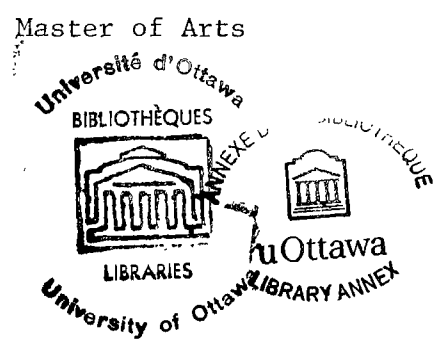


BILATERAL ELECTRODERMAL ACTIVITY
AND THE ORIENTING REFLEX IN
PATIENTS WITH UNILATERAL BRAIN DAMAGE
by John O'Kusky

Thesis presented to the School of
Graduate Studies of the University
of Ottawa as partial fulfillment of
the requirements for the degree of



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CURRICULUM STUDIORUM

John O'Kusky was born October 6, 1950, in Chicago, Illinois. He received the Bachelor of Science degree from Loyola University of Chicago in 1972.

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CHAPTER I

INTRODUCTION

Since the discovery of the galvanic skin response (GSR) at the end of the last century, measures of electrodermal activity have been of great utility in the fields of psychology and physiology. To attempt a comprehensive review of the history and technology of electrodermal recording would be beyond the scope of the present study, and a number of excellent reviews are already available (Edelberg, 1972; Montagu & Coles, 1966; Neumann & Blanton, 1969).

The concept of electrodermal activity includes all aspects of the electrical behavior of the skin, the observation of various phenomena depending on the recording technique employed. If two electrodes are placed on two similar skin sites or on the surface and interior of the body, an electrical potential difference between the electrodes can be measured on a sensitive, high impedance voltmeter. Potential differences vary from a few millivolts to as much as 50-60 millivolts, the surface being negative with reference to the interior. If a small alternating current is placed across two surface electrodes on similar skin sites, impedance measures can be obtained. By far the most commonly used measures in the literature on electrodermal activity are resistance and conductance. If two electrodes are placed on the skin surface and a small constant current is driven through them, the skin behaves as a resistor. A voltage develops across the electrodes and, by application of Ohm's law, the resistance of the skin can be calculated. Conductance, the inverse of resistance, can be calculated from resistance data or it can be measured directly.

Basal resistance values for the normal subject range between 10 and 500 kilohms (between 100 and 2μ mhos, respectively, in conductance units). When

recording basal resistance, one may occasionally record the spontaneous fluctuations in the basal skin resistance, known as spontaneous galvanic skin responses (spontaneous-GSRs). When a pin-prick or an electrical shock is applied to some part of the subject's body, or when the experimenter feigns a blow to the subject's face, a large action potential will appear in the basal resistance record, each action potential being known as an evoked galvanic skin response (evoked-GSR). These responses have a characteristic waveform, requiring about two seconds to reach peak. A typical response latency at room temperature is about 1.8 seconds and response magnitudes typically amount to about 5% of the basal level, although responses of as much as 50% of the basal resistance are not uncommon (Edelberg, 1972).

The behavioral significance of electrodermal activity is indicated by the vast volume of research concerned with delineation of the classes of stimuli responsible for both spontaneous- and evoked-GSRs. Edelberg (1972) estimated the number of English language publications concerned with behavioral variables and electrodermal activity to be over 1500. To the extent that electrodermal activity reflects sympathetic inflow to the skin area under the electrodes, it is related to the level of arousal or to the emotional activity of the subject. For example, Burch and Greiner (1958) demonstrated an increasing frequency of GSRs with increased level of arousal, manipulated pharmacologically. Stennett (1957) demonstrated a direct relationship between skin conductance and arousal as manipulated by tasks of increasing difficulty. Edelberg (1972) has reviewed the literature on the relationship of electrodermal activity to levels of arousal, emotional activity, personality and pathology, and conditioning. He concluded that electrodermal activity, while not a nonspecific index of overall arousal or emotional activity, is

useful as an index of the autonomic correlates of arousal and emotion in specific instances of behavior. For example, Schacter (1957) has shown that the autonomic correlates of such closely related emotions as fear and anger are not the same.

