COHERENCE, TRANSFER RATIO, AND POWER SPECTRA OF THE ELECTROENCEPHALOGRAM

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OF THE ELECTROENCEPHALOGRAM

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PREFACE

The research presented in this thesis embraces a study in an interdisciplinary area between engineering and medical science. Similarly, it encompasses the facilities of two universities, one the Oklahoma State University and the other the University of Oklahoma School of Medicine.

I have attempted to unite the two disciplines in the introductory chapter by drawing parallels between physiological and engineering systems. This is an attempt to provide a common ground for building future discussions. The human brain and its electrical manifestations as recorded in an electroencephalogram present intriguing and challenging problems which mystify both the biological scientist and the engineer. I hope that this research will help in some way to unravel some of the mysteries.

Acknowledgments of profound indebtedness must be made to several persons. First, to Dr. H. L. Jones and Professor P. A. McCollum of the College of Engineering, Oklahoma State University, I must express my gratitude for much help, advice, and encouragement. Likewise, I wish to express my appreciation to Drs. W. L. Hughes, W. J. Leivo, and H. E. Harrington for serving on my advisory committee.

It was almost exclusively through the efforts of Dr. C. G. Gunn of the University of Oklahoma School of Medicine that the opportunity to perform this work was made available to me. He obtained the

equipment, provided the facilities, and supervised the experimental work.

I estimate that nearly 600 hours of computer time have been required during the preliminary investigations and final data processing represented by the work presented in this dissertation. So, I feel a large debt of gratitude toward the personnel of the computer facility at the University of Oklahoma Medical Center, to its Director, Dr. Edward N. Brandt, Jr., and to Mr. Paul Costiloe, Statistician, for much help and advice, consultation, and computer programs. I also acknowledge my indebtedness to programmers Lee Henderson and Joe Briley, and to Mrs. Jan Bennett, the computing room supervisor.

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CHAPTER I

INTRODUCTION

The immediate goal sought in undertaking the research described in the following pages was to investigate the possibility of applying some of the mathematical techniques of statistical communication theory and feedback control systems to analyze the electroencephalogram (EEG). If this proves to be valuable, other long-term goals are to develop clinically useful diagnostic tests and experimental methods for neurophysiological research.

Classically, physiological experiments are based on stimulus-response techniques. That is, a controlled change in environment is induced in the physiological system being studied; and the resulting reaction to this change is recorded. By varying the stimulus, the response may be varied and patterns of response established. This enables one to formulate theories about the underlying physiological mechanisms.

In neurophysiological research this same general technique is employed. The stimulus to the neural system being studied can be presented in many ways, e.g., electrical, mechanical, chemical, and thermal. If intact conscious animals are used, a stimulus to the nervous system can be effected via any of the sensory modalities which may be sound, light, heat, touch, or smell. All are commonly used in experimentation.

For the benefit of readers having limited knowledge of the nervous system, a brief explanation is presented in the following paragraphs so the material in subsequent chapters may be better evaluated. A detailed discussion suitable for the non-physiologist may be found in Woolridge's book, Machinery of the Brain (1).

Two of the outstanding events in the history of electrophysiology are Galvani's discovery of nerve action in the frog's leg and Caton's discovery of the electrical activity of the brain (2). The former led researchers to dissection and exploration at the microscopic level, and the latter led to investigations at the macroscopic level. In 1924, Hans Berger (2) succeeded in recording electrical signals from the human scalp and was able to demonstrate a variation in these signals with eye opening, sensory stimuli, and mental activity.

Microscopic research has revealed that the basic building block of the nervous system is the "neuron." This is a highly specialized biological cell having three parts: (1) the soma or cell body; (2) the dendrites, which are tentacle-like afferent projections; and (3) the axon, a tail-like efferent projection which may or may not have single or multiple bifurcations. Throughout the nervous system, neurons appear in a variety of sizes and shapes. The inside of the quiescent cell is negatively charged with respect to the outside due to ionic concentration gradients; an electrical potential difference of about 70 MV can be measured across the cell wall. Ionized potassium and negatively charged protein molecules are the main elements contributing to this potential difference.

Neurons can be activated (or innervated) by application of an external stimulus. Commonly used forms of stimuli are electrical,

chemical, and mechanical. When inervated, the cell wall is electrically depolarized due to dramatic changes in ion concentration across the cell wall. This is a regenerative process; and once a stimulus adequate to exceed a threshold level is provided, the neuron is depolarized in a precipitious fashion, following which it can autonomously repolarize. The associated electrical potential change is referred to as an "action potential."

Engineers would liken the neuron to an analog threshold logic element in which, once the input exceeds the threshold, a monostable switching action occurs to provide an output pulse. Networks of neurons can be analyzed using Boolean algebra as commonly applied in switching circuit analysis (3).

The axon can be considered as the output trunk of the neuron. Once depolarization of the cell body occurs, a depolarization wave sweeps along the axon away from the cell body. Axons terminate on either dendrites or soma of other neurons at junctions referred to as synapses. At the synapse there is no protoplasmic continuity; but an electrochemical action occurs, and by some means, which is the subject of much contemporary investigation, an influence is exerted on the polarization and threshold level of the post synaptic neuron. A synapse may provide the incremental change to depolarize a neuron or it may decrementally depolarize it and sum together with synaptic inputs from other neurons. Some later synaptic input will then trigger depolarization. This phenomenon is referred to as spatial summation. There may also be temporal summation, which enables a repeated input from a single synapse to depolarize the post synaptic neuron. The times involved, over which this integrative influence is exerted, is in

the order of one-quarter millisecond.

More recently it has been shown that some synapses are inhibitory rather than excitatory, i.e., they hyperpolarize the post synaptic neuron and make it more difficult for the neuron to be depolarized (4). Herein lies a fundamental neurophysiological phenomenon which, it is believed, plays a major role in some diseases (such as epilepsy) and provides a mechanism for fine integrative control.

The axon is the specialized output extension of the neuron cell body and is commonly called a "nerve fiber." A depolarization wave (nerve impulse) moves along the axon with no diminution of amplitude. Some neurons have axons that are very lengthy (as long as 1 meter); while others are very small, having spans of less than a millimeter. These are referred to as interneuronal types. It is estimated that there are 20 billion nerve cells in the human central nervous system, and each of these may have as many as 200 synaptic inputs and in turn may provide synaptic outputs to 200 other neurons. There may be as many as one million neurons per cubic inch, and the combined metabolism rate of all these cells is such as to require less than the equivalent of 25 watts. These neurons are interconnected in very complex and intricate arrangements to form neural networks.

There are specialized neurons which play what may be regarded as the role of a transducer. They are innervated by pressure, temperature, chemicals, and light. They form a part of the sensory system and are called receptor neurons. Still others exert an electrochemical influence on muscle to cause it to relax or contract and are known as effectors. A simple chain can be visualized consisting of a receptor being innervated by, say temperature, and in turn activates an effector

which causes a muscle to contract and remove the receptor from the heat source. This is a trite example of a reflex action which is a vital neurophysiological phenomenon in the preservation of physiological integrity.

Not all action is reflexive. Sensory inputs are propagated via nerve trunks in the spinal cord to a massive differentiated conglomerate of neurons at the end of the spinal cord called the brain. The brain is in reality an extension of the spinal cord.

Here, neuronal inputs are received via afferent pathways. These inputs are, in effect, channeled to the proper area of the brain, analyzed, modified, and appropriate outputs generated and transmitted via efferent pathways to effect proper muscle movements (motor action). Engineers are tempted to label the brain as a data processor for obvious reasons.

The brain is highly differentiated, i.e., specific areas perform specific functions. These have been widely studied in man and are fairly well known.

Of special interest is one portion of the brain known as the reticular activating system. Nearly all afferent and efferent impulses are sensed by the reticular formation via collaterals. Neurons in the reticular activating system may respond to a stimulus anywhere on the body surface. Thus, they are said to be non-specific. It seems to sense the general neural condition of the whole body in a pattern-like manner and then properly govern sensitivities of the various other specific brain segments in a fashion appropriate to existing circumstances. For example, it is credited for enabling a mother to sleep undisturbed by a passing locomotive, yet instantly awaken at the

slightest abnormal sound from the baby's crib in an adjacent room. It seems to sort out that which is unimportant and minimize or reject it and pass on that which is important. The reticular formation can extend its influence to modify reflex actions (5).

If the central nervous system is regarded as an adaptive servo-system, then in all likelihood the adaptive role will be relegated to the reticular formation.

Sem-Jacobsen (6) found that pilots with quick well-coordinated motor responses have a lower seizure threshold than those with slower responses and poorer coordination. This suggests that the reticular activating system might keep the brain more "tightly bound" or "keyed up" in some people. The temptation is great to liken physiological systems to an adaptive servomechanism.

Awake or asleep, the brain is at work. Neurons are constantly being depolarized and repolarized in some regulated fashion to provide the living animal with an appropriately functioning brain.

Electroencephalogram

Hans Berger's (2) discovery that electric potentials can be measured on the scalp, which are, to some extent, indicators of brain function, has given medical science the electroencephalograph (EEG), a valuable clinical and research tool. The electric signals are thought to be derived from the dendritic potentials in the outer layers of the cortex in the vicinity where the measurement is made.

Exact relationships of these potentials, recorded by placing electrodes on the scalp, to the generating neuronal activity have not been elucidated. It is generally assumed to be a temporal summation

of dendritic potentials of hundreds of thousands of neurons in the vicinity of the electrode (7). The involved neurons have various physical arrangements with respect to the recording point. Thus, the surface electrical signals conceivably represent a weighted average of potentials generated by dipole layers of indescribable number and orientations which are transmitted through a volume-conducting medium having several layers of different resistivities.

Even in the face of such an unsolved complex situation, the EEG occupies a preeminent place in neurological investigations; though its use is based almost solely on empirical applications. The normal clinical EEG record consists of measuring scalp potentials at four or more places on the left and an equal number of places on the right hemisphere in various symmetrical patterns. Analysis of these records is based on signal frequency, patterns, waveforms, amplitude, symmetry, and similarity between channels. Many volumes have been written about EEG patterns in health and disease (8).

The Visual System

The human visual system provides a convenient means of making meaningful neurological investigations because a stimulus in the form of light flashes can be readily supplied to the nervous system.

Light is sensed in the eyes by the retinae which contain neuronal sensors called rods and cones. It is estimated that there are six million cones and 110 million rods in a human retina. These rods and cones are connected by a bipolar cell layer to a ganglion cell layer from which the optic nerve emerges. The optic nerve of man possesses an estimated one million nerve fibers; therefore, there is a considerable

degree of convergence (mostly in the ganglion cell layer), which necessarily results in a sacrifice of detail of the visual image. In addition to convergence, some cross-connections also exist, i.e., a given rod or cone may ultimately be connected to more than one fiber in the optic tract.

Light sources from a subject's right field of view are projected on the nasal half of the right retina and the temporal half of the left retina. Light from the subject's left field of view is projected onto the nasal half of the left retina and the temporal half of the right retina as shown in Figure 1.

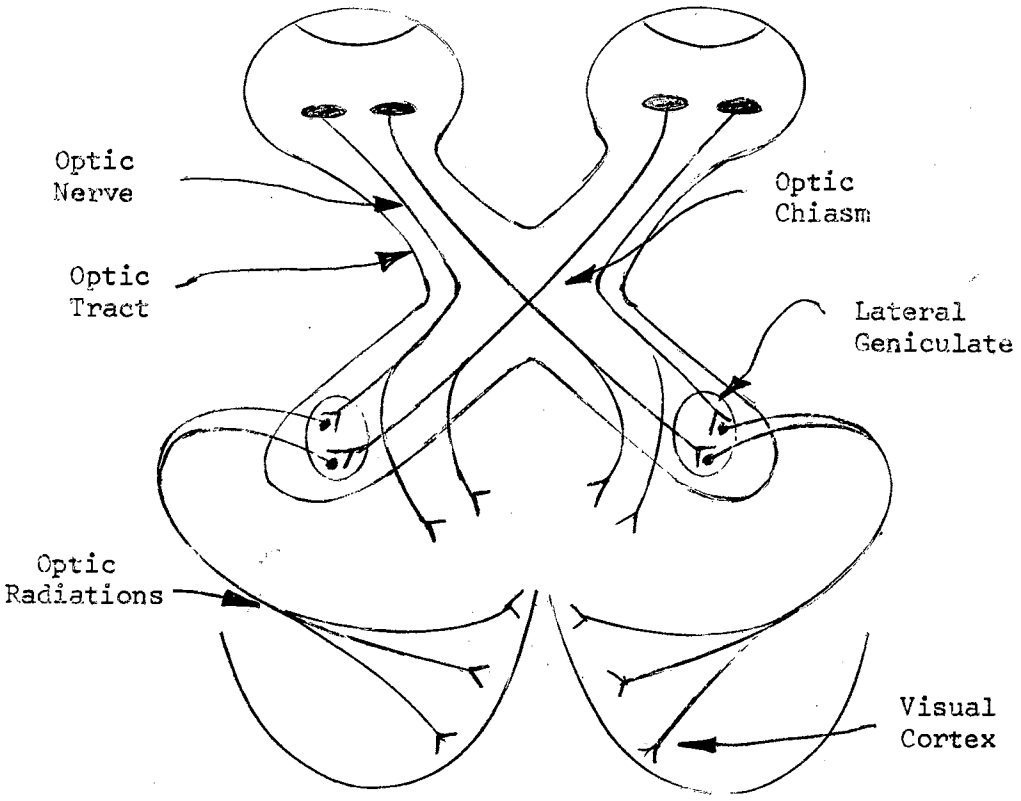

Figure 1. Visual System Pathways

The optic nerves connected to the nasal halves of the retinae cross to the contralateral side at the optic chiasma, while the nerves from the temporal halves of the retinae remain on the ipsilateral side. These optic tracts terminate at a nucleus called the lateral geniculate body which, in man, seems to serve only as a relay station with no discernible interaction between adjacent pathways at this point. From the lateral geniculate body, the visual information is relayed via the internal capsule to the visual cortex at the occipital lobe. Here, the nerve fibers enter into and arrange themselves throughout the fourth cell layer. They are so dense as to make a white layer visible to the naked eye, and for this reason the visual cortex is also referred to as the striate cortex.

From the visual cortex (which ranges from 20 to 45 sq. cm. in area), nerve impulses concerned with vision are extended to adjacent sections of the cortex. The afferent fiber density has been estimated at 25,000/mm^2 and the efferent density at 75,000/mm^2. Anatomically, there is no good evidence of any functional interconnections of the striate areas of the two hemispheres; however, the corpus callosum has been shown to play a role in the transfer of visual memory. Fine collaterals to the reticular formation are given off from the main branch of the optic tract both prior to the lateral geniculate body and the terminal organization. Other pathways project to subcortical regions.

From this point the mechanisms, pathways, and influence of the visual system become diffuse. It is known that the visual system is adaptive. Experiments have shown that visual memory is associated with the temporal lobes (9).

The visual system is utilized for tests in several ways. One is the flicker fusion diagnostic test wherein a repetitive light flash is presented from what is essentially a point source located at known coordinates in a patient's visual field. Each light flash should elicit a burst of impulses over the optic nerve; and, as the flash rate increases, the bursts of impulses merge into a continuous chain, and the patient no longer perceives distinct light flashes, and the light appears to be on continuously. The position, rate, and duration of the light flashes at which the fusion occurs conveys diagnostic information to the clinician.

A second method utilizes a technique known as "evoked response." Here, a repetitive light flash at a low rate (usually about 1 per second) is presented to a subject, and the EEG signals from the occipital area are recorded and analyzed by an averaging technique (10). Special purpose computers have been designed to accomplish these analyses in the laboratory while the test is being conducted. The original work was performed on the ARC-I (Average Response Computer) at the Massachusetts Institute of Technology (11).

This experimental technique has been used by many research workers. Stimuli are commonly provided in the form of light flashes, brief tones, clicks, or touching the body surface. Waveform analysis and delay time (latency) of the evoked potential has been the primary source of information gained from these experiments (12). They could conceivably be regarded in the same manner as an impulse response of a filter or servomechanism. A useful comparison can be made if the same experiment can be performed on normal brains and on brains having some diagnosed lesions. Such an opportunity is provided by comparing the

evoked potentials from normals and epileptics of the petit mal classification. This work has been reported and shows, in general, a lower amplitude response having the same general waveform in epilepsy as compared to normals (13).

By using a variable frequency light flash, a physician may search for flash rates to which a patient indicates a sensitivity as evidenced by his EEG when it shows an indication of "following" or "pacing." Psychomotor seizures may be provoked by this method (14).

Another analysis method which has received much attention from Brazier and Barlow (15, 16, 17) is the application of mathematical correlation of the EEG signals. They have demonstrated the correlograms derived from the EEG recorded on the same healthy subject at successive times over a period of months is essentially constant. They have also demonstrated changes in the autocorrelogram due to brain lesion. Brazier's work in this area has also included the analysis of single evoked responses (18). One interesting fact derived from these works was that even though the same predominant frequency existed in the right and left recording electrode (approximately 10 c.p.s.), they were not phased together. However, when the experimental subject was exposed to a light flash at this rate, both signals were phase locked.

Some of the most recent work reported in this area of research has been by Walter and Adey (19). They have employed cross-correlation and cross-power spectral analysis as a method of analyzing EEG recorded from depth electrodes implanted in cats' brains for experiments designed to investigate learning processes.

The work presented in this thesis is based on use of the photic stimulus. An experiment was constructed based upon an idea that a

flashing light could be considered as an input signal to a system of unknown characteristics, and the EEG recorded from the scalp could be considered as the output of the system. The EEG signal is certainly not the normal brain output. However, a system different from the normal physiological system can be defined to embody whatever elements are desired; and the interrelationship between any two points can be studied. They may be regarded as input and output for that system. Further discussion of the mathematical processes used in analyzing these data is presented in the following chapter.

CHAPTER II

EXPERIMENTAL METHODS AND DATA PROCESSING

Since the visual system has been so widely used in neurological investigations, it was decided to base the experimental processes used in this research on this same system. Doing so provides an opportunity to relate the results of this work to conclusions drawn from research of an allied nature.

The following experimental procedure was devised to produce the data to be used in the development of a useful analytical process.

Experimental Methods

Data were taken on subjects placed in a semi-darkened, electrically shielded room. Efforts were made to maintain a quiet surrounding.

Stimulation was provided by a Grass Instrument Company Photo Stimulator, Model PS1. The light was positioned 25 cm. from the nasal bridge and adjusted to flash squarely into the subject's eyes which were closed during the entire experiment. Flash rates of 5, 7, 10, 12, 15, 17, 20, 22, 25, and 30 per second were used. The rate was measured by a Hewlett-Packard Model 521C Electronic Counter. The stimulus intensity control on the Grass Photo Stimulator was adjusted to switch position 8, which provides a flash having a peak intensity of 9,500,000 lumens and a duration of 10 microseconds.

EEG was recorded on a Grass Instrument Company, Model 6,

Electroencephalograph. Recordings were made from the right and left occipital and temporal locations. The stimulus monitor capability of the EEG recorder was used to record stimulus events on one channel and timing marks on another channel. The Model 6 EEG provides signal outputs which can be adapted for recording on any standard magnetic tape recorder of instrumentation quality.

Right and left occipital and right and left temporal EEG signals were recorded (using vertex as a common lead), both on paper and on a Nemotron, Model 700 magnetic tape recorder. The stimulus monitor pulse recorded on paper was also used as a trigger to a Tektronix Type 161 Pulse Generator. For each trigger input the pulse generator produced a pulse of 0.75 volts magnitude and a width of 10 milliseconds which was recorded on magnetic tape. This step was necessary to record a useful pulse on magnetic tape as dictated by the bandwidth limitation of the instrument.

The sequence of events was programmed by a 120-second timer which closed a set of contacts for 35 seconds of each 120-second cycle. This contact closure was used to turn on a Tektronix Type 162 Waveform Generator. The waveform generator was adjusted to produce a sequence of 5 pulses spaced ten seconds apart. This occupies 40 seconds of the 120-second cycle and is the time during which the stimulation was applied. The remaining 80 seconds were used as a rest period for the subject and to adjust the stimulation to a new rate. This sequence of events is illustrated in Figure 2. The output pulse from the waveform generator was used to trigger a second pulse generator, and its output pulse was recorded on paper and on magnetic tape. The stimulation was started midway between the first two timing pulses and was terminated

with the last timing pulse.

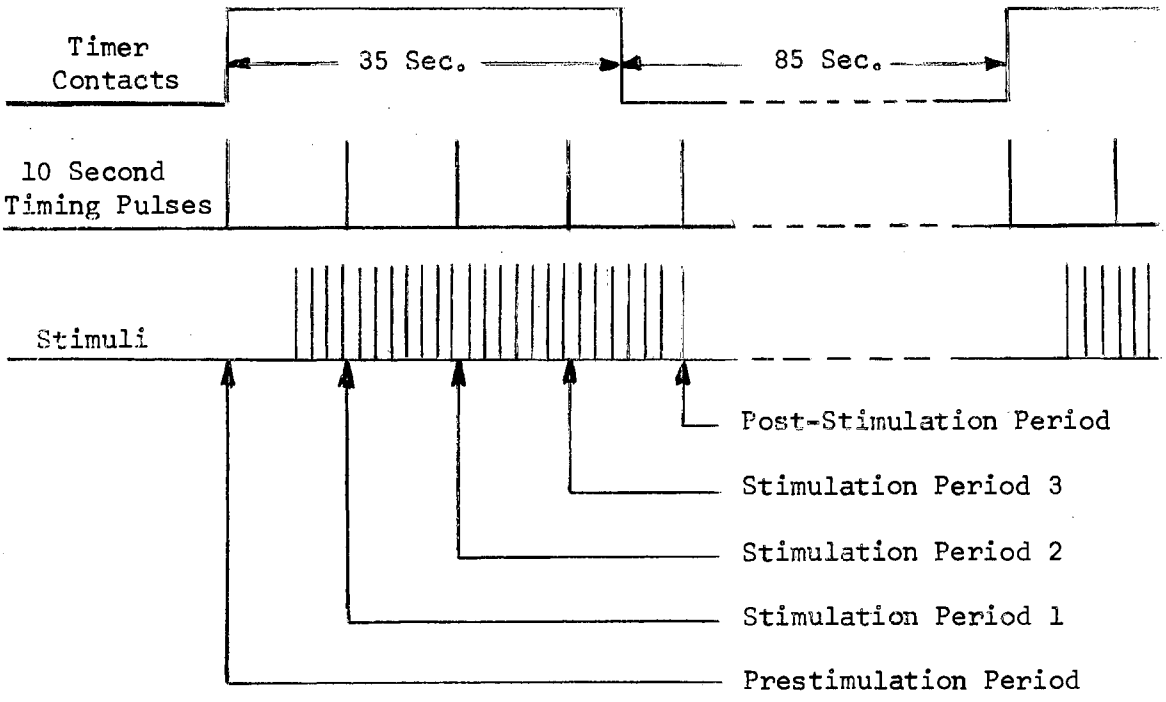

Figure 2. Sequence of Events

The first of these five pulses identified the segment of record to be analyzed as a prestimulation period; then, in turn, they identify the segment to be analyzed as: stimulation period 1, stimulation period 2, stimulation period 3, and a post-stimulation period. This cycle was repeated for each stimulation rate.

