## INFORMATION TO USERS

This dissertation was produced from a microfilm copy of the original document. While the most advanced technological means to photograph and reproduce this document have been used, the quality is heavily dependent upon the quality of the original submitted.

The following explanation of techniques is provided to help you understand markings or patterns which may appear on this reproduction.

1. The sign or "target" for pages apparently lacking from the document photographed is "Missing Page(s)". If it was possible to obtain the missing page(s) or section, they are spliced into the film along with adjacent pages. This may have necessitated cutting thru an image and duplicating adjacent pages to insure you complete continuity.

2. When an image on the film is obliterated with a large round black mark, it is an indication that the photographer suspected that the copy may have moved during exposure and thus cause a blurred image. You will find a good image of the page in the adjacent frame.

3. When a map, drawing or chart, etc., was part of the material being photographed the photographer followed a definite method in "sectioning" the material. It is customary to begin photoing at the upper left hand corner of a large sheet and to continue photoing from left to right in equal sections with a small overlap. If necessary, sectioning is continued again — beginning below the first row and continuing on until complete.

4. The majority of users indicate that the textual content is of greatest value, however, a somewhat higher quality reproduction could be made from "photographs" if essential to the understanding of the dissertation. Silver prints of "photographs" may be ordered at additional charge by writing the Order Department, giving the catalog number, title, author and specific pages you wish reproduced.

**University Microfilms**

300 North Zeeb Road
Ann Arbor, Michigan 48106

A Xerox Education Company

# Binaural Analysis as a Function of Physiological Masking: The Cardiac Cycle

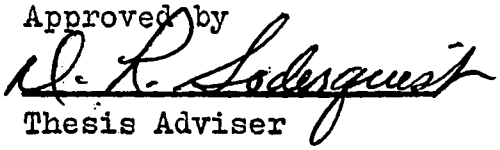
by

John W. Lindsey

A Thesis Submitted to
the Faculty of the Graduate School at
The University of North Carolina at Greensboro
in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Greensboro
April, 1972

Approved by



Thesis Adviser

APPROVAL SHEET

This dissertation has been approved by the following committee of the Faculty of the Graduate School at The University of North Carolina at Greensboro.

Dissertation Adviser A. R. Lodegaard

Oral Examination
Committee Members Ernest A. Lunsdaine Jr.

M. Russell Harter

Robert A. Eaves

Paul E. Lutz

March 27, 1972
Date of Examination

ACKNOWLEDGMENTS

The author wishes to thank the following Committee members: Dr. Robert Eason, Dr. Paul Lutz, and, the chairman, Dr. Ernest Lumsden. Gratitude for his invaluable assistance in making the blood volume measurements is extended to another Committee member, Dr. M. Russell Harter.

Special thanks belongs to Dr. David R. Soderquist, the Dissertation Adviser, for his critical assistance in the preparation of this dissertation. His conscientious involvement in my progress throughout graduate school deserves my sincerest thanks and appreciation.

My dedicated subjects: Mr. Richard Bowman, Mr. Ralph Donaldson, and Mr. Ronald Hughes are to be commended for their service. Finally, thanks are due to my wife, Carole, for her fiscal and emotional support.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

# INTRODUCTION

The psychophysical study of sensory systems has an extensive history. The sensitivity of sensory systems has often been quantified and evaluated in terms of various concepts of threshold. Generally, the threshold is viewed as a point on a continuum of stimulus intensity (or corresponding neural activity) below which detection of the stimulus does not occur and above which detection is possible. Furthermore, the statistical nature of the threshold concept has received renewed interest in the distinction between sensory sensitivity and decision mechanisms (Corso, 1963; Green & Swets, 1966; Swets, 1964).

Determination of the absolute sensitivity of sensory systems has occasionally revealed various sources of "noise" within, or internal to, the organism due to numerous biological processes. "Internal Noise" (IN), then, can be regarded as extraneous information which possesses characteristics similar to the relevant (i.e., "the signal") stimulus (Molyneux, 1963). Some authors have suggested that IN influences the rate at which information can be transmitted by the sensory channel (Stewart, 1965; Swets, Shipley, McKey, & Green, 1959). More commonly, however, IN has been viewed as limiting the amount of information which can be extracted from a sensory transducer. This implies that the functioning of a

sensory system must be evaluated in terms of the interaction, within the sensory channel, between neural activity due to the stimulus and its own background activity. In other words, detection based on input to a sensory tranducer is made difficult by confusion between a non-fluctuating, stimulus-specific, neural activity (e.g., a sinusoidal stimulus) and a fluctuating background of IN (Eijkman & Vendrik, 1965, Thijssen & Vendrik, 1968). In an organism's natural environment the disruption of the CNS due to IN is typically restricted by mechanisms of redundancy, attention, habituation, and inhibition which focus the system on only the most significant signals (Gasteiger & Brust-Carmona, 1964).

Nonetheless, it is clear that detectability measures of absolute sensitivity can be directly influenced by IN. Thus, the threshold of a sensory system must be regarded as "differential" rather than "absolute" (Diercks & Jeffress, 1962; Eijkman, Thijssen, & Vendrik, 1966; Ward, 1963). In other words, the precision of experimental measurements of the threshold may be decreased due to the inability of the S to distinguish between IN and neural activity due to the signal. There would appear to be two sources for this imprecision. First, masking may result when the signal and IN activate overlapping neural regions leading to increased "miss" responses. Second, the likelihood of a signal response may be increased when signal and IN share similar stimulus properties. Hence, the number of false-alarm (F/A)

responses may be increased by IN, dependent, of course, on an associated shift in the S's criterion (Eijkman & Vendrik, 1963; Moulin, 1969).

Before proceeding to a brief review of studies which have invoked various IN hypotheses it is well to be cognizant of the following suggestions of Green (1964). He notes that unless the specific effects of IN can be directly evaluated, the assumption of IN simply restates in an ad hoc fashion, but does not explain, the discrepanices in threshold actually observed. In order for the concept of IN to be useful, Green (1964) suggests, the researcher should: (1) be able to state exactly what the noise is; (2) specify in what way it interacts with the detection process; and (3) evaluate specifically what effect it will have on performance.

The present study concerns the effects of one aspect of IN--physiological masking (the heartbeat) on auditory signal detectability. It is probably clear at this point in the discussion that the concept of IN is quite broad. In an attempt to clarify the IN concept with respect to the auditory modality, Soderquist and Lindsey (1972) made the following distinctions:

> Neural Noise: Noise in the nervous system that results from spontaneous random neural firings (Davis, 1951; Fitzhugh, 1957).
>
> Physiological Noise: Any noise directly caused by the normal (or abnormal) activity of physiological systems within an organism. Such activities as peristalsis, breathing, muscular-joint movements or

tensions, and heartbeats are all causes of audible sounds which originate in living organisms.

Aural Noise: Low-frequency noise often attributed to mechanical vibrations and present in the external auditory meatus and middle ear. The noise, typically known as the "ocean's roar" or "seashell effect," is augmented by tight-fitting earphones and includes the mechanical vibrations of pulse actions and body movement.

Internal Noise: A generic term which may be applied to any of the three other types of noise. That is, internal noise may be aural, physiological, neural, or any combination of the three.

These distinctions will be maintained throughout the paper.

Despite the above delimitation it will become clear in the following survey of pertinent research that these distinctions are somewhat arbitrary and different types of "noise" often co-exist within an experimental situation. Directly related to the ambiguity of these terms is the fact that IN has generally been inferred, ad hoc, from changes in the dependent variable, contrary to Green's (1964) suggestions.

## Psychophysical Studies and Internal Noise

Several relatively independent areas of psychophysical research have obtained data which have indirectly supported the inference that low-frequency aural and physiological noise interfere with auditory performance.

First, the pioneering research of Sivian and White (1933) and Stevens and Davis (1938) revealed that when the

ear is sealed by an earphone mounted in a conventional cushion, aural noise is generated in the small volume under the earphone. A later study by Brogden and Miller (1947) asked Ss to match the quality of this aural noise by manipulating band-pass filters. These latter authors concluded that most of the energy was between 60-100 Hz and was negligible above 180 Hz. Recently, Moulin (1969) has obtained data bearing on this question using a yes-no procedure. It was found that an otosclerotic group (unhampered by their own IN due to the otosclerotic lesion) had steeper slopes at 80 Hz for their psychometric functions and made fewer F/As than normal hearing Ss and a control group having normal hearing but plugged external canals. Similarly, these group differences disappeared in a 50 dB SL masked condition, suggesting that the external masker was overriding the IN. In keeping with previous data, it was argued that the IN was in the lower portion of the frequency spectrum because F/As decreased at higher (125 and 1000 Hz) frequencies for the normal groups. Finally, the middle ear (rather than the external canal) was suggested as the origin of the masking noise since the two control groups were not significantly different.

Many authors have been interested in the masking properties of aural noise since it was first suggested by Sivian and White (1933). The latter found that minimum audible pressure and minimum audible field measurements for a 100 Hz tone depended on how the signal pressures were presented.

Since the earphone threshold pressures for the same S were higher than the free-field threshold pressures, this phenomenon came to be regarded as the "missing 6 dB" (Munson & Wiener, 1952). This pressure/free-field difference is clearly frequency-dependent. That is, it varies from about 16 dB at 60 Hz to 5 dB at 240 Hz, and finally disappears at 1000 Hz (Anderson & Whittle, 1971; Munson & Wiener, 1952). Likewise, the difference has been shown to disappear (for a 50 Hz tone) in the presence of a noise masker at 70 dB SPL and beyond (Anderson & Whittle, 1971). Rudmose (1962) has shown that the "missing 6 dB" effect can also be eliminated (at threshold levels) when an earmold system is used instead of the standard earphone system. His demonstration implies that masking by aural noise is dependent on the enclosed volume under the earphone as well as the pressure and physical contact of the headset.

This implication was studied further in a series of experiments by Anderson and Whittle (1971). They found that increasing the effective earphone volume by using circumaural rather than supra-aural earphones lowered both aural noise levels and thresholds (between 50 and 500 Hz). Subsequent study controlled the extent to which the aural noise was allowed to "leak out" from under the earcap. The amount of "leak" increased as the size of the hole in the earcap was increased from 1.1 to 1.65 mm. There was a corresponding drop of 15.5 dB in aural noise for the 50 Hz band, and a

drop of 8.5 dB in the 50 Hz threshold. The amount of "leak" is, of course, typically an unspecified source of inter-S variability in threshold measurements owing to differences in earphone fit. In this regard it is interesting to note that an experimental mounting of the TDH-39 has been developed (Villichur, 1970) which keeps the earphone-ear system constant across fittings and also can reduce low-frequency masking by aural noise (Shaw & Piercy, 1962a; 1962b).

Second, absolute thresholds at low frequencies have been shown to display considerable improvement with practice (4-6 dB at 150 Hz) while non-significant changes typically occur at higher frequencies (Corso & Cohen, 1958; Loeb & Dickson, 1961; Zwislocki, Maire, Feldman, & Rubin, 1958). Similarly, these practice effects are absent for masked thresholds (50 dB SPL) suggesting that the low-frequency practice effect at absolute threshold involves learning a discrimination between the low-frequency signal and the low-frequency IN (Loeb & Dickson, 1961).