For the most part, electrodermal activity is an epiphenomenon of physiological processes occurring peripherally in the skin. Edelberg (1972) has reviewed a number of the contemporary models of these peripheral processes with regard to their consistency with experimental evidence from the electrical and secretory behavior of the components of the skin. While there is no agreement as to the specific mechanisms of these peripheral processes, the generation of electrodermal baselines is seen as a result of the combined effects of the accumulation of sweat on the skin and the electrical properties of sweat glands and skin layers. When two electrodes are placed on the skin surface, the resistance between them is the sum of the resistances at the electrode sites, and the resistance of the interior of the body is negligible by comparison. Within the skin, the resistance lies in the layer known as the stratum corneum which acts as an insulator over the body surface. This layer is perforated by the ducts of the sweat glands, which act as conducting pathways through the layer depending on their level of activity. An increase in sweat gland activity results in a drop in resistance (an increase in conductance). This electrical model of the skin has been reviewed in greater detail elsewhere (Edelberg, 1972; Montagu & Coles, 1966).

The activity of the sweat glands is controlled by spinal sympathetic motoneurons in the ventral horn of the spinal cord; therefore, the fibers to the sweat glands are excitatory in function. Since there is no parasympathetic innervation, there are no inhibitory nerves to the sweat glands

(Wang, 1964). While electrodermal baselines are generated by local physiological processes, response activity results from signals reaching the periphery from higher centers in the central nervous system. The spinal sympathetic motoneurons are under constant and continuous bombardment from sweat centers at different levels of the brain, including the cerebral cortex, hypothalamus, and reticular formation, and these sweat centers appear to exert their influences on the contralateral body side (Wang, 1964). As a result, it is possible to monitor, to some extent, central nervous system activity through records of electrodermal activity.

The present study was concerned with investigating bilateral differences in electrodermal activity in both non-brain-damaged and unilaterally brain-damaged human subjects. Recent evidence, reviewed in detail in chapter II, suggests that the differences in electrodermal activity between the left and right body sides are greater for unilaterally brain-damaged subjects than for non-brain-damaged subjects. This greater difference is due to a lower resistance on the body side contralateral to the damaged hemisphere, resulting from damage to the sweat centers in that hemisphere. The present study examined the extent to which the recovery of function in the damaged hemisphere was paralleled by a decrease in the bilateral differences in electrodermal activity. It was predicted that the left-right differences in basal and response resistances for the brain-damaged subjects would approach normal values with the recovery of the function in cortical sweat centers. As a result of this relationship, it was predicted that bilateral measures of electrodermal activity could be used as a neuropsychological predictor of success in rehabilitation.

Chapter II reviews the literature concerning bilateral differences in electrodermal activity in normal and brain-damaged subjects and the central

nervous system mechanisms mediating these differences. Chapter III presents the design and procedure, chapter IV presents the results, and chapter V discusses these results. Concluding remarks are contained in chapter VI.

CHAPTER II

REVIEW OF THE LITERATURE

Bilateral differences in electrodermal activity

Numerous studies have reported bilateral differences in electrodermal activity in the normal subject: Baitsch, 1954; Fisher, 1958; Obrist, 1963; Galbrecht, Dykman, Reese, and Suzuki, 1965; Wyatt and Tursky, 1969; Varni, Doerr, and Franklin, 1971; Crocco, 1974. The findings of these studies suggest that the asymmetry of autonomic activity between left and right body sides as indicated by bilateral electrodermal records is typical rather than atypical for normal individuals.

Baitsch (1954), in a study investigating sex differences in basal skin resistance, measured resistance with a simple bridge circuit and galvanometer in a sample of 433 subjects. In approximately 80% of his subjects a higher basal resistance was encountered on the right side of the body. This difference was not related to handedness or sex. Fisher (1958), in a study investigating left-right differences in the perception of body image and corresponding electrodermal reactivity, reported similar findings of higher response resistance on the right side. Using a constant current technique he recorded resistance levels from corresponding points on the palms and three successive finger sites against a relatively inactive point on the upper arm in 78 subjects. The criterion for directionality of response was a difference of at least 80 ohms in the amplitude of spontaneous-GSRs between the left and right body sides, with at least twice as many responses favoring one side over the other. In 54 subjects the amplitude was found to be significantly greater on the right side, while the remaining subjects demonstrated no clear-cut directionality. With regard to body image, those

subjects having the higher resistance on the right side also demonstrated laterality of body image, rating the right body side as being smaller than the left side. These bilateral differences in electrodermal activity and body image were found to be unrelated to handedness.

Obrist (1963) conducted the first systematic study to determine the reliability of bilateral differences in electrodermal measures. Five male subjects participated in a 23-minute recording session each day for between 24 and 36 days. Each session consisted of 10 minutes of recording while the subject rested with his eyes closed followed by approximately 13 minutes of recording while he performed in a serial learning task, designed to be an appreciable stress. Separate measures of basal skin resistance and response resistance for spontaneous-GSRs were recorded from each side using a constant current technique. Significant differences were found between left and right basal skin resistance levels in three of the five subjects with the higher resistance levels encountered on the left side. An additional subject demonstrated a significantly higher basal resistance on the left side for the first 15 days of the experiment, while the right side had a higher resistance level for the remaining 21 days. In four subjects, these differences were smaller during the serial learning task than during the resting condition with basal resistance levels higher during rest than during learning. These results suggest that alerting or stressing the subject, lowering basal skin resistance levels, decreases the size of the bilateral differences in basal resistance.

