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A control systems analysis of electrically induced body sway.

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Introduction

The role of the vestibular apparatus is to contribute to the establishment of upright equilibrium and orientation in man and animals. This was most probably first determined by Flourens who published observations on the semicircular canals of pigeons in 1864 (Wendt, 1951). Engstrom, Lindeman, and Ades (1966, p. 35) described the anatomy of the end-organ as follows:

> In each vestibular organ there are three semicircular canals and two sacs, the utricle and the saccule. The canals and sacs are filled with endolymphatic fluid. Each canal forms a narrow duct which widens to a single ampulla. The ampullae of the anterior vertical and of the horizontal canals are situated close together, while the posterior vertical canal has its ampulla at the other end of the utricle, which forms a tubelike connection between all three semicircular canals. The planes of the canals are almost orthogonal to each other. Each canal has, in its widened ampulla, a crista ampullaris containing the sensory cells and topped by a jellylike cupula. The sensory areas of the utricle and the saccule are found in the utricular macula and the saccular macula upon which the jellylike otoliths rest.

Myelinated nerve fibers synapsing with sensory cells of the vestibular organ form the two division of the vestibular nerve, the utriculo-ampullar and the sacculo-ampullar divisions. The utriculo-ampullar branch is composed of fibers which synapse at the utricular macula and the cristae of both the anterior vertical and the horizontal semicircular canals. The sacculo-ampullar division synapses with the saccular

macula and the crista of the posterior vertical canal (Fischer, 1956).

The fibers comprising the vestibular portion of the eighth cranial nerve pass medialward together, through the bony labyrinth which encases the vestibular end organ, to the vestibular, or Scarpa's, ganglion, and from there to the four vestibular nuclei of the medulla or directly to the cerebellum (Morgan and Stellar, 1950). There is little information regarding the distribution of vestibular fibers within the nuclei and there is some disagreement regarding afferents from the nuclei. However, Brodal (1966) found that the superior and the medial nuclei are supplied chiefly by fibers from the cristae, while fibers from the utricular macula supply the lateral nucleus. The lateral nucleus is the vestibulo-spinal reflex center essential for the maintainance of postural tonus (Elliott, 1963). The medial vestibular nucleus sends axons to nerves controlling lateral eye movements and to the cerebellum (Brodal, 1966). The superior nucleus sends axons to the cerebellum and plays a role in the control of vertical eye movements (House and Pansky, 1960). The inferior nucleus has contact with the cervical cord for movement of the head and upper trunk (House and Pansky, 1960). Generally speaking, the vestibular nuclei receive afferents from the same regions to which they give off fibers (Brodal, 1966).

A new dimension has recently been added to vestibular physiology by Gacek (1960) who postulated that the lateral vestibular nucleus is the source of an efferent vestibular component. These efferent fibers are said to pass to Scarpa's ganglion and through the bony labyrinth following the routes of the utriculo-ampullar and the sacculo-ampullar afferent divisions. Efferent endings were found to synapse with sensory cells of the end-organ (Spoendlin, 1966).

Sala (1965) recorded afferent activity at the level of the vestibular nerve and receptors while stimulating in the area of the lateral vestibular nucleus using an electrical square wave. He found an increase followed by a decrease in vestibular resting potential and concluded that afferent activity of some receptors can be modulated through efferent nerve fiber activity. Groen (1960) reached a similar conclusion on a strictly theoretical basis by postulating that the inhibition of vestibular responding is attributable to the efferent impulses acting to suppress vestibular information of too long a duration..

Operationally, it is assumed that the vestibular apparatus is composed of two relatively separate systems. The semicircular canals (cupula-endolymph system) are sensitive to angular acceleration, each canal responding maximally to acceleration in its own plane. For the horizontal semicircular canals, excitation occurs when the ampulla follows the canal during angular accelerations, the stimulus

being cupula displacement caused by inertia movement of the endolymph toward the ampulla. Excitation of the vertical canals is effected by angular acceleration in which the ampulla is leading, the stimulus being cupula displacement caused by inertia movements of the endolymph away from the ampulla (Lowenstein and Sand, 1940). The utricle and the saccule (otolith system) are, according to Lowenstein (1966), "fundamentally capable" of responding to angular and linear accelerations, as well as to angular rotation at constant velocity and to vibration, but the otolith system is most sensitive to linear acceleration.

Investigators have attempted to describe the operations of the two systems comprising the vestibular apparatus by mechanico-mathematical models since Steinhausen (1933) showed that the cupula-endolymph system may be represented as a heavily-damped torsion pendulum. The characteristic equation of the system was defined by Egmond, Groen, and Jongkees (1949) as

$$\frac{d^2\xi}{dt^2} + \frac{d\xi}{dt}\,\frac{\text{Ⅱ}}{\text{Ⓘ}} + \frac{\Delta}{\text{Ⓘ}}\,\xi = 0$$

where

Ⓘ = moment of inertia of endolymph

Ⅱ = moment of friction at unit angular velocity

Δ = directional momentum at unit angle caused by cupula deflection

ξ = angular displacement of endolymph in relation to the skull

This equation considers stimulation of only one of the three semicircular canals on each side of the head and the response measures used in determining its accuracy were subjective report and nystagmus.

More recent models of the vestibular apparatus enlisted a control systems approach, but still were based on the damped torsion pendulum equation. Hixson and Niven (1961) used two transfer functions to describe the cupula-endolymph system. The first was the ratio of cupula displacement (output) to angular acceleration (input), and the second was the ratio of eye movement velocity during nystagmus (output) to cupula displacement (input). Their method of stimulation was whole body angular acceleration which stimulated more than one semicircular canal and nystagmus was the response measure (Niven and Hixson, 1961). Jones and Milsum (1965) also developed a transfer function for the cupula-endolymph system but considered stimulation of only one semicircular canal with nystagmus as output.

Young, Meiry, and Li (1966) presented a model for the otolith system. The form of the transfer function obtained was identical to the torsion pendulum model for the semicircular canals. Subjects were exposed to whole body linear accelerations and the response measures were subjective report and nustagmus. It was concluded that the otolith system is a linear "velocity indicator" in the same sense that the cupula-endolymph system is an angular "velocity

indicator". The systems act as velocity indicators insofar as they are sensitive to accelerative and decelerative forces involved in velocity.

The loci of stimulation on which the basic torsion pendulum model and the transfer functions are based are more limited than those found under normal conditions. The otolith and the cupula-endolymph systems are constantly being simultaneously activated in response to gravity-induced forces combined with angular and other positional movements of the head. Furthermore, a common response to normal stimulation is whole body movement. Therefore, the various transfer functions seem to be of limited practical applicability. The continuous operation of the vestibular apparatus as a whole should be recognized and methods should be devised which would enable researchers to work with the sense on a more realistic basis.

Sinusoidal electrical stimulation of the vestibular apparatus is an approach which may meet these requirements. This form of stimulation was first used by Barnett and Posner (1941). They impressed sinusoidally-varying, low-level current across the mastoid processes of humans via bilateral electrodes. The standing subject reported sensations of lateral oscillation, localizable in the head, trunk, or down to the thighs when he was stimulated by frequencies of up to 5.0 Hz. These sensations were reported to vary as the frequency of stimulating current was varied. The sub-

jective threshold curve of current level as a function of frequency was U-shaped with a minimum at 1.5 Hz.

Spiegel and Scala (1943) investigated the site of action of d-c electrical stimulation and concluded that the current acts on the peripheral vestibular neuron. The response which they obtained, when subjecting cats to monaural stimulation ranging from 2.0 to 15.0 ma using a bipolar electrode, was rotation of the head about the oro-occipital axis toward the nonstimulated ear. Binaural stimulation within the same range of current produced similar head rotation toward the anode and foreleg extension on the cathodic side. The same responses also were obtained when a-c electrical stimulation was used. Spiegel and Scala claimed that cathodic stimulation increased muscle tonus while anodic stimulation decreased it. Therefore, it seems that sway would be produced by increasing muscle tonus on one side of the body while decreasing it on the other, in an alternating fashion.

The more recent work of Fredrickson, Schwarz, and Kornhuber (1966) partially supported the findings of Spiegel and Scala. They stimulated the vestibular apparatus of cats binaurally using direct current of 0.05 to 0.12 ma and recorded from the area of the medial and descending vestibular nuclei, obtaining direction-dependent and direction-independent responses. The most common direction-dependent responses consisted of ipsilateral cathodic

activation and anodic inhibition with the opposite response occurring contralaterally. These responses of the vestibular nuclei to ipsilateral cathodic stimulation were found to be the same as those produced by stimulation of the horizontal semicircular canal using ipsilateral rotational acceleration about the vertical axis. Neurons were also found which responded in the same way bilaterally with cathodic stimulation yielding activation and anodic producing inhibition at the level of the vestibular nuclei. These were the responses similar to those reported by Spiegel and Scala. Fredrickson et al. postulated that this is the type of neuronal response one might expect from the otolith system. Direction-dependent responses were characterized by neuronal discharge regardless of the direction of polarizing current.

In order to continue this work in the area of behavioral responses, Dzendolet (1963) developed a technique for obtaining objective measures of lateral sway from humans exposed to sinusoidal electrical stimulation of the vestibular apparatus. This approach was used to avoid the necessity of relying on subjective reports as Barnett and Posner had done. Stimulation was induced via bilateral mastoid electrodes of the fluid Ag^{-}-AgCl-Cl^{-} type. Sway transduction was effected by means of a potentiometer connected to a headpiece worn by the subject. The potentiometer served as a voltage divider and deviations of its wiper from a zero position allowed current to flow, the magnitude changes of

which were recorded. The potentiometer wiper was affected only by lateral movements of the subject.

Dzendolet found the curves of simultaneously-recorded subjective and objective thresholds to be similar in shape, but the latter were lower. The results agreed with those of Barnett and Posner between stimulating frequencies of 0.5 and 4.0 Hz. The subjects gave qualitative reports of antero-posterior, as well as lateral, sway. Dzendolet agreed with Spiegel and Scala that the site of electrical stimulation is not the end-organ itself, but rather the peripheral vestibular neuron. This conclusion was based on the fact that both antero-posterior and lateral sway could be induced, implying the stimulation of different fibers yielding different sway responses.

On the basis of present evidence regarding electrical vestibular stimulation and vestibular anatomy, it seems likely that there is simultaneous stimulation of nerve fibers from at least one semicircular canal and one of the otoliths. The possible excitation of efferent, as well as afferent, fibers must be recognized. If Groen's postulation regarding the role of the efferents as inhibitory agents is correct, it is possible that the higher the current level, the greater is the participation of efferent, as well as afferent, fibers in the response. This would help to account for the differences in responses obtained by Spiegel and Scala and Fredrickson et al.

The present experiment employed sinusoidal electrical stimulation of the vestibular apparatus recognizing the possibility that both the cupula-endolymph and the otolith systems were thereby simultaneously stimulated along with efferent and afferent fibers. Body sway responses of humans were analyzed using a control systems approach. Sway output was assumed to be a continuous statistical signal rather than a periodic one because input-output relations have not yet been determined for stimulation of the vestibular apparatus as a whole. Systems analysis of such continuous signals involves the power spectral density (PSD) and the autocorrelation (ACF) functions. According to Milsum (1966), the former is a mathematical measure used to define a waveform when it cannot be broken down into a discrete set of harmonics or when it is constantly changing in an unpredictable manner. Power spectral density is expressed as power per unit frequency, where the "power" unit is proportional to signal amplitude squared. Therefore, frequency is on the abscissa and PSD value, expressed as watts/Hz, is on the ordinate. The total power in a signal equals the area which results from integrating the curve over all frequencies considered. This is expressed in watts.

The autocorrelation function is defined by Milsum (1966) as being the expected, or mean, value of the product of the signal at time t and its time-shifted value at time $t-\tau$, where τ is time shift or lag. The autocorrelation function identifies

waveform periodicities by correlation of output with itself at a later time. Time shift is on the abscissa and the value of the correlation is on the ordinate. The power spectral density and the autocorrelation functions are Fourier transforms of each other and contain the same mathematical information. However, they accentuate different aspects of a waveform since one is in the frequency domain and the other is in the time domain (Milsum, 1966). The crosscorrelation function (CCF) is a similar tool. It differs from the autocorrelation function in that the correlations are performed between two distinct sets of data (Bahrick and Noble, 1966).

The purpose of the present experiment was two-fold:

1. To specify better the effects of electrical stimulation of the vestibular apparatus on body sway by use of control systems analysis.

2. To determine whether or not the frequency of sway output could be varied as a function of electrical input frequency.

Method

Subjects - These were six undergraduate males enrolled at the University of Massachusetts. The subjects (Ss) were selected from a group of volunteers such that no S had had any type of head injury, muscle or bone injury to the legs or feet, a history of fainting spells, or a recent illness. No one taking any medication other than multipurpose vitamins was selected. The form of stimulation was described to each S and he was free to withdraw from the experiment at any time. The Ss received monetary reimbursement for their services.

Apparatus - The basic component of the stimulus-producing system was a function generator, the output of which was connected to S via a selection switch and two electrodes. There were two 5.0 ma fuses in each electrode lead. A 10 Ω resistor was placed in series with the generator so that stimulus current level could be obtained. A three-channel polygraph was also in series with the function generator and S. One channel of the polygraph was used to record the voltage output of the generator.

