination and in pure versus complex tone differentiation. His conception of intervals, octave, and other scale relationships would also usually be poor. Atkinson (1944, p. 743) wrote, "The patient likens his tinnitus for the purpose of description to sounds with which he is familiar, such as the roar of the sea, the hum of machinery, the hiss of steam escaping, yet he knows it's not the same as any of these sounds. It has a different

quality, often an unpleasant quality."

Graham (1960) found no correlation between the descriptive terms used by his subjects and the objective measurements of their tinnitus. He concluded that subjective descriptions offered little assistance in determining the psychophysical parameters of pitch, loudness and severity of tinnitus. Reed (1960) noticed that the subjective description of the tinnitus by his subjects did not correspond to the type of hearing impairment or probable site of lesion, and concluded that the description was of no value in the audiological evaluation. Kafka (1934) determined a relationship between the subjective description of his patients' tinnitus and its origin. For example, gurgling, boiling and whistling qualities were associated with diseases of rhinopharyngeal catarrh with the involvement of the ears. Vernon (1978) utilized the subjective evaluation to determine characteristics which could not be measured objectively. Patients were asked to rank the severity of the tinnitus on a scale of one to ten, with ten being the worst. It was found that the severity of the tinnitus does not correlate to the loudness. Patients normally matched the loudness of their tinnitus to a sensation level of 5 to 10dB, which is very mild considering that normal conversation levels are heard at 55 to 60dB.) Each patient is also questioned as to where the tinnitus is located. Vernon stated that eventually the initial site of trouble may be located, and the localization of the tinnitus sensation may be useful in this determination. It was found that patients were able to locate their tinnitus, with 37% being in one ear only (16% in the right ear and 21% in the left ear) and 58% reporting the problem in both ears.

Subjective descriptions are of value in understanding the patients'

ideas of what his own tinnitus sounds like to him. However, for tinnitus to be diagnostically significant, it appears necessary to obtain objective measures of the psychophysical parameters of this symptom.

Objective Measurement

When studying the process of tinnitus in humans, research shows that the investigation has been handicapped by the subjective nature of the symptom, the multiple theories on etiology and the frequent emotional instability of the patient. In addition, human study is further handicapped by the fact that the site of origin of tinnitus may be in the inner ear, the auditory nerve, or central auditory pathways, all of which are inaccessible to direct examination in the living (Reed, 1960). These drawbacks have caused investigators to develop alternative approaches in the study of tinnitus. Venters (1977, p. 43) wrote:

The need for an accurate and quantative description of tinnitus is essential in order to differentiate among different types of tinnitus as well as to have an objective means of evaluating various procedures designed to provide relief. It is not possible to accept patient reports of tinnitus at face value, for very few patients have sufficient knowledge about the characteristics of hearing experiences.

A review of the literature revealed that many contributions have been made to the subject of audiometric measurements of tinnitus. Included in these are the works of Josephson (1931), Wegel (1931), Fowler, Sr. (1912, 1938, 1944, and 1948), Mortimer (1940), Goodhill (1952, and 1954), Flottorp and Wille (1954), Reed (1960), Graham and Newby (1962), and Vernon (1978).

Wegel (1931) reported an account of some measurements made on one of his own ears, constituting a study of what he termed "the abnormal sensation areas". Measurements of his tinnitus found it was not very

loud, but it was continuous and sounded like a 3600 or 3700 cps tone. Wegel determined that he could plot a frequency and intensity curve of pure tones which masked his tinnitus. By increasing the intensity of a 3400 cps tone, its appearance at threshold was marked by a change in character of the tinnitus. When combined with the tinnitus, it produced a definite discord, whereas intensities above its threshold caused the tinnitus to be entirely masked. Wegel further observed that when the pitch of the tone was 3620 cps, threshold intensity caused the appearance of an unmistakable "beat" between the stimulus and the tinnitus. As the tone was decreased in pitch, the beats became faster resulting in the previously described discord upon reaching 3400 cps. Increasing the pitch higher than 3620 cps again caused the fast beats at threshold; however, when the intensity was increased to a certain range, a complete silence resulted. A careful search was made throughout the frequency range of 100 to 15,000 cps, which revealed no other areas where beats or distortion occurred. The transition between these masking boundaries was found to be abrupt and quite definite. Josephson (1931) reported similar findings to Wegel's. He further noted that the tinnitus was masked by increasing the intensity of the stimulus, and was found to be inhibited for a certain period of time after stimulation had ceased.

Goodhill (1952) recognized the difficulty in the task of subjective description of tinnitus. An attempt at audiometric analysis was recommended to determine the acoustic properties of the tinnitus, along with his tinnitus identification test. This recorded test was artificially created by sound technicians to mimic as closely as possible the acoustic components described by patients with tinnitus. The patients listen to the recording, picking out the components which most closely resembles

their sounds. Goodhill recognized the inadequacies and subjectivity of his test, but believed it to be equal in accuracy to other auditory tests which require judgements by the patient, i.e. the pure-tone threshold test and recruitment tests.

Many procedures have been reported for measuring the pitch, loudness and masking of tinnitus. These methods which attempt to objectively measure the characteristics of tinnitus are discussed below.

The pitch of a sound is the psychological attribute of that sound which corresponds to its frequency. That is, frequency is a physical, measurable, attribute of sound, while pitch is the psychological, subjective correlate of it. We place great importance upon the identification of the pitch of each patient's tinnitus. That information helps us determine whether or not masking is appropriate for the patient and if so what kind of masking will be needed. (Vernon, 1978, p. 2)

Fowler (1944, 1948) described how the frequency band or tone of tinnitus can be determined objectively by a comparison with similar applied frequencies. By utilizing a bracketing technique, matching begins by applying the discrete frequencies alternately, well above and well below the frequency tone or band of the tinnitus. The distance is lessened between the two exploring tones until they narrowly bracket the frequency band of the tinnitus. In this way the patient is able to determine within narrow limits the frequency which corresponds closest to that of his tinnitus. Fowler recommended that the exploring tones be intermittently applied (preferably to an opposite uninvolved ear) as an identical match in frequency may cause the test tone and the tinnitus to appear as one tone. In addition, when determining pitch, it is important to deliver exploring tones at intensities corresponding closely to the intensity of the tinnitus.

Fowler's procedure appears to have functioned as a guideline for

further studies on pitch identification. Reed (1960, p. 86) stated a similar but more detailed account of pitch identification.

After preliminary discussion with the patient in which the general objectives and aims of the matching procedure were explained, an attempt was made to deliver to the patient a sound somewhat similar to his own sound, as based on his description of his tinnitus. For example, if he described a high-pitched ringing tinnitus, he was given a pure-tone in the higher frequencies, e.g., 5000 to 6000 cps. If, on the other hand, he described a tinnitus sounding like escaping steam he was given a wide band of frequencies - - nearly "white noise" in character. The patient was then asked to describe in his own terminology the difference between his tinnitus and the mechanical noise. He was allowed to do this in his own terminology, and an attempt was made to arrive at an approximate match of the main pitch of his tinnitus by having him explain whether the applied sound was too high, too low, too deep, etc. The intensity of the applied sound was then roughly adjusted to match that of the patient's tinnitus. The bandwidth of noise on either side of the central frequency was then varied until the resultant sound resembled the patient's tinnitus. The central frequency was again varied upward or downward and smaller variations of bandwidth were made until the patient had chosen approximately the same matching spectrum of frequencies three times in a row.

Reed felt that this procedure was worth the effort since in most cases it was possible to determine a nearly perfect match of the patient's tinnitus. Patients with unilateral tinnitus received the exploring stimulus in the ear opposite the tinnitus, whereas cases with bilateral tinnitus received the stimulus in the ear being matched. These patients were asked to compare the applied noise with their tinnitus by alternately applying and removing the earphone.

Graham and Newby (1962) outlined a procedure which includes an initial and final pitch match of tinnitus. The initial pitch match test was a gross evaluation of the pitch characteristics of a subject's tinnitus and was made using the eleven frequencies available on the audiometer. This test was used to delineate a frequency range for the final assessment of the pitch of the tinnitus. The final pitch match utilized a

bracketing procedure similar to Fowler (1944, 1948), Reed (1960) and Vernon (1977), and will not be repeated here.

Vernon (1977) utilized a forced-choice bracketing technique in identifying the frequency characteristics of both tonal tinnitus and noise tinnitus. He stated that both types have an identifiable pitch, with the pitch of the noise type being more difficult to determine. In utilizing the matching procedure, Vernon (1978, p. 2) stated that there are two very important points to consider.

First of all, the physical sounds must be at the loudness level of the patient's tinnitus. If the physical sounds are too intense the patient will experience great difficulty in matching the pitch and, for some it is an impossible task. Secondly, the first identification selected by the patient is very apt to be off by about one octave. Thus, one must check various octave points around the identified point. If, for example, we have been moving steadily upward in pitch and at 2000 Hz the patient indicates a match with his tinnitus it is then imperative that 4000 Hz also be checked. When this matching is properly done it turns out that the octave above the first identification is the true identification in about 7 out of 10 cases.

The following table shows the agreement between the two studies concerning pitch identification of tinnitus in two groups of patients.

Table 2. A Comparison of the Pitch Characteristics of Tinnitus for Two Studies by Reed (1960) and Vernon (1978).

PITCH DISTRIBUTION OF TINNITUS

Pitch in Hz	Reed	Vernon
Below 2000 Hz	21.5%	21%
Below 2000 & 7000 Hz	64.0%	63%
Above 7000 Hz	14.5%	16%

In relation to hearing loss, Graham and Newby (1960) found no significant difference in the frequency characteristics of tinnitus between sensorineural and mixed losses; however, significant differences were seen between these two groups and those with conductive hearing losses. The sensorineural and mixed hearing loss groups matched the pitch of their tinnitus to a wide frequency range from 155-7800 cps for the former and from 40-6500 cps for the latter. In contrast, the conductive hearing loss group matched the pitch of their tinnitus to a restricted frequency range of 120-1400 cps. The normal hearing subjects who experienced tinnitus seemed to follow the distribution of the sensorineural and mixed hearing loss groups in matching the pitch of their tinnitus.

Atherly et al, (1968) reported that when subjects were exposed in one ear to a 1/3 octave band noise centered at a specific frequency for five minutes at 110dB SPL, the resulting pitch of the noise-induced tinnitus was found to bear a constant relationship to the frequency of the stimulus. Vernon (1978) was interested in the effects of excessive amounts of noise on the frequency of tinnitus. Originally, it was speculated that noise-induced tinnitus would share some common characteristics, such as a similar pitch. However, in a total of 231 patients with noise-induced hearing losses, the pitch appears to be everywhere. Thus, there appeared to be no distinction between noise-induced tinnitus and those of different causes in relation to pitch. In 513 of the patients seen in the clinic, 84% had high frequency tinnitus, i.e., the pitch is 2000 Hz or higher.

In comparing the pitch of tinnitus to the audiogram, Fowler (1941) determined that tinnitus usually occurs in the frequency areas near the

upper and lower edges of the audiogram slopes (hearing loss curve), rather than at the bottom of the slope, the point representing the greatest neural degeneration, as some have suggested. Josephson (1931) agreed that the frequency of the tinnitus is generally at the point where the auditory curve drops off sharply. Graham and Newby (1962) reported that the frequency of the subject's tinnitus coincided with sharp dips in the audiogram in only 36% of their cases. Loeb and Smith (1967) found that after subjects were exposed to intense pure-tone and broadband acoustic stimuli, both the frequency of the tinnitus and the frequency of the traumatic stimulus increased. However, the frequency revealing the greatest hearing loss and the frequency match of the tinnitus did not coincide.

Loudness

Josephson (1931) observed that when a sound of the same fundamental pitch as the tinnitus was presented to the affected ear, the superimposed sound was masked by the tinnitus tone. Originally, it was thought that if the mechanism of tinnitus and of hearing external sounds were similar, there would be a summation of the intensities of the two tones. As this was not the case, this observation was utilized to construct a fairly reliable measure of the intensity of tinnitus. Josephson determined that if an applied tone of the same frequency as the tinnitus was raised in intensity to the point where they were indistinguishable, then the amount of intensity above the normal threshold for that frequency was said to be the intensity of the tinnitus.

Fowler (1941) presented a procedure for determining the intensity of tinnitus which he termed the "loudness balance" method. He reasoned

that the loudness of tinnitus could be estimated objectively by comparing it with the loudness of an applied tone or noise of similar frequency, in essence balancing the tinnitus with the sound being used for comparison. The loudness of the tinnitus is reflected in the number of decibels, above the threshold of the non-test ear, required for a sound to match the tinnitus in loudness. This method is generally used with subjects who have unilateral tinnitus.

Minton (1923) described a method for determining the intensity of tinnitus in cases of unilateral involvement. In this technique, a tone of the same frequency as the tinnitus is presented to the uninvolved ear. The intensity of the tone is increased until the tinnitus appears to shift to the ear receiving the tone. This intensity is then assumed to be the intensity of the tinnitus. Reed (1960) and Graham and Newby (1962) both followed Fowler's loudness balance method for determining the loudness of tinnitus. Graham and Newby's method differed in that the variable stimulus used for comparison was a tone which was the next lower frequency on the audiometer from the perceived pitch of the subject's tinnitus, rather than the same frequency.

Atherly et al, (1968) studied the characteristics of short duration tinnitus which was induced temporarily by noise (Noise-Induced Short-Duration Tinnitus, NIST). Immediately following exposure to a 110dB SPL stimulus, a comparison tone was delivered to the non-stimulated ear which was adjusted by the experimenter for both frequency and loudness until the subject reported that it matched his tinnitus. The level of the comparison tone, expressed as sensation level in the non-stimulated ear, was considered to be the loudness of the NIST. Vernon (1977, p. 14) offered yet another method of loudness determination.

If tinnitus is present in both ears the forced-choice procedure is between a very feeble and a very loud presentation. According to the patient's response these presentations are systematically narrowed until the patient reliably indicates some particular intensity as the one best matching the loudness of his tinnitus. The intensity of the matching tone will be measured by a calibrated microphone placed at the opening of the patient's ear canal.

If the tinnitus is only present in one ear the loudness matching will be done independently in the two ears. It is highly likely that the tinnitus ear will reveal the phenomena known as recruitment. Recruitment is the abnormally rapid growth in loudness as sound intensity is increased. In a normal ear a sound which is say, 10 to 15dB above threshold in a recruiting ear is perceived as being extremely loud. This point is extremely important as it has consistently confused those few investigators who have attempted to determine the loudness of tinnitus. They have usually found that the loudness of tinnitus could be matched with tones only 15 to 20dB above the patient's threshold and thus erroneously concluded that the patient really had very little about which to complain The presence of recruitment then is very important here so that we must determine not only the particular intensity which matches his tinnitus but also the general loudness function over a wide range for the affected ear. Loudness functions will be determined in two ways. The usual and more routine procedure is to utilize an "alternate balance" procedure between the two ears which works well if at least one ear is normal. Another way to measure perceived loudness will make use of reaction time measures. Here the patient is asked to release a lever as rapidly as possible after hearing a sound. Reaction time is inversely related to the intensity of the sound.

The literature has consistently shown that the actual intensity of tinnitus, as measured with various loudness balance techniques, appears to be softer than was expected. As Fowler (1941, p. 639) remarked, "It was frequently found that although a patient complained of intense tinnitus, in many instances measurements might reveal that it required less than 10 decibels above threshold to equal the tinnitus in loudness (often less than five)". Graham and Newby (1962), Reed (1960), Atherly et al (1968) and Vernon (1978) reported similar studies which were in agreement

with Fowler. Although some individuals were found to experience tinnitus at intensities higher than 10dB (Vernon, 1978), the general agreement places it at lower intensity levels. The following chart indicates the distribution of tinnitus loudness in two studies.

Table 3. A Comparison of the Loudness of Tinnitus for Graham's (1960) and Reed's (1960) Studies.

LOUDNESS DISTRIBUTION OF TINNITUS

<u>Loudness in dB</u>	<u>Graham</u>	<u>Reed</u>
0-5	53.4%	41%
6-10	21.9%	28%
11-20	20.5%	18%
21-30	4.1%	8%
31-40	0.0%	3%
41-50	0.0%	2%

Reed (1960), Fowler (1943), and Vernon (1978) found that the patient's estimate of the loudness of his tinnitus rarely corresponded to intensities found with audiometric matching tests. Fowler (1943) recommended that this fact be used therapeutically by helping the patient realize that the actual intensity of his tinnitus was not loud as compared to other familiar noises. However, according to Vernon, it seemed apparent that tinnitus need not be loud in order to be severe and bothersome to the patient. The severity appeared to be related to some other facet than loudness.

Goodhill (1952) also found it difficult to explain the discrepancy of a patient's distress and the slight intensity factors in tinnitus measurements where the patient complained about an intensity of only 2

to 5dB. Explanations stated that possibly the emotional level and related psychosomatic factors colored the picture of tinnitus. Also it is important to realize that the logarithmic nature of the decibel is not just an expression of intensity; rather, a 20dB increment in intensity of tinnitus can mean 100 times the pressure in dynes/cm².

In general, researchers have found that efforts to link certain types of tinnitus to specific situations have been futile. Vernon (1978) and Reed (1960) agreed that when the central frequencies or other factors of the patient's tinnitus were compared, it was found not to be related to just one diagnosis, description, site of origin or disease.

Mechanisms of Tinnitus

Many theoretical explanations have been advanced to explain the way in which the symptom of tinnitus is produced, none of which have been proven experimentally. The etiologies associated with tinnitus were found to extend throughout the entire auditory system which also seems true for the mechanisms of tinnitus. Some of the more common explanations found in the literature will be discussed here.

Trowbridge (1949) related tinnitus to the intimate nerve connection between the neural elements of the middle ear and those of the inner ear. The tympanic plexus (trigeminal, sympathetic and glossopharyngeal nerves) which constitutes the neural elements of the middle ear, is connected with the cochlear neuromechanism of the internal ear. Tinnitus was said to be produced by pathologic changes in the middle ear and in neighboring structures such as the pharynx, nose and eustachean tube. Apparently the tympanic plexus acts as a central nerve plexus, receiving impulses through its connections with the ninth and fifth nerves and the carotid

sympathetic fibers. These impulses are manifested either by tinnitus or by aural pain. Supposedly, tinnitus is a result of a subthreshold phase of irritation whereas pain results from a suprathreshold phase.

Lathrop (1923) also advanced a theory dealing with the physiologic action of the middle ear muscles. He stated that disturbed ventilation, chronic adhesive processes, or displacement of the drum membrane by any factor, destroys the normal balance existing between the ossicular chain and the two middle ear muscles. In this theory, he considers that either the tensor tympani muscle, the stapedius muscle, or both, would be in a state of tension, resulting in an attempt by the muscles to restore a condition of balance. Lathrop suggested that the contractions of these muscles is an attempt to restore the ossicles to their normal position. These vibrations would then be communicated to the cochlea, resulting in tinnitus. Lathrop further stated that it would be reasonable to assume that the well known external and middle ear lesions which are associated with tinnitus are capable of producing this muscle imbalance.

According to Jones and Knudsen (1928), in the case of a cochlear lesion, an impairment of hearing may result from inadequate articulation between the active membrane and the contiguous auditory nerve endings whereas tinnitus might result from a continuous coaptation of the active membrane and the auditory nerve endings, resulting in continuous stimulation and auditory impulses.

Fowler (1941) explained tinnitus of cochlear origin with the "busy line" theory. He stated that tinnitus was always associated with hearing loss, which is to be expected considering that neural impulses, regardless of their source, always monopolize the neurons activated. These wires are busy and so cannot be used by superimposed irritations. Deaf-

ness occurs because the neurons producing the tinnitus were busy and could not be activated by external sound vibrations. However, in 1944, Fowler observed patients with tinnitus who had no apparent hearing loss and no observable aural or other disease.

Davis (1954) suggested that tinnitus is caused by conditions which increase the excitability of the cochlea, such as excessive noise exposure or any mechanical pressure which produces a slight but constant displacement of the tectorial membrane in relation to the hair cells. He further stated that the tonal quality of tinnitus depends upon the mechanical activation of a group of neural elements innervating a particular section of the basilar membrane and is not a tendency of the neural elements of the auditory nerve or elsewhere to synchronize their discharges. Fowler (1941), however, stated that the fibers from each area of the basilar membrane are spatially implicated as are their central distributions. Thus, spatial representation in the end organ or nerve does not mitigate against the hypothesis that there can be central as well as peripheral origins.