Significant bilateral differences in response resistance were found in two subjects, the left side showing greater values. A third subject demonstrated greater response resistance on the left side for the first 14 days and on the right side for the remaining 15 days. There were

appreciable day-to-day intrasubject variations in basal and response resistances and in the bilateral differences in each measure. Intraindividual correlations (a) between basal skin resistance and response resistance on each side and (b) between bilateral differences in basal resistance levels and response resistance failed to support the hypothesis that (a) reactivity would be greater when basal skin resistance was high and that (b) bilateral differences in response resistance would be larger when the bilateral differences in basal resistance levels were larger.

Galbrecht, Dykman, Reese, and Suzuki (1965) confirmed the existence of bilateral differences in skin resistance, but they could not relate these differences to body side. Resistance was recorded in 20 male subjects from the inferior surface of the plantar arch of each foot using a constant current technique. Basal resistance recordings were made during a 5-minute resting period, followed by a series of 12 tones (60 decibels, 5-second duration) spaced at approximately 1-minute intervals. The session terminated with a 3-minute stimulus-free period. Each subject participated in eight sessions with 2- or 3-day intersession intervals. For each subject in each session, bilateral differences in prestimulus levels of basal skin resistance were evaluated. Six subjects exhibited reliable differences in all eight sessions, 10 in seven sessions, and all 20 in at least five sessions. Across all sessions for all subjects ($N=160$) basal resistance was significantly higher on the left side in 63 instances and on the right in 78 instances, with only 19 instances in which there was no significant difference. Of the 63 sessions in which left-side basal levels were higher, 20 sessions showed a significant left-side dominance in response resistance for the evoked GSRs and 2 sessions showed a right-side dominance. Of the 78 sessions in which right-side basal levels were higher, 20 sessions showed a

significant right-side dominance and 7 showed a left-side dominance. Differences in basal resistance and response resistance demonstrated no relationship to handedness.

Wyatt and Tursky (1969) recorded bilateral skin potential levels from 12 left-handed and 12 right-handed male subjects in an attempt to delineate the relationship between bilateral differences and handedness. Skin potential was recorded from the palms of both hands during a 5-minute resting condition, followed by a series of stimuli, a second 5-minute resting condition, a second series of stimuli, and a final resting condition, total recording time being approximately 24 minutes. The stimuli consisted of audio, visual, or tactile stimuli, presented either unilaterally or bilaterally, such that each stimulus was presented to the left side, the right side, and to both sides simultaneously. The auditory stimulus was a tone (90 decibels) presented through a pair of stereo earphones, the visual stimulus was a flash of light delivered to the left eye, the right eye, or both eyes simultaneously (consequently, all three visual stimuli were bilateral with regard to input to the cerebral cortex), and the tactile stimulus was an electrical shock (4 milliamperes) to either the left or right leg (no bilateral shock was presented in the interest of subject safety). The type of stimulus and laterality of presentation were randomly ordered in the first series and repeated in the second series of stimuli.

Both right- and left-handed groups had higher skin potential levels on the right side. Since the correlation between resting skin potential and skin resistance has been reported to be negative (Gaviria, Coyne, & Thetford, 1969), these findings are consistent with those of Obrist (1963). Two right-handed and two left-handed subjects consistently showed levels higher on the

left side than on the right side. Unilateral stimuli and handedness did not unilaterally affect the maximum response amplitude. For example, similar response amplitudes were recorded from both left and right hands for stimuli presented to either the left or right side. The response amplitude appeared to be independent of skin potential level, although right-handed subjects demonstrated a consistently lower response amplitude than did the left-handed subjects.

Varni, Doerr, and Franklin (1971) obtained bilateral skin resistance measures and bilateral photoplethysmographic vasomotor measures for 16 female subjects. Skin resistance was recorded, using a constant current technique, from the middle finger of both hands. Subjects were asked to hold their breath on six occasions spaced at least five minutes apart during the 2-hour session, this procedure being employed to increase the number of spontaneous-GSRs. Of the 16 subjects, 10 were found to have significant bilateral differences in basal skin resistance levels. Of these 10 subjects, all of whom were right-handed, 5 had a higher basal resistance on the right side, while the other 5 had a higher resistance level on the left side. Two subjects who initially had a higher resistance on the right side reversed this relationship after approximately 40 minutes of the experiment. Bilateral differences in vasomotor resting levels, variability, and reactivity were demonstrated in most subjects. Left-right differences in electrodermal activity corresponded to those of vasomotor activity, indicating that certain autonomic variables form patterns of asymmetry.