After recording the data, the magnetic tape was played back for verification purposes. It was then digitized on an IBM 1710 Control System, under program control of an IBM 1620 digital computer, in the following fashion.

The IBM 1710 has a fixed sample rate of 6,000 points per second

which is much higher than needed. To compensate for this, the magnetic tape which was recorded at 1 7/8 i.p.s. was played back at 15 i.p.s. This is an 8:1 time speedup which decreased the real-time sample rate by a factor of 8, making it 750 points per second.

A schematic drawing of the equipment connections is shown in Figure 3.

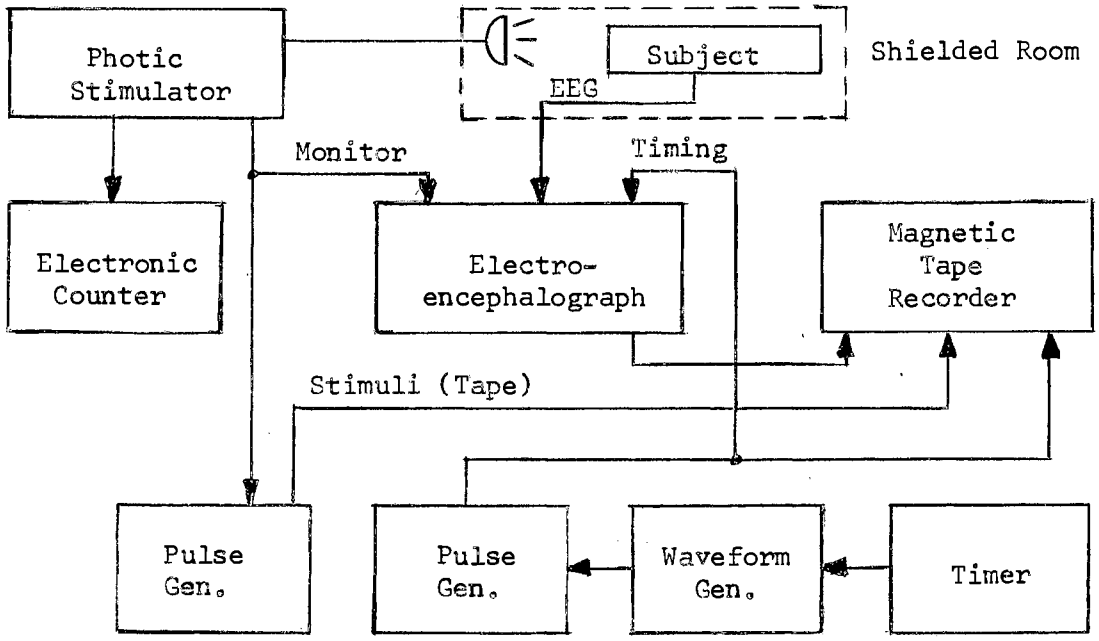

Figure 3. Equipment Connection Diagram

The IBM 1710 system was operated in the scan synchronized mode. Everytime it receives a synchronizing pulse, the analog-to-digital (A/D) converter is turned on. After digitizing 3,000 points (specified by program), it is turned off and remains off until another synchronizing pulse is received. The 5 timing pulses were used for synchronizing purposes. In this manner a 3,000 point data set was acquired for the prestimulation; stimulation periods 1, 2, and 3; and the post-stimulation period.

After the digitizing program was loaded into the computer, a heading card was read-in which identified the first 3,000 point set. When this set was digitized and the A/D converter shut off, another card was read-in to identify the next data set, etc. In this manner each digitized data record was identified.

When the five groups of 3,000 data points were completed for one stimulation rate, the magnetic tape was stopped; and these data were transferred from core storage to an IBM Model 1311 Disc File. The tape was started again, and the 5 data groups for the next stimulation rate were obtained. This cycle was continued until one data channel had been digitized properly at each stimulation rate. The tape was then rewound to the starting point, and a second channel was digitized. Between channels the data on the disc-file were transferred to magnetic digital tape. This process was repeated until the four EEG channels and the stimulation channel were digitized.

It should be pointed up that most mathematical analyses require simultaneous samples on all data channels. The foregoing procedure produces such digitized records when proper regard has been taken for synchronizing pulse rise-times and the A/D starting time.

While the digitizing process was being performed, the analog data were monitored on a Tektronix Model 561A oscilloscope to verify that proper points were taken and identifications made. A separate digital tape was made for each data channel.

For processing, two digital tapes (stimulation and a given data record) were merged point by point on an IBM 1401 computer to make a single data tape. In this merge program provisions were made to select data segments by stimulation rates and analysis periods. For example,

a merged tape could be generated that would contain the digital data from the first stimulation period of the 15 per second stimulation rate. Also, data points may be omitted to decrease the sample rate. As described previously, the real-time sample rate was 750 points per second; however, when the merged tape was generated, every seventh point was taken which reduces the sample rate to 107 points per second.

These data are now ready for processing on the IBM 1620 according to the methods described in the next section. Approximately four hours of computer time are required for processing one channel for one stimulation rate. Nine stimulation rates were processed per channel, which requires 36 hours. For all four channels this requires approximately 144 hours of computer time.

Data were acquired on three male subjects. Subject number 1 is a 23-year-old college senior with no history of brain trauma or neurological disease. He has a stammer which varies in severity with psychological stress. Subject number 2 is a well-studied 32-year-old epileptic patient with psychomotor seizures. He has a left temporal EEG abnormality. His medical history indicates seizures started after injuries were received in a childhood bicycle-automobile accident. He is a high school graduate and started college but dropped out in the first year. He is employed by the University Hospital as a laboratory technician. Subject number 3 is a 22-year-old medical student with no significant medical or psychological history.

Mathematical Processes

Initial efforts in the search for an analytic process were in the direction of correlation analysis of the EEG.

Correlograms of signals having many frequencies are very difficult to evaluate. They will emphasize predominant or fundamental frequencies, and with a little practice other frequency components can be discerned. They seemed inadequate and inconclusive as a diagnostic test, so the search for a better method was continued.

Power spectral density analysis was investigated and proved to be of greater value than correlation analysis but added considerable computational complexity. It was questionable that the power spectral density analysis yielded enough information to justify the additional computation. However, with a minor amount of additional analysis to compute coherence and transfer ratios, a significant advance was made in the amount of information produced by the data processing. It also provides an opportunity to gain a little insight as to the characteristics of the system which produced the data.

The light flash is considered to be the input to a system; and the EEG, as recorded on the scalp at both the occipital cortex and temporal lobes, is considered as the system output. Transfer ratios, phase information, cross-power spectral density, and coherence values can be obtained from these data by the following methods.

A system can be analyzed by several methods: (1) using a continuous input of varying frequency and measure the output magnitude and phase, (2) impulse response, (3) step response, (4) correlation analysis, and (5) power spectral density analysis. Lee (20) presents derivations of all but the first of these methods in Chapter 13. Here it is shown the system transfer function $H(\omega)$ can be obtained by

$$H(\omega) = \int_{-\infty}^{+\infty} h(t)\, e^{-j\omega t}\, dt \qquad (1)$$

where h(t) is the system unit impulse response. Conversely, by Fourier transformation, the unit-impulse response can be obtained from

$$h(t) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} H(\omega)\, e^{j\omega t}\, d\omega \quad . \tag{2}$$

Lee also shows how the autocorrelation of a system output $\phi_{oo}(\tau)$ can be determined by first autocorrelating both the system unit impulse response $\phi_{hh}(t)$ and the system input $\phi_{ii}(t)$ and then cross-correlating these two autocorrelations. In equation form this becomes

$$\phi_{oo}(\tau) = \int_{-\infty}^{+\infty} \phi_{hh}(t)\, \phi_{ii}\,(t + \tau)\, dt \quad . \tag{3}$$

In the frequency domain similar relations exist and are conveniently expressed in terms of the power spectral density. The output power spectral density $\Phi_{oo}(\omega)$, input power spectral density $\Phi_{ii}(\omega)$, and transfer function $H(\omega)$ are related by

$$\Phi_{oo}(\omega) = |H(\omega)|^2\, \Phi_{ii}(\omega) \tag{4}$$

and if the cross-power spectral density between output and input is used, this relation becomes

$$H(\omega) = \frac{\Phi_{io}(\omega)}{\Phi_{ii}(\omega)} \tag{5}$$

By use of Equation 5, it is possible to test a system in operation without disturbing it.

Another simple but important equation can also be derived which shows the cross-correlation between the system input and output to be

equal to a constant times the system unit impulse response when white noise is used as an excitation.

$$\phi_{io}(\tau) = 2\pi \, Kh(\tau) \tag{6}$$

Here, $K = \Phi_{ii}(\omega)$, which is a constant for white noise. It should also be noted Equation 6 permits the system unit impulse response to be determined in the presence of other disturbances so long as they are unrelated to the white noise excitation.

The procedure followed in this thesis is based on the use of Equation 5 with a stationary input of periodic light flashes at different flash rates. To implement Equation 5, the power spectral density of the input and the cross-power spectral density between output and input must be determined.

If the EEG data are regarded as a stationary time series $f_1(t)$, the autocorrelation $\phi_{11}(\tau)$ of this time function is determined by

$$\phi_{11}(\tau) = \lim_{T \to \infty} \frac{1}{2T} \int_{-T}^{+T} f_1(t) \, f_1(t + \tau) \, dt \tag{7}$$

(where τ is a continuous time displacement) which produces a filtered version of the original time function. However, all phase relations are lost and all components appear as cosine waves having a maximum value at $\tau = 0$. The Fourier transformation of the autocorrelation function produces the power spectral density

$$\Phi_{11}(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \phi_{11}(\tau) \, e^{-j\omega\tau} \, d\tau \tag{8}$$

Since the autocorrelation function is an even function, it may be

expressed as the real portion of Equation 8 by making a cosine transformation

$$\Phi_{11}(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \phi_{11}(\tau) \cos \omega\tau \, d\tau \quad . \tag{9}$$

The first step in acquiring cross-power spectral density is to produce the cross-correlogram between input and output by

$$\phi_{12}(\tau) = \lim_{T \to \infty} \frac{1}{2T} \int_{-T}^{T} f_1(t) \, f_2 \, (t + \tau) \, dt \quad . \tag{10}$$

In the same fashion as the auto-power spectral density was obtained for a single time series, the cross-power spectrum $\Phi_{12}(\omega)$, for two time functions (input and output), is produced by

$$\Phi_{12}(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \phi_{12}(\tau) \, e^{-j\omega\tau} \, d\tau \quad . \tag{11}$$

The cross-correlation function is not necessarily an even function; thus, the cross-power spectrum will, in general, have real and imaginary components which may be produced by making a cosine and sine transformation, respectively.

$$\text{Re } \Phi_{12}(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \phi_{12}(\tau) \cos (\omega\tau) \, d\tau \tag{12}$$

$$\text{Im } \Phi_{12}(\omega) = \frac{1}{2\pi} \int_{-\infty}^{+\infty} \phi_{12}(\tau) \sin (\omega\tau) \, d\tau \quad . \tag{13}$$

The magnitude and phase relation θ of $\Phi_{12}(\omega)$ can be calculated

in the usual way

$$\Phi_{12}(\omega) = \sqrt{[\text{Re } \Phi_{12}(\omega)]^2 + [\text{Im } \Phi_{12}(\omega)]^2} \tag{14}$$

$$\theta = \tan^{-1} \frac{\text{Im } \Phi_{12}(\omega)}{\text{Re } \Phi_{12}(\omega)} \tag{15}$$

The preceding equations are those required to calculate the transfer function $H(\omega)$ in Equation 5. Underlying assumptions upon which the derivation of all the foregoing relations rely are those of time stationarity and linearity. In actual practice the signal is not available for analysis from $-\infty$ to $+\infty$. When working with signals of finite duration, these processes produce estimates of the quantities involved. Sato (21) and Grindel (22) investigating the significance of short data samples found that a five-second segment of EEG data permits analysis possessing definite physiological value.

Most physiological systems are nonlinear, and most sensory systems approximate the Weber-Fechner law. This law relates a quantifiable change in a stimulus intensity to its perception by an observer. In these experiments the photic intensity was held constant and only the flash rate varied. The nonlinearities involved are not necessarily the same as those determined by varying the intensity of a constant stimulus. As is so often necessary, linear methods are used in the hope that the nonlinearities will not render the results meaningless. Correlation and spectral analysis are of proven value in other physiological data analyses. There is much evidence to suggest these techniques should serve equally well in the analysis of provoked

electrical activity of the brain.

The mathematical processes enumerated in the preceding pages are for continuous data systems. This implies that only analog techniques are applicable, which is true for the continuous data processes. However, sampled data and digital computing processes can be applied if the continuous signals are replaced by discrete values taken at equal sample intervals and integrations are replaced by summations. The choice of analog or digital process is dictated by preference or available equipment. In these experiments digital processing was used.

Jenkins (23) has presented derivations for discrete data analysis, and they are clearly presented from a statistician's background. Blackman and Tukey (24) also derive and discuss spectral analysis using discrete data. Walters (25) presents a discourse which produces formulations which lend themselves to format necessities of the modern digital computer. The more common mathematical symbols are replaced by abbreviations which are more compatible with characters available on computer output printers. These equations are summarized below and are the ones upon which the digital computer programs are based.

Prior to processing, the data are standardized by replacing the value of each sample point X_n by X(i) where

$$X(i) = \frac{[X_n - \bar{X}]}{S} \tag{16}$$

and

$\bar{X}$ = mean of the set of data points

S = standard deviation of the set of data points.

Calculation of the autocorrelation AC(L) is accomplished by

$$AC(L) = \frac{1}{N - L} \sum_{i=1}^{N-L} X(i)\, X(i + L) \tag{17}$$

where

N = total number of data points

L = lag

X(i) = standardized set of data points.

The cross-correlation CCXY(L) for two data channels X and Y is determined by

$$CCXY(L) = \frac{1}{N - |L|} \sum_{i=-b}^{b} X(i)\, Y(i + L) \tag{18}$$

where

b = N - L and negative values of L are allowed

Y(i + L) = standardized set of Y data points.

The formula for the auto-spectrogram AS(X) is

$$AS(X) = \frac{1}{M} \left[AC(0) + 2 \sum_{q=1}^{m-1} AC(q) \cos \frac{q\pi f}{f'} + AC(M) \cos \frac{m\pi f}{f'} \right] \tag{19}$$

where

M = maximum lag

f' = folding frequency

f = frequency point for which the power spectral density is calculated.

The real portion of the cross-power spectrogram, or cospectrum, COSP(f) is given by

$$COSP(f) = \frac{1}{2M}\left[CC(-M)\cos\left(-\frac{m\pi f}{f'}\right) + 2\sum_{q=-b}^{b} CC(q)\cos\left(\frac{m\pi f}{f'}\right) + CC(M)\cos\left(\frac{m\pi f}{f'}\right)\right] . \quad (20)$$

and for the imaginary, or quadrature component QUAS(f), the formula is

$$QUAS(f) = \frac{1}{2M}\left[CC(-M)\sin\left(-\frac{m\pi f}{f'}\right) + \sum_{q=-b}^{b} CC(q)\sin\left(\frac{q\pi f}{f'}\right) + CC(M)\sin\left(\frac{m\pi f}{f'}\right)\right] . \quad (21)$$

Correspondingly, the magnitude MAGS(f) of the complex cross-power spectrogram and its phase angle PHI are computed by

$$MAGS(f) = \sqrt{[COSP(f)]^2 + [QUAS(f)]^2} \quad (22)$$

$$PHI(f) = \arctan\frac{QUAS(f)}{COSP(f)} \quad (23)$$

and finally the transfer function TR(f) is determined from

$$TR(f) = \frac{MAGS(f)}{ASX(f)} \quad (24)$$

Jenkins (23) defined another factor called the coherence COH(f) as the ratio

$$COH(f) = \frac{MAGS(f)}{\sqrt{ASX(f)\ ASY(f)}} \quad (25)$$

This factor is similar to a correlation coefficient between channels defined at a particular frequency, f; e.g., a value of 1 and a phase angle of 0° indicates a high positive correlation. Goodman (26) has derived a means of statistically treating the transfer function and phase angle so upper and lower confidence bounds can be placed on them. The following relations are used in this connection.

$$\sin\theta\ (f) = \sqrt{\frac{1 - COH^2\ (f)}{COH^2\ (f)}\left[(1 - P)^x - 1\right]} \tag{26}$$

where

$x = -M/(N - M)$

P = confidence limit (a value of 0.5 was used in this work).

This is then used to calculate an upper UB(TR) and lower boundary LB(TR) of the transfer ratio and the upper boundary UB(PHI) and lower boundary LB(PHI) of the phase angle PHI(f):

$$UB(TR) = \frac{TR(f)}{1 - \sin\theta\ (f)} \tag{27}$$

$$LB(TR) = \frac{TR(f)}{1 + \sin\theta\ (f)} \tag{28}$$

$$UB(PHI) = PHI(f) + \theta(f) \tag{29}$$

$$LB(PHI) = PHI(f) - \theta(f) \tag{30}$$

Programs to implement the use of these formulae were written in the Alberta System for the IBM 1620 digital computer.

CHAPTER III

COMPUTED RESULTS AND ANALYSIS

From the EEG recorded in the experiments, the following items were computed for each analysis period and for all stimulation rates:

1. Auto spectra of the stimuli
2. Auto spectra of the EEG
3. Cross-power spectra
   (a) Quadrature component
   (b) Real component
4. Coherence
5. Transfer ratio

Only the auto spectra of the EEG has any meaning during the prestimulation and post-stimulation periods. The prestimulation power spectra are presented graphically in Figures 4, 5, 6, and 7 for the first subject; Figures 8, 9, 10, 11 for the second subject; and Figures 12, 13, 14, and 15 for subject number three.

They are presented graphically since the main information provided are basically patterns of activity. The fact that they are graphed in groups of threes has no specific meaning but was simply a convenient grouping which permitted displaying all prestimulation analyses for a given data channel. These analyses were made to examine the variability of the unstimulated EEG both within a subject and between subjects. It provides the background against which subsequent response to stimulation

