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ELECTRODERMAL INDICANTS OF AROUSAL IN THE BRAIN-DAMAGED

CHAPTER I

INTRODUCTION

Recent technical and theoretical advances in neurophysiology have provided new avenues of approach for investigations into the relationships between brain mechanisms and behavior. Particularly promising for psychology are findings concerning neural systems pertaining to arousal and activation of the organism. As a consequence of these data, the attention of psychologists has been focused on arousal mechanisms and their functions in regard to behavior. In instances where there are known deficits in behavior, as in cases of brain injury, disturbances of arousal may account for some of the observed deficits. The present study proposed to investigate arousal phenomena as a function of increasing situational demands placed on the adaptive mechanisms of the subject.

The neuropsychological conception, exemplified by Hebb (1949; 1955; 1958), Lindsley (1951), and Malmo (1957; 1959), provides a possibility of dealing with some of the problems that have perplexed psychologists. Through an understanding of neural action, the events that occur between stimulus and response may become more adequately conceptualized. For example, Hebb states, the "psychologist and neurophysiologist [use] some of the same fixed points and [are provided] with the opportunity of contributing to each other's results. The problem of understanding behavior is the problem of understanding the total action of the nervous system, and vice versa" (1949, p. xiv). According to Lindsley (1956), an understanding of the role of the central nervous system in behavior will provide anchoring points from which behavioral studies may proceed with benefit to both psychology and neurophysiology. The necessity to develop comprehensive theories of behavior also demonstrates the value of a neuropsychological approach. It is the position of Meyer (1957) that the evolution of such theories is dependent on careful study of the correlations between psychological and neurological facts.

The attempt to relate behavior and neurophysiology is difficult because of the complexity of the central nervous

system. In the study of brain functions, ideally, one would create specific disturbances in selected locations and record the behavioral results. The experimental neurophysiologist can employ such a technique, but the psychologist interested in human functions must depend upon naturally occurring disturbances such as those found in the brain-damaged patient.

A subject with brain injury or disease examined under standard conditions shows obvious differences in his behavior from a non-brain-damaged subject. But to which of the many variables is the difference to be attributed? A potentially satisfactory approach would identify delimited cerebral regions as being responsible for certain behaviors and functions. Then if a region is destroyed or damaged, predictable consequences would result. The modern representation of this classic view has been called by Magoun (1958) the Edwardian doctrine of specific localization. Evidence that this viewpoint cannot be completely disregarded comes from the findings of Jasper and Rasmussen (1958), Nielsen (1951; 1958), Olds (1958), Penfield (1958), Penfield and Milner (1957), and Reitan (1955). All have shown that specific portions of the brain are implicated in psychologically important functions.

Other data, however, are difficult to incorporate into a point of view that calls for specific localization of

function. For example, Lilly (1958) presents evidence that the classic motor and sensory areas are not as clearly differentiated as previously thought, in that motor fibers emanate from "sensory" areas and sensory fibers terminate in the "motor" area. Semmes, Weinstein, Ghent, and Teuber (1960) have demonstrated that strict localization is not adhered to even at a simple sensory level. Their subjects with damage to the left hemisphere showed impairment of sensory function in both hands. Roberts (1958) has shown that there is "functional plasticity" in the mechanisms of speech, an ability usually considered to be anatomically circumscribed.

In other functions, the results of Parsons and Huse (1958) and of Ax and Colley (1955) demonstrate that there may be impairment of temporal discrimination even where there is no direct evidence of involvement of primary areas or pathways of the sensory systems used in the discrimination process. Further, Wolff, Chapman, Thetford, Berlin, and Guthrie (1958) show that the disturbance of higher order functions is more related to the amount of tissue destroyed than to its specific location. The general conclusion drawn by these authors is that damage to the brain constitutes a stressful situation, and the effect will be shown in many behavior patterns. That is, the destruction of brain tissue has the

effect of interfering with "the proper interaction between organism and environment" (1958, p. 528).

The holistic or organismic conception of brain-behavior relationships is represented by Goldstein (1948), Lashley (1958), and Magoun (1958). These writers consider the brain to operate as a totality in terms of effecting behavior. The behavior observed in cases of brain damage is not due to specific loci being destroyed but rather to the total effect upon the organism. Impairment of psychological capacities is due to a breakdown of, or disturbance in, intracerebral organization and patterning and of coping mechanisms and personality. The emphasis in brain-behavior studies thus turns from consideration of specific connections within the nervous system to a recognition of other factors.

Behavior theories, in general, consider motivation as a primary factor in behavior. As used in these theories, the concept implies that there is a complete, organismic involvement. Conclusions from previously cited studies (Goldstein, 1948; Wolff et al., 1958) and the holistic conception of brain-behavior relationships would suggest that impairment of behavior in brain-damaged subjects may partially be due to motivational variables.

Behavior defined as motivated is frequently described

as varying in intensity (Bindra, 1959; Duffy, 1957; 1963; Hebb, 1955; Morgan, 1957; Woodworth, 1958). In the systems of Bindra (1959), Duffy (1957; 1963), Hebb (1955), Lindsley (1951), and Malmo (1959), the terms arousal and activation have been used to describe an intensity dimension of behavior. Arousal, then, specified as the intensity variable, becomes a significant aspect of behavior.

The Concept of Arousal

The significance for psychology of a concept of arousal is indicated in Duffy's statement that "variations in the degree of activation are, on the average, accompanied by certain variations in overt response" (1957, p. 268). A general concept of energy mobilization developed historically in psychology from observed correspondences between various physiological and behavioral measures. Energy mobilization has been largely replaced in recent literature by the terms activation and arousal, perhaps because of the physical and physiological connotations that the word energy has. However, mobilization, activation and arousal all are used to refer to the ways in which the resources of the organism are brought to bear on situational demands.

In the physiological literature arousal and activation

have been used to refer to the desynchronization of the resting electroencephalographic recording when the subject is stimulated. That is, the predominant alpha rhythm of the normal resting record is replaced by faster, less synchronous patterns (Moruzzi & Magoun, 1949). In psychological studies, precedent has been set for the use of arousal by Bindra (1959), Duffy (1957), Hebb (1958), Malmo (1959), and Woodworth (1958). Further, the arousal concept as used by these authors shows a close correspondence to activation as defined by Lindsley (1951) and to the energy mobilization concept of Freeman (1948). The use of arousal and activation in the present study follows that of the above authors.

That persons vary along an arousal dimension is a matter of common observation. Between the extremes of deep sleep and excitement can be seen a large number of response patterns which represent an almost infinite variety of states or levels of arousal. Behavioral observation and introspection provide justification for the postulation of an arousal continuum, and a rather considerable body of experimental evidence, e.g., Duffy (1963), Freeman (1948), Schlosberg (1954), also shows that arousal is a continuum.

The relationship between arousal levels and quality of performance has been experimentally demonstrated by Burch

and Greiner (1960), Duffy (1932), Freeman (1940), Payne, Hauty, and Moore (1957), Schlosberg (1954), Silverman, Cohen, and Shmavonian (1959), and Stennett (1957a). From these studies the conclusion can be drawn that the relationship between the quality of performance and arousal is best described as an inverted-U function. That is, in low or high arousal states, performance is likely to be poor, whereas at intermediate levels of arousal qualitatively better performance is more likely to result.

In the experiments cited, arousal levels have been inferred from various physiological measures, among which are the galvanic skin response (GSR), electromyography (EMG), electroencephalography (EEG), respiration, and heart rate. The use of such measures is justifiable on several grounds. Malmo emphasizes the objectivity of physiological measures and states that this objectivity "frees the investigator from dependence upon merely manipulating situations in the hope that he is producing intended changes in the arousal level" (1957, p. 278). Both Duffy (1957) and Malmo (1957; 1958) conclude that physiological measures provide the most direct measurement of the degree or intensity of activation. "In short, the physiological measures appear to be useful tools in establishing and precisely quantifying a dimension of

behavioral intensity" (Malmo, 1957, p. 279).

Granted that no single index has been found to be the best indicator of arousal, the electrodermal response could claim an eminent position on the basis of frequency of use. Also, its success as an indicator of the presence of emotion would argue for its possibilities as a measure of arousal, since emotion represents a variety of states or conditions that can be located on an arousal continuum (Duffy, 1957; Lindsley, 1951; Schlosberg, 1954). Other evidence of the value of electrodermal response as an indicant of arousal comes from the correspondence between EEG measures of central function and autonomic indicators (Darrow, 1950; Darrow, Pathman, & Kronenberg, 1946; Malmo, 1959; and Stennett, 1957b). Agreement has also been shown between somatic-skeletal indices of arousal (EMG) and electrodermal response by Malmo (1959) and Stennett (1957b).

From a neurophysiological point of view, autonomic indicators are further likely to prove valuable as measures of arousal. Gellhorn (1953) and Murphy and Gellhorn (1945) have provided evidence that there are hypothalamic effects of both a neural and humoral nature that affect cortical arousal and autonomic discharge. Stimulation of hypothalamic areas brought about simultaneous autonomic discharge and cortical

desynchronization (Gellhorn, 1953).

At least two electrodermal measures have utility as indicators of arousal. The first and most widely used is the level of skin resistance, or its reciprocal, conductance. This measure has consistently demonstrated its value as evidenced by the studies of Freeman (1948), Malmo (1957), Schlosberg (1954), and Stennett (1957a).

The second electrodermal measure which seems useful as an indicator of arousal is the momentary change in resistance, or galvanic skin response (GSR). This measure has been shown to be related to performance in a number of studies, for example, Burch and Greiner (1960), Duffy (1957; 1963), Lacey and Lacey (1958), Silverman et al. (1959), and Stern, Stewart, and Winokur (1961). These studies show this measure to have the same general relationship to behavioral measures as obtained with the level of resistance.

The relationship between level of resistance and GSR frequency has not been extensively studied. Correlations reported by Lacey and Lacey (1958), Silverman et al. (1959), and Stern (1962) are consistently low. In the Stern (1962) study, for example, correlations between the two measures over a series of experiments ranged between -.19 and .32. There appear to be differences in the reported correlations

which are a function of experimental conditions and subject variables, e.g., age, range of levels of resistance, that remain to be thoroughly investigated.

Performance on Psychological Tasks in Brain-Damaged Subjects

Reference to the literature (Hunt & Cofer, 1944; Meyer, 1957) leads to the conclusion that the subject with brain damage will perform at a relatively poorer level than the non-brain-damaged subject on a variety of conditions ranging from sensory threshold measurement to conceptual levels of functioning.

In sensory threshold measures, several studies have shown the disturbance of simple perceptual processes in the brain-damaged subject (Goldstein, 1948; Nielsen, 1951; Semmes et al., 1960). In addition to the experimental evidence, the testing of perception of simple stimuli is an integral part of the diagnostic procedure of the clinical neurologist.

Reaction-time experiments have also provided evidence of impaired function in brain-damaged subjects. Benton and Blackburn (1957), Blackburn (1958), and Blackburn and Benton (1955) have shown, over a variety of experimental conditions, that subjects with brain lesions are consistently slower in reaction time than control subjects. Similar evidence is

presented by Goldstein (1948).

In the area of learning, Meyer (1957), Reese, Doss, and Gantt (1953), Stark (1961), and Walter (1960), present data relating to the decreased ability of brain-injured individuals to perform adequately.

Studies involving temporal discrimination have also reported deficits in the behavior of the brain-injured. Ax and Colley (1955) have shown that temporal acuity thresholds in three modalities (vision, audition, and touch) are sufficiently lowered in cases of neuropathology as to provide information of diagnostic value. Parsons and Huse (1958) and Parsons and Gottlieb (1960) showed that the lowered flicker thresholds of their subjects were sufficiently valid and reliable indices for purposes of identification of brain-damaged individuals. Green, Reese, Pegues, and Elliott (1961) demonstrated that patients with brain lesions are markedly inferior in their ability to discriminate two stimuli separated by brief time intervals.

Halstead (1958) and Reitan (1955), utilizing the Halstead battery, show that brain-damaged subjects perform poorly over a wide range of tasks. This battery encompasses procedures from motor performance (finger-tapping rate) to conceptual level of function (Categories test) and has been

shown to be highly accurate in differentiating brain-damaged from control patients.

A single study which points up the deficits in many areas of behavior in subjects with brain injury is that of Wolff, Chapman, Thetford, Berlin, and Guthrie (1958). Using a large number of varied methods these investigators found that brain-damaged subjects are impaired not only in simple or unitary functions but also in higher integrative performances, e.g., reactions to stress and frustration, expression of needs and drives, mechanisms for goal achievement, and maintenance of defense reactions.

The determinants of impaired performance. With these easily demonstrated, well-documented, and frequently dramatic deficits or variations in behavior confronting the investigator, the question arises as to the possible determinants of the deficit or variation. The majority of the studies mentioned have not been concerned with seeking out possible causes; their nature is, for the most part, descriptive or empirical. If cause is considered, it is usually referred to the brain injury itself and little is said of other possible factors.

Obviously the lesion, its nature, and its location cannot be ignored as important determinants of impaired

functioning. At the same time, many of the studies cited, especially those of Wolff et al., Parsons and Huse, Blackburn, Ax and Colley, and Goldstein imply that variables other than destroyed or damaged tissue in some specific location may also play an important role. These studies suggest that some function is involved which underlies all behavior of the organism in adjusting to environmental demands.

Just as does a normal individual, the brain-injured person must, when confronted with stimuli, become appropriately aroused. He must mobilize his resources, energetic, intellectual, and emotional, to a sufficient level to meet the situation, and he must maintain an appropriate level of arousal in the face of changing conditions. If the environmental demands fluctuate, he must be ready to abandon the previous level and adopt a new one, whether it be higher or lower. Brain-damaged individuals seem to be particularly inadequate to make such adjustments in their arousal levels. The conclusions cited from the Wolff et al. and the Goldstein studies support such an interpretation.

If behavior and performance in various situations have an optimal level of arousal associated with them that is related to the situational demands, it may be that the brain-damaged person is consistently too low or too high on

the arousal continuum and the result is impaired performance.

Evidence relating to this question is inconclusive. Certain data point in the direction of higher than optimal levels of arousal in the brain-injured subject. For example, Goldstein (1948) speaks of the "catastrophic reaction." The patient, when faced with his inability to perform adequately, "changes color, becomes agitated, anxious, starts to fumble, his pulse becomes irregular . . . [and he may] even become aggressive" (1948, p. 71). These symptoms are interpreted by Goldstein as not being a direct result of damage to a part of the brain but rather as an inability on the part of the patient to cope with his defect. Goldstein further lists symptoms of abnormal fatigue, disturbances in maintaining attention, poor memory, and over-reaction to new stimuli, all of which may be indicative of high arousal states.

The lowered ability to tolerate frustration, less resistance to disorganizing influences, and general affective instability, observed by Wolff et al. in their subjects may also be considered suggestive of high levels of arousal.

Other findings, however, could lead to an opposite conclusion. A typical EEG record from a brain-damaged subject often shows a number of slow waves (1-5 per second) which are not found in a normal EEG. If the normal, awake,

relaxed record is one in which alpha frequencies (8-12 per second) predominate, then these slow frequencies present in the record of the brain-injured individual would seem to indicate a lowered level of arousal. This inference can be made from Lindsley's (1951; 1956) description of the relationship between EEG frequency patterns and arousal levels. The normal sleeping record shows slow waves, and sleeping conditions are near the low arousal extreme of the continuum.

Evidence from EEG data, in the study of Kooi, Eckman, and Thomas (1957), indicates that persons with brain lesions are less responsive to photic stimulation. That is, if a rhythmically flashing light is presented, the EEG recording will show a number of waves in phase with the frequency of the light. The group of organic patients studied by Kooi et al. showed a reduced effect of the light on their EEG patterns.

Grossman (1949), using auditory stimuli, and Li, Jasper, and Henderson (1952), using visual and auditory stimuli, demonstrated deficiencies in the arousal response of brain-damaged subjects. In both studies, especially in cases with lesions involving the cortex, arousal stimuli failed to produce EEG desynchronization. The abnormal pattern present in the EEG was usually not displaced upon stimulation, or, when there was an effect, it was most frequently of short

duration.

The inference of lowered arousal levels from these data is made on the basis that the response to stimulation, especially EEG response, can be considered as an index of relative excitability in the central nervous system. This is suggested by Li et al. in their statement that arousal produces an increase in the general excitatory state of the cortex and supported by Dustman, Boswell, and Porter (1962), who have shown that reaction time in normal subjects was slower when alpha frequencies were present than when faster waves were predominant. Thus, the effect of stimulation, which would ordinarily create a specific change in the EEG pattern, or cause a particular response, will depend on the level of excitability, or arousal, at the time of stimulation. This idea corresponds closely to Freeman's (1948) conception that the effect of stimulation, in terms of bringing about energy mobilization, depends on the background level of mobilization at the time of stimulation. Thus, if stimuli impinge on a system that is not at an "optimal" point in terms of energy level, arousal, or excitability, the response will not occur with the rapidity, ease, or efficiency that would otherwise obtain.

Summary. Numerous studies have been cited which

demonstrate that the performance of subjects with brain damage is impaired in comparison with subjects without such damage. The impairment has been shown to be present at all levels of function, from simple sensation to complex adjustments.

Two approaches, related to general theories of brain functioning, are considered as providing possible explanations for these findings. One explanation describes the impairment as due to the destruction of centers which are specific for a given function. The other and more promising approach brings into focus variables of a more general nature, not linked to any specific neural structure, and which seem more adequate to account for the reduced efficiency of performance of persons with brain injury. For example, the studies of Wolff et al. (1958) and Goldstein (1948) suggest a consideration of motivational variables. These studies show that the brain-injured individual's whole pattern of interpretation of stimuli and adjustment to his environment is impaired. It is suggested that the overall disturbance may be due to inability to arrive at and maintain arousal levels which are appropriate to the demands of the environment.

Arousal phenomena in brain-damaged subjects, however, have not been intensively studied nor has the relationship

between performance and arousal been investigated in these subjects. The nature of the available evidence would allow opposite predictions. Some electroencephalographic studies (Grossman; Kooi; Li) indicate that the subject with brain injury is at a low level of arousal. On the other hand, the conclusions from Goldstein and Wolff et al., by inference from the behavior of their subjects, indicate that these subjects are aroused to such a high level that impairment of performance results.

The general question of the degree of arousal in brain-damaged subjects under a variety of conditions has not been investigated. It was the purpose of this study to examine electrodermal indices of arousal in a group of patients with brain lesions during a series of conditions which place progressively greater demands on adjustive capacities.

CHAPTER II

STATEMENT OF THE PROBLEM

Previous studies have suggested that the deficits in performance in subjects with brain lesions may be related to deficiencies in adjustive capacities of these subjects. In the present context, the ability to arrive at and maintain appropriate levels of arousal or activation in relation to situational demands is considered to be a crucial aspect of the adjustive process. This study is concerned with the investigation of arousal phenomena in a group of brain-damaged subjects compared with a non-brain-damaged group under varying conditions.

For the purposes of the experiment, arousal is defined by two electrodermal variables which have been shown to have value in studies concerning activation and arousal: GSR frequency and skin resistance level. The stimuli, tasks, and order of presentation were designed to provide a series of qualitatively different situations which would make

progressively greater demands on the adjustive capacities of the organism. These conditions include rest, passive stimulation, and work.

Levels of Arousal During Rest

Interest in resting levels of arousal in subjects with brain lesions stems from three sources: first, as an empirical question, since no relevant data was found in the literature; second, a rest period preceding other experimental conditions usually provides a base line from which to make interpretative statements; and, third, data from experiments using other physiologically based measures and behavioral observation could lead to opposite expectations concerning resting levels of arousal in the brain-damaged (either higher or lower than non-brain-damaged). Thus, the following two questions are proposed:

1. What is the resting level of arousal in brain-damaged subjects compared with control subjects?

2. How does the level of arousal change during rest?

Change in Arousal During Passive Stimulation

Startle. Grossman (1949) and Li et al. (1952) showed that subjects with brain lesions did not have the

characteristic EEG activation pattern to simple stimuli, suggesting generally lower levels of cortical neural responsivity in such subjects. Electrodermal measures provide data concerning the effect of arousing stimuli at an autonomic level, rather than cortical, thus:

3. How does the level of arousal change in response to a startle stimulus?

Adaptation. A preliminary study (see Appendix A) indicated differences between brain-damaged and control groups in GSR adaptation to a visual stimulus. Studies of adaptation may indicate the degree to which the level of arousal in the brain-damaged is a function of stimulation, even when overt responses are not required, as opposed to situational demands. Thus:

4. What are the arousal characteristics of the adaptation process to repeated stimulation?

Levels of Arousal During Work

Deficits in performance on reaction time and learning tasks in subjects with brain lesions have been well established. The findings of Goldstein (1948) and Wolff et al. (1958) suggest that performance deficits may be related to inability to establish appropriate arousal levels. The

following questions are proposed to provide information regarding arousal in brain-damaged subjects during reaction time and learning situations.

5. How does the level of arousal change during a choice reaction time task?

6. How does the level of arousal change during a paired associates learning task?

The Effects of Failure on Arousal

Goldstein (1948) and Wolff et al. (1958) refer to the inability of subjects with brain lesions to maintain performance in stressful situations. However, the degree to which indices of activation change during failure stress has not been ascertained in brain-damaged subjects. Further, failure during a learning task represents a final step in a series of conditions designed to present the subject with situations which became progressively more demanding. Thus:

7. What is the effect of failure on arousal during a paired associates learning task?

Return to Resting Levels After Stimulation

Adaptation to situational demands necessitates not only increases in arousal as demands increase but also

decreases when requirements cease. The suggestion has been made that deficits in performance in the brain-damaged may be related to inabilities in adapting arousal levels to demands. Inquiry into returns to resting levels after stimulation provides information regarding another aspect of adaptation. Thus:

8. What is the pattern of return to resting level after the experimental conditions?

Relationships Between Performance and Arousal

No experimental data are available concerning the relationships between performance on psychological tasks and arousal in brain-damaged subjects. The electrodermal measures used in the present study can be correlated with performance on the experimental tasks to provide a tentative answer to the following question:

9. What is the relationship between performance on the reaction time and paired associates learning tasks and arousal in brain-damaged and control subjects?

CHAPTER III

METHOD

Subjects

The subjects for this study were 40 patients from the Veterans Administration and University Hospitals, Oklahoma City. Patients were rated for brain damage by the Senior Residents of the Neurology or Neurosurgical Services, or their physician, on the following scale:

1. Definitely indicated, no other evidence needed.

2. Strongly suspected, would like at least one more positive sign.

3. Suspected, but much more evidence needed.

4. Not likely, but cannot be ruled out at this point.

5. Definitely not indicated, no further evidence needed.

The brain-damaged group was restricted to patients who were placed in category 1. At the conclusion of the

study, a second rating, using the same scale, was made by the Consulting Physician to the Neurology Service[1] for the purpose of checking the initial rating. Degree of agreement between the two ratings was 100%. This same physician, using the scales presented in Appendix B, made ratings of localization, lateralization, variety, and severity of the brain disorder.