According to Atkinson (1944) the auditory system is subject to the same or similar disturbances of a peripheral sensory nerve tract, as the two are physiologically equivalent. Tinnitus can thus be regarded as a homologue of paresthesia in the peripheral sensory system, as both can be produced by internal and external factors. Hence the term auditory paresthesia arose. Similarly, Fowler (1948, p. 487) stated,

The mechanism of tinnitus may be roughly compared with the mechanism of itching. When the neural mechanism of the ear or that of the skin is sufficiently irritated by any intrinsic disturbances, there results an illusion of sensation. The sense organs of the skin set up their specific nerve impulses which are transmitted to the brain to be interpreted according to the special sensory nerves involved.

Goodhill (1954) divided tinnitus into two categories; 1) that which is a result of deafness, as the normal masking effect of the surrounding ambient noise is removed, and 2) tinnitus due to neural discharge which he described as suprathreshold auditory paresthesia, as seen in traumatic deafness, Menieres syndrome, etc.

Watkyn-Thomas (1953) considers tinnitus to be a paresthesia of the auditory nerve, agreeing with Lermoyez (1929) who stated that irritation of a sensory nerve produces pain, but depression of its function results in anaesthesia. He believed that the pain of the cochlea is tinnitus and deafness is a form of anaesthesia; therefore, deafness accompanied by tinnitus constituted the paradox of painful anaesthesia.

Fowler (1948) indicated that the auditory nerve functions in the same way as other sensory nerves; thus, it may respond to irritations applied at any level from the peripheral end organ (cochlea) to the cerebral cortex. Therefore, due to the frequency distribution of the neurons at each level, tonal tinnitus or band frequency tinnitus may be produced by irritations at any level of the auditory pathway, either directly or indirectly.

The conditions associated with tinnitus are all capable of causing these irritations possibly affecting the blood and lymph supply to the auditory neural elements or their supporting tissues. For example, sludging of blood in the area of auditory neural tissue may result in irreversible degeneration of nerve tissues, auditory ganglion cells, nerve fibers and insulating sheaths, often resulting in tinnitus.

In considering the subject from a psychological viewpoint, it has not been possible to determine if tinnitus is a direct result of emotional disturbances. In reference to this, Fowler (1950) stated that overacting

or underacting emotionally to the environment has been shown to be an underlying factor in many so called "functional disorders". Emotional reactions may be largely responsible for the time of onset, severity, duration, recurrence, variation, and ultimate outcome of various diseases, and tinnitus is no exception to the rule.

Finally, Harper (1959) stated that intractable tinnitus, that which has persisted unrelieved for any great length of time, is most likely related to the fundamental principle of neurophysiology; i.e. that the discharge of nervous impulses along any pathway tends to become facilitated by repetition. Similarly, Myers (1975) remarked that tinnitus may start in the periphery of the auditory system, such as in Menieres disease. However, the memory of the noise or tinnitus becomes fixed in the auditory center of the cerebral cortex.

TREATMENT

Morgan (1878) presented an outline of theories on various conditions producing tinnitus and their treatments. These theories were the same as those found throughout recent literature and the treatment was an imposing list of medications which were neither better or worse than those offered today. The uncertainty of treatment in cases of tinnitus appears to be due to the fact that there is no single known cause. Regarding this, Fowler (1948) stated that there is not now and most likely never will be one drug or surgical procedure which will cure tinnitus.

Graham (1958) remarked that the treatment of any disease or pathology is based upon an understanding of the dynamics of that condition. When there is no agreement as to the etiology of a condition, one would not

expect to see a common course of treatment being followed.] Graham further stated that part of the failure in treatment is by physicians who attempt to cure the tinnitus as a disease rather than a symptom. This could account for the number of treatments that have been tried at one time or another in attempting to cure the problem.

Heller and Bergman (1953) stated that treatment of tinnitus should be a therapeutic assault on the related etiological factors of tinnitus. It was noted, however, that frequently the etiological agent no longer exists, but the tinnitus persists.] These authors compiled a list of the various types of treatments that have been used, which although incomplete, is adequate in revealing the wide range of therapeutic procedures. (See Table 4) Several of the methods revealed in Table 4 have been widely used in treating tinnitus and evaluated for their effectiveness. Some of these therapies are discussed in the following section.

Medical Treatments

According to Myers (1975), definitive treatment is possible only in tinnitus patients who display a treatable condition. One area of investigation has been in the use of Vitamin A injections to arrest cochlear degeneration in vitamin deficient animals. Lobel(1949), Anderson et al (1950) and Bau and Savitt (1951) further found that this vitamin was also effective in improving hearing acuity and alleviating tinnitus in human beings. However, Atkinson (1954) found that administration of vitamin A parenterally and orally was without effect in reducing tinnitus.

Fowler and Fowler (1955) reported some success in alleviating tinnitus with intravenous procaine injections in psychosomatic cases exhibiting possible blood sludging. Trowbridge (1949) found relief for some

Table 4. Approaches to the treatment of tinnitus. (Heller and Bergman, 1953, p. 76).

Medical

1. Medication; bromides, barbiturates, other sedatives, potassium iodide, vitamins, benzyl cinnamate, antiallergic drugs, histamine therapy, intravenous procaine.
2. Local therapy to disease processes.
3. Elimination of drugs and intoxicants.
4. Elimination of foci of infection.
5. Correction of faulty gastrointestinal function.
6. Correction of metabolic diseases.
7. Control of diseases of the vascular system and blood-forming organs.
8. Dietary control of fluids, salt and water balance.
9. Dental rehabilitation.
10. Intratympanic medication.
11. Therapy directed to correct nose and throat pathology, including roentgen and radium therapy.
12. Politzerization, inflation, massage.
13. Removal of cerumen.
14. Psychotherapy.
15. Hearing aid.
16. Electrical therapies, i.e., ultra violet, quartz lamps, galvanism.

Surgical

1. Otologic; ossiculectomy, mastoidectomy, tympano-sympathectomy, fenestration of the labyrinth, obliteration of the saccus endolymphaticus.
2. Rhinologic.
3. Spinal tap.
4. Cranial surgery for tumor, vascular anomalies, section of 8th nerve.
5. Splanchnectomy and similar techniques for alleviation of hypertension.

patients with analgesic injections of the tympanic plexus, producing tympanosympathetic anaesthesia. In the majority of the cases the intensity of the tinnitus was reduced to a tolerable state, with few side effects and no harmful after effects of the drug.

Atkinson (1946) investigated the relationship of emotional states of the tinnitus patient and increased muscular tension in the intrinsic muscles of the ear. By using a drug curare to temporarily paralyze the intrinsic muscles of the middle ear, it was found that increased tension of these muscles was not a factor in the production of tinnitus except when emotional disturbance was also present. Therefore, those patients without emotional disturbance showed no lessening of tinnitus with curare treatments.

Flottorp and Wille (1955) reported some success in treating the problem by use of nicotinic acid as a vasodilator to increase the blood flow to the labyrinth. Atkinson (1944) observed best results with this procedure among patients with middle ear type deafness.

Intravenous lidocaine was used for the diagnosis of central pain in the Pain Clinic of the Auckland General Hospital (Melding, Goodey and Thorne, 1978). They observed that some pain patients with coincidental tinnitus reported temporary relief from tinnitus following intravenous lidocaine injection. The patients most responsive to lidocaine were those with acoustic trauma, endolymphatic hydrops and high tone sensorineural hearing loss, e.g. those with damage or degeneration of the Organ of Corti. The duration of temporary relief was found to be longer than the effects of the lidocaine, being anywhere from three to five minutes to as long as several days. The authors indicated that the effective-

ness of lidocaine in suppressing tinnitus in patients with presumed Organ of Corti damage suggests that tinnitus is produced by abnormal hyperactivity of neurons within the auditory pathway. However, the exact location of the hyperactive neurons was not ascertainable with this procedure.

Oral anticonvulsants have caused longterm remissions of pain whose rhythmic hyperactivity of the disturbed neurons is analogous to the epileptic state and tinnitus. Shea and Harrell (1978) used patients who responded favorable to the short-acting intravenous lidocaine treatment to study a longer-acting oral anticonvulsant, carbamazepine in order to obtain long-term relief from tinnitus. The suppressive effect of both drugs breaks the vicious circle of abnormal rhythmic neuronal hyperactivity in the auditory pathway reflex arc.

Responses to the carbamazepine treatment showed one patient (4%) with complete relief, 21 patients (78%) with at least partial relief, and 5 patients (18%) had no relief. The authors regarded chemical treatment of tinnitus as more promising than masking or biofeedback. However, at this point, a combination of the three was recommended until anticonvulsants with fewer side effects are produced.

Surgical Treatments

Due to the relationship between the sympathetic system and vasodilation and vasoconstriction, blocking of the stellate ganglion or the upper dorsal sympathetic has been used to produce relief from tinnitus. Johnson (1954) produced relief from tinnitus in Menieres patients with this procedure. If the tinnitus disappeared temporarily, the patient was considered a candidate for permanent relief with a dorsal sympath-

ectomy. Atkinson (1944) found some improvement in about 50% of his patients, indicating the limitations of such a procedure in many cases.

Graham (1965) reported that the stapes mobilization and the fenestration often result in the alleviation of tinnitus. It appeared that in these cases it was the improvement of the hearing acuity that caused the reduction of the tinnitus.

Hatton et al (1960) occasionally found that during routine galvanic stimulation tests, tinnitus could be increased or decreased in intensity according to the polarity of the current. The galvanic stimulus does not reach past the cochlea; thus, for those whom galvanic stimulation did not alter the tinnitus, either the current didn't reach the origin of the tinnitus in the cochlea, or the origin was placed in the central auditory system.

Cazals et al (1978) reported cancellation of tinnitus during positive electrical stimulation of the cochlea through a promontory or a round window electrode. This result occurred without affecting simultaneous acoustical or electrical stimulation of the hearing.

Lempert (1964) suggested disease of the tympanic plexus as a cause of tinnitus in that a tonus impulse could be transmitted to the inner ear from the malfunctioning plexus. It was reported that tympanosympathectomy, stripping of the plexus, improved the tinnitus of ten out of fifteen patients.

Some patients whose tinnitus has become unbearable, often to the point of mental depression, have consented to destruction of the affected ear by severing of the auditory nerve. Sadly enough, this procedure has not always been effective, leaving the patient with both tinnitus and deafness. Atkinson (1944, p.743) stated:

Just as in sensory disturbances division of nerves central to the lesion, and even amputation of a limb, may not relieve the patient because, by some mechanism which is not yet clear but which is gradually being clarified, the original peripheral disturbance has come to involve pathways more centrally placed, so with severe and long-standing tinnitus division of the cochlear nerve will often not relieve the patient of his noises.

Dandy (1965) also found that section of the auditory nerve in patients with Menieres disease resulted in relief of dizziness, but they experienced continuing tinnitus, indicating a problem which has become centrally fixed with time.

Psychotherapeutic Measures

It seems apparent that tinnitus is a symptom indicative of organic disease. The amount of distress due to tinnitus, however, is often related to the extent of the patient's preoccupation with the symptom, and his attitude of mind towards it. For this reason, Kennedy (1953) contended that it is impossible to consider the tinnitus without the patient. The following discussion on psychotherapeutic measures deals with those types of therapy which are not medical or surgical, with the exception of sedation.

House (1977) attempted biofeedback treatment for 41 patients with tinnitus of various etiologies. Many of these patients had other types of tension related to chronic pain disorders, including migraine headaches, low back pain, temporomandibular joint pain, depression and hypertension. Of these patients, 80% indicated some improvement, 10% reported that tinnitus had ceased and 2% reported no change. Almost all patients felt that the treatment was beneficial, realizing that they did have emotional difficulties and were misplacing much of their anxiety and depression on their tinnitus. House believed that tension exacerbates

the patient's perception of his tinnitus, causing it to become subjectively louder. He concluded that biofeedback is effective in that it reduces the subjective loudness of tinnitus, and allows the patient to better understand and cope with it.

Goodhill (1954) divided the possible means of treatment into the following steps:

1. Specific otologic therapy - The organic otologic disease focus must be approached with the idea of therapy directed specifically to eradication of the tinnitus.
2. Palliative Measures - Simple reassurance as to the reality of the tinnitus accompanied by encouragement and good prognosis, will go far in helping alleviate the anxieties of the patient with decompensated tinnitus.
3. Sound Sedation - Sound sedation is very helpful in many cases of tinnitus, especially in regard to the difficulty in sleep, which is a great problem with many patients . . . The use of a bedside or pillow radio or phonograph speaker is very helpful in providing an artificial source of ambient noise to mask out the subjective tinnitus.
4. Drug Sedation - Drug sedation is an important palliative measure, not only for daytime use but especially for bedtime tinnitus irritability . . . No one drug should be used for any long period
5. Surface Psychotherapy - . . . Such surface psychotherapy should include a thorough explanation as to the real nature of tinnitus with assurance that it is neither an hallucination nor an illusion.
6. Major Psychotherapy - Deep psychotherapy in psychiatric hands is indicated in every case of organized, symbolic, verbal or musical tinnitus. It is also indicated where surface psychotherapy, . . . has not solved the decompensated tinnitus problem

It is the patient with decompensated tinnitus, that is tinnitus which is recognized as a problem by the patient, that Goodhill directs these steps in managing tinnitus. It is recognized that tinnitus is not easily cured, therefore treatments are aimed more towards making the conditions bearable for the patient.

Hearing Aids and Masking

Saltzman and Ersner (1947) studied the use of a hearing aid for the relief of tinnitus. It was found that the benefits from a hearing aid were similar to those of the fenestration operation. Amplification allows much of the outside sound to reach the cochlea, crowding out and masking the patient's head noises. It was in the routine fitting of hearing aids that the therapeutic advantages of this mechanical means in regards to tinnitus were discovered. Thus, in nerve deafness, if no extraordinary tolerance difficulties are present, a hearing aid gives considerable relief from tinnitus as well as improvement in hearing.

Researchers continue to find hearing aids to be successful in alleviating tinnitus symptoms in some patients. Vernon (1977) indicated that it was widely recognized that one sound was capable of interfering with the detection of another sound. For example, tinnitus has been shown to interfere with the reception of some types of external sounds. The concept of masking tinnitus then is simply a reversal of this process.

In explaining this process, Vernon (1977, p. 18) stated, "The idea of masking came to us because of occasional reports from patients who said things like ' . . . the only time I experience relief from tinnitus is in the shower" As a result of these observations, a device called a tinnitus masker was designed. This masker generates a noise containing frequencies which have been previously shown to mask a patient's tinnitus. The device introduces the masking sound into the ear and is housed in a post auricular hearing aid case. The fact that 84% of the patients had high frequency tinnitus had in the past posed a problem for the masking program. The tinnitus maskers were made from components that are used to produce hearing aids, which limited the

frequency output capabilities of the masking noise to the lower frequencies. Steps have been taken, however, to produce maskers with high frequency characteristics enabling more people to benefit from this form of relief. There are currently available several high-frequency maskers which produce a substantial amount of energy out to 17,000 Hz. Vernon (1978) reported a successful case of masking in a young patient whose tinnitus was identified at 15,000 Hz.

In treating the symptom with masking, many patients have found no relief from the external noise. Fowler (1941) reported that neither the loudness of tinnitus nor its frequency alone determines the ease of masking it. He found that due to the recruitment phenomena, a mere lack of neural elements does not prevent the ear from being masked by loud sounds. In addition, he also stated that if the neural elements were entirely absent they could not be activated by any irritation; thus if there's no evidence of life there cannot be any tinnitus originating there. Therefore, it was suggested that the difficulty of masking and the origin of tinnitus may be due to a lesion medial or central to the cochlear end organ. This explanation was presented as a means of differentiating tinnitus of a peripheral (cochlear), medial (cochlear nerve), or central (cochlear nuclei and central pathways) cause.

During studies on the masking of tinnitus, a phenomenon termed residual inhibition has been observed. Residual inhibition is the term used for a continuation of the masking effect following the termination of the masking sound. This phenomena was first observed by Josephson (1931). Vernon (1978, p.3) described the procedure for measuring residual inhibition.

In the clinic we test for residual inhibition by completely masking the tinnitus for one minute. At the end of the masking period the patient is asked to describe their tinnitus. If they are displaying residual inhibition at that time they will say something like "it's gone", or ". . . it's greatly subdued", or ". . . it is much less". In any of these cases we time how long it takes to reestablish the normal level of tinnitus. Typically after one minute of masking, about 45 seconds or so are required to return to normal

It was found that of 513 patients tested, 22% experienced no residual inhibition, and 78% showed residual inhibition in one of two ways. Some patients see a complete relief of tinnitus, and some report only a partial suppression from their noise. Vernon stated that for residual inhibition to occur, the masking noise must be generated at the right frequency for a given patient's tinnitus. Thus, if tinnitus is at 5000 Hz then the masking noise should be a band of noise centered at 5000 Hz, or if a tone is used to produce masking it should, in this case, be a 5000 Hz tone. Furthermore, if tinnitus is at 5000 Hz, but a 25,000 Hz tone is used to mask it then masking can occur, but most likely residual inhibition will not occur. In some cases then the presence of residual inhibition can be used to help confirm the tinnitus pitch identity. Experience has shown that this concept is generally true of most patients. However, occasionally a subject's effective masking level has been identified at a frequency differing from the frequency of their tinnitus, and is usually an octave above that frequency.

Patients who wear tinnitus maskers may find extended periods of residual inhibition when removing the masker for the day. The mechanism responsible for residual inhibition is not yet understood; therefore, it is unexplainable why some people do not experience relief with the masker. According to Vernon (1978), however, patients who have dis-

played residual inhibition in the clinic have a good chance for using a tinnitus masker.

From the above review of the literature one can see that the vast amount of research reported on the subject of tinnitus has made it evident that there is no single cause, explanation or cure for the problem. For this reason, it has been difficult to provide relief or treatment for patients who suffer from tinnitus. Recent efforts have, instead, been placed on alleviating the problem by completely or partially covering it with masking. Although this method cannot be considered a cure for tinnitus, it does allow the patient an effective means of coping with the problem while efforts to unravel this mysterious symptom continue.

CENTRAL AUDITORY TESTS

General Considerations

It has recently become possible for the hearing researcher to get new and clearer insights into auditory functions central to the auditory nerve. Modern audiometric techniques have been able to determine hearing loss caused by damage in the peripheral auditory system; however, when lesions affect the central auditory pathway, even massive damage has been shown to result in no apparent peripheral hearing loss (Roeser, 1974).

The literature consistently shows that conventional pure-tone and speech audiometry do not identify cortical hearing impairments (Parker et al, 1962, Katz, 1962, Jerger, 1964, 1973, 1974, Bocca and Calero, 1968, Berlin and Lowe, 1972, Bengtsen, 1973, and Roeser, 1974). According to Berlin and Lowe (1972), central lesions are not easily detected with conventional audiometric techniques. As lesions progress from the peripheral auditory system towards the cortex, because of the redundancy, multiple crossings and interactions in the auditory tract, hearing loss becomes more difficult to demonstrate with simple frequency and intensity manipulations. Katz (1962) similarly reported that central hearing disorders may be uncovered by demanding the evaluation of unusually difficult audiological material by the patient. A heavier burden is placed upon the higher auditory mechanism, which is necessary in detecting disorders of the central auditory system.

Therefore, in view of the complex nature of the central auditory system, and the extremely elusive nature of the auditory symptoms associated with central disorders, researchers have been challenged to design innovative approaches which will tax the mechanisms involved in central

auditory processing.

Jerger (1973) discussed problems in the construction of a central auditory test battery. Largely, it is a strategic problem of designing test procedures that tap the patient's performance potential at the precise level of difficulty with a minimum of error variance. Central lesions do not share the same homogeneity of patient performance as do peripheral auditory disorders. Thus, a listening task that is too difficult for one patient may be simple for another. Therefore, when probing for central disorders it is necessary to adjust the difficulty of the task to match the extent of the patient's auditory disorder.

