1976

An Experimental Application of the Binaural Beat Phenomenon with Aphasic Patients

Pamela S. Herbert

University of Central Florida

This Masters Thesis (Open Access) is brought to you for free and open access by STARS. It has been accepted for inclusion in Retrospective Theses and Dissertations by an authorized administrator of STARS. For more information, please contact STARS@ucf.edu.

STARS Citation
https://stars.library.ucf.edu/rtd/222
AN EXPERIMENTAL APPLICATION OF THE BINAURAL BEAT PHENOMENON WITH APHASIC PATIENTS

BY

PAMELA S. HERBERT
B.A., Capital University, 1974

THESIS

Submitted in partial fulfillment of the requirements for the degree of Master of Arts: Communication in the Graduate Studies Program of the College of Social Sciences Florida Technological University

Orlando, Florida
1976
ACKNOWLEDGEMENTS

Throughout the course of this research numerous persons have given of themselves in order to make this a reality.

I would especially like to thank Dr. Thomas A. Mullin, my thesis advisor, for his professionalism and attention to detail and for the many hours he has spent with the material. Without his guidance and patience this thesis could never have been completed.

I would also like to thank Dr. David Barr for his assistance throughout this research, as well as the other members of my committee, Dr. Gladys Bennett and Dr. David Ingram. All have been instrumental in the completion of this work.

To Maggie Richardson for her time and cooperation in organizing and preparing the manuscript, I wish to extend thanks.

It is only fitting to thank the many friends who have supported me in my work. So to Joe and Marty McIlvaine, Ruth Hammock, Janis Fetter and many others I say thank you. I wish to also thank my employer, Little Friends Day Care Center for their cooperation in allowing me many days off to complete necessary details.

And finally I want to thank my family for their never ending faith and encouragement. They were always there and prepared to do anything I asked.
To the two men in my life,
my father, Donald Frantz Sr.
and my husband, Ron
I dedicate this thesis
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIST OF TABLES</td>
<td>vi</td>
</tr>
<tr>
<td>INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Binaural Beat</td>
<td>3</td>
</tr>
<tr>
<td>Aphasia</td>
<td>6</td>
</tr>
<tr>
<td>Cerebral Dominance</td>
<td>9</td>
</tr>
<tr>
<td>STATEMENT OF THE PROBLEM</td>
<td>13</td>
</tr>
<tr>
<td>METHODOLOGY</td>
<td>14</td>
</tr>
<tr>
<td>Test Site</td>
<td>14</td>
</tr>
<tr>
<td>Subjects</td>
<td>14</td>
</tr>
<tr>
<td>Instrumentation</td>
<td>14</td>
</tr>
<tr>
<td>Room</td>
<td>14</td>
</tr>
<tr>
<td>Tone Presentation</td>
<td>14</td>
</tr>
<tr>
<td>Stimulus Material</td>
<td>15</td>
</tr>
<tr>
<td>Procedure</td>
<td>15</td>
</tr>
<tr>
<td>Data Analysis</td>
<td>16</td>
</tr>
<tr>
<td>RESULTS</td>
<td>17</td>
</tr>
<tr>
<td>DISCUSSION</td>
<td>20</td>
</tr>
<tr>
<td>SUMMARY AND CONCLUSION</td>
<td>22</td>
</tr>
<tr>
<td>APPENDIX A. The Ascending (Afferent) Neuron Chain</td>
<td>24</td>
</tr>
<tr>
<td>APPENDIX B. The Descending (Efferent) Neuron Chain</td>
<td>26</td>
</tr>
<tr>
<td>APPENDIX C. Brief Case Histories for CVA Subjects</td>
<td>27</td>
</tr>
<tr>
<td>APPENDIX D. Schematic Block Diagram of Experimental Apparatus</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>Employed for Binaural Beat Stimulus Presentation</td>
</tr>
<tr>
<td>APPENDIX E. Graphic Representation of the Binaural Beat Phenomenon</td>
<td>31</td>
</tr>
<tr>
<td></td>
<td>vs. Pure Tone</td>
</tr>
<tr>
<td>LIST OF REFERENCES</td>
<td>32</td>
</tr>
</tbody>
</table>
LIST OF TABLES