The stimulus was a sinusoidally-varying current produced by the low-level function generator (Hewlett Packard, Model 202A). Four frequencies which were previously found to affect sway (Dzendolet, 1963; Moore, 1965) were used. These were 0.1, 0.2,

0.5, and 1.0 Hz. The levels of stimulating current were 0.005 and 0.05 ma, zero to peak. These values were determined to be below skin pain threshold in a pilot study.

Two stimulating electrodes[1] like that shown in Fig. 1 were placed on S's mastoid processes. The electrodes were made of a piece of clear plastic 25.40 mm long and 19.05 mm in diameter. The front view in Fig. 1 shows the area of the electrode which rested on the skin. The outer circle was 1.60 mm wide and 3.45 mm deep. The inner circle was 6.35 mm in diameter and 3.45 mm deep. "Fine" silver wire was put into the filler holes of each electrode. This silver wire was connected to the leads from the switch. The part of the electrode which touched the skin, indicated by the shaded areas in the figure, was cushioned with a thin layer of rubber to prevent skin abrasions. Saturated NaCl solution was the electrolyte used in the inner and outer circles. The electrodes were attached to a cloth head-band via spring-loaded metal plates. The springs exerted the pressure necessary to keep the electrodes firmly against the skin.

Between the function generator and the stimulating electrodes was a double-pole, triple-throw switch which allowed current to be passed through either or both electrodes. When only one electrode was used, the inner circle was one pole and the outer the other. When both electrodes were in use, the inner circles only were the poles.

The sway transducer was a square platform of 3/4 in.

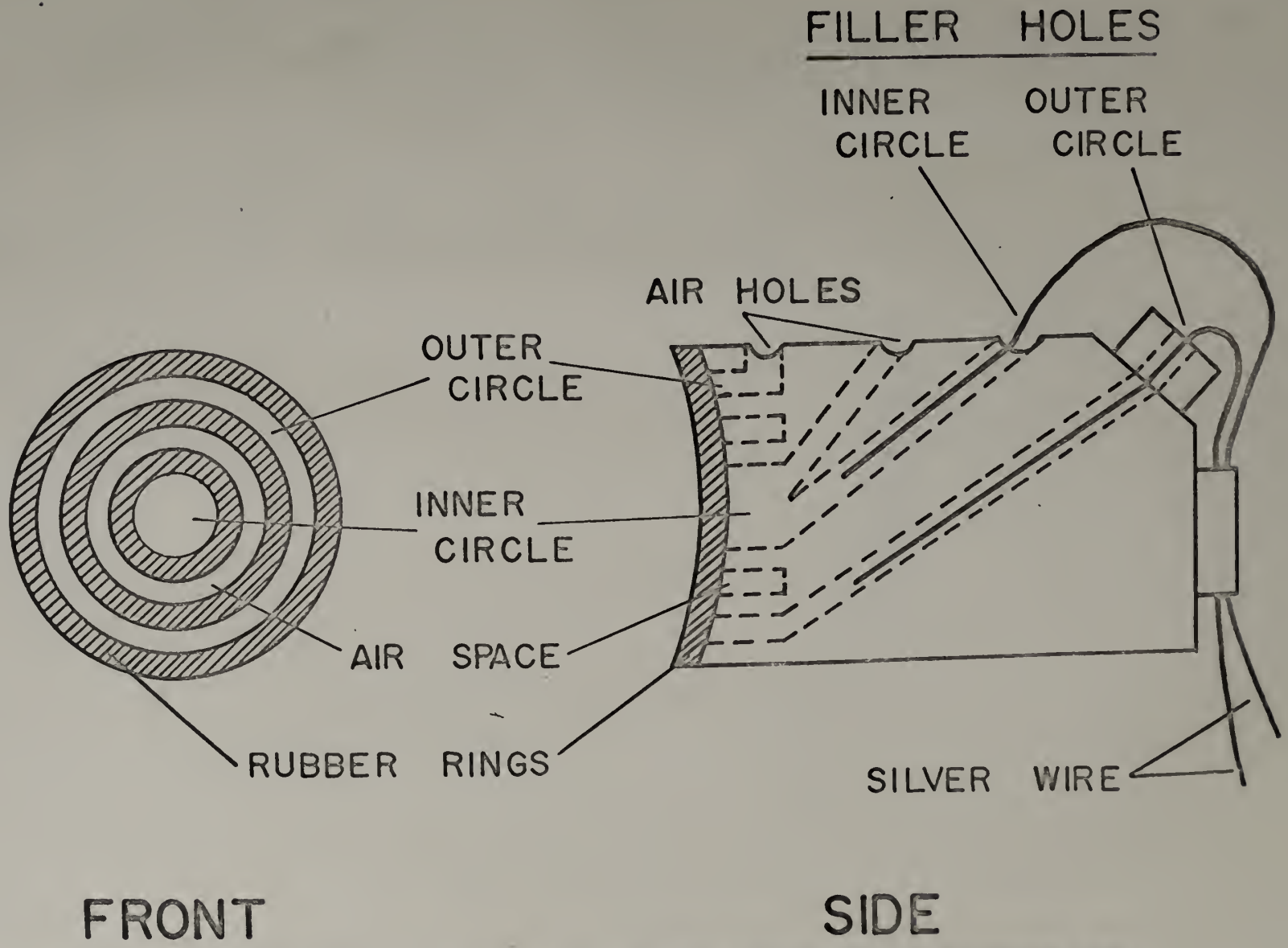

Fig. 1. Details of the construction of the plastic concentric electrodes used to stimulate Ss.

plywood supported at the center of each 27 1/4 in. long side by the end of each of four, short, horizontally-positioned, steel bars. The ends of the bars extended under the platform and made contact with it by means of machine screws which firmly attached the platform to the bars. The other end of each bar was rigidly fastened to a steel framework below the platform. Strain gauges were applied to all the bars. The two gauges on opposing bars were made part of a Wheatstone bridge circuit. Therefore, a force applied at any point except at the center of the platform, along a line joining two opposing bars, created an imbalance in the bridge circuit. The same situation held for any force with respect to the other two strain gauges. The overall characteristics of the platform were such that no sensation of movement or rocking occurred if S shifted his weight.

The outputs of the separate bridge circuits were led into preamplifiers (Grass Instrument, Model 5P1), and displayed on separate channels of a polygraph (Grass Instrument, Model 5). The output of the function generator was recorded on a third channel of the polygraph. Force on any portion of the platform was resolved into two, mutually perpendicular forces, the amplitudes of which were displayed on separate channels. With proper positioning of S, these two amplitudes were the results of S's swaying in the antero-posterior and in the lateral directions.

Procedure - At the start of the session, the seated S

was blindfolded and insert ear phones were placed in both ears. These were used to deliver white masking noise at 70 dB SPL. The head-band and electrodes were then placed so that the electrodes pressed firmly against each mastoid process. Saturated NaCl solution was injected into each filler hole by means of a blunted hypodermic needle. The following instructions were then read to S:

> Your task during this experiment is to stand on the platform. Stand without moving your feet or legs once their position has been set, and also without moving your hands, arms, or head. Please clasp your hands together and let them hang limply in front of you. Do not stand rigidly as if at attention. It is important that you relax. But relax without moving your feet or legs, your arms or your head. Also keep your weight evenly distributed on both legs.
>
> After each trial, you will be given a short rest during which you may shift your weight to one leg or unclasp your hands. However, do not move your feet. You will also have a 10 minute break sometime during the session during which time you may get off the platform and sit down. You will be told when these breaks occur. Are there any questions?

The S then stood on the platform and the experimenter guided him in positioning his feet in the approximate center of the platform with heels together and feet at about 45° to each other. Each trial consisted of a 30 sec prestimulus period (T_1) followed by a 30 sec stimulus-on period (T_2). This was followed by a 1 min break. The Ss were given a 10 min rest in the middle of each session.

During each session, S was stimulated with the two current levels at each of the four frequencies, but only one of the three electrode locations was used. The locations of

stimulation were right mastoid, left mastoid, and both mastoids together. The presentation of these variables was completely randomized. Each S was run on three separate days. The experimental design was of the form: Ss (1-6) by Electrode Location (right, left, and both mastoids) by Stimulus Frequency (0.1, 0.2, 0.5, and 1.0 Hz) by Current Level (0.005 and 0.05 ma) by Time Period (T_1 and T_2).

The sensitivity of the polygraph preamplifier was 0.05 mv/cm for all Ss. The system was calibrated by placing a 2.0 kg mass at 25.0 cm from the center of the platform. This was done for all four sides of the platform. This mass produced a pen deflection of 1.0 cm with preamplifier sensitivity set at 0.02 mv/cm.

The polygraph records of sway were scored every 0.2 sec in terms of millimeters of positive or negative pen deflections from an arbitrary baseline. Therefore, there were 150 observations or data points per 30 sec period. The analyses performed on the antero-posterior and the lateral sway separately were: (1) the calculation of the autocorrelation function (ACF). (2) the calculation of the power spectral density function (PSD), and (3) various analyses of variance. The crosscorrelation function (CCF) was calculated to determine the relationships between the two directions of sway. The different analyses will be discussed in detail below. The raw data for the analyses of variance were the arithmetic mean sway amplitudes of the prestimulus and the

stimulus-on periods. The means were obtained by summing over the absolute values of sway changes per 0.2 sec periods. The raw data for the ACF's, PSD's, and CCF's were the scored sway records.

Because the time between successive observations was 0.2 sec, the value of the time shift or lag, τ, in the computation of the ACF's was increased from 0 sec in steps of 0.2 sec. The number of lags extended from 0 to 71. Therefore, the value of the longest lag was 0.2 sec times 71, or 14.2 sec.

The highest frequency for which PSD's could be accurately computed was 2.5 Hz as determined from the Nyquist Sampling Theorem:

$$\# \text{ samples} = 2WT$$

where

W = maximum frequency

T = length of observation period (30 sec)

samples = 150

The number of PSD points obtained was 72. The value of each frequency increment was 0.0347 Hz according to the formula:

$$f_k = \frac{W}{P}$$

where

f_k = frequency increment

P = number of PSD points obtained

The overall power in the output was obtained by summing over the PSD values at each frequency computed and multiplying

this sum by the frequency increment, 0.0347 Hz.

The crosscorrelation function (CCF) was used to determine the relationship among the following measures: antero-posterior sway vs. lateral sway, antero-posterior sway vs. stimulus input, lateral sway vs. stimulus input. The number of lags again extended from 0 to 71 and the value of lag, τ, from 0 to 14.2 sec. For a given lag, correlations were computed for all combinations of the nonlagged and the lagged measures. One measure was held constant while the other two were lagged separately. Therefore, six combinations of relationships among measures were obtained.

Results

Sway Amplitude

A separate analysis of variance was performed on each of the two directions of sway recorded in the present experiment because antero-posterior and lateral sway were assumed to be independent processes. The analyses of variance were both completely factorial of the form: Ss (1-6) by Electrode Location (right, left, and both mastoids) by Stimulus Frequency (0.1, 0.2, 0.5, and 1.0 Hz) by Current Level (0.005 and 0.05 ma) by Time Period (30 sec prestimulus (T_1) and 30 sec stimulus-on (T_2) periods). The analyses of variance tables are presented in Appendix A and Appendix B.

Antero-Posterior Sway - The analysis of variance performed on the antero-posterior sway data yielded one significant source of variance. This was the second-order interaction of Stimulus Frequency by Current Level by Time Period ($F(3,15)=11.04$, $p<.001$). A histogram of the interaction is presented in Fig. 2. Sway amplitude during stimulation at 0.005 ma was generally greater than that during stimulation at 0.05 ma. Amplitude during stimulation at the lower current level increased with frequency while the opposite held for the higher current level. The means during T_1 were very similar for trials at either current level, being 1.06 mm during 0.005 ma trials and 1.08 mm during those at 0.05 ma. This was as expected since T_1