Central Auditory System Defined

Prior to discussing central auditory tests, a short introduction regarding the anatomical portions of the auditory system follows. Jerger (1973, p. 86) defined the central auditory system in the following manner.

The central auditory system may be broadly defined as that portion of the total auditory system lying within the central nervous system. It includes chiefly the brain stem pathways (Jungert, 1958) and the primary auditory projection areas on the superior temporal gyri (Tunturi, 1960). The central auditory system consists of two crossed pathways. Each pathway begins at the synapse between first- and second-order neurons in the cochlear nuclei, then crosses to the opposite side of the brain either directly or via the superior olivary complex. The pathway then rises in the lateral lemniscus to the medial geniculate body in the thalamus. From this synaptic level fourth-order neurons, the "auditory radiations", project to a small area on the superior gyrus of the temporal lobe, Heschl's gyrus or the primary auditory area on the cerebral cortex. This primary auditory area is by no means the end of the system. We know very little, however, about the subsequent pathways beyond this point. We can only speculate that further pathways ultimately link auditory cortex with language centers in the precentral and parietal areas, and with association areas linked to other sensory inputs (Penfield and Roberts, 1959).

We do know that each of the two crossed pathways is rather elaborately interconnected with the other pathways throughout the course of second-and-third-order neurons (Jungert, 1959). Furthermore, fourth-order neurons are probably interconnected via the corpus callosum (Milner et al, 1968).

Jerger (1973) reported that through auditory testing some manifestations of central auditory disorders will reflect this elaborate interconnection, while other manifestations do not. The crossed pathway will further demonstrate its reality in that lesions of either pathway proximal to the crossings in the brain stem will invariably produce symptoms in the ear opposite to the affected side of the brain.

Dunn and Johnson (1977, p. 1) outlined the functions of the central auditory system as related to acoustic stimuli.

Lower Brain Stem

- Relay to cortex; approximately 80% of signal crosses to contralateral side before reaching cortex.
- Inhibition and facilitation of selected portions of signal.
- Processing of signal intensity for activation of acoustic reflex (connections to CN₇) and also eyeblink reflex (connections to CN₆).
- Processing of temporal relationship between the signal from each ear for localization of sound in free field.

Midbrain

- Relay to Cortex
- Localization of sound resulting in eye movements and head turning.

Cortex

- Perception
- Recognition
- Discrimination
- Association
- Memory

Liden (1969) indicated that the development of tests which would sufficiently assess the condition of the central auditory system has been a long and often fruitless process. Many investigations have been

The following illustration outlines the pathways of acoustic stimuli from primary centers to the cerebral cortex.

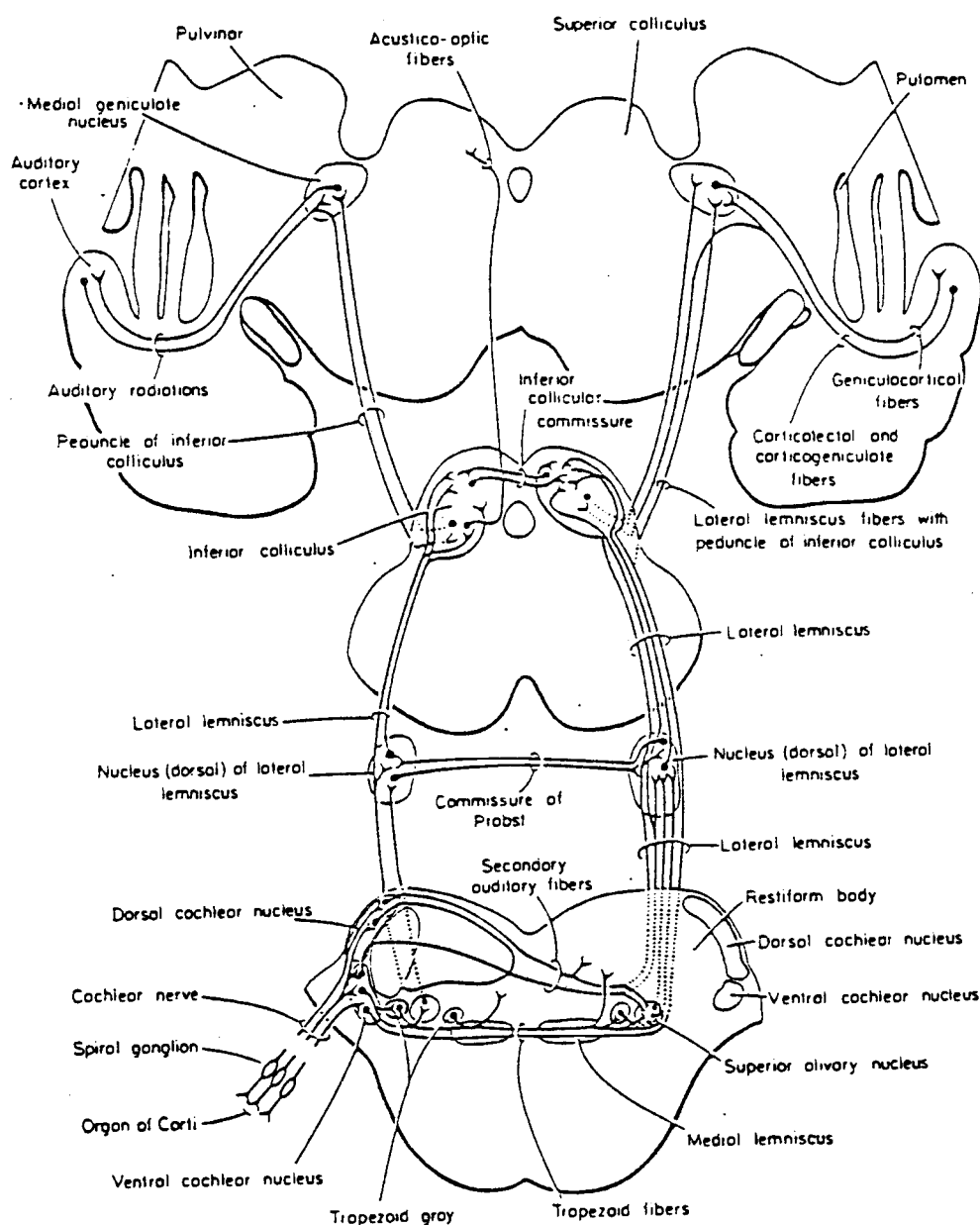


Figure 1. Diagrammatic illustration of pathways for projection of auditory impulses from primary centers to cerebral cortex. (Crosby, E.C., Humphrey, T., Lauer, E.W., from Keith, R.W., 1977, p. 4.)

made regarding the occurrence of pure-tone threshold changes in cortical pathology, but the outcome has generally been discouraging. Experience has shown that a mild pure-tone threshold loss may occur, but does not differ from a peripheral impairment. In measurement of speech discrimination abilities, greater demands are placed on the system; however, one seldom sees any significant impairment in the central lesions. The normal speech message contains such an abundance of information that the individual, even with relatively diffuse damage in the central auditory pathways and cortex, has normal or nearly normal discrimination. Thus, tonal stimuli and simple speech messages are not complex enough to uncover a lesion of the central nervous system.

Bocca and Calearo (1963) also stated that neurological lesions have been studied by every possible method of pure-tone audiometry with the results that threshold curves showed no deterioration as related to the lesions. Those who fixed their attention on speech audiometry for subjects affected with lesions of the auditory cortex were no more fortunate. There seemed to be no middle way between speech deafness and the frequent complete normality of speech hearing. Alajouanine et al (1955, p. 206) spoke the feelings of many in stating: "An audiometric symptomatology of disorders at the level of the cortical auditory center has to be considered impossible".

The first really significant breakthrough on this problem was achieved when Bocca (1955) and his colleagues reported that patients with temporal lobe tumors showed a modified ability to understand distorted speech in the ear contralateral to the lesion. This unilateral deficit was demonstrable despite virtually normal pure-tone sensitivity in both ears. In a series of experiments, Bocca systematically varied

the redundancy of speech messages by means of low-pass filtering, acceleration of rate, periodic interruption and variation in message length. Most importantly they found that any reduction in redundancy of speech produced effects in test results, but that low-pass filtering provided the most effective clinical technique in diagnosing temporal lobe tumors. In essence, Bocca's work provided the ground work for the multitude of studies using distorted or difficult speech for central auditory testing.

Research has indicated that patients with unilateral temporal lobe lesions (Penfield and Evans, 1934, Hodgson, 1967) invariably display normal pure-tone hearing. Despite this, studies on tests using pure-tones, clicks and masking noise have been applied to the diagnosis of central auditory dysfunctions. Berlin and Lowe (1972) noted that these tests have been applied with only a modicum of success. Therefore, the review will involve only a brief summary of the non-speech tests.

Non-Speech Tests

Calearo and Antonelli (1968) stated that investigations with pure-tone materials have attempted to analyze the particular aspects of auditory integration which are characteristic of every level of the brain stem. Attempts have also been made to understand the relationship between each level and its function. However, due to the impossibility of microscopic analysis of lesions and the limited knowledge of the relevant neurophysiology, this relationship has been difficult to assess.

Calearo and Antonelli (1968) reported results from previous studies involving tonal stimuli. They indicated that discrepant and sometimes conflicting data have been obtained with these tonal tests, such as the results of lateralization and spatial localization tests (Jonkees and

Veer, 1957, Sanchez-Longo et al., 1957, Walsh, 1957, Sanchez-Longo and Forster, 1958, Matzker and Welker, 1959), indications of an abnormal adaptation on affected frequencies (Parker et al, 1962; Pestalozza and Cioce, 1962, Sorenson, 1962, Eickel et al, 1966) and tests based on binaural beats (Demichelis et al, 1966).

Tests which indicated more positive results dealt with the influence of contralateral masking with white noise on hearing adaptation, (Brunetti, 1960, Maspétiol et al, 1961) tests based on binaural supplementation or inhibition (Chochelle, 1960, Maspétiol et al, 1960), and studies on the reflex reaction times using a monaural supraliminal pure-tone (Chochelle, 1955, Maspétiol et al., 1960).

Pure-tones were further studied by comparing auditory encephalographic responses with an observer's sensations when his ears were stimulated by single or paired clicks via earphones (Rosenzweig, 1954, Hirsch, 1969).

Other researchers reported that in order to determine that a temporal sequence anomaly was unique to the auditory system, tests which compare sequencing capabilities in other modalities (e.g., visual or tactile) in relation to auditory performance were utilized (Gescheider, 1966, Hirsh and Sherrick, 1961, Efron, 1963, Jerger et al., 1969).

Results of Bekesy audiometry procedures has shown unusually wide excursions by some temporal lobe patients (Berlin et al., 1965, Jerger et al., 1969). This effect was seen in both sweep and fixed-frequency tracings.

Katz (1972) discussed the masking level difference test (MLD) as related to central auditory disorders. The test required two ear interaction, thought to be at the level of the trapezoid body and related

midbrain structures. Katz (1972, p. 284) described the phenomena as follows:

If a masked threshold for a signal embedded in a given noise is established in one ear, subsequent presentation of noise 180° out of phase in the contralateral ear can release the tone in the test ear from as much as 12 to 13dB of masking; similar results are obtained when the noises are left in phase, and a contralateral tone is introduced out of phase with the test signal.

Quaranta and Cervellera (1977) studied the relationship between the tonal MLD performance and the behavior of the "sensitized speech tests", based on the work of Bocca and Calearo (1973) who studied this effect in normal hearing subjects suffering with central nervous system diseases. The studies have shown that pathological MLD values were present in all cases with pathological sensitized speech tests. These MLD values were variable and were related to the bulbo-pontine lesions and the central hearing function asymmetry. In normal hearing subjects suffering from central nervous system disease, pathological tonal MLD's were attributed to central hearing lesions.

Speech Tests

Calearo and Antonelli (1966) believed that speech is a more adequate testing material as it allows for exploration of central levels of auditory integration, is a more global test, and can be performed regardless of tonal deficits. One of the cardinal principles of central auditory evaluation has been the reduction of redundancy in the signal, because of the complexity and neural redundancy of the central nervous system (Berlin and Lowe, 1972). Speech signals are ideal in that they lend themselves to more complicated manipulations than do tones. Words and sentences can have their redundancy reduced by being filtered, split,

interrupted, competed, accelerated, and reversed. Beasley and Freeman (1977) acknowledged that the use of altered speech as a part of the clinical test battery has grown out of the need to detect subtle neurological lesions which may go unnoticed by use of standard pure-tone and word discrimination measures of audition.

Speech audiometry tests fall into two main groups; monaural and binaural. Monaural tests present words, sounds or signals separately to one ear at a time, comparing the results obtained on one side with those on the other. In binaural tests both ears are tested simultaneously with equal or different words or signals. The result is a summation of the hearing capacity in both ears.

Frequency-Distorted Speech

With the use of electronic filters, speech signals can be made less redundant by attenuating parts of the frequency spectrum. The ability to discriminate filtered speech varies considerably depending on the type of filter used, i.e. high-pass, low-pass or band-pass filters, as well as with verbal material (Korsan-Bengtson, 1973).

As stated previously, Bocca, Calero and Cassinari (1954) were the first to show reduced scores on the ear opposite to the brain lesion using low-pass-filtered speech. Test material consisted of disyllabic meaningful phonetically balanced (PB) words processed through a low-pass filter with a cut-off frequency of 800 Hz. Test results showed that the score for the contralateral ear was reduced by 20-25% as compared to the ipsilateral ear.

In a follow-up investigation, Bocca, Calero, Cassinari and Miglia-
vacca (1955) showed the general validity of their filtered speech test

in 18 patients with confirmed unilateral temporal lobe disorders. In those patients whose test results were essentially equal for both ears, surgery confirmed that the tumor had not affected the auditory cortex. Thus, the potential use of filtered speech as a means of exploring function of the central auditory nervous system was shown by Bocca and his co-workers.

A review of the literature indicated that filtered speech tests have been widely used for diagnosing temporal lobe disorders (Linden, 1960, 1964, Jerger et al, 1960, 1964, Maspetiol, 1964, Berlin et al, 1965, 1972, Korsan-Bengtson, 1970, 1978, and Lynn et al, 1972). Results of these studies are in agreement with Bocca et al (1954).

Low-pass-filtered speech tests are generally considered as a means of assessing the integrity of the temporal lobe. However, patients with brain stem pathology have also demonstrated difficulty with this task. Jerger (1964) reported auditory test results from four groups of patients with central pathology. These groups consisted of patients with (A) brain stem lesions not involving the auditory system, (B) unilateral brain stem involvement involving the auditory system, (C) unilateral temporal lobe lesions involving the auditory cortex, (Heschl's gyrus), and (D) cortical lesions not involving Heschl's gyrus. Results showed normal and bilaterally symmetrical scores for Group A; a reduction in intelligibility of about 20% in the ear contralateral to the lesion for group B; similar results in group C; and a smaller but measurable reduction in discrimination ability in the contralateral ear for group D. It appears then, that the filtered speech test is sensitive to lesions of the brain stem; however, the amount of difficulty tends to be less

than those reported for lesions of the temporal lobe.

Calearo and Antonelli (1968) reported test results from 24 patients with brain stem lesion. Twelve of these subjects showed abnormally poor performance for low-pass-filtered speech. Of these, four had a bilateral reduction, and eight a unilateral deficit in discrimination. Of these eight, the reduction was contralateral to the lesion in one half of the patients and ipsilateral to the lesion for the remainder. The outcome of this investigation suggested that in the confined area of the brain stem, small differences in the location or extent of the lesion can produce markedly different auditory results.

In conclusion, past research studies suggest that the low-pass-filtered speech test is a widely used tool for assessing the function of the central auditory nervous system. The expected outcome for patients with temporal lobe lesions would be a reduction of the score in the ear contralateral to the lesion, whereas patients with lesions of the brain stem auditory pathways tend to produce more variation in test scores. The discrimination deficit may be seen bilaterally or unilaterally and either ipsilateral or contralateral to the lesion.

Interrupted Speech

Further studies based on the redundancy principle initiated the use of interrupted or electronically chopped speech. Speech was periodically interrupted with an electronic switch, causing amplitude modulations of the speech waves, i.e. cancelling parts of the speech message. Bocca (1958), Calearo and Antonelli (1963) and Teatini (1970) have studied the effects of interrupted speech on discrimination abilities in normals. Their studies indicated that discrimination abilities de-

pended on the number of interruptions/second, and also on the ratio between the duration of the periods of speech and of the intervals.

Studies using interrupted speech on patients with unilateral temporal lobe disorders showed that discrimination scores will be reduced on the side opposite to the lesion (Bocca, 1958, Calero and Antonelli, 1963, and Antonelli, 1970). Patients with brain stem disorders similarly show reduced performance on discrimination tasks, with one or both ears showing a poor score (Bocca, 1963, 1967, Calero and Antonelli, 1968, and Jerger, 1970).

Bocca (1967) and Teatini (1970) investigated the effects of a patient's intelligence on scores for interrupted speech tests. Their studies demonstrated that an individual's intelligence quotient (IQ) correlated with his performance on interrupted speech tests, the discrimination score varying with the IQ.

Time Compressed Speech

The measurement of the intelligibility of verbal material as a function of an increased speech rate has also been investigated as a tool for central auditory testing. Korsan-Bengtson (1973) stated that normal speech rates are from 110-140 words/minute depending on the habit of the speaker and the language used. Altering the speed of speech is easily done by increasing the playback speed of an ordinary tape recorder, with the effect on the speech being both a change in the rate and a frequency shift of the speech sounds.

Garvey (1953) used this method and showed that the intelligibility of spondee words, that is, words composed of two syllables that are equally stressed, decreased to 65% when accelerated at a rate of 2.0,

with the score reducing to only 10% when accelerated further to 2.5. Klumpp and Webster (1961) and Fairbanks et al (1957) showed that sentences are affected less by time-frequency distortions, being almost 90% intelligible at a time-compression of 0.67.

Korsan-Bengtzen (1973) showed that speech-rate can also be increased without changing the frequency spectrum either by quick reading of the verbal material, or by use of a special tape-recorder with a group of rotating playback heads. With this method, speech-rate can be altered from .5 to 2.0T; however, to maintain intelligibility, intensity must also be increased. Calero and Lazzaroni (1957) tested normals with this method and found that to maintain intelligibility at a speed of 350 words per minute, intensity needed to be increased 10-15dB. Thus discrimination curves kept their shape, but were shifted to the right.

Studies of time-compressed speech on patients with lesions involving the central auditory pathways and auditory cortex indicate results similar to those of other tests for central function. Calero and Lazzaroni (1957), Bocca (1958), Bocca and Calero (1963) and Korsan-Bengtzen (1968, 1970) found that well defined lesions in the auditory cortex significantly reduced scores in the ear contralateral to the lesion for time-compressed speech. A bilateral decrease in test scores was generally seen in patients with diffuse central nervous system lesions. Studies of time-compressed speech in patients with brain stem lesions also found a decrease in performance, usually unilaterally (Calero and Antonelli, 1968). Administration of these tests to aged people sometimes produced a decreased score which has been interpreted as a result of de-

generative changes in the central nervous system (Korsan-Bengtson, 1968, and Antonelli, 1970).

Performance-Intensity for Phonetically-Balanced Words (PI-PB)

Performance-intensity (PI) functions for monosyllabic phonetically balanced (PB) words are constructed by presenting lists of 25 words each at several intensities until the shape of the function is well defined. Generally, PI-PB functions range from that speech level yielding 0-20% correct up to a maximum speech intensity of 110dB sound pressure level (SPL). In some patients the PI-PB function reaches a maximum, then declines substantially with further increase in speech level. This performance drop-off or "roll over" at high speech intensities is quantified by analyzing each PI-PB function for PB maximum (PBmax) and PB minimum (PBmin). PBmax is defined as the maximum discrimination score at any speech level; PBmin is defined as the lowest discrimination score at any speech level above PBmax. The difference between these two scores (PBmax minus PBmin) defines the degree of rollover.

Jerger (1973, p. 89) stated that the PI-PB function is useful in central disorders for two reasons.