Third, data from binaural analysis (cf. Green & Henning, 1969; Lindsey, 1970) for masking-level differences (MLDs) at low frequencies have often involved the postulation of an "internal noise hypothesis" (Diercks & Jeffress, 1962; Dolan, 1968; Dolan & Robinson, 1967; McFadden, 1968; Soderquist & Lindsey, 1971a; Wilbanks & Cornelius, 1969). In general, it has been argued that the absolute threshold is actually a masked threshold due to the presence of IN. Further, the

data have suggested that the relative contribution of IN seems to be inversely related to both the intensity of the external masker (noise via earphones) and the frequency of the signal to be detected. This suggestion was tested by Watson, Franks, and Hood (1967). They estimated their S's critical bandwidth by assuming that the critical ratio equalled 1.0 and then measured the amount of signal energy necessary for S to achieve a d'= 1.00. Signal energy was measured in the presence of a moderate intensity, wide-band masker. Assuming that the critical bandwidth did not change at absolute threshold, signal energy was again manipulated to yield d'= 1.00. Results indicated that the level of IN was relatively constant above 500 Hz, but increased as frequency decreased. The level of IN was estimated to be about 19 dB greater at 125 than 250 Hz.

## Possible Origins and Measurement of Internal Noise

Before reviewing several studies which have attempted direct measurements of IN on a relatively molar level, it is interesting to note an observation made by Wever and Lawrence (1954) concerning the recording of cochlear potentials in anesthesized animals. They reported that random variations of mechanical parts (especially of the tympanic muscles), the presence of metabolic and chemical activities in the cells, and the flow of blood (and other bodily activities) produced "noise" which imposed a lower limit on the satisfactory

recording of cochlear potentials. The limit was extended tenfold when recordings were made from dead animals.

Although numerous psychophysical studies have inferred the presence of low-frequency "noise" from various sources (such as the above), direct measurement of the intensity/frequency characteristics of the "noise" has not received as much attention. The available studies have typically measured aural noise by using a condensor microphone sealed to the head and fitted with a probe tube attachment. Shaw and Piercy (1962a; 1962b) found that the level per 1/3 oct band was approximately 70 dB SPL at 16 Hz and fell steadily to 34 dB SPL at 125 Hz and 12 dB SPL at 250 Hz. Anderson and Whittle (1971) have recently replicated these findings but noted substantial inter-S variability, presumably due to variations in earphone fit, and hence, different amounts of "leak."

Shaw and Piercy (1962a; 1962b) suggested that some of the aural noise was generated by the circulatory system since the overall noise level in the cavity enclosing the ear (volume= 60 $cm^3$) increased and decreased by a few decibels at the heart-pulse frequency. Anderson and Whittle (1971) have also argued that blood flow is the crucial variable in the generation of noise in the external auditory canal and offered two possible hypotheses. First, since the pinna expands and contracts due to changes in local blood pressure these changes could influence corresponding

changes in the volume under the coupled earphone. However, this "pinna expansion hypothesis" was rejected since physiological noise was found to be the same under either an absorbent or hard earcap. In other words, absorptions of pulsations of the pinna with the "soft" earcap should, according to the hypothesis, have resulted in less noise. Second, the activity of the blood vessels in the wall of the meatus may influence the sound pressure by changing meatal volume in relation to ongoing blood pressure changes. Since the blood vessels are unable to expand outward due to the bone encasing the meatus, the hypothesis suggests that their inward expansion induces an increase in sound pressure by decreasing meatal volume. Support for this hypothesis was obtained when measurements of maximal blood pressure at the external carotid artery were found to correspond with periods of greatest meatal sound pressure. The "pinna expansion hypothesis" would have predicted the opposite, i.e., expansion of the pinna under increased blood pressure would lift the earphone, increasing the volume and decreasing the pressure. Thus, Anderson and Whittle (1971) concluded that the "meatal pulsation hypothesis" was a more likely explanation of changes in aural noise associated with the circulatory system.

Some additional data concerning the acoustic aspects of circulatory noise have been offered by Saito, Kobayashi, Yasuda, Inagaki, Nakamura, Tokumasa, Yaguchi, and Oose (1969).

They measured the frequency spectrum of intracardiac sounds with a transducer fixed at the tip of a double lumen intracardiac catheter. Heart sounds were measured at 6 positions of the right heart catherization of clinical Ss. The frequency spectrum of the intracardiac sound at the root of the pulmonary artery and outflow tract of the right ventricle indicated that intensity was about 50 dB at 50 Hz and went to about 10 dB at 1000 Hz. Peak intensity (55 dB) was at 100 Hz.

It has been recognized that muscle tension, particularly of the neck, is a source of IN (Brogden & Miller, 1947). Similarly, Piercy and Shaw (1963) studied the effects of rigid head motion and earphone inertia. Subjects had their heads driven by a pure-tone vibrator and were instructed to adjust the level of excitation for a loudness balance (with TDH earphones) when their ears were covered (supra-aural or MX-41/AR) or not. Subtraction of the values for these two conditions showed free-field physiological noise levels of 40, 15, 10, -10, and -10 dB at 40, 62.5, 125, 250, and 500 Hz, respectively. It appears, then, that studies measuring IN support the inference from psychophysical studies that IN is characterized by low frequencies and its intensity is inversely related to frequency.

## The Problem

### Immediate Background

It is apparent at this point that the notion that the absolute threshold is actually a masked threshold has a rather lengthy history of about 40 years in auditory research. An example of the phenomenology of physiological (cardiac) masking is well-expressed by Lawson (1948). He observed that: "when we are listening to a continuous source of sound of uniform intensity, an apparent dimunition in intensity will be observed for the duration of each "beat," and between beats the intensity will be greater and sensibly uniform [p. 782]." Soderquist and Lindsey (1972) studied this phenomenon in a quantitative and controlled fashion. They found that detection of a binaural, in-phase (S0), 100 Hz, 100 msec tone showed changes in detectability (d') that were related to the temporal delay in the signal onset following the S's own EKG R-wave (cf. Ganong, 1969; Guyton, 1971). Results indicated depressed sensitivity with signal delays of: 0.0, 0.3, and 0.7 sec following the R-wave, while maximal sensitivity occurred at 0.5 sec. These results were discussed in terms of the correlation between the drops in sensitivity and physiological masking produced by heart sounds. In other words, the heart sounds: "lub" (closing of mitral and tricuspid valves) and "dub" (closing of aortic and pulmonic valves) occur at 0.0 and 0.3 sec after the R-wave, the same points at which sensitivity dropped. The

possibility that the data could be explained by changes in blood volume (measured with a plethysmograph) at the earlobe was ruled out since neither maxima nor minima of the blood volume measures corresponded to highest or lowest d' sensitivity. Similarly, the fact that cochlear blood volume measures (Suga & Snow, 1969) seem essentially similar to the pattern measured at the earlobe was cited by Soderquist and Lindsey (1972) as further support for the "cardiac masking hypothesis."

Finally, there are data available suggesting that increasing the flow of blood to the brain (by inducing higher heart rates) has the effect of increasing (or failing to change) an auditory threshold when compared to a resting baseline (Boys & Curry, 1956; Saxon & Dahle, 1971). It may be argued that increasing the heart rate, which reduces the inter-R time, increases the number of heart sounds and hence eliminates sensitivity improvements due to "cardiac arousal" by increasing the amount of physiological masking. In this regard, Delfini & Campos (1971) tested an hypothesis that auditory detection of a binaural, S0, 1000 Hz tone (in a background of 53 dB SPL ventilator noise) would be poorer during the QRS and T phases of the EKG. This was predicted from evidence that the baroreceptor input to the nucleus tractus solitarius triggers maximal inhibition of cortical arousal during these phases. It was found, however, that there were no significant relationships between d', S's

criterion, and the EKG. Hence, they concluded that "cardiac arousal effects" are minute or nonexistent.

The Present Study: Design and Hypotheses

The present study is an extension of the Soderquist and Lindsey (1972) experiment and an attempt to further investigate the masking properties of cardiac sounds by using the EKG in a signal detection task. Data are lacking regarding the extent to which internal masking (in this case the sounds produced by the physiological activity of the heart) is similar to external masking. For instance, it is well-known that the threshold of a binaural signal (S0) can be decreased by shifting the interaural phase of the signal 180° (S$\pi$) and presenting it in a noise background (cf. Green & Henning, 1969; Hirsh, 1948a; Kikuti & Yosida, 1940; Lindsey, 1970). These effects are known as masking-level differences (MLDs) and the two typical binaural listening conditions are labelled N0S0 and N0S$\pi$ when the external noise is binaural, in-phase (N0). The MLD size, then, represents the threshold difference expressed in dB between these two listening conditions (or others) and depends on numerous parameters (frequency, masker intensity, interaural correlation, etc.).

The present study investigated the S0 and S$\pi$ listening conditions at "absolute threshold." Two major questions were of particular interest. First, can the presence of an

internal, physiological masker (cardiac sounds) influence SO and SΠ thresholds in a manner similar to that for external noise? Second, do the masking effects of cardiac sounds extend to frequencies higher than 100 Hz?

Two experiments involving the following variables were performed with repeated measures designs in an attempt to answer these questions.

Experiment I: (1) Signal Delay: 0.0 and 0.5 sec following the EKG R-wave

(2) Frequency: 100, 200, and 300 Hz

(3) Listening Condition: SO and SΠ

Experiment II: The same delay and listening conditions were used as in Experiment I but at 5000 Hz. This experiment was run essentially as a control. In other words, since the ear shows little phase sensitivity at 5000 Hz (Green & Henning, 1969) and physiological noise does not extend beyond 1000 Hz, any significant differences between signal delays would presumably implicate a variable other than physiological masking in the observed detection differences.

The rationale underlying the choice of the parameters listed above requires some elaboration. Since it was predicted that non-significant differences would be found in Experiment II, the following hypotheses pertain primarily to Experiment I.

First, the level of physiological noise was manipulated by selecting the signal delays at which Soderquist and

Lindsey (1972) found the most (0.0 sec) and least (0.5 sec) difficult detection. Thus, their results seemed to reflect masking and release from masking, respectively. This suggestion was examined in the present study by comparing performance in the SO and S$\pi$ conditions. In other words, the inferred presence of cardiac noise in the cochleas at 0.0 sec delay suggests that these conditions are actually noSO and noS$\pi$. (The abbreviation "no" rather than "NO" designates the masker as internal and positively correlated). Because a noise background is known to facilitate detection when interaural cues are available, it was expected in Experiment I that the detection advantages for S$\pi$ *re* SO would be largest when the signal was coincident (0.0 sec delay) with the R-wave of the EKG; that is, when the signal to be detected occurred simultaneously with the cardiac sound resulting from the closing of the mitral and tricuspid valves.

Second, four frequencies (100, 200, 300, and 5000 Hz) were studied to determine if the masking effects of the cardiac sounds extend to higher frequencies than the 100 Hz used by Soderquist and Lindsey (1972). The data from Saito, *et al.* (1969) suggest that the frequency spectrum for cardiac sounds extends to 1000 Hz. Hence, it was predicted that masking, i.e., depressed sensitivity at 0.0 sec signal delay (*re* 0.5 sec), would be found for 100, 200, and 300 Hz, but not at 5000 Hz. Likewise, it was expected that detection would be less difficult at 0.5 sec than 0.0 sec for all frequencies except 5000 Hz.

# METHOD

## Design

Two experiments were done. Experiment I involved a 2x2x3 repeated measures design to investigate three experimental parameters: (1) signal delay: 0.0 and 0.5 sec; (2) listening condition: SO and S$\pi$; (3) signal frequency: 100, 200, and 300 Hz. The twelve conditions yielded by all possible combinations of these parameters were "randomized" in blocks of 80 trials. The two restrictions imposed on randomization were the following. First, no two sequential blocks of trials were the same experimental condition. Second, no condition systematically occupied a given ordinal position across sessions. Hence, the dependent variable, signal detectability as expressed in d' units (cf. Elliott, 1964), was based on a mean of 400 trials or 5 blocks (replications). Experiment II used a 2x2 repeated measures design to study the same signal delay and listening conditions as Experiment I with a 5000 Hz signal. The same quasi-random procedure and dependent variable were used as in Experiment I. Experiment II followed completion of Experiment I.