In a recent study investigating left-right differences in electrodermal activity in different types of French-English bilinguals, Crocco (1974) recorded skin resistance levels in 82 female subjects. Records were obtained using a constant current technique and electrodes were placed on the volar

and distal segments of the first and third fingers on each hand. The experimental session lasted approximately 40 minutes, with a 5-minute resting condition followed by a 35-minute stimulus condition during which 30 French and English words were presented to the subject. Bilateral differences in basal skin resistance varied from 0-20 kilohms with the left side having a higher resistance in approximately half of the subjects. Left-right differences in basal resistance demonstrated no relationship to handedness. Response amplitude was found to be consistently greater on the left side by an average of 570 ohms.

In the foregoing account of current research in bilateral differences in electrodermal activity it is apparent that the asymmetry of these measures is typical rather than atypical for the normal individual. Although the relationship between asymmetry and body side is still a controversial issue, there is a basic agreement concerning the existence of bilateral differences in the normal subject and the independence of these differences from the handedness of the subject.

Bilateral differences in brain-damaged subjects

While the determinants of normal bilateral differences in electrodermal activity are unknown, a consideration of these bilateral differences in brain-damaged subjects suggests a possible central nervous system mechanism. Electrodermal records obtained from patients with bilateral and unilateral brain damage and from animal ablation studies indicate that the neural control of electrodermal activity is essentially bilateral and that functional asymmetry of the cerebral hemispheres could be responsible for normal bilateral differences.

Studies concerned with cortical damage and electrodermal activity in humans have been somewhat limited in number. Sourek (1965) demonstrated

higher amplitude skin potential responses (conversely, lower amplitude response resistance) in human subjects on the side contralateral to unilateral damage in the cerebral cortex. Guttman and List (1928) reported that unilateral damage of the cerebral cortex produced greater sweating in response to increases in room temperature on the side of the body contralateral to the damaged hemisphere. This greater accumulation of sweat on one side of the body would be expected to increase skin conductance levels (to reduce basal resistance levels) on that side of the body. Parsons and Chandler (1969) demonstrated that brain-damaged subjects have higher skin conductance levels than normal subjects and that these differences are consistent over conditions ranging from rest and passive auditory stimulation to conditions requiring verbal and motor responses. In a relatively thorough investigation of skin conductance levels in patients with unilateral and bilateral brain damage, Holloway and Parsons (1969) reported higher palmar skin conductance levels on the side contralateral to the damaged hemisphere in the unilaterally brain-damaged subjects. This bilateral difference was not reported for the bilaterally brain-damaged subjects or for hospital controls. The theoretical importance of this experiment to the present study requires that it be examined in greater detail.

Holloway and Parsons (1969) obtained records from 23 brain-damaged subjects (left hemisphere lesions, right hemisphere lesions, and bilateral lesions) and 19 hospital controls. Laterality of damage was determined by a qualified neurologist, although it was not possible to determine the exact site or the extent of the lesion. Bilateral skin conductance was measured directly using a constant voltage bridge circuit, electrodes being placed on the palms and on the dorsal aspect of the third finger of each hand. The experiment consisted of five conditions: (1) a 5-minute

rest period, (2) 10 habituation trials with a 2-second buzzer, (3) 10 habituation trials with a 10-second buzzer, (4) 10 trials of a motor reaction test (subject required to push a foot pedal on command), and (5) 10 trials of a simple perception task (subject required to report the position of the second-hand of a clock face on command).

Left minus right skin conductance scores were computed for both palmar and dorsal recordings to assess the relationship between laterality of lesion and possible bilateral differences in skin conductance measures. Results indicated that (a) unilateral lesion groups had higher palmar skin conductance levels (lower basal resistance) on the side contralateral to the lesion than on the ipsilateral side, and (b) this contralateral effect was greater during conditions involving passive sensory stimulation (conditions 2 and 3) than during rest, perception, or motor conditions. No significant differences were obtained for the bilateral lesion group or for hospital controls across all conditions. These differences were not found for measures taken from the dorsal electrodes. Analyses were performed on skin conductance levels for each hand to assess the effects of the different treatment conditions for each side of the body. Results indicated that there was a functional asymmetry between the left and right body sides. The left palmar skin conductance data differentiated the two unilateral lesion groups while the right palmar data did not. These large differences were due to the right-lesion group having higher conductance levels on the left side than the left-lesion group had on the right side. The authors suggested that these differences could be due to the relative magnitude of the cortical lesions, right-lesion subjects having the more extensive cortical damage of the two unilateral groups. Finally, no consistent differences were found between left- and right-handed subjects.