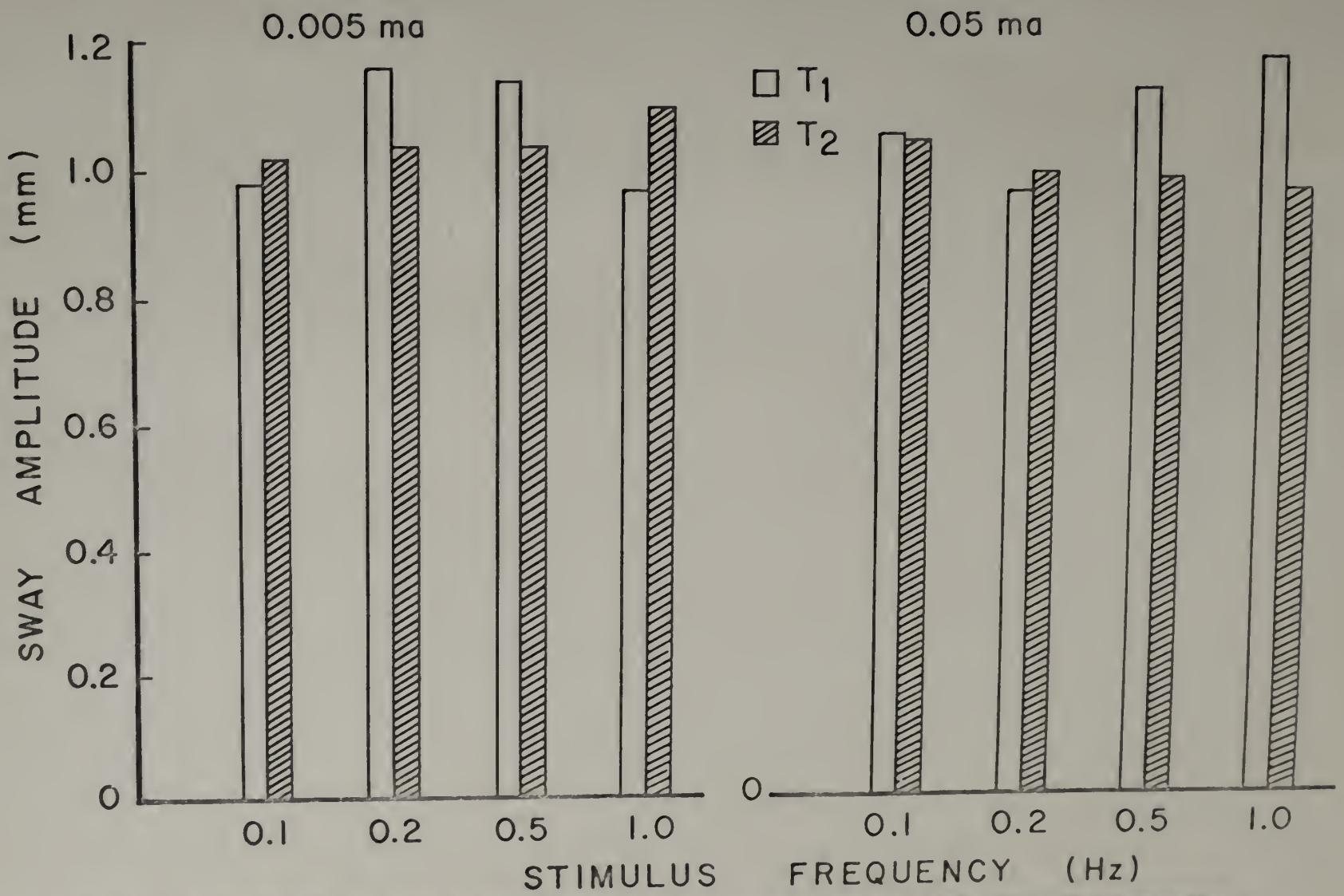

Fig. 2. Histogram of the significant relationship among stimulus frequencies, current levels, and time periods for antero-posterior sway amplitude.

served as a control period. Sway amplitudes during stimulation were slightly less than those during T_1. The mean amplitude during stimulation at 0.005 ma was 1.05 mm and, during stimulation at 0.05 ma, was 1.00 mm. Therefore, stimulation tended to decrease antero-posterior sway amplitude. This effect was heightened as current level was increased.

A plot of the relationships among electrode location, current level, and time period for antero-posterior sway is presented in Fig. 3. The source of variance involving these variables was not significant ($F(2,10)=1.63$). The reason for presenting these results is to compare them with those for lateral sway a little later. It should be noted that the means of the sway amplitudes during T_1 and T_2 at 0.005 ma were very similar at each electrode location. This was also the case at 0.05 ma on trials during which both mastoids were stimulated. However, for 0.05 ma trials during which only one or the other mastoid was stimulated, the mean amplitude of sway during T_1 was greater than that during T_2. Mean sway amplitude during stimulation of the right mastoid was greater than during stimulation of the left on 0.05 ma trials. There was only a slight difference between these on 0.005 ma trials.

The overall mean antero-posterior sway amplitude was 1.05 mm with the mean amplitudes of the individual Ss ranging from 0.75 to 1.30 mm. There was a slight tendency for sway during T_1 to be greater than that during T_2. The mean

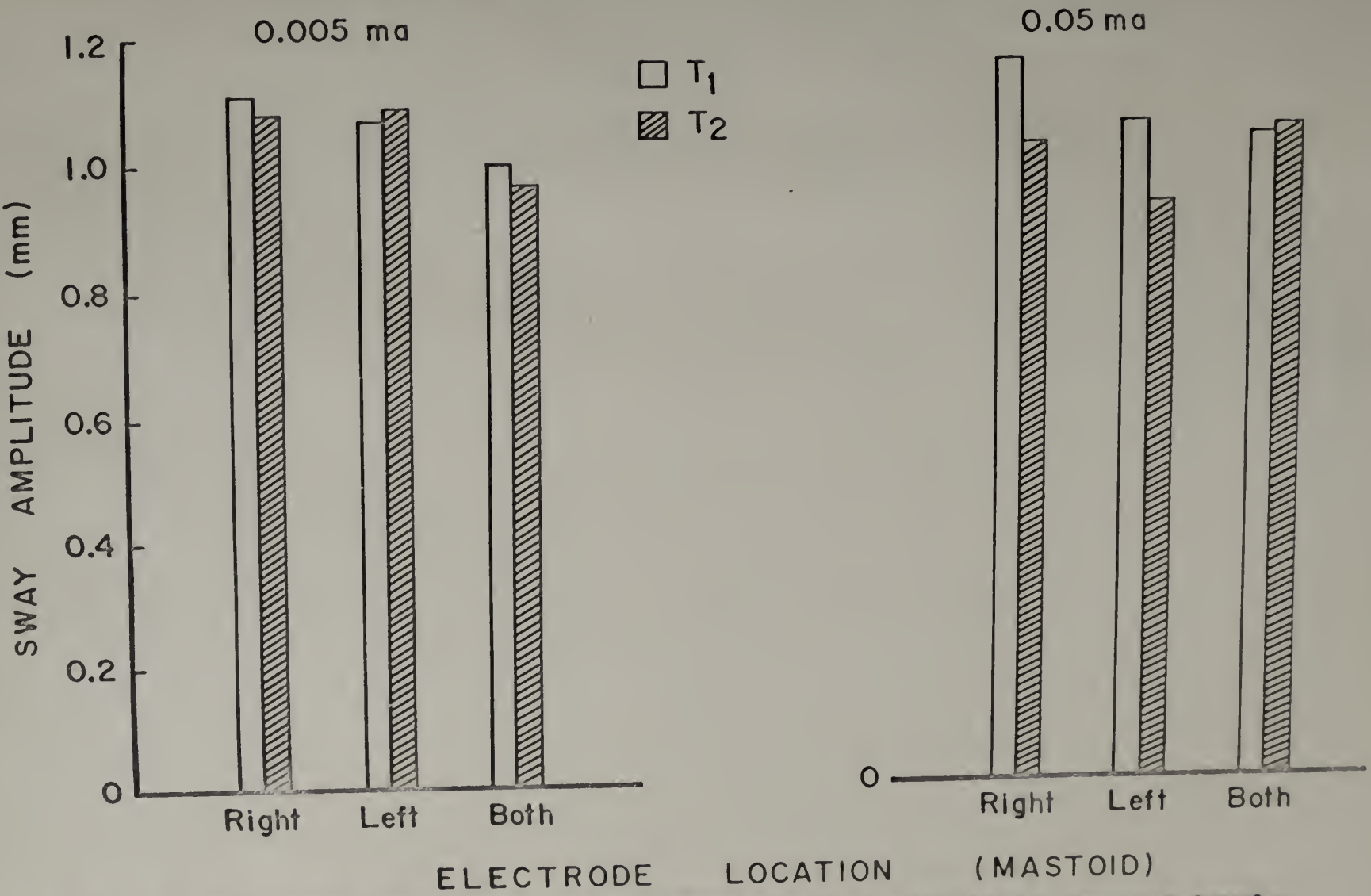

Fig. 3. Histogram of the relationship among electrode locations, current levels, and time periods for antero-posterior sway amplitude.

amplitude during the former was 1.07 mm and, during the latter, was 1.03 mm.

Lateral Sway - The Stimulus Frequency by Current Level by Time Period interaction was also significant in the analysis of variance performed on the lateral sway amplitude data ($F(3,15)=4.32$, $p<.025$). A histogram of this interaction (Fig. 4) is similar on 0.005 ma trials to that obtained for antero-posterior sway. However, at 0.05 ma of stimulation, sway amplitude did not decrease as stimulus frequency increased. Instead, mean amplitudes were similar at all frequencies with the exception of 0.2 Hz, where amplitude increased. The means during T_1 were again very similar being 0.97 mm for 0.005 ma trials and 0.98 mm for those at 0.05 ma. Sway amplitude during stimulation was 0.97 mm and 1.00 mm for 0.005 ma and 0.05 ma trials, respectively. Therefore, lateral sway amplitude tended to increase slightly during stimulation with amplitude increasing more during stimulation over its T_1 value as current level was increased.

An analysis of lateral sway also yielded a significant Electrode Location by Current Level by Time Period interaction ($F(2,10)=5.04$, $p<.05$). As can be seen in Fig. 5, the means during the prestimulus periods were very similar being 0.97 mm on 0.005 ma trials and 0.98 mm on 0.05 ma trials. Similarly, mean sway amplitude during stimulation at 0.005 ma was 0.97 mm. However, during stimulation at

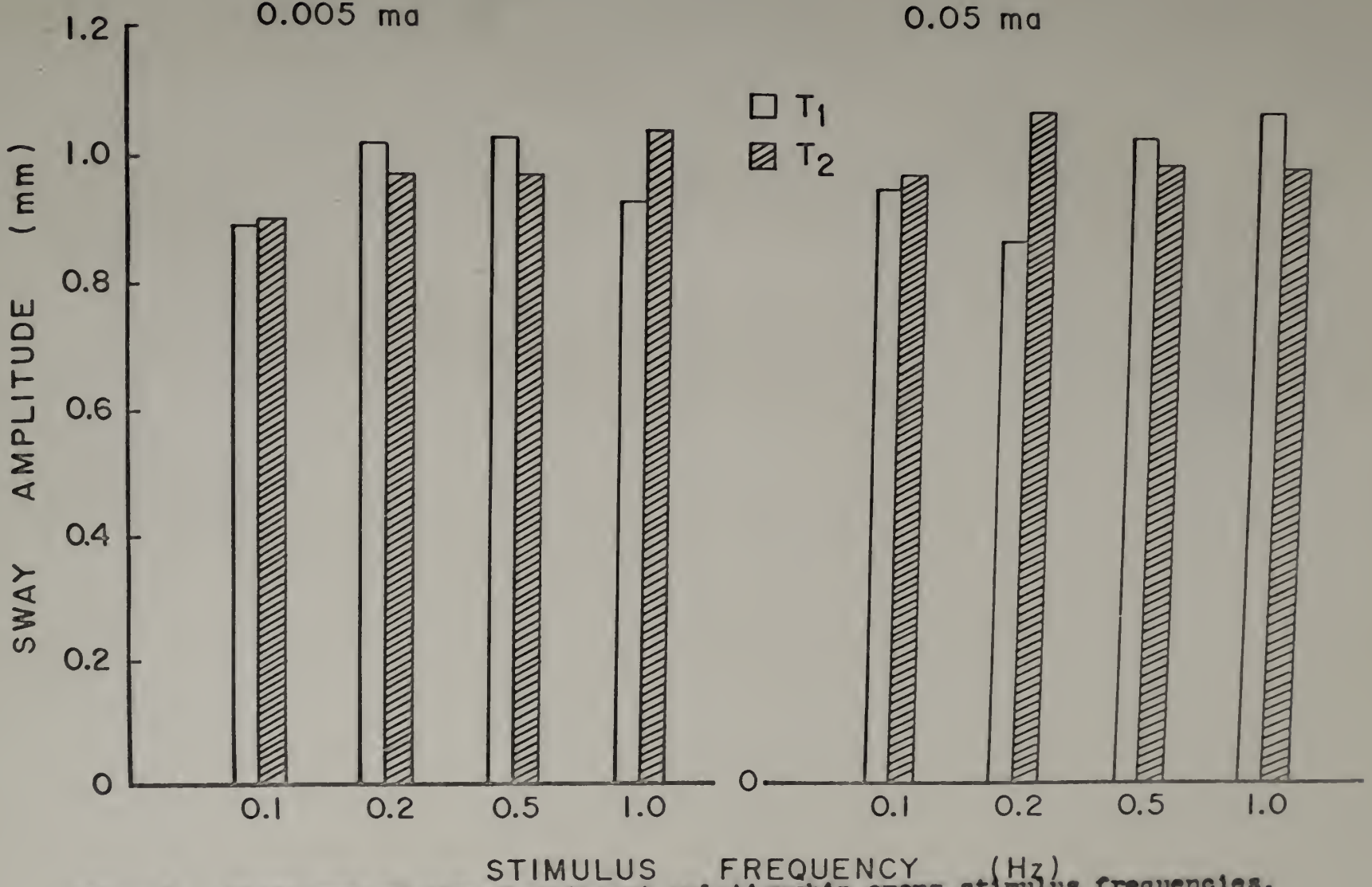

Fig. 4. Histogram of the significant relationship among stimulus frequencies, current levels, and time periods for lateral sway amplitude.