## Subjects and Training

Three graduate assistants (males 22-26 yrs) with clinically normal hearing were listeners in both experiments. Training prior to Experiment I consisted of at least 4000

trials and included instructions on how to eliminate extraneous sources of self-induced noise (e.g., movements, breathing rate, earphone cords rubbing against objects, etc.). Additionally, approximately, 1000 training trials preceded Experiment II. During training (for both experiments) each S's sensitivity was determined for all conditions of the experiment. Those signal intensities (one for each frequency) were chosen at the conclusion of training which had yielded a d' of around 1.00 (76%) for the 0.0 sec, S$\pi$ conditions. All conditions of the experiment were then run at these determined intensities; i.e., the intensity for a given frequency was held constant and detection (d') was allowed to vary.

Experimental Sessions and Apparatus

A yes/no signal detection paradigm (cf. Green & Swets, 1966; Swets, 1964) was used to investigate binaural (S0 and S$\pi$) sensitivity in both experiments. Each S was connected to a Grass Model 7 polygraph and his heartrate monitored periodically with a Lehigh Valley 4 digit counter (384-04). Gold cup electrodes were placed approximately 8 in below either the right or left clavicle. The site chosen was the one which yielded relatively large R-waves. Dermal resistance was lowered with Redux electrode paste. The S was provided with a pair of calibrated earphones (TDH-49) mounted in MX-41/AR muffs. The observer was then seated in a sound-attenuated room before a panel of indicator lights and response keys. He was instructed to respond on each trial

indicating (by pressing one of two microswitches) his decision regarding the presence or absence of the signal. Responses (Hits and F/As) were automatically recorded on electromechanical counters. Feedback lights informed S after each trial whether a signal had been presented or not. An experimental session lasted approximately one hr and 15 min and contained 5 blocks of 80 trials, each preceded by about 20 warmup trials. A rest period of about 5 min followed each block. Subjects were run 5 days per wk for approximately 2.5 months. Figure 1 shows a schematic of the experimental apparatus.

The output of the Grass Model 7 polygraph (S's EKG R-wave) triggered a fixed interval timer (Lehigh Valley adjustable time base 351-05). Interfacing was accomplished by a Lehigh Valley dual Schmidt trigger (321-03). The fixed interval timer (Timer A) prevented a trial from being initiated every 0.8 sec, or with each R-wave. A trial was begun only by the first R-wave following the offset of Timer A. Trial onset (either a signal or no signal) automatically reset Timer A and began a programmed sequence controlled by a Lafayette 8-Bank Timer. The latter bank of timers determined the overall timing of the experimental intervals consisting of the following sequence: intertrial interval (0.9 sec); light for onset of observation interval (0.1 sec); observation interval (2.4 sec); light for offset of observation interval (0.1 sec); response interval (1.9 sec); feedback light (0.1 sec).

The signal a priori probability of 0.5 was determined by a Lehigh Valley probability gate (335-11). For trial blocks when a signal was presented at the 0.5 sec delay, the R-wave (output of Timer A) keyed the probability gate and triggered a Davis model D-501 time interval generator (Timer C). At the conclusion of the preset interval (0.5 sec), Timer C then triggered the signal generating circuit. Alternatively, the pulse from Timer A was used in the 0.0 sec signal delay condition to key the signal circuit directly (i.e., the Grason-Stadler 471-1 interval timer). When the signal was presented it always occurred in the middle of the observation interval. This was accomplished by including an additional Davis model D-501 time interval generator (Timer B) prior to the sequence control operations of the Lafayette 8-Bank timer. Timer B was keyed by the output pulse of Timer A. Thus, when the 0.5 sec signal delay was in effect the first light of the observation interval was also delayed by 0.5 sec to prevent the signal from occurring later in the observation interval for this condition than with the 0.0 sec signal delay.

The signal was generated by a Hewlett-Packard 201-C audio oscillator and frequency-calibrated by a Hewlett-Packard 5221-B electronic counter. Rise-decay time (10 msec) was determined by a Grason-Stadler 829-C electronic switch. Signal duration (100 msec) was gated by a Grason-Stadler 471-1 interval timer. Gating was dependent on the offset

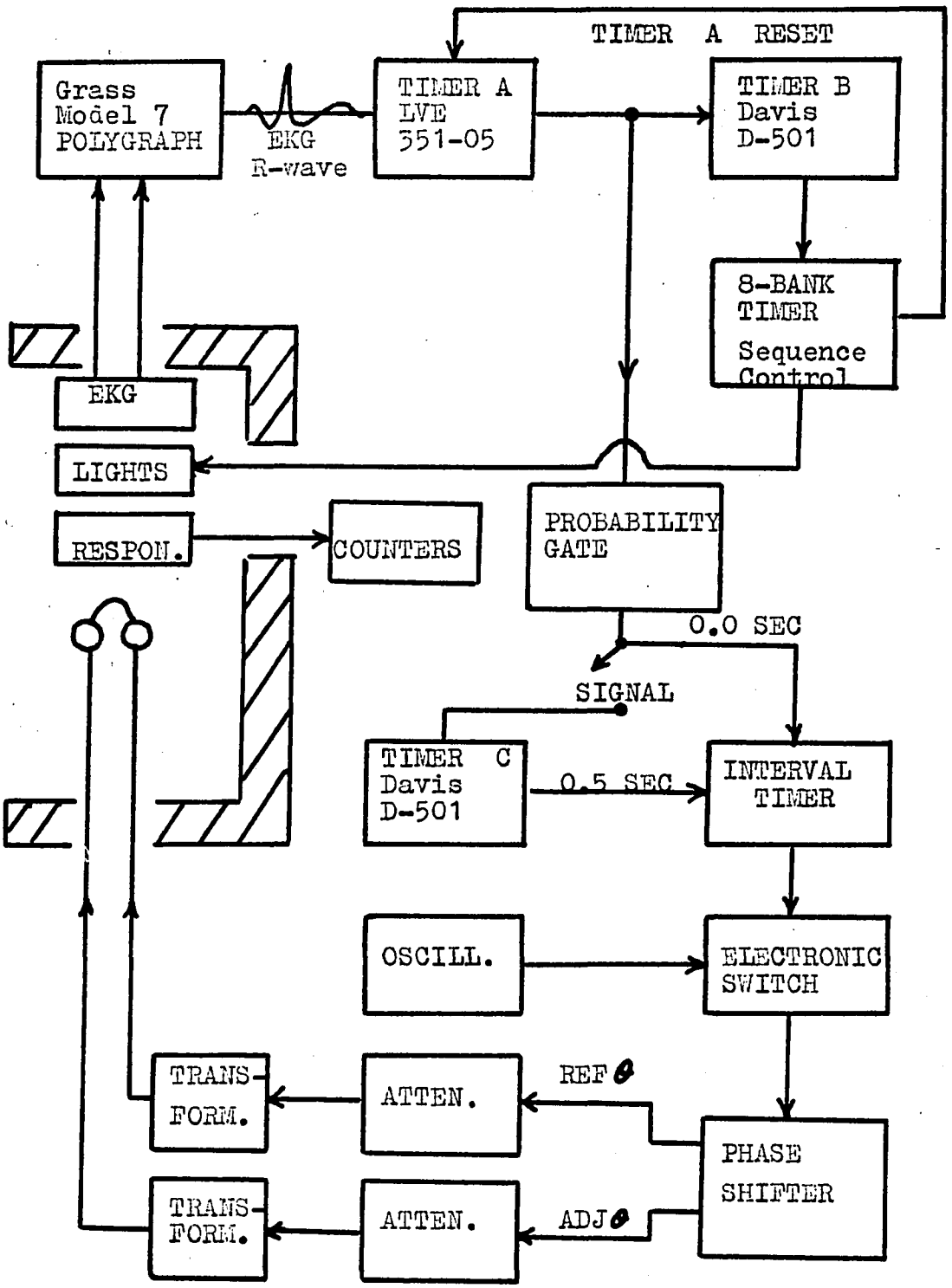



Figure 1. A schematic representation of the experimental apparatus.

pulse of Timer C in the 0.5 sec delay conditions and on the R-wave after offset of Timer A in 0.0 sec conditions. Signal phase (0° and 180°) was controlled by a Grason-Stadler Model E35208 phase shifter. Two Hewlett-Packard 350-D attenuators controlled reference and adjustable phase signal intensity for the right and left ears, respectively. Measurements of signal level were made at both earphones prior to each experimental session with a Ballantine true RMS voltmeter. Impedance matching antecedent to the earphones was done with two Grason-Stadler E 10589 A impedance matching transformers.

Blood volume measures were obtained by placing a Grass Model PTTI photoelectric transducer on S's earlobe and feeding the information through appropriate amplifiers into a Fabri-Tek Model 201 averaging computer. The plethysmographic measurement involved a computer average of 64 heartbeats triggered by the EKG R-wave and were made before the start of Experiment I.

# RESULTS

## Experiment I

A three factorial repeated measures analysis of variance (all factors within) was performed on the data presented in Table 1. The dependent variable, d', represents an average (mean) of the five replications for each condition of the experiment. Signal intensities (in dB SPL) at 100, 200, and 300 Hz for each S are shown in Table 3 (see Appendix).

Cochran's C statistic (Kirk, 1969, p. 62) indicated that the hypothesis of homogeneity of variance could not be rejected ($p > .05$). Thus, F ratios were determined by testing mean squares with pooled error variance. The analysis (see Table 4 in Appendix) indicated significantly larger values of d' associated with the 0.5 sec signal delay in comparison with 0.0 sec ($p < .01$). Similarly, the phase main effect showed S0 to be significantly greater than S$\pi$ ($p < .01$). Furthermore, the main effect for frequency indicated significant differences ($p < .01$). The only significant interaction was for signal delay-frequency ($p < .05$). The estimate of variance accounted for ($w^2$) was determined after variation due to subjects was removed from the analysis. Under the assumption that all factors were fixed, $w^2$ showed that the four significant factors accounted for approximately 70% of the total

TABLE 1

Mean Sensitivity (in d') for the Conditions of Experiment I

| Signal Delay | 0.0 Sec | | | | | | 0.5 Sec | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Phase | S0 | | | Sπ | | | S0 | | | Sπ | | |
| Frequency | 100 | 200 | 300 | 100 | 200 | 300 | 100 | 200 | 300 | 100 | 200 | 300 |
| S1 | 0.83 | 1.05 | 0.38 | 0.48 | 0.84 | 0.14 | 1.80 | 1.10 | 1.16 | 1.15 | 1.90 | 0.96 |
| S2 | 1.53 | 1.43 | 1.14 | 0.91 | 0.67 | 0.67 | 3.06 | 1.89 | 1.72 | 1.92 | 1.55 | 1.00 |
| S3 | 1.39 | 1.55 | 1.05 | 1.43 | 1.42 | 0.66 | 2.57 | 1.59 | 1.75 | 2.76 | 2.01 | 0.67 |
| Mean | 1.25 | 1.34 | 0.86 | 0.94 | 0.98 | 0.49 | 2.48 | 1.53 | 1.54 | 1.94 | 1.82 | 0.88 |
| Phase Mean | 1.15 | | | 0.80 | | | 1.85 | | | 1.54 | | |
| Delay Mean | 0.97 | | | | | | 1.69 | | | | | |

variance in the following manner: signal delay (36%), listening condition (7%), frequency (23%), and the signal delay-frequency interaction (4%). A Newman-Keuls post hoc test (Kirk, 1969, p. 91-93) on the significant main effect for frequency resulted in one significant comparison between 100 and 300 Hz ($p < .05$). Finally, a simple effects analysis (see Table 5 in Appendix) on the significant signal delay-frequency interaction showed all factors significant ($p < .0250$ or .0167) except frequency at the 0.0 sec signal delay (Kirk, 1969, p. 181).