Table | Page
---|---
1 | 19

Table of Chi Square of CVA and Normal Subjects Perception of Binaural Beats
INTRODUCTION AND RATIONALE

Zemlin (1968) describes the auditory pathway as a series of way-stations which carry impulses away from the cochlea (afferent pathway) toward the auditory cortex as well as away from the cortex (efferent pathway) toward the cochlea. The fibers leading from the hair cells of the cochlea collect at the spiral ganglion and emerge from the temporal bone through the internal auditory meatus. These fibers are joined by the fibers of the vestibular branch of the VIIIth and the VIIth nerve before emerging from the temporal bone at the medulla area of the brain stem. The cochlear neurons then proceed to the ventral and dorsal cochlear nuclei on the ipsilateral side of the medulla and the pons area of the brain stem. At this point there is no cross over of fibers to the other side of the brain. The fibers then proceed to the superior olivary complex. The superior olivary complex is located at the pons area of the brain stem. It appears that the superior olivary complex is the first area of cross over of fibers in the ascending system. It is also felt that this area is the first area to receive a major number of fibers from both ears, thus it is the first area of binaural fusion (Stotler, 1953; Deatherage, 1966; Wernick, 1968; Moushegian, et al 1969). From the superior olivary complex the fibers proceed via one of the major ascending tracts to the lateral lemniscus and to the nuclei known as the inferior colliculi, where cross over of fibers is
also experienced. The inferior colliculi are the second area of primary binaural fusion, as well as being the first area of visual and auditory fusion (Minkler, 1972). From here the neurons proceed to the medial geniculate bodies in the thalmic region of the brain stem. This area is the primary receptive area for localization of high frequency sounds (Rose, et al 1966). From these bodies auditory radiations spread through the limbic system then to the cortex of the cerebrum, specifically to the temporal lobe (Luria, 1965). A diagram of the afferent pathways of the auditory nerve is illustrated in Appendix A.

Minkler (1972), describes the major descending tract of the auditory pathway as the olivocochlear bundle, or Rasmussen's bundle. This efferent tract originates in the area of the superior olivary complex. Projections form a stria just below the IVth ventrical and these neurons cross the brain stem to the organ of Corti of the opposite ear. The total function of the olivocochlear bundle is still not known, but it is believed to exert a primary inhibitory effect upon the hair cells of the cochlea (Newby, 1972). Further subserving the main ascending pathways is the reticular formation running from the upper spinal cord to the temporal cortex. This system along with the limbic system (particularly the hippocampus and amygdala) work in a "push-pull", facilitation-inhibition fashion to filter, gate, and transmit auditory stimuli to the prime cortical areas for final interpretation and long term memory storage. The
limbic system exerts affect or emotionality upon incoming stimuli and is a prime area for short term memory storage (Smythies, 1970; Minkler, 1972; and Barr, 1976). Appendix B illustrates the efferent pathways of the auditory nerve.

**Binaural Beat**

Binaural beats are the appearance of subjective fluctuations in the loudness of two dichotically presented tones of constant amplitude differing only in frequency (Perrott and Nelson, 1969). Dichotic presentation is the use of auditory stimuli presented to both ears simultaneously (Kimura, 1967).

Since 1945 there has been an increase in the study of binaural interaction, both in localization of external sounds and in lateralization of earphone-delivered dichotic stimuli (Deatherage, 1966). In 1930 Von Bekesy described a model of binaural interaction which perhaps describes the phenomena in simplest terms. Briefly, the model explained that it is assumed that within the brain there exists a nucleus comprised of many neurons. In theory each neuron is innervated by fibers arising from each ear. As a signal reaches the neuron from, say, the left ear, the neuron is "tuned" Left; another neuron receiving a signal from the right ear would be tuned Right. Since each neuron is innervated by fibers from both ears, it is assumed that it is "tuned" according to the first signal to arrive. The fibers enter the nucleus from opposite sides and the signals
traverse the nucleus in opposite directions in some short time span. As the signals progress through the nucleus they "tune" the neurons along their path until the two events collide and extinguish each other. The brain then counts the number of left tuned and right tuned. If the numbers are equal a center position of binaural image is perceived. If one side is in excess of the other, the image perceived is on that side (van Bergeijk, 1962). It is apparent then, if the model is to be accepted that several areas of the brain are related to the binaural fusion function.