First, it performs an important role as a screening technique for detecting central problems. Any patient who shows a consistent ear difference on PI-PB functions in the absence of a pure-tone sensitivity difference sufficient to account for the ear difference on a peripheral basis must be suspected of central auditory disorder. Second, the PI-PB functions help to define the extent of the central problem. The performance deficit will typically appear on the ear contralateral to the affected side of the brain. In addition, rollover of the PI-PB function may occur. The presence of rollover, in the absence of sensitivity loss, is a further confirmatory sign of a central problem. The PI-PB function works well as a screening device primarily because for patients with central disorder, PB word repetition is

usually a relatively difficult listening task. Hence an ear difference of 20 - 30% is often demonstrable.

Auditory results in patients with brain stem disorders often present inconsistent findings between individuals. The PI-PB rollover phenomenon is a good example of this inconsistency. In a series of studies, Jerger and Jerger (1974) found that depressed PB maximum performance was found either on both ears or on the ear contralateral to the site of lesion. In contrast, patients with unilateral temporal lobe lesions are usually characterized with no PI-PB rollover (Jerger and Jerger, 1975). However, in a unique study of bilateral temporal lobe lesion, the patient showed a marked inability to recognize any PB words or sentences even under ideal listening conditions (Jerger et al, 1972).

The concept of hemisphere dominance was studied in relation to monaural testing techniques. Calero and Antonelli (1963) demonstrated on normal subjects that with interrupted and distorted voice, discrimination curves did not show significant differences between ears. The same tests repeated with subjects affected with pathological lesions of the temporal cortex showed that the loss in contralateral discrimination is the same whether the lesion is located on the right or the left.

Binaural Speech Tests

Korsan-Bengtson (1973) found that when normal speech is presented to both ears simultaneously (diotic listening) the discrimination score was higher than when presentation was to only one ear (monaural listening). In light of this, Groen and Hellema (1960) investigated monaural and binaural speech perception in hard-of-hearing children and in

children with central deafness. In the former, who had peripheral lesions, speech perception was much better in the binaural listening condition. In children with central deafness, the difference between binaural and monaural discrimination did not differ much. Implications were that an intact central auditory system is necessary for securing binaural summation of speech.

Bocca and Calero (1963) defined binaural speech integration tests as based on the principle of the central summation of the two parts of a monaural message, each of these two parts being insufficient for identification. Bocca (1955) demonstrated this by presenting speech of low intensity to one ear, and the same speech, well above threshold but low-pass-filtered, to the other ear. Monaurally, discrimination did not exceed 50% in either ear. Simultaneous binaural stimulation scores however were approximately equal to the sum of the monaural scores.

Calero (1957) used this test in patients with temporal lobe disorders, finding no summation when the low-intensity speech was presented to the ear opposite the lesion. A normal summation score occurred when the stimuli were reversed, indicating the value of this method in the diagnosis of central auditory disorders.

Matzker (1958) demonstrated the concept of binaural fusion, which is the combining or synthesizing of incomplete speech sounds from the two ears to a meaningful message. The same vocal message is passed through two filters, one low-pass and one high-pass, each too scanty to understand alone. Delivered simultaneously the two fractions achieved good integration, which allowed normal subjects to attain 10% scores. In lesions of the auditory pathway, Matzker found that in 80%

of these patients the effect of binaural summation was completely abolished and discrimination becomes poor.

Studies by Hall (1965) and Tillman et al (1966) indicate similar findings in patients with brain stem and temporal lobe lesions.

In a study using dichotic stimulation, Broadbent (1954) found that when two simultaneous sets of digits were presented, one set to each ear, the subjects tended to respond more often to the set from the right ear before they responded to the left. He believed that this showed the effects of a short term memory system. Kimura (1961) also used the concept of dichotic stimulation in studying patients with temporal lobe disorders. When different digits were presented simultaneously, patients with unilateral temporal lobectomy demonstrated difficulty in recognizing digits in the ear opposite the removal. Furthermore, overall efficiency, defined as the total number of digits reported from both ears, was affected by left temporal lobectomy but not by right temporal lobectomy. Kimura interpreted this data to mean that the crossed auditory pathways in man were dominant over the uncrossed auditory pathways and that the left hemisphere was more important than the right hemisphere in the perception of spoken material. Kimura's work has been the basis for an abundance of studies since then which have supported her conclusions.

The dichotic tests are those in which a different message is presented to each ear. The signals may also be synchronized to reach the ears simultaneously. The Staggered Spondaic Word Test (SSW) is another type of dichotic test developed by Katz (1962). The test uses English spondaic words in a competing message mode to evaluate the higher auditory pathways. The patient is required to attend first to one side then

to both sides simultaneously and then only to the opposite side, with different words being presented to each ear. The subject is expected to repeat both messages. The two spondaic words are partially overlapped in the following manner:

Ear		Time	
Right	UP	STAIRS	
Left		DOWN	TOWN
		1	2
			3

In comparing the SSW test results of normals and patients with central lesions, the latter demonstrated considerable difficulty on the test. Those individuals with unilateral central trauma behaved normally on conventional audiometric measures; however, marked deviations from normal subjects were seen on the ear contralateral to the lesion with the SSW test. This contralateral effect is again consistent with other tests of higher auditory cortex behavior.

Jerger (1973) developed and later modified the Synthetic Sentence Identification Test (SSI) in order to explore the possibility of a central lesion in greater detail. The materials for SSI are artificial or synthetic sentences constructed as approximations to real English sentences, grammatically correct, but meaningless. During the test, the sentences are made more difficult to identify by using a competing message, i.e. a recording of continuous discourse. This may be an ipsilateral competing message (ICM) or a contralateral competing message (CCM), the former being presented to the test ear along with the synthetic sentences and the latter being presented to the opposite ear.

With the SSI test materials fixed at a given intensity level, the competing messages may be varied in intensity, allowing a number of message-to-competition ratios (MCR's) to be obtained (Jerger, 1973). In using the sentences diagnostically, a performance intensity (PI) function, such as with PB words, is formed. The area under the curve is calculated and can be compared to one for PB words or exclusively.

Jerger (1966, 1973, 1977) used the performance on the two tasks, SSI-ICM and SSI-CCM, to assist in the differentiation of brain stem and temporal lobe lesions. A significantly greater deficit for ICM than for CCM has been indicative of a brain stem lesion, whereas a significantly greater deficit for CCM than ICM indicates a temporal lobe lesion.

Jerger and Hayes (1977, p. 123) compared performance versus intensity functions for both phonetically balanced (PB) words and synthetic sentence identification (SSI), yielding patterns useful in differentiating peripheral from central sites of auditory disorder. The information was found to be useful in four ways.

First, if the relationship between the two functions is consistent with audiometric contour, and significant rollover is not present in either function, then a purely peripheral cochlear site may be reasonably inferred. Second, if the difference between the two functions is out of proportion to either degree or shape of audiometric contour, and if substantial rollover is apparent in either function, an eighth nerve site must be suspected. Third, whenever the SSI function falls below the PB function, and the difference cannot be explained as a peripheral effect due to an audiometric configuration that rises from low to high frequencies, then a central auditory site must be suspected. Fourth, in the elderly patient with presbycusis, the combination of audiometric contour and direction and magnitude of the PB-SSI discrepancy yields a rough estimate of the relative contributions of peripheral and central effects to the patients total auditory impairment.

The SSI test is of value in that it can yield useful information either with or without a hearing loss. At this point, its only major drawback has been that the patient must be able to read in order to perform the task.

SUMMARY

Extensive research with tonal and speech tests has determined that the redundancy of speech stimuli (i.e. words or sentences) can be varied in order to sufficiently tax a certain individuals central auditory system at the precise level of difficulty, whereas tonal stimuli cannot. The most common methods of reducing speech redundancy have been frequency filtering, splitting, acceleration of rate, periodic interruption and variation in message length, competing messages, reversals, and alternating signals. Of interest to the present study will be the competing message, alternating signals and low-pass filtering methods.

CHAPTER III

EXPERIMENTAL METHOD

The purpose of the proposed investigation was to evaluate the integrity of the central auditory system in tinnitus patients who exhibit normal peripheral hearing. As a means of testing the hypothesis, a battery of peripheral and central auditory tests was administered to adult tinnitus patients who were determined to have normal hearing. The patients were also evaluated to determine the parameters of their tinnitus.

Subjects

The subjects for this experiment included ten normal hearing adults, ranging in age from 26-57 years, who experienced tinnitus. The mean age of the group was 41.8 years. Selection of the subjects was from the files of tinnitus patients at Kresge Hearing Research Laboratory, Portland, Oregon. Of the group of ten, four were males and six were females. Subject inclusion was based on the following criteria:

1. An age of 20-60 years, in order to eliminate possible effects due to presbycusis.
2. Responses within normal limits on a peripheral battery of tests. Normal limits for this study are considered to be:
 - a. pure-tone air conduction responses of 20dB or better for frequencies 250-8000 Hz.
 - b. a speech reception threshold (SRT) which is ± 6 dB from the pure-tone average (average of 500, 1000 and 2000 Hz).
 - c. A speech discrimination score of at least 90%.

- d. Jerger Type A tympanograms bilaterally, acoustic reflexes elicited at 500, 1000, 2000 and 4000 Hz at 65dB to 90dB SL, and negative reflex decay at 500 and 1000 Hz.
3. Tinnitus in at least one ear.
4. A minimum of a high school education (necessary for SSI test standards).

TEST STIMULI

Peripheral Auditory Tests

Each prospective subject was seen previously at the Tinnitus Clinic at the Kresge Hearing Research Laboratory. The audiometric data available in the files were used to aid in the initial selection of subjects. However, in order to confirm that a patient met the specific criteria for inclusion as a research subject, a battery of audiological tests were administered to determine the integrity of the three major portions of the peripheral auditory system: the outer and middle ear, the cochlea (inner ear) and the auditory (8th) nerve. This battery consisted of the following tests:

1. Pure-Tone Air Conduction Thresholds - Pure-tone thresholds were determined by employing the Hughson-Westlake procedure for pulsed pure-tone stimuli at 250, 500, 1000, 2000, 4000, and 8000 Hz. Threshold was considered to be the lowest level (in dB HL) at which the subject responded two out of three times.
2. Speech Reception Thresholds (SRT) - The subjects threshold for speech was determined by presenting a list of spondee words in a descending manner. The level of presentation was decreased until the subject repeated 50% or 3 out of 6 words correctly, or until the minimum limits of the audiometer was reached. This level was considered as the sub-

ject's speech reception threshold.

3. Speech Discrimination - The subject's speech discrimination score was determined using a taped version of the Northwestern University Auditory Test No. 6 (NU-6) phonetically balanced word list (50 words per list) presented at 50dB HL. The speech discrimination scores were recorded as the percentage of words correctly repeated by the subject.

4. Impedance Audiometry (included tympanometry, acoustic reflex thresholds and reflex decay).

Impedance audiometry is a relatively new procedure, and for this reason is discussed in greater detail than the other peripheral tests. The impedance tests are designed to yield information in three areas:

1. The tympanogram is a graphic portrayal of the relationship between air pressure in the external canal and impedance in the plane of the eardrum. The static compliance is also portrayed with the tympanogram, and is a factor proportional to the mobility of the ossicular mechanism.
2. The threshold of the stapedius reflex in response to pure-tones of varying frequencies is a sensitive measure of the entire reflex mechanism.
3. The reflex decay test determines the ability of the stapedius muscle to sustain a contraction in response to a ten second suprathreshold pure tone stimulus.

Results of impedance audiometry have been valuable in determining site of lesion. Through careful study of numerous subjects, specific patterns of response have been determined for the various pathologies. However, impedance testing does not yield conclusive information in itself, but as part of a test battery can provide a basis for important diagnostic decisions.

Jerger (1973) indicated that the above battery of tests is sufficient for screening the functional abilities of the ear from the outer ear through the eighth auditory nerve. Therefore, performance on these tests served as the final criteria for the acceptance or rejection of the patient as a research subject.

Central Auditory Tests

In order to test the integrity of the central auditory system, a battery of tests which will decrease the redundancy of the external stimuli being presented is needed. However, considering the complicated design of the central auditory system, the construction of a battery that will tap a patient's performance potential at exactly the right level of difficulty has been perplexing. Research has demonstrated that a listening task that is too difficult for one patient may be surprisingly simple for another. Therefore, the ability to vary the level of difficulty of the task is an essential characteristic of tests for central auditory assessment. With this consideration in mind, the following tests were chosen for inclusion in the study.

1. Performance-Intensity for Phonetically Balanced Words (PI-PB)
2. Synthetic Sentence Index - Ipsilateral Competing Message and Contralateral Competing Message (SSI-ICM and SSI-CCM)
3. Staggered Spondaic Words (SSW)
4. Low-Pass-Filtered Speech (LPFS, Low-pass cut-off frequency at 500 Hz)

The above group of tests are widely used in audiology clinics as tools for evaluating the central auditory system. The tests are easy to administer, the subject's tasks are not too difficult, and when considered as a battery yield useful information concerning the integrity

of the various levels of the central auditory system. The tests are essentially useful only with subjects who have normal peripheral hearing, since any peripheral lesion would influence performance on the test.

Each test was specifically chosen for its ability to interact with other test scores in providing diagnostic information. When considered individually, the tests have been most useful in the following ways.

1. The PI-PB test generally performs as a screening technique for detecting central problems. An ear score difference on PI-PB functions in the absence of a pure-tone sensitivity difference sufficient to account for the difference on a peripheral basis may indicate a possible central disorder. The PI-PB also helps define the extent of the central problem.
2. The performance on the two tasks, SSI-ICM and SSI-CCM is used to assist in the differentiation between brain stem and temporal lobe lesions. A significantly greater deficit for ICM than for CCM has been indicative of a brain stem problem, whereas a significantly greater deficit for CCM than for ICM usually indicates a temporal lobe problem.
3. The SSW is usually considered as a test for temporal lobe function. Considerable difficulty with the task is often evident in patients with temporal lobe lesions. Marked deviations from normal are seen on the ear contralateral to the lesion. By use of the standard norms, the test has also been used to differentiate auditory reception from non-auditory reception dysfunction.
4. The Low-pass-filtered speech test is generally used as an additional test of central auditory function. Depressed scores do not necessarily

imply a lesion in one specific region of the brain as do other tests. Rather, an abnormal low-pass-filter score in one ear can be the result of either a lesion of the brain stem or one in the temporal lobe. This test has proven to be quite sensitive to central nervous system lesions and thus was included in the battery.

Equipment

The evaluation procedures for this study were accomplished with the audiometric equipment at the Kresge Hearing Research Laboratory, Portland, Oregon. The equipment required to present the test stimuli for pure-tone and speech tests were located in the control booth of a small IAC sound proof room conforming to ANSI (1969) standards for ambient noise. A clinical audiometer (Grason-Stadler, Model 1701), coupled to TDH-49 earphones mounted in Mx 41 AR cushions were utilized to present the pure-tone and speech stimuli. Recorded monaural and binaural speech signals were transmitted from a dual channel AKAI tape recorder, Model 4000 DB, to the same two channel audiometer and earphones mentioned above.

Impedance audiometry was accomplished with an electroacoustic otoadmittance meter (Grason-Stadler, Model 1720) and a pure-tone audiometer.

The equipment utilized for the tinnitus evaluation procedure included a tinnitus synthesizer (Norwest, Model SG-1) and Koss earphones (ESP10). These earphones have the capability of transducing frequencies as high as 20,000 Hz.

The experimental equipment was calibrated to ANSI (1969) standards prior to, during and after the experiment. These measurements were accomplished electroacoustically with a sound level meter (Bruel & Kjaer,

model 2203) coupled to a condensor microphone (Brue1 & Kjaer, model 4145), housed in an NBS-9A coupler.

Experimental Procedure

A questionnaire regarding tinnitus, taken during a previous tinnitus evaluation, was utilized to determine any commonalities which may exist between subjects. The questionnaire can be found in the appendix.

The evaluation procedure began by seating each subject in the sound treated room at a right angle to the tester. An otoscopic examination was made to ensure against wax occlusion, any obvious perforation of the ear drum, or any other observed condition which appeared atypical to the examiner.

Audiological testing began with instructions to the subject concerning the peripheral test battery. Questions from the subject about the test procedures were answered at this time or during test administration if it was necessary. The peripheral battery was given in the following order:

1. Pure-tone air conduction tests
2. Speech reception thresholds
3. Speech discrimination tests
4. Impedance audiometry (tympanometry, acoustic reflex thresholds, and reflex decay)

With the completion of these tests, the subject was given a short break. At this point, test results were interpreted and a determination was made as to the patient's eligibility for inclusion as a research subject. If the subject met the criteria for the study, the tinnitus evaluation was then administered.

Tinnitus Evaluation

The tinnitus evaluation followed procedures identical to those utilized by tinnitus researchers at Kresge Hearing Research Laboratory (Johnson, 1979, p. 1). The procedures are as follows:

PROCEDURE FOR DETERMINING THE PITCH OF TINNITUS

The pitch of tinnitus will be measured in the following manner. Patients are first trained as to definitions of noise and tone with ample illustrations of each. They are then required to indicate whether their tinnitus is more like tone or more like noise . . . Depending upon the response of the subject, either a pure tone of 1000 Hz or a band of noise centered at 1000 Hz will be presented to the ear opposite the side in which the tinnitus resides for unilateral cases. For bilateral cases the comparison stimulus is placed in the ear opposite the one to be tested. The intensity of that signal will be increased until the patient responds that it is just audible. The sound will then be increased 5dB above threshold and the patient will be asked to make a judgement regarding whether the comparison tone in the opposite ear is higher or lower in pitch than their tinnitus. Following this determination, the stimulus will be increased or decreased one octave and the preceding procedure will be repeated for that frequency and all other frequencies until the patient indicates that the external tone and the tinnitus are similar. For each octave change the external signal will be adjusted so that the sensation level of the stimulus is 5dB.

When the patient indicates that the external stimulus and the tinnitus are similar in pitch, the stimulus will be slowly adjusted either continuously for pure tones or in small steps for a noise band until the patient indicates that a "good" match has occurred. At this point, the second oscillator will be set one octave above the selected stimulus. It will be adjusted to 5dB SL then the two sounds will be presented alternately until a decision is made by the patient regarding the most appropriate pitch match. This frequency or band of noise will be recorded as pitch #1. After a rest period, the same procedure will be repeated once more and the pitch match for that measurement will be recorded as pitch # 2. If these two pitches are different, the patient will be given an opportunity to select between the two frequencies as to which one most closely simulates his or her tinnitus. This choice will be recorded as the final pitch for that patient.

PROCEDURE FOR DETERMINING THE LOUDNESS OF TINNITUS

Once the subject has made a final decision regarding the stimulus that best matches the pitch of his tinnitus, that signal will then be utilized for measuring the loudness of the tinnitus. When possible, the stimulus will be presented to the ear opposite the side in which the tinnitus is being measured. The tone or noise band will be increased from a subthreshold level in 1dB steps until the tone is barely audible. This dial setting will be recorded as threshold #1. The stimulus will then be further increased in 1dB increments until the patient indicates that the tinnitus and the external stimulus are of equal intensity. This level will be recorded as tinnitus level #1. The difference between the threshold and the tinnitus level will be the loudness of the tinnitus in sensation level.

The above procedure will be repeated a second time and recorded as threshold #2 and tinnitus level #2. The two loudness measures will be averaged and that magnitude will be considered as the loudness of that patient's tinnitus.

PROCEDURE FOR DETERMINING THE MINIMUM MASKING LEVEL OF TINNITUS

Once the pitch and loudness of the tinnitus has been determined, the next procedure involves finding the Minimum Masking Level (MML) for that patient. This level can be measured either ipsilaterally, contralaterally or bilaterally. The process is essentially the same for all three modes except that the sound is presented to different ears. The process of making this measurement is as follows.

The external stimulus which best matches the patient's tinnitus is presented to the appropriate ear. The signal is increased in 1dB steps until the patient indicates that he barely hears the sound. This level is recorded as threshold #1 for that patient. The intensity of the noise is then further increased in 1dB steps until the patient's tinnitus is just masked. This is masking level #1 and is recorded as such. The difference in the intensity between the subject's threshold and the level which will just mask their tinnitus is the Minimum Masking Level #1 (MML) in sensation level (SL).

The above procedure is repeated and recorded as threshold #2 and masking level #2. Again the MML is determined and recorded as MML #2. The two MML's are then averaged and the resultant figure becomes the final Minimum Masking Level.