The overall results are shown in Figure 2 and portray each S's sensitivity as a function of signal delay at 100, 200, and 300 Hz with S0 and S$\pi$ as parameters. In general, it is clear that all Ss showed substantial improvements in detection at the 0.5 sec signal delay for both listening conditions and all three frequencies. The exceptions to this trend are with the S0 condition at 200 Hz (S1 and S3) and the S$\pi$ condition at 300 Hz (S2). The significant detectability differences for the listening conditions show S0 at higher d' levels than S$\pi$ with an exception for S3 at 100 Hz. Additional reversals at the 0.5 sec delay were shown by S1 and S3 at 200 Hz. Finally, the significant main effect for frequency can be seen by the tendency for detectability to be better at 100 Hz than 300 Hz; i.e., there is a decrease in d' as frequency increases.

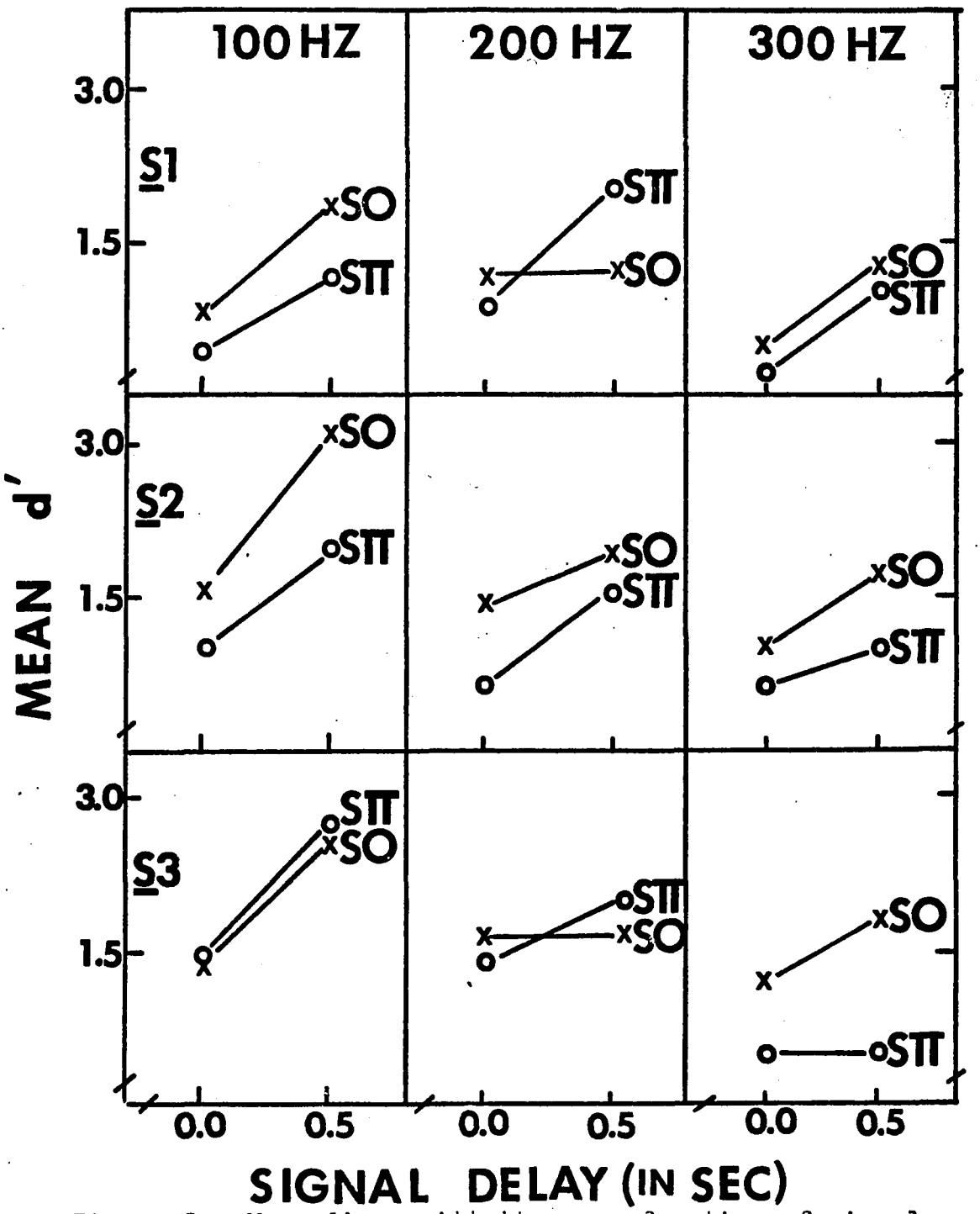



Figure 2. Mean d' sensitivity as a function of signal delay at 100, 200, and 300 Hz with SO and STT as parameters. Data are plotted for individual Ss across five replications.

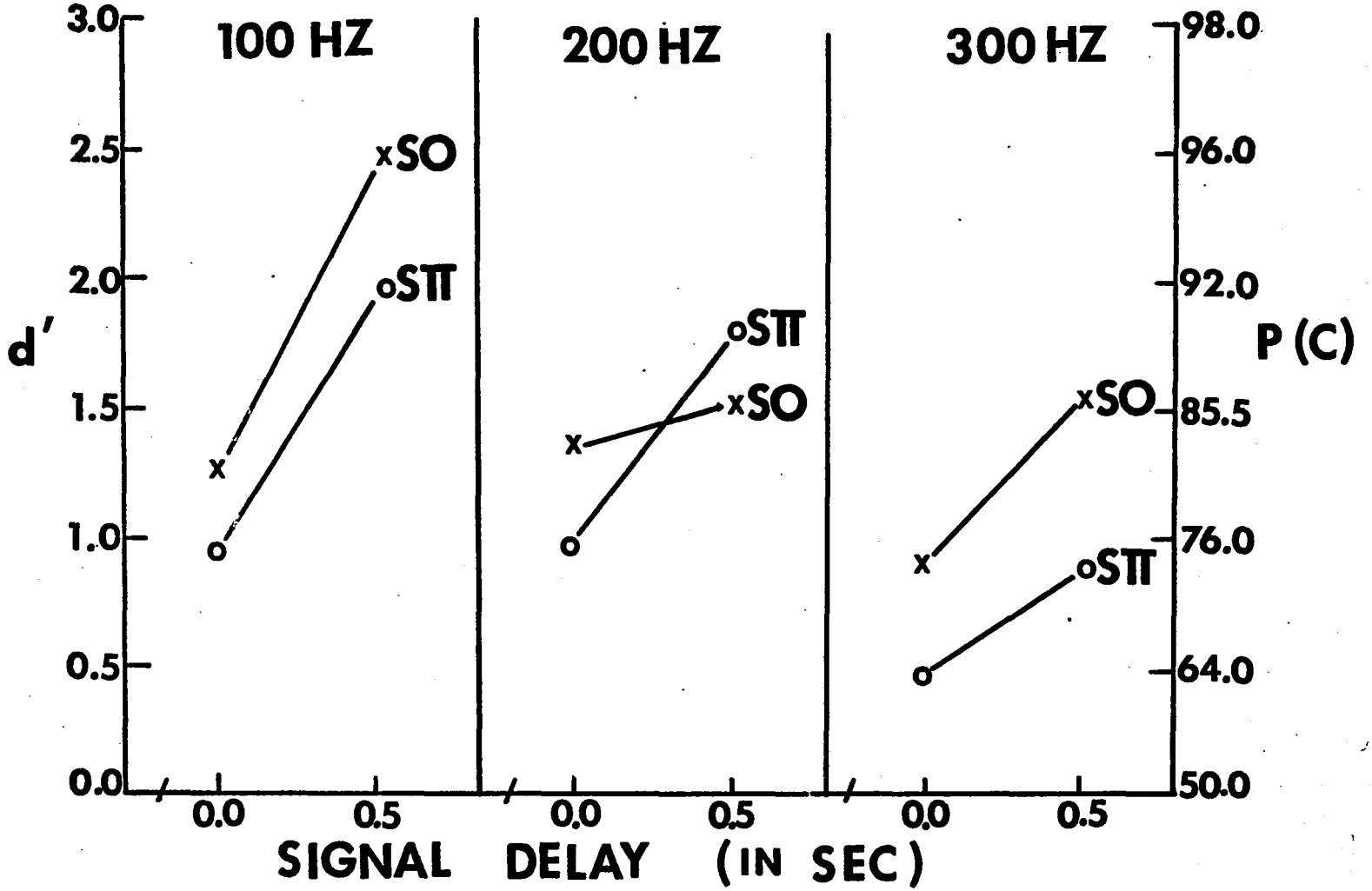



Figure 3. Mean d' values averaged across Ss as a function of the signal delay. The listening condition ($S\bar{O}$ or $S\Pi$) is plotted as the parameter at 100, 200, and 300 Hz. Equivalent P(C) is shown on the right ordinate.

The mean values of the individual data presented in Figure 2 are shown in Figure 3. The significant ($p < .01$) increase in detectability at the 0.5 sec delay is apparent. Also, the significant ($p < .01$) advantage for S0 re Sπ is evident, with the exception of a reversal at 200 Hz. Specifically, the extent of the improvement due to signal delay and listening condition can be readily understood by evaluating changes in mean d' in terms of equivalent percent correct [P(C)] values (cf. Elliott, 1964). For example, Sπ detection improved from about 75%, 76% and 64% at the 0.0 sec signal delay to 92%, 90%, and 73% at 100, 200, and 300 Hz, respectively. A similar pattern can be seen for S0 but with slightly higher P(C) or d'. That is, S0 detection increased from about 81%, 83%, and 73% at the 0.0 sec signal delay to approximately 96%, 86%, and 86% for the 0.5 sec delay at 100, 200, and 300 Hz, respectively. A further indication of the significant detection improvement for the 0.5 sec signal delay is shown in Figure 4 where mean d' is plotted as a function of frequency for both signal delays.