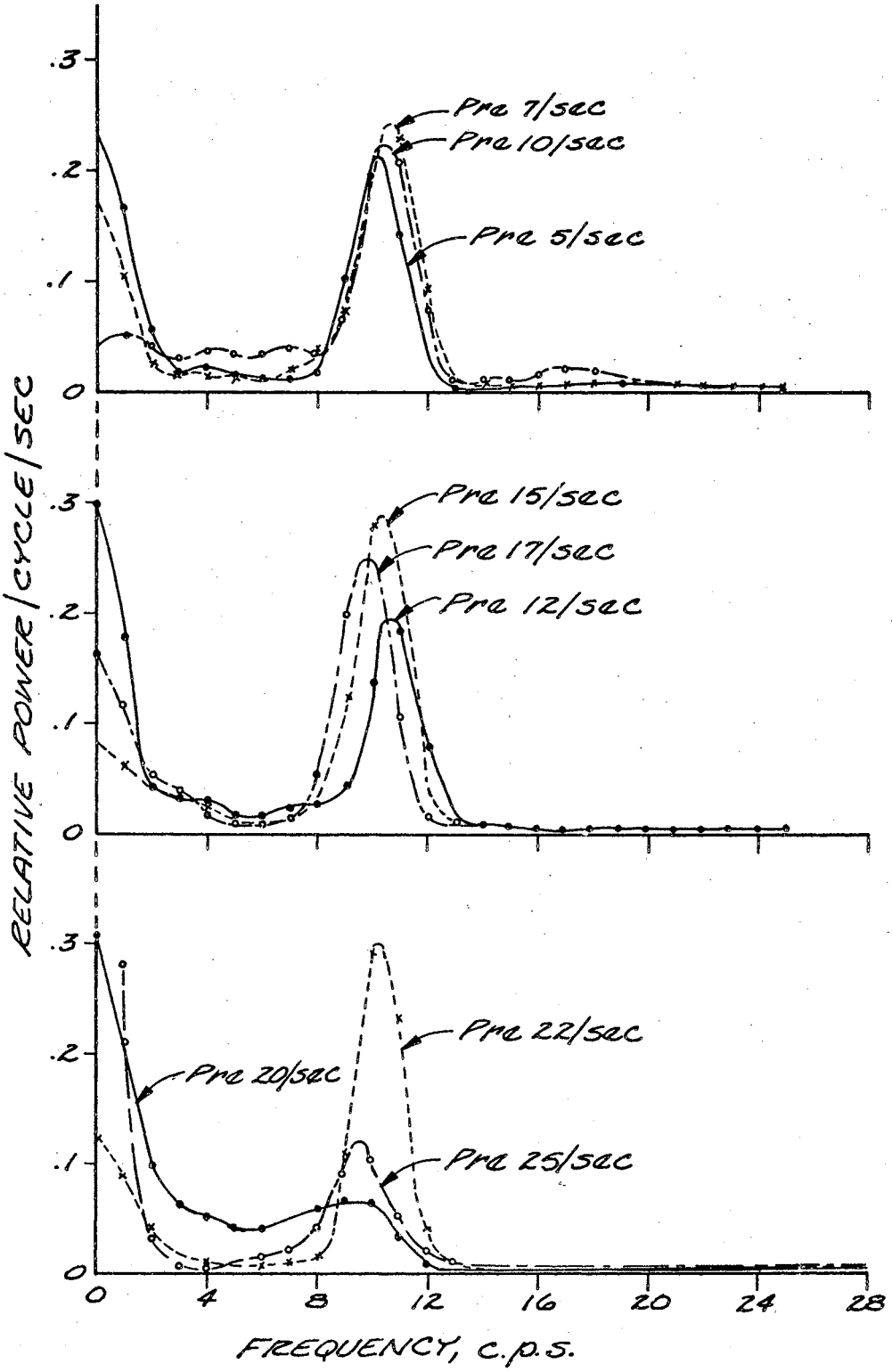

Figure 4. Subject No. 1. Left Occipital Prestimulation Auto Spectra

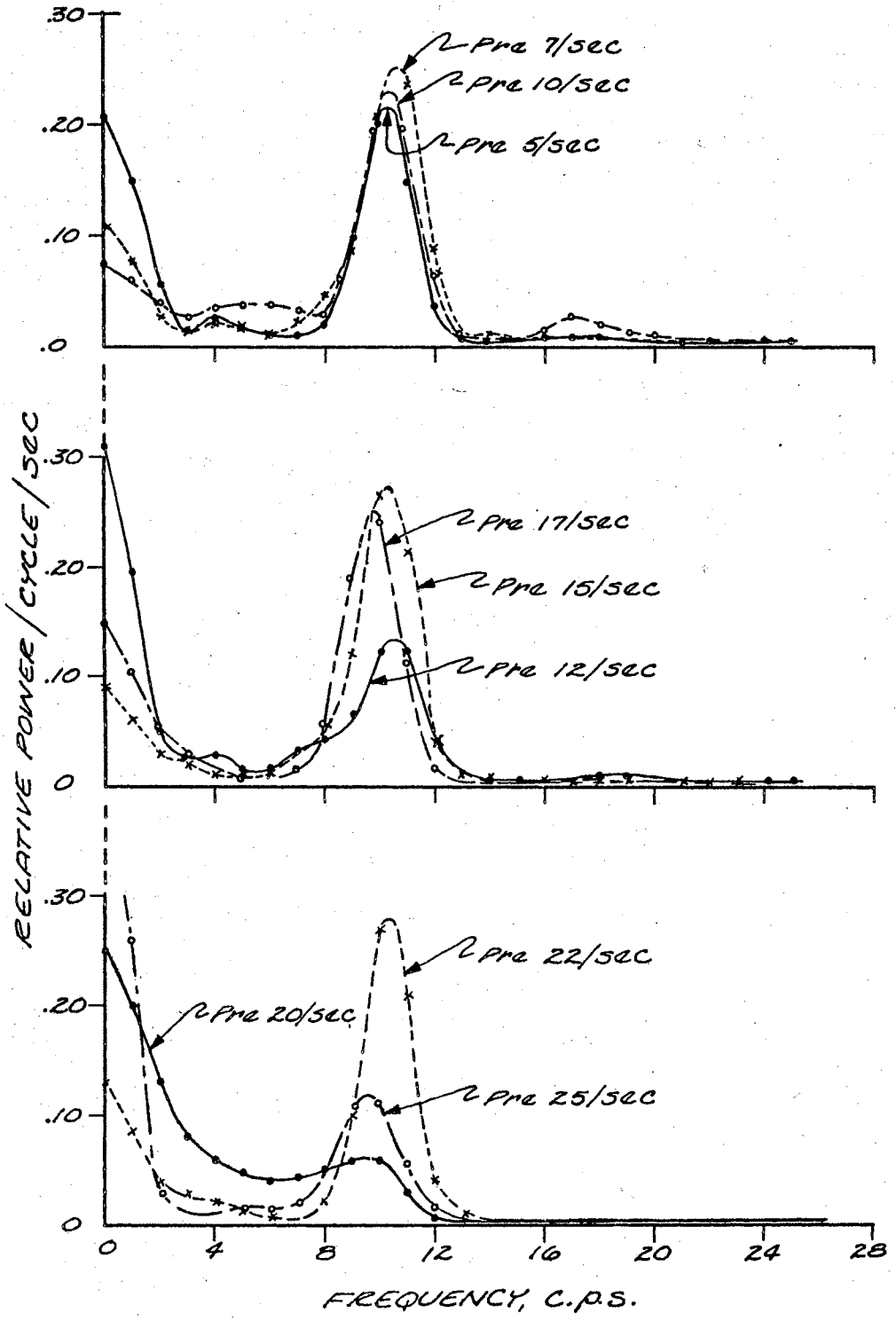

Figure 5. Subject No. 1. Right Occipital Prestimulation Auto Spectra

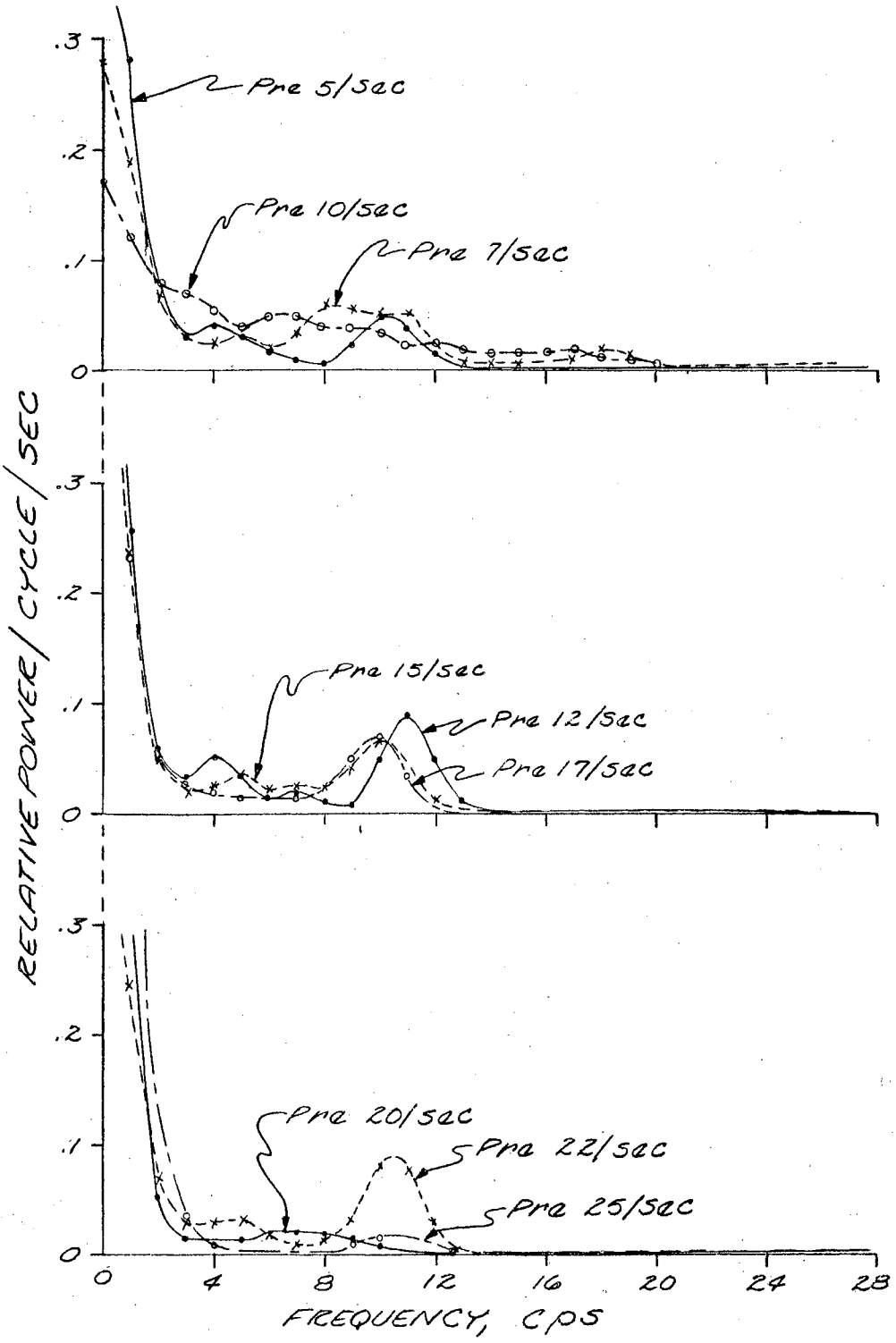

Figure 6. Subject No. 1. Left Temporal Prestimulation Auto Spectra

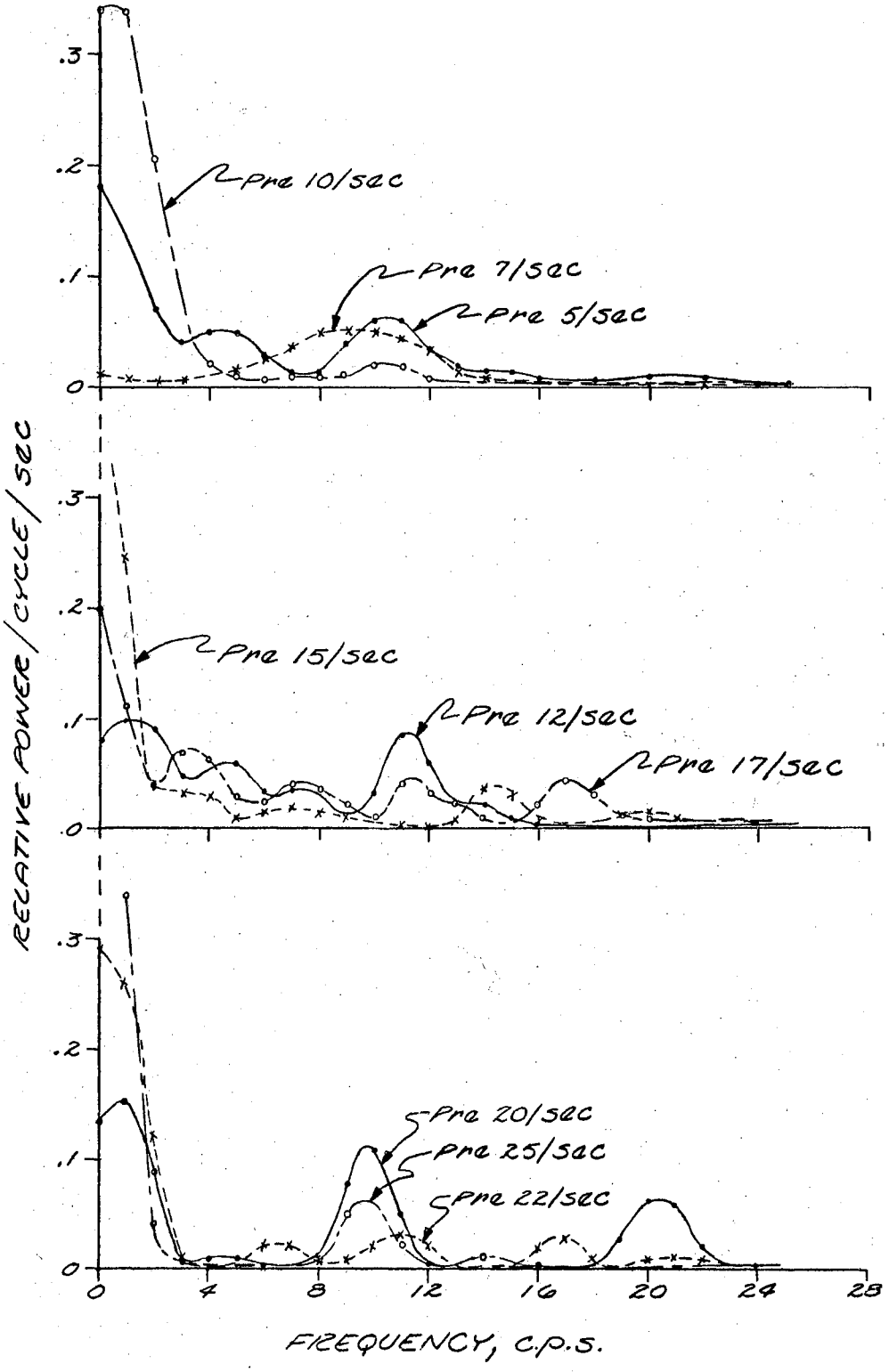

Figure 7. Subject No. 1. Right Temporal Prestimulation Auto Spectra

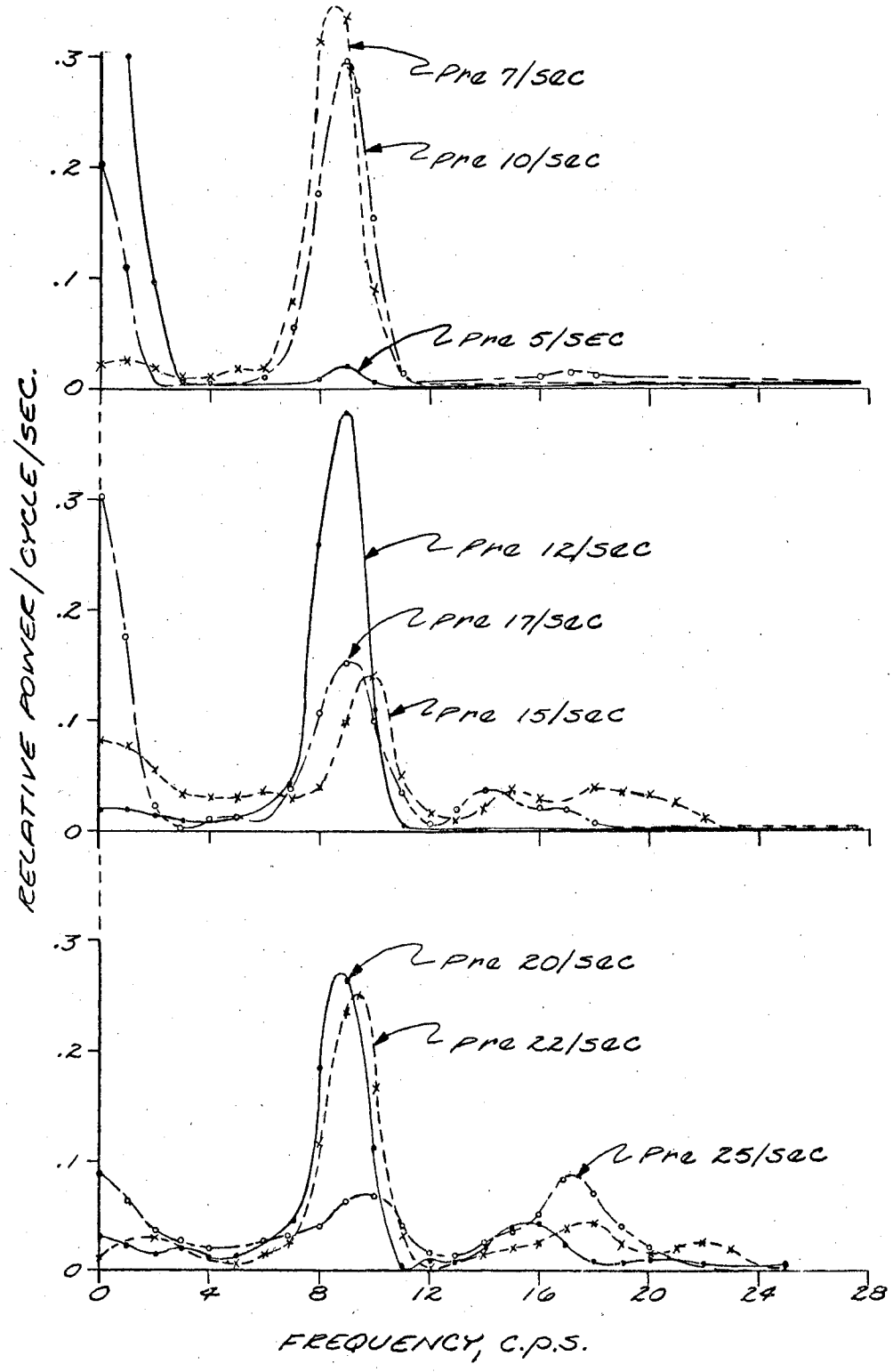

Figure 8. Subject No. 2. Left Occipital Prestimulation Auto Spectra

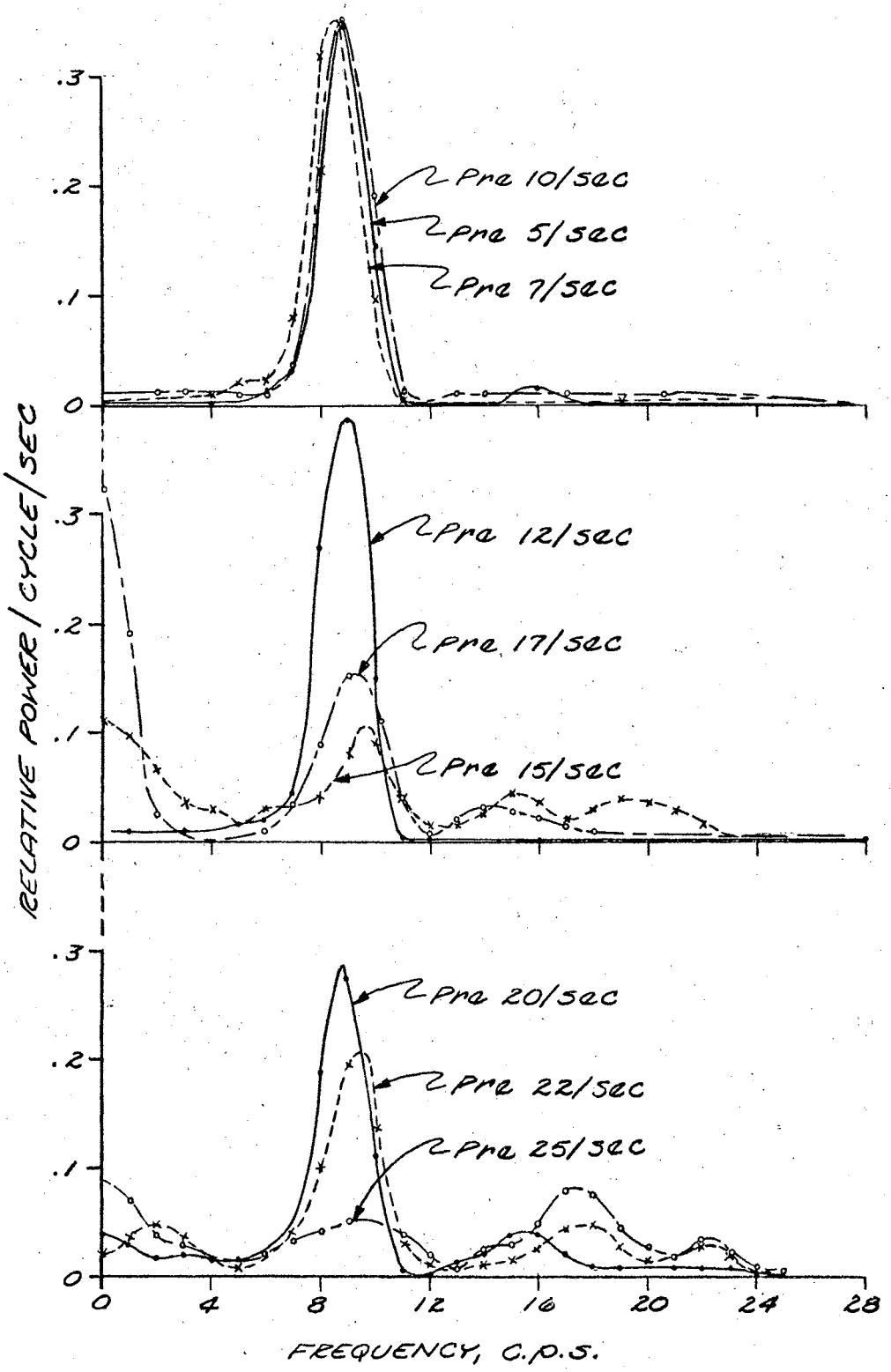

Figure 9. Subject No. 2. Right Occipital Prestimulation Auto Spectra

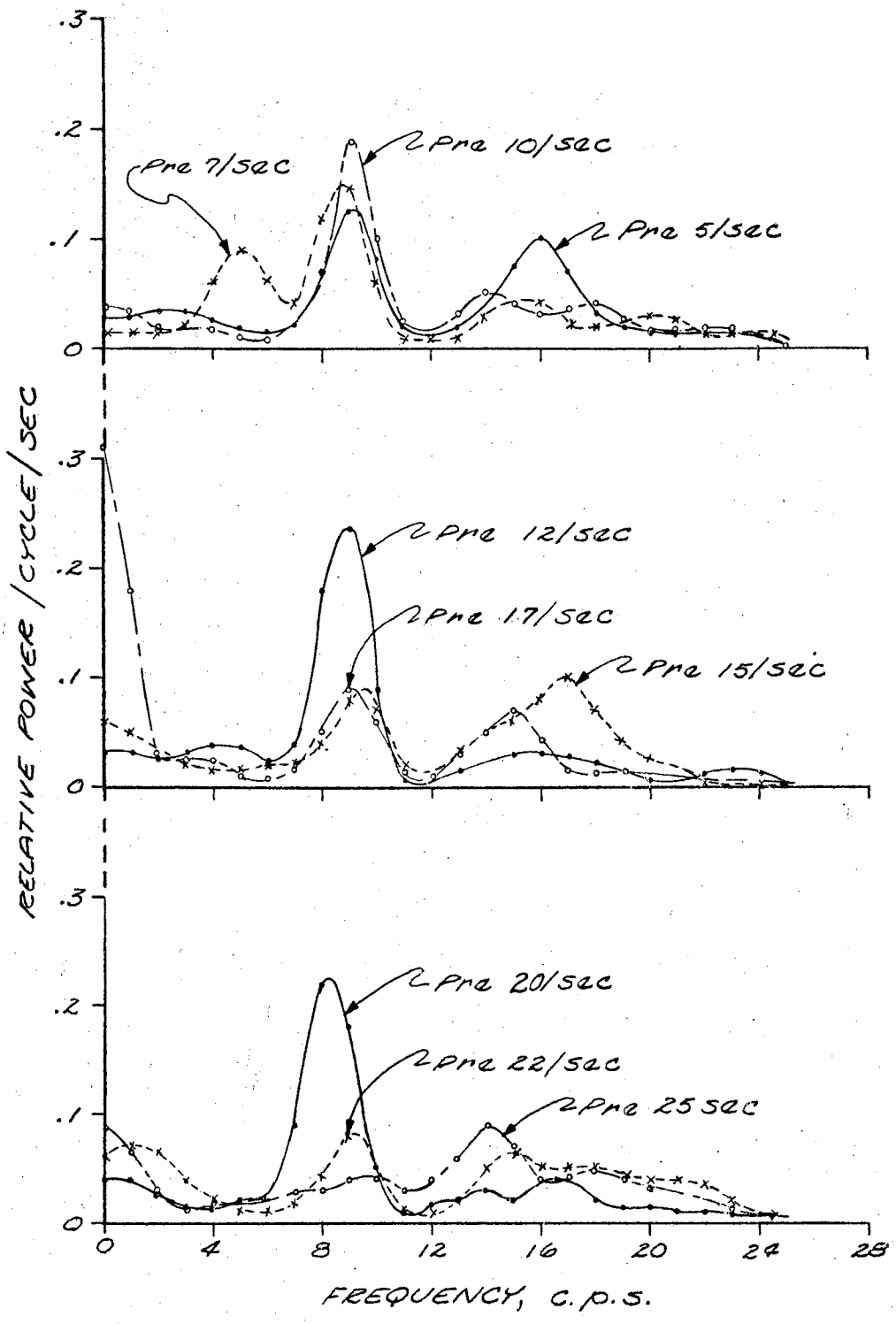

Figure 10. Subject No. 2. Left Temporal Prestimulation Auto Spectra

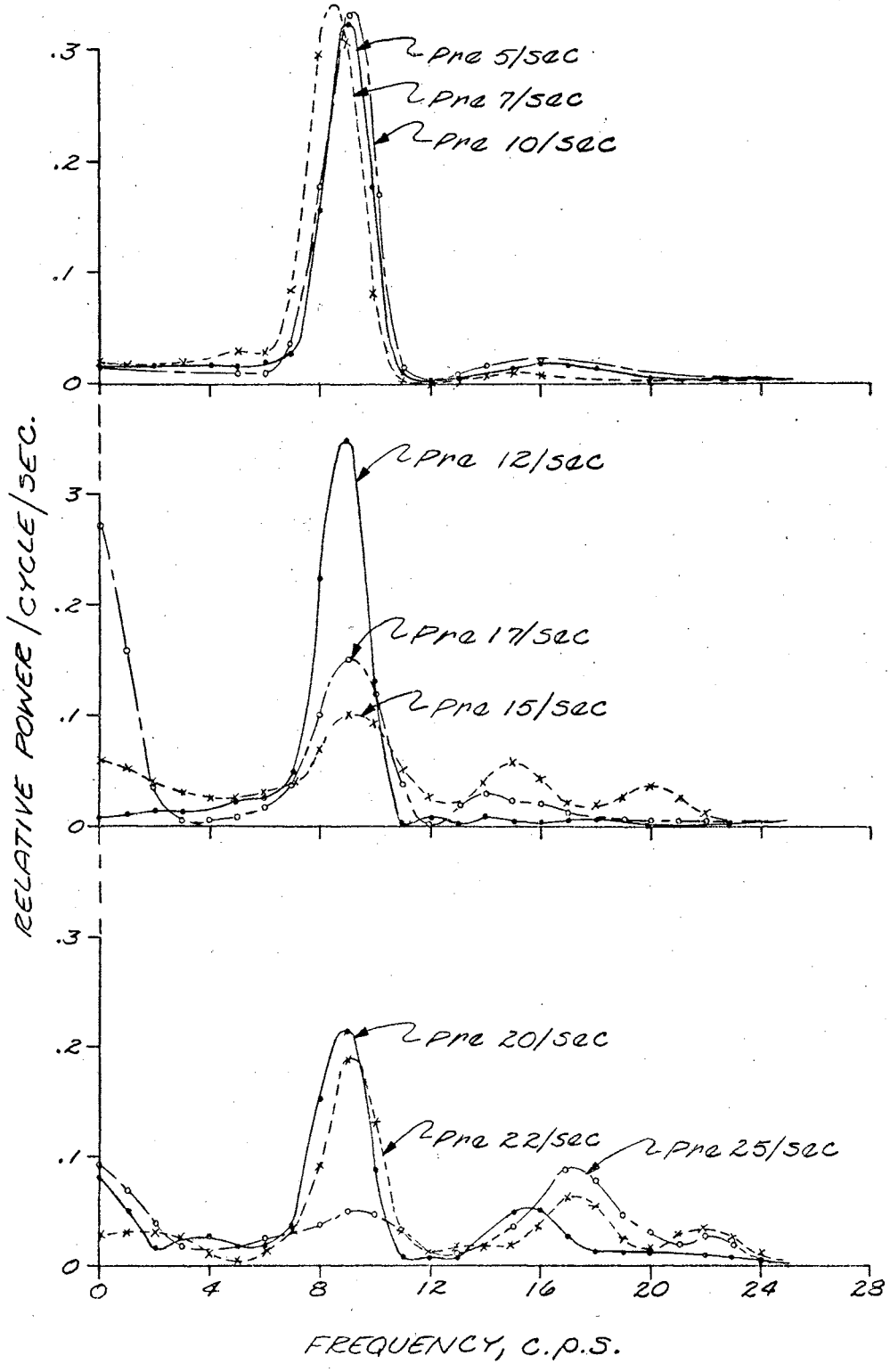

Figure 11. Subject No. 2. Right Temporal Prestimulation Auto Spectra

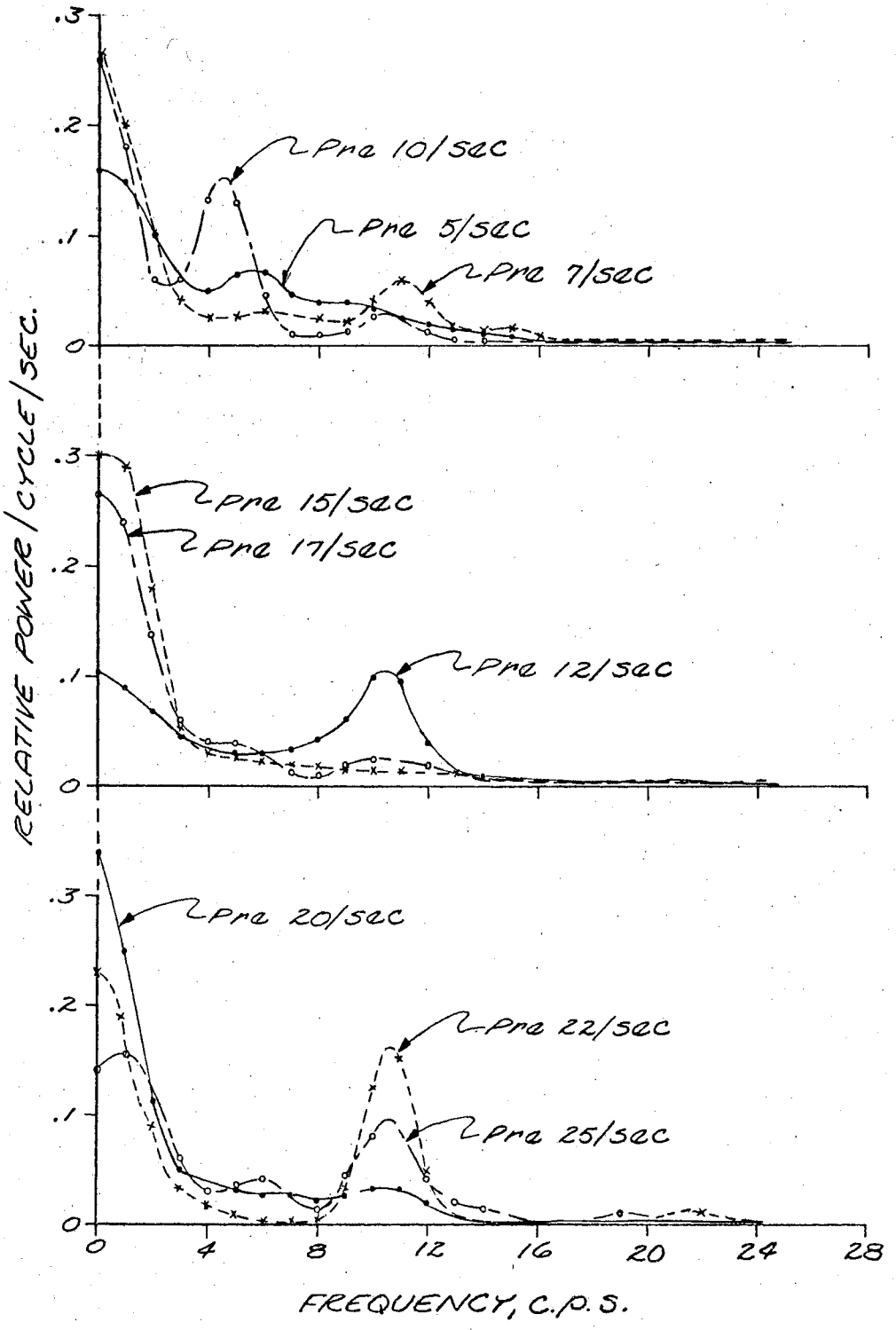

Figure 12. Subject No. 3. Left Occipital Prestimulation Auto Spectra

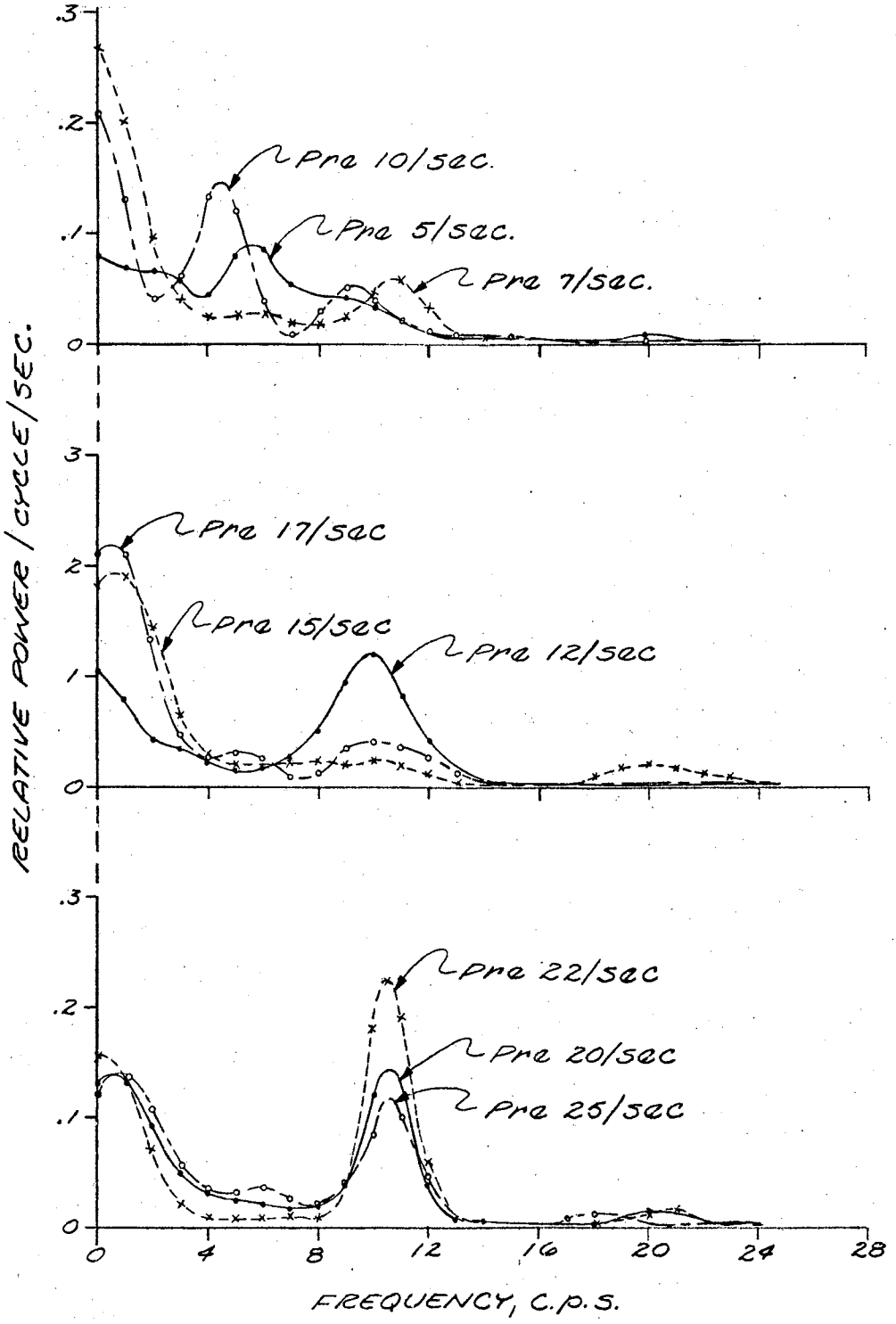

Figure 13. Subject No. 3. Right Occipital Prestimulation Auto Spectra

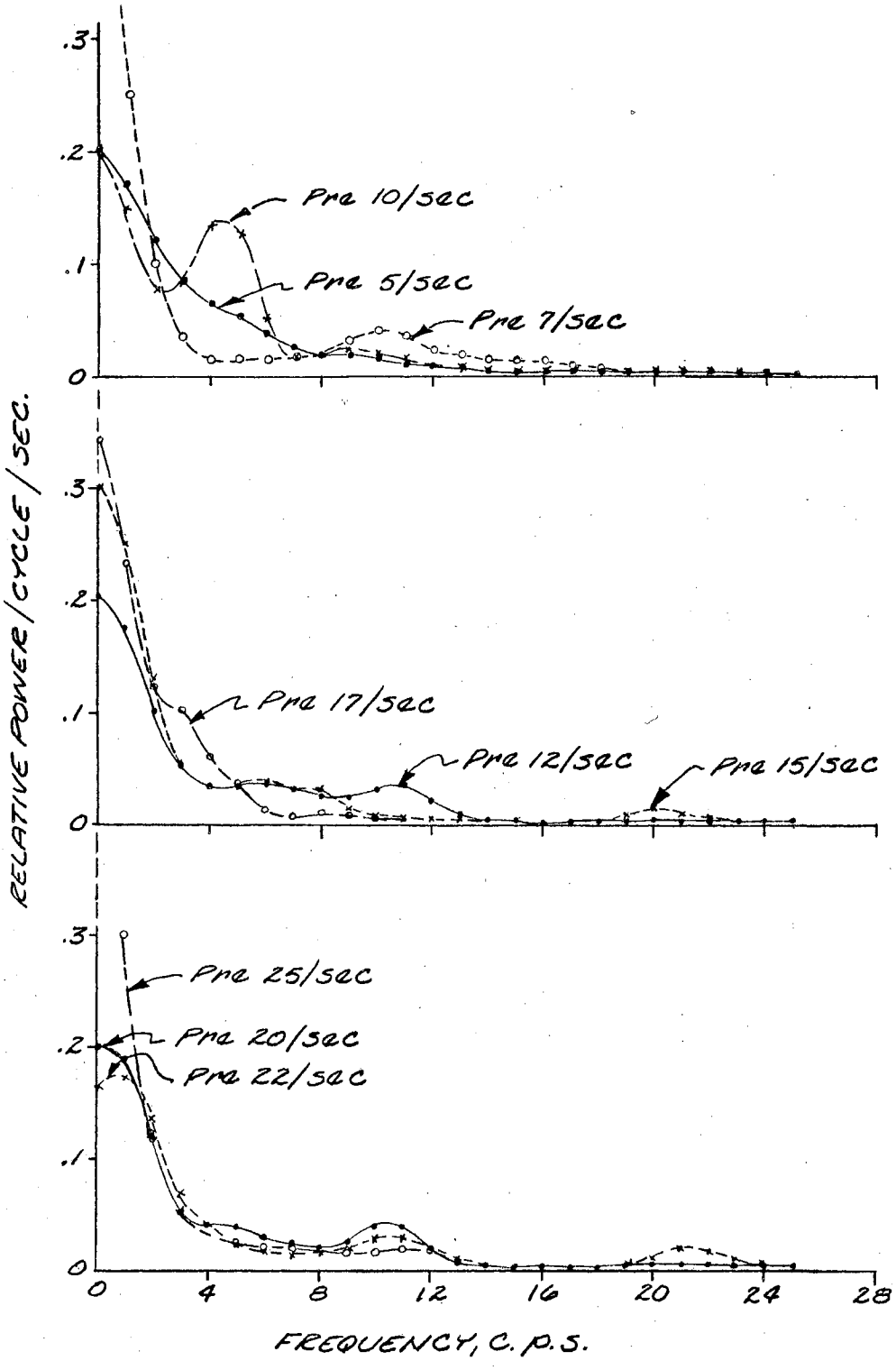

Figure 14. Subject No. 3. Left Temporal Prestimulation Auto Spectra

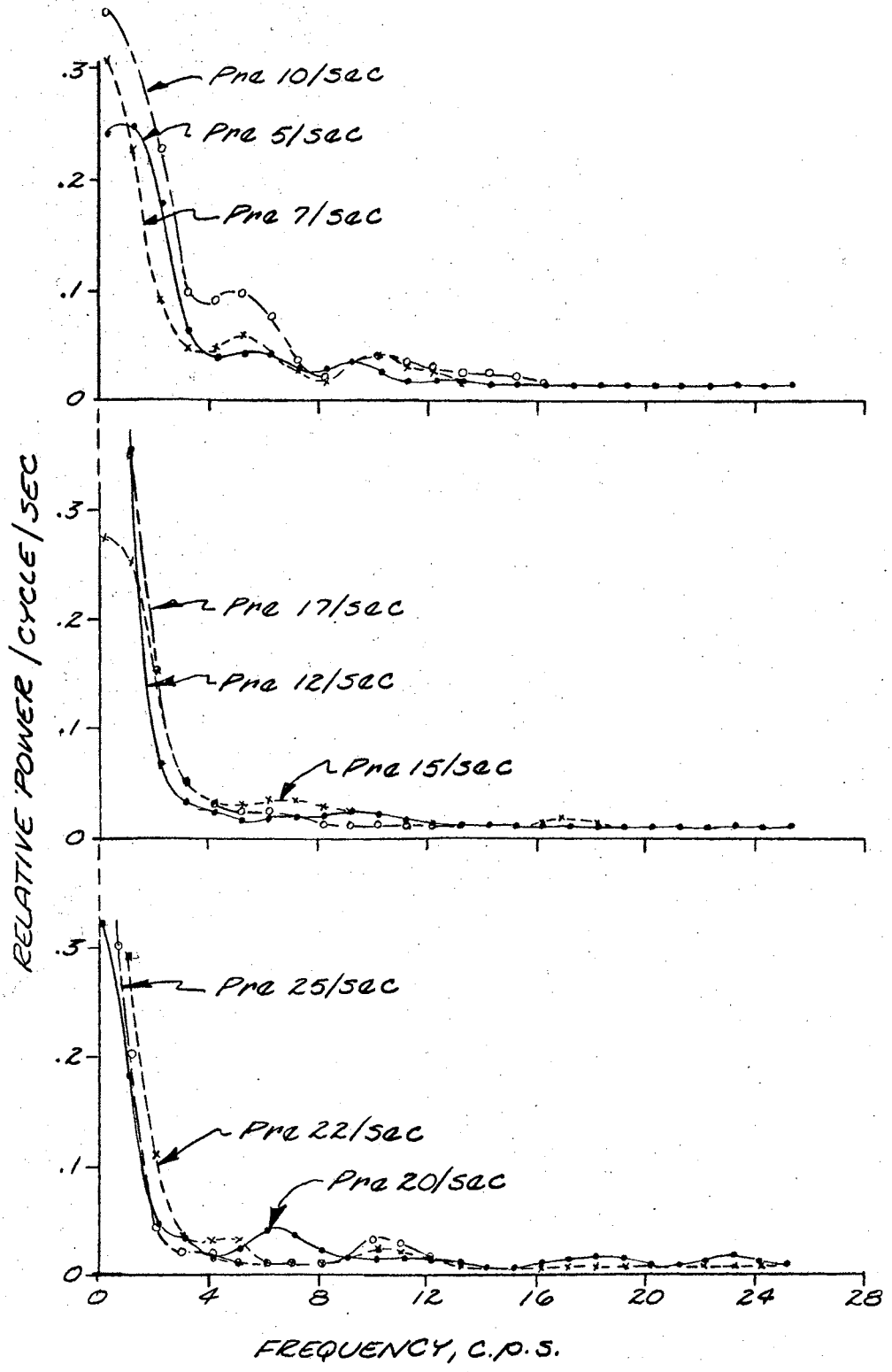

Figure 15. Subject No. 3. Right Temporal Prestimulation Auto Spectra

must be evaluated.

Post-stimulation spectrograms are presented for subject number one in Figures 16, 17, 18, and 19; subject number two in Figures 20, 21, 22, and 23; and subject number three in Figures 24, 25, 26, and 27. These analyses were made to explore the residual effect of the stimulus, i.e., to see if the post-stimulation power spectral density was equivalent to prestimulation power spectral density or if there were lingering effects of the stimulation.

Coherence, as defined by Equation 25 in Chapter II, was computed for each analysis period for every stimulation rate. These are presented in tabular form for the left occipital (LO), right occipital (RO), left temporal (LT), and right temporal (RT) of each subject in Table I. It should be recalled that this is a measure of the extent that the cross-power spectral density at each frequency can be attributed to the stimulation.

Transfer ratios are presented in analogous fashion in Table II. This is the ratio of cross-power spectral density to the stimulation power spectral density at each flash rate. In terms of servomechanism analysis, it is the system gain.

The actual cross-power spectral density magnitude (MAGS), the real component (COSP), and quadrature component (QUAS) are tabulated in Tables III, IV, V, and VI. For ease of comparison, each table lists computed results for the same lead of all three subjects. The left occipital is given in Table III, right occipital in Table IV, left temporal in Table V, and right temporal in Table VI.