Animal ablation studies, in general, have indicated that experimentally produced unilateral cortical lesions produce an increase in skin conductance levels (a decrease in basal resistance levels) on the body side contralateral to the lesion (Wang, 1964).

Temporary inactivation of one hemisphere in human subjects produces similar results. One technique for determining hemisphere dominance for speech in patients who are candidates for neurosurgery involves injecting a quantity of sodium amytal into the left or right internal carotid artery of the patient. This substance produces a temporary inactivation of the corresponding cerebral hemisphere, one result of which is a decrease in basal skin resistance (an increase in conductance) and a total abolition of spontaneous GSR activity on the body side contralateral to the deactivated hemisphere.¹ These results are consistent with the findings of Holloway and Parsons (1969).

Central nervous system influences on electrodermal activity

Two relatively independent centers in the central nervous system participate in the initiation and control of electrodermal activity. The premotor cortex (area 6, of Brodmann) is the best documented center of influence, capable of eliciting GSRs when stimulated (Schwartz, 1937; Wilcott, 1969). The descending pathway from this area courses through the pyramidal tract, bypassing the hypothalamus. Figure 1 illustrates this descending pathway. Stimulation of the pyramidal tract or the cerebral peduncles can elicit GSRs, and the section of one peduncle blocks the responses elicited by stimulation of the ipsilateral premotor cortex (Wall & Davis, 1951). Ablation of the hypothalamus does not prevent the eliciting of GSRs by stimulation of premotor cortex (Wang & Lu, 1930). The influences of this control center appear to be exerted contralaterally (Wang, 1964).