PROCEDURE FOR DETERMINING RESIDUAL INHIBITION

Once the Minimum Masking Level of the patient's tinnitus was de-

terminated, the next procedure involved determining the presence or absence of Residual Inhibition. Residual Inhibition is measured by presenting a constant stimulus at 10dB above the MML of the tinnitus for a period of one minute. Immediately following cessation of the stimulus, the patient is asked to describe his tinnitus. A stop watch is started at this time, and the duration of both partial and complete residual inhibition is determined. When the patient indicates that the tinnitus has returned to previous levels, the time accumulated on the stop watch is considered to be the length of residual inhibition for the patient's tinnitus.

Following the determination of the subject's tinnitus characteristics, a short rest period was given. The evaluation then proceeded to the central auditory test battery. Instructions to the subject concerning test procedures was given prior to each test, and are provided in the appendix.

Central Auditory Test Battery

As stated previously, the experimental procedure consisted of four tests which are designed to measure central auditory dysfunction. These four tests are described separately in terms of description, procedure, and scoring. In order to guard against an undesirable ordering effect, each subject was preassigned in random order to a counterbalanced presentation order of the central test battery.

Performance-Intensity for Phonetically Balanced Words

The PI-PB test consisted of tape recorded NU-6 phonetically balanced word lists. These words were presented through the earphones monaurally at a constant level of 90dB HL. Performance at the 50dB HL

level was obtained during preliminary speech discrimination testing and was considered along with PI-PB scores in the articulation gain function.

The PI-PB score will be considered positive for retrocochlear pathology if the PBmax score falls 20% or more at supra-threshold levels, i.e., positive rollover.

SSI-ICM and SSI-CCM

The SSI test was first described by Speaks and Jerger (1965). The authors suggested that discrimination tests incorporating nonsense syllables and PB monosyllabic words were not capable to assess the temporal processing abilities of the auditory system. They indicated that in order to evaluate the temporal aspects of speech, verbal materials should consist of sentences of controlled length, and controllable relative information content.

The use of synthetic sentences was pursued in order to avoid the problems involved in using "real sentences", i.e., the meaning of the sentence can be conveyed by one or two key words. It is also difficult to construct equivalent message sets of real sentences because of factors of vocabulary, word familiarity, word length, sentence length and syntactical structure. In order to avoid these contaminating variables, sentences were constructed that were both artificial and synthetic; artificial in that they were not real, and synthetic in that each new word was conditional on the preceding word(s) according to normal syntactical structure. Words used in the sentences were chosen from the 1000 most common words of the Thorndike-Lorge count (Keith, 1977).

The general rule for constructing "synthetic" sentences was to

select successive words for a sentence solely on the basis of the "conditional probabilities" of word sequences. Thus, each new word was conditional on the preceeding word or words. Third-order approximations were developed by having any new word conditioned on the two words preceeding it. An example of ten typical sentences constructed using these methods are shown below:

1. Small boat with a picture has become.
2. Built the government with the force almost.
3. Go change your car color is red.
4. Forward march said the boy had a.
5. March around without a care in your.
6. That neighbor who said business is better.
7. Battle cry and be better than ever.
8. Down by the time is real enough.
9. Agree with him only to find out.
10. Women view men with green paper should.

Early applications of the SSI showed that the task was too easy if the sentences were presented in quiet conditions, since no matter how poor a patient's discrimination score for monosyllables, the SSI score reached a maximum of 100% at high intensities. To make the task more difficult, both an ipsilateral and a contralateral competing speech message of continuous discourse was added to the sentences. Jerger (1970) has since reported the SSI test materials to be especially useful in the study of retrocochlear disorders as it is possible to reduce the redundancy of the speech message. Administration procedures for the SSI-ICM and SSI-CCM tests were as follows.

The SSI test consisted of a 2-channel tape recording of ten synthetic sentences (third order sentence approximations), as well as a recording of continuous discourse for the ICM and CCM modes of stimulation. The sentences were presented through Channel 1 of the speech circuit in the audiometer, and the continuous discourse was presented

through Channel II.

The test stimuli were presented through the earphones, monotically for the ICM (both stimuli in the test ear), and dichotically for the CCM (message in the test ear and discourse in the opposite ear). Performance was measured at several different message-to-competition ratios (MCR's) for both ICM and CCM. For the CCM, the recorded sentences were administered at MCR's of 0, -20 and -40dB, that is, with SSI at 30dB SL re SRT in the test ear, the competing message is presented at 30, 50 and 70dB SL in the opposite ear. For ICM, the test was administered at MCR's of +10, 0, -10 and -20dB. That is, with the SSI at 30dB SL re SRT, the competing message was presented at 20, 30, 40 and 50dB SL in the same ear. These levels of presentation are commonly used and are recommended by Jerger (1973).

The subject was provided with a list of the ten synthetic sentences. The responses for the SSI required the subject to identify the sentences by reporting the appropriate number of the sentences. The examiner recorded the subjects' responses on the proper form.

According to Jerger (1973), normal hearing persons will perform on the SSI-ICM at approximately the 100% level with an MCR of 0dB, 80% with an MCR of -10dB, 55% with an MCR of -20dB, and 20% with an MCR of -30dB. On this test patients with lesions of the brain stem have shown large differences in scores between both ears. With increase in the level of the competing message, scores deteriorate more rapidly than normal when the test is performed in the ear contralateral to the lesion. For example, given a patient with a lesion of the left brain stem, SSI-ICM scores will be poorer in the right ear

than in the left ear.

Jerger (1973) suggested that persons with normal hearing will perform very well (90%-100%) on the SSI-CCM test even at MCR's of -40dB. Patients with lesions of the temporal lobe perform well when the sentences are presented to the ear on the same side as the lesion with the competing message presented to the opposite ear. However, deterioration of scores is observed when the sentences are presented to the ear on the unimpaired side and the competition presented to the ear on the same side as the cortical lesion. For example, in a left cortical lesion, SSI scores will be good with sentences in the left ear and competition in the right ear, but scores will be poor with sentences in the right ear and competition in the left ear. SSI scores for patients in the proposed study were interpreted according to Jerger's (1973) standards. Characteristic SSI results on patients with eighth nerve, brain stem, and temporal lobe lesions can be found in Table 4 on the following page.

Table 4. Summary of Characteristic SSI Test Results on Patients with Eighth Nerve, Brain Stem and Temporal Lobe Lesions (Keith, 1977, p. 87).

NERVE VIII TUMOR - RIGHT

<u>SSI-Right</u>	<u>SSI-Left</u>
<u>ICM</u>	<u>ICM</u>
Poor Score	Good Score
<u>CCM</u>	<u>CCM</u>
Poor Score	Good Score

BRAIN STEM LESION - RIGHT

<u>SSI-Right</u>	<u>SSI-Left</u>
<u>ICM</u>	<u>ICM</u>
Good or Poor Score	Poor Score
<u>CCM</u>	<u>CCM</u>
Good Score	Good Score

TEMPORAL LOBE LESION - RIGHT

<u>SSI-Right</u>	<u>SSI-Left</u>
<u>ICM</u>	<u>ICM</u>
Good or Poor Score	Poor Score
<u>CCM</u>	<u>CCM</u>
Good Score	Poor Score

Staggered Spondaic Word Test

The SSW test consisted of a 2-channel tape recording of spondaic words (40 test items). A spondaic word was presented to each ear at 50dB above threshold and the subject was required to repeat both words. Each test item was composed of two spondees recorded in a partially overlapped fashion. One spondee was presented to each ear, with each ear receiving stimulation in isolation as well as in competition with each other. The time sequence of the SSW test items is illustrated below.

Table 5. An SSW Item That Starts in the Right Ear (Katz, K., In Keith, R. (ed.), 1977, p. 104.)

<u>TIME SEQUENCE</u>			
	1	2	3
RIGHT EAR	UP	STAIRS	
LEFT EAR		DOWN	TOWN
	R-NC	R-C	L-NC
		L-C	

R (Right)
 L (Left)
 C (Competing)
 NC (Non-competing)

The items were presented so that the competing and non-competing messages were reversed in terms of which ear is stimulated first. As the subject repeated the words presented, the examiner monitored responses both visually and auditorily, marking responses on the special

form prepared for SSW score interpretation.

Scoring standards for the SSW test were presented by Katz (1968). Individual errors are summed first for each ear, with both competing and non-competing syllables. The total SSW score is the total number of errors obtained for both ears. Each monosyllable is given a weight of 2.5% and is subtracted from 100% to give the percentage of correct responses. In order to avoid penalizing the patient's SSW scores for possible peripheral discrimination difficulties, the word percentage error for PB words is credited to each ear by subtracting the percentage error for PB words from the percentage error on the SSW test (Raw-SSW scores) for the same ear. This corrected SSW score (C-SSW) is then used for determining a patient's performance level.

Katz (1972) suggested that individuals from 11-60 years of age will make few errors on the SSW test (mean total R-SSW equaled 98%, standard deviation equaled 2.2%). Poor scores on the SSW test were said to suggest a lesion in the higher brain centers (auditory cortex) on the contralateral side.

SSW scores for subjects in the proposed study were interpreted using the standards presented by Katz (1968) based upon a portion of normal hearing subjects and patients with surgically confirmed or medically diagnosed central nervous problems. Performance was evaluated by examining the total C-SSW score, the C-SSW scores for each ear separately, and the C-SSW score for any condition (e.g., right competing or left competing), as shown in Table 6 on the following page.

Table 6. Upper limits for Evaluation of Performance on the SSW Test (Katz, 1968, p. 344.)

C-SSW	Normal	Mildly Abnormal	Moderately Abnormal	Severely Abnormal
Total Score	5	15	35	100
Ear Score	10	20	40	100
Condition Score	15	25	45	100

Katz stated that normal hearing subjects will have C-SSW scores no greater than the upper limits of normal given in the table above. A central auditory problem would be suspected if the total C-SSW score was over 15, the C-SSW score for either ear was over 20, or the C-SSW score for any condition was over 25.

Low-Pass-Filtered Speech

The filtered speech test consisted of tape recorded NU-6 phonetically balanced word lists which have been low-pass filtered with a cut-off frequency of 500 Hz and rejection rate of 24dB/octave. The test stimuli were presented through the earphones monaurally at a constant level of 50dB SL above the SRT (Bocca, 1958). The subject was required to repeat each stimulus word during the test. The examiner observed the responses as with the other tests and recorded the percentage of corrected responses on the scoring form.

Bocca (1958) presented normative data for low-pass filtered speech tests. Normal hearing persons displayed scores of 70-80% when tested monaurally at 50dB SL with a cut-off frequency of 800 Hz. Presentation of the test to cases of temporal lobe tumors resulted in scores of about 50% in the ear contralateral to the tumor, and 65 to 75% in the ipsi-

lateral ear. Jerger (1960) similarly reported intelligibility for filtered speech (cut-off frequency of 500 Hz) about 30% poorer on the ear contralateral to the lesion in patients with temporal lobe pathology who had essentially normal hearing sensitivity and normal discrimination scores for undistorted speech signals.

Studies on patients with brain stem lesions (Calearo and Antonelli, 1968) indicated that due to the confined area of the brain stem, slight variations in the location or extent of the lesion produced significantly different auditory results on filtered speech tests, i.e., the discrimination deficit was ipsilateral to the lesion in some patients and contralateral in others. Interpretation of test scores in the present study utilized the standards described by Bocca (1958) and Jerger (1960).

Data Analysis

As stated previously, central auditory tests do not yield conclusive information when test scores are considered separately. Research has shown that results will vary among individuals, largely depending upon the location, size and type of disorder that exists. Therefore, the results of this study are described and discussed for individual test results and later are presented in terms of individual patient performance. These results are presented utilizing descriptive statistics only. Thus, each subject served as his/her own control, with importance placed on the interrelation between the specific test scores. Results of the central auditory tests are analyzed according to the current standardized norms, as explained earlier in this chapter.

CHAPTER IV

RESULTS AND DISCUSSION

INTRODUCTION

This study was guided by the hypothesis which states that those subjects with tinnitus who do not have an accompanying peripheral hearing loss, have tinnitus as a result of a disorder in the central auditory nervous system.

The procedure used in this study was to administer a battery of peripheral and central auditory tests and a tinnitus evaluation to adult patients who were determined to have normal hearing and tinnitus. The peripheral auditory test battery included pure-tone air conduction measures, speech reception thresholds, speech discrimination tests, tympanometry, acoustic reflex thresholds and acoustic reflex decay tests. Final subject inclusion was dependent upon normal results on the peripheral auditory battery of tests.

Following inclusion as a research subject, a tinnitus evaluation was performed. The procedures followed were identical to those utilized at the Kresge Hearing Research Laboratory. Tinnitus measurements included the parameters of pitch, loudness, minimum masking level and residual inhibition.

The final stage of testing involved administration of a central auditory test battery. This battery included the Performance-Inten-

sity for Phonetically Balanced Words (PI-PB) test, a Low-Pass-Filtered Speech (LPFS) test, the Synthetic Sentence Index - Ipsilateral Competing Message (SSI-ICM) and Contralateral Competing Message (SSI-CCM), and the Staggered Spondaic Word (SSW) test. These tests were administered in a counterbalanced order to ensure against an undesirable order effect.

As stated previously, there is no single audiologic test which will yield definitive information concerning the existence and severity of a hearing loss, the locus of the lesion or lesions responsible for the pathology and possible areas of remediation of the disorder. Some tests are designed specifically for assistance in pinpointing the site of lesion while others are primarily used to determine the existence of an auditory deficit (Rosenberg, 1978). It is well known in the audiologic profession that due to individual variability, peculiarities of different audiologic pathologies and other factors, complete agreement and consistency on all the different tests performed is not to be expected. Thus, the importance of utilizing a large number of overlapping tests to create a picture that is much clearer than that presented by any single examination finding is realized.

It will be remembered that of interest to this study is the state of the central auditory nervous system in tinnitus patients who exhibit normal hearing. The peripheral auditory testing battery was administered only for the sake of determining if a subject qualified for the study. Therefore, results of these tests will not be discussed as they are assumed to be within normal limits if the subject is a participant of this study.

RESULTS

The results of this study are reported by examining the outcome for each individual central auditory test and by comparing these results of individual performance between subjects. These results are reported in Table 7. In addition to the results of the central auditory battery used in this investigation, Table 7 also includes a description of the subjects' sex and age as well as a description of the localization, pitch and quality of the tinnitus. As stated previously, six of the subjects were females and four were males. The subjects ranged in age from 26 - 57 years. The age range for this group of patients is somewhat younger than Vernon (1978) found in a survey of 513 tinnitus patients. Vernon found that 58% of his population were between the ages of 45 - 65 and only 11% were between the ages of 20 - 35 years. In the present study, 40% of the subjects were between the age of 26 - 33 years while 60% were between 42 - 57 years of age. These percentages must be considered in a somewhat guarded fashion because of the size of the present study. It seems reasonable to assume, however, that the population for the present investigation may be somewhat younger from the standpoint of the fact that none of these patients have hearing losses. It is a well established fact that a large majority of tinnitus patients present with hearing losses resulting from either noise exposure or aging process which they have accumulated over time.

The location of the tinnitus was confined to only one ear in two of the ten subjects (20%). The largest group, six out of ten subjects (60%) had it in both ears, while 20% localized the tinnitus within their head. A comparison to Vernon's (1978) data reveals figures that are in

Table 7. Results for the Central Auditory Test Battery for Each
each of the Ten Subjects in Addition to their Age, Tinnitus
Location, Quality, and Pitch and the Speech Discrimination
Score.

					Score.											
▲ Abnormal Score					L	R	L	R	L	R	L	R	L	R	L+R	
■ Abnormal Ear Difference																
F-1	26	Ears Bilat.	Tonal	5,800	100	98	96	100	84	84	93.3	83.3	96.6	100	5.0	
F-2	55	Head	Tonal	10,000	100	100	96	100	70▲	82	73.3	70.0	100	100	5.0	
M-3	42	Ears Bilat.	Cricket	12,250	100	96	88	92	88	92	66.6	50.0	100	100	0.0	
F-4	47	Ears Bilat.	Tonal	12,000	100	96	80▲	86	80	86	60.0	66.6	90	83.3	22.5	
M-5	57	Ear Right	Tonal	6,300	100	100	88	96	84	62▲	80.0	86.6	100	100	2.5	
F-6	52	Head	Tonal	8,000	100	96	84	88	84	88	60.0	80.0	100	100	0.0	
F-7	26	Ear Left	Combin.	850 7,500	100	100	96	92	78	88	76.6	46.6	100	100	0.0	
M-8	26	Ears Bilat.	Tonal	7,500	100	100	100	96	82	88	96.6	83.3	100	100	0.0	
M-9	33	Ears Bilat.	Tonal	8,000	100	100	100	100	76	80	80.0	80.0	100	100	0.0	
F-10	54	Ears Bilat.	Tonal	6,000	100	100	100	100	80	72	83.3	70.0	100	100	2.5	
Subj.#	Sex	Age	Tinni- tus Loca- tion	Tinni- tus Quality	Tinni- tus Pitch	Speech Discrim Score	Percent Correct	PI-PB 90dB HL	Percent Correct	LowPass Filter Percent Correct	SSI-ICM Percent Correct	Avg. of MCR's	SSI-CCM Percent Correct	Avg. of MCR's	SSW-Raw Score % Error	

close agreement to those of this study, i.e., in 30% it was confined to one ear only, 58% had tinnitus in both ears and 5% heard it within their head. The localization of tinnitus, therefore, does not seem to be determined by the presence or absence of a hearing impairment.

The quality of tinnitus can generally be classified as either tonal tinnitus or noise tinnitus, both of which usually have an identifiable pitch (Vernon, 1978). Table 7 reveals that in the population of the present study, 80% of the subjects had tonal tinnitus, 10% had a combination of both noise and tone and 10% reported the sensation as a cricket-like sound. Vernon found that 59% of his population had tonal tinnitus, 25% had tinnitus which simulated noise and 16% had a combination of the two types. In the present study, a larger number of patients had tonal tinnitus since no patient reported a tinnitus which they described as noise-like. Again one must be careful in making comparisons because of the differences in size between the two populations. However, the tinnitus patients who displayed normal hearing most generally have their problem as the result of such things as head trauma, viral infection or vascular problem and most of these causes are generally associated with a tonal type of tinnitus.

When the subjects were asked to match the pitch of their tinnitus to an external stimulus, the resulting matches ranged from 5800 - 12,500 Hz. Although the number of subjects precludes making any generalized statement regarding pitch identification, it certainly is in agreement with most of the previous investigations which indicate that the tinnitus in the majority of cases is above 2000 Hz (Graham, 1968 and Vernon, 1978).

Performance-Intensity for Phonetically Balanced Words (PI-PB)

The results obtained with the PI-PB rollover test are reported in Table 7 in the form of raw percentage scores and in Figure 2 as a graphic illustration comparing the results of the speech discrimination test administered at 90dB HL. The degree of rollover was defined by Jerger and Jerger (1975) as the difference between the maximum (PBmax) and minimum (PBmin) phonetically balanced word discrimination scores, with PBmin being the lowest percentage correct score at any speech level above the level yielding PBmax. Rollover indices were considered to be abnormal when performance declined more than 20% as intensity was raised above the level yielding maximum performance. For the purposes of this study, degree of rollover is defined as the difference between performance on PB word discrimination at the 50dB HL and 90dB HL levels.