Patients were excluded from either group who:

1. were over 55 years of age;

2. had symptoms of aphasia;

3. had evidence of spinal cord or peripheral nerve injury to sympathetic pathways serving the arms and hands;

4. had uncorrected visual deficits (Snellen acuity below 20/50, or extreme color vision defects);

5. had a diagnosis of psychosis;

6. had skin conditions which might influence skin resistance recordings, e.g., skin disease or infection, hypo- or hyperhydrosis;

7. had less than five years of education;

8. had a diagnosis of narcolepsy;

9. were taking tranquilizing (e.g., Chlorpromazine)

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[1]Gunter R. Haase, M. D.

or excitant drugs;

10. were diagnosed as, or known to be, alcoholic.

Table 1 presents descriptive data for the brain-damaged group. The control subjects were selected from the Neurology and Psychosomatic wards and only patients placed in category 5 on the brain damage rating scale presented above were included. As can be seen from Table 2, which presents descriptive data for the control subjects, no patients were selected who had diseases associated with cerebral dysfunction.

The control and brain-damaged groups were equated for anxiety as measured by the Minnesota Multiphasic Personality Inventory A scale. The mean scaled score for the brain-damaged group was 54, and the mean score for the control subjects was 52. By the U test (Siegel, 1956) this difference was not statistically significant ($U = 185$, $p > .10$).

The mean MMPI T scores of the two groups are given in Appendix D. It can be seen that the amount of elevation present suggests at least mild states of psychological disturbance.

The two groups were also equated for mean age. The mean age of the control subjects was 41.7 years, and the mean age of the brain-damaged was 41.4 years. This difference was

Table 1

Descriptive Data for Brain-damaged Subjects

S no.	Diagnosis[a]	Location[b]	Age	Education	Time since injury
1[c]	CVA	LFP	48	11	acute
2[c]	T	RF	45	13	5 years
3[c]	CVA	LFT	56	10	3 years
4[c]	T	LT	33	12	acute
5[c]	T	RFT	38	16	3 years
6[c]	Tr	LT	40	14	1 year
7[c]	Tr	BiF	41	15	3 years
8[c]	CVA	RFP	44	12	1 year
9	Tr	LT	42	12	3 years
10	MS	D	42	14	1 year
11	Tr	D	40	12	3 years
12	Tr	LFT	43	7	3 years
13[c]	CVA	LFT	53	8	acute
14[c]	TLE	RT	31	13	5 years
15	CVA	RFP	47	9	1 year
16	T	BiF	25	12	5 years
17	T	LO	30	15	1 year
18[c]	CVA	LFP	38	8	5 years
19	CVA	LF	41	14	1 year
20	Tr	D	52	10	3 years

[a]CVA = cerebral vascular disease (hemorrhage, aneurysm, etc.); T = Tumor; Tr = Trauma; MS = Multiple sclerosis; TLE = Temporal lobe epilepsy.

[b]L = Left; R = Right; Bi = Bilateral; D = Diffuse; F = Frontal; T = Temporal; P = Parietal; O = Occipital.

[c]Subjects receiving Phenobarbitol and Dilantin. Dosages are given in Appendix C.

not significant (U = 165, $p > .10$).

A comparison of Tables 1 and 2 shows that the brain-damaged subjects had a higher educational level. The control group had an average education of 10.5 years, while the brain-damaged had a mean of 11.8 years of education. The difference between groups was significant (U = 106, $p < .02$).

Table 2

Descriptive Data for Control Subjects

S no.	Diagnosis	Age	Education
41	Peripheral neuropathy	48	10
42	Tension headaches	42	12
43	Osteoarthritis (left leg)	54	12
44	Peripheral nerve injury	33	8
45	Peripheral nerve injury	43	9
46	Post-traumatic neuropathy	41	6
47	Gastro-intestinal disorder	33	13
48	Gastro-intestinal disorder	45	12
49	Gastro-intestinal disorder	36	8
50	Colitis	44	18
51	Peripheral nerve injury	31	12
52	Conversion reaction	42	10
53	Gastro-intestinal disorder	43	8
54	Diarrhea	44	11
55	Gastro-intestinal disorder	50	12
56	Cordotomy for leg pain	40	12
57	Ulnar nerve injury	46	8
58	Tension headaches	40	10
59	Trigeminal neuralgia	40	10
60	Radial nerve injury	39	9

From Table 1 it can be seen that 11 of the brain-damaged subjects were taking drugs (Dilantin and Phenobarbitol) which might be expected to have an effect on the skin resistance measures. However, analysis of the data revealed that there were no significant effects. Appendix E gives the results of these tests.

Apparatus

GSR apparatus. Electrodermal measures were recorded on two channels of a polygraph (Physiograph, E and M Instrument Co.). The preamplifiers associated with this equipment allowed recording of resistance level by use of a DC bridge circuit, and concurrent recording of momentary changes (GSR). The circuit supplied a constant current of 20 microamperes applied across the electrodes.

The GSR circuit had a condensor-coupled input to block base-level changes and was constructed to return to a pre-set zero (with a time constant of five seconds) after a deflection had occurred. The instrument was calibrated by inserting a precision (0.5%) 2500 ohm resistor in series with the subject. The gain of the GSR circuit was adjusted so that large amplitude deflections would be maintained on the record, and calibrations were made for every adjustment

of the gain. Tests indicated that changes as small as 300 ohms could be measured before excessive hum occurred.

The DC bridge circuit was also calibrated by connection of a resistor in series with the subject. In this case, the gain was typically set so that a 10,000 ohm change caused a two millimeter recording pen deflection. Test recording showed that amplifier drift was zero over a one hour period with zero signal input.

The electrodes were a one-fourth inch zinc electrode used with zinc chloride paste (Lykken, 1959), for the index finger, and a 1½" by 2¼" silver plate electrode used with electrocardiograph paste for the forearm (ground electrode). The electrocardiograph paste was rubbed into the skin to minimize resistance at the ground electrode. The arm electrode was attached with a rubber strap and the finger electrode was attached with adhesive tape.

The polygraph was equipped with two marker pens, one of which provided a time base and event marker, and the other was used to record subject's responses.

Reaction time apparatus. A red and a green light (one inch in diameter) mounted six inches apart on a light gray panel six feet in front of the subject provided the stimuli for the choice reaction time task. On the subject's

chair, under his preferred (or usable) hand, was a telephone-type lever switch, adjusted so that ¼" movement to either side turned off the appropriate light. A Standard Electric timer recorded the reaction time of the subject in units of .01 second.

Paired associates learning apparatus and materials. The stimuli for this task were projected as a negative image on a white screen six feet in front of the subject by means of a remotely-controlled film-strip projector. The projected size of the stimulus words was approximately one inch in height.

The materials for this task, adapted from Stark (1961), were as follows:

Stimulus	Easy associate	Difficult associate
Dark	Light	Salt
Slow	Fast	Needle
Carpet	Rug	Whistle
High	Low	Child
Long	Short	Health
Bed	Sleep	Street
Blossom	Flower	Hammer

All subjects were first given three trials of the list of seven easy associates, e.g., dark-light, and then the stimulus word from the easy associate was paired with a difficult associate, e.g., dark-salt, for seven trials. The interval between stimuli was five seconds.

Procedure

The subjects were taken from the ward by the experimenter or an assistant. Many of the subjects had been to the laboratory on other occasions, so that both the surroundings and the experimenter were familiar. Rapport was established during the time the subject was being brought to the laboratory. The subjects were brought into the sound-insulated, air-conditioned testing room, where they were seated in a comfortable chair and connected to the equipment. During this process, they were reassured that no shock would be used, that no blood would be taken, and that the experimenter would be in the adjoining room at all times. The apparatus was described as being similar to the electroencephalograph and electrocardiograph, since these are familiar instruments to hospitalized patients. After a preliminary description of the conditions of the experiment, the experimenter left the room. All recording equipment and controls for presenting stimuli were located in an adjoining room. Further instructions were given over an intercommunication system.

Experimental Conditions and Procedure

The experimental conditions and procedure were as follows:

1. Initial rest period. The subject was instructed to relax, refrain from gross movements, and to keep awake with his eyes open. This period continued for 20 minutes, and throughout the period instrumental music, selected to promote relaxation, was played through a speaker in the subjects' room. The music ended within the last half-minute of the period.

2. Startle stimulus. At the end of the twenty minute period, a buzzer was sounded for two seconds.

3. Rest I. A three minute rest period followed the startle stimulus.

4. Adaptation period. Pilot studies indicated that the repeated buzzer brought about varied responses, even in normal subjects, therefore, at the end of Rest I, the subject was told:

> You heard that buzzer a few minutes ago. It will sound a few more times, but you don't need to pay any special attention to it.

The buzzer was then sounded for 15 two-second trials, with 30 second intervals between trials.

5. Rest II. A three minute rest period followed the adaptation trials.

6. Reaction time period. At the end of Rest II, the following instructions were given:

> This is a test to see how fast you can react when you have to make a choice. On the board in front of you are two lights. I want you to look at the black dot between the lights. The light on the right is red (light was turned on). On the right (or left) arm of your chair is a small black handle. If you turn the handle to the right, the light will go off. Would you do that now, please. The other light is green (light was turned on). Now if you turn the handle to the left, the light will go off. Turn the handle now, please. All you have to do is push the handle over and it will spring back by itself. I will turn on one or the other of the lights from out here, and you are to turn it off as fast as you can by turning the handle.
>
> Remember to turn the handle to the right to turn off the red light, and turn the handle to the left to turn off the green light. Do you have any questions? Now let's try it a few times for practice.

Six practice trials followed, and then the subject was told:

> That was fine. Now let's go on with the test. Remember to turn the handle as fast as you can to turn off the light.

Thirty test trials followed. Both the six practice trials and the test trials were equally divided between right and left responses, presented in a pre-arranged order. Stimuli were presented with two, four, or six second inter-trial intervals in a pre-arranged order to prevent a temporal set. The order was the same for all subjects. At the end of the task the subject was told: "That's fine. Now just relax for a couple of minutes."

7. Rest III. At the end of the reaction time period,

a three minute rest was given.

8. Paired associates learning period. At the end of Rest III, the projector was turned on, and the following instructions were given:

> I am going to show you some words on the screen in front of you. At first a word will come on, and then that same word will come on paired with another word. I want you to learn the words that are paired together, so that when the first word comes on the next time, you can tell me the word that goes with it. For example, I'll show you the word Chair (shown to subject) and then I'll show you Chair and Table together (shown to subject). Now if I were to show you Chair again, what would you say?

If the subject did not give the correct response to the Chair-Table example, the instructions were repeated and the example explained until it was apparent that the subject understood the task.

The subject was then told:

> Of course, the first time through, you won't know the words that are paired, so I will show them to you, and I want you to read them out loud as they come on the screen. Remember I want you to learn the words that are paired together.

The first trial of the easy list followed the instructions. After this trial, the subject was told:

> Now when the next word comes on the screen, tell me the word that goes with it. After a word appears, you have five seconds to give me the answer.

The easy list was then presented for two more trials.

The order of pairs was presented in a pre-arranged sequence which was the same for all subjects. After the last pair of the easy list, the subject was told:

> Now I am going to show you the same words that you saw before, but this time they have different words paired with them. I want you to read them aloud, just like you did the first time, and learn the words that are paired together.

The first trial of the difficult list followed. At the end of the trial, the subject was told:

> Now when the next word comes on the screen, tell me the word that goes with it. After a word appears, you have five seconds to give me the answer.

If a subject failed to give responses after his first few failures, which occurred commonly in both brain-damaged and control subjects, he was told to read the stimulus words aloud and urged to try to guess its associate.

The difficult list was then presented for six more trials, with the order of pairs within trials in a pre-arranged sequence. At the end of the sixth trial the experimenter said:

> How do you think you are doing, Mr. _____?

After the subject's response, which was uniformly negative, the experimenter said:

> Well, you are not doing very well at all. What do you suppose is the matter?

After the subject had responded, the experimenter said:

> Maybe you are not trying hard enough. Now this time, I want you to try real hard and see if you can't do better.

At the end of the trial, the subject was told:

> That time you did better than before. In fact, over that whole test, you did much better than most people do on a test like that. Now the tests are over and I want you to sit and relax for a few more minutes.

The score consisted of the number of errors or omissions made during the two easy trials, the six difficult trials, and the last, or failure, trial.

9. Final rest period. After the instructions at the end of the failure trial, a ten minute rest period was given. At the conclusion of this period, the electrodes were removed from the subject, he was reassured as to the quality of his performance, and given the booklet form of the Minnesota Multiphasic Personality Inventory to be completed on his ward.

Derivation of Electrodermal Measures

As previously indicated, two measures of electrodermal activity were used in this study. The measures were GSR frequency and level of skin resistance.

In order to take into account a possible, but unknown, relationship between GSR amplitude and skin resistance level, a procedure for counting GSR frequencies was established on the following basis: It was established that the GSR recording equipment could reliably measure changes of the order of 300 ohms. The ratio between this change and the lowest level of skin resistance recorded (20K ohms) was converted to a percentage. This percentage (one and one-half percent) was used as a criterion for GSR counting. That is, in order for a deflection to be counted, its amplitude had to exceed one and one-half percent of the skin resistance level at the time of deflection. Frequencies of GSRs which exceeded this criterion were recorded for each minute of the experimental session, with the exception of the paired associates period, where the recording was made on the basis of trials.

Readings were taken for skin resistance level at each minute of the experiment, and at the beginning and end of each condition, with the exception of the paired associates condition, where readings were taken by trials. At the conclusion of the experiment, distributions of the resistance readings were made for the initial rest, adaptation, and reaction time conditions. In order to correct for the non-

normality of these distributions, and to make interpretation of the level readings correspond to statements about arousal, the resistance readings were converted to log conductance (the logarithm of the reciprocal of the resistance).

Since there were two measures of electrodermal activity, their relationships were examined. This procedure was felt necessary since it was possible that the two measures would provide essentially the same information, obviating the necessity of examining both measures in relation to the experimental conditions. Table 3 presents the results of rank-order correlations (Siegel, 1956) computed between mean log conductance scores and GSR frequencies for the various experimental conditions in the control group.

Table 3

Rank-order Correlations Between Log Conductance and GSR Frequency in the Control Group

Condition	r_s	p
Initial rest	.27	
Adaptation	.23	
Reaction time	-.36	.05
Paired associates	-.12	
Final rest	.13	

The lack of a consistent relationship between GSR frequency and log conductance level in the control group justifies the consideration of both measures in relation to the experimental questions.

CHAPTER IV

RESULTS

The purpose of this experiment was to investigate arousal phenomena in brain-damaged subjects in comparison with a control group and also to derive information regarding the relationship between performance and arousal in the brain-damaged. The findings of the experiment will be presented in the following order:

1. Results concerning task performance;
2. Results relating to the various experimental questions for the conductance measures;
3. Results relating to the various experimental questions for the GSR measures;
4. Additional findings;
5. Summary of the results.

Group Differences in Task Performance

The subjects included in the brain-damaged group were defined by a neurological criterion. However, unless

they had impaired performance on the experimental tasks, there would be little basis for making inferences about relationships between performance and electrodermal indicants of arousal. Thus, before taking up the results relevant to the experimental questions, the findings relative to the reaction time and paired associates tasks will be briefly presented.

Reaction time. For the choice reaction time task, the mean score for the brain-damaged group was .530 seconds, and for the controls, .410 seconds. The difference between the groups for this measure was significant by the Mann-Whitney U test (Siegel, 1956) ($U = 122$, $p = .04$).

Paired associates learning. For the learning task, Stark's (1961) results were, in part, supported by the findings of this study. For the easy list, the mean errors score for the brain-damaged was 2.2, while the mean error score for the control group was 0.8. The difference between the groups was highly significant ($U = 68$, $p < .001$, one-tailed test).

The mean error scores for the difficult task were also significantly different. The mean score for the brain-damaged was 26.4, and for the controls, 22.4 ($U = 126$, $p = .025$, one tailed test).

For the failure trial, the brain-damaged and control groups were again different in mean error scores. The mean score for the brain-damaged was 3.7, while the control group had a mean score of 2.7. This difference is also significant ($U = 122$, $p = .04$).

Thus, the subjects classified as brain-damaged show behavioral deficits similar to those of subjects in other studies.

Electrodermal Measures of Arousal

In Chapter II a series of questions was asked regarding the effects of the experimental treatments on the arousal of brain-damaged subjects. The results relevant to these questions will be considered in the order in which the questions were presented. Since there were two measures of arousal, log conductance and GSR, the questions will be considered first for the conductance measure and then for the GSR measure. The lack of a consistent relationship in the control group justifies the inclusion of both measures.

Although the most preferable and powerful test for group comparisons would have been a repeated measurements analysis of variance, it became apparent that the data did not meet the fundamental assumptions for this technique.

The results of tests for homogeneity of variance, given in Appendix F, indicated that, by and large, the data were not suitable for parametric statistical tests. Thus, two-tailed non-parametric statistical techniques were used for the comparisons made to answer the experimental questions.

Arousal as indicated by log conductance. 1. What is the resting level of arousal in brain-damaged subjects compared with control subjects?

Figure 1 presents the minute-by-minute mean log conductance levels for the two groups for the 15 minute initial rest period. A general downward trend is apparent in both groups, with the brain-damaged having a higher conductance level.

Using the mean log conductance level for the entire fifteen minute period as an estimate of the central tendency of the two groups during rest, a Mann-Whitney U test indicated a significant difference between levels for the two groups. Table 4 includes the results of this test.

A U test was also performed on the log conductance measures for the first minute of the initial rest period. Here the result approached significance, as shown in Table 4.

For the last minute of the initial rest period, Table 4 indicates a significant difference between the groups.

Table 4

U Test Comparisons of Differences in Log Conductance in BD and Control Groups

Comparison	U	p[a]
Initial rest		
Mean level (15 minutes)	123	.04
First minute	135	.08
Fourteenth minute	132	.07
Last minute	122	.04
Startle	135	.08
Rest after startle		
First minute	129	.06
Last minute	118	.03
Adaptation		
Mean level (seven minutes)	115	.02
Rest after adaptation		
First minute	122	.04
Last minute	114	.02
Reaction time		
Mean level (seven minutes)	147	>.10
First minute	160	>.10
Last minute	128	.05
Rest after reaction time		
First minute	129	.06
Last minute	130	.06
Paired associates		
Mean level (seven minutes)	144	>.10
Easy list (mean of three trials)	178	>.10
Difficult list (mean of six trials)	160	>.10
Failure trial	165	>.10
Final rest		
Mean level (ten minutes)	116	.02
First minute	138	.10
Last minute	115	.02

[a]For a 20 x 20 comparison (two-tailed test) the expected U = 200.

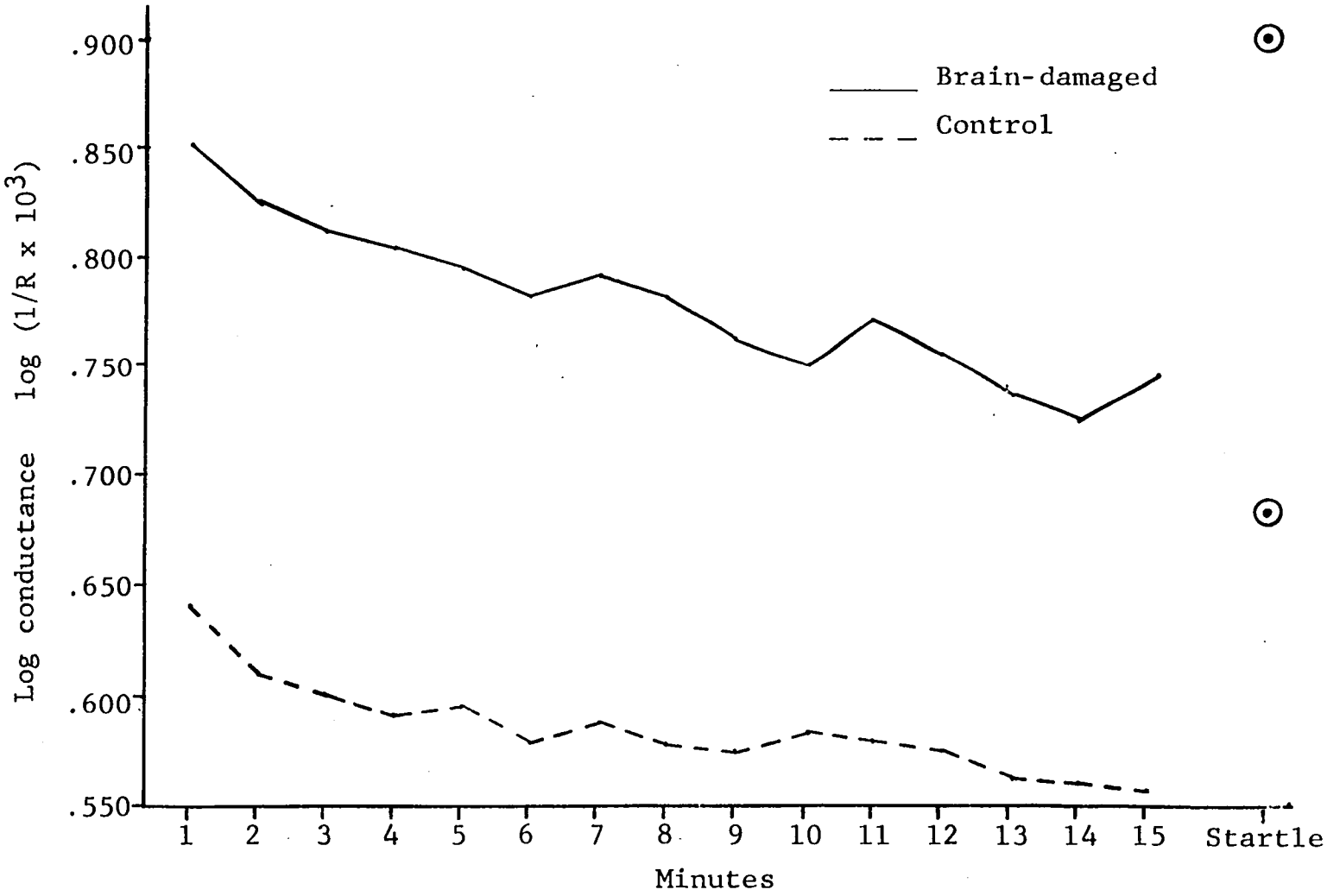

Fig. 1. Mean log conductance in brain-damaged and control groups during the initial rest condition.