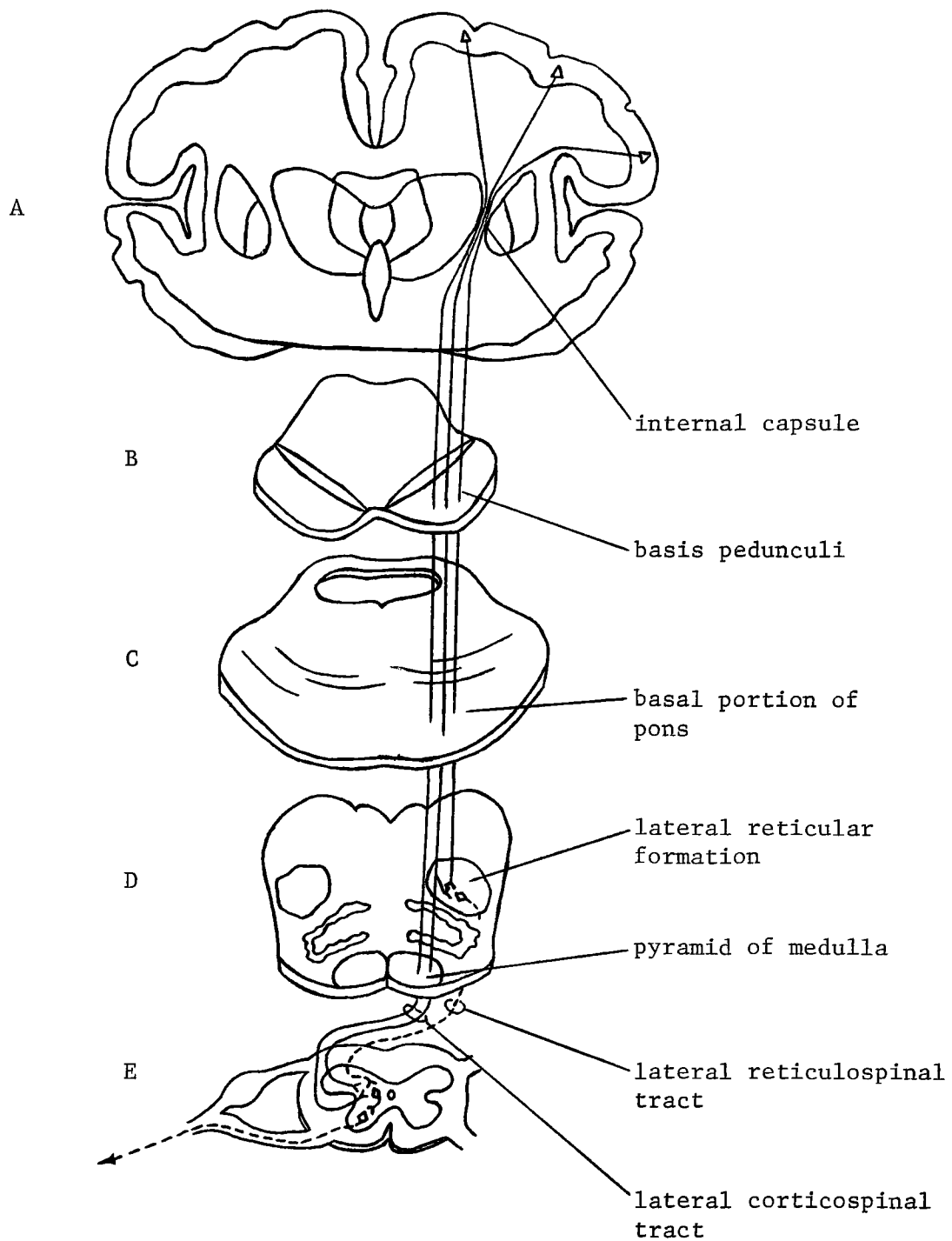


Figure 1. Theoretical descending pathway for premotor cortex influences on electrodermal activity. A: coronal section through premotor cortex. B: transverse section through midbrain. C: transverse section through pons. D: transverse section through medulla. E: transverse section through spinal cord.

In addition to premotor influences, there is a second source of influence involving the hypothalamus. The anterior limbic cortex apparently functions as a control center that is relatively independent of the premotor cortex. Isamat (1961) explored the anterior limbic and the infralimbic areas of the cerebral cortex in cats with electrical stimulation, while recording evoked-GSRs from all four feet. Results indicated that stimulation of the anterior limbic cortex produced evoked-GSRs (recorded as potential changes) in all four feet, although the response amplitudes were significantly greater from the contralateral feet. Descending fibers from these cortical areas make contact with the lateral portion of the anterior hypothalamus and the prechiasmatic area which, when stimulated, elicit GSRs (Hasama, 1929; Wang & Richter, 1928). Fibers from these areas impinge upon the preganglionic spinal sympathetic neurons. Figure 2 illustrates this descending pathway. The hypothalamic centers, which can be activated by direct warming and by stimulation of temperature receptors in the skin, appear to be one of the centers for thermoregulation (Kuno, 1956). In addition, since limbic areas have been implicated in emotional behavior (MacLean, 1955; Papez, 1937), input to the hypothalamus from the limbic cortex may represent the mechanism for eliciting electrodermal reflexes in response to emotional stimuli.

Animal ablation studies indicate that (a) while the cerebral cortex has both excitatory and inhibitory effects on subcortical centers, most of the frontal cortex has an inhibitory effect (Wang & Brown, 1956), and that (b) the cortical inhibitory effects are distributed contralaterally at the periphery (Wang, 1964). The higher skin conductance levels (lower resistance levels) on the side contralateral to unilateral cortical lesions have been interpreted as being due to the phenomenon of cortical release. Presumably, brain damage affects normal cortical inhibitory functions, releasing