Two other relationships are also evident in Figure 4. First, the significant frequency effect ($p < .01$) is once again indicated by the tendency for d' to decrease as frequency increased. However, the Newman-Keuls post hoc analysis demonstrated that only the difference between 100 and 300 Hz was significant ($p < .05$). Second, the significant ($p < .05$) signal delay-frequency interaction can be seen by noting the

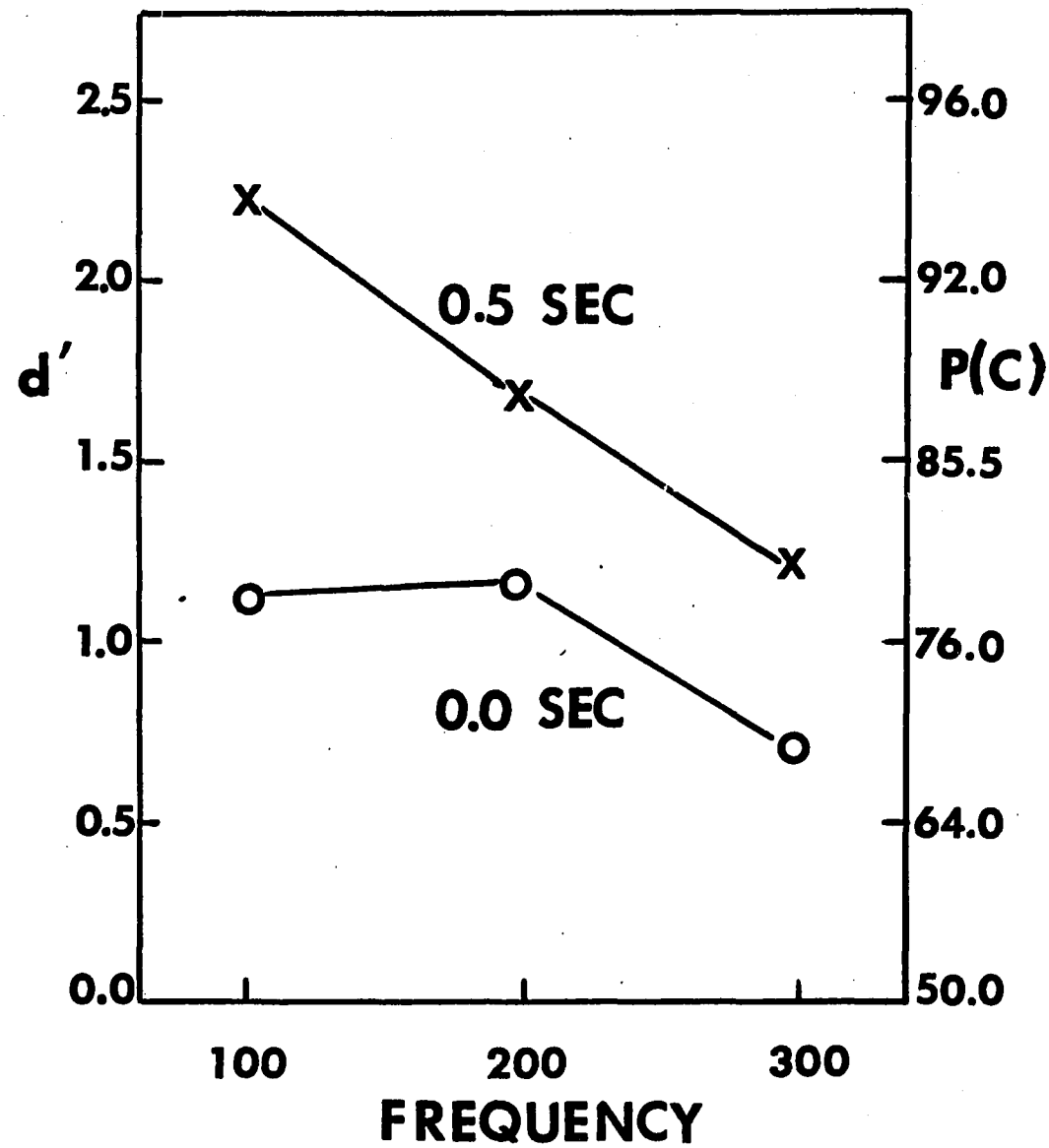



Figure 4. Mean d' [or P(C)] as a function of frequency for the 0.0 and 0.5 sec signal delay times. The data have been averaged across listening conditions and Ss.

divergence of the curves describing the 0.0 and 0.5 sec delay parameters. Clearly, there is a larger relative increase in d' (for 0.5 re 0.0 sec) at 100 Hz (1.11) than at 200 Hz (0.52) and 300 Hz (0.53). The simple effects analysis showed the interaction to be due to the significant frequency difference at 0.5 sec. Further analysis of the significant simple effects showed that 66% of the variance ($w^2$) between 0.0 and 0.5 sec was accounted for by the 100 Hz frequency, while 200 and 300 Hz showed only 13% and 14%, respectively. A further indication of this same relationship is that 71% of the variance due to frequency is accounted for by the 0.5 sec signal delay.

Experiment II

The individual data, averaged across five replications, are presented in Table 2 with the respective mean d' values. Signal intensities for the 5000 Hz tone are shown in Table 3. A repeated measures analysis of variance (see Table 6 in Appendix) indicated that S0 was significantly ($p < .01$) greater than S$\pi$ ($w^2 = 59\%$). Error variance was again pooled since Cochran's C statistic indicated that the hypothesis of homogeneous variances could not be rejected ($p > .05$). Inspection of Table 2 reveals that although the significant advantage for S0 is indicated in the data of all Ss, S3 contributed most to the magnitude of this difference. All other factors in Experiment II were non-significant.

TABLE 2

Mean Sensitivity (in d') for the Conditions of Experiment II

| | Signal Delay | | | |
|---|---|---|---|---|
| | 0.0 Sec | | 0.5 Sec | |
| Phase | S0 | Sπ | S0 | Sπ |
| S1 | 2.39 | 1.55 | 2.55 | 2.05 |
| S2 | 1.32 | 0.45 | 0.56 | 0.72 |
| S3 | 1.66 | 0.38 | 1.73 | 0.53 |
| Mean | 1.79 | 0.79 | 1.61 | 1.10 |
| Delay Mean | 1.29 | | 1.35 | |

Blood Volume and Heartrate Measures

Mean heartrates for S1, S2, and S3, respectively were: 78.7, 78.3, and 72.5 in Experiment I and 75.3, 83.0, and 70.4 in Experiment II. Figure 5 shows a typical EKG from each S along with a corresponding plethysmographic measure of his blood volume as determined at the earlobe. A single cardiac cycle has been plotted rather than the computer record of the EKG since the latter is less clear graphically due to the averaging across variable inter-R times. The single EKG which is shown does, however, represent the heart rate obtaining for each S at the time his blood volume was measured. It can readily be seen in Figure 5 that maximum blood volume for all Ss occurs in the neighborhood of 0.3 to 0.5 sec after the R-wave. Likewise, the lowest point in blood volume was between 0.1 and 0.2 sec after the R-spike.

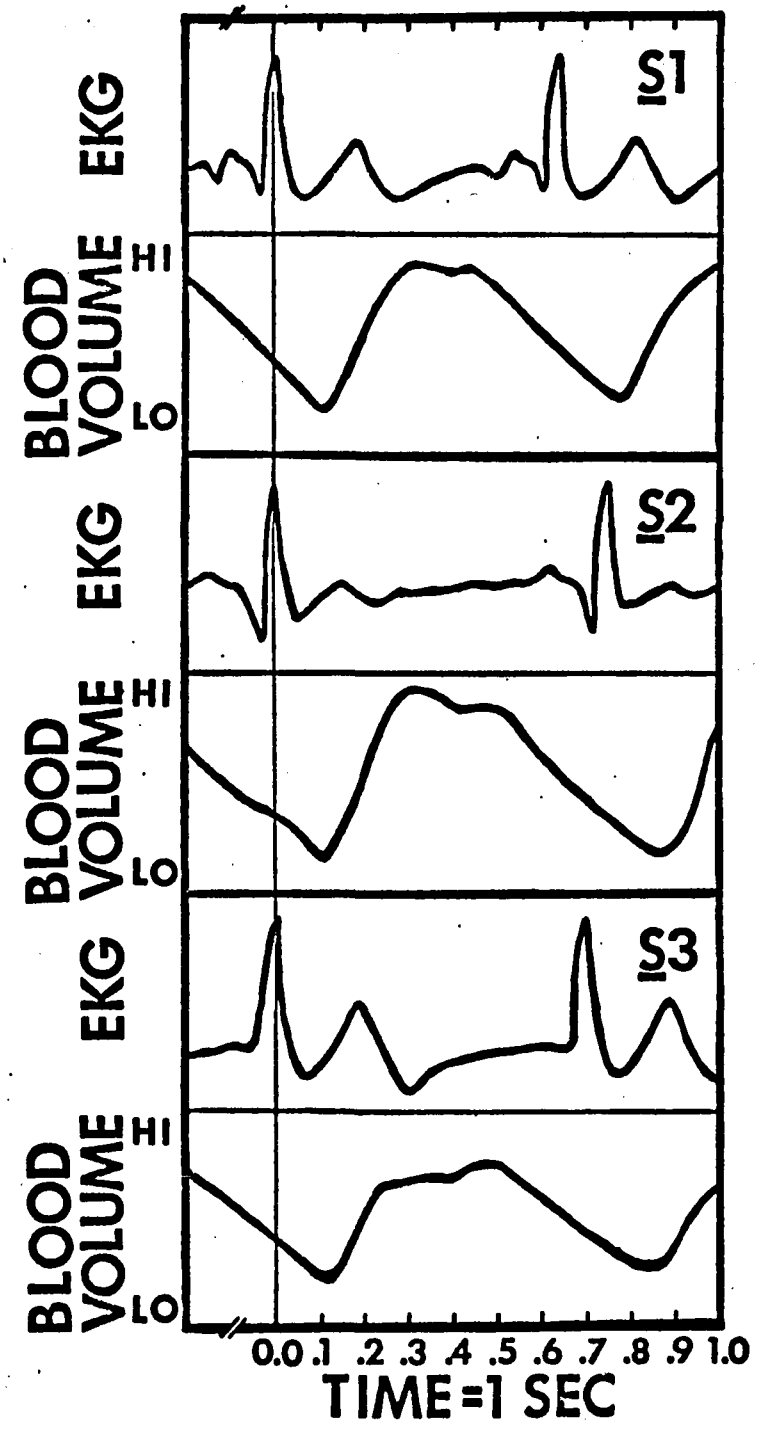



Figure 5. An illustration of each S's typical EKG plotted in temporal correspondence with changes in his blood volume as measured with a plethysmograph on the earlobe. The blood volume measures represent a computer average of 64 trials (heartbeats).

# DISCUSSION

The results of the present experiments indicate that binaural (S0 and Sπ) detection was strongly influenced by the S's cardiac cycle. Specifically, sensitivity was substantially improved by presenting the signal 0.5 sec after the EKG R-wave rather than coincident (0.0 sec) with it. This delay-advantage, however, appears to be frequency-dependent since it was largest at 100 Hz, decreased somewhat at 200 and 300 Hz, and disappeared at 5000 Hz. Contrary to the hypothesis under study, detection differences between the in-phase (S0) and inverted phase (Sπ) conditions were not influenced by signal delay; that is, the advantage for Sπ re S0 was not larger at 0.0 sec than 0.5 sec as predicted. More unexpectedly, sensitivity was, in fact, better for S0 than Sπ at all frequencies and delay conditions with a single exception.

## Subject Variability and Individual Differences

Despite the extensive (at least 4000 trials) pre-training, both experiments evidenced some variability. Numerous factors were very likely involved in this finding. First, subtle changes in motivational and attentional states introduced both intra- and inter-subject variability. The extent to which these factors were operating is difficult to assess. It may be suggested, however, that subject-state

changes were of greater magnitude in Experiment II than in Experiment I. Experiment II was, unavoidably, conducted in a post hoc fashion. That is, it was not realized at the start of the study that a higher frequency (5000 Hz) would be an ideal control for the signal delay differences observed in Experiment I. It could be argued, then, that greater motivational differences took place in Experiment II because Ss were requested to serve longer than planned. Furthermore, an additional factor in Experiment II is the task difficulty. In Experiment II the signal level used for each S resulted in detection tasks of differential difficulty (Table 2). In other words, the task was much easier for S1 than the other Ss. Thus, differences in the established baseline signal levels introduced spurious individual differences in addition to those genuinely present.