2. How does the level of arousal change during rest?

The change in log conductance was assessed by taking the difference between the mean of the measures for the first five minutes of the initial rest period and the mean of the last five minutes of this period. The difference between brain-damaged and control groups for this index of change was not significant, as shown in Table 5.

3. How does the level of arousal change in response to a startle stimulus?

Figure 1 presents the mean log conductance level for the two groups for the reading taken after the presentation of the startle stimulus (labeled S in the figure).

The difference in log conductance level for the two groups for the startle condition is of questionable significance, as can be seen in Table 4.

The change in level was determined by comparing the difference between the last minute of the initial rest period and the log conductance measure after the startle stimulus. The two groups were not significantly different in amount of change to this stimulus (see Table 5).

4. What are the arousal characteristics of the adaptation process to repeated stimulation?

Table 5

U test Comparisons of Differences in Change in Log Conductance in BD and Control Groups

Comparison	U	p[a]
Initial rest		
Mean (min. 1-5) - Mean (min. 11-15)	188	>.10
Startle	164	>.10
Adaptation		
Min. 1 - min. 4	118	.03
Min. 4 - min. 7	169	>.10
Reaction time		
Min. 1-7	162	>.10
Min. 1-4	187	>.10
Min. 4-7	146	>.10
Paired associates		
All trials (excluding failure)	193	>.10
Easy task	153	>.10
Difficult task	126	.05
Failure instructions	151	>.10
Failure trial	186	>.10
Final rest		
Mean (1-5) - Mean (6-10)	125	.05

[a]For a 20 x 20 comparison (two-tailed test) the expected U = 200.

Figure 2 gives the mean log conductance values for the groups for the adaptation period. The level for the entire period was higher in the brain-damaged subjects, and

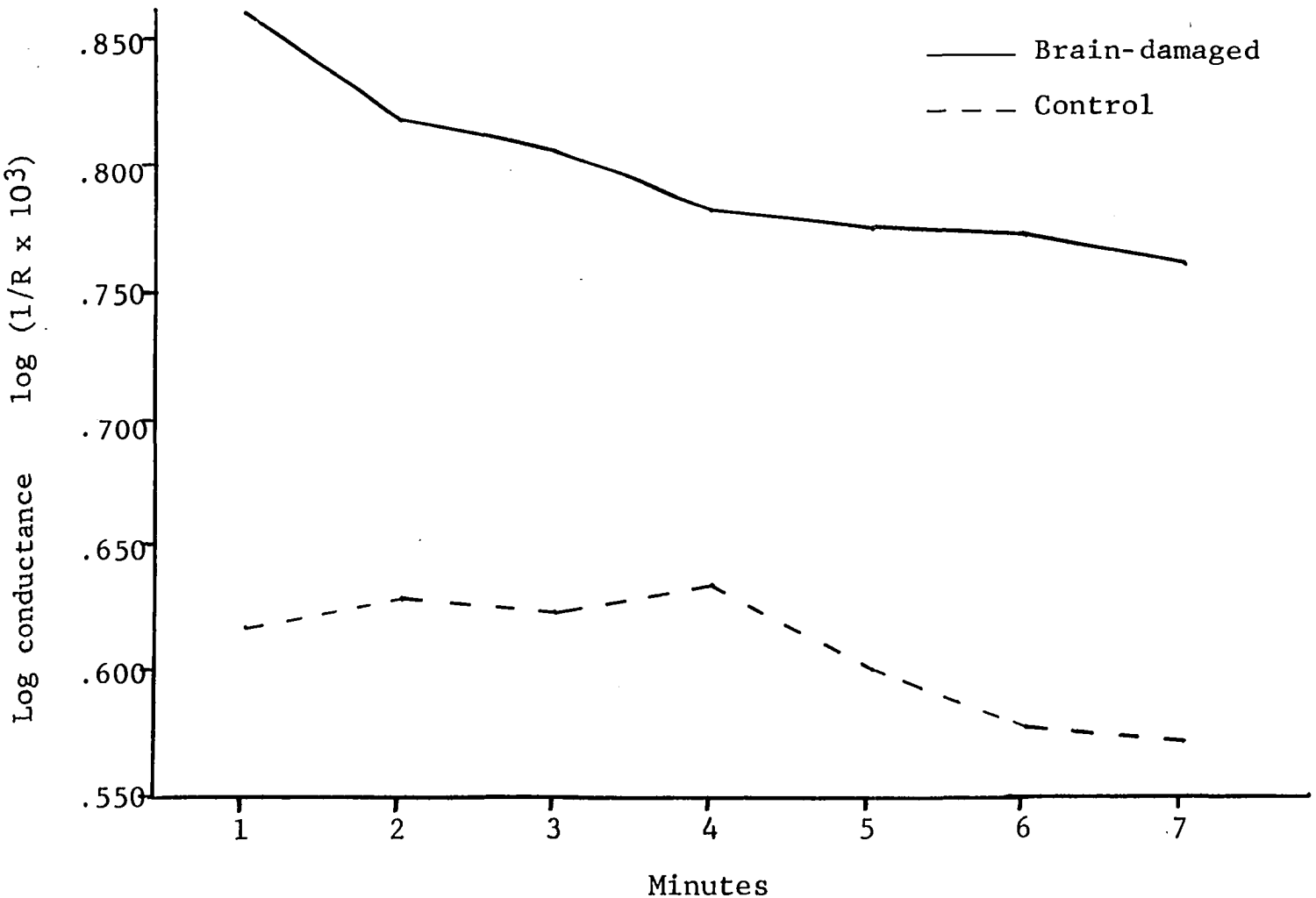

Fig. 2. Mean log conductance in brain-damaged and control groups during adaptation.

both groups showed a general adaptation effect.

As shown in Table 4, the mean level for the entire period was significantly different for the two groups.

In order to determine the change in log conductance over the adaptation period, the period was divided into two equal parts, and differences were taken between minute one and minute four, and between minute four and minute seven (see Figure 2).

From Table 5 it can be seen that there was a significant difference in the change in log conductance during adaptation only for the first half of the period, with the brain-damaged subjects showing the greater change.

5. How does the level of arousal change during a choice reaction time task?

Figure 3 shows again a consistently higher log conductance level for the brain-damaged. The two groups were not significantly different in mean level for the seven minutes of the reaction time period, nor for the first minute, but did differ significantly at the last minute, as shown in Table 4.

Table 5 demonstrates that the brain-damaged and control groups did not show significantly different changes in level, either over the entire period, or when the period was

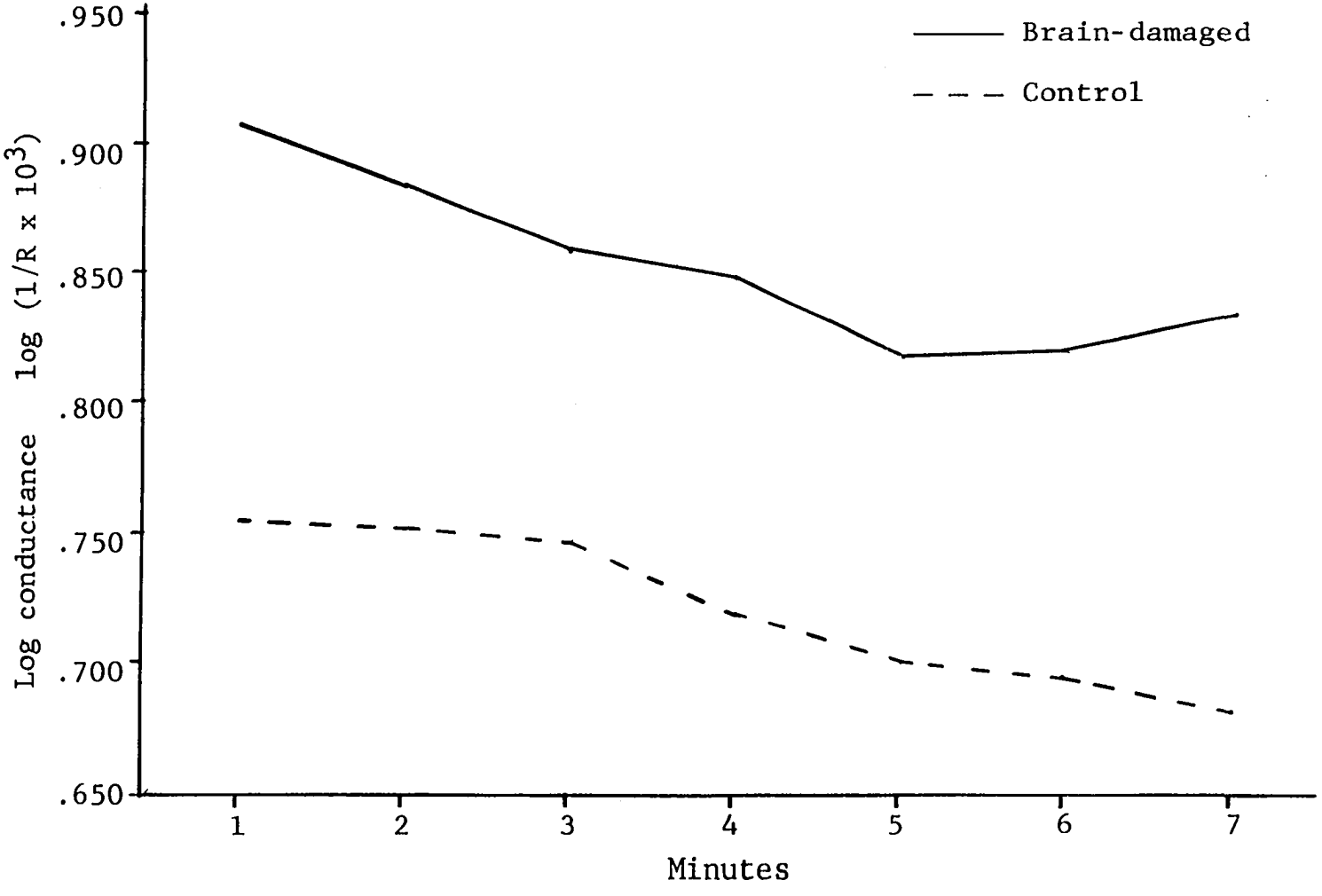

Fig. 3. Mean log conductance in brain-damaged and control groups during choice reaction time.

divided into halves.

6. How does the level of arousal change during a paired associates learning task?

The data for this period, by trials, is presented in Figure 4. The analysis for this condition was made on the basis of trials, as opposed to minutes, since the paired associates period was composed of three parts. The two groups did not differ in mean level for all conditions combined, as indicated in Table 4, although the brain-damaged showed consistently higher log conductance scores. When the data for the paired associates period were subdivided into the separate conditions, the mean log conductance levels were again not significantly different, as shown in Table 4.

When the change in log conductance during paired associates learning is viewed in relation to task difficulty, Table 5 demonstrates that there was a significant difference between brain-damaged and control subjects for change during the difficult task; but no significant differences in change were found for the easy task.

7. What is the effect of failure on arousal during a paired associates learning task?

Figure 4 presents the log conductance data for the failure instructions and failure trial. Both groups

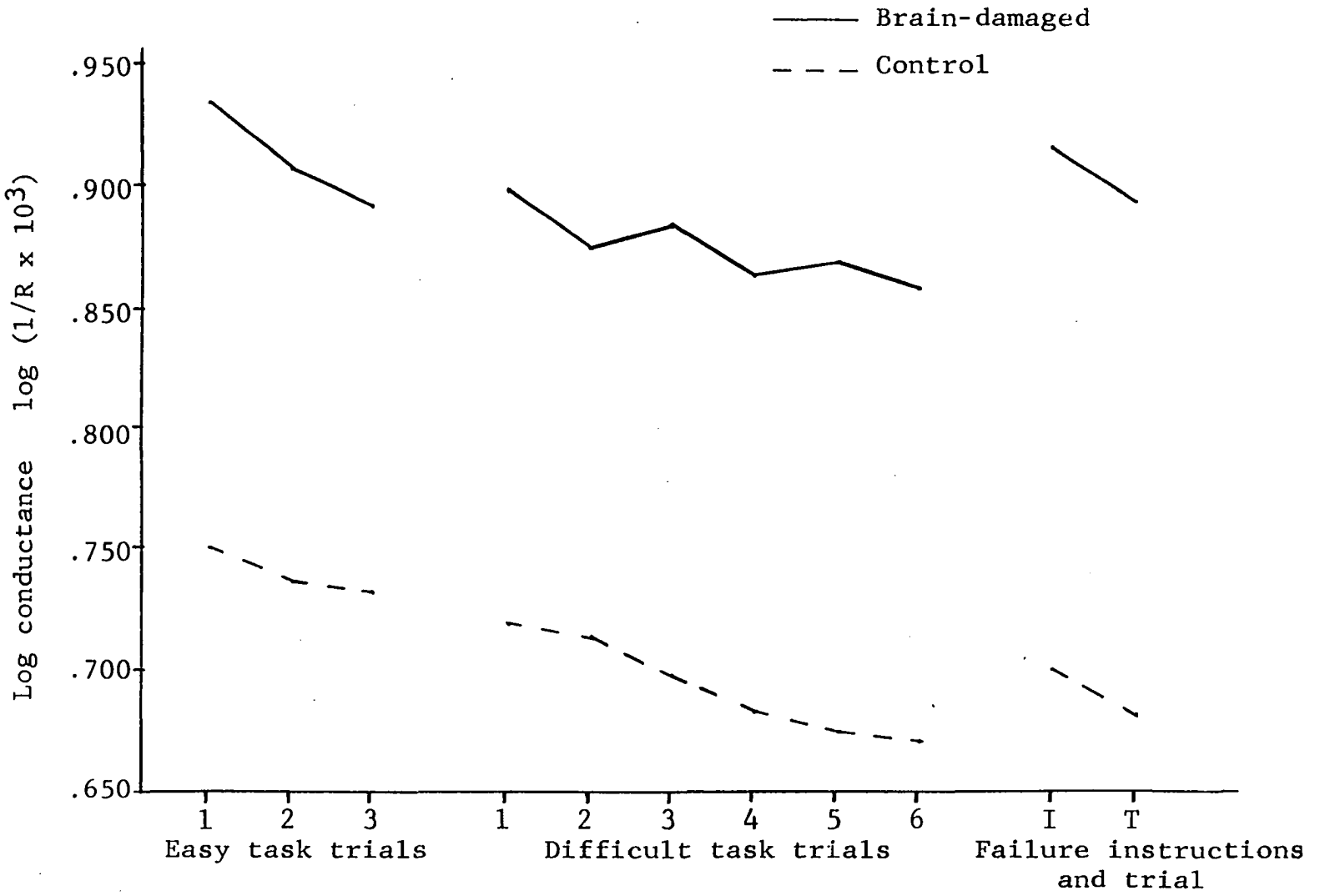

Fig. 4. Mean log conductance in brain-damaged and control groups during paired associates learning.

responded to the failure instructions with an increase in conductance; but there was no significant difference between the groups in change to the failure instructions (Table 5), or in change during the failure trial (Table 5), or in mean log conductance level during the failure condition (Table 4).

8. What is the pattern of return to resting level after the experimental conditions?

The results of U tests for differences in log conductance for the rest periods which followed the various experimental conditions are given in Table 4. The brain-damaged subjects had consistently higher log conductance levels for all these periods, and, as shown in Table 4, were generally significantly different from the control subjects.

The data for the ten minute final rest period are presented in Figure 5. Table 4 indicates that the brain-damaged and control groups did not differ significantly for the first minute of the period, but were different for mean log conductance level for the entire period, and also for the last minute of the final rest period.

The change in log conductance for the final rest period was measured by the difference between the mean of the measures for the first five minutes minus the mean of the last five minutes. Table 5 indicates that there was a

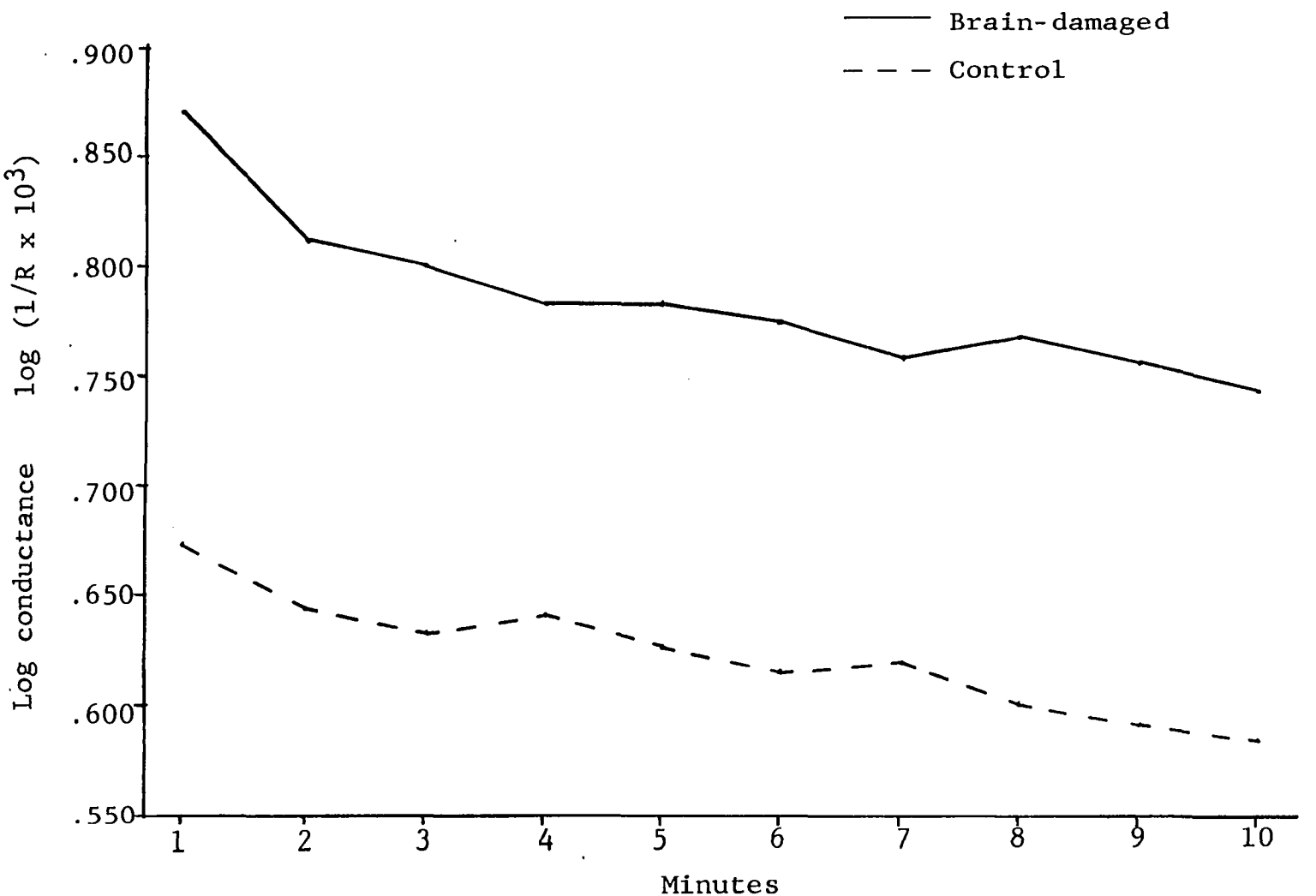

Fig. 5. Mean log conductance in brain-damaged and control groups during final rest.

significant difference between the groups in change in log conductance when the two halves of the final rest period were compared.

9. What is the relationship between performance on the reaction time and paired associates learning tasks and arousal in brain-damaged and control subjects?

In Table 6 are given rank-order correlation coefficients for the brain-damaged and control groups between the log conductance measure of arousal and performance on the experimental tasks. The order of ranking resulted in a positive coefficient indicating high arousal and good performance. The most apparent feature of this comparison is the difference between the groups in signs of the coefficients for the paired associates learning task.

To test for the significance of the difference between the coefficients given in Table 6, recognizing the assumption that r_s is an approximation to r, the z transformation (McNemar, 1949) was used. By this test, the difference between the coefficients approached significance in the case of initial rest mean level and paired associates errors ($z = 1.7$, $p = .08$, two-tailed test). For the paired associates period mean log conductance level and paired associates errors, the difference was significant ($z = 2.2$,

p = .03, two-tailed test).

Table 6

Rank-order Correlations Between Performance on Reaction Time and Paired Associates Learning Tasks and Log Conductance in BD and Control Subjects

Period	BD	Control
Reaction time		
Mean level initial rest and mean RT score	-.30	.14
Mean level reaction time period and RT score	-.03	-.10
Paired associates learning		
Mean level initial rest and PA errors	.38*	-.21
Mean level PA period and PA errors	.35	-.38*

*p = .05

Arousal as indicated by galvanic skin response. In this section, the experimental questions are considered in sequential order relative to the GSR measure of arousal.