These computed data can best be analyzed by making comparisons between right and left leads for each subject and lead-for-lead

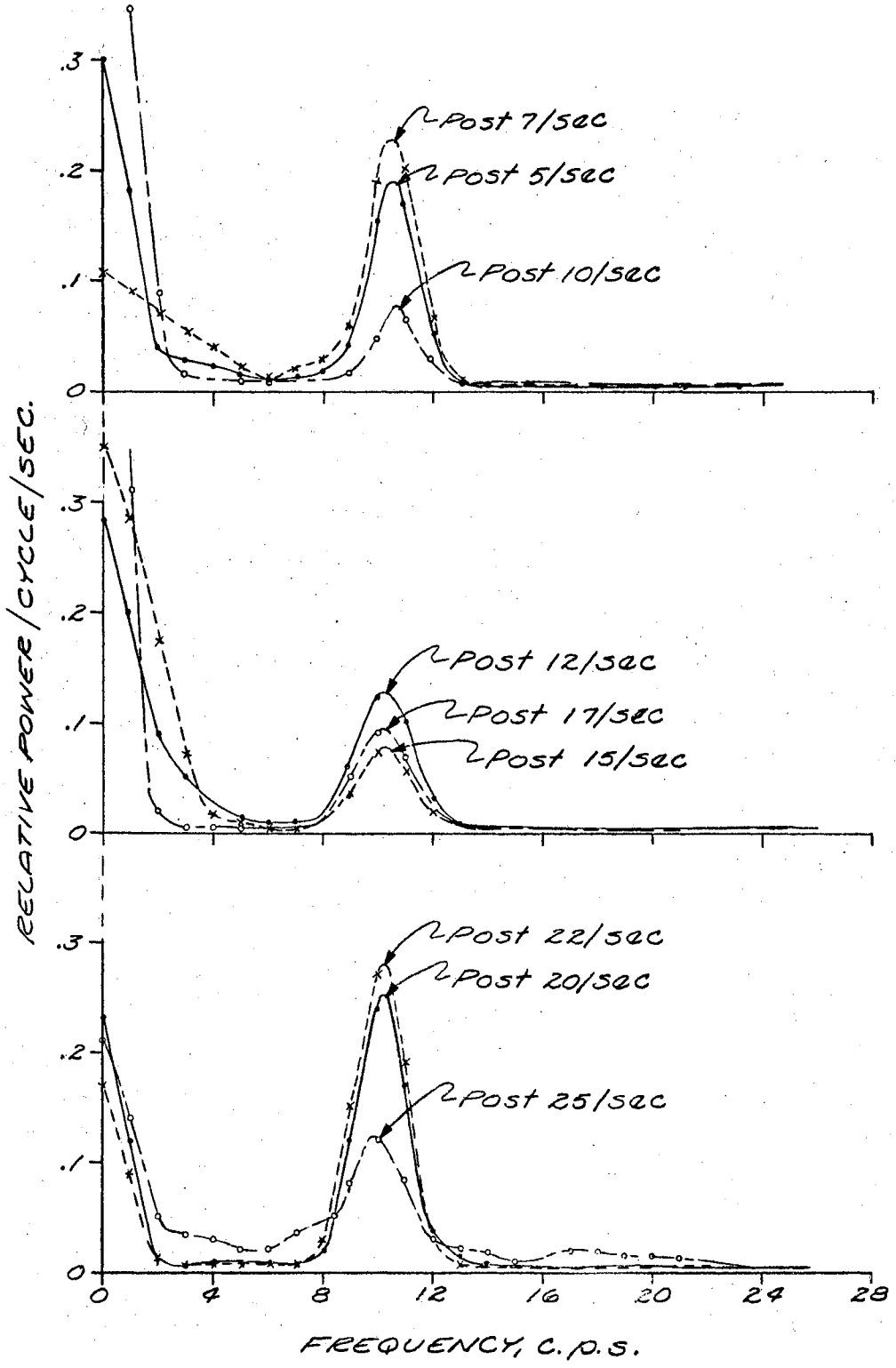

Figure 16. Subject No. 1. Left Occipital Post-Stimulation Auto Spectra

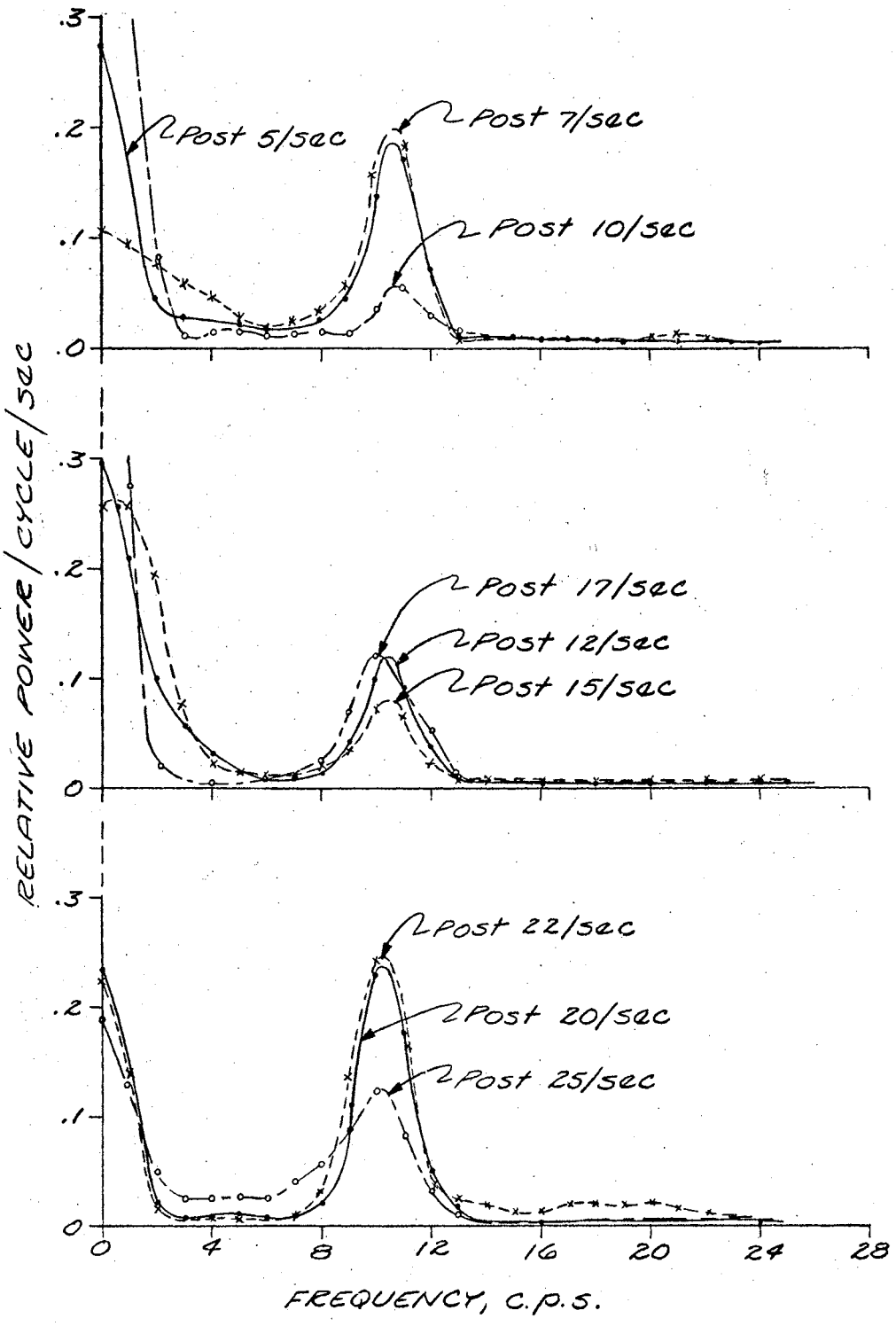

Figure 17. Subject No. 1. Right Occipital Post-Stimulation Auto Spectra

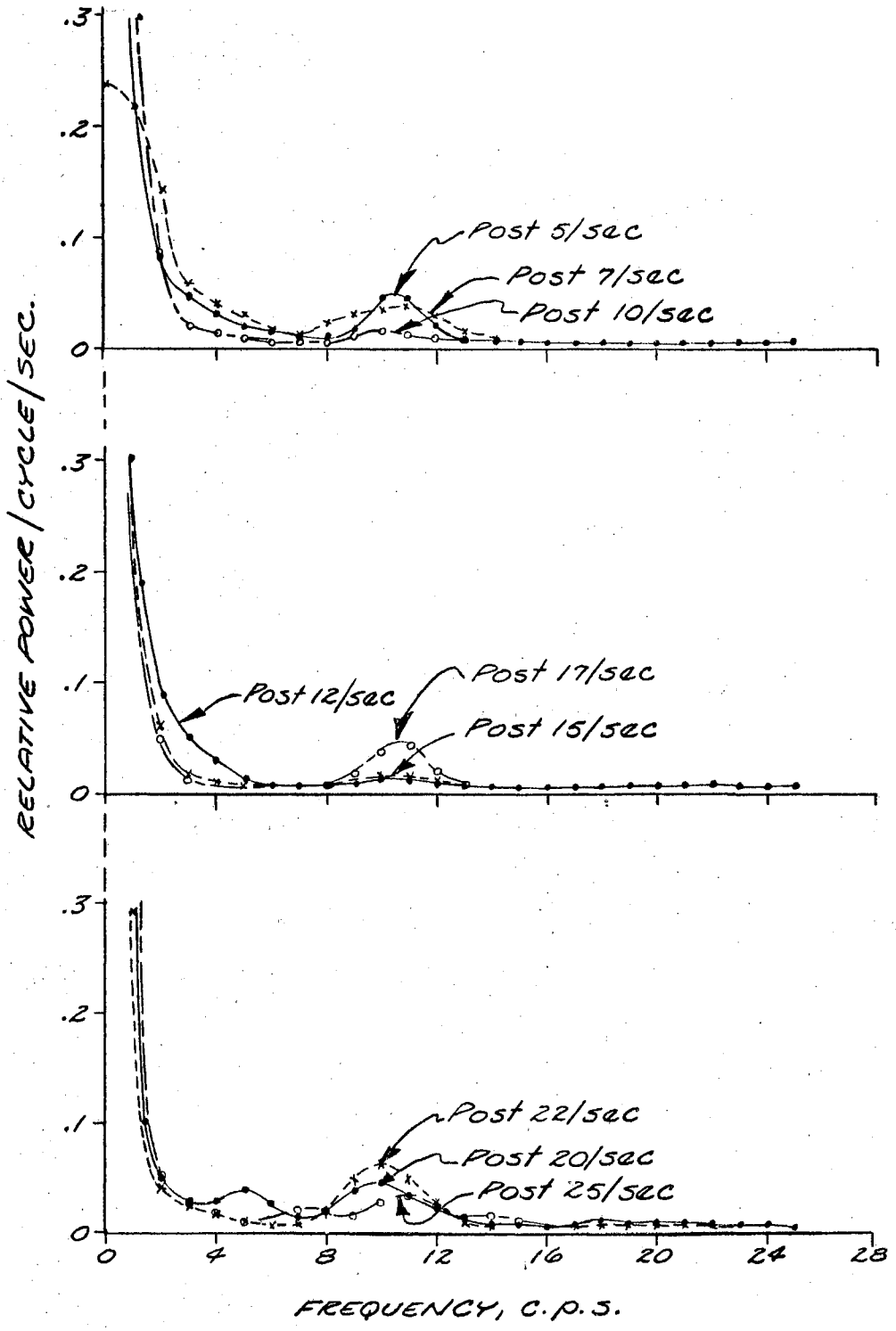

Figure 18. Subject No. 1. Left Temporal Post-Stimulation Auto Spectra

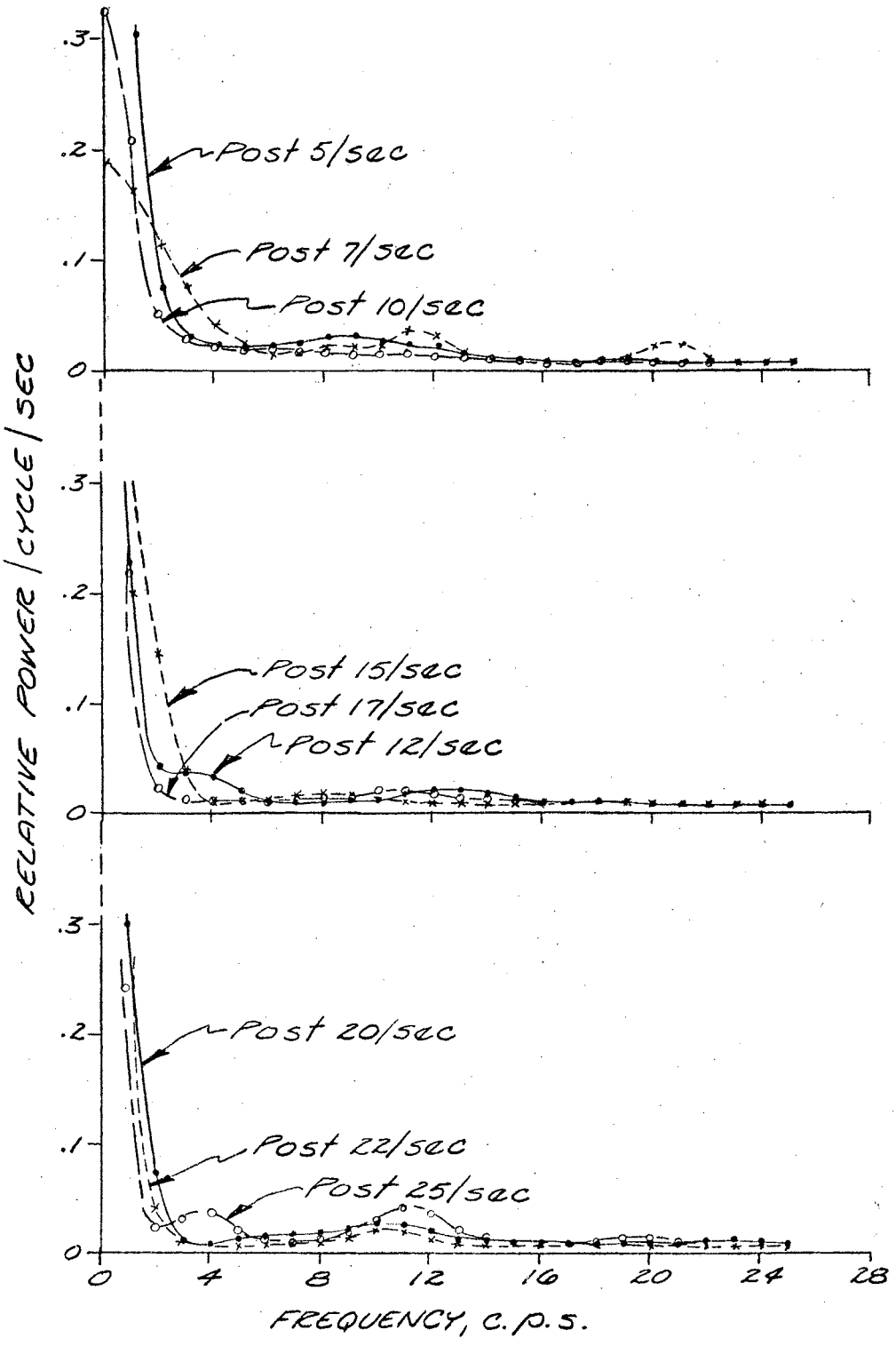

Figure 19. Subject No. 1. Right Temporal Post-Stimulation Auto Spectra

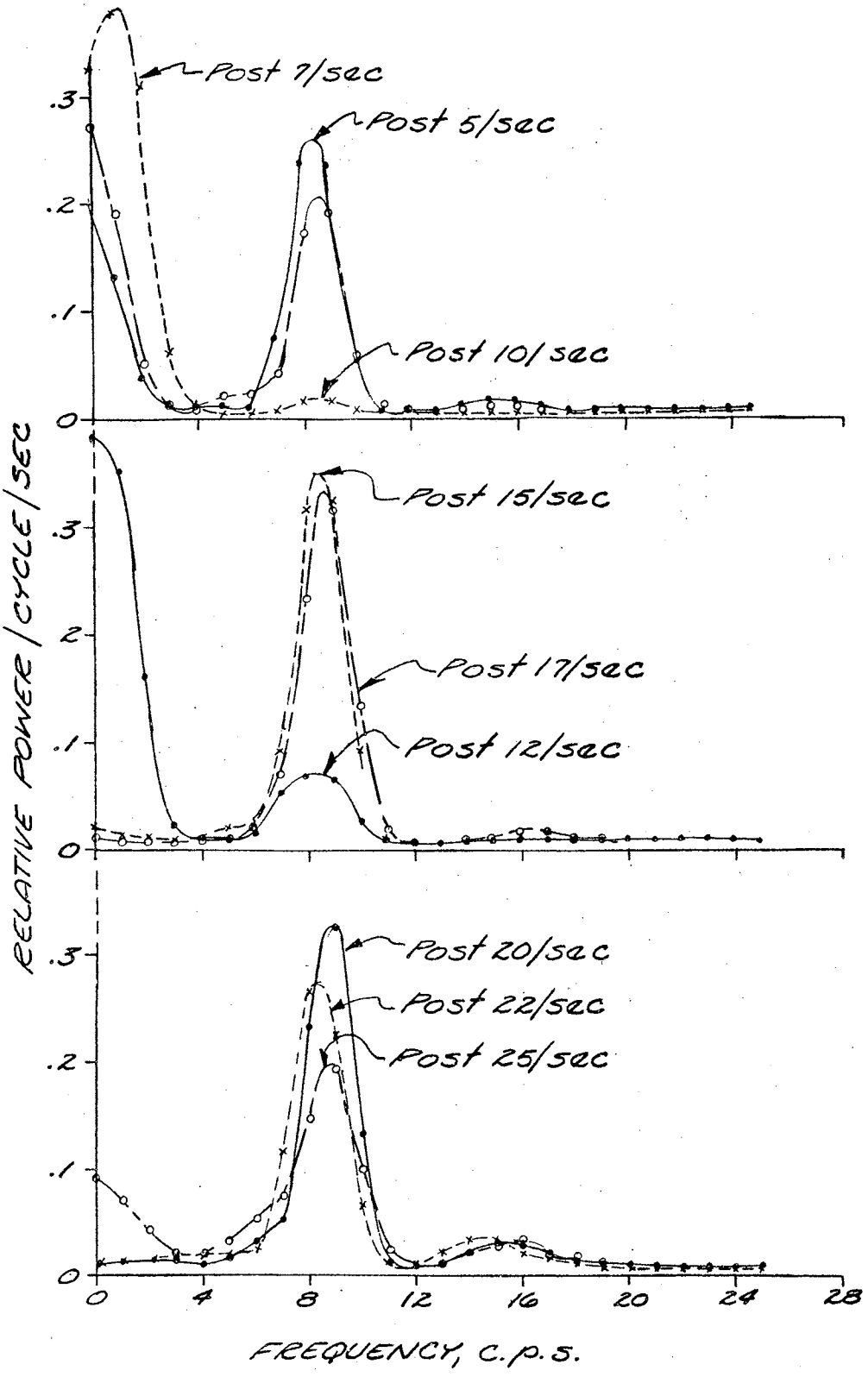

Figure 20. Subject No. 2. Left Occipital Post-Stimulation Auto Spectra

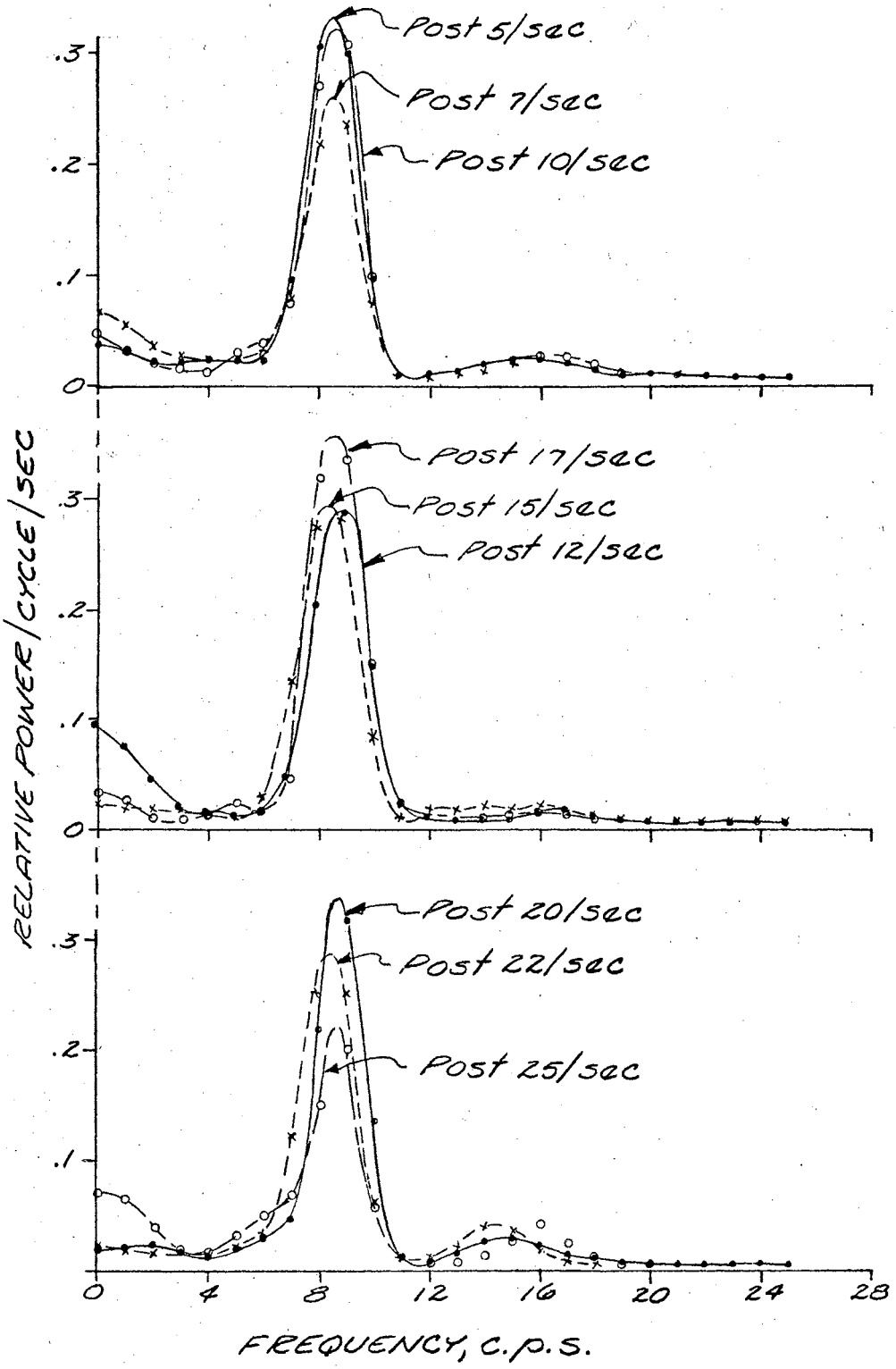

Figure 21. Subject No. 2. Right Occipital Post-Stimulation Auto Spectra

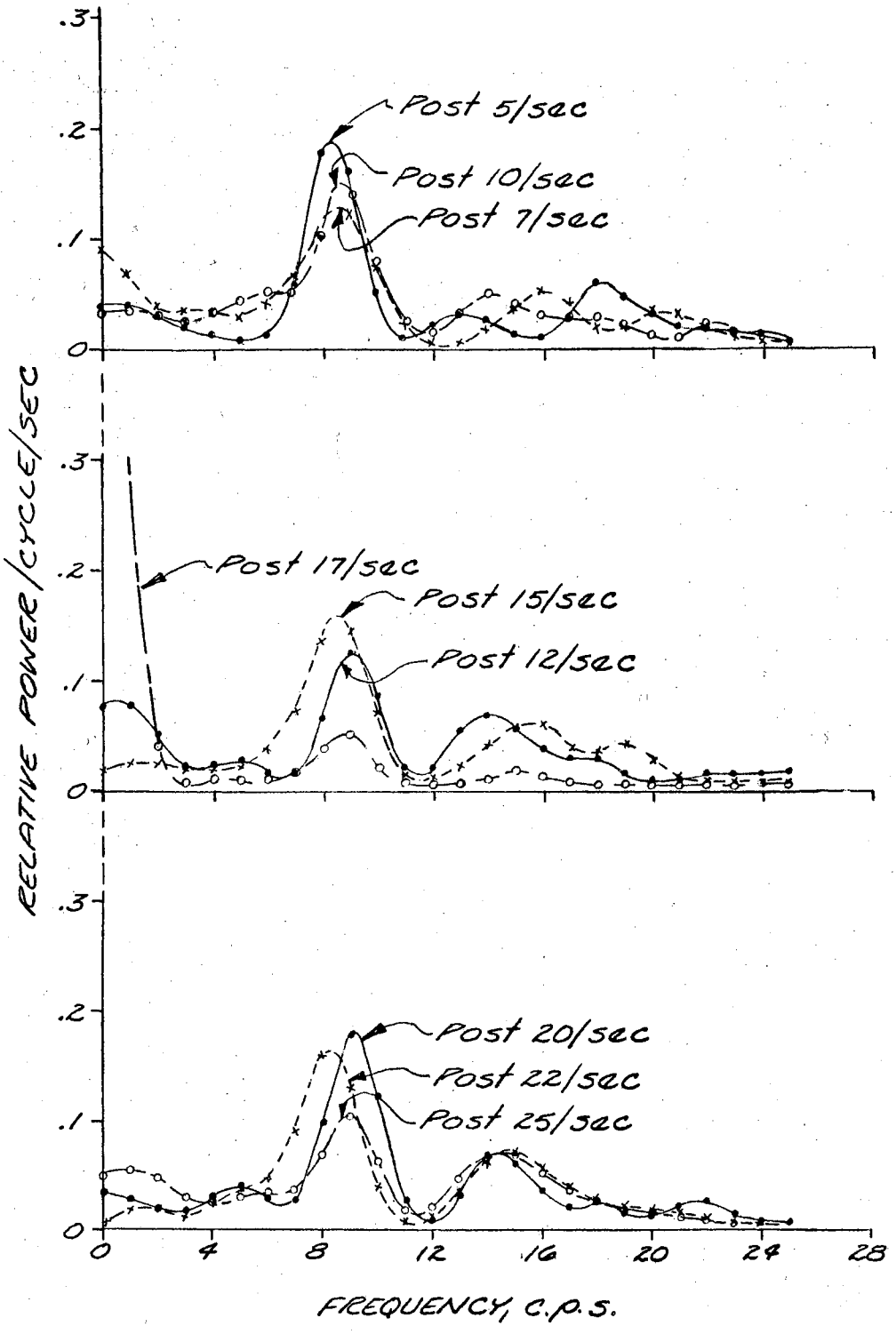

Figure 22. Subject No. 2. Left Temporal Post-Stimulation Auto Spectra

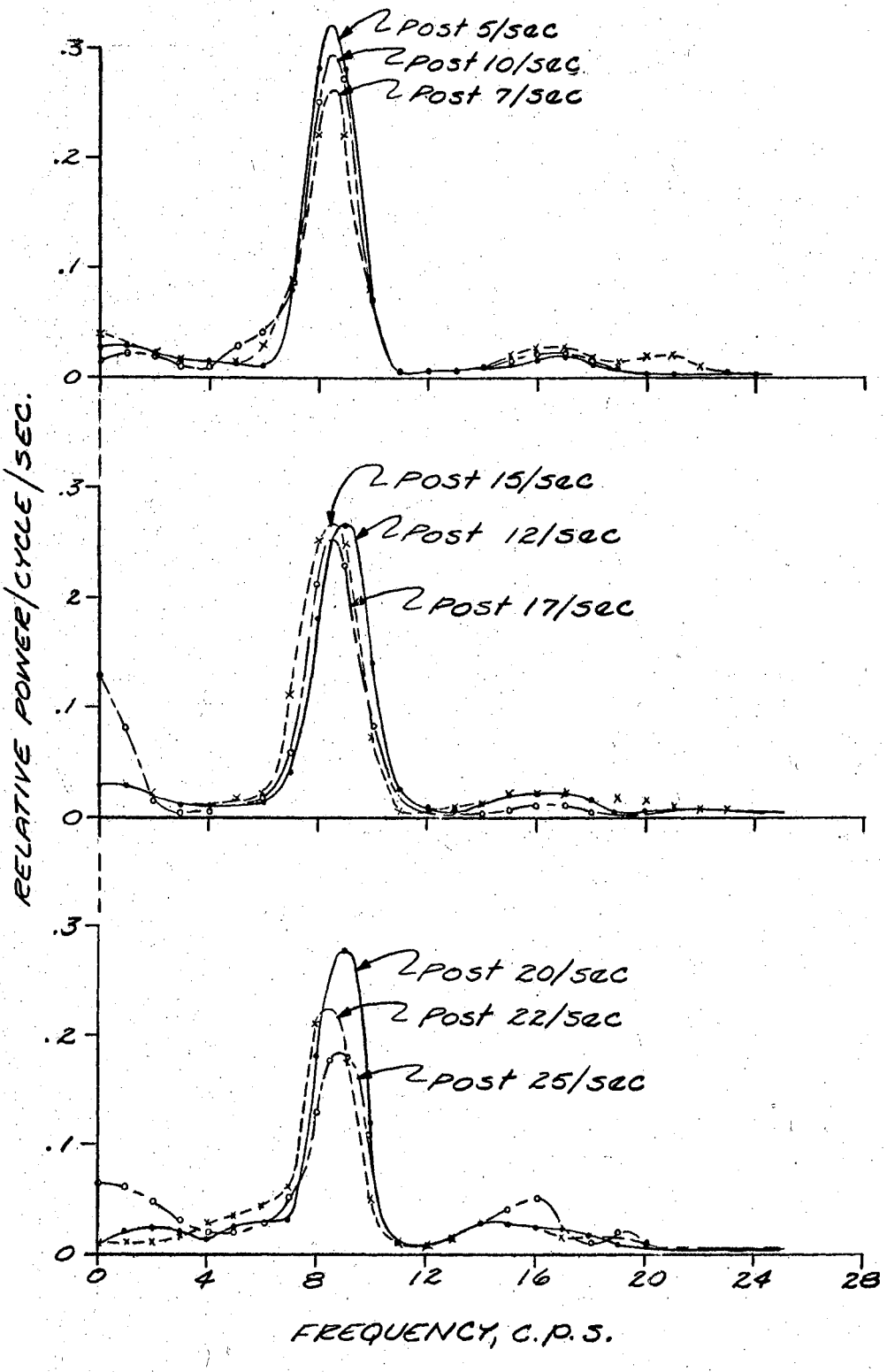

Figure 23. Subject No. 2. Right Temporal Post-Stimulation Auto Spectra

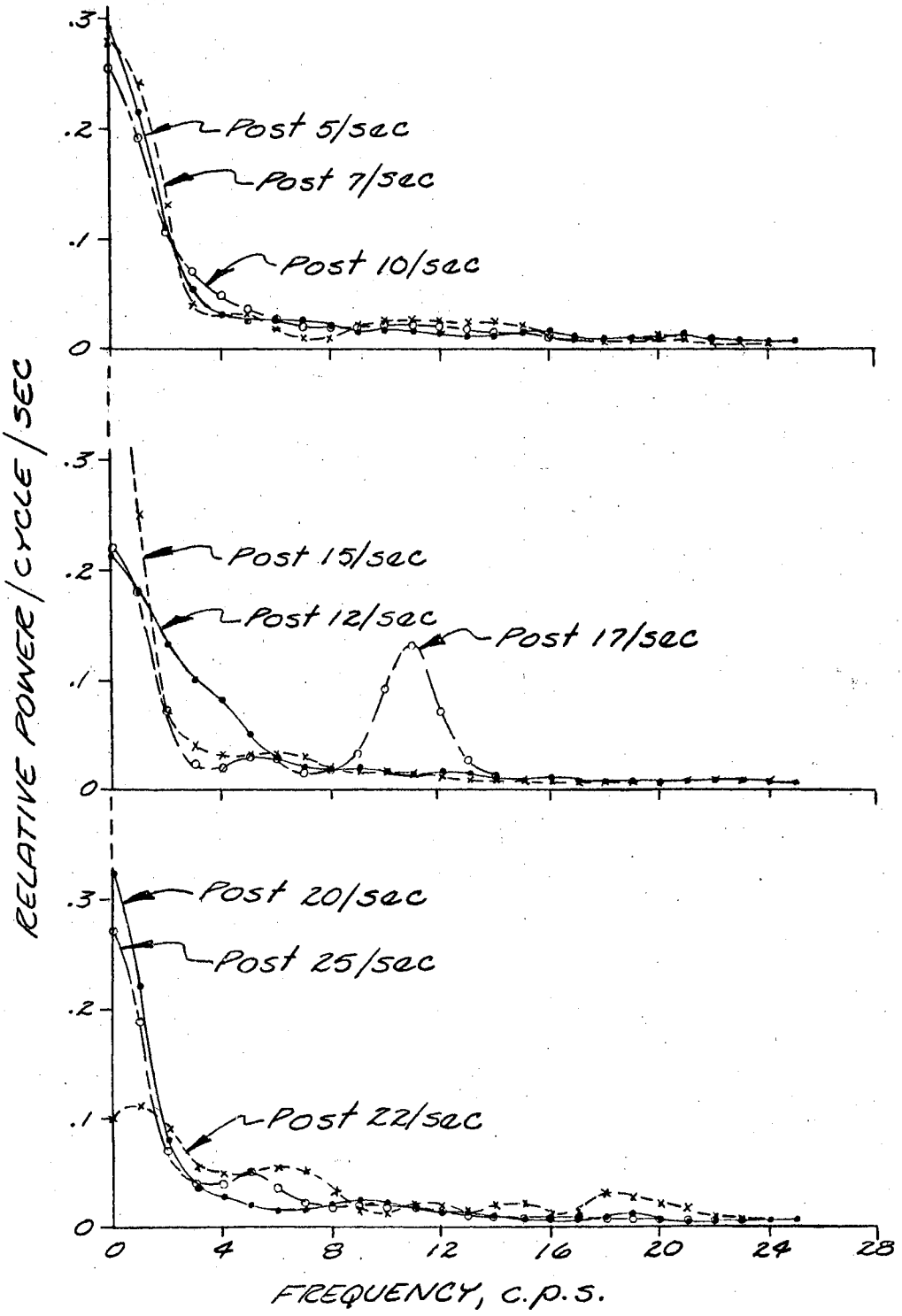

Figure 24. Subject No. 3. Left Occipital Post-Stimulation Auto Spectra

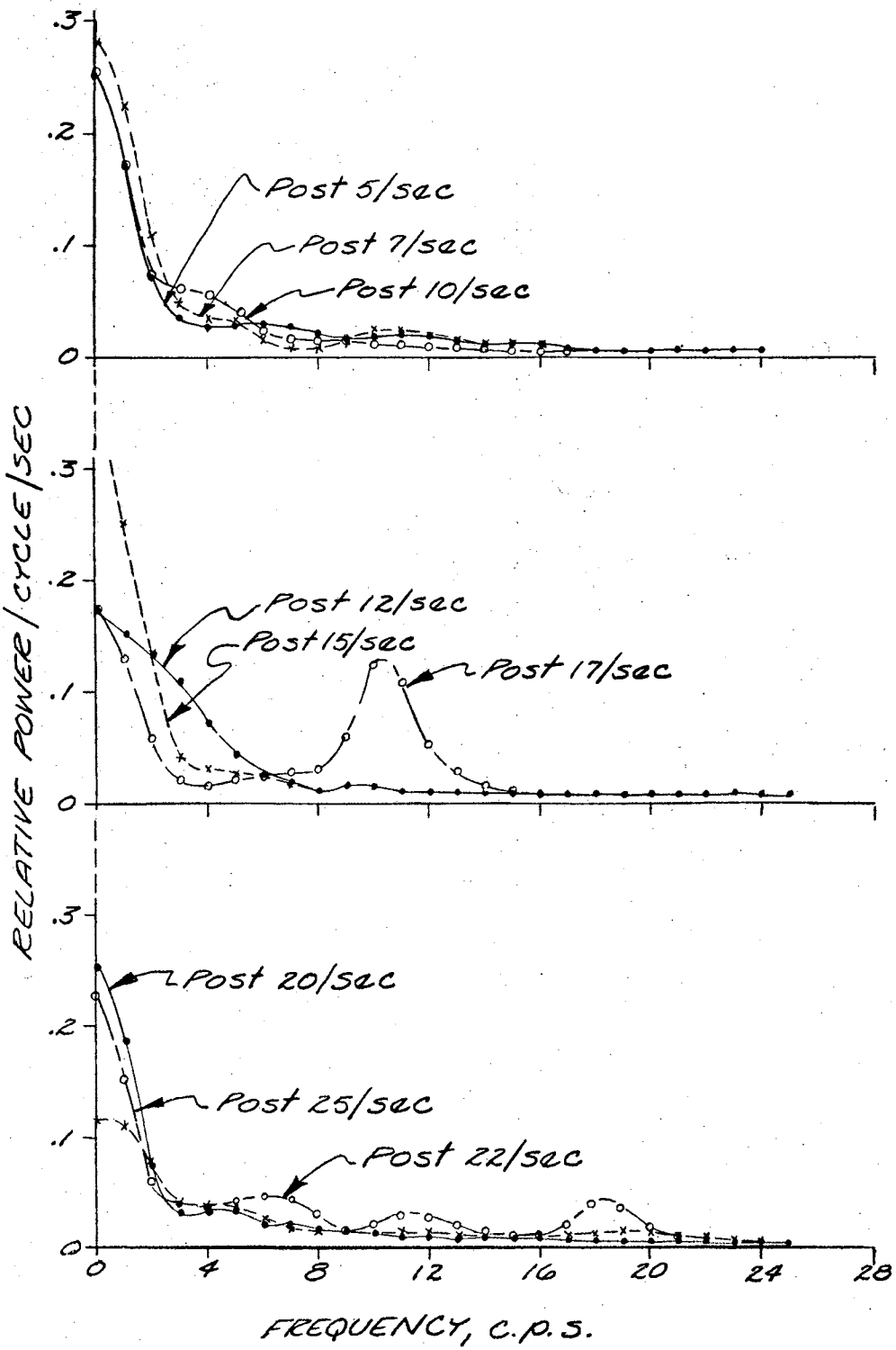

Figure 25. Subject No. 3. Right Occipital Post-Stimulation Auto Spectra

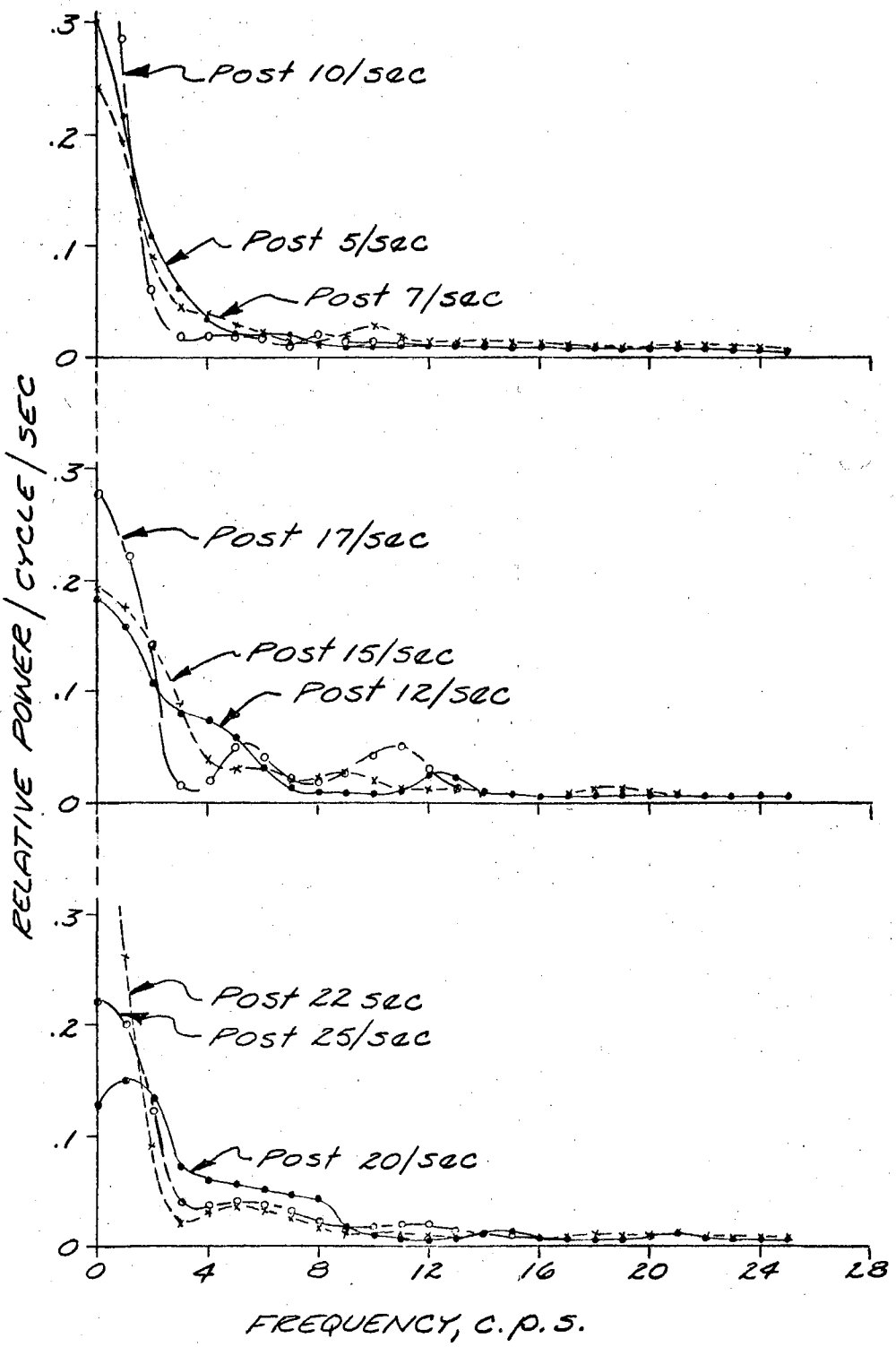

Figure 26. Subject No. 3. Left Temporal Post-Stimulation Auto Spectra

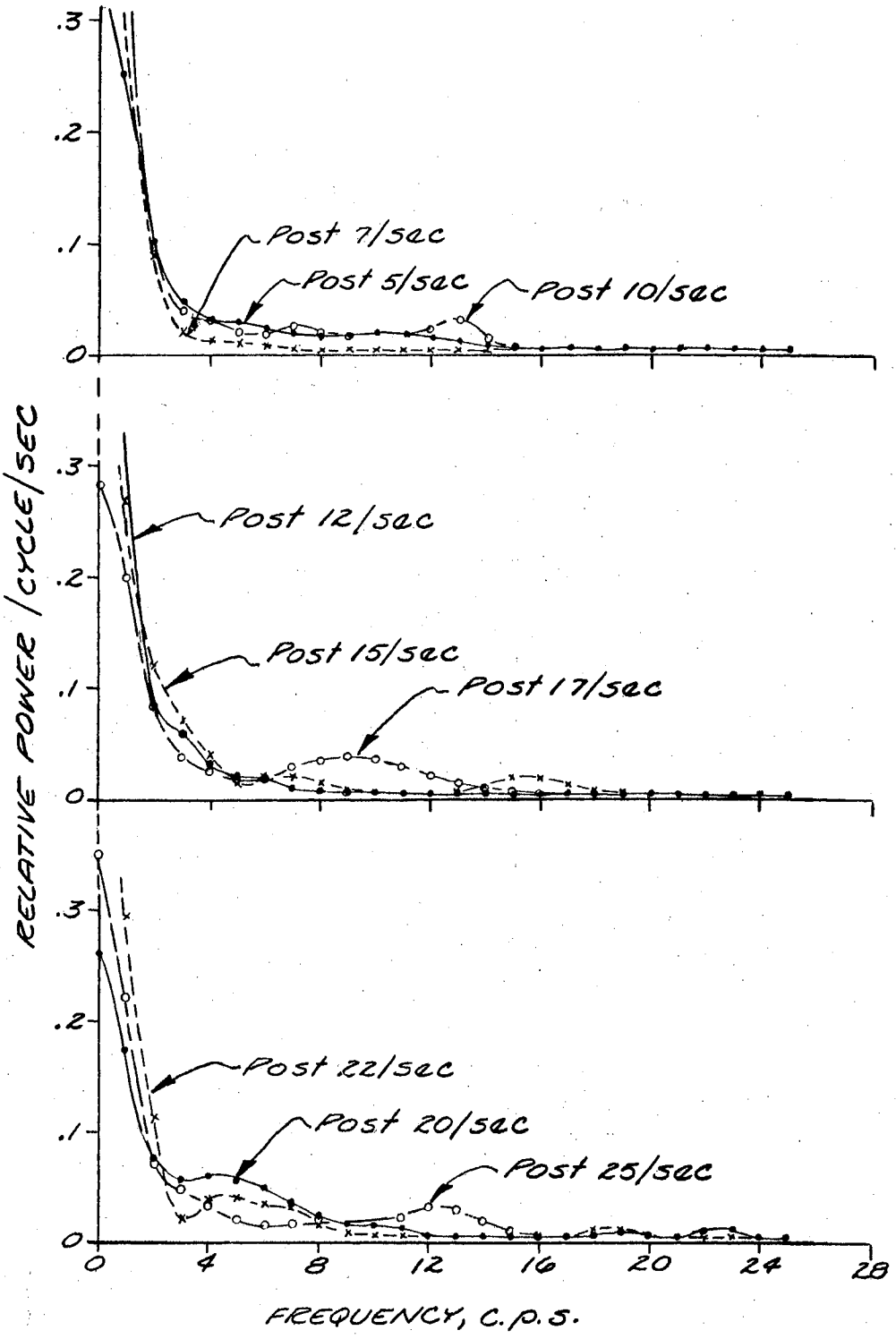

Figure 27. Subject No. 3. Right Temporal Post-Stimulation Auto Spectra

TABLE I

COHERENCE

Stimulus	Subject No. 1				Subject No. 2				Subject No. 3			
	LO	RO	LT	RT	LO	RO	LT	RT	LO	RO	LT	RT
5/Sec. No. 1	0.51	0.79	0.38	0.15	0.31	0.32	0.39	0.35	0.85	0.92	0.40	0.51
No. 2	0.53	0.52	0.33	0.48	0.41	0.43	0.39	0.56	0.69	0.84	0.35	0.12
No. 3	0.67	0.73	0.09	0.40	0.54	0.59	0.52	0.59	0.81	0.88	0.25	0.30
7/Sec. No. 1	0.93	0.94	0.48	0.55	0.30	0.31	0.23	0.35	0.82	0.80	0.42	0.49
No. 2	0.92	0.95	0.70	0.52	0.11	0.09	0.33	0.14	0.87	0.87	0.66	0.45
No. 3	0.95	0.94	0.57	0.63	0.20	0.12	0.21	0.11	0.95	0.97	0.64	0.76
10/Sec. No. 1	0.88	0.88	0.63	0.90	0.56	0.62	0.67	0.69	0.86	0.87	0.72	0.59
No. 2	0.72	0.69	0.25	0.59	0.50	0.56	0.61	0.53	0.81	0.88	0.52	0.31
No. 3	0.33	0.51	0.11	0.47	0.34	0.42	0.48	0.45	0.92	0.95	0.88	0.67
12/Sec. No. 1	0.60	0.69	0.22	0.68	0.72	0.81	0.60	0.82	0.77	0.81	0.79	0.58
No. 2	0.74	0.75	0.57	0.43	0.79	0.86	0.68	0.78	0.90	0.92	0.65	0.73
No. 3	0.38	0.47	0.39	0.70	0.51	0.63	0.10	0.44	0.75	0.81	0.39	0.42
15/Sec. No. 1	0.33	0.29	0.40	0.83	0.84	0.87	0.40	0.79	0.54	0.88	0.92	0.88
No. 2	0.77	0.76	0.25	0.67	0.54	0.68	0.28	0.68	0.84	0.89	0.82	0.79
No. 3	0.28	0.42	0.03	0.45	0.76	0.76	0.70	0.74	0.79	0.89	0.89	0.89
17/Sec. No. 1	0.76	0.75	0.66	0.81	0.01	0.17	0.70	0.23	0.48	0.67	0.80	0.88
No. 2	0.68	0.73	0.30	0.69	0.51	0.56	0.12	0.48	0.47	0.59	0.66	0.76
No. 3	0.80	0.77	0.65	0.11	0.55	0.56	0.51	0.26	0.58	0.65	0.86	0.57
20/Sec. No. 1	0.86	0.87	0.57	0.63	0.81	0.81	0.66	0.78	0.92	0.91	0.85	0.92
No. 2	0.82	0.82	0.52	0.65	0.69	0.71	0.70	0.74	0.72	0.60	0.29	0.63
No. 3	0.54	0.58	0.19	0.10	0.63	0.58	0.28	0.58	0.73	0.58	0.69	0.21
22/Sec. No. 1	0.96	0.94	0.59	0.57	0.75	0.70	0.23	0.49	0.95	0.94	0.81	0.68
No. 2	0.65	0.71	0.37	0.24	0.71	0.79	0.39	0.68	0.86	0.86	0.71	0.65
No. 3	0.89	0.89	0.50	0.56	0.72	0.74	0.56	0.59	0.49	0.40	0.30	0.44
25/Sec. No. 1	0.73	0.70	0.51	0.60	0.20	0.35	0.40	0.19	0.97	0.96	0.76	0.62
No. 2	0.03	0.16	0.29	0.65	0.68	0.73	0.24	0.63	0.92	0.90	0.63	0.76
No. 3	0.82	0.80	0.41	0.69	0.80	0.82	0.50	0.76	0.97	0.96	0.59	0.27

TABLE II

TRANSFER RATIO

Stimulus	Subject No. 1				Subject No. 2				Subject No. 3			
	LO	RO	LT	RT	LO	RO	LT	RT	LO	RO	LT	RT
5/Sec. No. 1	0.73	0.80	0.22	0.10	0.19	0.25	0.24	0.28	1.05	1.25	0.37	0.51
No. 2	0.48	0.56	0.22	0.44	0.25	0.20	0.33	0.27	0.96	1.44	0.32	0.10
No. 3	0.69	0.82	0.05	0.31	0.48	0.66	0.43	0.64	1.11	1.46	0.24	0.26
7/Sec. No. 1	1.24	1.33	0.36	0.41	0.34	0.42	0.19	0.49	0.87	0.89	0.27	0.43
No. 2	0.80	0.89	0.51	0.30	0.12	0.13	0.34	0.21	1.63	1.63	0.55	0.39
No. 3	1.33	1.34	0.33	0.51	0.26	0.17	0.18	0.13	1.26	1.33	0.37	0.44
10/Sec. No. 1	1.50	0.44	0.55	0.53	0.61	0.68	0.50	0.75	0.77	0.95	0.61	0.42
No. 2	0.92	0.82	0.13	0.38	0.48	0.55	0.46	0.47	0.63	0.85	0.30	0.12
No. 3	0.34	0.51	0.07	0.26	0.28	0.39	0.29	0.38	0.72	0.88	0.51	0.31
12/Sec. No. 1	0.69	0.81	0.10	0.46	0.32	0.38	0.27	0.43	0.71	0.87	0.48	0.29
No. 2	0.85	0.90	0.43	0.22	0.13	0.32	0.30	0.32	0.77	0.70	0.33	0.20
No. 3	0.47	0.54	0.28	0.35	0.11	0.24	0.04	0.16	0.37	0.45	0.12	0.13
15/Sec. No. 1	0.08	0.07	0.07	0.37	0.50	0.65	0.37	0.61	0.47	0.46	0.45	0.57