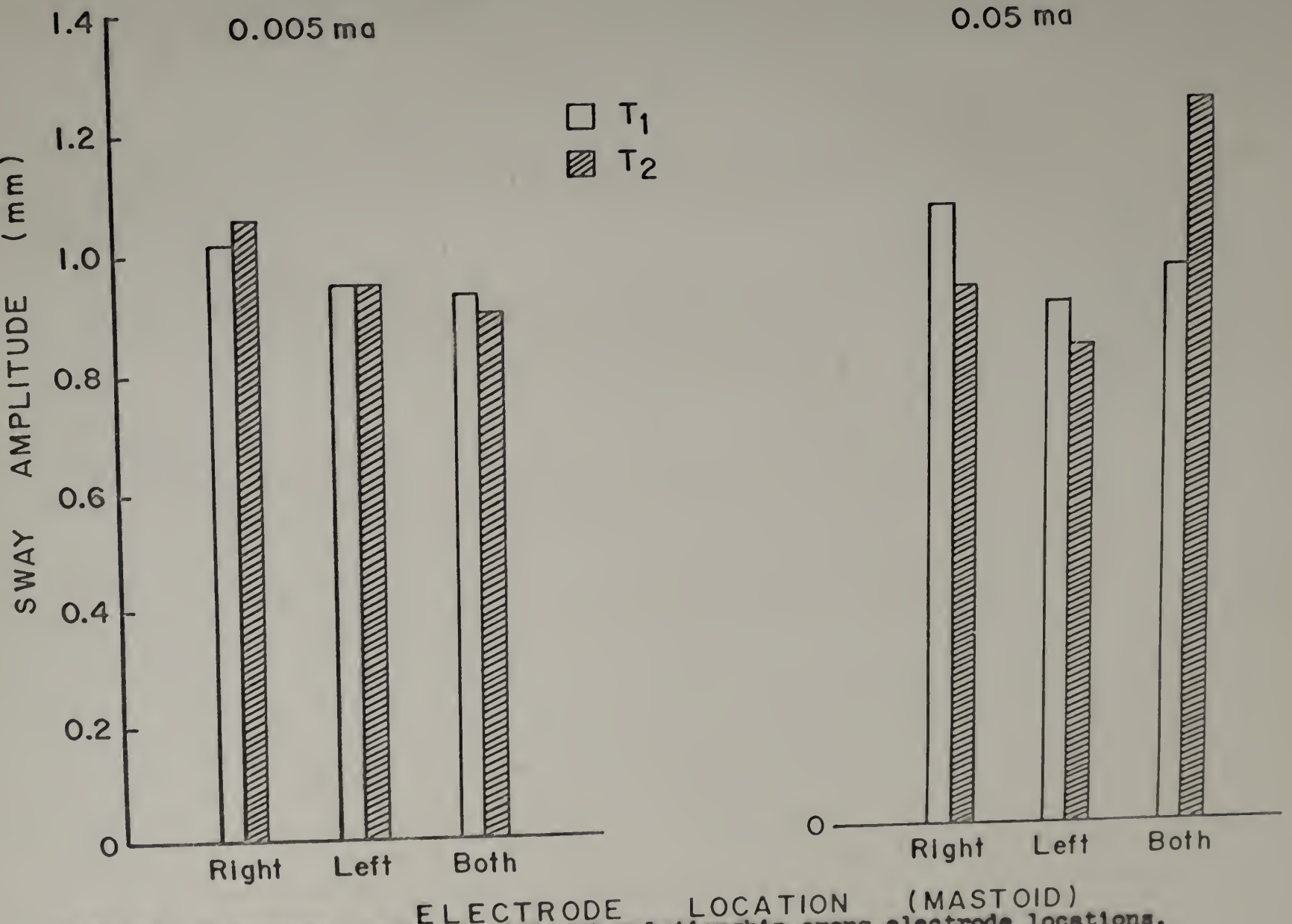

Fig. 5. Histogram of the significant relationship among electrode locations, current levels, and time periods for lateral sway amplitude.

0.05 ma, sway amplitude was less than that during the prestimulus periods when either the right or left mastoids were stimulated. When both mastoids were simultaneously stimulated at 0.05 ma, lateral sway amplitude increased greatly over that during the prestimulus period. Mean sway amplitude during stimulation of the left mastoid was less than that during stimulation of the right on both 0.005 and 0.05 ma trials.

Mean lateral sway amplitude was 0.98 mm with the means of the Ss ranging from 0.71 to 1.14 mm. Mean sway amplitude during T_1 was 0.97 mm and, during T_2, was 0.99 mm.

Sway Frequency

Autocorrelation functions and power spectral densities were obtained for all Ss individually with antero-posterior and lateral sway being analyzed separately. To obtain information regarding the effects of the electrical input on the frequency of sway output, mean PSD's were obtained by summing over Ss. It is these mean PSD's which are presented here as most relevant to the purposes of the present experiment, since they allow the frequencies represented in the sway to be readily identified. The ACF's will not be presented since they contain the same information as the PSD's, but in a different form. Furthermore, only the power in the sway lying at frequencies from 0.0 through 1.5 Hz will be presented since power

at the frequencies beyond 1.5 Hz was negligible.

Antero-Posterior Sway - Regarding the mean PSD's obtained from the antero-posterior sway data, for both the prestimulus (T_1) and the stimulus-on (T_2) time periods, the highest values were found to lie between 0.0 Hz, or d-c, and 0.2 Hz with the values of PSD's at 0.0 Hz ranging from 70 to 250 watts/Hz. At frequencies beyond 0.2 Hz, PSD's fell continuously toward 0.0 watts/Hz. There was a slight indication that the power in antero-posterior sway was affected by the frequency of electrical input. On 0.1 Hz stimulation trials, the power of the sway based on mean PSD's, obtained by summing over current levels, was greater during T_2 than during T_1, being 24.95 and 14.21 watts, respectively. This also held at 0.2 Hz where power during T_1 was 19.44 watts, while, during T_2, it was 23.28 watts. During 0.5 and 1.0 Hz stimulation trials, power during T_1 was greater than that during T_2. For 0.5 Hz trials, power was 19.08 watts during T_1 and 15.99 watts during T_2. The values were 25.57 watts during T_1 and 21.67 watts during T_2 for 1.0 Hz trials.

The presence of the stimulating current and its levels also had a slight affect on the power of the antero-posterior sway. Power during T_1 on 0.005 ma trials was 18.94 watts and, during T_2, was 20.69 watts. For 0.05 ma trials, power during T_1 was 20.21 watts and, during T_2, was 22.75 watts. With regard to electrode location,

the power in the antero-posterior sway was greater when the left or both mastoids were stimulated than when the right mastoid was stimulated. The values of power based on mean PSD's were 22.35 watts during stimulation of the left mastoid, 23.39 watts during stimulation of both, and 19.44 watts during stimulation of the right mastoid. Power during trials on which the left or both mastoids were stimulated was greater during T_2 than during T_1. The opposite held for right mastoid stimulation.

The PSD's obtained for antero-posterior sway gave no indication of changes in the frequency of sway as a function of the frequency of electrical input. Figs. 6 through 9 are the PSD curves. These were plotted on log-log coordinates with the current level being 0.05 ma and the electrodes on both mastoids. It is evident that the power computed over all frequencies considered was greater during T_2 than during T_1 with the exception of 1.0 Hz trials, when the current level was 0.05 ma.

Lateral Sway - The mean PSD's obtained from lateral sway were generally lower than those for antero-posterior sway with the range being from 25 to 100 watts/Hz for the former at 0.0 Hz. For lateral sway also, most of the power lay between 0.0 and 0.2 Hz. There was no indication that the frequency of sway was affected by stimulation of either the right or the left mastoids singly. However, the frequency of sway output did vary directly as a function

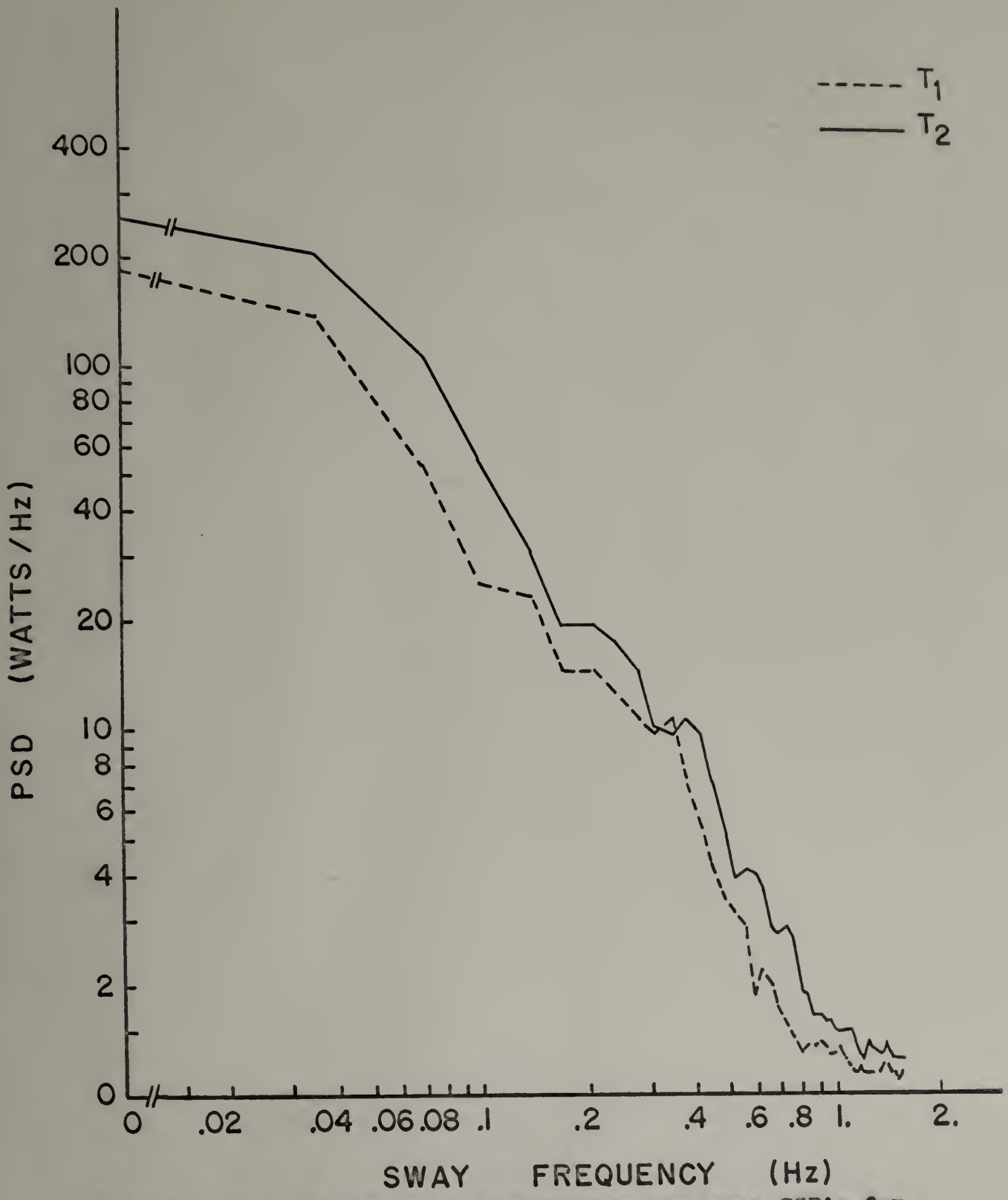

Fig. 6. Plot of the mean antero-posterior sway PSD's for T_1 and T_2 during stimulation of both mastoids at 0.1 Hz and 0.05 ma.

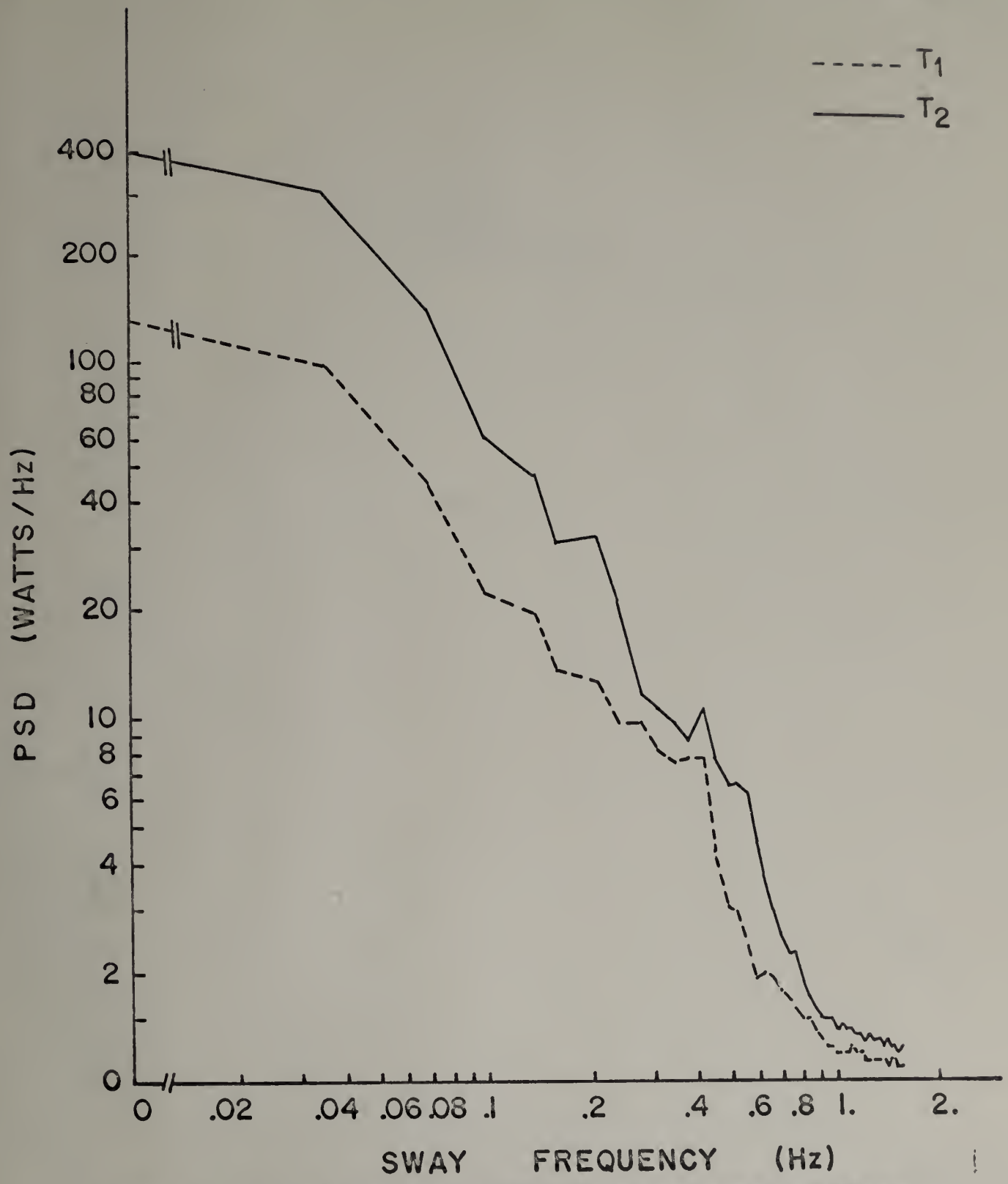

Fig. 7. Plot of the mean antero-posterior sway PSD's for T_1 and T_2 during stimulation of both mastoids at 0.2 Hz and 0.05^2 ma.