1. What is the resting level of arousal in brain-damaged subjects?

Figure 6 gives the data for the initial rest period for the frequencies of GSRs by minutes. In Table 7 are

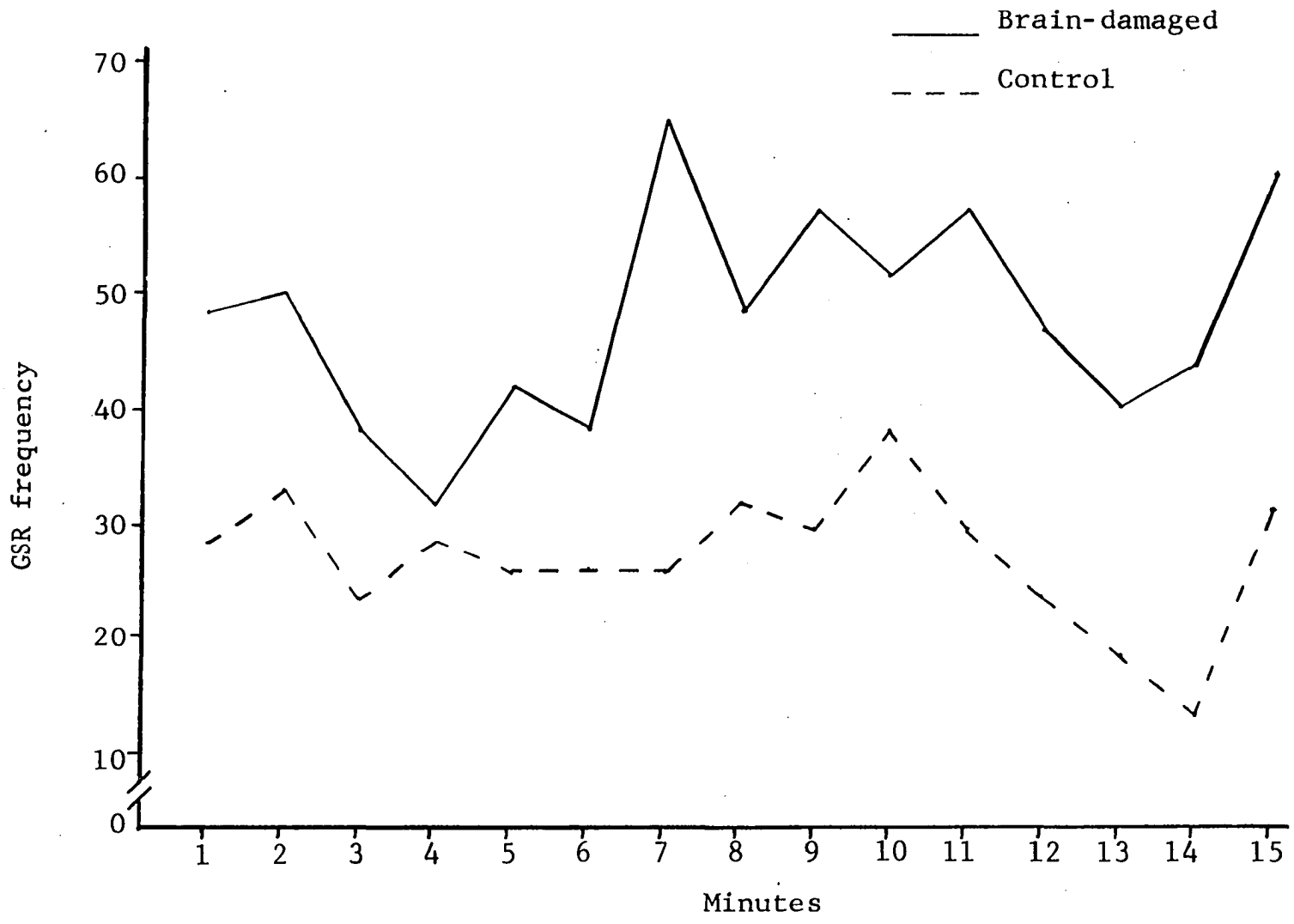

Fig. 6. Total GSR frequencies in brain-damaged and control groups during initial rest.

Table 7

U Test Comparisons of Difference in GSR Frequency in BD and Controls

Comparison	U	p
Initial rest		
Total GSR (15 minutes)	156	> .10
First minute	150	> .10
Fourteenth minute	64	< .002
Fifteenth minute	160	> .10
Startle		
GSR amplitude	184	> .10
Rest after startle		
Minute 1	157	> .10
Minute 3	120	.03
Adaptation		
Total GSR	106	< .02
Rest after adaptation		
Minute 1	117	.025
Minute 3	109	< .02
Reaction time		
Total GSR	178	> .10
Rest after reaction time		
Minute 1	169	> .10
Minute 3	142	> .10
Paired associates		
Total GSR (easy and difficult lists combined)	170	> .10
Easy list		
Total GSR	135	.08
Trial 1	146	> .10
Trial 2	134	.08
Trial 3	142	> .10
Difficult list		
Total GSR	162	> .10

Table 7--Continued

Comparison	U	p
Difficult list (continued)		
Trial 1	134	.08
Trial 2	132	.07
Trial 3	129	.06
Trial 4	130	.06
Trial 5	187	>.10
Trial 6	145	>.10
Failure		
Instructions	140	>.10
Trial	114	.02
Final rest total	154	>.10
Minute 1	166	>.10
Minute 4	102	<.02
Minute 10	28	<.002

given the results of U tests for the GSR measure.

In terms of the total number of GSRs during the initial rest period, the two groups were not significantly different, nor were they different for the last minute of the period. The groups did not differ significantly at the first minute, but were highly significantly different at the fourteenth minute. The comparison for the fourteenth minute was made since the last minute of measurement included GSRs which were caused by the end of the music played during the initial rest period.

2. How does the level of arousal change during rest?

From Figure 6 it can be seen that there is a slight trend toward a decrease in the number of GSRs per minute in the control group as the rest period continues. Such a decrease is not so apparent in the brain-damaged, although the difference between groups in the amount of change is not statistically significant, as shown in Table 8.

Table 8

U Test Comparisons of Differences in Change in GSR Frequencies in BD and Controls

Comparison	U	p
Initial rest		
Mean (1-5) - Mean (11-15)	168	>.10
Adaptation		
Min. 1-4	128	.05
Min. 4-7	100	<.02
Reaction time		
Min. 1-7	115	.02
Min. 1-4	122	.04
Min. 4-7	100	<.02
Paired associates		
All trials (excluding failure)	165	>.10
Easy task	131	.06
Difficult task	162	>.10
Failure trial	127	.05
Final rest		
Minute 1-10	97	<.02
Mean (1-5) - Mean (6-10)	102	<.02

3. How does the level of arousal change in response to simple stimulation?

The change in arousal to the startle stimulus was assessed by converting the amplitude of the GSR to this stimulus to conductance units ($1/R \times 10^3$). A U test for the significance of the difference between groups in GSR amplitude to the startle stimulus was not significant ($U = 184$, $p > .10$). The mean response (in conductance units) for the brain-damaged group was .543, and .768 for the control subjects.

The change in arousal to the startle stimulus was also considered in terms of the number of GSRs to the stimulus. The total number of responses given by the brain-damaged group was 29, and the control subjects gave a total of 21 responses. Although the difference in frequencies of GSRs was not extreme, it indicated a greater tendency on the part of the brain-damaged subjects to give more than one response. Since there were many subjects who gave only one response, with a resulting large number of ties, the U test was not used for this comparison. The result of a median test (Siegel, 1956) approached significance ($\chi^2 = 2.77$, $p = .10$).

4. What are the arousal characteristics of the adaptation process to repeated stimulation?

Figure 7 indicates that the brain-damaged have a uniformly greater frequency of GSRs during the adaptation period. The total number of responses for the period was significantly different for the two groups, as shown in Table 7.

There was also a significant difference between the brain-damaged and control groups in the amount of change over the adaptation period. This result, as shown in Table 8, was computed by dividing the adaptation period into halves. Inspection of Figure 7 reveals that the control group showed the greater change in GSR frequency.

5. How does the level of arousal change during a choice reaction time task?

The GSR frequency data for the reaction time period are presented in Figure 8. It is apparent from the figure that the brain-damaged and control groups were quite similar in the frequency of GSRs during the task. The groups were not significantly different in total GSR frequency during the reaction time task, as shown in Table 7.

The brain-damaged and control groups were found to be significantly different in the amount of change in GSR frequency during the reaction time period, when the difference was taken between the first and last halves of the

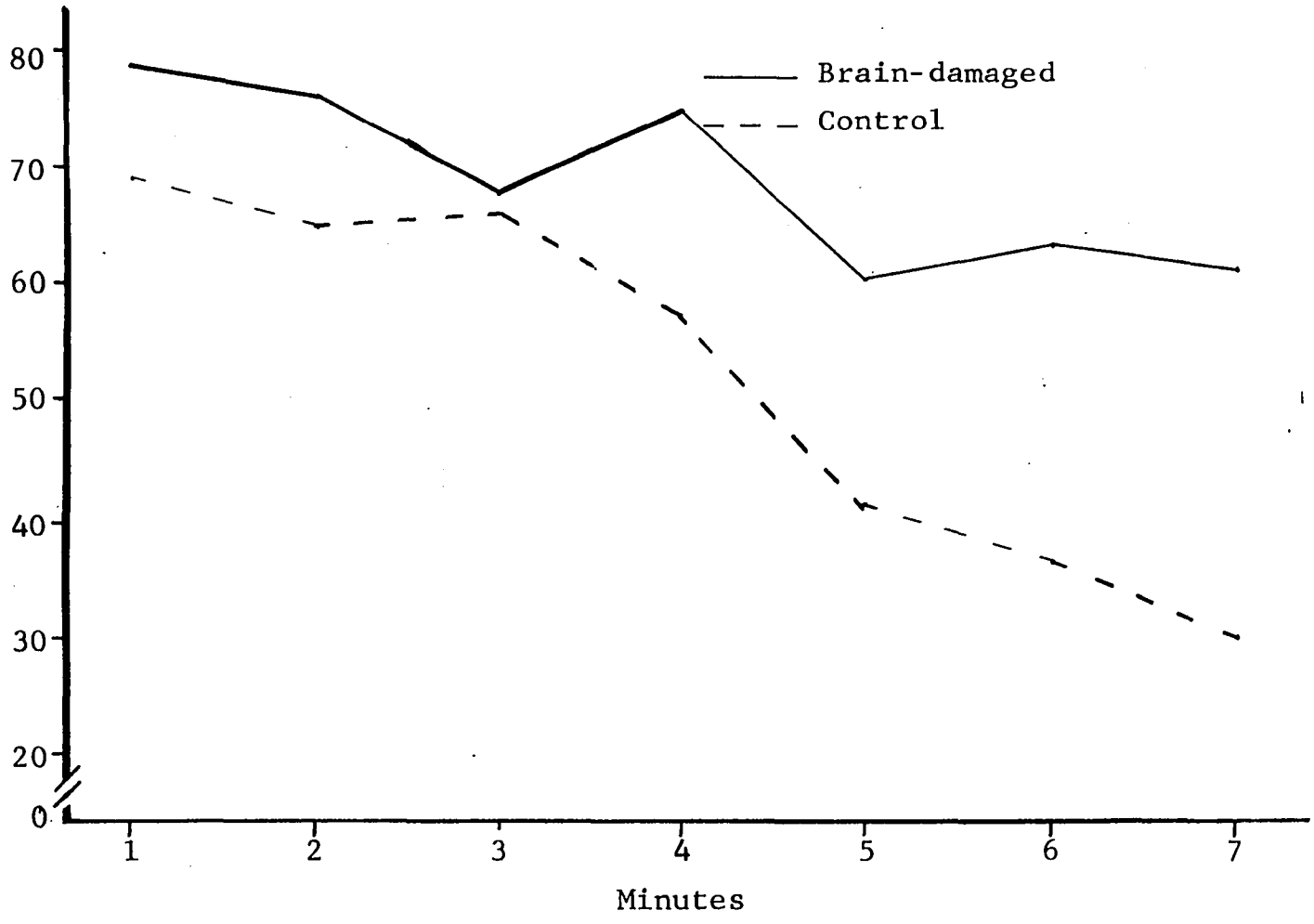

Fig. 7. Total GSR frequencies in brain-damaged and control groups during adaptation.

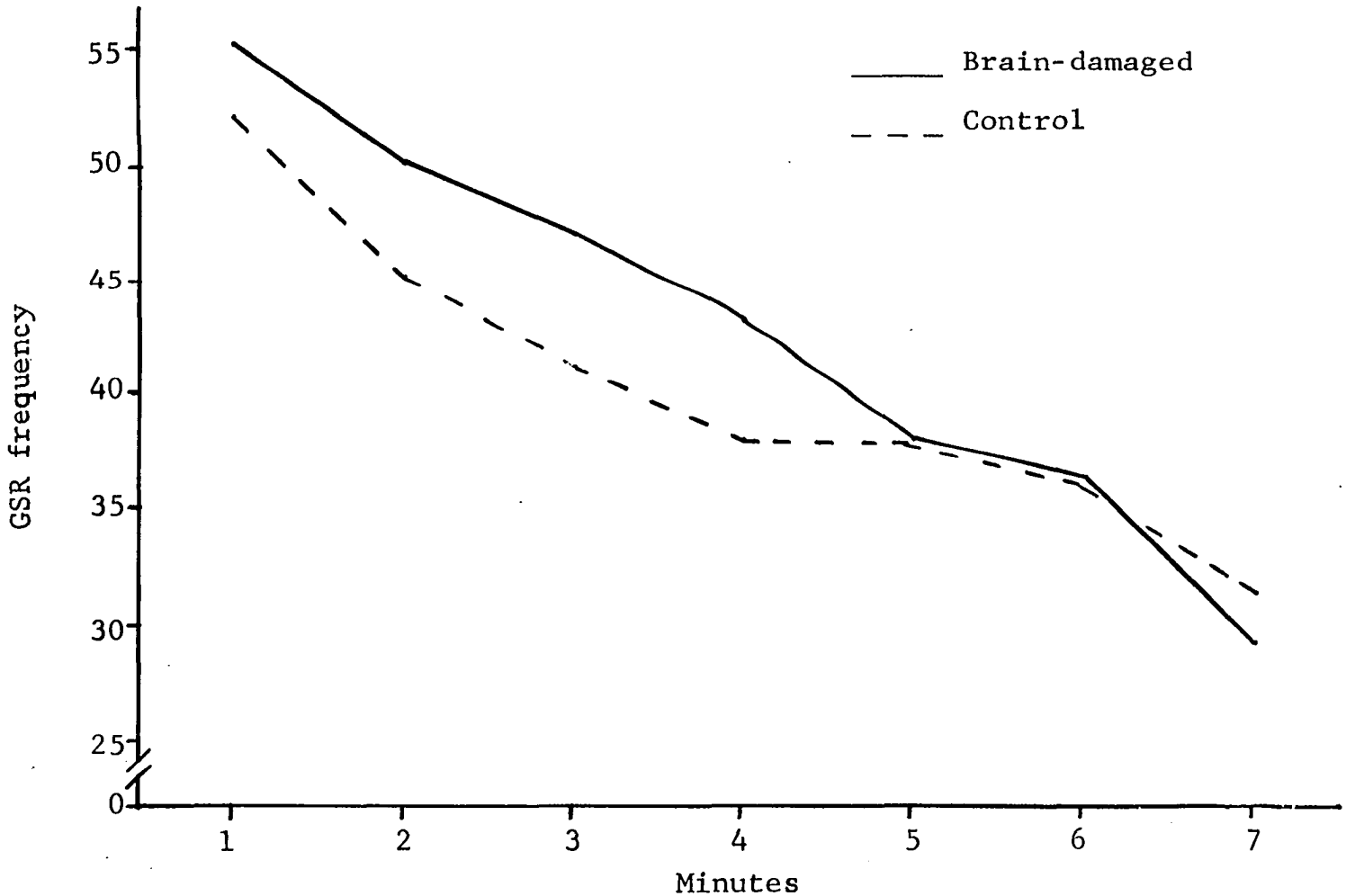

Fig. 8. Total GSR frequencies in brain-damaged and control groups during choice reaction time.

period, as shown in Table 8.

6. How does the level of arousal change during a paired associates learning task?

Figure 9 gives the GSR frequency data for this period. As was the case with the log conductance measure, the difference in the conditions within the period was taken into account by recording frequencies by trials. The brain-damaged group, as evidenced by Figure 9, was generally more reactive during this period.

In Table 7 are given the results of U tests for the paired associates conditions. The two groups were not significantly different in the frequency of GSRs during the learning tasks. However, when the easy and difficult tasks were analyzed on the basis of individual trials, some of the differences between groups approached significance, as indicated by Table 7.

The results of tests relating to change in GSR frequency during the paired associates period are given in Table 8. The change during the easy task approached statistical significance, while change in GSR frequency during the difficult task did not.

7. What is the effect of failure on arousal during paired associates learning?

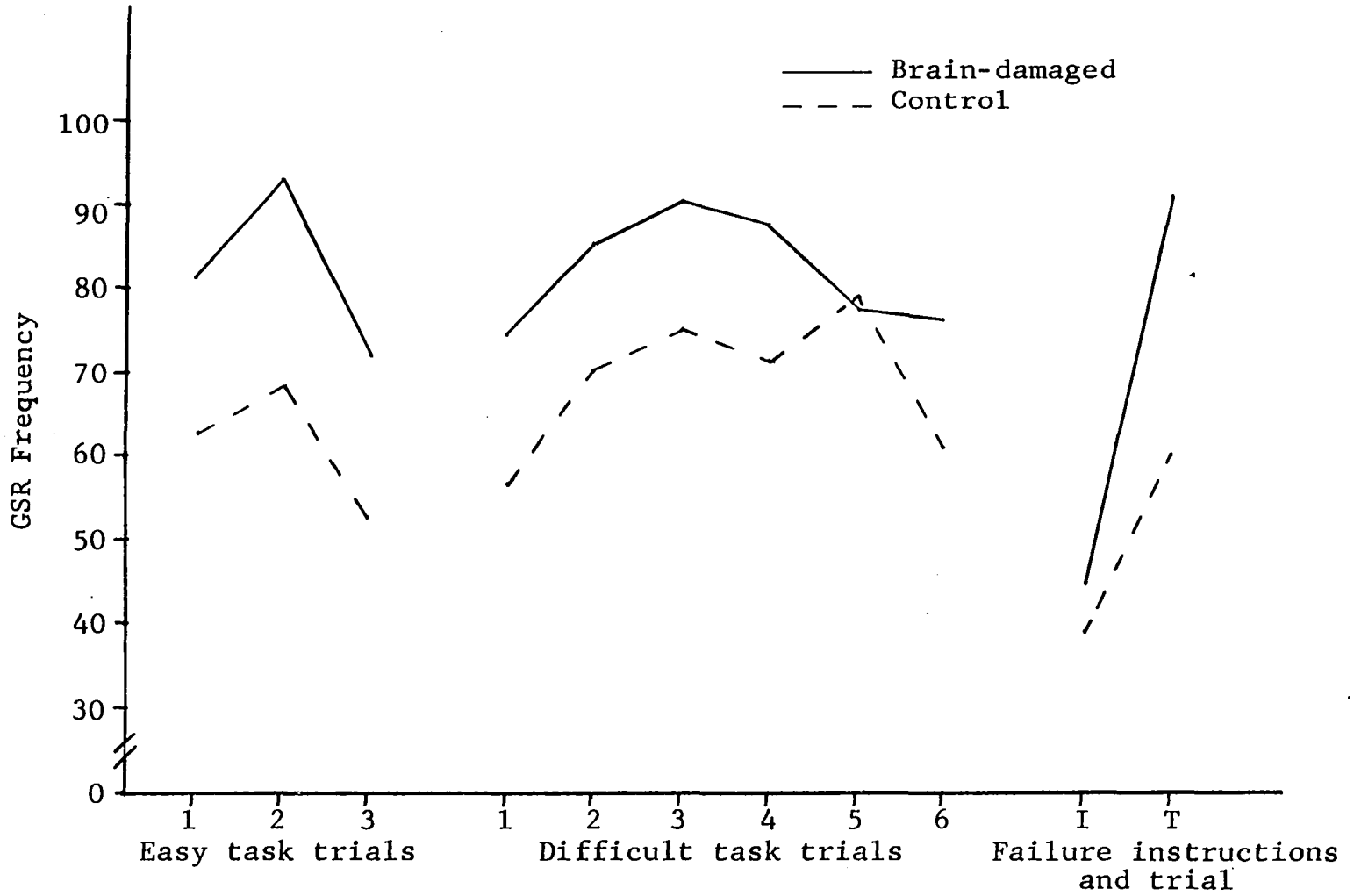

Fig. 9. Total GSR frequencies in brain-damaged and control groups during paired associates learning.

The data relevant to this condition are also given in Figure 9. The figure indicates that the groups responded to essentially the same degree to the failure instructions, but quite differently during the failure trial itself. These observations are borne out by the results of U tests, given in Table 7, which show a significant difference between groups for the failure trial while the difference between groups in response to failure instructions is not significant.

Table 8 includes the results of tests concerned with change to the failure situation. The brain-damaged and control subjects show significant differences in the amount of change during the failure trial, as shown in the table.

8. What is the pattern of return to base level after the experimental conditions?

The tests for differences in frequencies of GSRs during the rest periods which followed the experimental conditions are given in Table 7. The two groups do not show consistent differences, as evidenced by the table.

Figure 10 indicates striking differences between the brain-damaged and control groups in their GSR frequency during the final rest period. The control group shows a rather typical curve of relaxation or habituation. The brain-damaged, on the other hand, appear to be relaxing for the first half of the period, and then show an increase in GSR

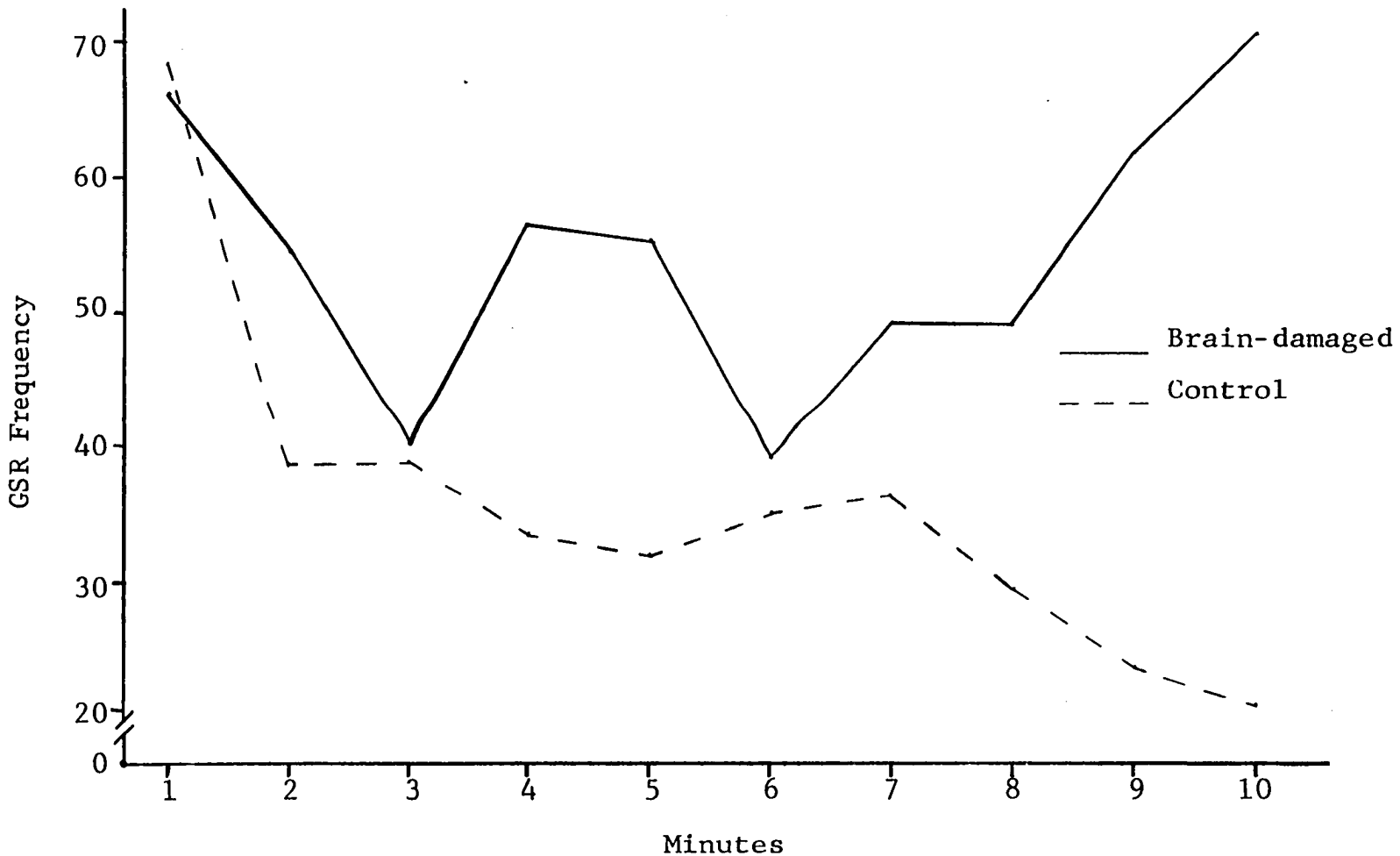

Fig. 10. Total GSR frequencies in brain-damaged and control groups during final rest.

frequency over the latter half.