As stated previously, along the auditory pathway, the first area to receive innervation from both ears is the superior olivary complex, (Stotler, 1953; Moushegian, Rupert and Whitcomb, 1964; Deatherage, 1966; Wernick and Starr, 1968). This complex can be divided into five major subdivisions, with the accessory segment (the medial geniculate body) being of primary concern for binaural interaction (Moushegian, Rupert and Whitcomb, 1964). Moushegian, et al (1964) report that "Cells located in this nuclear mass appear to receive bilateral innervation and electrophysiological investigations of their properties have shown them, in fact, to be delicately tuned to binaural stimulation". Deatherage (1966) reports that great emphasis has been placed on the structures from the cochlea to the superior olive along the auditory pathway and concludes that "This prodigious research activity attests to the importance attributed to this neural region, particularly as it concerns binaural interaction".
Investigations of binaural interaction have been reported by several researchers. Moushegian, et al (1964) report using 20 cats in an investigation to show how single units of the accessory nucleus respond to monaural and binaural clicks. Their study concluded that some units of the accessory nucleus are sensitive to the binaural clicks and showed their sensitivity through inhibitory and excitatory processes. In a similar experiment conducted by Wernick and Starr (1968) the investigators again used cats and found the binaural interaction to take place in the accessory nucleus and in the superior olivary complex, thus supporting the findings of Moushegian, et al (1964). In 1969 Perrot and Nelson reported work done with binaural interaction and human subjects. The study indicated that the probability of beat detection was greatest at 500 Hz and decreased as the frequency increased up to 1500 Hz. This result would be expected if one accepts the view that binaural fusion takes place at the superior olivary complex, which is sensitive to low frequency sounds (Rose, 1966). Tobias (1963) employed 500 Hz and 503 Hz as the stimulus frequencies based upon his previous research which indicated these frequencies to yield the most discernable binaural beats. He found that in the presentation of absolute stimuli, subjects felt confident only when the intensity levels in the two ears were matched. He also reported finding that a strong beat was reported by subjects when the sound image was centered in the head and only when the sound was centered were they sure they were
hearing the beat. Only one female subject in this pilot could not hear the beat, regardless of the amount of training induced. Tobias lowered the frequencies for the subject several times and only when the frequency was dropped to 350 Hz could she hear the beats. Tobias has concluded from this that "preliminary data indicate a reliable sex difference in maximum frequency at which binaural beats are heard" (Tobias, 1963). Oster (1973) has also noted that women do not hear the beats at the same level as do males. His study indicates that the level of estrogen in the blood may influence the ability to hear binaural beats. If this is true, then typically the menstrual cycle would effect the ability to perceive the beats. During the menstrual cycle estrogen levels are low, while the level of monoamine oxidase (a central nervous system neuroinhibitor) is elevated. Further the key central nervous system neurotransmitters, norepinephrine, acetylcholine and serotonin levels are low, thus there is no binaural fusion (Rothballer, 1959 and Klaiber, et al, 1974).

Aphasia

Aphasia is a general term used for disorders of language resulting from brain injury or trauma. The major cause of aphasia is cerebral vascular accident (CVA) or what laymen term "stroke" (Schuell, 1974). Brain damage resulting from a CVA is believed to occur due to a change in the brain cells resulting from a
depletion of oxygen supply (Luria, 1965). He further points out that this supply can be disrupted in any of three ways:

1) a blood clot in a cerebral artery;
2) a ruptured artery and
3) a compression of the artery

These are the four forms of CVA which may deplete the oxygen supply to the brain possibly causing aphasia. Thrombosis, a blood clot which may form inside a cerebral artery or cerebral thrombosis which is a stationary clot inside an artery which obstructs the cerebral supply is one such form. A second type of CVA is the embolism. As in the case of thrombosis, the embolism is also found inside an artery; however, the embolism is a free flowing embolus (clot) which may at some point become caught or wedged in one of the cerebral arteries thereby restricting the oxygen supply to the cortex. Hemorrhage, the third form of CVA is the result of a diseased artery bursting causing surrounding brain tissues to be flooded with blood. Hemorrhaging may result from a bursting aneurysm (an abnormal pouch-like structure filled with blood which balloons out from the arterial wall), or from a blow to the head.