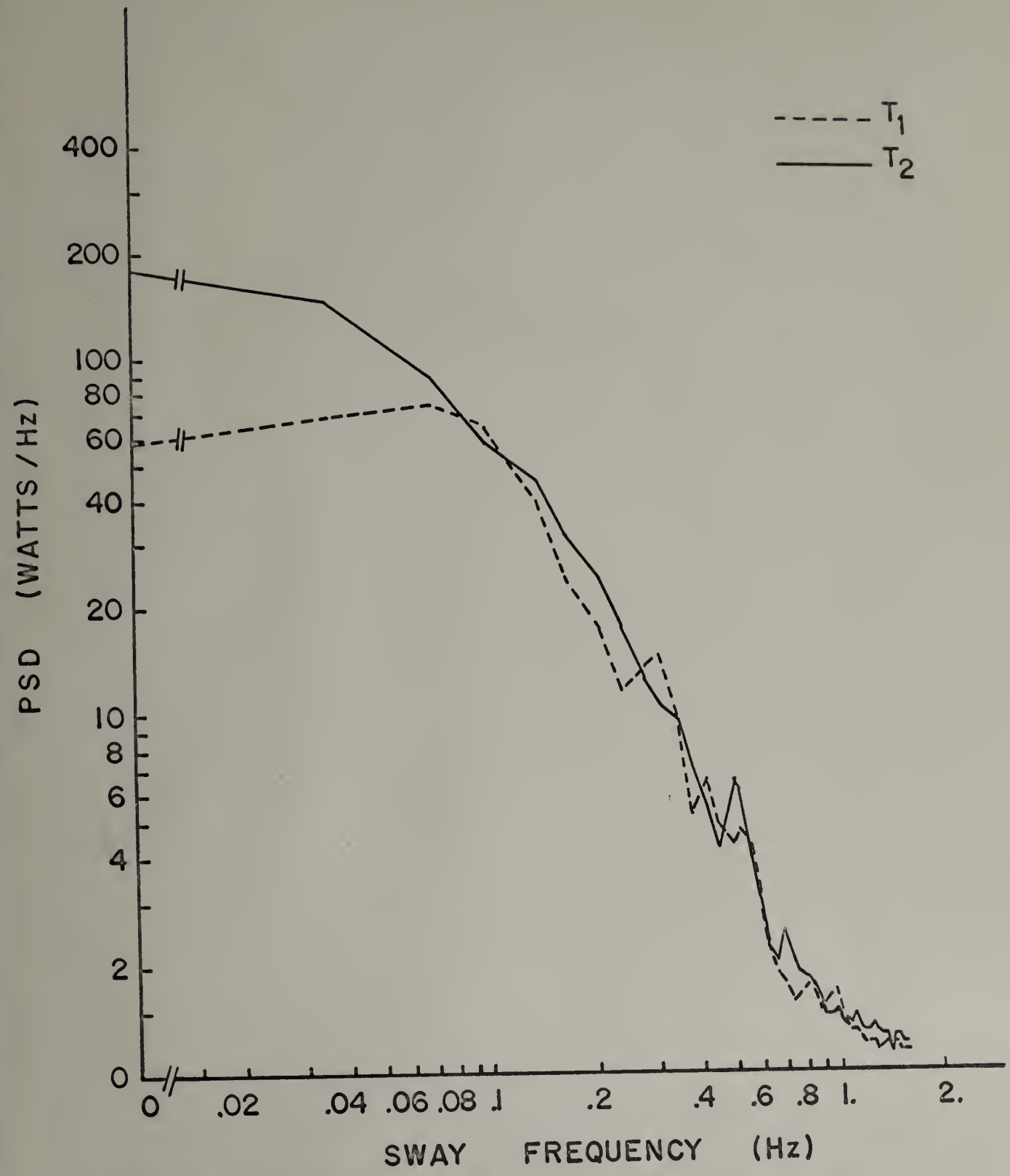

Fig. 8. Plot of the mean antero-posterior sway PSD's for T_1 and T_2 during stimulation of both mastoids at 0.5 Hz and 0.05 ma.

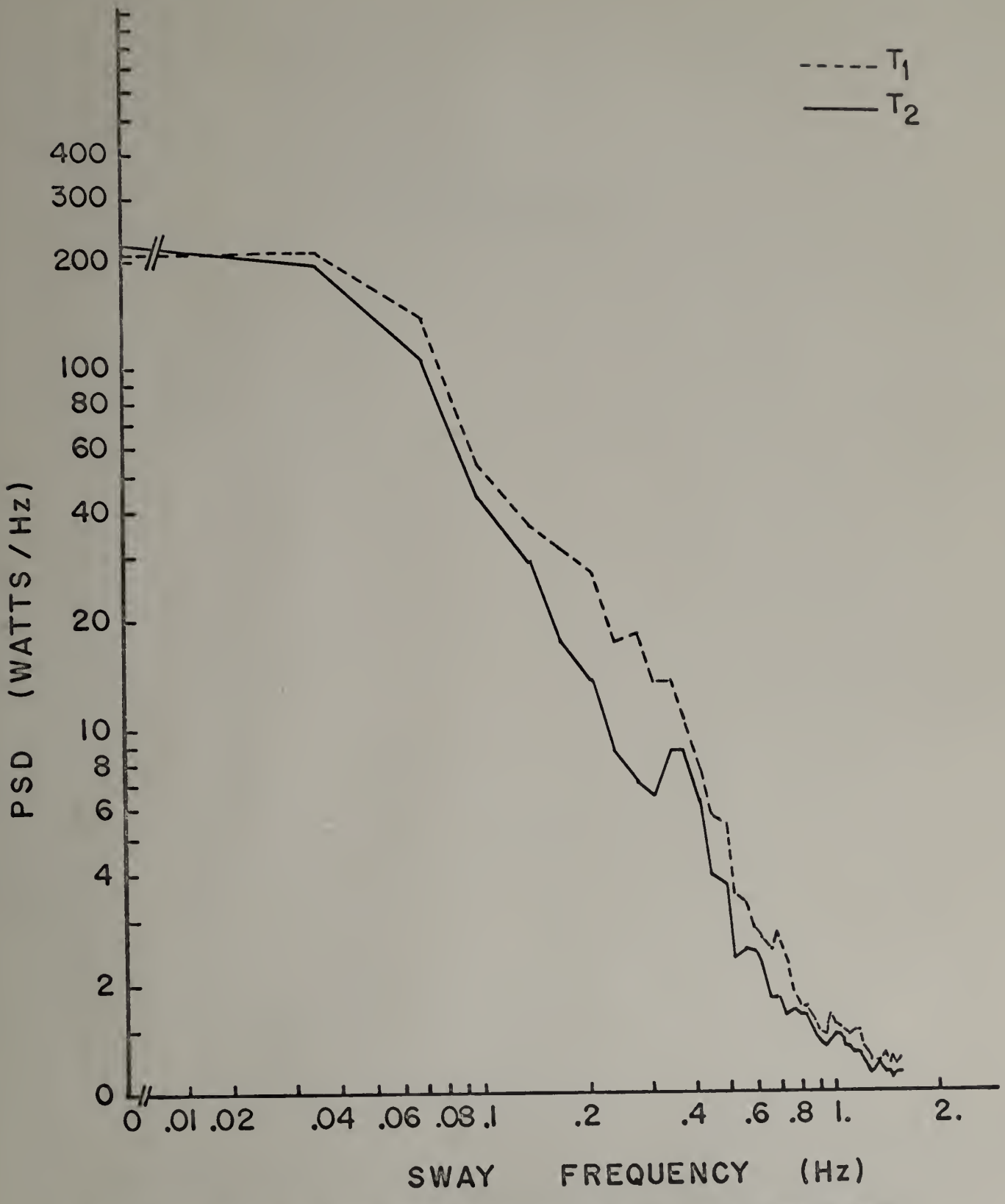

Fig. 9. Plot of the mean antero-posterior sway PSD's for T_1 and T_2 during stimulation of both mastoids at 1.0 Hz and 0.05 ma.

of the frequency of electrical input when both mastoids were simultaneously stimulated at 0.05 ma. Figs. 10 through 13 are log-log plots of these findings. By comparing PSD's during the prestimulus, or control, periods with those of the stimulus-on periods, the extent of the effect of stimulation can be seen to have varied in magnitude depending upon how far removed stimulation frequency was from the normal range of sway frequencies (0.0 to 0.2 Hz). However, even stimulation at 0.5 and 1.0 Hz (Figs. 12-13) did definitely affect sway frequency. It should be noted that the potent electrical stimulation did not change the amount of power in normal sway frequencies, but, instead, increased the amount of power at the stimulation frequency over that which would normally lie there with the exception of 1.0 Hz trials. When both mastoids were stimulated at 0.1 Hz with 0.05 ma, the power based on mean PSD's during T_1 was 6.96 watts while it was 14.96 watts during T_2. At 0.2 Hz, power was 7.67 watts during T_1 and 16.07 watts during T_2. The value of power was 10.48 watts during T_1 at 0.5 Hz stimulation and 12.04 watts during T_2. At 1.0 Hz, the value of power during T_1 was 15.13 watts while it was 13.83 watts during T_2.

When mean PSD's were obtained by summing over Ss, current levels, and electrode locations, there was no definite indication that the power in lateral sway was

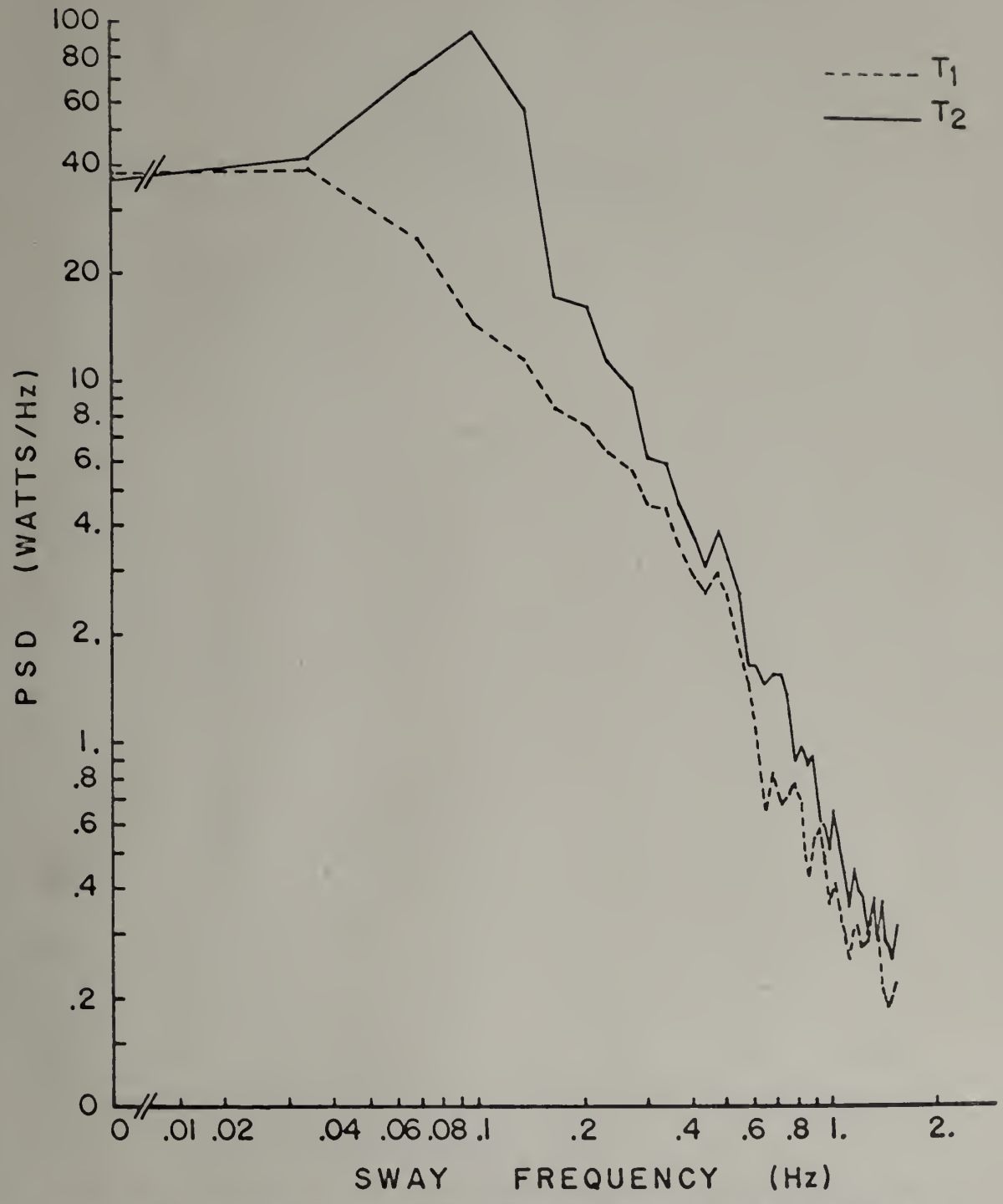

Fig. 10. Plot of the mean lateral sway PSD's for T_1 and T_2 during stimulation of both mastoids at 0.1 Hz and 0.05 ma.