The results of U tests for the final rest period are given in Table 7. As shown in the table, the brain-damaged and control subjects are not significantly different in GSR frequency at the first minute of the period, do differ at the fourth minute, are not significantly different for the sixth minute, but at the end of the final rest period are highly significantly different.

Analysis of change in GSR frequency for the final rest period is given in Table 8. As indicated by the table, the brain-damaged and control groups show extreme differences in the amount of change over the final rest period, with the control subjects showing the greater change. This difference is due to the divergence in the groups' response during the latter half of the period.

9. What is the relationship between performance on the reaction time and paired associates learning tasks and arousal in brain-damaged and control subjects?

Table 9 gives rank-order correlation coefficients between GSR frequency and performance in the brain-damaged and control groups.

None of the correlation coefficients reported in Table 9 are significant, although the reaction time period

Table 9

Rank-order Correlations Between Performance on Reaction Time and Paired Associates Tasks and GSR Frequency in BD and Controls

Period and Task	BD	Control
	r_s	
Reaction time		
Initial rest GSR and RT score	-.11	-.02
Reaction time period GSR and RT score	.35	.18
Paired associates Initial rest GSR and PA errors	.29	-.21
Paired associates Period GSR and PA errors	.24	-.13

GSR and reaction time score coefficient approaches significance (p approximately .08). The differences between coefficients are similarly not significant, the largest difference being between initial rest GSR and paired associates errors. For this comparison, $z = 1.1$, $p = .13$. It is of interest to note that the direction of relationship is generally consistent with the results of the comparison with the conductance measure (Table 6).

Additional Findings

The incidence of "compound" GSRs. As the GSR records were being analyzed, it was noted that in response to the startle and adaptation stimuli, certain subjects would give a GSR with a more complex wave form than usually seen. That is, the recording pen would describe what appeared to be one response which was superimposed on another. The recording pen would reach an apparent peak deflection and begin the return to base line, but before reaching zero would immediately begin another deflection. This pattern, referred to as a compound GSR, was noted to occur with greater frequency in control subjects than in the brain-damaged. The frequency of such GSRs was counted by the experimenter and an assistant independently after the criteria for counting had been defined. The recorded frequency of compound responses differed by a total of two for the two independent assessments.

The total number of compound GSRs in the control group was 52, whereas the brain-damaged subjects had 26 such responses. This difference is highly significant ($U = 77$, $p < .002$).

Location of lesion and indicants of arousal. As the data were being collected, it became apparent that there were essentially two sub-groups within the brain-damaged. Certain

subjects gave many GSRs, both specific and non-specific, while others gave relatively few. There were no immediately obvious reasons for this difference, in that these subjects were not markedly different in age, drug intake, anxiety level, etc. Reference to the neurologist's ratings of these patients provided the clue to a possible determining factor.

Of the brain-damaged Ss, 17 were rated as having "localizable" lesions, and three were rated as "diffuse" (see Table 1). Of the 17 with localized lesions, eight were judged as having involvement of the temporal lobes, either limited to temporal areas, or in combination with other brain regions, e.g., temporal-frontal, temporal-parietal, etc. The remaining nine localized lesion cases did not, in the opinion of the judge, have temporal lobe involvement. They presented evidence of damage to other areas, or combinations of areas, e.g., frontal-parietal, but with the temporal areas excluded.

The basis of the dichotomy in terms of GSR frequency was found in this localization rating. The subjects with temporal lobe involvement were those who consistently had few GSRs. Table 10 gives the result of U test comparisons on the GSR data for subjects with temporal lobe involvement compared with localized cases without temporal lobe damage.

In Appendix G GSR frequencies for these brain-damaged

sub-groups for the experimental conditions are given. Appendix H gives log conductance data for these same subjects.

Table 10

U Test Comparisons of GSR Frequency of Temporal and Non-temporal BD Ss

Comparison	U[a]	p
Initial rest total GSR	9	.02
Startle	18	>.10
Rest after startle	9	.02
Adaptation	11	.02
Rest after adaptation	7	.02
Reaction time total GSR	4	.002
Rest after reaction time	5	.002
Paired associates total GSR	10	.02
Final rest total GSR	21	>.10
Minute 1	13	.03
Minute 10	17	.08
Total GSRs all conditions	9	.02

[a]For 8 x 9 comparisons, the expected U = 36.

It is apparent from inspection of the data that the two groups are not totally distinct. However, the differences between the groups are evidenced by the tests of significance

in Table 10. The groups are significantly different for all conditions except startle and the final rest condition, although the table indicates a significant difference at the beginning of the final rest period.

The distinction within the brain-damaged group on the basis of lesion location was not found for the log conductance measure. U tests between temporal and non-temporal cases failed to reach significance in any instance where applied. Table 11 gives the results of these tests. Distributions of the conductance measures suggested higher arousal levels in the subgroup without temporal lobe damage.

Summary of the Results

The results of this study may be summarized as follows:

1. The brain-damaged and control groups were significantly different in performance on the reaction time and paired associates learning tasks, with the brain-damaged showing poorer performance on both tasks.

2. The brain-damaged group was consistently higher in arousal as indicated by the log conductance measure. The difference between the groups was generally statistically significant during the various rest conditions and during

Table 11

U Test Comparisons of Temporal and Non-temporal BD Subjects on Log Conductance

Comparison	U	p
Initial rest mean level	19	>.10
Minute 1	22	>.10
Minute 15	24	>.10
Startle	21	>.10
Rest after startle	25	>.10
Adaptation	26	>.10
Rest after adaptation	22	>.10
Reaction time	28	>.10
Rest after reaction time	26	>.10
Paired associates	27	>.10
Final rest	25	>.10

passive stimulation, but not during either of the task, or work, conditions.

3. With few exceptions, the brain-damaged and control groups did not show differential changes in log conductance during the experiment. The exceptions were the first half of the adaptation period, the difficult task during the paired associates learning task, and the final rest period.

4. Although the brain-damaged group had generally higher frequencies of GSRs throughout the experiment, the groups were not, for the most part, significantly different. The brain-damaged had significantly more responses during adaptation and toward the end of the rest periods, but not during the work periods.

5. The control group had greater decreases in GSR frequency than the brain-damaged during the adaptation, reaction time, failure trial of paired associates learning, and the final rest period.

6. The correlations between performance on the experimental tasks and the electrodermal measures were generally not statistically significant. The correlations were minimal for both brain-damaged and control groups during the reaction time task, and somewhat higher during the paired associates task. The outstanding feature of the relationships was the tendency for the brain-damaged to have positive relationships between the electrodermal measures and learning performance, while the control group had negative correlations. The difference between groups for the relationship between arousal and performance was significant only for the correlation between mean log conductance for the paired associates period and total paired associates errors.

7. The brain-damaged and control groups were shown to be significantly different in the number of compound GSRs to the startle and adaptation stimuli, with the control group having twice as many of these responses as the brain-damaged.

8. Further analysis of the brain-damaged group data revealed that there were two fairly distinct sub-groups in regard to the frequency of GSRs, defined on the basis of the location of lesion. The patients having temporal lobe involvement showed relatively low frequencies of GSRs under all conditions, while subjects who did not have temporal lobe damage showed higher GSR frequencies. These sub-groups were not significantly different on log conductance, nor in performance on the experimental tasks, but were usually significantly different in the number of GSRs.

CHAPTER V

DISCUSSION

The results of this experiment, based on comparisons of brain-damaged and control groups, indicate rather clear distinctions between the groups in performance,[1] in certain features of arousal, and in the relationships between performance and arousal. The discussion of these results is somewhat speculative for two principal reasons; first, no comparable experiments were found in the literature; and second, current theories of brain function do not lead to generation of inferential statements in regard to the complex relationships between brain disorders, arousal states, and psychological performance. Although the terms arousal and activation have a specific neurophysiological meaning, i.e., EEG activation, these terms are used in the present discussion to refer to an intensity aspect

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[1] A discussion of the findings relating to reaction time and paired associates learning performance will be found in Appendix I.

of behavior, following Duffy (1957) and Malmo (1957), and imply involvement of the whole organism.

The various aspects of the experiment will be discussed in the following order:

1. Methodological considerations, including subject variables and task difficulty and order effects.

2. Proposal of an interpretation of the data in terms of two processes: first, cortical release; and, second, increased activation resulting from attempts to cope with deficits in information processing.

These interpretations are based on the findings of differences between brain-damaged and control groups on indicants of arousal during rest, passive stimulation, and work.

Methodological Considerations

Subjects

The inferences that might be made from the results of this experiment are necessarily related to the degree to which the subject samples can be considered representative of the populations from which they were selected.

Control subjects. The control subjects were drawn principally from the neurology and psychosomatic services. Patients on these services reside on the same ward in this

hospital. Therefore, the general hospital environment is the same for this group of control subjects and the brain-damaged. Further, the neurology patients were under the care of the physicians who treated the brain-damaged cases, thus the diagnostic techniques were similar.

In regard to representativeness, the control patients were not significantly different from a sample of 40 non-brain-damaged subjects, selected from the same wards and using the same rating system, seen in other studies currently in progress in age ($z = .94$, $p > .10$) or education ($z = .87$, $p > .10$) nor were they different in MMPI A-scale scores from a sample of 23 drawn from these other studies ($z = 1.2$, $p > .10$). To this degree the control subjects might be considered representative of patients who come to this VA hospital for psychosomatic and peripheral neural conditions.

Brain-damaged subjects. The present group of subjects is different in one respect from other brain-damaged subjects who have been seen in other current experiments in this laboratory. Two samples of 20 were randomly drawn from a pool of 70 brain-damaged subjects, and in both cases the mean educational level was lower than for the subjects in the present study, and in one of the comparisons the difference was significant by the U test ($U = 128$, $p = .05$).

In regard to other characteristics, the present sample was not different from brain-damaged samples seen in other experiments. By U tests, neither MMPI A-scale scores (z = 1.15, $p > .10$) nor age (z = .32, $p > .10$) were significantly different from a sample of 32 other brain-damaged subjects who have been seen in this laboratory. The degree of impairment of the present group was also not significantly different from the 70 subjects referred to above. This comparison was derived from the ratings of impairment made by the neurologist. The result of a U test on the impairment ratings was not significant (z = .97, $p > .10$).

Considering the types of lesions in the group of brain-damaged in this study, it can be seen from Table 1 that this is a heterogeneous group. However, the proportions compare with the distribution of the group of 70 brain-damaged subjects referred to above, in that in both instances the total group is roughly divided into one-third each of tumor, trauma, and cerebral vascular disease cases.

An important variable in regard to brain damage is that of chronicity, or the extent of time that diagnosed brain disease has been present. Table 1 shows that one-half of the subjects in the present study had been diagnosed for their present illness for three or more years, and only three

cases had been diagnosed for one year or less. The brain-damaged sample in this study would thus be more representative of a chronic than an acute population. However, when the group was sub-divided into those cases diagnosed for more than three years and less than three years, no significant differences were found in any of the measures employed. The results of these tests are given in Appendix J.

A general conclusion in regard to the brain-damaged subjects in the present study is that for the most part, they are not different from the larger population of brain-damaged found in the VA hospital.

Task Difficulty and Order Effects

The present experiment was designed to present the subjects with a series of conditions calling for progressively greater degrees of arousal as a function of stimulus complexity and task demands. In a general way, this expectation was upheld by the results, supporting the proposal of Malmo (1957) regarding the value of physiological measures in studies presuming to manipulate the arousal of a group of subjects.

Group means for the nine experimental conditions are presented in Figure 11 based on individual mean conductance scores for each of the conditions. An analysis of variance (Lindquist, 1953) yielded a significant result for the

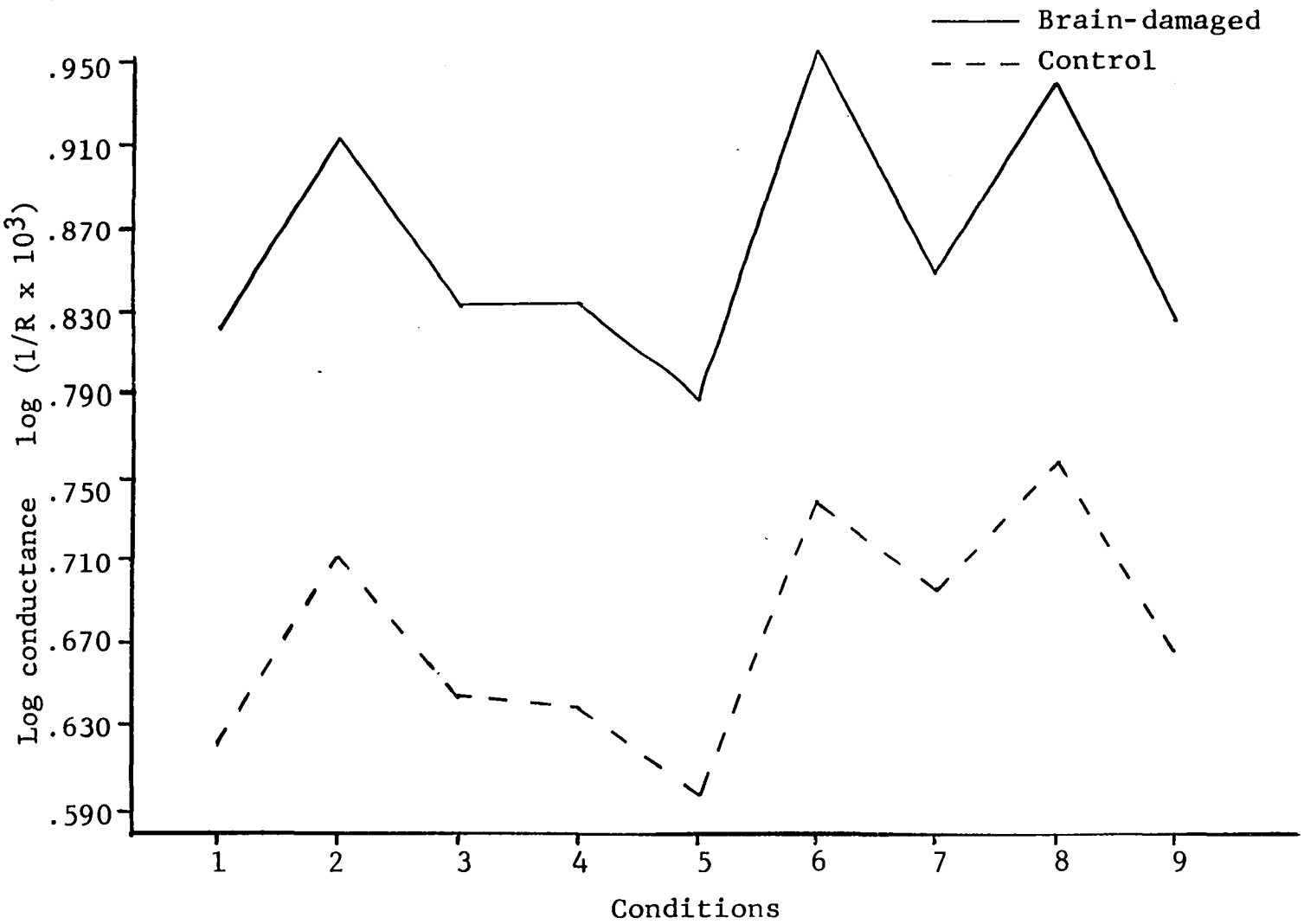

Fig. 11. Mean log conductance in brain-damaged and control groups for nine conditions. (1-initial rest; 2-startle; 3-rest I; 4-adaptation; 5-rest II; 6-reaction time; 7-rest III; 8-paired associates learning; 9-final rest)

Conditions variable ($F = 13.78$, $p < .001$). The analysis is presented in Appendix K. The same general trend was seen for the GSR measures (Figure 12) in that both brain-damaged and control groups had higher frequencies of response as task complexity increased.

Interpretations of the Results

In the earlier chapters, increased electrodermal response as reflected in higher conductance levels and greater GSR frequency was considered to be indicative of higher arousal levels. The general findings of the experiment demonstrate increased arousal or activation in the brain-damaged subjects which is not ascribable to chronic anxiety, since these subjects were not different from the control subjects on the anxiety measure.

By grouping the experimental conditions into rest, passive stimulation, and work periods, the greater portion of the results can be briefly summarized as follows:

1. Rest. Both the conductance measures and GSR frequency were significantly higher in the brain-damaged group, with the control subjects showing greater decreases during any given period.

2. Stimulation. The results were similar to the

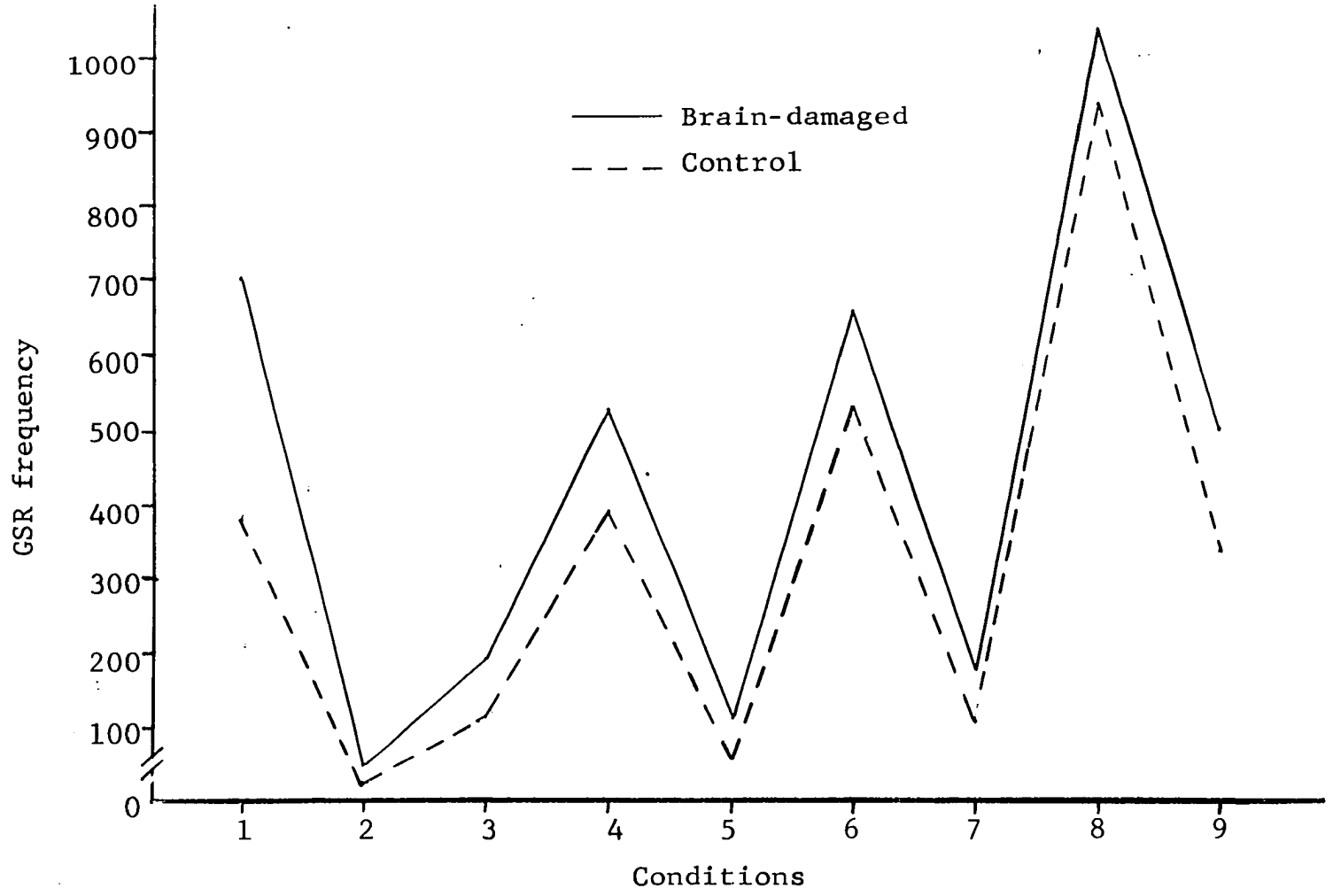

Fig. 12. Total GSR frequencies for brain-damaged and control groups for nine conditions. (1-initial rest; 2-startle; 3-rest I; 4-adaptation; 5-rest II; 6-reaction time; 7-rest III; 8-paired associates learning; 9-final rest)

rest condition, with the exception that there was no difference between the groups in conductance change.

3. Work. Neither of the measures was significantly different between the groups, although the control group tended to show greater decreases in GSR frequency, and the brain-damaged had higher scores on both measures.

In Chapter I the suggestion was made that behavioral deficits in subjects with brain damage might be due to an inability on the part of these subjects to arrive at and maintain arousal levels, as an aspect of motivation, appropriate to the demands of the situation. The implication was that the level of activation in these subjects would be either too high or too low, following a postulated inverted-U relationship between level of activation and performance efficiency (Bindra, 1958; Duffy, 1957; Freeman, 1948; Hebb, 1955; Malmo, 1959). The consistency of the indications of high states of arousal in the brain-damaged compared with the control group, in all the experimental conditions, discounts any possibility of considering the performance deficits as being due to low states of activation. The alternative possibility, i.e., that the activation levels were too high, cannot be either completely discounted or supported, since in one of the experimental tasks positive correlations

were found between performance and arousal in the brain-damaged. The conclusion is that, at least for the brain-damaged subjects in this study, high arousal levels are not necessarily causally related to poor performance in all tasks.

In the following section, interpretations of the finding of higher activation levels in the brain-damaged will be discussed, and tentative proposals made regarding their relationship to performance.