No. 2	0.24	0.27	0.08	0.16	0.23	0.25	0.18	0.22	0.57	0.57	0.27	0.23
No. 3	0.07	0.12	0.00	0.11	0.38	0.36	0.66	0.38	0.36	0.36	0.32	0.21
17/Sec. No. 1	0.32	0.29	0.24	0.21	0.02	0.04	0.26	0.05	0.24	0.24	0.25	0.20
No. 2	0.21	0.26	0.06	0.19	0.17	0.20	0.02	0.17	0.19	0.19	0.23	0.24
No. 3	0.34	0.34	0.11	0.03	0.23	0.24	0.10	0.08	0.14	0.14	0.27	0.12
20/Sec. No. 1	0.28	0.32	0.09	0.13	0.20	0.21	0.11	0.17	0.54	0.53	0.34	0.53
No. 2	0.21	0.21	0.10	0.09	0.31	0.34	0.25	0.30	0.27	0.27	0.06	0.22
No. 3	0.14	0.14	0.04	0.01	0.10	0.10	0.10	0.11	0.22	0.22	0.17	0.04
22/Sec. No. 1	0.26	0.26	0.11	0.11	0.14	0.14	0.04	0.08	0.60	0.60	0.21	0.19
No. 2	0.09	0.14	0.04	0.02	0.21	0.28	0.09	0.20	0.57	0.57	0.17	0.12
No. 3	0.15	0.18	0.08	0.10	0.33	0.40	0.15	0.25	0.15	0.15	0.06	0.10
25/Sec. No. 1	0.07	0.07	0.04	0.05	0.02	0.02	0.05	0.02	0.51	0.51	0.13	0.11
No. 2	0.00	0.01	0.03	0.09	0.18	0.22	0.03	0.15	0.39	0.39	0.12	0.13
No. 3	0.20	0.23	0.07	0.12	0.34	0.36	0.09	0.25	0.48	0.48	0.12	0.05

TABLE III

LEFT OCCIPITAL CROSS-POWER SPECTRA

Stimulus	Subject No. 1			Subject No. 2			Subject No. 3		
	COSP	QUAS	MAGS	COSP	QUAS	MAGS	COSP	QUAS	MAGS
5/Sec. No. 1	-0.031	+0.005	0.031	-0.007	+0.005	0.009	-0.053	-0.008	0.053
No. 2	-0.020	+0.004	0.020	+0.002	+0.012	0.012	-0.042	-0.005	0.043
No. 3	-0.018	+0.025	0.031	-0.014	+0.017	0.022	-0.041	+0.011	0.043
7/Sec. No. 1	+0.046	+0.062	0.078	+0.016	-0.017	0.023	+0.025	+0.049	0.055
No. 2	+0.029	+0.041	0.050	-0.007	-0.004	0.008	+0.088	+0.033	0.094
No. 3	+0.033	+0.055	0.064	+0.016	-0.009	0.019	+0.080	+0.050	0.094
10/Sec. No. 1	+0.106	+0.067	0.125	+0.015	-0.060	0.062	+0.031	-0.061	0.068
No. 2	+0.049	+0.069	0.085	-0.013	-0.046	0.048	-0.004	-0.057	0.058
No. 3	+0.019	-0.025	0.032	-0.012	-0.026	0.029	-0.014	-0.062	0.063
12/Sec. No. 1	-0.046	-0.059	0.075	-0.039	-0.009	0.040	-0.032	-0.077	0.083
No. 2	-0.074	-0.061	0.096	-0.016	-0.006	0.017	-0.052	-0.080	0.095
No. 3	-0.007	-0.056	0.057	-0.014	-0.005	0.015	-0.030	-0.030	0.043
15/Sec. No. 1	+0.001	-0.012	0.012	-0.012	+0.080	0.080	0.000	+0.010	0.010
No. 2	+0.029	+0.022	0.037	+0.007	+0.038	0.039	-0.028	+0.027	0.039
No. 3	-0.003	+0.011	0.011	-0.032	+0.053	0.061	-0.028	+0.027	0.040
17/Sec. No. 1	-0.045	+0.032	0.055	0.000	+0.004	0.004	+0.027	+0.004	0.027
No. 2	-0.008	+0.036	0.037	+0.030	-0.009	0.031	+0.022	-0.017	0.027
No. 3	-0.057	+0.016	0.059	+0.035	-0.025	0.044	+0.015	-0.019	0.025
20/Sec. No. 1	-0.020	-0.054	0.058	+0.001	-0.043	0.043	+0.088	-0.070	0.112
No. 2	-0.021	-0.038	0.044	+0.042	-0.052	0.066	+0.060	+0.010	0.061
No. 3	-0.012	-0.026	0.029	-0.005	-0.022	0.023	-0.002	-0.059	0.059
22/Sec. No. 1	-0.059	+0.019	0.063	-0.034	-0.005	0.034	+0.113	-0.086	0.142
No. 2	-0.014	+0.017	0.022	-0.039	+0.031	0.050	+0.021	-0.133	0.134
No. 3	-0.032	+0.016	0.036	-0.071	+0.030	0.077	+0.035	-0.030	0.045
25/Sec. No. 1	+0.015	+0.010	0.018	+0.001	+0.005	0.005	-0.165	-0.024	0.167
No. 2	+0.001	0.000	0.001	-0.009	+0.059	0.059	-0.126	+0.046	0.134
No. 3	-0.023	+0.048	0.053	-0.076	+0.087	0.115	-0.160	+0.056	0.170

TABLE IV

RIGHT OCCIPITAL CROSS-POWER SPECTRA

Stimulus	Subject No. 1			Subject No. 2			Subject No. 3		
	COSP	QUAS	MAGS	COSP	QUAS	MAGS	COSP	QUAS	MAGS
5/Sec. No. 1	-0.034	-0.006	0.034	-0.007	+0.010	0.012	-0.063	+0.006	0.064
No. 2	-0.023	+0.003	0.024	-0.001	+0.010	0.010	-0.063	+0.011	0.064
No. 3	-0.030	+0.022	0.037	-0.019	+0.024	0.031	-0.052	+0.023	0.056
7/Sec. No. 1	+0.045	+0.079	0.090	+0.017	-0.024	0.029	+0.028	+0.049	0.056
No. 2	+0.029	+0.049	0.056	-0.007	-0.006	0.009	+0.087	+0.036	0.094
No. 3	+0.038	+0.075	0.084	+0.012	-0.003	0.012	+0.085	+0.051	0.099
10/Sec. No. 1	+0.113	+0.043	0.121	+0.020	-0.066	0.069	+0.041	-0.073	0.084
No. 2	+0.066	+0.038	0.076	-0.011	-0.054	0.055	+0.012	-0.077	0.078
No. 3	+0.025	-0.042	0.049	-0.011	-0.037	0.039	-0.009	-0.077	0.078
12/Sec. No. 1	-0.068	-0.057	0.089	-0.045	-0.017	0.048	-0.042	-0.094	0.103
No. 2	-0.085	-0.054	0.101	-0.039	-0.014	0.041	-0.050	-0.071	0.087
No. 3	-0.036	-0.054	0.065	-0.027	-0.014	0.031	-0.030	-0.043	0.053
15/Sec. No. 1	-0.010	-0.005	0.011	-0.028	+0.100	0.104	-0.006	+0.074	0.074
No. 2	+0.024	+0.035	0.042	-0.006	+0.042	0.043	-0.061	+0.066	0.090
No. 3	-0.006	+0.017	0.018	-0.034	+0.047	0.058	-0.041	+0.039	0.057
17/Sec. No. 1	-0.024	+0.043	0.049	+0.007	+0.002	0.007	+0.030	+0.030	0.043
No. 2	0.000	+0.046	0.046	+0.036	-0.013	0.038	+0.034	-0.003	0.034
No. 3	-0.056	+0.017	0.059	+0.039	-0.024	0.046	+0.024	-0.009	0.025
20/Sec. No. 1	-0.032	-0.058	0.066	0.000	-0.046	0.046	+0.064	-0.083	0.110
No. 2	-0.023	-0.036	0.043	+0.039	-0.062	0.073	+0.052	-0.010	0.053