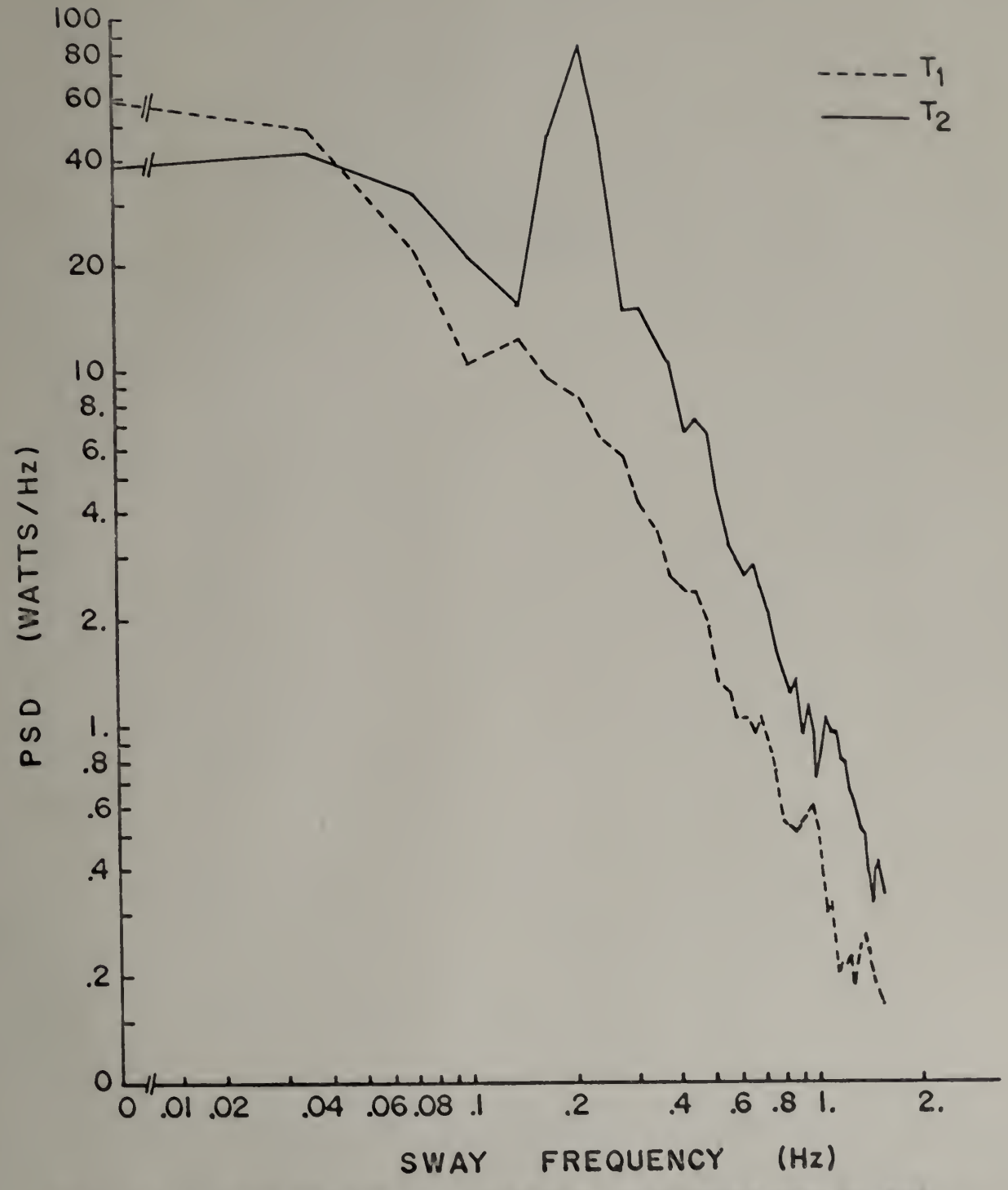

Fig. 11. Plot of the mean lateral sway PSD's for T_1 and T_2 during stimulation of both mastoids at 0.2 Hz and 0.05 ma.

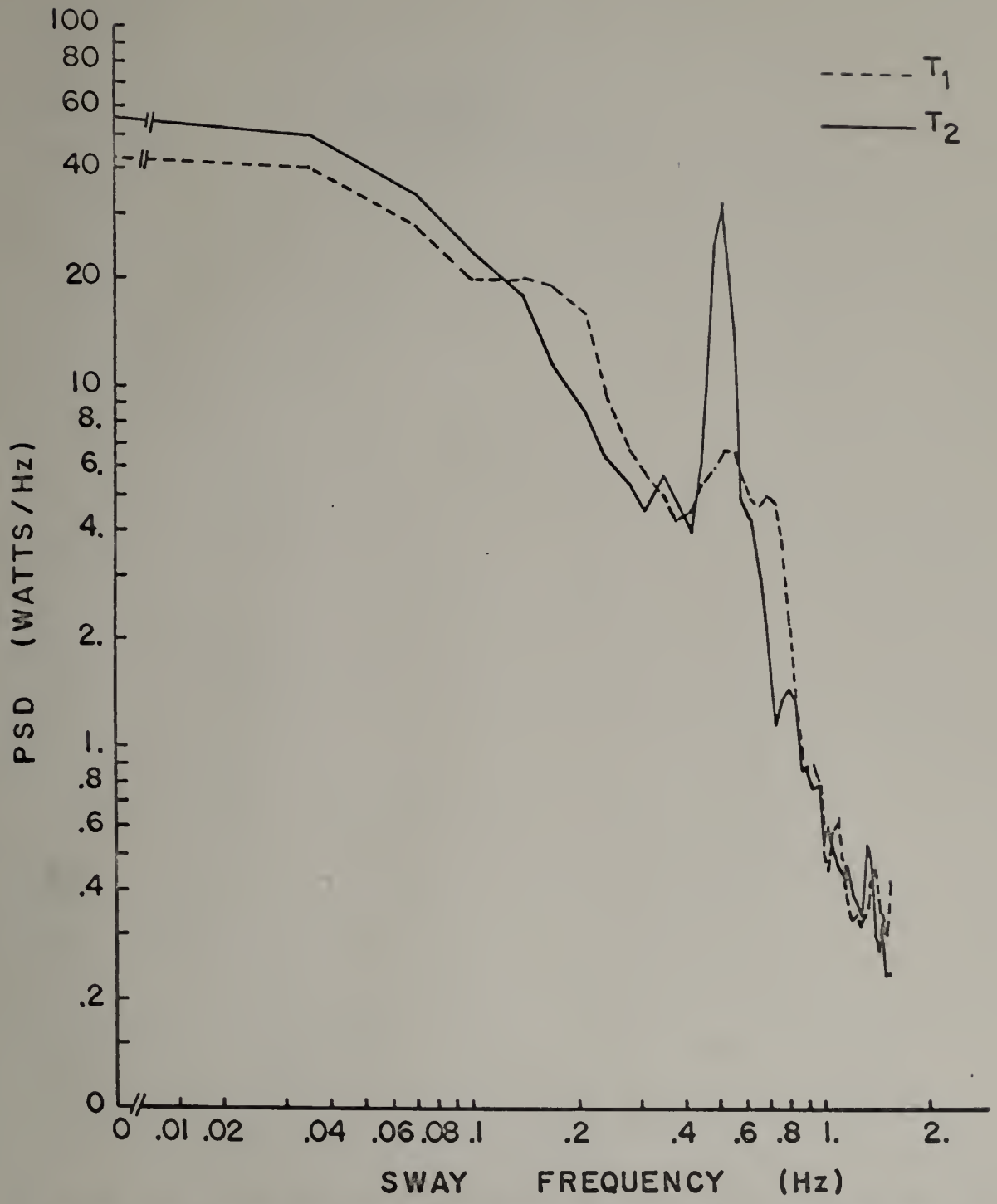

Fig. 12. Plot of the mean lateral sway PSD's for T_1 and T_2 during stimulation for both mastoids at 0.5 Hz and 0.05 ma.

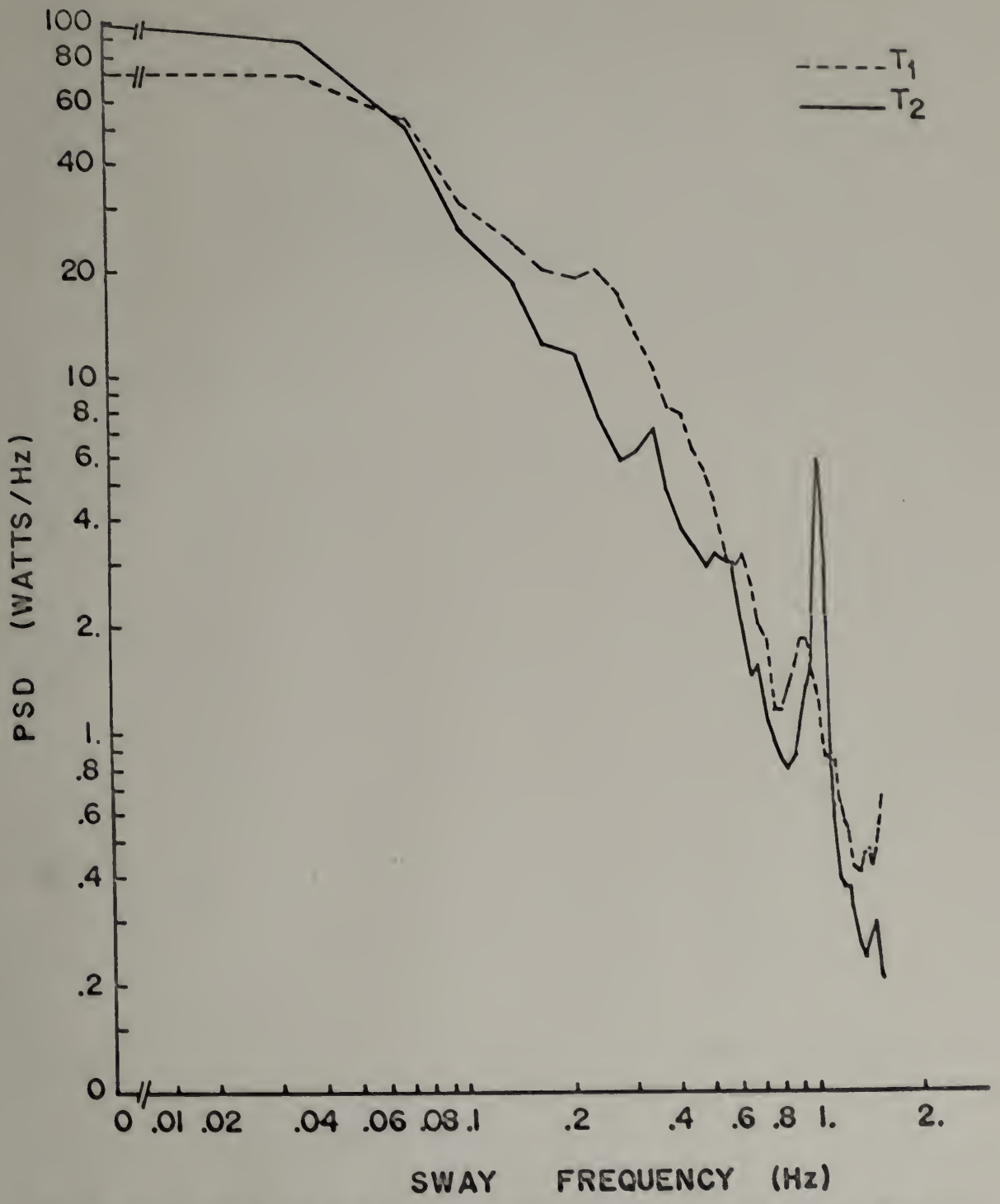

Fig. 13. Plot of the mean lateral sway PSD's for T_1 and T_2 during stimulation of both mastoids at 1.0 Hz and 0.05 ma.

affected by stimulation frequency as antero-posterior sway was. However, the presence of the stimulating current did increase the amount of power in lateral sway as it did in antero-posterior sway, but current level did not affect lateral sway power. Power during T_1 on 0.005 ma trials was 9.71 watts and, during T_2, was 11.15 watts. For 0.05 ma trials, power during T_1 was 10.57 watts and, during T_2, was 11.10 watts. Electrode location did not have any apparent affect on lateral sway with the exception of simultaneous stimulation of both mastoids. Here power was slightly higher than during stimulation of only one mastoid.