Interpretations of High Activation in the Brain-damaged in Terms of Cortical Release and Attempts to Cope with Deficits

Two possible interpretations of the indications of higher arousal in the brain-damaged are proposed. The first accents neurological or neurophysiological systems, accounting for the higher arousal in the brain-damaged through a cortical release phenomenon. That is, the destruction of higher centers allows subcortical networks to operate independently of the controlling and inhibitory influences of the cortex. Second, an interpretation may be made which puts more emphasis on psychological constructs and adjustmental patterns of the brain-damaged. In this context, activation or arousal is seen as an aspect of the attempt to

adjust to situational demands. The subject, in an attempt to cope with, or compensate for, his poor performance, becomes more motivated or involved. The higher level of motivation is reflected in physiological indices of the intensity aspect of motivation (Duffy, 1957).

Cortical release. Many studies have demonstrated the complexities of interaction between cortical and lower neural mechanisms as well as indicating that the usual role of the cortex in these interactions is to modify or inhibit the activities of the lower mechanisms. In a comprehensive review of work in this area, Ingram (1960) concludes that autonomic systems are, in normal circumstances, subject to influence from higher areas. Ingram further states that when cortical centers are disturbed, the possibility rises that reverberatory circuits will be set up, through the internuncial neurons of the reticular formation. This results in autonomic systems, stimulated through reticular efferents, showing higher than normal rates of activity. Further, with the disturbance of midbrain-cortex relationships resulting from cortical lesions, the hormonal component of autonomic function may also lead to continuation of neural discharge (Ingram, 1960). In the present context, the increased frequency of GSRs and the higher conductance levels found in

the brain-damaged may be due to cortical release.

In considering the concept of cortical release in reference to the results of the present study, two aspects seem likely. First, release involves some structure which ordinarily exercises inhibitory control over orienting responses. Second, autonomic mechanisms are no longer subject to cerebral control and function, by analogy, as unbiased neural networks, firing without external stimulation.

The first aspect maintains some relationship with activation or arousal, to the extent that orienting responses may be considered as indices of activation. Autonomic orienting responses represent an undifferentiated alerting reaction, poorly integrated and non-selective, ordinarily capable of being influenced by higher centers (Berlyne, 1960; Pavlov, 1927). Cerebral lesions serve to make the lower mechanisms independent of central control (Ingram, 1960), leading to high frequencies of response and maintenance of high levels of tonic activity which are not necessarily related to situational demands. The autonomic responses, as orienting reactions to stimuli, indicate that something of potential significance has occurred, and thus the findings may be interpreted in terms of a general kind of arousal or activation system.

The second possibility does not require any relationship between high activity rates and activation or arousal. Cortical release simply frees lower centers from control and inhibition, so that the lower structures fire without external stimulation and show very little adaptation to continued stimuli. By way of analogy, the cortex normally serves to bias lower networks, so that certain incoming stimuli will be gated out as a function of intensity, relevance, etc. (Bruner, 1957). From this analogy, cortical lesions would interfere with proper bias levels so that any event would bring about firing of the lower centers and response at some level.

The greater frequency of GSRs and the higher conductance levels found in the brain-damaged can be accounted for by either of these possibilities. Both measures were consistently higher in the brain-damaged and also showed a general pattern of greater difference between groups with continuation of time within any given condition. The data do not provide a basis for determining the relative contribution of the two possibilities. It is possible that both were operating to produce the obtained results.

A further indication of freedom from cortical control at an autonomic level may be seen in the higher correlations

between GSR frequency and conductance found in the brain-damaged. Table 12 gives these correlations, showing the positive relationship consistently found in the brain-damaged, as well as the generally higher values. While the only significant difference between the correlations was during the reaction time period (z = 1.98, p = .05), the results indicate a consistent difference between the groups. It is of particular importance to note that the correlations found in the control group approximate those reported by Lacey and Lacey (1958) and Stern (1962), indicating some generality of the findings of the present study. No studies were found with which to compare the results of the brain-damaged group.

In addition to the indications of greater activity and the higher correlations between GSR frequency and conductance found in the brain-damaged, certain other findings of the present study point toward cortical release as a factor. For example, GSR latency to the startle stimulus, and also to the first two adaptation trials, was found to be significantly shorter in the brain-damaged. The mean latency to the startle stimulus was 2.31 sec. in the brain-damaged, and in the control group, 2.48 sec. (U = 116, p = .02). The mean latency to the adaptation stimuli was 2.39 sec. in the brain-damaged, and 2.56 sec. for the control group (U = 125,

Table 12

Rank-order Correlations Between GSR Frequency and Conductance in Brain-damaged and Control Groups

Condition	r_s	
	Brain-damaged	Control
Initial rest	.71**	.27
Adaptation	.61**	.23
Reaction time	.41*	-.36
Paired associates	.35	-.12
Final rest	.43*	.13

Note.--Correlations were computed between total GSR frequency and mean log conductance level for the various conditions.

*$p < .05$.

**$p < .01$.

$p = .05$). These findings may be interpreted as indications of decreasing inhibitory influence as a function of cerebral damage, and the inference may be made that, in the brain-damaged, there is a greater readiness to respond, or higher level of excitability, in subcortical centers.

As stated previously, there are no studies available using brain-damaged subjects from which to make comparisons

with the results of the present study. However, if inadequate functioning on tasks is taken as an indicator, schizophrenics form a comparable group. Studies have shown higher correlations in schizophrenic subjects, compared with controls, in body temperature (oral and rectal) (Linder & Carmichael, 1933) and between diastolic and systolic blood pressure (Hoskins & Jellinek, 1933).

The results of the latter studies have been interpreted by Shakow (1963) in a way which seems applicable to the correlations in the present experiment. Shakow refers to the work of Coghill and his theory of individuation and integration and develops a concept of segmentalization which is similar to individuation. In Shakow's thinking, the cerebrospinal system becomes more automatized as life progresses, so that higher centers can be freed for more complex activities. In schizophrenics there is a weakening of the control centers that serve organizing and integrating functions and an accompanying tendency for individuated, segmented, automatized systems to become predominant. The higher correlations in the disturbed subjects mean that certain processes have become independent of higher centers and less amenable to the kind of control necessary for effective adaptation.

Because of the similarities in performance between brain-damaged and schizophrenic subjects, the question arises as to the possibility of applying Shakow's scheme to the findings of the present study. The disturbance of integrating functions, directly through brain lesions or indirectly through interference with cognitive activities in general, allows greater independence of autonomic response systems from cortical influence, allowing them to function in a way not necessarily related to adaptation.

The concept of cortical release has been suggested as accounting for the indications of higher levels of arousal in the brain-damaged. The positive correlations found between the electrodermal measures and learning performance in the brain-damaged group, however, do not seem to be attributable to decreased inhibition. Cortical release may, in effect, add a constant to indices of arousal, and there would be no reason to expect that this "artificial" heightening of activation indices would result in positive relationships with performance.

Granted that cortical release seems to provide an adequate explanation for the majority of the findings, the lack of ability of the concept to account for the correlations

discussed above suggests the consideration of other factors.

Increased activation in the brain-damaged as a result of attempts to cope with performance deficits. A second general interpretation of the findings of the present study is related to the deficits in performance of subjects with brain lesions. The suggestion is made that heightened levels of arousal are a function of attempts to cope with situational demands in the face of recognized inadequacies.

The proposal requires assuming that the brain-damaged were a random sample from a normally distributed population in regard to emotional disturbance and also that their reaction to situational demands was basically adaptive. The latter implies effective means of coping with environmental conditions. The disturbance at the time of their hospitalization, shown by the MMPI profile elevation (Appendix D), is considered to be largely a reaction to the brain lesion itself, as opposed to signifying long-term psychological abnormality. The control group, on the other hand, had no profound organismic threat in the etiology of their psychological disturbance. In fact, the nature of the illnesses of certain of the subjects (the psychosomatic patients) suggests chronic maladaptive patterns of response to stressful situational demands.

Common experience and experimental evidence testify to the general effect of increasing effort in improving performance (Duffy, 1957). The brain-damaged subjects no doubt had had opportunity to learn this relationship. In the subject with brain pathology, the inadequacies in his performance are obvious, and the subject himself may compare his present capacity with the past. It is suggested that the subjects with brain damage, at least those who were capable of performing the tasks of the present experiment, recognize their own deficits, although they may not be able to verbalize this recognition. Such a subject, having an expectation that if he tries harder his performance will be more adequate, becomes more involved or more highly motivated. One result of this effort to cope with deficits is a higher level of activation, generalized to all situations, reflected in higher conductance levels and GSR frequencies, even at rest and during conditions where no overt response is required. The positive correlations found between learning performance and arousal indices indicate that the attempts of the brain-damaged to cope with situational demands by increased effort (and correspondingly higher activation) met with some success.

A more detailed approach to interpretation of performance deficits and arousal levels in brain-damaged subjects

may be formulated in terms of information processing. Of the three main aspects involved in processing of information; input, output, and central integrative and evaluative processes, it is the last on which the present discussion is focused.

The deficits in the integrative processes in the brain-damaged, indicated by their well-established perceptual and cognitive deficits, may be interpreted in terms of a system which describes perception (and cognitive activities in general) as involving a process of categorization or assigning cues to appropriate classes (Bruner, 1957; Bruner, Goodnow, & Austin, 1956). Brain damage may result in the disruption of the categorization process, so that the subject is no longer able to rapidly and appropriately classify events, nor is he able to easily make decisions (not necessarily conscious) as to which features of the environment should be attended to and which disregarded. As an outcome of these disabilities, all stimuli must be responded to as potentially important. These responses, not necessarily related to the externally defined meaning of the stimulus, are reflected in autonomic changes, the GSR. At the same time, the inability to categorize stimuli may result in relative deficits in the psychological elaboration of the response,

indicated by fewer compound GSR patterns in the brain-damaged.

A recent proposal by Deutsch and Deutsch (1963) directly involves activation levels in the processing of information. Deutsch and Deutsch cite evidence that the number and importance of stimuli which bring about responses are directly related to arousal, in that in sleep only the most significant stimuli (loud sounds, the subject's name_ will result in a response. When the subject is drowsy, more stimuli will be responded to, but some will still be missed. In general, as arousal increases up to some optimal point (Lindsley, 1956), more stimuli have a probability of being responded to.

The efforts of an individual with a brain lesion, who is attempting to function in his environment adequately, may be directed toward missing as few stimuli as possible. Responses, including GSRs, should occur indiscriminately and frequently, even in situations where, by external criteria, continued response is inappropriate.

From these admittedly speculative proposals, a systematic way is possible to understand both the higher conductance levels (as an index of general arousal) and the higher GSR frequency (interpreted as indiscriminate responses) in

the brain-damaged group. The higher ongoing activation levels increase the probability of detection of possible information-giving stimuli, and the greater number of GSRs signify undifferentiated assignment of signal value to all stimuli.

The foregoing proposal, emphasizing efforts directed toward coping with deficits in information processing, seems capable of accounting not only for the indications of higher arousal, but also the positive correlations between performance and arousal in the brain-damaged. However, the lack of group differences on arousal indices during work, is contrary to what would be expected. If attempts at coping with performance deficits are responsible for the higher activation levels in the brain-damaged, even greater differences would be expected during work, since it is toward these situations that coping efforts should be directed. A suggestion as to a possible solution to this apparent contradiction is seen in the negative correlations between performance and arousal indices in the control group. Recalling that these subjects had a composite MMPI profile suggesting at least mild states of psychological disturbance, it would not seem unreasonable to state that this group was actually too highly aroused. The lack of difference between the groups, then, is not due to inadequate levels of activation in the brain-damaged, but

instead to abnormally high levels in the control group.

Suggestions for Further Research

It will be recalled that this study was designed as an exploratory project in an area where there are few theoretical and empirical guidelines. It was expected that from the findings of the experiment and the interpretations of those findings, issues would emerge which would require exploration before there could be a thorough understanding of activation processes in the brain-damaged. That such an expectation was realized is indicated in the following suggestions for further investigations.

However, before discussing future experimentation, an additional methodological note should be considered. The interpretations of the data have been founded on the assumption that sufficient experimental controls were exercised. However, it is possible that the brain-damaged subjects had disturbances in skin temperature controlling mechanisms, circulatory changes, etc., which could have influenced the electrodermal measures. Evidence has recently been presented of the effects of such variables on skin resistance measures (Edelberg, 1963). Although there is a possibility that central nervous system damage could have affected the mechanisms

responsible for skin temperature, etc., there was no reason, prior to the experiment, to expect that specific local changes would occur. A necessary control in future experimentation would involve taking into consideration those factors shown by Edelberg to be capable of influencing the measures.

Comparisons of brain-damaged subjects and other impaired groups and explication of cortical release effects. The interpretation of certain of the results of the experiment in terms of cortical release was compared to an interpretation of Shakow of similar findings in schizophrenics. An obvious experiment would involve direct comparisons of schizophrenics, and others with impaired psychological functions (extremely disturbed neurotics, retardates), with brain-damaged subjects on a series of stimulus conditions and tasks with concurrent measurement of several psychophysiological variables. It would be of interest to show that deficits in psychological performance and changes in physiological function were comparable in the various groups.

The intercorrelation of several variables (e.g., skin resistance, heart rate, blood pressure, skin temperature) could lead to more definitive statements in regard to the role of cortical release and segmentalization in producing high frequencies of activity in subcortical mechanisms.

Further evidence relating to cortical release would come from comparisons of responses in which the inhibiting role of the cortex varies. That is, reflexes, which may (or can) be influenced by cortical processes, might be more rapid in the subject with a brain lesion. The response time of a segmental reflex, in other words, might be less affected by cerebral damage than a suprasegmental one, since in the latter there would be greater likelihood of cortical influence.

Elaboration of arousal in relation to adjustmental patterns. Emphasis was placed on attempts by brain-damaged subjects to cope with deficits in one of the interpretations of the findings. Support for this proposal was drawn from the finding of positive correlations between learning performance and activation in these subjects. The question arises as to the relationships between adjustmental patterns, performance, and arousal. By selecting subjects who varied along a continuum of adjustment, particularly in their premorbid patterns, this assumption would be open to test. For example, measures such as the Worcester Scale for Social Attainment (Phillips & Cowitz, 1953) would provide means for estimating pre-morbid adjustment patterns.

Further studies of relationships between performance and arousal in the brain-damaged. The finding of positive

correlations between learning performance and arousal indices in the brain-damaged subjects in the present study leads to a question as to the generality of this finding. Whether this result was related to the relatively high educational level of the group could be determined from a study with larger numbers of subjects and a broader range of educational levels.

It is also possible that this relationship would tend to hold in verbal but not in performance tasks, since studies generally show greater deficits in the brain-damaged on performance measures (Meyer, 1957). That is, since deficits are more obvious on perceptual-motor tasks, they might present a greater threat to the brain-damaged subject, leading to over-mobilization, and negative relationships between performance and arousal.

Autonomic lability, information processing deficits, and inappropriate responses. Lacey and Lacey (1958) postulate and support a hypothesis concerned with relationships between autonomic lability and motor "impulsivity." Subjects were defined as labile on the basis of high rates of spontaneous activity on autonomic variables. The results of the present study would classify many of the brain-damaged subjects as labile, and the prediction would be made, from

Lacey's position, that they would make more "impulsive" errors in overt responses than the subjects showing lower rates of spontaneous activity. Using his experimental method, inferences could be made from the results concerning the degree to which brain-damaged subjects respond to deficits by indiscriminate or inappropriate response.

Comparisons of central and peripheral indices of activation in the brain-damaged. Since arousal and activation are usually defined in terms of EEG changes, it seems appropriate to make a comparison between autonomic measures which have been interpreted as indices of arousal and more direct indications. Concurrent recording of EEG and autonomic (e.g., electrodermal) indices of arousal in brain-damaged subjects would resolve the apparent conflict between the findings of Li et al. and Grossman (little EEG activation with stimulation) and those of the present study. Also, a study of this kind would provide information relevant to the cortical release hypothesis, since, if high rates of autonomic activity were present while cortical arousal was low, support for cortical release would be obtained.

Central measures might also explicate the nature of the deficits in the brain-damaged. For example, relationships between cortical rhythms and reaction time have been shown in

the previously cited study of Dustman et al., demonstrating faster reaction times with higher frequencies. Lansing (1957) showed that reaction time was also related to alpha phase, implying relationships between cortical excitability cycles and performance. If these results were to hold in brain-damaged subjects, combined with measurement of EEG response latency and cortical evoked potentials, understanding of deficits in the brain-damaged in relation to information processing might be furthered.

The final suggestion for future research develops from the finding of the study which has yet to be considered. Neither of the interpretations of the results seems able to account for the lower GSR frequencies in subjects with temporal lobe involvement compared with those without temporal lobe damage. It is suggested that the observed effects in these subjects are due to direct neurological involvement as opposed to disturbances in arousal mechanisms.

Temporal lobe structures have manifold interconnections with areas (e.g., rhinencephalic structures) which, either directly or indirectly through other connections, are important in autonomic functions (Ingram, 1960). There is also some evidence that there are more or less direct connections between temporal areas and the reticular system, since

stimulation in the temporal lobe can result in behavioral arrest (cessation of ongoing behaviors, Lilly, 1958). The low frequency of GSRs in the subjects with temporal lobe damage may be due to at least two effects. First there may be direct interference with centers or pathways necessary for the propagation or passage of impulses which result in the GSR. Second, cortical release of inhibitory systems may have occurred. The data of the present experiment do not lead to a choice between these alternatives.

The findings of low GSR frequencies of patients with temporal lobe involvement compared with non-temporal damage could be explicated by using subjects upon whom brain surgery had been performed. The data from the operation should provide needed information regarding structures involved and perhaps lead to more definitive statements concerning the contribution of temporal areas to autonomic function. The low GSR frequency in these patients also suggests comparing sensory threshold measurements in a group with temporal lobe damage with a brain-damaged group without temporal damage. If the GSR may be considered to be an indication of information coming through lower centers, the low frequency in subjects with temporal lobe damage might indicate deficits in the input stage of an information processing model. It would

also be of interest to record several measures of autonomic function in these subjects in order to ascertain if similar effects (i.e., decreased activity) would be found.

Summary

In this chapter, methodological issues were discussed, and the interpretation was made that, for the most part, the subjects in the present study were representative of patient groups seen in this laboratory in other experiments. Also it was seen that the expectation that the order, stimulus conditions, and tasks representing progressively greater degrees of complexity was upheld by the results.

Two proposals with particular emphasis on the brain-damaged group were considered as interpretations of the experimental results, one being cortical release and the other emphasizing increased activation as a result of attempts to cope with deficits in information processing. Cortical release was shown to be capable of accounting for the major portion of the findings, particularly since shorter GSR latencies were found in the brain-damaged. High activation levels in relation to attempts to cope with performance deficits and related to an information-processing model were also considered to be applicable to the findings, especially

in regard to positive correlations between learning and arousal in the brain-damaged.

From these findings and interpretations, suggestions for future research were made.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Numerous studies have demonstrated deficits in performance in brain-damaged subjects at all levels of function. Two approaches, related to general theories of brain function, have been considered as possible explanations for these findings. The first approach described impairment as due to the destruction of areas which are specific for a given function based on direct relationships between structure and function. The second brings into focus more general variables, in particular motivation, which seem more adequate to account for the overall reduced efficiency of performance in individuals with brain injury. Studies, especially Goldstein (1944) and Wolff et al. (1958), show that the brain-injured individual's whole pattern of interpretation of stimuli and adjustment to his environment is impaired. After considering these findings, the suggestion was made that the disturbances in performance in brain-damaged subjects may be due to

inability to arrive at and maintain levels of arousal which are appropriate to the demands of the situation.

Arousal phenomena in the brain-damaged, and their relationship with performance, have not been previously intensively investigated. The available evidence (from EEG studies and behavioral observation) lead to opposite expectations regarding the state of arousal in the brain-damaged. One line of evidence leads to the conclusion that the brain-damaged individual is highly aroused, while the other suggests low states of activation. The present study was designed to provide experimental evidence regarding the nature of the arousal process in subjects with brain damage.

For the purpose of the study, arousal was defined in terms of two electrodermal measures, conductance level and GSR frequency, which have previously been shown to have value in studies concerned with arousal. The stimuli, tasks, and order of presentation were designed to provide a series of conditions which would place progressively greater demands on the adjustive capacities of the organism.

Since relevant experimental data and theory are not sufficient to generate hypothetical statements, a series of empirical questions was asked concerning comparisons of brain-damaged and control groups on the arousal measures

during the various conditions. The experimental conditions were grouped into three classes: rest, passive stimulation, and work; and the questions were concerned with the effect of these conditions on states of arousal in the two groups.

The subjects for the study were 40 patients from the VA and University Hospitals, Oklahoma City, 20 of whom were defined by a neurologist as definitely brain-damaged and the remaining 20 defined as definitely not brain-damaged. The mean age of the two groups was approximately equal, 41.5 years, and the two groups were not significantly different on the MMPI A scale. However, it was found that the brain-damaged had a significantly higher mean level of education, 11.8 years compared with 10.5 for the control group. The brain-damaged group was composed of approximately one-third each of tumor, trauma, and cerebral vascular disease cases, and the majority of the subjects would be regarded as chronic cases. The control subjects were from the Neurology and Psychosomatic Services and had primarily spinal cord and peripheral neural damage and gastrointestinal disorders.

The electrodermal measures were recorded concurrently on two independently calibrated channels of a polygraph. The electrodes used were found to minimize artifact. The purpose of the design was to present the subject with a

series of conditions of increasingly greater complexity, i.e., startle, adaptation, choice reaction time, and paired associates learning. The reaction time and learning tasks were chosen because previous studies have shown brain-damaged subjects to be impaired on these tasks.

The order of the experimental conditions was as follows: rest, startle, adaptation, reaction time, paired associates learning, and rest. Readings were made of conductance levels, and GSR frequencies were obtained, for each minute of the experiment. A GSR was defined as a response which exceeded 1.5% of the resistance level at the time the response occurred.

The results of the study can be summarized as follows:

1. Despite the higher educational level of the brain-damaged they were significantly poorer in performance on both the reaction time and paired associates learning tasks.

2. The brain-damaged group was consistently higher in arousal as indicated by log conductance. The difference between the groups was generally significant during the various rest conditions and during passive stimulation but not during either of the work conditions.

3. With few exceptions, the brain-damaged and control groups were not significantly different in changes in log conductance during the experiment.

4. Although the brain-damaged had generally higher GSR frequencies throughout the experiment, the groups were not, for the most part, significantly different. An exception was seen during passive stimulation, where the brain-damaged gave significantly more responses.

5. The control group had greater decreases in GSR frequency during most of the experimental conditions.

6. The correlations between the arousal measures and performance, while not generally statistically significant, tended to be different in the two groups, especially during the learning task. The brain-damaged, during this task, had positive correlations between the electrodermal measures and performance, while the correlations in the control group were negative.