In a study to determine the clinical validity of central auditory tests, Jerger and Jerger (1975) reported that abnormal rollover occurred in four of the ten brain stem patients. Two patients had abnormal rollover on both ears and two patients had abnormal rollover on the ear contralateral to the lesion only. The amount of rollover was approximately 20% on both the ipsilateral and contralateral ears of all four patients. Rollover effects were reported to be within normal limits for the six temporal lobe patients in their study. In a similar study, Jerger and Jerger (1975) found that brain stem patients with relatively good PBmax scores in both ears did not show a substantial rollover effect. In seven out of ten patients with good PBmax scores (above 80%), the amount of rollover was only 4% which is well within the range of normal performance. The remaining three patients showed a rollover

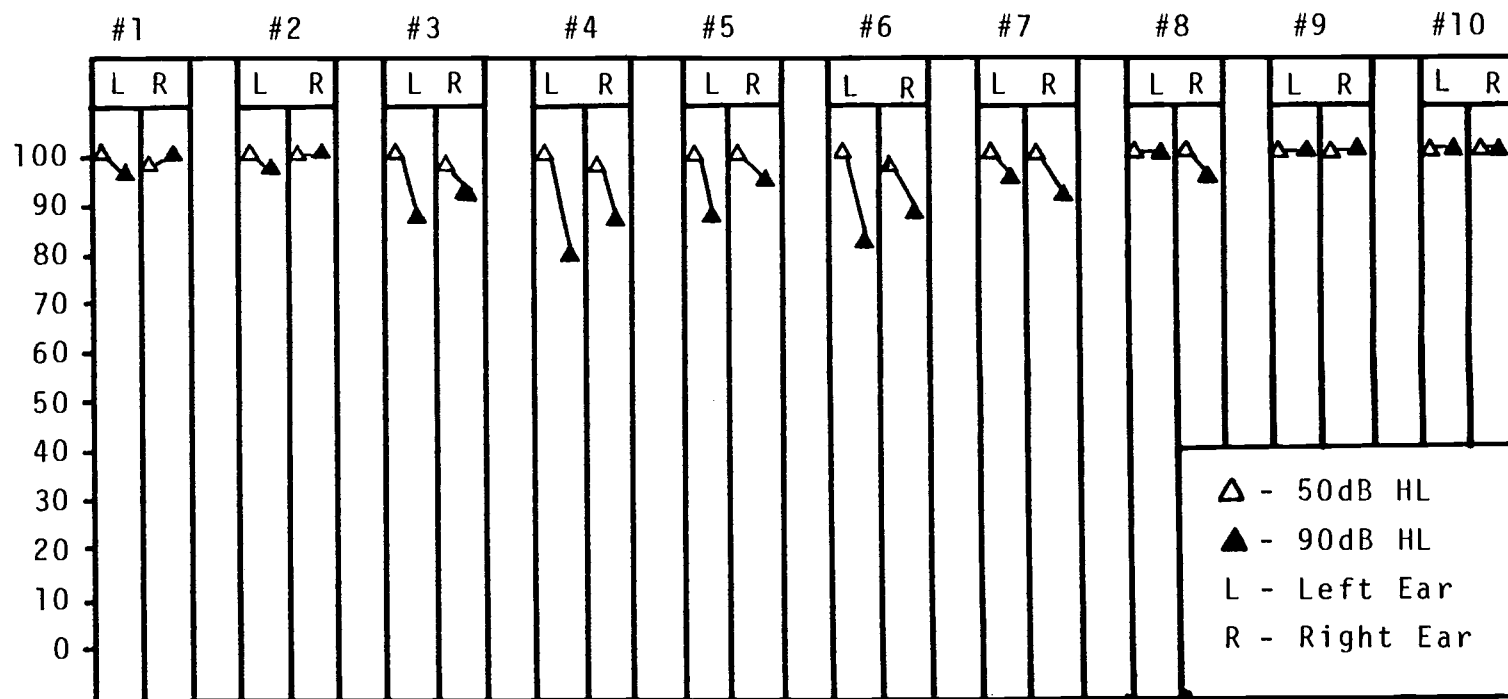


Figure 2. Results of the Performance-Intensity for Phonetically Balanced Words Test administered at 90dB HL in comparison to the Speech Discrimination Test administered at 50dB HL for each of the ten subjects.

effect of approximately 28%, with the deficit occurring on the contralateral ear for two patients and on both ears for one patient.

Inspection of the PI-PB data for this study reveals that only one of the ten subjects (F-4) shows a significant rollover effect (20%) in the left ear only. The remaining nine subjects performed normally on this test.

Low-Pass-Filtered Speech (LPFS)

The results of the filtered speech test are reported in Table 7 as the percentage of correct number of words identified and in Figure 3 as a graphic illustration of these scores. These results indicate that three of the ten subjects performed abnormally on this test. Furthermore, two subjects had abnormally low scores on filtered speech in one ear only. The remaining seven subjects performed normally on this test.

The reader will recall that the criteria established for the LPFS test can be interpreted in two ways. Bocca (1958) reported normative data for the LPFS test, with normals receiving scores of 70% and above for each ear tested monaurally. Scores below 70% are considered to be abnormal for normal listeners, i.e., possible retrocochlear involvement. As stated previously in the literature review, patients with temporal lobe lesions show a reduction of scores in the ear contralateral to the lesion. On the other hand, patients with lesions of the brain stem auditory pathways tend to produce more varied test scores with either a bilateral deficit or a unilateral deficit being either contralateral or ipsilateral to the lesion. If one interprets the data in this study according to Bocca's criteria of 70%, only two of the ten subjects (#2 and #5) fall within the abnormal category. However, a third subject

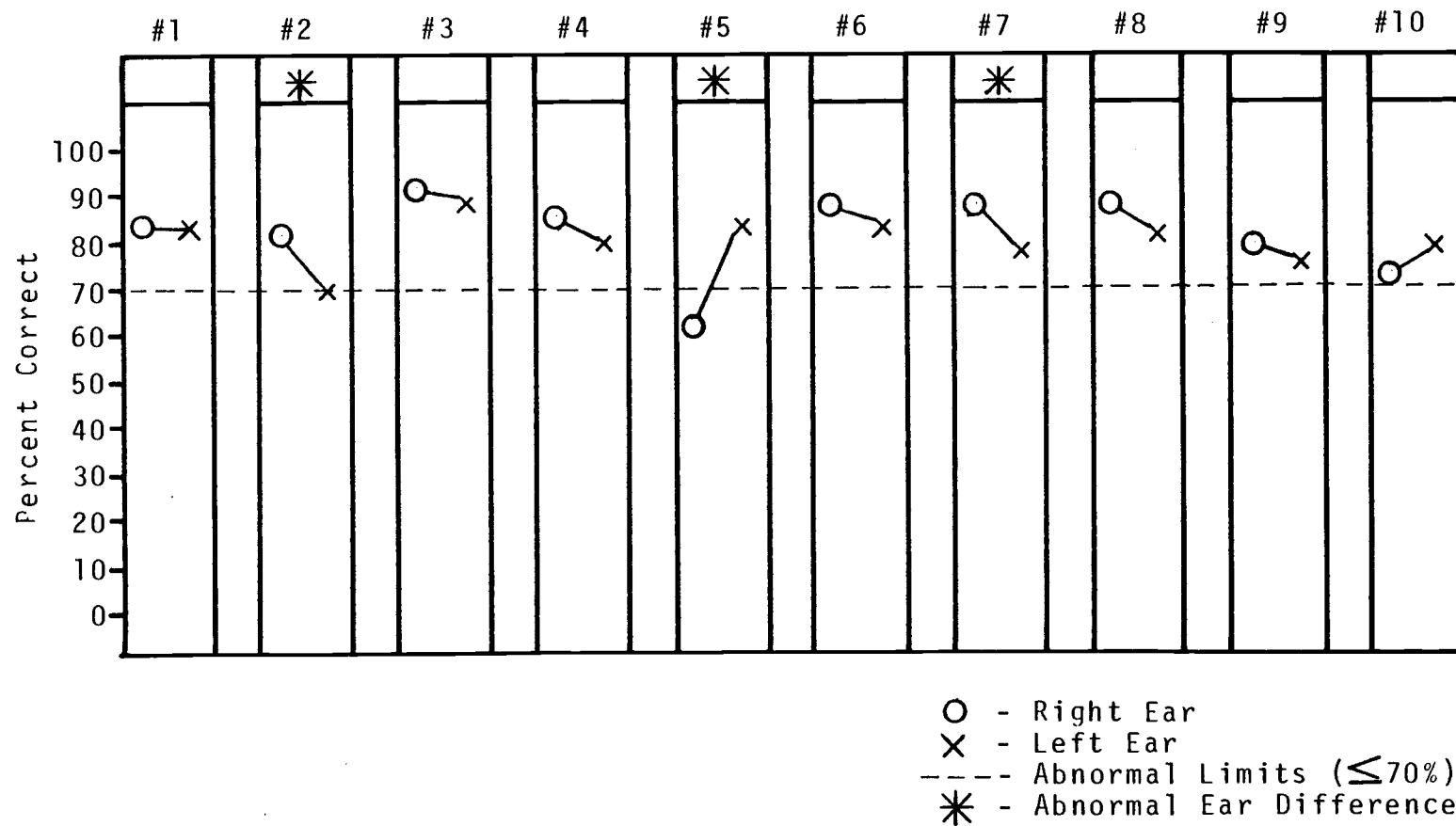


Figure 3. Results of the Low-Pass Filtered Speech Test for each of the ten subjects in this study.

(F-10) displays a score on the right ear of only 72% as opposed to 100% for unfiltered PB words which may or may not be of significance.

More importantly, however, when probing for lesions of the central auditory pathways, consideration of the performance of one ear as compared to the other is extremely important. Clinically, a 10% difference score between the two ears is considered significant and indicates a possible central nervous system lesion. In these patients with normal peripheral test scores, the ear ipsilateral to the lesion serves as the control ear.

Interpreting the test results using the differences between the ears as a comparison, three out of ten subjects (#2, #5 and #7) are considered to be abnormal listeners on the basis of the LPFS test.

Synthetic Sentence Index - Ipsilateral Competing Message (SSI-ICM)

The data pertaining to the SSI-ICM test is reported in Table 7 and shown graphically in Figure 4. Table 7 indicates the mean SSI-ICM score for each ear. This number is obtained by averaging subject performance at the 0, -10 and -20 Message to Competition Ratio (MCR) levels (Jerger, 1974). The stipled area in Figure 4 displays the mean and one standard deviation above and below the mean for each MCR. Jerger (1973) reported that normal hearing persons will perform at approximately a 100% level with an MCR of 0dB, at an 80% level with an MCR of -10dB and at a 55% level with an MCR of -20dB with an overall mean score of 77%. Those subjects falling below 77% are therefore designated as abnormal listeners on this test.

Utilizing this criteria, six of the ten subjects in this study performed abnormally. Of these six, three performed abnormally in both

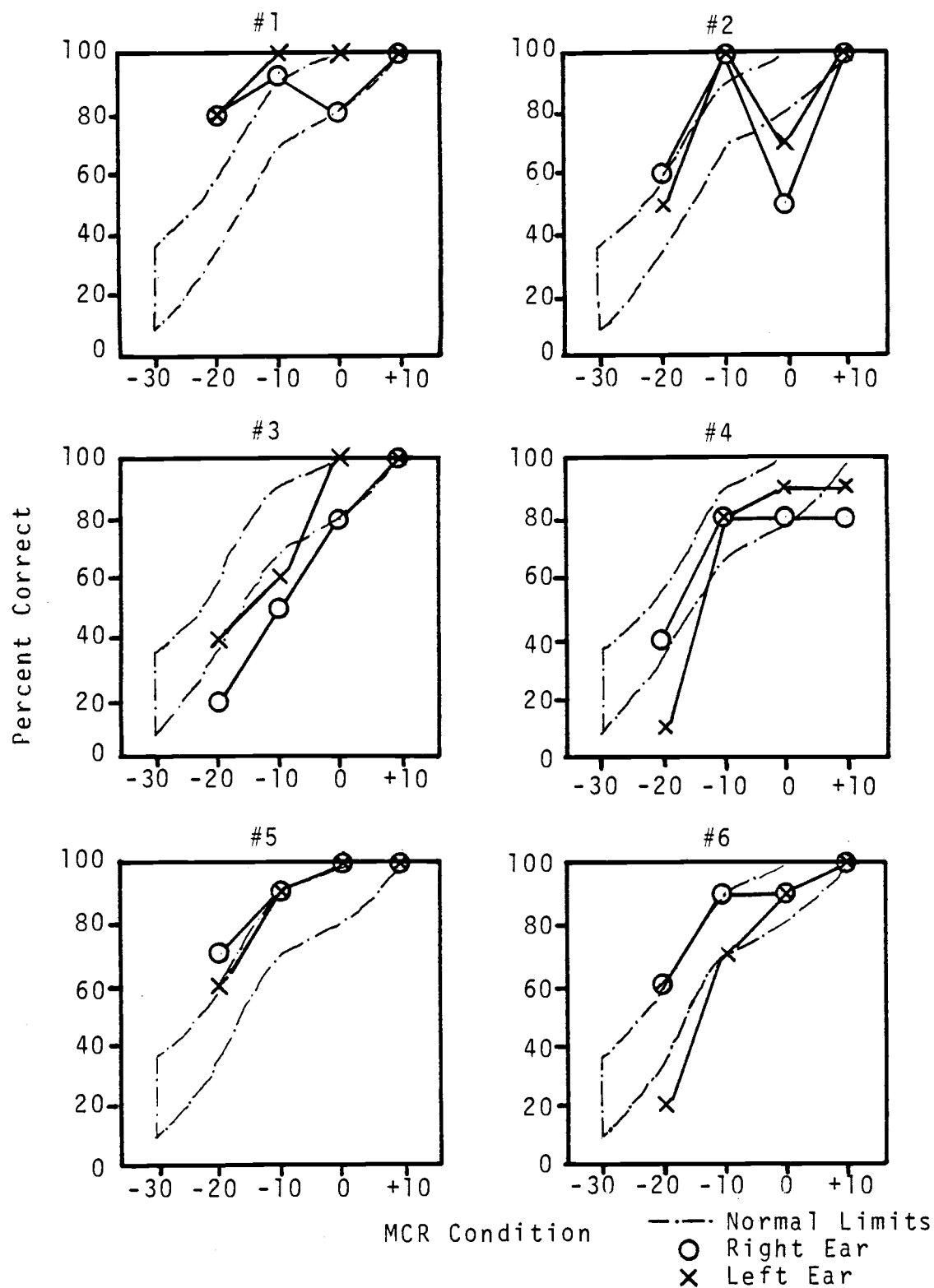


Figure 4. Results of the Synthetic Sentence Index - Ipsilateral Competing Message for each of the ten subjects in this study. (Continued on p. 94).

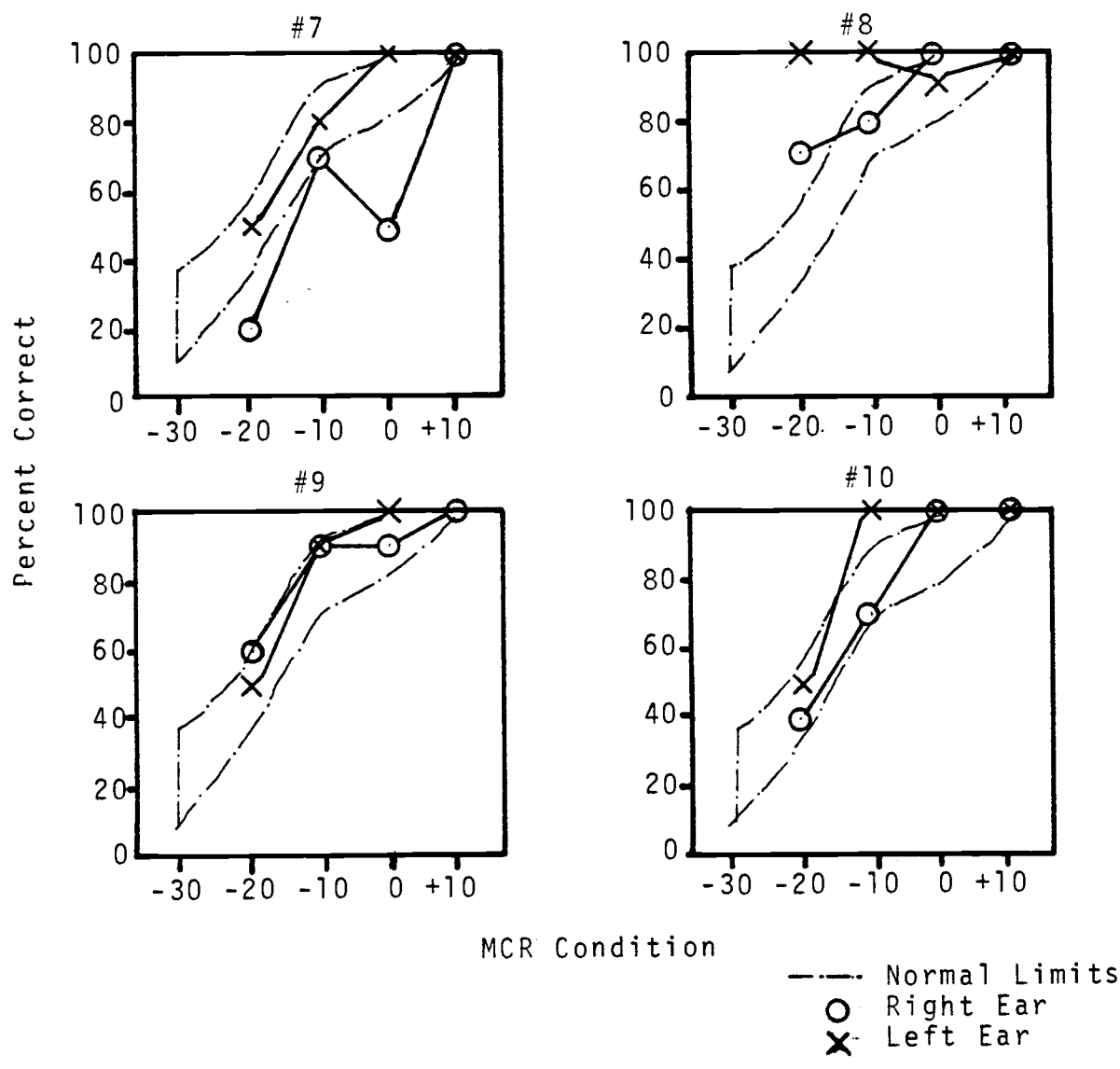


Figure 4. Synthetic Sentence Index - Ipsilateral Competing Message continued.

ears, two in the right ear and one in the left ear.

As stated previously in the literature review, the results of SSI testing are interpreted in the following manner:

Eighth nerve lesions are characterized by poor performance on the ear ipsilateral to the lesion on both the ICM and CCM tasks.

Brain stem lesions are characterized by poor performance for ICM and relatively good performance for CCM. The ICM deficits are observed on the ear contralateral to the lesion unless the lesion extends across the midline, in which case ICM performance is depressed bilaterally.

Temporal lobe lesions are characterized by poor performance on ICM and CCM. ICM deficits are observed on both ears or the ear contralateral to the lesion, and the CCM deficit observed on the ear contralateral to the affected temporal lobe. The overriding principle in these patients is that they have relatively more difficulty for the CCM than for ICM tasks. (Keith, 1977, p. 86)

Synthetic Sentence Index - Contralateral Competing Message (SSI-CCM)

The results of the SSI-CCM are reported in Table 7 and shown graphically in Figure 5. Table 7 shows data in the form of mean SSI-CCM performance for both ears and indicates abnormal performance where applicable. Studies by Jerger (1974) revealed that performance for normal listeners ranged from 90 - 100% for the three MCR's (0, -20 and -40dB) tested. Thus, on the basis of these criteria, results indicate that only one subject (F-4) out of ten performed abnormally on this test. As stated previously, brain stem lesions are characterized by relatively good performance on the ear contralateral to the lesion.