Sway and Stimulus Relationships

Mean crosscorrelation functions were obtained by summing over Ss. Figs. 14 and 15 are plots of the mean correlations between various stimulus frequencies and lateral sway, where lateral sway was being lagged. These CCF's are for stimulus-on periods during which both mastoids were being stimulated with 0.05 ma. The effect of stimulus frequency on sway frequency is again evident here as it was in the PSD's (Figs. 10-13). As stimulus frequency was increased, the correlations between it and sway frequency decreased with the correlations at 1.0 Hz being lowest. The length of time by which the sway was lagging behind the stimulus

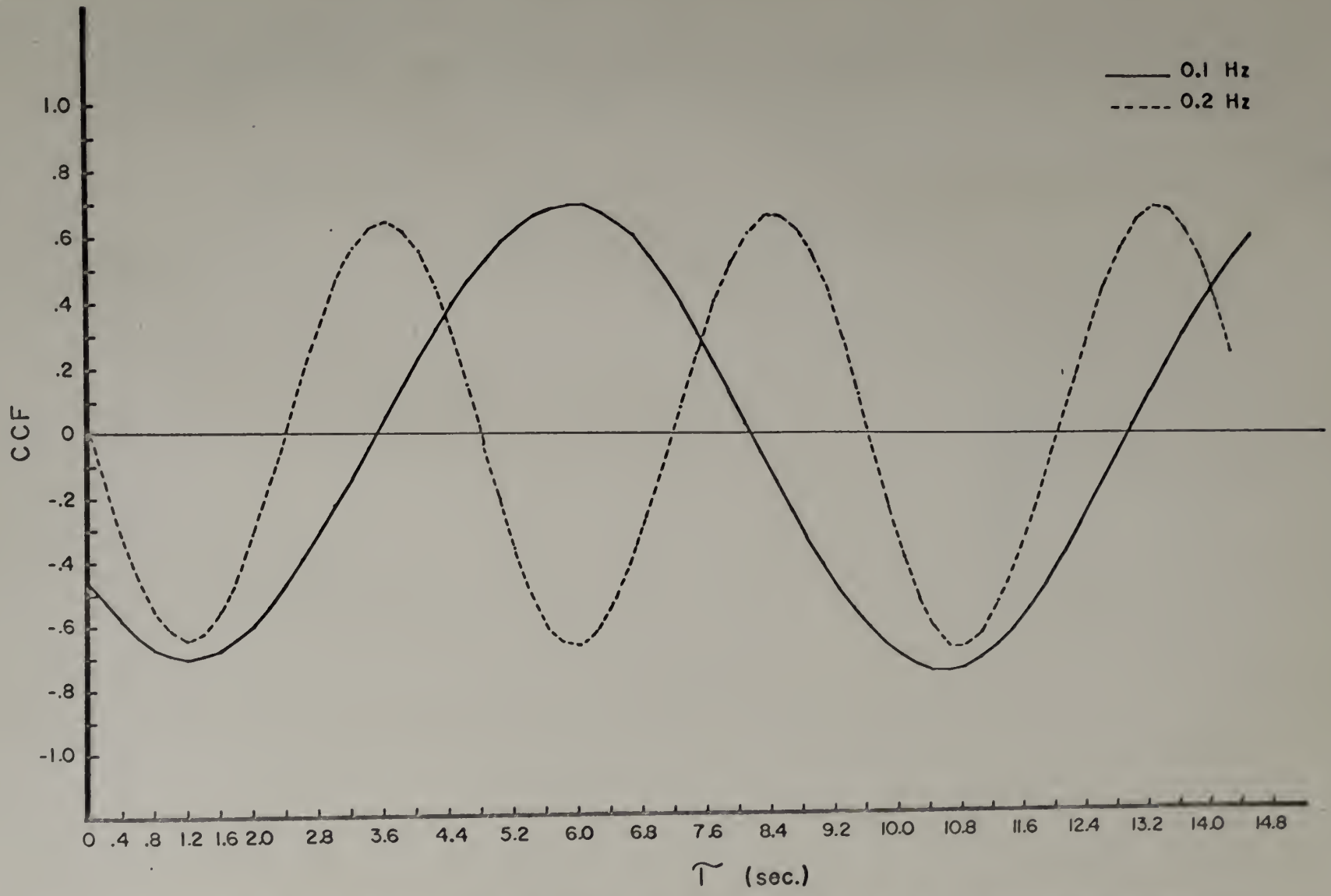

Fig. 14. Plot of the mean CCF's between a stimulus of 0.1 Hz and lateral sway and a stimulus of 0.2 Hz and lateral sway during stimulation of both mastoids at 0.05 ma. Lateral sway is being lagged.

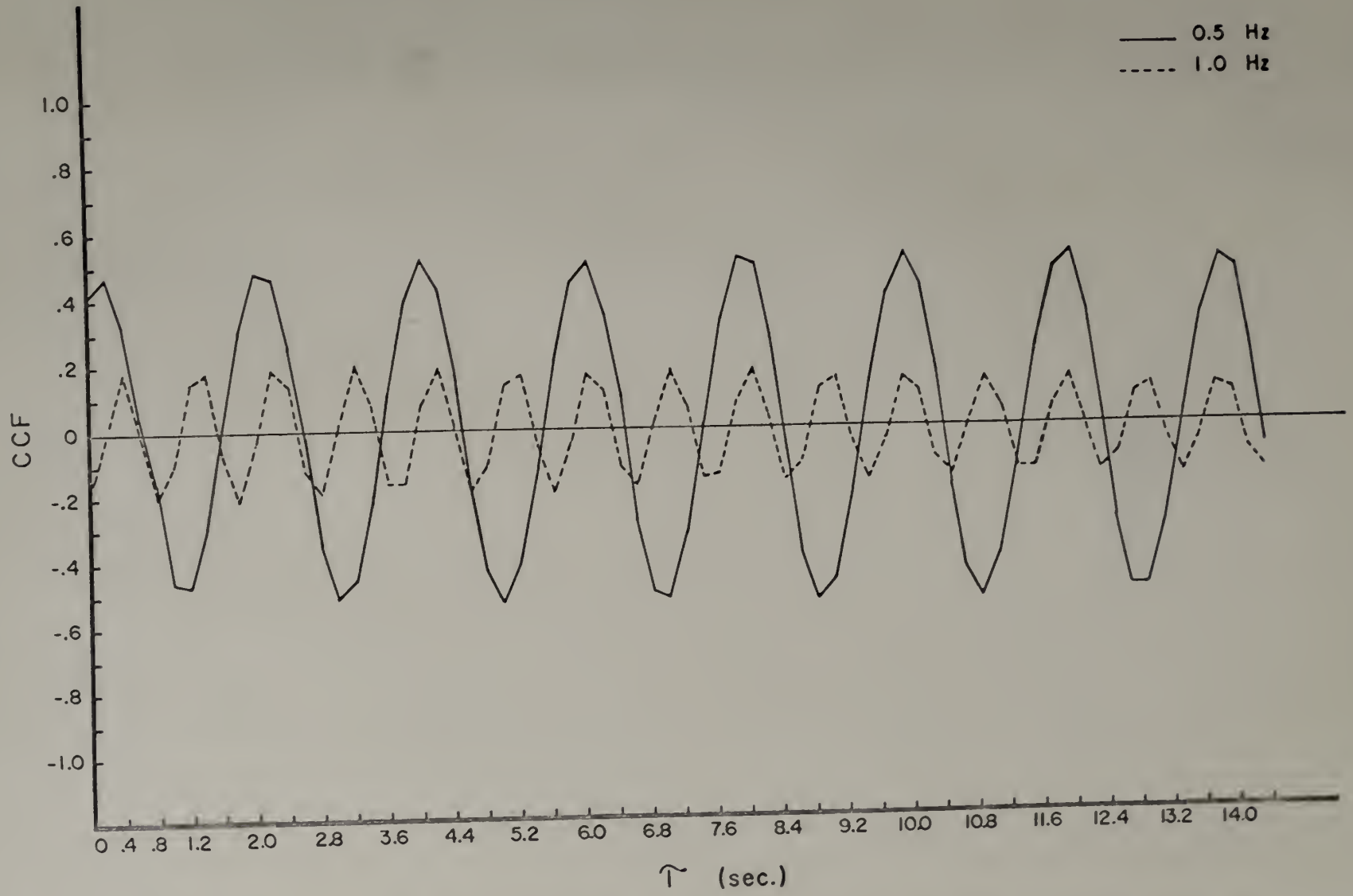

Fig. 15. Plot of the mean CCF's between a stimulus of 0.5 Hz and lateral sway and a stimulus of 1.0 Hz and lateral sway during stimulation of both mastoids at 0.05 ma. Lateral sway is being lagged.

varied with stimulus frequency. At At 0.1 Hz, the lag was 5.8 sec and, at 0.2 Hz, was 3.6 sec. The lag was 0.2 sec at 0.5 Hz and 0.4 sec at 1.0 Hz. Therefore, with the exception of 1.0 Hz trials, the lag time decreased as stimulus frequency increased. Furthermore, the time for one wavelength of the cross-correlations was approximately equal to the time for one wavelength of the stimulus frequency corresponding to the correlations. This indicated a close relationship between lateral sway frequency and stimulus frequency. It should also be noted in Figs. 14 and 15 that, at stimulus frequencies of 0.1 and 0.2 Hz, minimum correlations were achieved before maximum whereas, at 0.5 and 1.0 Hz, the opposite held. These phase relationships should not be accepted as being correct since the CCF values presented are means over Ss and the Ss were not exposed to identical stimulus trances. For some trials the anode was first on the right mastoid, while for others it was first on the left.

The remainder of the mean CCF's were close to 0.0 although various CCF's of individual Ss showed high positive or negative relationships between the two directions of sway or between sway and stimulus. These results showed no consistency within or among Ss. However, there was a higher probability that Ss would have high positive or negative correlations between antero-posterior and lateral sway when the left mastoid was

stimulated than when the right mastoid was stimulated. This was also the case when the relationship between sway in either direction and stimulus was considered.

Discussion

The amplitudes of both directions of sway recorded in the present experiment were affected only slightly by the presence of the stimulating current and its level. Antero-posterior amplitude decreased during stimulation relative to its prestimulus value, with the higher current level (0.05 ma) yielding the greater decrease. The opposite relationships were found for lateral sway. Here amplitude increased during stimulation, and the higher the current level, the greater the increase. To determine whether or not this indicated that the vestibular-motor system expended a constant amount of energy so that antero-posterior sway would always decrease if lateral sway increased, the amplitudes of the two directions of sway were compared at each stimulus frequency and current level during T_2 in terms of what percent sway in one direction was of that in the other. The percentages were found to vary with no consistency. A t-test was also done to see if there was a significant difference between the T_1 and T_2 means when both directions of sway were added together. The obtained t of 0.05 was not significant. The power in both directions of sway was slightly greater during stimulus-on periods than during the prestimulus times. These effects on amplitude and power were of such small magnitude that there is no basis for maintaining that current has an

excitatory or inhibitory effect upon sway amplitude which varies with the level of the current and the direction of sway.

Figs. 2 and 4, in which stimulus frequency results, as well as those for current level, can be examined, show that the amplitude relationships between T_1 and T_2 remained relatively stable regardless of sway direction. On trials during which the stimulus was 0.1 or 1.0 Hz at 0.005 ma, T_2 amplitude was greater than T_1. An increase in T_2 over T_1 was also found during trials at 0.2 Hz with 0.05 ma. At the other frequencies, T_2 amplitude either decreased or remained constant relative to the T_1 value, with the exception of 0.1 Hz trials at 0.05 ma for lateral sway. Here T_2 amplitude increased slightly over that during T_1. Amplitude may be confounded by stimulus frequency. This relationship among stimulus frequency, current level, and time period does suggest inhibitory or excitatory effects upon sway governed by these variables. The findings of Barnett and Posner (1941) and Dzendolet (1963), who used human Ss, gave no indication as to the reason for these differential effects, nor did those of Spiegel and Scala (1943) and Fredrickson *et al.* (1966) who experimented on cats. However, Fredrickson *et al.* did find that an increase in d-c stimulation increased, but never decreased, neuronal activity.

Another aspect of the findings for which antero-posterior

and lateral sway results were again similar involved the relationship between stimulus location and current level. On 0.005 ma trials, T_2 amplitudes were approximately equal to their respective T_1 values suggesting that this current level had little or no effect on sway. For 0.05 ma trials, sway amplitude during T_2 was less than that during T_1 when either the right or the left mastoid was stimulated. During stimulation of both mastoids at 0.05 ma, lateral sway amplitude for T_2 was much greater than that for T_1 while, for antero-posterior sway, T_2 amplitude was only slightly greater than that at T_1. Therefore, stimulation of one mastoid at 0.05 ma depressed sway in both directions and stimulation of both mastoids increased lateral sway.

These findings are in accord with those of Spiegel and Scala, who suggested that body sway is produced by alternating increased muscle tonus on one side of the body and decreased tonus on the other. The slight change in antero-posterior amplitude during stimulation of both mastoids suggests that the sides of the body on which the mastoids are located are the foci of the effect. It cannot be ascertained from the present data whether or not it was anodic stimulation that decreased muscle tonus while cathodic increased it. However, there is no reason to maintain that the present Ss responded differently to the current poles in terms of body tonus changes than did the cats examined by Spiegel and Scala. According to

Fredrickson *et al.*, the increased sway response obtained when both mastoids were stimulated suggests possible involvement of the otolith system and, therefore, the vestibulo-spinal reflex center.