7. The brain-damaged and control groups were found to be significantly different in the number of compound GSRs during the passive stimulation condition, with the control subjects having the greater frequency of these responses.

8. The brain-damaged were found to have significantly shorter GSR latencies to the startle and adaptation

stimuli.

9. The brain-damaged had consistently higher correlations between conductance and GSR frequency than did the controls.

10. In terms of GSR frequency in the brain-damaged, a distinction was found on the basis of lesion location. Subjects with temporal lobe involvement consistently had relatively low frequencies of GSRs under all conditions when compared with brain-damaged subjects without temporal lobe involvement. These sub-groups were not found to be significantly different in conductance levels.

Two proposals were tentatively considered as being capable of accounting for the results. The first involves cortical release, with damage to higher centers resulting in decreasing cortical influence over lower mechanisms. Cortical release was considered to be capable of accounting for the findings through two effects: decreased cortical inhibitory influence over orienting responses, and reverberatory circuits resulting from lessened cortical control. The consistent positive correlations between the electrodermal measures found in the brain-damaged were interpreted in terms of a system developed by Shakow to account for similar findings in schizophrenics.

The second proposal brought into consideration the adjustive patterns of the brain-damaged. It was suggested that these subjects, attempting to cope with deficits, become more highly motivated and involved. As a result of this higher involvement, higher levels of activation are seen. The high activation levels in relation to attempts to cope with performance deficits were also considered in more detail in terms of an information processing model. It was suggested that the coping attempts are directed toward minimizing information processing inadequacies.

From the findings and interpretations, a number of proposals for future research were made. They included:

1. Attempts to explicate cortical release effects through intercorrelation of several autonomic variables, studies of reflex functions in which cortical control varies, and comparisons of brain-damaged groups with others in which performance deficits are found.

2. Elaboration of arousal and adjustmental patterns by studying psychological processes in the brain-damaged, focusing on pre-morbid patterns of adjustment.

3. Further studies of relationships between arousal and performance in the brain-damaged, with the suggestion that differences might be found which would be related to the

nature of the task, e.g., perceptual-motor as opposed to the verbal task used in this study.

4. Extension of the concept of autonomic lability to studies of brain-damaged subjects, expecting that such subjects would show greater motor impulsivity and tendencies to make inappropriate responses.

5. Comparisons of central (EEG) and autonomic indices of arousal in the brain-damaged in response to standard stimuli.

6. Further studies concerned with the effects of temporal lobe involvement, using subjects on whom reports of brain operations would be available.

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APPENDIX A

GSR ADAPTATION TO VISUAL STIMULI: AN EXPLORATORY STUDY

AROUSAL STATES, RESPONSES TO FLICKER, AND BRAIN DAMAGE: AN EXPLORATORY STUDY

Purpose. This experiment was designed to investigate relationships between flicker thresholds, adaptation to a flickering light of low frequency, arousal states, and brain damage.

Silverman, Cohen, and Shmavonian (1959) report experiments in which low perceptual thresholds were associated with low arousal states. The question arose as to whether the known deficits in perceptual performance in subjects with brain damage might be ascribed to low arousal states, in addition to other factors, e.g., interruption of visual pathways.

The adaptation of GSR to a flickering light of low frequency might also indicate the tendency of brain-damaged subjects to maintain levels of arousal under continued stimulation.

Method. The subjects were patients from the VA Hospital, Oklahoma City. Both groups, brain-damaged and control,

were composed of ten subjects each.

Flicker thresholds were determined by the method described by Parsons and Huse (1958). Thresholds were determined for nasal and temporal points, on the horizontal, at the 30° position. The low-frequency light stimulus was the same source used in determining thresholds, set to flicker at ten cycles per second.

The GSR apparatus was a Wheatstone bridge with a DC amplifier and an Esterline-Angus Recorder calibrated in conductance units. Zinc electrodes described by Lykken (1959) were used.

Conductance measures were taken for the following conditions: initial level, change in level during rest, change in level to instructions, amplitude of GSRs during stimulation, and change in level during stimulation. The number of GSRs which occurred during the presentation of the stimuli were counted.

All subjects were given an initial ten-minute rest period followed by instructions to look at the light and then were given ten trials of five seconds each of low-frequency stimulation.

Results. The two groups were not significantly different in initial and resting levels of conductance.

Correlations between conductance level and flicker thresholds were not significant for either group (Controls: r = -.20; Brain-damaged: r = .30). It can be concluded that under the conditions of the experiment low states of arousal are not necessarily linked to poor perceptual performance.

The two groups differed significantly in their response to instructions (U test, $p < .05$), the number of GSRs during stimulation (U test, $p < .01$), and change in level during stimulation (U test, $p < .05$), with the brain-damaged being consistently higher.

The adaptation results are as follows:

GSR Frequencies by Adaptation Trials

Trials	1	2	3	4	5	6	7	8	9	10
Controls	7	6	2	5	4	4	2	1	3	0
Brain-damaged	14	12	9	5	11	9	9	7	5	4

The results indicate that the curve of adaptation in the two groups is similar but that the brain-damaged subjects remain more responsive through all trials.

Conclusions. This study demonstrates that differences in performance on perceptual tasks between brain-damaged and control subjects cannot be ascribed to differences in arousal as measured by skin conductance levels. The

relatively greater responsivity of the brain-damaged subjects to stimulation, whether verbal or visual, and their relative lack of adaptation to continued stimulation, indicates a greater response, perhaps emotionally toned, to demands placed upon them from external sources.

APPENDIX B

NEUROLOGICAL RATING SCALES

Brain Damage Scale

Please rate the patient in question on the following scale, relative to your judgment concerning the presence of brain damage.

_____1. Definitely indicated, no other evidence needed.

_____2. Strongly suspected, would like at least one more positive sign.

_____3. Suspected, but much more evidence needed.

_____4. Not likely, but cannot be ruled out at this point.

_____5. Definitely not indicated, no further evidence needed.

Cerebral Neural Involvement Scale

The following scale is designed to give an estimate of magnitude of cerebral neural deficit. Extent of deficit in terms of area of dysfunctioning neural tissue and degree of dysfunction of that area are components of "cerebral neural involvement."

Ratings on the scale are accomplished in two steps. In step 1 ratings of impairment of function and severity of symptoms are made. In step 2 considering all evidence an overall rating of degree of cerebral neural involvement is given.

I. Ratings of impairment of function:

Please use the following five point scale:
1-none; 2-mild; 3-moderate; 4-severe; 5-very severe

___A. Sensory disturbance due to cerebral involvement.
___B. Motor disturbance due to cerebral involvement.
___C. Speech disturbance due to cerebral involvement.
___D. Intellectual impairment due to cerebral involvement.
___E. Emotional disturbance and/or personality change due to cerebral involvement.
___F. Seizure severity.
___G. Seizure frequency (rate from 1=none to 5=very frequent).

II. Considering the above and evidence from sources listed below please give an overall rating: H. ___

Neurological Localization Scale

I. Surgical Lesion ____ Non-Surgical Lesion ____

II. Severe ____ Moderate ____ Mild ____

III. Diffuse ____ Focal ____ Focal and Diffuse ____
Bilaterally Focal ____

IV. Right ____: Frontal ____ Temporal ____
Parietal ___ Occipital ___

V. Left ____: Frontal ____ Temporal ____
Parietal ___ Occipital ___

VI. Static ____ Slowly prog. ____ Moderately prog. ____
Rapidly prog. ____

VII. Cerebral Vascular Disease:_____
Hemorrhage _____ versus Insufficiency ____

Arteriosclerotic _____ A. V. Malformation _____
A. V. Malformation ____ Hypertensive _____
Aneurysm _____ Encephalopathy _____
No source found _____ Arteriosclerosis _____

VIII. Tumor:_____
Intrinsic _____ versus Extrinsic _____

Glioma ____ versus Metastatic _____
Fast growing ____ Lung _____
Slow growing ____ Not lung ____

Meningioma ____
Craniopharyngioma ____
Pituitary adenoma ____
Acoustic neurinoma ____

IX. Inflammatory or Infectious Disease: _____

Encephalitis _____ Syphilis _____
Meningitis _____ Gumuna _____
Abscess _____ Tuberculoma _____

X. Degenerative or Demyelinating Disease: _____

Multiple sclerosis _____ Anemia _____
Alzheimer's disease ____ Metabolic disease ___
Pick's disease _____ Cerebral atrophy ____

XI. Trauma: _____

Birth trauma _____
Penetrating head injury _____
Closed head injury _____

APPENDIX C

DOSAGES OF BRAIN-DAMAGED SUBJECTS RECEIVING ANTI-CONVULSANT MEDICATION

Dosages of Brain-damaged Subjects Receiving Anti-convulsant Medication

S#	Dilantin	Phenobarbitol
1	240	150
2	150	60
3	300	150
4	150	0
5	200	60
6	300	200
7	150	90
8	100	50
13	300	150
14	250	100
18	300	50

Note.--Dosages are given in total milligrams per day.

APPENDIX D

MEAN MMPI T-SCORES FOR BRAIN-DAMAGED AND CONTROL GROUPS

Mean MMPI T-Scores in Brain-damaged and Control Groups

Scales	Brain-damaged	Control
L	52	50
F	60	54
K	54	51
Hs	72	72
D	77	71
Hy	70	69
Pd	70	64
Mf	55	55
Pa	60	53
Pt	66	66
Sc	69	62
Ma	61	57
Si	54	52
A	54	52
R	57	53

APPENDIX E

U TESTS OF DIFFERENCES BETWEEN DRUG VS. NO-DRUG BRAIN-DAMAGED GROUPS ON ELECTRODERMAL MEASURES

U Test Comparisons of Log Conductance of Brain-damaged Subjects Receiving Drugs vs. Subjects not Receiving Drugs

Condition	U	p
Initial rest	41	> .10
Startle	43	> .10
Adaptation	48	> .10
Reaction time	40	> .10
Paired associates	44	> .10
Final rest	45	> .10

U Test Comparisons of GSR Frequency of Brain-damaged Subjects Receiving Drugs vs. Subjects not Receiving Drugs

Condition	U[a]	p
Initial rest	40	> .10
Adaptation	38	> .10
Reaction time	39	> .10
Paired associates	40	> .10
Final rest	41	> .10

[a]For 11 x 9 comparisons, the expected U = 44.

APPENDIX F

TESTS OF HOMOGENEITY OF VARIANCE

Bartlett's Tests[a] for Homogeneity of Variance on Log Conductance Data

Condition	Chi-square	df	p
Initial	62.87	39	.05
Startle	89.05	39	.01
Adaptation	94.70	39	.01
Reaction time	85.67	39	.01
Paired associates	106.43	39	.01
Final rest	73.29	39	.01

[a]Edwards, 1950.

APPENDIX G

GSR FREQUENCY DATA

BRAIN-DAMAGED GROUP GSR FREQUENCIES: INITIAL REST

	Minutes														
S#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	5	6	5	6	3	5	12	6	7	6	4	4	4	3	6
2	6	1	4	2	4	4	7	8	5	9	8	6	6	6	11
3	0	2	0	0	0	0	0	1	1	0	1	1	0	0	2
4	0	1	0	1	0	0	1	0	0	0	0	0	0	2	2
5	3	1	3	1	2	1	3	2	1	3	3	1	0	1	2
6	0	0	0	0	1	0	1	1	0	0	1	1	0	0	1
7	7	7	5	6	3	5	6	10	13	7	3	6	4	5	2
8	0	0	1	0	1	1	1	1	0	1	0	1	1	0	1
9	2	0	2	2	0	2	3	3	1	4	0	1	0	2	0
10	3	1	3	0	1	1	4	1	4	3	4	2	1	4	3
11	2	6	2	2	3	1	6	5	7	5	11	3	8	6	4
12	1	0	0	0	0	1	0	0	0	0	0	0	0	0	0
13	0	1	0	0	0	0	1	0	0	2	1	1	0	0	0
14	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15	3	0	2	2	2	2	1	0	0	3	5	3	4	3	3
16	6	9	6	5	8	6	8	7	4	6	2	5	7	7	13
17	2	4	3	3	4	2	4	3	4	2	4	2	2	2	2
18	7	10	6	7	11	6	6	2	7	2	7	6	3	2	7
19	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20	1	1	2	1	1	0	1	0	3	1	0	0	0	0	0

CONTROL GROUP GSR FREQUENCIES: INITIAL REST

	Minutes														
S#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
41	1	0	3	6	0	3	3	0	1	0	0	1	0	0	0
42	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
43	1	1	0	1	2	1	0	3	1	2	2	5	0	0	7
44	2	3	2	4	4	1	0	1	3	3	2	2	0	0	0
45	4	0	1	0	0	0	1	2	0	1	1	0	0	1	1
46	3	2	3	1	3	1	3	4	5	6	2	3	4	5	4
47	4	5	0	4	1	4	1	6	1	0	5	0	0	1	3
48	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
49	2	1	0	0	0	0	0	0	0	0	0	0	0	0	0
50	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
51	6	9	5	9	11	6	6	6	7	12	5	3	7	2	5
52	0	3	1	0	0	0	0	0	3	3	2	0	0	0	1
53	2	2	4	2	1	1	2	5	1	1	2	1	2	2	0
54	0	0	2	1	1	0	0	0	0	0	0	0	0	0	0
55	0	0	1	2	0	3	2	3	5	5	2	3	2	0	4
56	0	0	0	0	0	0	0	0	0	1	0	1	1	0	1
57	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0
58	1	0	0	1	0	0	1	0	0	0	1	1	1	0	2
59	1	1	1	0	0	2	0	0	0	2	4	0	0	0	1
60	0	4	2	1	0	1	2	1	1	0	0	1	2	1	1

BRAIN-DAMAGED GROUP GSR FREQUENCIES: STARTLE, REST I, ADAPTATION, AND REST II

		Minutes												
	Startle	Rest I			Adaptation							Rest II		
S#		1	2	3	1	2	3	4	5	6	7	1	2	3
1	1	15	5	7	10	14	8	12	3	4	8	1	1	4
2	1	7	10	5	6	7	5	6	8	8	14	2	5	3
3	1	0	0	1	2	1	0	0	0	1	0	3	0	0
4	2	2	2	2	2	1	0	0	1	1	1	0	1	0
5	1	1	8	1	4	2	6	3	5	4	6	3	1	1
6	2	0	0	0	2	1	2	2	2	2	3	0	0	0
7	3	8	4	9	7	10	6	6	6	4	8	4	3	8
8	1	2	1	2	2	4	1	0	2	1	3	1	0	2
9	3	4	2	4	5	6	6	7	7	7	11	3	1	1
10	1	5	0	1	3	4	3	2	3	3	5	4	2	2
11	1	3	1	1	4	3	2	3	1	3	5	2	4	0
12	1	0	0	0	0	0	0	0	1	0	0	0	0	0
13	1	0	0	0	1	0	1	0	0	0	0	0	1	1
14	0	0	2	0	2	0	0	0	0	0	0	0	0	0
15	1	1	2	1	2	3	3	6	2	4	6	7	2	0
16	3	11	6	2	5	4	4	8	5	7	10	4	3	3
17	1	6	4	4	4	3	3	3	2	4	2	0	1	2
18	3	12	4	6	15	9	11	10	11	9	11	11	3	3
19	1	1	1	0	2	2	2	3	0	1	1	0	1	0
20	1	1	1	2	2	2	4	1	0	0	1	0	0	0

CONTROL GROUP GSR FREQUENCIES: STARTLE, REST I, ADAPTATION, AND REST II

		Minutes												
	Startle	Rest I			Adaptation							Rest II		
S#		1	2	3	1	2	3	4	5	6	7	1	2	3
41	1	0	0	2	2	2	2	1	2	2	2	0	0	0
42	1	0	0	0	2	2	2	3	2	2	4	0	0	0
43	1	4	1	4	4	1	5	2	2	1	4	2	2	2
44	1	3	2	1	2	3	1	1	1	0	1	0	0	0
45	1	2	1	0	4	4	3	4	4	4	2	1	1	1
46	1	4	4	3	6	5	4	5	4	3	4	3	1	5
47	2	4	6	6	5	4	5	2	4	2	3	4	3	0
48	1	0	0	1	2	2	0	0	0	0	0	0	0	0
49	1	2	2	1	4	2	2	3	1	2	1	0	0	0
50	1	1	1	1	4	4	3	5	3	2	6	1	1	0
51	1	14	7	11	11	13	10	6	8	4	8	5	7	10
52	1	2	2	2	3	2	4	2	2	1	1	1	2	0
53	1	8	5	3	6	5	2	6	3	2	2	2	2	2
54	1	2	0	0	2	2	2	2	1	0	2	0	0	0
55	1	3	1	0	3	3	5	4	2	5	5	2	2	5
56	1	2	2	0	3	2	3	1	2	3	3	0	0	1
57	1	0	0	0	1	1	2	1	1	0	3	1	0	0
58	1	0	0	1	0	0	1	0	1	1	1	0	0	0
59	1	2	1	0	2	4	5	1	0	0	0	0	2	1
60	1	2	3	0	3	3	4	2	2	3	1	2	1	1

BRAIN-DAMAGED GROUP GSR FREQUENCIES: REACTION TIME AND REST III

	Minutes									
	Reaction Time							Rest III		
S#	1	2	3	4	5	6	7	1	2	3
1	4	4	4	4	4	5	5	7	5	6
2	4	4	4	4	4	5	5	7	7	4
3	1	1	0	1	0	0	0	2	1	1
4	3	4	3	4	3	4	2	3	5	7
5	4	4	4	4	3	3	1	1	0	0
6	4	3	1	1	2	1	2	0	0	0
7	4	4	2	1	1	1	0	4	5	10
8	3	4	4	2	1	3	3	3	1	2
9	3	0	2	1	1	1	1	2	1	0
10	3	1	1	1	2	0	2	2	1	1
11	2	3	3	3	2	0	0	4	2	1
12	1	0	0	0	0	1	1	0	0	0
13	1	0	1	0	1	0	0	1	0	2
14	0	0	1	1	2	0	2	0	0	0
15	3	4	4	4	4	4	5	7	6	2
16	4	4	3	4	3	3	4	7	11	6
17	2	2	2	2	0	1	1	1	4	2
18	4	4	4	4	4	5	5	5	4	2
19	4	4	4	4	3	3	4	0	1	2
20	0	0	1	1	1	0	0	0	1	0

CONTROL GROUP GSR FREQUENCIES: REACTION TIME AND REST III

	Minutes									
	Reaction Time							Rest III		
S#	1	2	3	4	5	6	7	1	2	3
41	4	4	4	4	4	5	5	2	4	2
42	1	2	2	2	0	0	0	0	0	0
43	2	1	0	3	0	2	2	4	3	3
44	4	4	4	4	4	4	5	1	2	9
45	1	0	1	0	0	1	2	1	1	1
46	2	2	3	0	2	1	3	4	3	5
47	2	3	4	4	2	5	5	4	4	1
48	1	0	0	1	0	0	2	0	0	1
49	4	4	4	4	3	5	5	6	2	4
50	4	3	4	4	3	4	4	8	9	4
51	4	3	3	3	3	4	3	9	7	4
52	4	3	2	2	3	4	3	0	0	1
53	4	4	4	4	4	5	5	2	3	4
54	3	3	3	0	0	1	0	3	2	1
55	3	1	2	1	2	1	1	3	1	0
56	1	0	1	0	0	0	0	1	0	0
57	0	0	0	1	0	1	0	1	0	0
58	1	1	0	1	0	0	1	0	0	0
59	3	2	0	0	1	1	1	1	2	0
60	4	2	0	2	3	3	2	3	2	0

BRAIN-DAMAGED GROUP GSR FREQUENCIES: PAIRED ASSOCIATES

	Trials												
	Easy List				Difficult List							Failure	
S#	I[a]	1	2	3	I[a]	1	2	3	4	5	6	I[a]	1
1	20	7	7	7	5	10	9	8	9	6	6	3	10
2	8	7	4	4	1	5	9	11	8	5	6	3	3
3	3	1	6	1	1	2	4	3	5	3	2	1	2
4	6	3	2	6	2	6	5	6	5	4	6	1	5
5	5	5	6	4	1	4	4	7	7	4	3	4	4
6	2	1	2	0	0	2	2	0	0	0	0	1	2
7	8	4	6	4	1	4	7	5	4	3	5	2	7
8	8	1	5	1	3	2	4	6	1	3	3	2	4
9	7	4	5	3	1	3	2	0	4	4	1	2	0
10	8	1	4	6	1	2	3	3	4	2	2	1	1
11	5	6	7	5	1	5	4	5	6	8	6	1	5
12	2	1	1	1	0	2	1	1	1	1	1	2	6
13	5	4	8	4	1	3	2	5	5	3	5	2	4
14	0	0	1	0	0	1	0	1	1	3	4	1	3
15	7	4	5	4	2	3	5	5	7	6	4	4	6
16	8	8	4	5	3	5	5	6	6	3	5	4	5
17	6	4	2	0	1	1	4	2	2	1	1	1	1
18	11	14	10	14	3	10	10	11	7	11	8	3	11
19	15	4	7	4	2	4	4	4	3	5	5	2	8
20	3	3	0	0	0	0	1	0	1	0	2	3	2

[a]Instructions.

CONTROL GROUP GSR FREQUENCIES: PAIRED ASSOCIATES

	Trials												
	Easy List				Difficult List							Failure	
S#	I[a]	1	2	3	I[a]	1	2	3	4	5	6	I[a]	1
41	6	3	4	8	2	6	9	7	4	6	6	2	5
42	3	1	3	0	1	1	2	1	2	2	0	1	1
43	2	0	0	0	1	0	1	1	0	4	1	1	3
44	8	4	4	4	2	6	5	4	3	3	5	2	4
45	3	1	0	0	0	1	2	1	3	1	0	0	0
46	1	1	3	3	1	1	2	3	2	2	3	1	2
47	6	2	3	3	2	2	3	5	5	5	2	2	0
48	2	2	6	5	2	1	3	2	6	5	4	1	5
49	7	7	3	4	2	3	2	3	3	5	2	1	2
50	14	10	8	5	3	6	6	7	4	7	6	3	7
51	11	8	7	5	2	7	5	6	9	12	7	7	4
52	3	2	3	2	1	2	3	2	2	0	1	1	3
53	10	6	7	8	4	5	10	14	11	10	8	4	5
54	3	5	5	2	0	4	5	4	6	5	7	2	5
55	6	6	5	2	1	5	4	6	3	3	4	2	6
56	2	1	1	0	0	1	0	2	1	0	0	2	2
57	2	0	0	0	2	1	1	1	1	2	1	2	2
58	1	1	1	0	0	0	0	1	1	0	0	1	0
59	1	2	3	1	1	2	2	2	3	3	3	1	2
60	1	3	3	2	1	2	3	3	2	2	3	2	2

[a]Instruction.