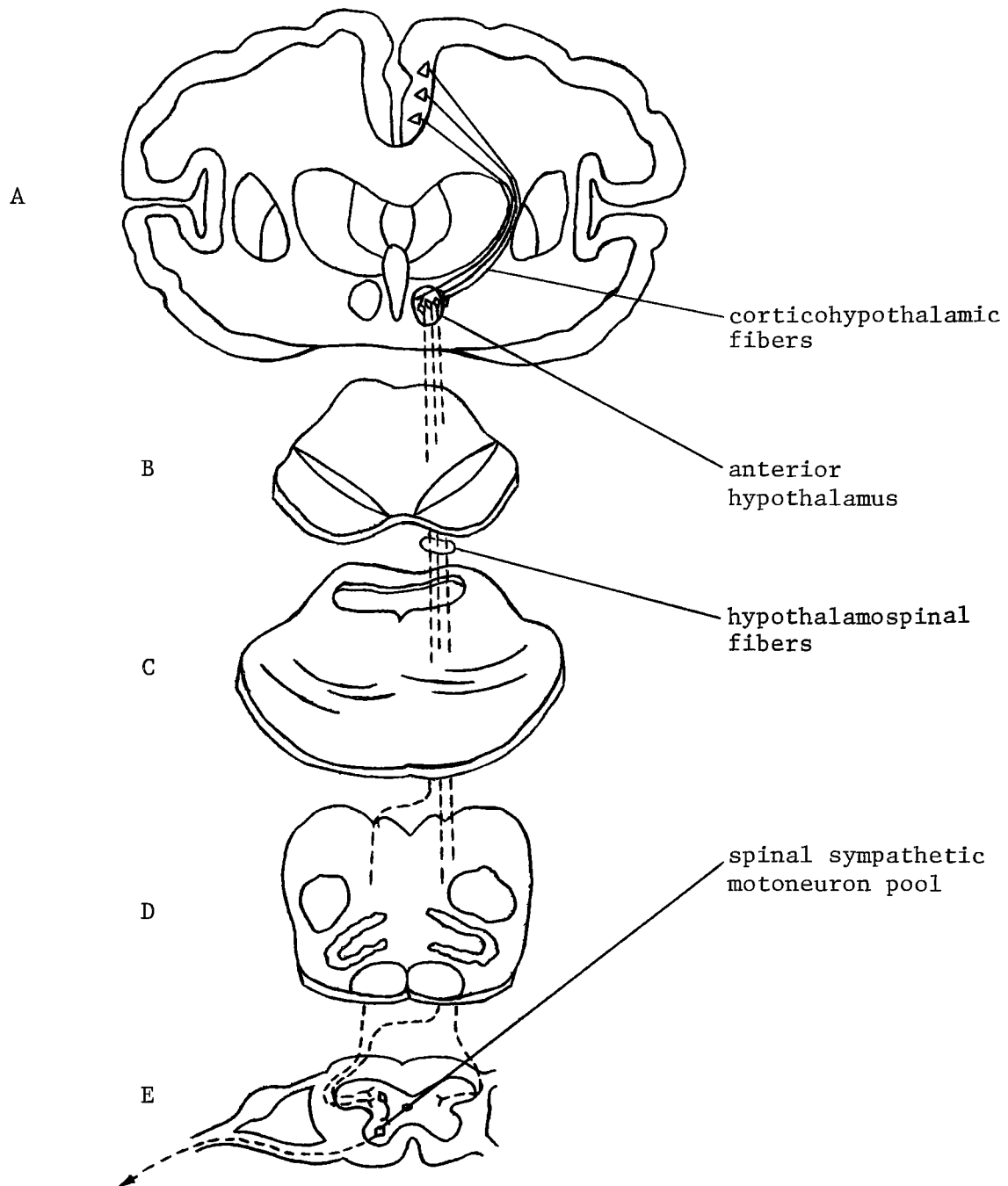


Figure 2. Theoretical descending pathway for limbic cortex influences on electrodermal activity. A: coronal section through hypothalamus. B: transverse section through midbrain. C: transverse section through pons. D: transverse section through medulla. E: transverse section through spinal cord.

reticular and autonomic networks from normal tonic cortical inhibition. Wang (1964) has demonstrated excitatory centers for the control of sweating in the anterior hypothalamus and in the lateral reticular formation of the midbrain. Both centers have direct connections with the spinal cord and spinal sympathetic motoneurons. In addition, there are direct cortico-spinal connections originating in the premotor cortex, also distributed contralaterally. Unilateral cortical lesions of premotor and limbic cortex would increase sweating and raise the skin conductance level (lower basal resistance) on the contralateral body side by means of (1) direct disinhibition of the anterior hypothalamus from the influences of the limbic cortex, (2) direct disinhibition of the lateral reticular formation of the midbrain from the influences of the premotor cortex, and (3) the termination of inhibitory impulses from the premotor cortex to the spinal sympathetic motoneurons via the direct route (bypassing the lateral reticular formation in the midbrain).

The functional asymmetry of the cerebral hemispheres in the normal brain could conceivably produce the bilateral differences in electrodermal activity seen in normal subjects by means of these cortical release mechanisms. Penfield and Roberts (1959) have indicated that, at least for language functions, there is a certain degree of asymmetry in the function of the cerebral hemispheres. However, the relationship between bilateral asymmetry of electrodermal activity and of hemisphere function is not simply one of handedness or cerebral dominance. No study, to date, has demonstrated a clear-cut relationship between handedness and bilateral differences in electrodermal activity. In addition, while 90% of the population is estimated to show left hemisphere dominance, at least for language, no consistent findings have emerged to link left-right differences in electrodermal measures to either body side. Significantly higher resistances (or the

equivalent in conductance or potential) have been reported on the right side (Baitsch, 1954; Fisher, 1958), on the left side (Obrist, 1963; Wyatt & Tursky, 1969), and on both the right and left sides with equal frequency (Crocco, 1974; Galbrecht, et al., 1965; Varni, et al., 1971). A further consideration of the mechanisms of normal bilateral differences in electrodermal activity will be made in chapter V.