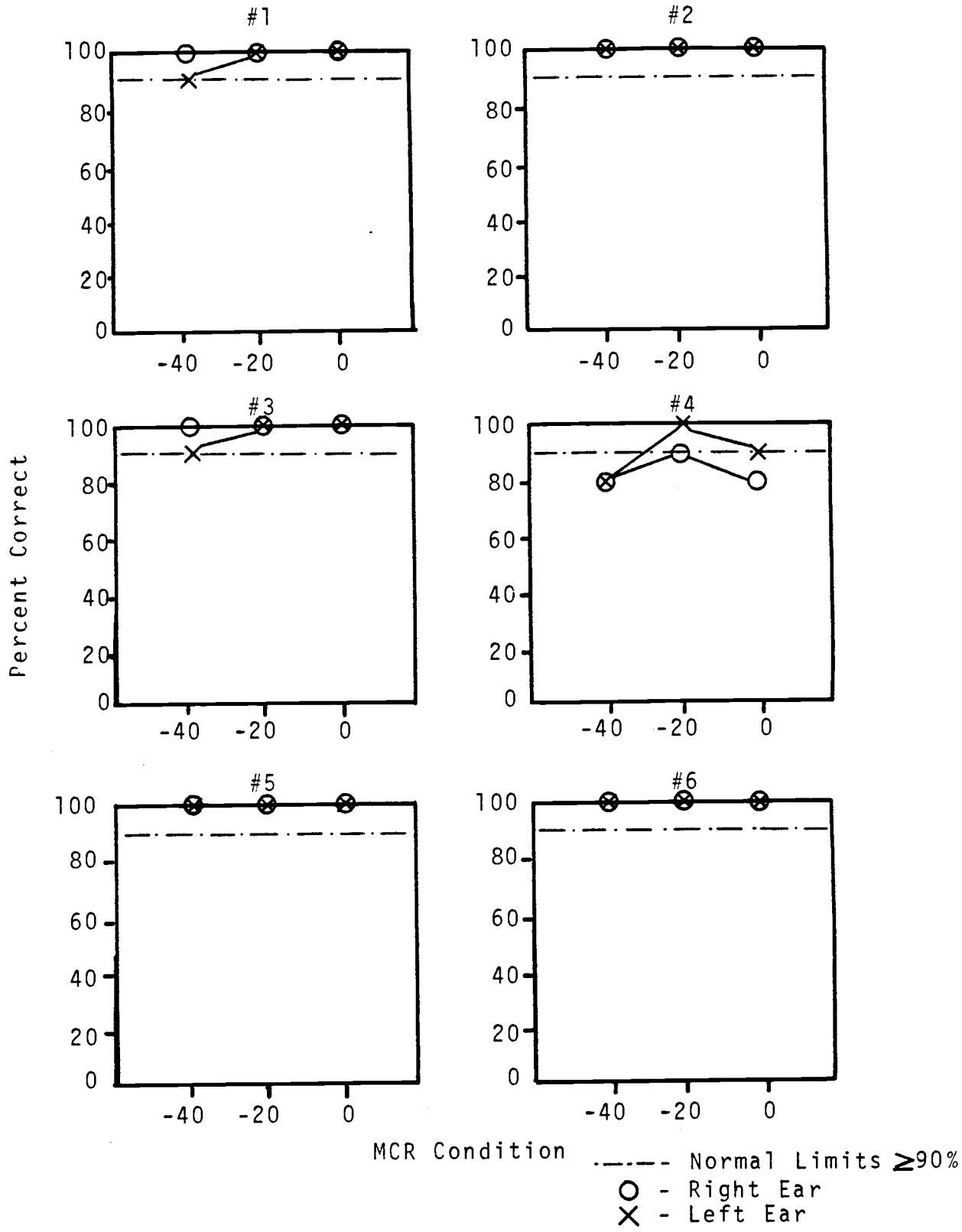


Figure 5. Results of the Synthetic Sentence Index - Contralateral Competing Message for each of the ten subjects. (Continued on p. 97)

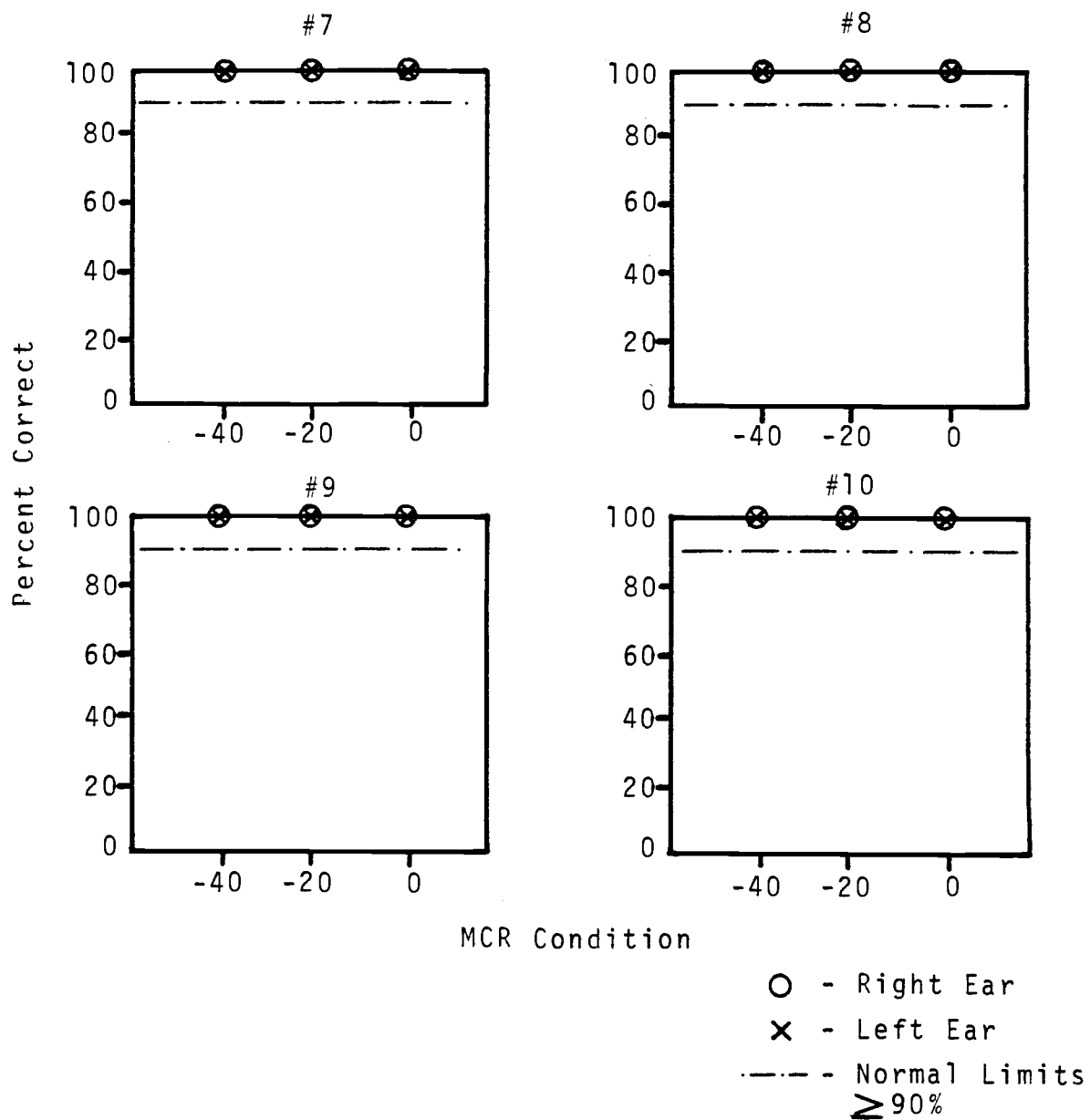


Figure 5. Synthetic Sentence Index - Contralateral Competing Message continued.

Staggered Spondaic Word (SSW) Test

The results of the SSW test are reported in Table 7 and Figure 6. Table 7 represents data in the form of the Total Raw-percentage scores and indicates whether results are within the normal or abnormal range. Figure 6 is a graphic illustration of the Total Raw-percentage scores with the normal, mildly abnormal, moderately abnormal and severely abnormal limits indicated on the graph.

Standardized normative studies by Katz et al (1963) suggested that normal hearing individuals from 11 - 60 years of age would make few errors on the SSW test. The test was administered to 40 normal hearing subjects, with the mean Total Raw-percentage score being 98% and the poorest individual score being 94%. Studies by Brunt (1969) performed in a typical clinical setting revealed similar findings. In order to determine abnormal performance on the SSW test, percentage scores for the right and left ear are averaged to produce a Total Raw SSW score. A central auditory problem is suspected if the total SSW score exceeds 15%

Inspection of the SSW results in Table 7 shows that only one subject's performance was in the abnormal limits when scores for both right and left ears were combined to make the Total Raw-Percentage score. Reference to Figure 6 shows this score to be in the mild to moderately abnormal range, with the remaining nine subjects in the normal to mildly abnormal ranges. Katz (1972) stated that subjects whose scores are in the mildly abnormal range are not suspected of having a central auditory reception disorder.

Clinical findings of Katz and others suggest that the SSW test is most useful in detecting cortical auditory problems especially in pa-

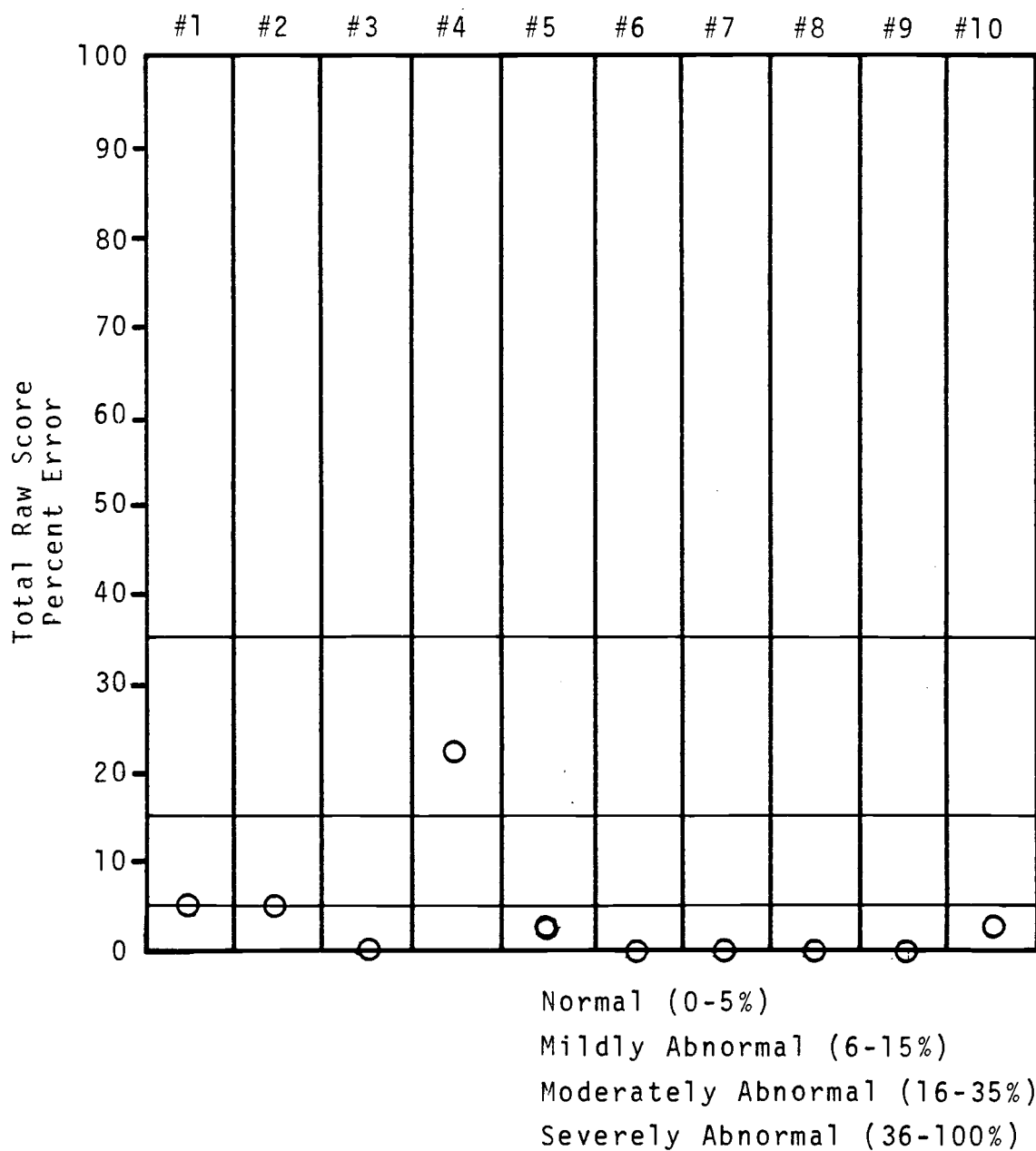


Figure 6. Summary of the Staggered Spondaic Words Test results indicating the Total Raw Score, obtained by combining the right and left ear scores for each individual subject.

tients with primary auditory reception disorders. Damage outside of this area was found to result in normal or mildly abnormal performance expect in cases of corpus callosum disorder. In general, however, past studies lend support to the SSW test as being sensitive to cerebral auditory dysfunction, showing reduced performance in the ear opposite the involved hemisphere.

DISCUSSION

Although the results reported above are certainly not definitive in nature, they are most interesting and allow for one to speculate regarding possible site of lesion for certain subjects in this study. One can immediately divide these subjects into two groups, one in which findings were completely normal and the second group where one or more tests indicate a possible abnormality. It is again important to remind the reader that these tests cannot be used separately to identify central auditory disorders, but must be viewed as a test battery.

For the three patients with normal test results (F-1, F-8 and M-9), the tinnitus is most probably idiopathic in nature. However, it is interesting to note that although the hearing of these subjects is within the normal range of hearing, mild hearing losses corresponding to the region of the tinnitus pitch can be evidenced for each case.

Figure 7 graphically illustrates a comparison of the tinnitus pitch to the pure-tone audiogram results for the three patients with normal test results. Reference to this Figure reveals that the frequency of the tinnitus for each of these subjects corresponds closely to the point at which a mild decline in hearing is evidenced.

Fowler (1940) suggested that the frequency of the tinnitus may

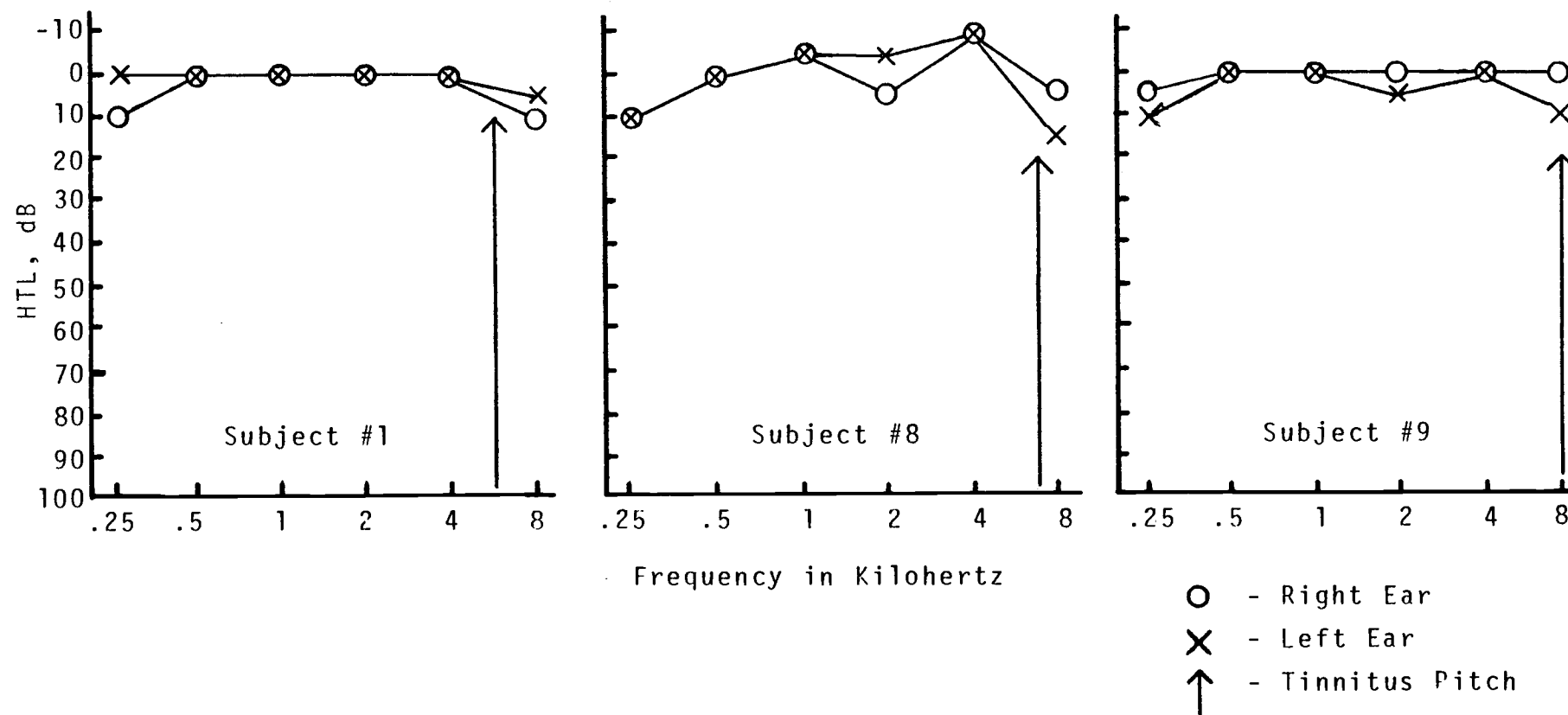


Figure 7. Pure-tone air conduction audiograms for 3 subjects with normal performance on the Central Auditory Test Battery in addition to indications of tinnitus pitch for each subject.

coincide with the edge of the lesion on the organ of Corti in the cochlea, which in turn would find the frequency of tinnitus corresponding to the frequency at which the hearing decreases on the audiogram. This phenomenon does not always occur, however, as numerous patients have reportedly identified the pitch of their tinnitus at various frequencies within the depressed area (Johnson, 1979).

It is evident from the literature that there is not as yet an agreement concerning the pitch of tinnitus in relation to side of lesion within the cochlea. Therefore, the reality and significance of this finding under consideration must be questioned until it is further studied. In spite of this however, some interesting speculations concerning this phenomenon can be made.

1. The tinnitus causes a masking effect at it's specific frequency creating the illusion of a hearing loss in that frequency region.
2. As the hearing loss progresses to the lower frequencies, the pitch of the tinnitus does not change, i.e., if the patient has a high frequency hearing loss that in its initial stages affects only the frequencies above 4000 Hz the tinnitus may very readily be matched to that frequency.- As the hearing loss progresses over time, it may be that the pitch of the tinnitus will not change in a corresponding way. (See Figure 8.)

For the remaining seven subjects in this study (F-2, M-3, F-4, M-5, F-6, F-7 and F-10), abnormalities on at least one of the central auditory tests was found. Reference to Table 7 reveals that of all the tests in the central battery, the SSI-ICM was the most sensitive. Six of the seven subjects under consideration performed abnormally on this test,

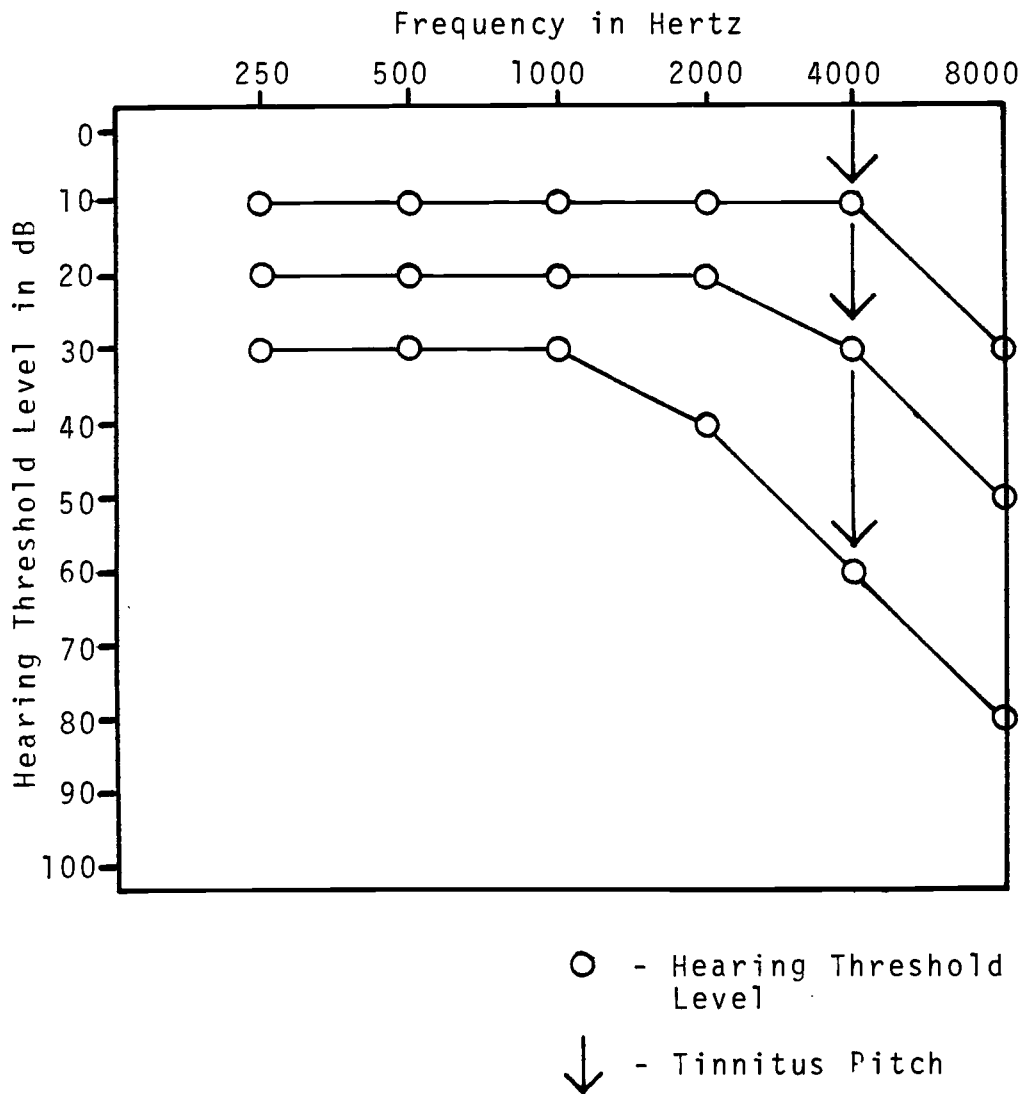


Figure 8. Pure-tone air conduction audiogram of a hypothetical tinnitus patient, indicating that as the hearing loss progresses over time, the pitch of the tinnitus does not change in a corresponding way.

with four subjects showing reduced scores bilaterally and two subjects revealing performance deficits in one ear only. It is remembered that the SSI-ICM test is a test of brain stem auditory function.