A fourth form of CVA, compression of a cerebral artery, is a result of pressure exerted on brain tissue or cerebral arteries. Brain tumors may apply pressure causing compression and the depletion of oxygen to the surrounding cells (Luria, 1965). CVAs are the chief cause of aphasia, but aphasia may also be caused by tumors, infectious
diseases and degenerative pathologies such as multiple sclerosis (Eisenson, 1973).

Aphasia has often been described as a multi-modality disorder encompassing disorders of expressive and/or receptive behavior (Van Riper, 1972). Aphasics experience difficulty in formulating, comprehending and expressing meanings with their basic problem lying in symbolic behavior. Not only do aphasics have difficulty in speaking, but they may also experience difficulties in reading, writing, gesturing and understanding the speech of others (Van Riper, 1972). Schuell (1974) has concluded that there is some degree of impairment of the auditory process in all aphasics. In some patients, although no hearing loss can be identified by audiometric examination, there is defective auditory perception, which prevents the aphasic from perceiving words; or if he does perceive words, he does so in such a distorted form as to make their recognition impossible. Schuell (1953) formulated the idea that a basic problem of the aphasic is one of "reauditorization" or the "ability to retain and evoke auditory patterns of language". Of 99 patients who recovered functional speech after having aphasia, only four made errors identifying common objects named by the examiner, although all made errors following oral and written directions (Schuell, 1974).

Aphasics show an impairment in the ability to understand spoken language. This impairment is characterized by reduced comprehension.
of spoken words, reduction of auditory retention span and sometimes
difficulty in discriminating between similar auditory patterns
(Schuell, 1974). Liles and Brookshire (1975) report that aphasics
may require more time than non-aphasics to process auditory stimuli.
They further state that this increased time requirement is not
limited to verbal auditory stimuli but applies to verbal and nonverbal
materials. Brookshire (1975) reports that aphasics demonstrated a
deficit in auditory sequencing, reflecting a deficit in the ability
to perceive, recognize or process such sequences. They did not,
however, reflect deficits in the ability to organize and carry out
sequences or responses necessary to report these sequences. It is
feasible then, for one questioning the relationship between brain
injury and the ability to perceive and report auditory stimuli. It
would seem necessary to delineate the role hemispheric dominance
plays with functions of auditory perception.

Cerebral Dominance and Audition

Cerebral dominance is defined as "the processing and control of
functions by a localized area within one of the hemispheres of the
brain" which is clearly differentiated in man (Kimura, 1967).
Language functions have most often been attributed to left cerebral
dominance (Kimura, 1967). Tsunoda (1969) presents the viewpoint
that the right hemisphere is usually dominant for nonverbal behavior
and the left for verbal behavior. Meyer (1961) further supports
this contention and states that "The most consistent claims are that patients with the left hemisphere lesions (dominant side) are relatively poor at verbal tasks, while those with right-sided lesions ... are relatively poor at practical tasks ...". Among the population at large, aphasic pathologies are most frequently of the middle cerebral artery within the left hemisphere (Eisenson, 1973).

In an attempt to study cerebral dominance, dichotic listening tasks are being employed. Kimura (1961) found that signals such as words or digits presented dichotically were heard better by most subjects at the right ear-left hemisphere. She also found that the ear opposite the dominant hemisphere is the more efficient ear. It is apparent then, that in tasks involving dichotic listening the influence of contralateral and ipsilateral stimulation to each hemisphere must be considered. Each ear has connection with the auditory reception area in each hemisphere and the pathways connecting the ears to their opposite hemispheres are apparently more effective than the ipsilateral pathways (Kimura, 1967). Rosenzweig (1951) stated that "at the auditory cortex of both cerebral hemispheres, each ear is represented by a population of cortical units". 

He further reports, "The population representing the contralateral ear is larger than the population representing the ipsilateral ear. The two populations overlap; that is some units belong to both populations". 
This uneven distribution of cortical units from both sides would appear to offer a partial explanation to the dominance of the contralateral over the ipsilateral signal.