A second and perhaps more important source of variability may be attributed to the physical relation of the headphones on the ears, variations in earphone-fit, and consequently, changes in the "leak" of aural noise (Anderson & Whittle, 1971; Dolan, 1968; Villichur, 1970). This account is presumably most applicable to variability in Experiment I since most of the energy for aural noise is concentrated at the lower frequencies (Anderson & Whittle, 1971; Shaw & Piercy, 1962a; 1962b). This suggests, once again, that motivational and individual differences (spurious and actual) were perhaps more important factors than earphone-fit in Experiment II.

Third, the data in Experiment I showed relatively small individual differences (Figure 2). The variability which did occur is more likely a reflection of the previously mentioned motivational and earphone-fit factors than significant individual differences. On the other hand, in Experiment II (Table 2), the differential binaural sensitivity of one subject (S3) appears to have contributed most to the overall advantage for S0.

Binaural Analysis

The cardiac masking hypothesis (Soderquist & Lindsey, 1972) was used in Experiment I to argue that the interaural cues generated by phase reversal (Sπ) would lead to larger detection improvements (re S0) in the presence of physiological noise. It was inferred that larger MLDs would be found during the primary heart sound (0.0 sec) than in its absence (0.5 sec). The data argued against this logic and indicated a mean superiority for S0 (negative MLDs) at all frequencies and delays (except at 0.5 sec, 200 Hz). This finding was somewhat surprising and will be discussed in terms of previous work.

Comparison of the low-frequency data with extant MLD research is difficult. Most studies have determined MLD size in decibels (rather than d') by comparing S0 and Sπ attenuation thresholds (method of adjustment; Békésy audiometer) or by generating psychometric functions (two alternative temporal forced choice--2ATFC). More importantly, the available

data on interaural phase shifts at absolute threshold are conflicting. Diercks and Jeffress (1962), for example, report a significant (0.9 dB) advantage for Sπ re S0 at 250 Hz using a 50% threshold method and experienced Ss. Tempest, Marsh, and Bryan (1969) also found a significant (0.72 dB) phase effect at 250 Hz. However, only untrained Ss showed the effect, suggesting their method of study (Békésy audiometer) allowed for variable detection criteria. Gerber, Jaffe, and Allford (1970) continue the confusion by failing to find a significant Sπ effect for frequencies between 500 and 4000 Hz (with a Békésy audiometer). Additionally, Soderquist and Lindsey (1971b) found non-significant differences at 150 and 200 Hz which became significant ($p < .05$) only at 300 Hz for experienced Ss in a 2ATFC procedure. Thus, the current literature for low-frequency interaural phase effects presents a very ambiguous picture. The extremely small magnitude of the effect (often the result of averaging across Ss) suggests that different psychophysical methods and restrictions of subject sampling are largely responsible for the lack of agreement between studies. The situation is different at higher frequencies (e.g., 5000 Hz) since interaural time differences are too small to produce MLDs unless a high intensity masker is present (cf. Green & Henning, 1969).

Unfortunately, the present study does not clarify the low-frequency situation since negative MLDs were found.

Negative MLDs in binaural masking studies are atypical and seem to depend on the signal and masker being gated on simultaneously (McFadden, 1966; Wightman, 1969) or an extremely low (5 dB spectrum level) masker (Lindsey, 1970). Since no external masker was provided in the present study these explanations may be unlikely. Another explanation for the SO advantage can be offered. The presence of cardiac noise (0.0 sec) seems to be phenomenally perceived as located at the ears. Similarly, the $S\pi$ signal is perceived at the periphery. Therefore, it could be claimed that the detection advantages found for SO reflect the separation in phenomenal space between the noise (periphery) and signal (median plane). Thus, the absence of an $S\pi$ advantage at the 0.0 sec signal delay would presumably suggest peripheral masking. However, there are two problems with this explanation for the SO superiority. First, SO is also significantly better at 0.5 sec, where the inferred noise is absent. Secondly, Jeffress, Blodgett, Sandel, and Wood (1956) have argued against the "phenomenal space" explanation for MLDs. They state that:

> Under binaural conditions, when the masked threshold is lower, it is lower because we hear the sound move when the signal is added to the noise, not because we hear the signal in one place and the noise in another. This experience does occur, but only for strong signals, not for signals near threshold [p. 426].

Hence, the data fail to conclusively support the hypothesis that interaural cues available to the binaural system are facilitated by the inferred physiological noise.

There appears to be a discrepancy, then, between two bodies of research literature. Numerous studies have shown that an increase in the intensity of external noise results in larger MLDs (Dolan, 1968; McFadden, 1968; Hirsh, 1948a). On the other hand, evidence to support the presence of IN is also substantial (Anderson & Whittle, 1971; Piercy & Shaw, 1963; Shaw & Piercy, 1962a; 1962b; Soderquist & Lindsey, 1972). This contradiction was not evident in the study of Diercks & Jeffress (1962) who inferred that the S$\pi$ advantage they observed reflected a binaural unmasking effect. It could be argued that the opposite (SO advantage) finding of the present study implies that the assumption that 0.0 and 0.5 sec correspond to different levels of IN should be questioned. However, discussion in a later section will demonstrate that an IN explanation is probably the most suitable account for the detection differences observed between 0.0 and 0.5 sec. Thus, this leaves the relatively weak intensity of the IN as a possible focus for the discrepancy. Previous data have shown only small low-frequency MLDs when spectral levels were below 15 dB (Soderquist & Lindsey, 1971a; 1971c; Wilbanks & Cornelius, 1969). Perhaps, then, it was unreasonable to expect substantial binaural unmasking from a brief 150 msec (cf. Ganong, 1969) and weak intensity masker (the first cardiac sound). Apparently, the binaural system was unable to utilize the available noise background to facilitate processing of the interaural cues in the S$\pi$ condition. In

fact, it is possible that the phase-sensitive detection mechanism was degraded.

In summary, the superiority of SO detection over SΠ cannot be readily explained except in terms of the out-dated hypothesis of binaural power summation (Hirsh, 1948b; Shaw, Newman, & Hirsh, 1947). Nonetheless, several additional variables can be regarded as relevant to the SO advantage. First, there are methodological differences between this study and others which have measured MLDs at absolute threshold. The latter studies have allowed the signal to vary randomly in relation to S's EKG and hence, were unable to assess associated changes in binaural analysis. However, it is not immediately obvious what variables or interactions would be necessary to account for the discrepant MLD sizes. Perhaps, aural and physiological noise combined in the present study to produce an uncorrelated masker (NU) and hence the advantage for SO (e.g., Dolan, 1968). Second, the discrepant findings in the literature concerning the influence of interaural phase effects at absolute threshold remain to be resolved. It appears that S's criterion and training may indeed be relevant variables. This study, for example, eliminated criterion problems by using a TSD task with highly trained Ss. This suggests, as did the Tempest et al. (1969) study, that experience may remove phase effects at absolute threshold. Finally, it is possible that some feature of subject selection or nuance of stimulus presentation

produced unrepresentative MLDs, although this is unlikely. Thus, the resolution of the effect of phase at absolute threshold is incomplete.

Frequency, Signal Delay, and the Cardiac Masking Hypothesis

Previous research by Soderquist and Lindsey (1972) showed changes in detection of a binaural (S0) 100 Hz tone as a function of temporal delay in signal presentation following the EKG R-wave. Experiment I replicated the significantly better (than 0.0 sec) sensitivity they observed at 0.5 sec signal delay and showed that this advantage was inversely related to frequency (Figure 4). The non-significant delay effects at 5000 Hz (Experiment II) reiterated this frequency-dependency (Table 2) and imply that "unauthorized" detection cues or listening strategies were not operating in the study.

Brief mention should be made of the significant frequency effect in Experiment I. It is clear from Figure 4 that, despite considerable effort, detectability in the baseline condition (0.0 sec) was not held at d'= 1.00 as frequency varied. Although the means do not differ significantly at 0.0 sec (Table 5) detection was somewhat worse at 300 Hz. Apparently, the drop at 300 Hz is indicative of an error in signal level estimation for S1 (Table 1). The significant frequency effect was primarily due to detectability changes at 0.5 sec ($w^2$= 71%) and hence, qualifies the following discussion of the delay-frequency interaction (Table 5)

only slightly. Similarly, the significant frequency difference between 100 and 300 Hz seems to be due to the relatively greater 0.5 sec detection ($w^2$= 66%) at 100 Hz (Table 5). Nonetheless, it is unfortunate that baseline performance was not strictly constant across frequency. Slightly better detection at 300 Hz (0.0 sec) may have indicated the magnitude of the signal delay-frequency interaction even more clearly.

The fact that detectability was influenced by the position of the signal in the EKG cycle suggests that the cardiac masking hypothesis of Soderquist and Lindsey (1972) may also be a reasonable explanation for the present data. Further alternatives require consideration before this conclusion can be reached, however. For instance, Delfini and Campos (1971) failed to find poorer detection for 1000 Hz (SO) during the QRS and T phases of the EKG as suggested by the cardiac masking hypothesis. There are essentially two methodological differences between their experiment and the present study. First, they correlated detection performance with relatively large (0.2 sec) EKG segments of the polygraphic record. Hence, the size of the segments allowed for less exact time specification that the triggering approach of the present study. Second, the relatively intense level (53 dB SPL) of ambient, ventilator noise served (as they suggest) a masking function; this may have overriden any IN present. Another relevant factor for the null effect

between detection and EKG phase which is not related to these methodological considerations concerns the frequency of the signal. Measurements of aural and cardiac noise have indicated that the level of IN at 1000 Hz is negligible (Anderson & Whittle, 1971; Munson & Wiener, 1952; Saito et al., 1969). The Delfini and Campos (1971) data, then, do not argue against an IN explanation and could, in fact, be viewed as congruent with the inverse relation between frequency and the magnitude of the 0.5 sec detection advantages.

In order to suggest that the above contradiction is apparent requires further analysis of interpretations relevant to the interaction between EKG phase and frequency. Numerous authors have suggested that a cardiac-cortical system modulates sensory sensitivity (Delfini & Campos, 1971; Lacey & Lacey, 1970; Saxon & Dahle, 1971). However, such a "cardiac arousal" explanation would seem to require (for the present data) the unreasonable assumption that inhibition of cortical arousal (the observed QRS detection decrement) was influenced not only by cardiac but auditory afference as well. Hence, two acoustically-oriented hypotheses: "cardiac masking" (Soderquist & Lindsey, 1972) and "meatal pulsation" (Anderson & Whittle, 1971) will be evaluated.

The latter hypothesis, "meatal pulsation," suggests that an increase in sound pressure in the external auditory canal is caused by an increase in local blood pressure. This inference relies on the finding that concurrent

oscillographic records of carotid blood pressure and aural noise demonstrate substantial correspondence. Peak carotid blood pressure was coincident with the maximum of the oscillogram for aural noise. The timing after the R-wave for these events was, unfortunately, not contained in the Anderson and Whittle (1971) report. Hence, viewing the present data in terms of the meatal pulsation hypothesis is somewhat speculative. Their oscillogram traces are separated by about 0.8 sec, the same as for an average heart rate of about 75 bpm. Interpolation from another source (Ganong, 1969) suggests that the peak carotid pulse follows the R-spike of the EKG by about 0.3-0.4 sec at 75 bpm. Thus, it would appear that the blood pressure measured by Anderson and Whittle (1972) is quite similar to the blood volume data obtained in the current study and shown in Figure 5. If the above interpolation is reasonably accurate, it implies that the meatal pulsation hypothesis would predict maximal masking at 0.3-0.4 sec after the R-wave. Although Soderquist and Lindsey (1972) did observe a detection drop at 0.3 sec, that data and the present study found the greatest sensitivity loss considerably prior (i.e., 0.0 sec) to the period of peak pulsation suggested by Anderson and Whittle (1971). The meatal pulsation hypothesis, then, seems to provide a logical basis for the generation of aural noise. However, the temporal course of either blood pressure (Anderson & Whittle, 1971), blood volume (Figure 5), or S's heart rate

shows only a weak correspondence to the observed detection differences. Likewise, cochlear blood flow is not essentially different from the plethysmographic data presented in Figure 5 (Suga & Snow, 1969).