Although the amplitude of antero-posterior sway was generally greater than that of lateral sway, in Figs. 3 and 5 it is evident that stimulation of both mastoids at 0.05 ma yielded a much greater amplitude in the lateral direction than in the antero-posterior. Furthermore, on the basis of the plots of the PSD's (Figs. 6-13), it is evident that lateral sway yielded results clearly indicating that the frequency of sway did vary as a function of the frequency of electrical input while antero-posterior sway did not. From the present study, it is impossible to predict whether or not a high sway amplitude will always be linked to increased sway power at the stimulus frequency though alternating increases and decreases in muscle tonus would be expected to yield a high lateral sway amplitude.

The decrease in sway amplitude during stimulation of one mastoid relative to its prestimulus period value was in agreement with the findings of Spiegel and Scala (1943) who obtained rotation of the head toward the nonstimulated side when the stimulation was monaural and this did not involve oscillation. However, stimulation of one mastoid did not increase PSD values at 0.0 Hz above their T_1 level

indicating that monaural stimulation did not cause Ss to lean.

Although a comparison of amplitudes during T_2 on the 0.05 ma trials in Figs. 3 and 5 indicates that amplitudes during stimulation of the left mastoid were less than those during stimulation of the right, it must be remembered that electrode location was confounded with sessions or days. Therefore, sway during T_2 is most validly considered in relation to its respective T_1 level. On this basis, it can be seen that sway amplitudes during stimulation of either the right or the left mastoid decreased by approximately the same amount relative to their respective T_1 amplitude values. However, the crosscorrelations did indicate that stimulation of the right and the left mastoids cannot be equated. With stimulation of the left, there was a higher probability of obtaining a high correlation between the two sway directions or between sway and the stimulus than if the right mastoid was stimulated. This suggests the occurrence of some phenomenon akin to handedness. Although no information regarding the dominant side of the body was obtained from the Ss, it may be assumed that most were right dominant. This could mean that muscular control of the right side of the body was more highly developed than that of the left. Therefore, Ss could have been more readily affected by experimental manipulations applied to the left mastoid than those applied to the right.

The results of this study have shown conclusively that the frequency of whole body lateral sway output does vary as a function of the frequency of electrical input if certain conditions are met. These conditions include stimulation of both mastoids at an adequate current level. A level of 0.005 ma proved to be below sway threshold, but 0.05 ma was above it. On the basis of the present study, it is impossible to determine what values of current below 0.05 ma would be effective in eliciting the sway response. Furthermore, it is evident that normal sway frequency in both the antero-posterior and the lateral directions lies between 0.0 and approximately 0.2 Hz. It is possible that, if more readings of the raw data were made per second and the number of PSD points was increased, high frequency components of sway could be obtained. However, it seems unlikely that so great a mass as the whole body does emit movements of high frequency.

Of the four stimulus frequencies used in this study, two (0.1 and 0.2 Hz) were within the range of normal sway frequency and two (0.5 and 1.0 Hz) were not. As stimulus frequency was increased, the amount of power added to the sway at that frequency decreased as can be seen in Fig. 16. The four points were obtained by subtracting PSD value for T_1 from that for T_2 at the appropriate stimulus frequency. The plot is an indication of the frequency sensitivity of the system.

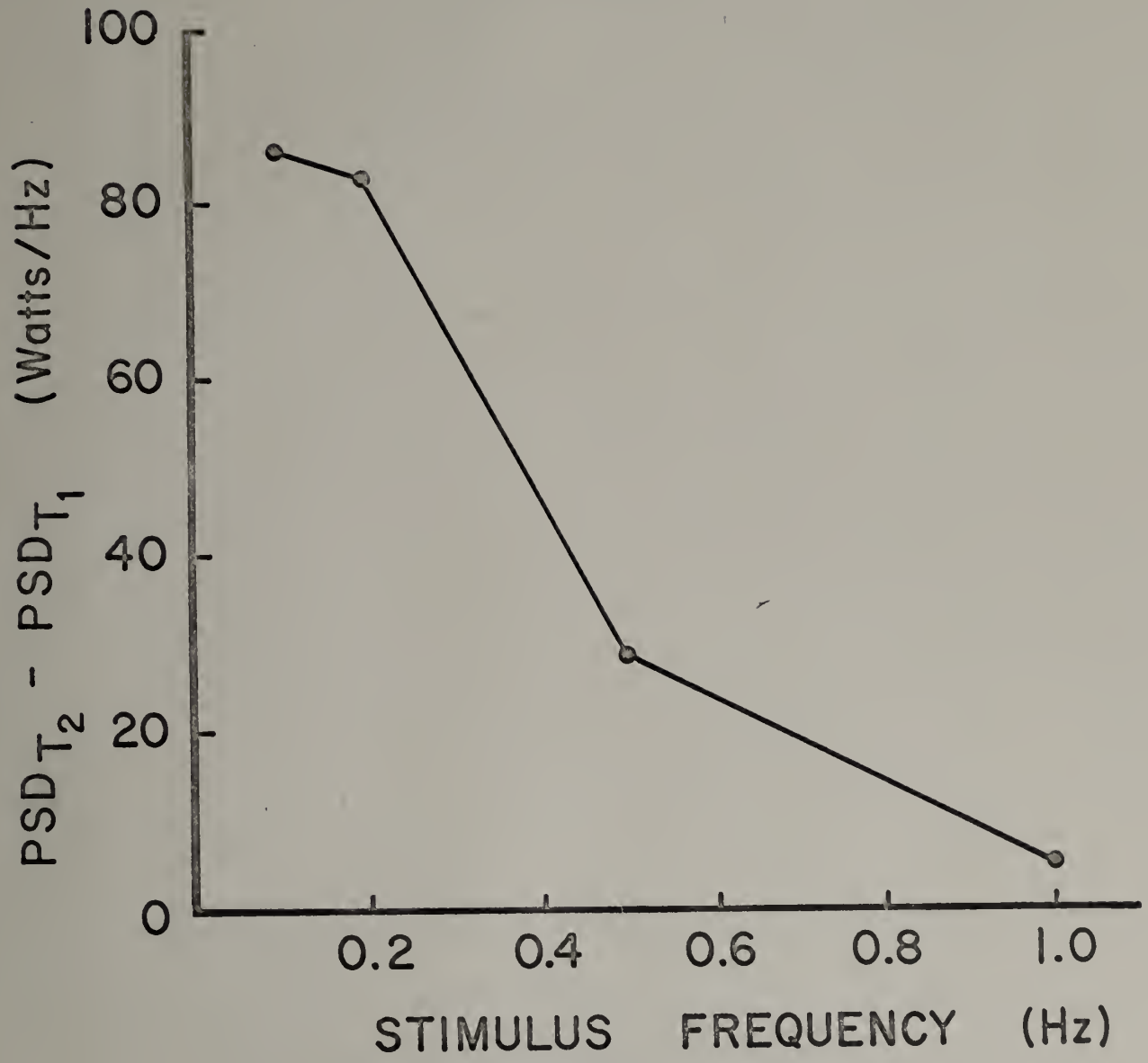

Fig. 16. Plot of the frequency sensitivity of the vestibular-motor system.

It is possible that the potent effect of the stimulus would disappear if its frequency were further from the normal frequency of sway. It could also be conjectured that a higher level of current would be needed if the effect were to be obtained at higher frequencies. On the basis of the findings of Barnett and Posner and Dzendolet, both of the possibilities seem likely. Barnett and Posner found that, above 5.0 Hz, the sensation of oscillating fused. To obtain a sway response at 4.0 Hz stimulation, Dzendolet found that current of 0.4 ma was required.

No conclusions regarding the site of action of electrical stimulation can be reached from the present study. However, this experiment and the system analysis used did show that the electrical method of vestibular stimulation does permit experimental control and manipulation of the frequency and amplitude of the sway response. Further studies of this kind in which the Ss have diagnosed vestibular impairments would be needed to gain information regarding the anatomical elements affected by electrical stimulation of the vestibular apparatus.

Another aspect of the findings which is of interest is the variation in sway lag, or response, time with stimulus frequency that was obtained when the crosscorrelations were calculated between lateral sway and the stimulus. As stimulus frequency was increased, lag time decreased. Also, Figs. 14 and 15 show that,

when the stimulus was 0.1 or 0.2 Hz, the first peak correlations achieved were negative. When 0.5 or 1.0 Hz were used, maximum correlations were obtained before minimum. A possible explanation may be that, since the higher stimulus frequencies were outside the normal range of sway frequencies, more control could be exerted over the system when they were used causing shortened lags and positive correlations between the response and the stimulus. The lower stimulus frequencies, on the other hand, were represented in the sway by high PSD values even when the system was not being stimulated. Therefore, a longer lag time could have been required before the sway was affected by the stimulus because the stimulus could only serve to accentuate ongoing sway.

In conclusion, present research did support the use of the electrical method of vestibular stimulation and the measurement of whole body sway as a means of obtaining further information regarding the operation of the vestibular system. The frequency and the amplitude of sway output would seem, as a result of this study, to be valid response measures.

Summary

Six standing male Ss were stimulated with sinusoidally-varying, low-level electric current via electrodes attached to their mastoid processes. Each S was exposed to stimulus frequencies of 0.1, 0.2, 0.5, and 1.0 Hz at zero to peak current levels of 0.005 and 0.05 ma, on three separate days. Either the right, the left, or both mastoids were stimulated at each session. Each trial consisted of a 30 sec prestimulus period followed by a 30 sec stimulus-on period. The response measures, recorded on separate channels of a polygraph, were whole body sway in the antero-posterior and in the lateral directions. These measures were analyzed using the autocorrelation, the power spectral density, and the crosscorrelation functions, and also analyses of variance.

The conclusions from the present study were:

1. The amplitude of sway in both the antero-posterior and the lateral directions was affected by current level, stimulus frequency, and electrode location.

2. Lateral sway output varied as a function of stimulus input frequency when both mastoids were stimulated at a current of 0.05 ma.

3. A sharp increase in the power of lateral sway at the stimulus input frequency was seen in lateral sway when both mastoids were stimulated.

4. Most of the power in S's sway, for both the antero-posterior and the lateral directions, was between 0.0 and 0.2 Hz whether or not S was being stimulated.

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Footnote

1. I am indebted to Dr. Ernest Dzendolet and to Mr. Allen Williams for their aid in the design of these electrodes.

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Appendix A

Analysis of Variance of Antero-Posterior Sway Measure

Source of Variance	df	Mean Square	F-ratio
Subjects (Ss)	5	1.572	
Electrode Location (L)	2	0.180	1.642
Ss x L	10	0.110	
Frequency (F)	3	0.028	0.438
Ss x F	15	0.065	
F x L	6	0.032	0.934
Ss x F x L	30	0.035	
Current Level (I)	1	0.005	0.071
Ss x I	5	0.071	
I x L	2	0.133	2.128
Ss x I x L	10	0.062	
I x F	3	0.102	1.399
Ss x I x F	15	0.073	
I x F x L	6	0.015	0.265
Ss x I x F x L	30	0.055	
Time (T)	1	0.153	2.271
Ss x T	5	0.067	
T x L	2	0.029	0.663
Ss x T x L	10	0.043	
T x F	3	0.056	1.545

Appendix A (continued)

Source of Variance	df	Mean Square	F-ratio	
Ss x T x F	15	0.037		
T x L x F	6	0.027	0.667	
Ss x T x L x F	30	0.041		
T x I	1	0.080	0.565	
Ss x T x I	5	0.142		
T x L x I	2	0.027	1.631	
Ss x T x L x I	10	0.036		
T x F x I	3	0.183	11.042	$p < .001$
Ss x T x F x I	15	0.017		
T x F x L x I	6	0.039	1.132	
Ss x T x L x F x I	30	0.034		

Appendix B

Analysis of Variance of Lateral Sway Measure

Source of Variance	df	Mean Square	F-ratio	
Subjects (Ss)	5	1.131		
Electrode Location (L)	2	0.346	2.492	
Ss x L	10	0.139		
Frequency (F)	3	0.099	2.069	
Ss x F	15	0.048		
F x L	6	0.048	1.371	
Ss x F x L	30	0.035		
Current Level (I)	1	0.035	0.287	
Ss x I	5	0.123		
I x L	2	0.488	8.091	$p<.01$
Ss x I x L	10	0.060		
I x F	3	0.030	0.545	
Ss x I x F	15	0.054		
I x F x L	6	0.015	0.213	
Ss x I x F x L	30	0.071		
Time (T)	1	0.012	0.180	
Ss x T	5	0.066		
T x L	2	0.253	4.381	$p<.05$
Ss x T x L	10	0.058		
T x F	3	0.045	1.159	

Appendix B (continued)

Source of Variance	df	Mean Square	F-ratio	
Ss x T x F	15	0.039		
T x L x F	6	0.014	0.299	
Ss x T x L x F	30	0.045		
T x I	1	0.006	0.028	
Ss x T x I	5	0.218		
T x L x I	2	0.392	5.035	$p < .05$
Ss x T x L x I	10	0.078		
T x F x I	3	0.153	4.321	$p < .025$
Ss x T x F x I	15	0.036		
T x L x F x I	6	0.053	1.116	
Ss x T x L x F x I	30	0.048		

A CONTROL SYSTEMS ANALYSIS OF ELECTRICALLY INDUCED BODY SWAY

A Dissertation

By

Carolyn K. Bensel

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July 1962
(Month) (Year)