The Low-pass filtered speech test was the second most effective test with three of the seven subjects (F-2, M-5 and F-7) performing abnormally in one ear only. The LPFS test is essentially used for determining the integrity of the auditory portion of the temporal lobe; however, individuals with lesions in the brain stem have also shown difficulty with this task.

Only one of the seven subjects (F-4) performed abnormally on the remaining three tests, the PI-PB test, the SSI-CCM test and the SSW test. While the PI-PB test is essentially a screening device for lesions of the central auditory system in general, the SSI-CCM and SSW tests were designed for use in detecting disorders of the temporal lobe. Therefore, the findings in this case indicate that the site of lesion may not be limited to any specific area of the auditory system but may be more diffuse in nature.

Auditory findings in this series of patients varied considerably on some tests of central auditory function, yet, showed a degree of consistency on other tests. Listening tasks that were easy for some subjects were more difficult for other subjects. When one considers the overall test results in Table 7, there emerges some interesting patterns for the seven abnormal subjects in this study. Initially it is clear that a disorder of the brain stem auditory pathways is a strong possibility among most of these subjects (F-2, F-3, F-4, F-6, F-7 and F-10) due to the widespread difficulty on the SSI-ICM test.

Among the six subjects who performed abnormally on this test, three performed abnormally in both ears and three performed abnormally in one ear only. Performance scores varied for these subjects, ranging from 46.6% - 73.3%. It is remembered that a score equal to or greater than 77% is considered to be normal performance.

Results on the SSI-ICM test for the six subjects under consideration are similar to those reported by Jerger and Jerger (1974) concerning eleven patients with intra-axial brain stem disorders. In short, the ICM task produced unusually poor performance scores in all eleven of his patients. Six patients had abnormal scores on both ears and five patients had difficulty on the contralateral ear only. No patient had poor performance on the ipsilateral ear only. Furthermore, in the six patients with abnormal ICM scores for both ears, scores for the contralateral ear were generally poorer than for the ipsilateral ear, but not greatly. Performance scores (mean SSI-ICM scores) were approximately 37% on both the ipsilateral and contralateral ears. For the remaining five patients with abnormal scores on the contralateral ear only, mean scores were 82% on the ipsilateral ear and 44% on the contralateral ear.

This study (Jerger and Jerger, 1974) and others have reported an overall picture of brain stem symptoms that is generally characterized by normal hearing, little or no impairment in PBmax scores or PI-PB rollover and consistent impairment on the SSI-ICM test with either bilateral or contralateral symptoms. Jerger and Jerger (1975) also compared results of the SSI-CCM and SSW tests and found significantly reduced scores for the temporal lobe group, but not for the brain stem group alone.

If one interprets the SSI-ICM and CCM data in the present study according to the above criteria, two subjects (F-4 and F-6) are suspected of a lesion specific to the brain stem on the right side and four subjects (F-2, F-3, F-7 and F-10) have a possible lesion specific to the left brain stem.

Considering results on the remaining central test battery, Table 7 reveals that three of the six subjects (M-3, F-6 and F-10) who performed abnormally on the SSI-ICM test did not experience difficulty on any of the other central tests, with the exception of borderline abnormal performance on the LPFS test for one of these three subjects. Recall, however, that many patients with brain stem lesions do not show deficits for the PI-PB test, and the remaining tests in the battery are essentially used for assessing temporal lobe functions. Thus, performance scores on the SSI-ICM test were considered to be reduced enough to raise the suspicion of a brain stem disorder in at least two of the three subjects under consideration (M-3 and F-6). One subject (F-10) reveals an abnormal mean score in the right ear; however, when results of the scores at each MCR condition are considered separately (see Figure 4) they are found to be in the normal range of performance. Scores for both the LPFS test and the SSI-ICM test were borderline abnormal in the right ear for this subject; therefore, suspicion of a lesion in the brain stem pathways is seemingly not as strong as it is for the other subjects in this study.

Further reference to Table 7 reveals that two of the six subjects (F-2 and F-6) with abnormal scores on the SSI-ICM test also performed abnormally on the LPFS test. One subject (F-2) performed abnormally in

both ears on the SSI-ICM test (poorer in the right ear) and showed an abnormal ear difference and an abnormal score in the left ear on the LPFS test. The second subject (F-7) performed abnormally in the right ear only on the SSI-ICM test and showed an abnormal ear difference (poorer in the left ear) on the LPFS test.

While the SSI-ICM test is a test for brain stem function, the LPFS test is generally used in detecting temporal lobe lesions. As previously mentioned, however, sometimes patients with lesions of the brain stem perform abnormally on the LPFS material. Researchers have not seen consistent findings or patterns which are unique to brain stem lesions, with abnormal scores being in the ear contralateral to the lesion in some cases and in other instances scores may be reduced in the ipsilateral ear or both ears.

For the two patients with abnormal performance on both the SSI-ICM and the LPFS test, a lesion specific to the brain stem is more highly suspected than a lesion in the temporal lobe on the basis that the scores for the two more sensitive tests of temporal lobe function, the SSI-CCM and the SSW test, were within normal limits in both cases. Although performance was abnormal in opposite ears on the two tests (SSI-ICM and LPFS) for these subjects, this is consistent with other studies concerning audiometric patterns of brain stem lesions.

Reference to Table 7 reveals that the one remaining subject (F-4) of the six who performed abnormally on the SSI-ICM test also experienced difficulty on three of the other four central auditory tests. An overall review of the test scores for this subject reveals that performance was abnormal in the left ear on the PI-PB test, in both ears on the SSI-ICM test (poorer to the left), in the right ear on

the SSI-CCM test and in both ears (poorer to the right) on the SSW test. The LPFS test was the only test with a normal performance score for this subject.

When dealing with tests that are designed to detect specific lesions along the central auditory pathway, it is necessary to determine which task was most difficult for the patient to perform. Examination of scores in Table 7 reveals that within the battery of tests administered, the SSI-ICM test posed the greatest amount of difficulty, whereas scores were borderline between normal and mildly impaired performance on the SSI-CCM and the SSW tests. Although scores reveal a greater amount of difficulty on the brain stem test, if a lesion exists in the central auditory nervous system it is possible that it is affecting a broader region than just the brain stem.

Table 7 reveals that one subject (M-5) performed abnormally on the LPFS test in the right ear only and experienced no difficulty on the remaining central test battery. While the LPFS test is considered a useful instrument in assessing the integrity of the central auditory system, it is not as definitive as the SSI and SSW tests are on its own. Recall that the LPFS test reveals abnormal scores in patients with both brain stem and temporal lobe lesions.

A lesion in either the brain stem or temporal lobe is not as strongly suspected for this subject (M-5) on the basis that the scores for the more sensitive SSI and SSW tests were in the normal range. It is evident that there is a definite difference in performance between the two ears which is not explained by the pure-tone audiogram or speech discrimination performance. Other factors such as age, dis-

traction and motivation to respond must also be considered when considering a subject's performance. In the author's opinion, however, these factors were not felt to contribute to this subject's performance. Therefore, it is not possible to make a definite statement concerning site of lesion for subject M-5 on the basis of the abnormal scores on the LPFS test alone.

On the basis of the information presented above the following conclusions seem reasonable.

1. Tinnitus subjects with normal peripheral hearing appear to be younger in age than those with hearing impairments.
2. The location of the tinnitus and the sex of the subject do not appear to be significantly different from other tinnitus patients.
3. The tinnitus pitch of normal hearing tinnitus subjects is restricted to the high frequencies.
4. The quality of tinnitus for this group of subjects is tonal rather than noise-like.
5. Not all tinnitus subjects with normal hearing experience difficulty on tests of central auditory function.
6. The SSI-ICM test appears to be the most sensitive instrument for detecting possible lesions in the central auditory nervous system of tinnitus patients with normal peripheral hearing.
7. Overall results of this study strongly suggest that the abnormalities found in seven out of ten subjects were due to disorders of the brain stem rather than the temporal lobe.

On the basis of the results and conclusions discussed above, the hypothesis which states that those patients with tinnitus who do not

have an accompanying peripheral hearing loss, have tinnitus as a result of a disorder in the central auditory nervous system can be accepted.

CHAPTER 5

SUMMARY AND CONCLUSIONS

INTRODUCTION

The phenomenon described by many individuals as "roaring", "buzzing" or "ringing" in the ears is referred to as tinnitus. These noises, reportedly heard either in the ears or in the head, have been a common symptom in otologic practice throughout history. Many attempts have been made to determine the nature and causes of tinnitus although there is no complete agreement concerning this as yet. While great strides have been made in providing relief for these individuals, the diversity and variety of symptoms among tinnitus patients continues to perplex researchers.

Early experimenters dealing with tinnitus reported that it was always accompanied with a hearing loss; however, continued research revealed that normal hearing individuals are also afflicted with tinnitus.

Recent experiments in the field of auditory disorders have revealed that most individuals with disorders of the auditory region of the brain stem and temporal lobe also have normal hearing. Further study revealed, however, that in spite of normal hearing these individuals were unable to perform well on more difficult tests designed for assessing central auditory function. The problems these individuals had on these tests appeared to be the result of disorders of the central auditory system.

In light of this, it seemed reasonable that disorders of the central auditory nervous system could be responsible for initiating the neural activity that produces tinnitus.

It seemed evident that because of the uncertainty surrounding tinnitus and the fact that the central auditory system had not been formally studied among normal hearing tinnitus patients, further investigation was necessary in this area. Therefore, the present study was designed to investigate the condition of the central auditory nervous system in subjects experiencing tinnitus in the presence of a normal peripheral auditory system.

EXPERIMENTAL DESIGN

The present study was guided by the hypothesis which stated that those patients with tinnitus who do not have an accompanying peripheral hearing loss, have tinnitus as a result of a disorder in the central auditory nervous system. The procedure designed to test this hypothesis was to administer a battery of central auditory tests to adult tinnitus patients who had been previously determined to have normal hearing. The tests were chosen specifically to assess the integrity of the central auditory nervous system from the brain stem through the temporal lobe. The test battery included: 1) the Performance vs. Intensity for Phonetically Balanced Words test (PI-PB) which is generally a central auditory screening device; 2) the Low-Pass-Filtered Speech test (LPFS) which tests the function of both the brain stem and temporal lobe; 3) the Synthetic Sentence Index - Ipsilateral Competing Message (SSI-ICM) which tests the function of the brain stem; 4) the Synthetic Sentence Index - Contralateral Competing Message (SSI-CCM) which tests

the function of the temporal lobes; and 5) the Staggered Spondaic Word (SSW) test which tests the function of the temporal lobes. A tinnitus evaluation was also performed in order to investigate the parameters of tinnitus location, pitch, quality, loudness, minimum masking level and residual inhibition. Test scores of the central battery were interpreted by utilizing the standards described in the literature.

RESULTS AND DISCUSSION

The results for this investigation were discussed by reporting the scores of each individual central auditory test and by comparing these results of individual performance between subjects. In addition, the subject's age and sex as well as tinnitus location, pitch and quality were reported and compared to a previous study. This comparison revealed that the normal hearing tinnitus subjects of this study did not differ greatly from hearing impaired tinnitus patients regarding sex and tinnitus location. There was a higher degree of high pitched tonal tinnitus among the subjects in this study as well as a younger age range; however, these differences must be considered cautiously due to the size of the present study.

Four central auditory tests were administered to the subjects in this study. For the PI-PB test, abnormal rollover occurred for one of the ten subjects in one ear only. For the LPFS test, three of the ten subjects performed abnormally in one ear only. The SSI-ICM test was the most sensitive test in this study as six of the ten subjects performed abnormally. Of these six subjects, three performed abnormally in both ears, two in the right ear and one in the left ear. For the

SSI-CCM test, one of the ten subjects performed abnormally in one ear. Finally, for the SSW test, one of the ten subjects performed abnormally in one ear.

The results of this study, although not entirely definitive, are most interesting. Three subjects performed normally on the entire central test battery and the cause of their tinnitus is unknown. The remaining seven subjects performed abnormally on at least one of the central auditory tests. According to standardized criteria for interpreting the separate tests, five of the seven abnormal subjects were suspected of a disorder of the brain stem, one of the seven subjects was suspected of a more diffuse lesion involving the brain stem and temporal lobe and site of lesion was questionable for the remaining subject.

CONCLUSIONS

From these results the following conclusions were drawn.

1. Tinnitus patients with normal peripheral hearing appear to be younger in age than those with hearing impairments.
2. The location of the tinnitus and the sex of the subjects do not appear to be significantly different from other tinnitus subjects.
3. The pitch of normal hearing tinnitus subjects is restricted to the high frequencies.
4. The quality of tinnitus for this group of subjects is tonal rather than noise-like.
5. Not all tinnitus patients with normal hearing experience difficulty on tests of central auditory function.
6. The SSI-ICM test appears to be the most sensitive instrument for de-

tecting possible lesions in the central auditory nervous system of tinnitus patients with normal peripheral hearing.

7. Overall results of this study strongly suggest that the abnormalities found in seven out of ten patients were due to disorders of the brain stem rather than the temporal lobe.

8. Finally, the possible disorders of the brain stem in these seven subjects could be responsible for initiating the neural activity that produces tinnitus; thus, the hypothesis is accepted.

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APPENDICES

APPENDIX A

Instructions to patients prior to central auditory tests

PI-PB

This is a test of your ability to understand and repeat single words in each ear separately. You will hear a man's voice presenting some lists of one syllable words. The word lists will be presented at three different loudness levels. When the words are being heard in the test ear, you will be hearing a wind-type noise in your opposite ear. Try to ignore the noise and listen only to the words. Repeat each word exactly as you heard it immediately following its presentation. If you're not sure of the word please take a guess.

SSI-ICM & SSI-CCM

This is a test of your ability to identify the nonsense sentences you see on this paper in front of you. After you hear the sentences, please indicate the number of the sentence you think you heard. You will listen to seven sets of sentences in each ear. There will be two different ways that the sentences will be presented to you. During the first three sets, the sentences will be heard in one ear and a story about Davey Crockett will be heard in the opposite ear. During the second four sets, the sentences and the story will both be heard

in the same ear. Throughout the entire test, try to ignore the story and listen to the sentences.

SSW

This is a test of your ability to hear and repeat pairs of two syllable words, such as upstairs and downtown. The words will be overlapped so you will hear the first syllable of one word in one ear, and while the second syllable is presented, the first syllable of the other word is presented to the other ear, and then the second syllable of the second word is presented alone. The ear receiving the first syllable will change with each test item. Try to listen to both words and repeat them in the order that you hear them. Two practice items will be given before we start. Please repeat them both immediately after you hear them.

LPFS

This is a test of your ability to hear and repeat distorted one syllable words. You will hear a tape recording of a mans voice presenting a list of words in one ear at a time. Listen carefully and repeat the word immediately after you hear it. These words may be difficult to understand, but please take a guess even if you are uncertain. You will hear the same wind-type noise you heard previously in the opposite ear, and as before, try to ignore it and listen only for the words.

APPENDIX B

TINNITUS CLINIC QUESTIONNAIRE

1. I have had tinnitus in its present form for:

(circle the appropriate
letter)

- a. less than a year
- b. one to two years
- c. two to three years
- d. three to five years
- e. longer than five years

2. Prior to my present form of tinnitus I had a mild tinnitus for
_____ years.

(number)

3. My tinnitus seems to be primarily located in:

(circle the appropriate
letter)

- a. the left ear
- b. the right ear
- c. both ears equally
- d. both ears but unequal
- e. my head

4. The severity of my tinnitus in its worse form, according to the scale below, is represented by the number:

1	2	3	4	5	6	7	8	9	10
mild				moderate					extremely
tinnitus				severity					severe

5. The loudness of my tinnitus is:

(circle the appropriate
letter)

- a. fairly constant from day to day
- b. fluctuates widely being very loud on some days and very mild on other days
- c. usually constant but on rare occasions will decrease markedly

6. The loudness level of my tinnitus is best compared to:

(circle the appropriate
letter)

- a. the loudness of a jet aircraft taking off
- b. the loudness of a diesel truck motor
- c. the loudness of a jackhammer
- d. the loudness of a police siren
- e. the loudness of an electric fan
- f. other _____

Referred to the Tinnitus Clinic by: _____

7. On the scale below indicate the pitch of your tinnitus. It might help to imagine the scale as if it were a piano keyboard.

1	2	3	4	5	6	7	8	9	10
low				middle					high
pitch				pitch					pitch

8. Check any items below which describe how your tinnitus sounds:

(circle the appropriate letter)

- a. hissing
- b. cricket-like
- c. pounding
- d. pulsating
- e. whistle
- f. ringing
- g. steam whistle
- h. bells
- i. clanging
- j. ocean roaring

9. My tinnitus appears worse:

(circle the appropriate letter)

- a. when I am tired
- b. when I am tense and nervous
- c. when I am relaxed
- d. after use of alcohol

10. Do you smoke?(circle one) YES NO

If so for how long have you been a smoker _____ years

If so how many cigarettes per day _____

11. Do you drink coffee? (circle one) YES NO

If so how many cups per day? _____

12. Check any of the following items which give you any relief from your tinnitus.

(circle the appropriate letter)

- a. listening to radio or T.V.
- b. traffic sounds
- c. sounds of running water(e.g., shower)
- d. medication (_____ Kind)
- e. changes in altitude
- f. Other _____

13. Have you ever received a head injury? (circle one) YES NO

If so were you knocked unconscious? (circle one) YES NO

How long ago was the accident? _____ years

14. Have you been exposed to loud sounds? (circle one) YES NO

explain briefly _____

15. Are you presently working in or exposed to loud sounds? (circle one) YES NO

explain briefly _____

16. Do you wear ear protection in the presence of loud sounds? _____

17. Have you ever worn a Hearing Aid? (circle one) YES NO
If yes do you currently wear it? (circle one) YES NO

18. Do you have any of the following?
(circle the appropriate letter) a. high blood pressure
b. diabetes
c. allergies
d. other _____

19. Tinnitus causes me problems getting to sleep. (circle one) YES NO

20. If you are a Hearing Aid user, how does the aid affect your tinnitus _____

21. Are you taking any medications? (circle one) YES NO

22. What medications? _____

23. Have you any history of ear disease? Explain _____

24. Have you a hearing loss? (circle one) YES NO
Right ear Left ear

additional comments