Thus, the blood-related concomitants of the EKG do not appear to indicate that physiological changes have a clear causal role in the explanation of the psychophysical data. The heart sound generated by the closing of the mitral and tricuspid valves (ventricular depolarization) appears, by exclusion, to be the only cardiac event concurrent with the poorer detection at 0.0 sec. Thus, the cardiac masking hypothesis argues that the process underlying the observed EKG-detectability differences is acoustical rather than physiological. Consideration of the frequency trend observed in the present study appears to add substance to this hypothesis. That is, since the effect of signal delay varies with frequency, the data suggest that the spectrum of IN consists primarily of low frequencies and can be conceptualized relative to the temporal course of the EKG. Hence, the improvement in detectability offered by delaying the signal 0.5 sec became irrelevant at 5000 Hz when the frequency difference between the signal and "noise" exceeded that sufficient to generate masking. Frequencies at or near the signal are known to be more effective maskers than those farther removed (Jeffress, 1970; Mayer, 1894; Wegel & Lane, 1924). In these terms, the low-frequency facilitation at 0.5 sec can

be looked upon as a "release from masking." The greatest spectral energy of the cardiac noise is centered at 100 Hz and decreases with frequency (Saito et al., 1969). Hence, the decrease of the 0.5 sec detectability advantage from 100 to 5000 Hz is in keeping with the spectral characteristics of heart sounds and their probable masking properties.

In summary, the detectability gains shown by locating a signal 0.5 sec after the S's EKG R-wave seem to be frequency-dependent. The frequency-dependency coupled with the inadequacy of physiological explanations suggests that cardiac noise is present at the cochleas and has masking properties. It is assumed that the skeletal system and body tissues serve as adequate transmission media (cf. Butterworth, Chassin, McGrath, & Reppert, 1960) for the conduction of heart sounds to the cochlea. This assumption awaits direct empirical support. Overall, the causal role for cardiac sounds suggested by the cardiac masking hypothesis seems reasonable. However, it relies heavily on a correlational analysis. As such, the inability to demonstrate MLDs in the presence of the inferred noise qualifies the explanatory power of the hypothesis. Future research will hopefully determine the interaural correlation of the cardiac masker as well as clarifying the extent to which the presence of MLDs (at absolute threshold) and the inference of IN are necessarily and sufficiently related.

Suggestions for Future Research

Several suggestions for future research utilizing the paradigm of the present study will be briefly outlined. They are the following: (1) A signal known exactly (SKE) procedure (e.g., Egan, Schulman, & Greenberg, 1964) whereby the observation interval is specified by a single light coincident with the signal should be used. This method would reduce any variability between Ss due to listening strategy and serve as an excellent control for differential delay effects not due to cardiac masking. (2) The inference of cardiac masking should be examined with a larger range of frequencies. Likewise, since the primary cardiac sound (at the R-wave) has a duration of only 150 msec (Ganong, 1969), perhaps a shorter duration signal than the 100 msec used in this study would allow a better test of the MLD/IN hypothesis. (3) Comparison between normal and otosclerotic Ss such as that used by Moulin (1969) would more completely test the inference that masking occurs within the cochlea rather than by aural noise in the external auditory meatus. That is, evidence that the inner ear is the site of tonal masking (as suggested by the cardiac masking hypothesis) would be indicated by the demonstration of signal delay effects for both (normal and otosclerotic) groups. In other words, the undetected IN in the external canal for the otosclerotic group should have no significant effect on detectability changes due to EKG phase although, threshold baselines would, of

course, differ. (4) The interaction between aural noise and the signal delay effect could also be examined by manipulating headband pressure (e.g., Anderson & Whittle, 1971) or by using an earphone which reduces this source of IN (e.g., Villichur, 1970). Additionally, it is possible that insert earphones could be used in an attempt to differentiate between the aural and cardiac masking hypotheses. For instance, Rudmose (1962) eliminated the "missing 6 dB" phenomenon by decreasing the enclosed volume under the earphone with an earmold system. Thus, it could be expected that an insert earphone would decrease aural noise but not influence cardiac masking.

## SUMMARY

Two experiments were conducted in an effort to determine the relationship between auditory signal detectability and the S's EKG (cardiac cycle). Previous work in the literature had indicated that physiological noise (heart sounds) generated by the valve closures of the heart tended to mask a low-frequency tone. Thus, two questions were of particular interest: (1) Does the binaural listening system utilize internal noise (IN) to lower inverted phase ($S\pi$) [relative to in-phase (S0)] thresholds as it does with external noise? (2) What is the relationship between the masking effects of cardiac sounds and the frequency of the signal to be detected?

Data were collected according to a yes/no, signal detection paradigm in repeated measures designs. Three males served as trained subjects. The S's task was to indicate (by pushing one of two microswitches) his decision concerning the presence of a 100 msec signal with a 0.5 *a priori* probability of occurrence. Experiment I investigated the following parameters: (1) Signal Delay: 0.0 and 0.5 sec following the EKG R-wave; (2) Signal Frequency: 100, 200, and 300 Hz; (3) Listening Condition: S0 and $S\pi$. Experiment II examined the same delay and listening conditions as Experiment I but at 5000 Hz. Subjects received a quasi-random schedule of these parameters. The dependent variable was

signal detectability as measured by d'. Two hypotheses were studied. First, it was predicted (based on the frequency spectrum of cardiac sounds) that depressed detectability (masking) would be found at 0.0 sec signal delay (re 0.5 sec) at 100, 200, and 300 Hz, but not at 5000 Hz. Second, detection advantages for Sπ re S0 were predicted to be largest when the signal was coincident (0.0 sec) with the R-wave of the EKG. This prediction was suggested by the masking-level difference (MLD) literature on facilitation of interaural cues by the addition of noise, in this case, cardiac sounds.

The experiments indicated support for the first hypothesis since binaural (S0 and Sπ) detection was significantly improved by presenting the signal 0.5 sec after the EKG R-wave rather than coincident with it (0.0 sec). This delay advantage, however, depended significantly on frequency since it was largest at 100 Hz, decreased somewhat at 200 and 300 Hz, and disappeared at 5000 Hz. However, contrary to the second hypothesis, detection differences between S0 and Sπ were not influenced by signal delay; that is, the advantage for Sπ re S0 was not larger at 0.0 sec than 0.5 sec. Rather, sensitivity for S0 was significantly better than Sπ for all conditions except one.

The results were discussed in terms of the cardiac masking hypothesis. The frequency-dependency of the signal delay effect suggested that an acoustical rather than a

physiological (blood pressure/volume) process was implicated, and that the inferred noise was of low-frequency spectrum. It was suggested that cardiac noise due to the closing of the mitral and tricuspid valves produced masking at the 0.0 sec signal delay and release from masking at 0.5 sec. The correlational analysis of the data and the absence of interaural phase effects were regarded as limitations on the explanatory power of the cardiac masking hypothesis. Several suggestions for future research were offered.

## REFERENCES

Anderson, C. M. B., & Whittle, L. S. Physiological noise and the missing 6 dB. Acustica, 1971, 24, 261-272.

Boys, F., & Curry, E. T. Measurement of auditory thresholds before and after strenuous physical exercise. Annals of Otology, Rhinology, and Laryngology, 1956, 65, 190-197.

Brogden, W. J., & Miller, G. A. Physiological noise generated under earphone cushions. Journal of the Acoustical Society of America, 1947, 19, 620-625.

Butterworth, J. S., Chassin, M. R., McGrath, R., & Reppert, E. H. Cardiac auscultation. New York: Grune and Stratton Co., 1960.

Corso, J. F. A theoretico-historical review of the threshold concept. Psychological Bulletin, 1963, 60, 356-370.

Corso, J. F., & Cohen, A. Methodological aspects of auditory threshold measurements. Journal of Experimental Psychology, 1958, 55, 8-12.

Davis, H. Psychophysiology of hearing and deafness. In S. S. Stevens (Ed.), Handbook of experimental psychology. New York: John Wiley and Sons, 1951.

Delfini, L. F., & Campos, J. J. Signal detection and the "cardiac arousal cycle." Paper read at the Society for Psychophysiological Research, St. Louis, 1971.

Diercks, K. J., & Jeffress, L. A. Interaural phase and the absolute threshold for tone. Journal of the Acoustical Society of America, 1962, 34, 981-984.

Dolan, T. R. Effect of masker spectrum level on masking-level differences at low signal frequencies. Journal of the Acoustical Society of America, 1968, 44, 1507-1512.

Dolan, T. R., & Robinson, D. E. An explanation of masking level differences that result from interaural and intensive disparities of noise. Journal of the Acoustical Society of America, 1967, 42, 977-981.

Egan, J. P., Schulman, A. I., & Greenberg, G. Z. Memory for waveform and time uncertainty in auditory detection.

Cited in J. A. Swets (Ed.), Signal detection and recognition by human observers. New York: John Wiley and Sons, 1964, 510-515.

Eijkman, E., Thijssen, J. M., & Vendrik, A. J. H. Weber's law, power law, and internal noise. Journal of the Acoustical Society of America, 1966, 40, 1164-1173.

Eijkman, E., & Vendrik, A. J. H. Detection theory applied to the absolute sensitivity of sensory systems. Biophysical Journal, 1963, 3, 65-78.

Eijkman, E., & Vendrik, A. J. H. Can a sensory system be specified by its internal noise? Journal of the Acoustical Society of America, 1965, 37, 1102-1109.

Elliott, P. A. Tables of d'. In J. A. Swets (Ed.), Signal detection and recognition by human observers. New York: John Wiley and Sons, 1964, 651-684.

Fitzhugh, R. The statistical detection of threshold signals in the retina. Journal of General Physiology, 1957, 40, 925-948.

Ganong, W. F. Review of medical physiology. Los Altos, California: Lange Medical Publications, 1969, 447-451.

Gasteiger, E. L., & Brust-Carmona, H. On the relation of spinal reflex variability to internal noise. Transactions of the New York Academy of Sciences, 1964, 26, 688-696.

Gerber, S. E., Jaffe, P. G., & Allford, B. J. Binaural threshold and interaural phase angle differences. Journal of the Acoustical Society of America, 1970, 48, 85-86. (Abstract)

Green, D. M. Psychoacoustics and detection theory. In J. A. Swets (Ed.), Signal detection theory and recognition by human observers. New York: John Wiley and Sons, 1964, 86-87.

Green, D. M., & Henning, G. B. Audition. In. P. H. Mussen and M. R. Rosenzweig (Eds.), Annual Review of Psychology. Palo Alto, California: Annual Reviews, Inc., 1969, 105-128.

Green, D. M., & Swets, J. A. (Eds.). Signal detection theory and psychophysics. New York: John Wiley and Sons, 1966.

Guyton, A. C. Basic human physiology: Normal function and mechanisms of disease. Philadelphia: W. B. Saunders Co., 1971, 125-268.