No. 3	-0.018	-0.024	0.030	-0.010	-0.018	0.021	+0.004	+0.043	0.044
22/Sec. No. 1	-0.059	+0.018	0.062	-0.033	-0.003	0.033	+0.112	-0.077	0.136
No. 2	-0.020	+0.027	0.033	-0.051	+0.042	0.066	+0.013	-0.129	0.129
No. 3	-0.037	+0.022	0.043	-0.084	+0.039	0.093	+0.014	-0.032	0.035
25/Sec. No. 1	+0.007	+0.016	0.018	+0.005	+0.010	0.011	-0.166	-0.037	0.171
No. 2	-0.002	+0.003	0.003	+0.001	+0.075	0.075	-0.121	+0.055	0.133
No. 3	-0.031	+0.051	0.059	-0.071	+0.099	0.121	-0.148	+0.053	0.157

TABLE V

LEFT TEMPORAL CROSS-POWER SPECTRA

Stimulus	Subject No. 1			Subject No. 2			Subject No. 3		
	COSP	QUAS	MAGS	COSP	QUAS	MAGS	COSP	QUAS	MAGS
5/Sec. No. 1	+0.002	-0.009	0.009	-0.011	-0.005	0.012	+0.018	-0.006	0.019
No. 2	+0.004	-0.008	0.009	-0.016	-0.002	0.016	+0.014	+0.004	0.014
No. 3	+0.002	-0.001	0.002	-0.019	-0.007	0.020	-0.007	-0.006	0.009
7/Sec. No. 1	+0.014	+0.018	0.023	+0.002	-0.013	0.014	+0.003	-0.003	0.004
No. 2	+0.028	+0.016	0.032	-0.021	+0.012	0.024	+0.031	-0.006	0.032
No. 3	+0.002	+0.021	0.021	+0.012	-0.003	0.013	+0.026	+0.008	0.028
10/Sec. No. 1	+0.042	+0.019	0.046	+0.035	-0.037	0.051	-0.035	+0.041	0.054
No. 2	+0.007	+0.010	0.012	+0.042	-0.018	0.046	-0.027	-0.007	0.027
No. 3	+0.007	+0.001	0.007	-0.005	+0.029	0.030	-0.039	-0.009	0.038
12/Sec. No. 1	-0.008	-0.008	0.011	-0.033	-0.007	0.034	+0.020	+0.052	0.056
No. 2	-0.024	-0.041	0.048	-0.036	-0.014	0.038	+0.028	+0.031	0.041
No. 3	+0.029	-0.018	0.034	+0.004	-0.002	0.005	-0.005	+0.013	0.013
15/Sec. No. 1	+0.010	+0.002	0.010	+0.017	+0.056	0.059	+0.060	-0.039	0.071
No. 2	+0.004	+0.012	0.013	-0.024	+0.018	0.030	+0.035	-0.022	0.042
No. 3	0.000	+0.001	0.001	-0.035	+0.101	0.107	+0.038	-0.032	0.050
17/Sec. No. 1	-0.025	-0.034	0.042	+0.041	-0.024	0.047	-0.030	-0.031	0.043
No. 2	-0.008	+0.006	0.010	-0.001	+0.006	0.007	-0.015	-0.037	0.041
No. 3	-0.010	-0.016	0.019	+0.001	-0.019	0.019	+0.002	-0.047	0.047
20/Sec. No. 1	-0.005	+0.019	0.019	-0.004	-0.024	0.025	-0.030	-0.059	0.066
No. 2	+0.011	-0.018	0.021	+0.005	-0.053	0.054	+0.002	-0.011	0.011
No. 3	+0.009	+0.003	0.009	+0.013	-0.017	0.021	-0.015	+0.025	0.029
22/Sec. No. 1	-0.012	-0.025	0.028	+0.009	-0.001	0.009	+0.032	-0.035	0.048
No. 2	+0.009	-0.005	0.010	+0.015	+0.015	0.021	-0.009	-0.036	0.037
No. 3	-0.005	-0.020	0.020	-0.018	+0.031	0.035	+0.005	+0.012	0.013
25/Sec. No. 1	-0.008	+0.009	0.012	-0.002	-0.017	0.017	-0.039	-0.020	0.043
No. 2	-0.001	-0.012	0.012	-0.008	-0.008	0.011	-0.022	+0.034	0.040
No. 3	-0.017	-0.010	0.020	-0.036	+0.007	0.036	-0.017	+0.035	0.039

TABLE VI

RIGHT TEMPORAL CROSS-POWER SPECTRA

Stimulus	Subject No. 1			Subject No. 2			Subject No. 3		
	COSP	QUAS	MAGS	COSP	QUAS	MAGS	COSP	QUAS	MAGS
5/Sec. No. 1	+0.004	+0.002	0.005	-0.009	+0.010	0.013	-0.005	-0.026	0.026
No. 2	-0.010	-0.018	0.021	-0.004	+0.013	0.013	+0.003	-0.003	0.004
No. 3	-0.007	-0.012	0.014	-0.022	+0.019	0.029	-0.009	+0.004	0.010
7/Sec. No. 1	-0.000	+0.026	0.026	+0.015	-0.031	0.035	+0.006	-0.027	0.027
No. 2	+0.011	+0.016	0.019	-0.014	-0.005	0.015	0.000	-0.022	0.022
No. 3	+0.002	+0.033	0.033	+0.007	-0.007	0.010	+0.017	-0.028	0.033
10/Sec. No. 1	+0.048	-0.006	0.049	+0.019	-0.073	0.075	-0.028	+0.024	0.037
No. 2	+0.033	-0.007	0.033	-0.017	-0.044	0.047	-0.006	-0.009	0.011
No. 3	+0.012	-0.022	0.025	-0.009	-0.037	0.038	-0.021	-0.009	0.022
12/Sec. No. 1	-0.041	-0.034	0.054	-0.049	-0.021	0.053	-0.000	+0.034	0.034
No. 2	-0.022	-0.010	0.024	-0.038	-0.016	0.041	+0.018	+0.017	0.025
No. 3	0.000	-0.034	0.034	-0.016	-0.013	0.021	+0.007	+0.013	0.015
15/Sec. No. 1	-0.048	-0.025	0.054	-0.035	+0.090	0.097	+0.073	-0.053	0.091
No. 2	-0.022	+0.009	0.023	-0.008	+0.035	0.036	+0.035	-0.007	0.036
No. 3	-0.015	-0.007	0.016	-0.048	+0.039	0.062	+0.030	-0.013	0.033
17/Sec. No. 1	-0.028	+0.024	0.036	+0.009	-0.001	0.009	-0.033	-0.013	0.035
No. 2	-0.012	+0.030	0.032	+0.026	-0.017	0.032	+0.015	-0.039	0.042
No. 3	+0.002	+0.005	0.005	+0.012	-0.007	0.014	+0.003	-0.020	0.020
20/Sec. No. 1	+0.003	+0.027	0.027	-0.011	-0.034	0.036	-0.076	-0.070	0.103
No. 2	+0.004	-0.018	0.018	+0.035	-0.055	0.065	-0.024	-0.034	0.042
No. 3	-0.001	+0.003	0.003	-0.019	-0.013	0.023	-0.004	-0.006	0.007
22/Sec. No. 1	-0.025	+0.003	0.026	-0.015	-0.012	0.019	-0.010	-0.041	0.042
No. 2	-0.005	-0.003	0.006	-0.024	+0.041	0.048	-0.012	-0.025	0.028
No. 3	-0.021	-0.014	0.025	-0.056	+0.022	0.060	-0.019	+0.011	0.022
25/Sec. No. 1	-0.013	-0.008	0.015	-0.006	+0.003	0.007	+0.010	-0.037	0.038
No. 2	-0.025	-0.007	0.026	+0.011	+0.051	0.052	+0.003	+0.045	0.045
No. 3	-0.013	-0.010	0.033	-0.073	+0.043	0.085	-0.011	-0.010	0.015

comparisons between subjects.