Rosenzweig (1951) concludes that "Simultaneous stimulation of the ears usually results in partial summation; that is, the response is somewhat larger than the response of the contralateral ear, but it is not so large as the sum of the contralateral and the ipsilateral responses". It is apparent that although summation occurs, in dichotic listening both hemispheres receive stronger signals from the contralateral ear than from the ipsilateral ear (Kimura, 1967).

Numerous studies have been conducted to further delineate cerebral dominance. Papcun, et al (1974) used dichotic listening tasks in their investigation of subjects who were both primarily left handed and those who were primarily right handed. From the research they concluded "In experiments over the last 10 years, normal right-handed subjects have consistently shown a right ear superiority for the perception of competing dichotically presented verbal stimuli". It was assumed that in each case, if the subjects demonstrated a preference for the right hand that the left hemisphere was dominant and vice versa for the left-handed subjects. They also concluded that the left hemisphere is specialized to deal with "time related properties" as well as for the processing of sequential subparts that comprise a stimulus.
Bryden (1963) conducted a study based on the findings of Kimura's 1961 study which investigated the influence of bias as a result of instructions. Bryden instructed subjects to report material presented to the right ear first, for half of the sets of digits presented, and to report the digits presented to the left ear on the remaining half. His data supported Kimura's findings, in that he found that subjects had more errors when reporting the digits presented to the left ear first. He concluded that the difference of reporting the material presented to the two ears was due to a perceptual difference between the hemispheres and not due to instructional bias or the tendency to report material presented to the right ear first.

In a study involving subjects with hemispheric lesions, Gordon (1967) found that left lesioned subjects performed worse than right lesioned subjects or subjects without lesions on tasks in which they were to say which of two stimuli was the longer. This data supports the findings of Meyer (1961),
Numerous studies have been conducted involving aphasic subjects and dichotic listening tasks, but no studies have been reported concerning aphasic patients and their perception of the binaural beat phenomenon. It has been shown that binaural fusion occurs at the superior olivary complex and the inferior colliculi, indicating that if one hears the binaural beat these bodies are intact and functioning. It is not known however, if these bodies are intact and functioning in aphasics. It is the purpose of this study to determine if aphasics perceive the binaural beat phenomenon as do normals. Specifically to be questioned are the following:

1. Does binaural fusion occur in the aphasic at the levels stated above?
2. Is there a difference in the recognition of the binaural beat between normals and aphasics?
3. Is there a difference in responses of the severe aphasics vs. the mild aphasic?

This information could lead us to rethink our therapeutic methodology with aphasics and would indicate the degree and extent of the trauma caused by the CVA.
METHODOLOGY

Test Site

All testing was done at Florida Technological University's Communicative Disorders Clinic in Orlando, Florida.

Subjects

Six male individuals who were previously diagnosed by speech pathologists and neurologists as having aphasia were included in the experimental group for this study. All subjects were between the ages of 55 and 75 and had hearing no poorer than 25 dB (ISO-1964) at 500 Hz in both ears. (Brief case histories of all CVA subjects appear in Appendix C). Two of the six subjects were considered to have only mild receptive or expressive problems while the remaining four, though ambulatory experienced moderate to severe language problems. All subjects in the experimental group were right handed prior to the CVA and all had experienced a left cortical lesion within the last 15 months.

Six males with no history of CVA, matched in age and hearing abilities with the experimental group, comprised the control group. No individual presently taking testosterone or other hormones were included in this study.
Instrumentation

Room - A dual walled Industrial Acoustics Company Series 1200 Sound Treated Booth was used for all testing.

Tone Presentation - Two audio generators (RCA model WA44C) coupled to two audio amplifiers (RCA model 1421) were connected to the attenuators of a Clinical and Research dual channel audiometer (Grayson Stadler Model 1702-A). Calibration of the audio generators were monitored by a frequency counter-timer (Monsanto Model 100B). The waveform composition of the stimulus tones was monitored by an oscilloscope (Telequipment Model D54). The stimulus was presented through earphones (Telephonics TDH-50). (See diagram Appendix C).

Stimulus Material

A tone of 534 Hz, 40dB sensation level (SL) was presented through the right earphone, and a tone of 540 Hz, 40 dB SL was presented through the left earphone. A chart with a graphic representation of a waveform (beat and a straight line (no beat) was used for nonverbal description of the stimulus tones (see Appendix D).