Hirsh, I. J. The influence of interaural phase on interaural summation and inhibition. Journal of the Acoustical Society of America, 1948, 20, 536-544. (a)

Hirsh, I. J. Binaural summation--A century of investigation. Psychological Bulletin, 1948, 45, 193-206. (b)

Jeffress, L. A. Masking. In J. V. Tobias (Ed.), Foundations of modern auditory theory. New York: Academic Press, 1970, Vol. I, 95-114.

Jeffress, L. A., Blodgett, H. C., Sandel, T. T., & Wood, C. L. Masking of tonal signals. Journal of the Acoustical Society of America, 1956, 28, 416-426.

Kikuti, Y., & Yosida, T. Behaviour of time-difference and phase-difference in the binaural effect. Japanese Journal of Medical Sciences, 1940, 7, 71-77.

Kirk, R. E. Experimental design: Procedures for the behavioral sciences. Belmont, California: Brooks/Cole Publishing Co., 1969.

Lacey, J. I., & Lacey, B. C. Some autonomic-central nervous system interrelationships. In P. Black (Ed.), Physiological correlates of emotion. New York: Academic Press, 1970, 205-227.

Lawson, R. W. Aural sensitivity and heart beats. Nature, 1948, 162, 782-783.

Lindsey, J. W. Masking-level difference (MLD) as a function of noise spectrum level, frequency, and signal duration. Master's thesis. University of North Carolina at Greensboro, 1970.

Loeb, M., & Dickson, C. Factors influencing the practice effect for auditory thresholds. Journal of the Acoustical Society of America, 1961, 33, 917-920.

Mayer, A. M. Researches in acoustics. No. IX. Philosophical Magazine 5th Series, 1894, 37, 259. Cited in D. D. Greenwood, Critical bandwidth and the frequency coordinates of the basilar membrane. Journal of the Acoustical Society of America, 1961, 33, 1344.

McFadden, D. Masking-level differences determined with continuous and burst masking noise. Journal of the Acoustical Society of America, 1966, 40, 1414-1419.

McFadden, D. Masking-level differences determined with and without interaural disparities in masker intensity. Journal of the Acoustical Society of America, 1968, 44, 212-223.

Molyneux, L. Biological signals and noise. Nature, 1963, 197, 855-856.

Moulin, L. K. The effects of physiological noise on the auditory threshold. (Doctoral dissertation, Case Western Reserve University). Ann Arbor, Michigan: University Microfilms, 1969, No. 69-9363.

Munson, W. A., & Wiener, F. M. In search of the missing 6 dB. Journal of the Acoustical Society of America, 1952, 24, 498-501.

Piercy, J. E., & Shaw, E. A. G. Estimation of equivalent free field physiological-noise levels by loudness balancing. Journal of the Acoustical Society of America, 1963, 35, 778. (Abstract)

Rudmose, W. Pressure vs. free field thresholds at low frequencies. In A. K. Nielsen (Ed.), Fourth International Congress on Acoustics (Copenhagen). Amsterdam: Elsevier Publishing Co., 1962, Paper H52.

Saito, K., Kobayashi, K., Yasuda, T., Inagaki, Y., Nakamura, Y., Tokumasa, Y., Yaguchi, T., & Oose, S. Measurement of intracardiac sounds. In Y. Kohasi (Ed.), Sixth International Congress on Acoustics (Tokoyo). Tokoyo: Maurzen Publishing Co., 1969, Paper M-1-7.

Saxon, S. A., & Dahle, A. J. Auditory threshold variations during periods of induced high and low heart rates. Psychophysiology, 1971, 8, 23-29.

Shaw, E. A. G., & Piercy, J. E. Physiological noise in relation to audiometry. Journal of the Acoustical Society of America, 1962, 34, 745. (Abstract) (a)

Shaw, E. A. G., & Piercy, J. E. Audiometry and physiological noise. In A. K. Nielsen (Ed.), Fourth International Congress on Acoustics (Copenhagen). Amsterdam: Elsevier Publishing Co., 1962, 1-4. (b)

Shaw, W. A., Newman, E. B., & Hirsh, I. J. The difference between monaural and binaural thresholds. Journal of Experimental Psychology, 1947, 37, 229-242.

Sivian, L. J., & White, S. D. On minimum audible sound fields. Journal of the Acoustical Society of America, 1933, 4, 288-321.

Soderquist, D. R., & Lindsey, J. W. Masking-level differences as a function of noise spectrum level, frequency, and signal duration. Journal of Auditory Research, 1971, 10, in press. (a)

Soderquist, D. R., & Lindsey, J. W. Interaural phase effects at absolute threshold. Paper presented at 17th meeting of the Southeastern Psychological Association, Miami, 1971. (b)

Soderquist, D. R., & Lindsey, J. W. Masking-level differences determined for low intensity maskers and different reference conditions. Psychonomic Science, 1971, under editorial review. (c)

Soderquist, D. R., & Lindsey, J. W. Physiological noise as a masker of low frequencies: The cardiac cycle. Journal of the Acoustical Society of America, 1972, 51, in press.

Stevens, S. S., & Davis, H. Hearing: Its psychology and physiology. New York: John Wiley & Sons, Inc., 1938, 53-54.

Stewart, J. L. Fundamental constraints to sensory discrimination imposed by two kinds of neural noise. Behavioral Science, 1965, 10, 271-276.

Suga, F., & Snow, J. B., Jr. Cochlear blood flow in response to vasodilating drugs and some related agents. The Laryngoscope, 1969, 79, 1956-1979.

Swets, J. A., (Ed.). Signal detection and recognition by human observers. New York: John Wiley & Sons, Inc., 1964.

Swets, J. A., Shipley, E. F., McKey, M. J., & Green, D. M. Multiple observations of signals in noise. Journal of the Acoustical Society of America, 1959, 31, 514-521.

Tempest, W., Marsh, J. A., & Bryan, M. E. The monaural and binaural thresholds of hearing. International Audiology,

Thijssen, J. M., & Vendrik, A. J. H. Internal noise and transducer function in sensory detection experiments: Evaluation of psychometric curves and of ROC curves. Perception and Psychophysics, 1968, 3, 387-400.

Villichur, E. Audiometer-earphone mounting to improve inter-subject and cushion-fit reliability. Journal of the Acoustical Society of America, 1970, 48, 1387-1396.

Ward, W. D. Auditory fatigue and masking. In J. Jerger (Ed.), Modern developments in audiology. New York: Academic Press, 1963.

Watson, C. S., Franks, J. R., & Hood, D. C. Detection of tones in the absence of external masking. Journal of the Acoustical Society of America, 1967, 42, 1194. (Abstract)

Wegel, R. L., & Lane, C. E. The auditory masking of one pure tone by another and its probable relation to the dynamics of the inner ear. Physical Review, 1924, 23, 266-285.

Wever, E. G., & Lawrence, M. Physiological acoustics. Princeton: Princeton University Press, 1954.

Wightman, I. L. Binaural masking with sine-wave maskers. Journal of the Acoustical Society of America, 1969, 45, 72-78.

Wilbanks, W. A., & Cornelius, P. T. Binaural detection of low-frequency tones as a function of noise level. Paper read at 77 th meeting of the Acoustical Society of America, Philadelphia, 1969.

Zwislocki, J., Maire, F., Feldman, A. S., & Rubin, H. On the effect of practice and motivation on the threshold of audibility. Journal of the Acoustical Society of America, 1958, 30, 254-262.

# APPENDIX

TABLE 3

Mean Signal Intensity in SPL for each Subject and all Four Frequencies

| Subjects | Frequency | | | |
|---|---|---|---|---|
| | 100 Hz | 200 Hz | 300 Hz | 5000 Hz |
| S1 | 53.6 | 26.6 | 11.6 | 8.6 |
| S2 | 46.6 | 18.6 | 8.6 | 10.6 |
| S3 | 49.6 | 22.6 | 7.6 | 10.6 |

TABLE 4

Summary of Analysis of Variance for Experiment I

| Source of Variation | SS | df | MS | F | $w^2$ |
|---|---|---|---|---|---|
| Signal Delay | 4.68 | 1 | 4.68 | **46.80 | 0.36 |
| Listening Condition | 0.95 | 1 | 0.95 | ** 9.50 | 0.07 |
| Frequency | 3.14 | 2 | 1.57 | **15.70 | 0.23 |
| Subjects | 2.34 | 2 | 1.17 | | |
| Signal Delay x Listening Condition | 0.01 | 1 | 0.01 | 0.10 | |
| Signal Delay x Freq. | 0.71 | 2 | 0.36 | *3.60 | 0.04 |
| Listening Condition x Frequency | 0.39 | 2 | 0.20 | 2.00 | |
| Signal Delay x Listening Cond. x Frequency | 0.42 | 2 | 0.21 | 2.10 | |
| Pooled Error | 2.18 | 22 | 0.10 | | |
| Signal Delay x Ss | 0.04 | 2 | 0.02 | | |
| Listening Cond. x Ss | 0.55 | 2 | 0.28 | | |
| Frequency x Ss | 0.66 | 4 | 0.16 | | |
| Signal Delay x Listening Cond. x Ss | 0.05 | 2 | 0.02 | | |
| Signal Delay x Frequency x Ss | 0.26 | 4 | 0.06 | | |
| Listening Cond. x Frequency x Ss | 0.50 | 4 | 0.12 | | |
| Residual | 0.12 | 4 | 0.03 | | |
| Total | 14.82 | 35 | | | |

* Significant at $p < .05$

** Significant at $p < .01$

TABLE 5

Summary of Simple Effects Analysis of Variance on the Signal Delay--Frequency Interaction

| Source of Variation | SS | df | MS | F | $w^2$ |
|---|---|---|---|---|---|
| Signal Delay | 4.68 | 1 | 4.68 | **46.80 | |
| Signal Delay at 100 Hz | 3.73 | 1 | 3.73 | ++37.30 | 0.66 |
| Signal Delay at 200 Hz | 0.79 | 1 | 0.79 | ++ 7.90 | 0.13 |
| Signal Delay at 300 Hz | 0.87 | 1 | 0.87 | ++ 8.70 | 0.14 |
| Frequency | 3.14 | 2 | 1.57 | **15.70 | |
| Frequency at 0.0 sec | 0.84 | 2 | 0.42 | 4.20 | |
| Frequency at 0.5 sec | 3.01 | 2 | 1.50 | +15.00 | 0.71 |
| Signal Delay x Frequency | 0.71 | 2 | 0.36 | * 3.60 | |
| Pooled Error | 2.18 | 22 | 0.10 | | |
| Total | 10.71 | 34 | | | |

* Significant at $p < .05$

** Significant at $p < .01$

Adjusted Confidence Limits:

\+ Significant at $p < .0250$

++ Significant at $p < .0167$

TABLE 6

Summary of Analysis of Variance for Experiment II

| Source of Variation | SS | df | MS | F | $\omega^2$ |
|---|---|---|---|---|---|
| Signal Delay | 0.01 | 1 | 0.01 | 0.02 | |
| Signal Delay x Ss | 0.17 | 2 | 0.08 | | |
| Listening Condition | 1.71 | 1 | 1.71 | **14.25 | 0.59 |
| Listening Condition x Ss | 0.40 | 2 | 0.20 | | |
| Subjects | 4.14 | 2 | 2.07 | | |
| Signal Delay x Listening Condition | 0.18 | 1 | 0.18 | 1.50 | |
| Residual | 0.12 | 2 | 0.06 | | |
| Pooled Error | 0.69 | 6 | 0.12 | | |
| Total | 6.73 | 11 | | | |

** Significant at $p < .01$