BRAIN-DAMAGED GROUP GSR FREQUENCIES: FINAL REST

	Minutes									
S#	1	2	3	4	5	6	7	8	9	10
1	5	7	4	3	8	10	6	11	14	19
2	7	6	4	4	11	3	4	5	5	7
3	2	4	3	4	1	2	2	4	6	7
4	4	4	5	4	0	0	1	0	2	0
5	6	6	3	7	7	8	9	3	5	9
6	1	0	0	0	0	0	0	0	0	0
7	7	6	5	6	6	1	3	2	1	2
8	4	0	1	2	2	2	3	2	6	5
9	1	2	1	2	2	1	3	2	2	2
10	1	0	4	7	3	0	3	1	1	2
11	2	1	3	3	1	0	2	5	8	5
12	2	1	0	0	0	2	1	0	2	0
13	1	2	0	1	1	1	2	0	0	1
14	1	0	0	0	0	0	0	0	0	1
15	6	3	2	2	3	1	1	0	1	3
16	5	6	3	5	6	4	2	5	4	6
17	0	0	0	0	0	0	1	0	0	0
18	9	5	2	4	3	3	2	8	4	2
19	0	0	0	2	0	0	0	0	0	1
20	2	1	0	0	0	1	3	0	0	1

CONTROL GROUP GSR FREQUENCIES: FINAL REST

	Minutes									
S#	1	2	3	4	5	6	7	8	9	10
41	9	7	2	4	0	0	1	0	0	0
42	1	0	0	0	0	0	0	0	0	0
43	5	3	4	2	2	1	2	1	3	1
44	4	5	2	2	3	0	5	3	0	1
45	5	5	2	4	5	9	5	8	6	11
46	3	1	2	2	1	2	3	1	1	1
47	0	1	1	3	2	3	0	2	0	0
48	2	0	0	0	0	0	0	0	0	0
49	2	1	1	0	1	0	0	0	0	0
50	7	6	5	4	4	4	1	2	2	0
51	7	1	4	4	5	7	6	4	4	3
52	2	0	1	0	0	0	0	0	0	0
53	5	3	6	3	2	2	2	2	1	0
54	4	0	0	0	0	0	0	0	0	0
55	6	0	1	2	1	1	2	1	5	2
56	1	1	0	1	2	0	2	3	2	0
57	0	1	1	1	2	1	1	1	0	0
58	0	0	0	1	0	0	0	0	0	0
59	3	2	3	0	0	0	1	0	0	0
60	3	2	4	0	2	5	5	0	0	0

APPENDIX H

LOG CONDUCTANCE DATA

BRAIN-DAMAGED GROUP LOG CONDUCTANCE: INITIAL REST

S#	Minutes 1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1	1051	1046	1229	1066	1060	1009	983	1154	1076	1061	1222	1079	1051	1066	1174
2	866	816	767	836	854	804	866	824	785	807	830	848	830	774	821
3	483	474	473	474	481	474	456	456	456	456	449	450	449	441	428
4	924	1027	951	876	818	757	688	647	616	596	585	576	580	678	870
5	1194	1155	1066	1236	1155	1092	1051	1036	951	1022	1086	1022	928	863	812
6	542	522	498	496	467	468	462	446	428	389	389	384	358	336	338
7	740	740	734	734	734	722	777	785	792	761	734	747	734	747	722
8	314	312	312	305	314	344	322	330	334	310	301	310	312	303	303
9	857	844	824	830	807	785	785	827	816	790	821	785	764	721	757
10	928	914	936	883	863	845	818	863	842	810	788	788	772	793	836
11	1013	1061	1034	939	896	1036	1114	1131	932	943	1036	983	924	979	907
12	253	269	261	269	279	282	290	309	303	303	303	305	305	321	318
13	468	551	514	499	499	587	554	521	564	567	545	529	520	516	511
14	695	699	695	695	686	695	692	692	690	690	688	690	719	719	719
15	782	796	757	742	728	728	717	703	678	699	684	676	752	684	703
16	1252	1027	987	987	1031	947	1174	928	928	839	991	936	951	863	966
17	866	883	879	900	879	893	903	900	900	879	910	886	883	860	910
18	1658	1620	1569	1538	1569	1481	1456	1387	1444	1337	1328	1387	1284	1236	1236
19	634	638	638	631	633	629	625	625	622	622	631	627	614	614	612
20	1022	1018	1119	1125	1125	1114	1114	1108	1102	1102	1086	1076	1051	1027	1013

Note.--Decimal points have been omitted.

CONTROL GROUP LOG CONDUCTANCE: INITIAL REST

	Minutes														
S#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
41	378	364	348	299	288	255	330	230	155	120	114	093	083	083	086
42	281	258	233	220	220	220	210	207	207	207	207	207	207	207	207
43	1284	1301	1284	1292	1292	1319	1301	1337	1301	1337	1357	1337	1347	1357	1337
44	630	616	589	572	572	517	465	413	400	647	647	559	468	386	328
45	932	910	900	880	873	841	832	860	848	854	832	801	769	996	812
46	536	504	488	521	493	517	483	550	545	528	521	526	526	491	551
47	1022	1004	979	996	1161	1060	1149	1027	1070	1056	987	1027	971	928	889
48	481	471	461	461	440	440	425	417	410	417	405	399	390	382	375
49	857	839	821	796	775	762	750	750	745	745	759	810	798	798	790
50	409	409	403	403	394	394	394	394	394	394	394	397	397	397	397
51	962	851	827	827	857	788	810	810	780	824	827	785	745	839	780
52	553	551	527	517	492	472	461	446	638	638	588	542	523	535	517
53	461	414	429	356	394	308	398	457	331	261	272	310	315	327	496
54	607	609	604	616	607	587	562	559	550	550	540	540	533	533	513
55	333	333	344	330	409	445	467	460	493	462	481	472	472	458	523
56	208	179	166	139	123	112	108	105	119	125	152	173	186	167	224
57	533	533	533	538	551	551	547	551	551	564	572	572	570	570	570
58	851	839	848	848	848	851	851	866	866	857	857	860	866	860	860
59	567	502	500	493	486	482	491	484	480	474	476	462	451	460	414
60	701	708	701	694	652	608	591	602	621	625	620	611	624	625	565

Note.--Decimal points have been omitted.

BRAIN-DAMAGED GROUP LOG CONDUCTANCE: STARTLE, REST I, ADAPTATION, AND REST II

		Minutes												
	Startle	Rest I			Adaptation							Rest II		
S#		1	2	3	1	2	3	4	5	6	7	1	2	3
1	1268	1102	1091	1108	1125	1091	1086	1060	996	979	958	955	921	1009
2	900	900	854	801	844	787	787	769	774	741	804	747	719	703
3	434	434	439	439	455	480	470	460	454	450	444	455	455	450
4	987	910	836	1051	1523	987	896	726	708	714	674	641	636	621
5	1161	1060	1229	1149	1194	1168	1143	1131	1137	1086	1076	1051	1004	1097
6	465	442	444	431	520	554	526	549	509	516	467	446	426	396
7	761	747	747	761	785	754	747	728	728	722	722	722	716	769
8	360	362	332	377	380	365	354	314	299	312	358	326	297	320
9	924	917	880	823	943	932	932	932	932	932	896	886	876	876
10	928	841	770	750	790	818	815	785	770	735	735	742	724	714
11	983	914	889	834	907	896	866	836	907	921	932	900	839	796
12	335	327	327	327	334	337	342	349	362	358	363	367	370	367
13	642	585	548	599	611	592	556	583	540	520	506	506	594	548
14	719	717	709	815	767	770	746	721	695	695	692	686	686	684
15	813	699	672	672	664	676	692	717	724	730	708	672	658	660
16	1456	1071	939	917	1027	962	951	900	951	1017	963	932	928	886
17	963	903	879	900	936	917	889	869	854	857	848	827	818	813
18	1620	1469	1347	1347	1509	1509	1509	1456	1432	1432	1456	1420	1409	1409
19	686	668	648	627	754	719	724	721	708	674	638	633	640	627
20	1086	1066	1081	1076	1143	1108	1086	1086	1086	1036	1018	1009	996	974

Note.--Decimal points have been omitted.

CONTROL GROUP LOG CONDUCTANCE: STARTLE, REST I, ADAPTATION, AND REST II

		Minutes												
	Startle	Rest I			Adaptation							Rest II		
S#		1	2	3	1	2	3	4	5	6	7	1	2	3
41	225	225	207	182	190	260	246	225	152	146	170	190	120	117
42	212	238	233	220	250	279	301	348	373	373	346	324	312	303
43	1347	1347	1337	1347	1367	1377	1398	1420	1420	1432	1468	1444	1444	1444
44	664	653	559	459	400	575	529	438	394	348	326	276	238	217
45	943	917	880	857	914	920	947	977	928	917	917	906	889	876
46	697	589	526	516	576	604	602	602	618	575	533	508	474	496
47	1174	1070	1041	987	996	983	939	943	910	893	932	883	848	866
48	382	404	394	368	409	416	385	359	339	334	327	327	320	320
49	991	914	876	842	906	860	830	810	796	785	770	759	752	742
50	432	422	439	412	407	427	446	433	407	400	421	407	398	395
51	1027	854	796	796	870	857	780	863	812	750	796	804	777	777
52	547	551	530	518	500	573	539	520	496	488	475	467	474	466
53	889	625	511	418	530	524	431	484	381	313	296	289	271	242
54	538	620	582	556	590	590	590	564	550	542	542	529	529	520
55	548	569	564	493	672	658	668	587	538	503	542	527	498	484
56	313	251	235	204	264	264	254	239	228	233	214	217	209	228
57	567	567	567	567	580	582	609	609	600	588	593	595	612	588
58	876	873	863	863	870	870	870	860	867	857	854	854	836	830
59	554	591	584	441	505	602	600	591	593	572	400	561	550	397
60	647	592	585	571	590	603	591	585	584	561	578	555	551	543

Note.--Decimal points have been omitted.

BRAIN-DAMAGED GROUP LOG CONDUCTANCE: REACTION TIME AND REST III

	Minutes									
	Reaction Time							Rest III		
S#	1	2	3	4	5	6	7	1	2	3
1	1149	1051	1051	1032	1031	1066	1091	1018	1031	1055
2	782	821	790	769	754	774	882	882	780	737
3	465	461	455	453	447	456	583	586	580	579
4	1602	1236	1102	1066	1018	1060	1125	1468	1119	1031
5	1194	1168	1161	1248	1114	1102	1102	1081	1055	1004
6	542	542	554	501	465	431	433	380	352	336
7	754	754	740	728	722	710	710	699	699	716
8	441	441	430	406	380	393	393	423	373	362
9	900	900	900	873	854	854	857	844	830	812
10	810	810	880	833	830	824	824	801	775	738
11	943	943	943	943	896	857	857	857	815	772
12	370	421	434	436	436	446	434	438	429	420
13	594	594	606	618	590	558	582	545	521	561
14	684	684	804	804	793	767	767	767	721	688
15	633	738	726	712	701	686	676	714	738	699
16	1456	1456	1097	1125	1004	966	1018	962	943	959
17	924	924	917	886	866	854	863	851	833	824
18	1854	1770	1569	1569	1569	1569	1553	1495	1468	1432
19	889	889	883	830	810	804	804	780	752	730
20	1149	1131	1091	1091	1070	1114	1091	1081	1066	1055

Note.--Decimal points have been omitted.

CONTROL GROUP LOG CONDUCTANCE: REACTION TIME AND REST III

	Minutes									
	Reaction Time							Rest III		
S#	1	2	3	4	5	6	7	1	2	3
41	470	551	518	444	418	426	352	352	305	283
42	582	582	529	474	452	452	436	394	358	344
43	1468	1444	1444	1444	1432	1432	1444	1420	1410	1410
44	996	903	839	790	777	777	839	772	735	745
45	924	917	921	924	900	900	958	943	910	892
46	593	623	542	502	509	567	508	487	478	467
47	1027	979	979	951	932	921	921	921	870	833
48	404	432	409	407	396	394	383	705	654	623
49	1200	1236	1137	1120	1092	1086	1045	921	879	863
50	692	620	699	721	678	660	567	495	466	429
51	932	860	928	830	762	830	827	752	721	721
52	634	646	606	638	575	569	553	526	521	491
53	775	775	710	691	691	578	578	411	342	386
54	654	529	654	654	600	600	568	568	600	547
55	662	777	728	602	602	567	567	750	632	592
56	300	436	445	445	432	401	401	390	340	295
57	607	592	592	592	592	583	583	604	618	627
58	851	860	857	857	844	844	832	824	812	801
59	613	692	690	571	583	618	600	610	610	532
60	690	598	682	704	708	680	667	651	600	600

Note.--Decimal points have been omitted.

BRAIN-DAMAGED GROUP LOG CONDUCTANCE: PAIRED ASSOCIATES

	Trials												
	Easy List				Difficult List							Failure	
S#	I[a]	1	2	3	I[a]	1	2	3	4	5	6	I[a]	1
1	1102	1009	1125	1075	1036	1041	1009	1000	1013	1009	1031	1070	1018
2	863	798	752	738	740	726	759	735	717	703	701	761	745
3	587	594	595	592	594	587	595	594	602	609	607	618	625
4	1620	2222	1398	1398	1214	1523	1187	1468	1155	1180	1131	1699	1357
5	1194	1168	1143	1137	1125	1131	1102	1108	1119	1097	1086	1102	1092
6	372	408	516	518	518	509	499	445	465	438	409	478	467
7	1456	1432	1420	1420	1432	1420	1426	1426	1426	1426	1444	1481	1444
8	401	423	441	412	429	424	412	374	356	338	358	386	400
9	876	876	876	845	845	833	824	824	824	824	824	854	824
10	752	857	857	857	857	870	870	842	842	842	827	866	845
11	762	830	1013	1013	924	983	924	932	924	951	939	955	979
12	420	436	453	450	394	414	414	414	414	409	420	442	466
13	573	638	648	656	648	648	633	644	623	650	644	724	676
14	688	666	660	670	678	678	664	733	754	746	750	782	740
15	699	752	733	701	721	730	769	769	774	767	767	721	699
16	1377	1009	979	939	939	932	983	962	921	893	889	914	1102
17	854	886	886	870	839	848	833	827	807	796	780	772	780
18	1699	1699	1658	1553	1602	1602	1602	1583	1553	1658	1602	1658	1602
19	801	836	824	824	830	836	860	821	807	848	807	854	824
20	1161	1174	1149	1131	1137	1125	1108	1155	1155	1168	1131	1131	1174

[a]Instructions.

Note.--Decimal points have been omitted.

CONTROL GROUP LOG CONDUCTANCE: PAIRED ASSOCIATES

	Trials												
	Easy List				Difficult List							Failure	
S#	I[a]	1	2	3	I[a]	1	2	3	4	5	6	I[a]	1
41	428	438	426	426	419	475	506	509	419	419	429	569	455
42	423	529	529	532	511	511	489	462	462	450	450	472	488
43	1420	1420	1444	1444	1444	1420	1420	1420	1420	1420	1398	1398	1420
44	833	833	824	824	796	790	782	790	782	790	793	815	810
45	928	917	903	903	896	886	886	906	906	896	886	886	873
46	524	597	533	523	533	533	514	498	462	452	452	504	509
47	848	939	939	914	921	914	903	870	870	870	833	818	879
48	583	658	775	775	810	764	735	733	710	750	754	836	775
49	959	1100	1027	1027	1060	983	979	921	903	860	860	883	863
50	770	770	770	770	609	585	544	535	518	556	526	556	495
51	854	854	810	810	775	782	730	730	730	730	730	745	745
52	507	517	517	517	547	531	548	524	517	517	509	517	517
53	815	815	686	686	636	656	613	577	524	467	540	585	550
54	559	620	642	666	618	606	614	594	598	590	594	674	600
55	719	719	676	676	656	656	620	629	636	625	600	606	620
56	338	353	353	374	374	374	368	366	349	360	332	358	356
57	604	618	618	618	627	638	642	633	633	618	618	618	618
58	830	842	854	845	845	845	845	839	830	830	830	830	830
59	700	704	698	702	700	700	692	698	700	688	692	651	651
60	701	701	700	700	680	669	660	672	660	675	680	660	651

[a]Instructions.

Note.--Decimal points have been omitted.

BRAIN-DAMAGED GROUP LOG CONDUCTANCE: FINAL REST

	Minutes									
S#	1	2	3	4	5	6	7	8	9	10
1	996	936	1018	955	1004	959	974	991	1000	947
2	812	780	807	701	735	876	848	914	857	857
3	604	604	620	602	602	611	607	600	625	625
4	1244	1155	1180	1180	1119	1046	979	924	939	940
5	1076	1076	1031	1013	1000	991	991	958	962	962
6	373	344	332	318	310	316	322	326	326	322
7	699	694	694	683	673	664	659	659	641	641
8	378	340	330	352	356	369	346	364	356	356
9	793	793	793	793	793	780	793	762	785	772
10	775	790	777	798	854	842	810	790	807	809
11	886	863	860	851	824	851	785	854	812	813
12	495	516	504	523	475	463	456	474	474	474
13	604	604	577	550	642	595	597	606	567	550
14	723	680	660	646	640	625	606	597	594	594
15	687	666	660	662	721	686	654	690	674	674
16	907	943	951	917	939	898	848	896	879	870
17	764	750	730	719	708	703	719	703	701	692
18	1658	1658	1658	1602	1509	1495	1456	1538	1444	1420
19	780	759	728	703	684	692	662	646	642	682
20	1161	1143	1125	1114	1097	1086	1091	1066	1046	1004

Note.--Decimal points have been omitted.

CONTROL GROUP LOG CONDUCTANCE: FINAL REST

	Minutes									
S#	1	2	3	4	5	6	7	8	9	10
41	422	380	356	322	299	322	262	220	225	225
42	470	440	410	394	394	380	380	373	373	362
43	1410	1409	1410	1410	1444	1495	1456	1432	1432	1456
44	780	769	777	754	745	787	764	740	721	721
45	928	932	958	967	987	1031	1060	1108	1070	1070
46	465	436	465	438	476	459	459	450	426	426
47	796	730	717	738	719	706	695	699	688	688
48	764	721	697	656	580	548	498	475	460	475
49	863	839	818	796	780	772	759	757	735	747
50	516	470	453	444	436	419	470	413	403	403
51	745	678	676	699	695	719	684	678	656	656
52	526	507	496	488	484	480	480	481	484	487
53	550	394	347	580	476	350	573	407	369	369
54	627	578	570	556	540	529	527	531	518	508
55	695	594	556	538	597	544	559	656	604	561
56	287	263	255	276	260	258	258	246	223	230
57	620	611	616	614	620	604	593	582	573	567
58	836	836	832	807	801	790	782	774	754	754
59	509	642	642	631	591	582	581	591	584	467
60	640	650	649	640	636	602	585	585	571	481

Note.--Decimal points have been omitted.

APPENDIX I

DISCUSSION OF REACTION TIME AND PAIRED ASSOCIATES RESULTS

GROUP DIFFERENCES IN PERFORMANCE

As indicated earlier, many studies have shown that brain-damaged subjects show poor performance on a variety of psychological tasks. The results of this study provide further evidence of the impairment and specifically support the findings of experiments which used the tasks involved in this study. In the case of both the reaction time task and paired associates learning, however, there were differences from reported results.

Reaction time. The mean reaction times found in this study, for both control and brain-damaged groups, were somewhat longer than those reported in other experiments. However, the brain-damaged group had significantly longer reaction times than did the control group. There are certain differences in the equipment and stimulus situation which may account for the obtained differences. For example, the double-throw switch probably required more force to actuate it than did the micro-switches used by Blackburn and Benton

(1955). The fairly long time intervals between trials also could have contributed to the longer reaction times of the subjects in the present study. Other work (Woodworth & Schlosberg, 1958) has indicated that reaction time increases with inter-trial intervals beyond two seconds. Another feature of the reaction time situation which may have brought about the obtained results could be related to the strength of the stimulus. It has been shown that reaction time is related to stimulus intensity (Woodworth & Schlosberg, 1958) and since the subjects in this study were six feet from a fairly dim light, the light was necessarily less bright at that distance. It is also possible that reaction time is related to proximity to the stimulus, even when brightness, duration, and area are held constant.

It is not considered likely that these features of the experimental situation contributed differentially to the performance of the subjects so as to produce the significant difference in reaction time. The reason for this conclusion is that the control group performed relatively more poorly than did the subjects of Blackburn and Benton (1955) and Blackburn (1958), actually lessening the difference between the groups. Thus, notwithstanding the differences in experimental conditions when this study is compared with others,

the results demonstrate that the brain-damaged subjects were impaired on the choice reaction-time task.

Paired associates learning. Although both the brain-damaged and control groups in the present study had higher mean error scores than in the study reported by Stark (1961), the brain-damaged were significantly poorer in performance, on all aspects of the task, than the control group. The differences in mean error scores reported in the two studies may be partially due to the fact that the subjects in Stark's study were private patients. It may be assumed that such patients would have generally higher educational and socio-economic levels than VA patients, and also greater familiarity with verbal tasks. It is also considered possible that the subjects used by Stark had generally higher levels of motivation to perform adequately on verbal learning tasks. This is felt to be most applicable to the control group, who performed particularly poorly in comparison with Stark's results.

Further, it was found that the brain-damaged subjects in the present study had a significantly higher educational level than did the control group. Despite this higher level, the brain-damaged made significantly more errors in the learning tasks than did the control subjects.

APPENDIX J

U TESTS OF ELECTRODERMAL MEASURES BETWEEN BRAIN-DAMAGED SUB-GROUPS GROUPED ON THE BASIS OF TIME SINCE ONSET OF SYMPTOMS

U test Comparisons of Electrodermal Measures of Brain-damaged Sub-groups Divided on the Basis of Time since Onset of Symptons

Log Conductance

Condition	U[a]	p
Initial rest	35	>.10
Startle	38	>.10
Adaptation	39	>.10
Reaction time	37	>.10
Paired associates	41	>.10
Final rest	40	>.10

GSR Frequency

Condition	U[a]	p
Initial rest	38	>.10
Adaptation	40	>.10
Reaction time	36	>.10
Paired associates	39	>.10
Final rest	38	>.10

[a]For 11 x 9 comparisons, the expected U = 44.

APPENDIX K

ANALYSIS OF VARIANCE OF LOG CONDUCTANCE MEASURES

Analysis of Variance of Mean Log Conductance Measures

Source	df	Sum of squares	Mean squares	F	p
Total	359	3121.5			
Between Ss	39	2844.2			
Groups	1	261.2	261.20	3.84	$>.05$ $<.10$
Error	38	2583.0	68.00		
Within Ss	320	277.3			
Conditions	8	73.0	9.10	13.78	$<.001$
G x C	8	4.8	0.60	1.0	
Error	304	199.5	.66		