Procedure

All individuals were given a pure tone air conduction threshold test at 1000 Hz, 2000 Hz, 4000 Hz, 8000 Hz, 500 Hz and 250 Hz. All individuals were required to have hearing no poorer than 25dB (ISO-1964) at 500 Hz in both ears in order to be considered eligible for subject inclusion.

The right earphone was placed against the right ear and the subject was asked, "Do you hear the tone?" The same procedure then followed
for the left ear. Both earphones were then placed on the subject and he was asked to listen carefully. He was allowed to listen to the stimuli for approximately one minute. He was then asked, "What do you hear?" He was then asked to point to the chart and tell the examiner which response looked most like what he heard. He was then asked to tell where on his head he seemed to hear the beats. All responses, verbal and gestural were transcribed by the examiner.

Data Analysis

A $X^2$ (chi square) statistical analysis was run to determine the significance of data found. The Yates Correction was employed due to the number of subjects involved to further insure reliability of results.
RESULTS

Twelve subjects were tested in the study, six comprised the experimental group and six the control group. The experimental group was composed of those subjects who had experienced a CVA and the control group were non-CVA individuals. Within the experimental group a further subdivision was made based on the severity of the CVA and the degree of observable damage due to the CVA. Two of the six subjects (group 1a) in the experimental group were classified as mild aphasics with no hemiperesis and with only a slight loss of gripping ability at the right side and slight numbness in their right legs. The remaining four (group 1b) exhibited right hemiperesis as well as expressive and receptive function impairment as a result of the CVA. These four were considered to have experienced a more serious CVA in comparison with the subjects in group 1a. All subjects were ambulatory and did not require the aid of a cane or walker.

The subjects in group 1b of the experimental group could not perceive the beats. These four were found to have experienced the more severe CVA and expressed their perception of the beats through verbal (limited to yes or no) and gestural means. The two subjects
comprising group 1b did perceive the beat phenomenon and expressed this through verbal and gestural means. All gestural responses were made in conjunction with the chart shown in Appendix D. All six subjects in the control group perceived the beats and their responses were recorded in the same way as for the experimental groups.

Each response was recorded as either a yes, perceived the beats, or no, could not perceive the beats. These yes and no values were then compared as the number of yes and no responses for the experimental group vs. the number of yes and no responses for the control group. The values were compared according to the chi square statistical analysis and found to be significant ($X^2 = 5.24$, d.f. = 1, $p < .05$).
Table 1

Table of Chi Square of CVA and Normal Subjects Perception of Binaural Beats

<table>
<thead>
<tr>
<th>Group</th>
<th>Yes</th>
<th>No</th>
<th>$x^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>1a</td>
<td>2.5</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>1b</td>
<td>0</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>5.5</td>
<td>0</td>
<td>5.24*</td>
</tr>
</tbody>
</table>

* with df = 1 $p < .05$
DISCUSSION

Results of the data found indicated that for subjects in group 1b (moderate – severe aphasia) either binaural fusion was occurring or that some form of auditory interference was occurring at the level of the superior olivary complex or the inferior colliculi. One possible explanation may be a phenomenon we shall call the 'Depression Theory'. The major contention of this theory is that pressure results from hemorrhaging occurring during the CVA. This hemorrhaging causes pressure within the brain and depresses the tissue structure of the midbrain which in turn interferes with brain stem functioning. Since it is known that binaural fusion is a function of two main brain stem areas (superior olivary complex and the inferior colliculi), it would follow that if these areas were interfered with by the lesion in some way, the binaural beat phenomenon could not occur, or it would at the very least become depressed. This would explain why only two of the CVA subjects could perceive the beats. The more severe the CVA the possibility of greater damage and pressure is present, thereby increasing the probability of the cortex exerting pressure to the stem, if the 'depression theory' is to be accepted.
A second theory which may explain the reason why only two of the four CVA subjects perceived the beats we will call the "Temporal Time Sequencing Theory". This theory asserts that it takes two hemispheres to perceive the beats. With the onset of the CVA, one of the hemispheres experiences damage thereby limiting in some fashion the temporal time sequencing occurring at the corpus callosum between the hemispheres. If the function of one of the hemispheres is impaired, the binaural beat phenomenon would not be heard. In the case of a less severe CVA the extent of damage would be limited and the possibility of neuronal transmission interference is decreased.