Subject Number 1

In the first subject a high degree of similarity can be noted between the left and right occipital power spectra shown in Figures 4 and 5. The spectrogram reveals a concentration of signal frequencies in a low band of 0-2 c.p.s. and in one other band between 9 and 11 c.p.s. The latter is the easily visualized alpha rhythm, a common background frequency in all species.

The left and right temporal prestimulation spectrograms of Figures 6 and 7 display a striking dissimilarity. The right temporal area exhibits high frequency power in bands centered at 13, 17, and 22 c.p.s., and these frequencies do not appear in the left temporal area. It should be recognized that these became more pronounced as the experiment progressed and are possibly due to previous epochs of stimulation. The low frequency power peaks in the temporal data are larger than the low frequency power spectral peaks in the occipital leads, and the spectral peaks at higher frequencies have lower amplitudes. It can also be observed that during analysis times when the power spectral density is large in, say the left temporal area, it is diminished in the right temporal and vice versa. There appears to be an out-of-phase or interchange in signal frequency and amplitude between the left and right temporal regions.

Power spectral density analysis of the occipital post-stimulation data (Figures 16 and 17) shows a suppression of the magnitude of the spectral peaks and a more strict confinement to the alpha frequency band. The same analysis for the temporal data in Figures 18 and 19

also emphasize the suppression by showing almost complete lack of all activity in the 8-11 c.p.s. region. This has a clinical counterpart called "alpha blocking."

Subject Number 2

Figures 8 and 9 are prestimulation spectrograms of the occipital area, and those for temporal areas are shown in Figures 10 and 11. Examination of these figures show that initially the main signal frequencies are in two bands, 0-2 c.p.s. and 8-10 c.p.s. As the experiment progressed, the magnitude of these spectral peaks diminished, and small spectral peaks appeared at 13, 16, 17, and 22 c.p.s. Again, this is possibly due to previous stimulation periods. Good similarity exists between right and left occipital leads, except the pre five per second shows lack of 8-10 c.p.s. power in the left occiput.

The temporal leads exhibit large magnitude peaks in the power spectra diversely centered at frequencies of 0, 5, 9, 14, 15, 16, 17, and perhaps 22 c.p.s. Some of these are not evident in the pre 5, -7, or -10 per second stimulation periods. The great similarity between right temporal and right occipital area is unusual and worth noting.

Occipital post-stimulation spectrograms for the same subject in Figures 20 and 21 show suppression of the high frequency spectral peaks and a confinement of activity to the 0-2 c.p.s. and the 8-10 c.p.s. bands. Observation of Figures 22 and 23 show signals of comparable amplitude and frequency distribution as those in the prestimulation analysis of the left temporal region. The right temporal data shows lowered high frequency power spectral density peaks; and nearly all activity is in the 8-10 c.p.s. band, which is significantly different

from the left temporal analysis.

Subject Number 3

Spectrograms from this subject, who is clinically normal, convey the impression of a less orderly signal in both the occipital (Figures 12 and 13) and the temporal regions (Figures 14 and 15). The peaks are broad in spectrum and moderate in amplitude. The occipital activity appears primarily in the 0-2 c.p.s. and 8-10 c.p.s. bands. Two exceptions are the peak at 4 c.p.s. in the pre ten per second analysis and some indication of activity at 6 c.p.s. There are even less pronounced power spectral bands in the temporal regions.

Post-stimulation analysis for this subject produces spectrograms which exhibit depressed activity following each stimulation.

Comparison Between Subjects

The prestimulation spectrograms of the occipital signals in subjects one and two are similar and differ from three, while spectrograms of the temporal regions of subjects one and three are similar but differ from two.

Power spectra of the occipital prestimulation data from the first and second subject are quite similar yet differ in two respects. One difference is the second subject (epileptic patient) has occipital spectral peaks appearing at higher frequencies as the experiment progressed through epochs of higher frequency photic stimulation. This was not evidenced in either subjects one or three. Next, the magnitude of the 8-12 c.p.s. (alpha) spectral peaks in the second subject's prestimulation occipital analysis is slightly greater than those of the

first subject and much larger than those of the third subject.

Number three's occipital prestimulation spectrograms differ markedly from the others. They exhibit low magnitude peaks and less indication of a unified frequency even though there is a dominant frequency of about 10 1/2 c.p.s. There are also indications of possible spectral peaks at a frequency of one-half cycle per second which is not present in the other two subjects.

Prestimulation analysis of the temporal data of subjects one and three demonstrate low magnitude peaks at various frequencies and exhibit only low frequency power densities. There is correspondingly less indication of low frequency activity in the second subject.

Magnitudes of the temporal spectral peaks in the second subject are much greater than for the other two. This person has peaks at the higher frequencies which are also evidenced in the right temporal lead of subject number one but are nonexistent in number three.

Power spectral density graphs of the post-stimulation analysis of the occipital data present a picture of slight suppression in the first subject (Figures 16 and 17), greater suppression in the third subject (Figures 24 and 25), and virtually no suppression in the second subject (Figures 20 and 21). However, the high frequency activity essentially disappears in the post-stimulation analysis of number two. In addition to suppression there seems to be a consolidation of activity to the dominant frequency bands in all subjects.

In the temporal area there are few discernible power spectral density peaks except in the 0-2 c.p.s. band in the post-stimulation analyses of the first and third subjects as shown in Figures 18, 19, 26, and 27. The left temporal area of the second subject continues to

exhibit high frequency peaks while the dominant frequency of 8-10 c.p.s. appears decreased in power. Activity continues undiminished in the right temporal area in this subject, but the high frequency signals are absent by comparison with the prestimulation data. There is little evidence of low frequency (0-2 c.p.s.) components in the temporal signals of the second subject, and this is the major band in the other subjects during this epoch.

In the post-stimulation analysis the first and third subjects are comparable, and the second differs radically from both of these.

Coherence

In the preceding sections of this chapter a subjective examination is presented of the prestimulation and post-stimulation power spectral density analysis of the EEG taken from the three experimental subjects. This is important in that it explores the natural on-going EEG and presents a picture which portrays the natural variability encountered between subjects under conditions where pathology is involved and where no pathological conditions are suspected. The difficulties of making subjective interpretations should be evident from the preceding discussion. Inescapably, such phrases as "somewhat larger," "similar but with minor differences," "quite different," and "a little less pronounced" fill such wordy descriptions. Very often the end result is inconclusive, questionable, and rarely produces decisive results.

Attempts are made in the following section to present analyses of EEG during photic stimulation having numerical results in the hope of arriving at something more quantitative.

Coherence is defined by Equation 25 in Chapter II. The computed

coherences for all three subjects are given in Table I. The tabulated numerical results are the items of direct interest and are presented in a form for greatest ease in making comparisons; however, general patterns can also be observed. In fact, there are many ways these data could be analyzed, graphed, or compared; but care should be taken not to detract from the main analysis which is a subject-to-subject comparison of the coherence values in each area at each analysis period.

The results are seen in the tabulated data (Table I). In summary, it can be said subject number three shows consistently higher coherence values, subject number one has slightly less coherence (especially in the temporal leads at the high stimulation rates), and subject number two has markedly lower values of coherence throughout all stimulation periods up to 15 per second. So, while the second subject's pre-stimulation and post-stimulation spectrograms had large peaks indicating considerable power density, his EEG is influenced less by the photic stimulation.

Generally, there is good agreement in coherence values between left and right occipital areas in all three subjects. Each subject has a photic stimulation rate where a minimum of coherence appears. For the first subject this was at 15 per second and at 17 per second for the second and third subjects.

Coherence in the two occipital areas increases and decreases in unison in all subjects as they progress through higher photic frequencies. In the temporal leads a different picture is presented. In these areas there is usually a high-low combination; i.e., when one temporal area shows a high coherence, the other temporal has a lower value. This is less evident in subject number three.

Transfer Ratios

Computed transfer ratios are tabulated in Table II for all subjects. Again, the result is lower values of transfer ratio for the second subject, especially in the occipital data. The third subject has consistently higher transfer ratios, and those from the first subject are comparable but slightly lower.

Both number one and number two demonstrate less influence at higher stimulation rates than subject number three. An item of great interest is the fact that in the temporal data analysis of the second subject (temporal lobe epileptic patient), there appears transfer ratios equal to or greater than those from the occipitals. In the other two subjects the temporal transfer ratios are less than the corresponding occipital values. One exception is at the 17 per second stimulation rate with the third subject.

The first subject shows a minimal response to the 15 per second flash, and subjects two and three demonstrate a similar minimal response to the 17 per second stimulation.

Upper and lower boundaries of this transfer ratio were computed according to the process explained in Chapter II, Equations 27 and 28. These boundary values varied insignificantly from the transfer ratio proper so they were not included with these data.

Cross-Power Spectral Density

Tables III, IV, V, and VI show the cross-power spectral density for the left occipital, right occipital, left temporal, and right temporal areas, respectively.

These results are interesting for two reasons. One is the resolution of the cross-power spectra into its real and quadrature components reveals the relative phase changes at the various stimulation rates. The other item of value is the magnitude of the cross-power spectral density.

It does not seem to be worthwhile to examine phase relations in detail, but a gross study as to which quadrants the phase angle falls is interesting. Unresolvable anomalies arise due to inability to either ascertain multiple values of 360° or in which direction the phase angle changes (increasing or decreasing phase) as the stimulation rate is varied. A summary of phase angle quadrants is given in Table VII. The most logical conclusion is that the initial phase angle at a five per second stimulation is large and lagging, and the lag angle increases with stimulation rate to a maximum of around 1,000 degrees. This portrays a non-minumum phase system. The variation of phase angles at a given stimulation rate may be a characteristic induced by the adaptive mechanisms of the visual system. No apparent significance can be attached to differences between subjects in this respect.

The magnitudes of the cross-power spectra show again that of the three subjects the third consistently exhibits the largest values in the occipital regions at nearly every stimulation rate. The exceptions are at stimulation rates around 15-17 per second. At stimulation rates beyond these values, the differences between subjects one and three are less evident; and differences between the third and second subject remain but are less pronounced.

In the temporal areas the cross-power spectral density magnitudes

TABLE VII

PHASE ANGLE QUADRANTS

Stimulation Rate	Subject No. 1				Subject No. 2				Subject No. 3			
	LO	RO	LT	RT	LO	RO	LT	RT	LO	RO	LT	RT
5/Sec.	2	2	4	3	2	2	3	2	2	2	3, 4	3, 4
7/Sec.	1	1	1	1	4	4	2, 4	4, 3	1	1	4	4
10/Sec.	1, 4	1, 4	1	4	4, 3	4, 3	4	4, 3	4, 3	4, 3	2, 3	2, 3
12/Sec.	3	3	3, 4	3	3	3	3	3	3	3	1	1
15/Sec.	2, 1	2, 1	1	3	2	2	1, 2	2	2	2	4	4
17/Sec.	2	2	3	2	4	4	4	4	4	1	3	3, 4
20/Sec.	3	3	4, 1	1, 4	4, 3	4, 3	4	3, 4	1, 4	1, 4	3, 2	3
22/Sec.	2	2	3	3	2	2	1, 2	3, 2	4	4	4, 3	3, 2
25/Sec.	1, 3	1, 3	3	3	2	2	3	2, 1	3, 4	3, 4	3, 2	4, 3

of subject number two generally equal or exceed those of the first and third subject.

CHAPTER IV

CONCLUSIONS

From the clinical viewpoint the pictures presented by the pre-stimulation and post-stimulation spectrograms are valuable. They point up, in inescapable fashion, differences in the three experimental subjects. Continued applications of these processes in a study of a larger group of patients and normal subjects will allow these spectrograms to be interpreted in terms of pathological involvement and to be examined for physiological significances.

The relations between coherence and transfer ratios in the pathological EEG have been revealed to be significantly different. Statistical limits of these differences can be established by analyzing a group of normal subjects.

These results relate very well to other recent studies. Cernacek (13) has compared the cortical evoked potentials in a group of petit mal epileptics with those in a group of non-epileptics.

This comparison showed the primary waves of the evoked potentials recorded from the occipital area of epileptics presented with a photic stimulus were much reduced in amplitude from the evoked response seen in normal subjects. Since the epileptic group demonstrated a reduced EEG response to a photic stimulus, it is in keeping that the coherence and transfer ratios should also be low, as they were found to be.

Creutzfeldt (27) has published results of research aimed at

linking the surface EEG to underlying neuronal functions. In this work a correlation is established between certain portions of the surface EEG recorded from exposed motor cortex in cats and inhibitory post synaptic potentials (IPSP) of a sub cortical neuron. Other segments of the surface EEG correlate with excitatory post synaptic potentials (EPSP) of the same neuron. If it is valid to assume the existence of similar relations between the surface EEG and neuronal activity in the sub cortical layer of the occipital cortex in man, then the following conclusions can be reached.

In epilepsy it is generally believed that a deficiency exists in the inhibitory facilities of the brain. If the surface EEG recorded from the occipital areas is a reflection of both IPSP and EPSP and the inhibitory functions are blunted, then it would be expected that the EEG recorded during photic stimulus would contain components related to IPSP in reduced amounts. The coherence and transfer ratios of subject number two (a patient with temporal lobe epilepsy) show a much reduced influence of the photic stimulus in the EEG. This could be a measure of a failing inhibitory capability. Creutzfeldt's work also shows that the number of second and third EPSP's with corresponding surface EEG components increase while the IPSP and its related surface component dimish at increased stimulation rates. At 15 to 20 per second rates the IPSP component disappears. Data analyses from subject number two show coherence values and transfer ratios comparable to those of the third subject when the stimulation rate reaches 15 to 17 per second, similar to Creutzfeldt's observations on cats. Clinically, some epilepsy patients can be driven into seizures with these faster stimulation rates. One logical deduction is that

if lack of IPSP in subject number two is responsible for reduced coherence and transfer ratios at lower stimulation rates and this element vanishes at higher stimulation rates (around 15-17 per second), then there should be less discrepancy between coherence and transfer ratios of subjects two and three at these higher rates. This was found to be true.

Returning to the post-stimulation spectral analyses, it should be recalled that the second subject exhibits virtually no suppression in the 8 1/2-10 c.p.s. power spectral band; while subjects one and three show a marked suppression in this alpha frequency band. The alpha frequency is normally blocked by arousal or cognitive efforts. It is often associated with an orienting reflex which is an innate effort on behalf of the brain to search for and find those sensory inputs which demand concentrated analyses at the moment. Once a subject has adjusted to a repetitive light flash, cessation of the flash becomes a change in environment, thus an "off" stimulus to the neurophysiological system. The suppression of the alpha frequency in the post-stimulation periods could be an indication of the brain arousal due to the cessation of photic stimulation. If arousal is associated with an inhibitive reflex producing alpha rhythm blocking and the inhibitory mechanisms are weak (as postulated in subject number two), then suppression of this frequency band would not be expected.

Grindel (22), whose work was published after the data analyses in this dissertation were completed, autocorrelated human EEG recorded while presenting the subject with a photic stimulation. The auto-correlograms of the occipital EEG revealed a periodic component related

to the photic stimulation, and in the temporal EEG this component was virtually nonexistent. Use of autocorrelograms as a measure of photic driving was investigated in the initial phases of this dissertation, and the results coincided with the findings of Grindel. Auto-power spectral density analysis, which is derived from the autocorrelogram, also shows the same results.

Summary

This dissertation presents a power spectral density analysis of 18 samples of the unstimulated EEG recorded from the right and left occipital and right and left temporal areas. Nine of these samples were taken five seconds prior to presenting a photic stimulation to each subject. Nine samples were also taken at the cessation of photic stimulation. Each succeeding epoch of stimulation used a higher stimulation rate, and 85 seconds elapsed between stimulation periods.

Each rate of photic stimulation was presented for 35 seconds, and three samples of EEG were taken during this time. The first record analyzed was taken five seconds after the start of the stimulation, the second sample 15 seconds after the start, and the third sample 25 seconds after a period of stimulation was begun.

These data were acquired on three male subjects; two were healthy males (one of whom has a speech stammer) and the third has temporal lobe epilepsy.

Spectral analysis of the data samples just prior to each epoch of stimulation showed similarities and differences between subjects that would not be revealed by analysis of the normal routine EEG.

The post-stimulation power spectra exhibited marked depression of

periodic activity in the two healthy males, but there was virtually no suppression with the epileptic. The greatest difference was exhibited in the temporal area. The suppressed power spectral density shown by the post-stimulation analysis differs markedly from the pre-stimulation analysis made 80 seconds later just prior to presenting a stimulation at a higher rate. This difference was not present in the epileptic subject.

The data taken during stimulation periods were analyzed for coherence and transfer ratio with respect to the stimuli. Cross-power spectra were also computed and presented in absolute magnitude as well as with real and quadrature components. Computed values of these items again present significant differences within each subject. Phase relations between subjects appeared to have little differences, but this is not clear.

The goal of exploring the EEG to produce an analysis system which can make available quantitative numerical results of value to the clinician has, it is felt, been achieved. Likewise, the other goal of having a system of value in neurophysiological research has also been achieved.

Future Work

It is believed that these techniques developed to analyze the human EEG are applicable to basic neurophysiological research and can be of great value in creating electrophysiological models of the central nervous system. Such investigations have already been started.

The data processing described herein is being used to study a group of ten normal, healthy males. From this study, variations will

be analyzed to estimate normal values and standard deviations for coherence, transfer ratio, and cross-power spectral density.

The use of a random stimulation rate is also under study.

A SELECTED BIBLIOGRAPHY

1. Woolridge, P. E. The Machinery of the Brain. McGraw-Hill, New York, New York (1963).

2. Brazier, Mary A. B. A History of the Electrical Activity of the Brain--the First Half-Century, 1st ed. Pitman Medical Publishing Company, Ltd., London, England (1961).

3. McCullough, W. S. and W. Pitts. "A Logical Calculus of the Ideas Imminent in Nervous Activity." Bulletin of Mathematical Biophysics. Volume 5. (1943) 115-137.

4. Eccles, J. C. "Inhibitory Controls on the Flow of Sensory Information in the Nervous System." Proceedings of the International Union of Physiological Sciences, XXII, International Congress, Leiden, 1962 (editors: Gerard, R. W. and Duyff, J. W.) International Congress Series No. 49, Excerpta Medica Foundation, New York City, New York.

5. Magoun, H. W. The Waking Brain, 2nd ed. Charles C. Thomas, Springfield, Illinois (1963).

6. Sem-Jacobsen, E. W. "Electroencephalography Studies of Pilot Stresses in Flight." Aerospace Medicine. (November, 1959) 797-801.

7. Bremer, F. "Cerebral and Cerebellar Potentials." Physiological Reviews. Volume 38, No. 3. (1953) 357-388.

8. Hill, J. D. N. and G. Parr. Electroencephalography. Macmillan, New York, New York (1950).

9. Penfield, W. and L. Roberts. Speech and Brain Mechanisms. Princeton University Press, Princeton, New Jersey (1959).

10. Davis, J. F. and H. A. Ferris. "A Review of Systems for Recording Average Evoked Response in the Human Electroencephalogram." Proceedings of the Second International Conference on Medical Electronics. Iliffe, London, England (1960) 103.

11. Clark, W. A., Jr. "Average Response Computer (ARC-1)." Research Laboratory for Electronics. M.I.T. Quarterly Progress Report, April 15, 1958.

12. Dawson, G. D. "The Latency and EEG Form in Man of the Occipital Potentials Evoked by Bright Flashes." Journal of Physiology. Volume 152. (1960) 108-121.

13. Cernacek, F. M. and L. Ciganek. "The Cortical Electroencephalographic Response to Light Stimulation in Epilepsy." Epilepsia. Volume 3. (1962) 303-314.

14. Foster, F. M., et al. "Stroboscopic Induced Seizure Discharges." Archives of Neurology. Volume 11. (1964) 603-608.

15. Brazier, M. A. B. and J. S. Barlow. "Some Applications of Correlation Analysis to Electroencephalography." Electroencephalography and Clinical Neurophysiology. Volume 8. (1956) 325-333.

16. Barlow, J. S. and R. M. Brown. "An Analog Correlator System for Brain Potentials." Research Laboratory for Electronics. M.I.T. Technical Report 300, July 14, 1955.

17. Barlow, J. S., M. A. B. Brazier, and W. A. Rosenblith. "The Application of Autocorrelation Analysis to Electroencephalography." Proceedings of the First National Biophysics Conference. Columbus, Ohio, March, 1957.

18. Brazier, M. A. B. "Paired Sensory Modality Stimulation Studied by Computer Analysis." Annals of the New York Academy of Sciences. Volume 92. (1961) 1054-1063.

19. Walter, D. O. and W. R. Adey. "Spectral Analysis of Electroencephalograms Recorded During Learning in the Cat." Experimental Neurology. Volume 7. (1963) 481-501.

20. Lee, Y. W. Statistical Theory of Communication. John Wiley and Sons, Inc., New York, New York (1963).

21. Sato, K. N., et al. "A Simplified Method for Cross-Correlation Analysis." Electroencephalography and Clinical Neurophysiology. Volume 14. (1962) 767-768.

22. Grindel, O. M., et al. "Possible Application of Correlation Analysis to Human Electroencephalogram." Federation Proceedings Translation Supplement. Volume 24, No. 5, Part II. (1964) T753-T757.

23. Jenkins, G. M. "Some Examples of and Comments on Spectral Analysis." Proceedings of the IBM Scientific Computing Symposium Statistics. IBM Data Processing Division, White Plains, New York (1963) 205-246.

24. Blackman, R. B. and J. W. Tukey. The Measurement of Power Spectra. Dover, New York, New York (1959).

25. Walters, D. O. "Spectral Analysis for Electroencephalograms: Mathematical Determinations of Neurophysiological Relationships from Records of Limited Duration." Experimental Neurology. Volume 8. (1963) 155-181.

26. Goodman, N. R. "Measuring Amplitude and Phase." Journal of Franklin Institute. Volume 270. (1960) 437-450.

27. Creutzfeldt, O. D., S. Watanbe, and H. D. Lux. "Relations Between EEG Phenomena and Potentials of Single Cortical Cells: I." Electroencephalography and Clinical Neurophysiology. Volume 20. (1966) 1-18.

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