It is important to note that it appears that the amount of damage incurred by the CVA and the retained language function of the CVA patient are keys to determining whether or not the beats will be perceived. The subjects in group 1a had experienced the less severe CVA and in both cases, expressive abilities were greater than in the case of the four subjects in group 1b. These findings would tend to further support the two theories presented.
A review of relevant literature reveals that numerous studies have been conducted involving dichotic listening tasks with various populations, but no studies have been reported involving the binaural beat phenomenon and aphasic patients. It was decided to investigate how or if aphasic patients binaurally fuse two slightly differing frequencies of constant amplitude.

The stimuli was presented to groups of subjects, one control group consisting of six males and one experimental group divided into two subgroups: 1a - two males who experienced a mild CVA within the past 15 months, and group 1b - four males experiencing a severe CVA within the past 15 months.

Two tones of different frequency levels, but equal in intensity were presented dichotically to the subjects at 40dB SL. All subjects had normal hearing at 500 Hz (0-25dB).

A 2 x 2 design resulted from the study and was statistically compared with a Chi square and results were found to be significant (p < .05).

From this data one may assume that in patients who have experienced a severe CVA binaural fusion does not occur at the
level of the superior olivary complex or the inferior colliculi. It may also be assumed that some form of auditory interference has occurred as a result of the CVA. In cases where the CVA is less severe, the superior olivary complex and the inferior colliculi remain intact and function normally.

These results have application in current aphasic therapeutic techniques. Aphasia therapy currently does not differentiate the ear to which stimuli is presented. From the results of this study one may find that stimuli presented to the left ear-right hemisphere may be more effective as these areas could in some fashion further subserve the language function of the usually dominant left hemisphere.

This study indicates a great need for further research involving binaural beats. A possible study would involve the investigation of the levels of monoamine oxidase before and after CVA and the effects of the MAO level on the perception of the binaural beats. It should also be noted that only the ascending auditory tracts were of consideration in this study and that perhaps there is some interference with the descending tract resulting in an inability to perceive binaural beats.
APPENDIX A

The Ascending (Afferent) Neuron Chains
APPENDIX B

The Descending (Efferent) Neuron Chains

Diagram showing various structures including Auditory Cortex, Reticular Formation, Medial Geniculate Body, Inferior Colliculi, Lateral Lemniscus, Dorsal Nucleus, Dorsal and Ventral Cochlear Nuclei, and Cochlea.
APPENDIX C

Brief Case Histories for CVA Subjects


Severity: Moderate Date of CVA: Jan. 7, 1975 Release date: Jan. 17, 1975

CVA (2) 55 years. Left cerebral aneurysm. Little language involvement. No hemiplegia but some loss of strength in extremities. Occupation prior to CVA: Sanitation worker.

Severity: Moderate Date of CVA: Feb. 17, 1975 Release date: March 6, 1975

CVA (3) 57 years. Left middle cerebral aneurysm. Right side affected especially the right hand. Severe expressive language involvement. Occupation prior to CVA: Farmer.

Severity: Severe Date of CVA: Nov. 19, 1974 Release date: Dec. 12, 1974

CVA (4) 63 years. Left hemispheric hemorrhaging from severe blow to head. Numbness/reduced sensitivity in right extremities. Moderate expressive language involvement. Occupation prior to CVA: Plumber.

Severity: Severe Date of CVA: Dec. 12, 1974 Release date: Dec. 27, 1974


Severity: Moderate/Severe Date of CVA: Mar. 13, 1975 Release date: Mar. 23, 1975

Severity: Severe Date of CVA: Jan. 20, 1975
Release date: Feb. 10, 1975
APPENDIX D

Schematic Block Diagram of Experimental Apparatus Employed for Binaural Beat Stimulus Presentation
Audio Oscillator (1) 
Audio Oscillator (2) 

Hz Counter (1) 
Hz Counter (2) 

Attenuators in Dual Channel Clinical and Research Audiometer 

Monitor Oscilloscope 

Sound Treated Booth 

DH-50 Earphones
APPENDIX E

Graphic Representation of the Binaural Beat Phenomenon Vs. Pure Tone

No beat perceived

Beat Pattern Perceived
LIST OF REFERENCES