Nature and properties of the orienting reflex

The concept of the orienting reflex (OR) was first introduced by Ivan Pavlov and his students in the early years of this century (Pavlov, 1960). Pavlov called the OR the "investigatory reflex" and the "What-is-it?" reflex. His description of the OR is as follows:

It is this reflex which brings about the immediate response in man and animals to the slightest changes in the world around them, so that they immediately orientate their appropriate receptor organ in accordance with the perceptible quality in the agent bringing about the change, making full investigation of it. The biological significance of this reflex is obvious. If the animal were not provided with such a reflex its life would hang at every moment by a thread (p. 12).

Pavlov attached a great deal of importance to the OR as a tool for investigating the discrimination of stimuli by the central nervous system:

It is obvious that the investigating reflex can be used to determine the degree to which the nervous system of a given animal is capable of discriminating between various stimuli. If, for example, among the differing environing agencies there is present a definite musical tone, any, even slight, alteration of its pitch will suffice to evoke an investigatory reflex in the form of a definite orientation of the ears and maybe of the whole body of the animal in relation to the tone. The same is true even of slight changes in various other elementary or compound stimuli (p. 112).

The physiological constituents of the OR have been studied extensively by Sokolov (1963). He asserted that the OR developed in the organism on any change of stimulus irrespective of the relative strength of the stimulus or of the quality of the stimulus. The development of an OR can be

interpreted as a sign that the central nervous system has detected a change of either magnitude or quality in the stimulus, that it has differentiated one stimulus from another. In addition, the time at which the OR develops relative to stimulus onset can indicate the time at which this differentiation was first effected. The physiological events heralding the development of an OR include (1) orienting movements of the body, head, eyes, ears, etc., (2) an increase in muscle tone, (3) cardiovascular and respiratory changes, (4) electrodermal changes (GSR activity), (5) desynchronization of electroencephalographic activity, (6) vascular changes (vasoconstriction in the limbs, vasodilation in the head), (7) pupil dilation, and (8) an increase in the sensitivity of the sense organs (lowering of the visual and auditory thresholds). These changes appear to orientate the organism to maximize on the detection of incoming stimuli and to mobilize the body to act in response to these stimuli.

The OR exhibits two additional qualities which functionally differentiate it from other classes of reflex (Lynn, 1966). The OR is an unspecific reflex. It develops in response to changes in the stimulus environment of the organism (changes in the novelty, intensity, quality, complexity, uncertainty, or incongruity of stimuli, or in situations of surprise or conflict). As such it is distinguished from adaptive reflexes (homeostatic reactions), defensive reflexes (startle reactions), and specific skeletal reflexes. The OR tends to extinguish or habituate with repeated presentations of the same stimulus, 10-30 presentations being sufficient to produce total habituation in most cases. The habituation is selective in that the presentation of a novel stimulus produces a characteristic OR, even when the differences between the habituated and novel stimuli are only slight.

The most comprehensive model for the OR is that proposed by Sokolov (1960).

This model suggests that stimulus analysis takes place in the cerebral cortex and that, after analysis, the cortex initiates the excitation or inhibition of the OR. Figure 3 illustrates the stages involved in this process.

Afferent stimulation ascends the classical sensory pathways to the cortex (pathway 1 in Figure 3), also sending afferent collaterals into the reticular formation (pathway 2). If the stimulus is novel or significant, the cerebral cortex sends excitatory impulses to the reticular formation (pathway 5), initiating the OR. Sokolov postulated that incoming stimuli leave traces in the cortex, these traces being called nervous models. He maintained that any incoming stimulus is compared with the nervous models existing in the cortex. If the stimulus does not match any of the stored nervous models, the OR is initiated. If the stimulus does match a nervous model, the OR is blocked.

The mechanism for the activation of the OR consists of (1) non-specific stimulation via collateral afferents activating the reticular formation (pathway 2), and (2) the cortex sending excitatory impulses to the reticular formation (pathway 5). If an incoming stimulus is matched to an existing nervous model, the OR is blocked by impulses from the cortex preventing transmission in the afferent collaterals to the reticular formation (pathway 3 blocking transmission in 2). Output from the cerebral cortex (pathway 6) initiates specific responses caused by coincidence between incoming stimuli and nervous models (habitual responses), while output from the reticular formation (pathway 7) initiates the somatic and autonomic components of the OR.

The habituation of the OR is a particularly suitable experimental paradigm for studying the nature and extent of psychophysiological responsiveness. Habituation has been shown to be an extremely variable, reversible process

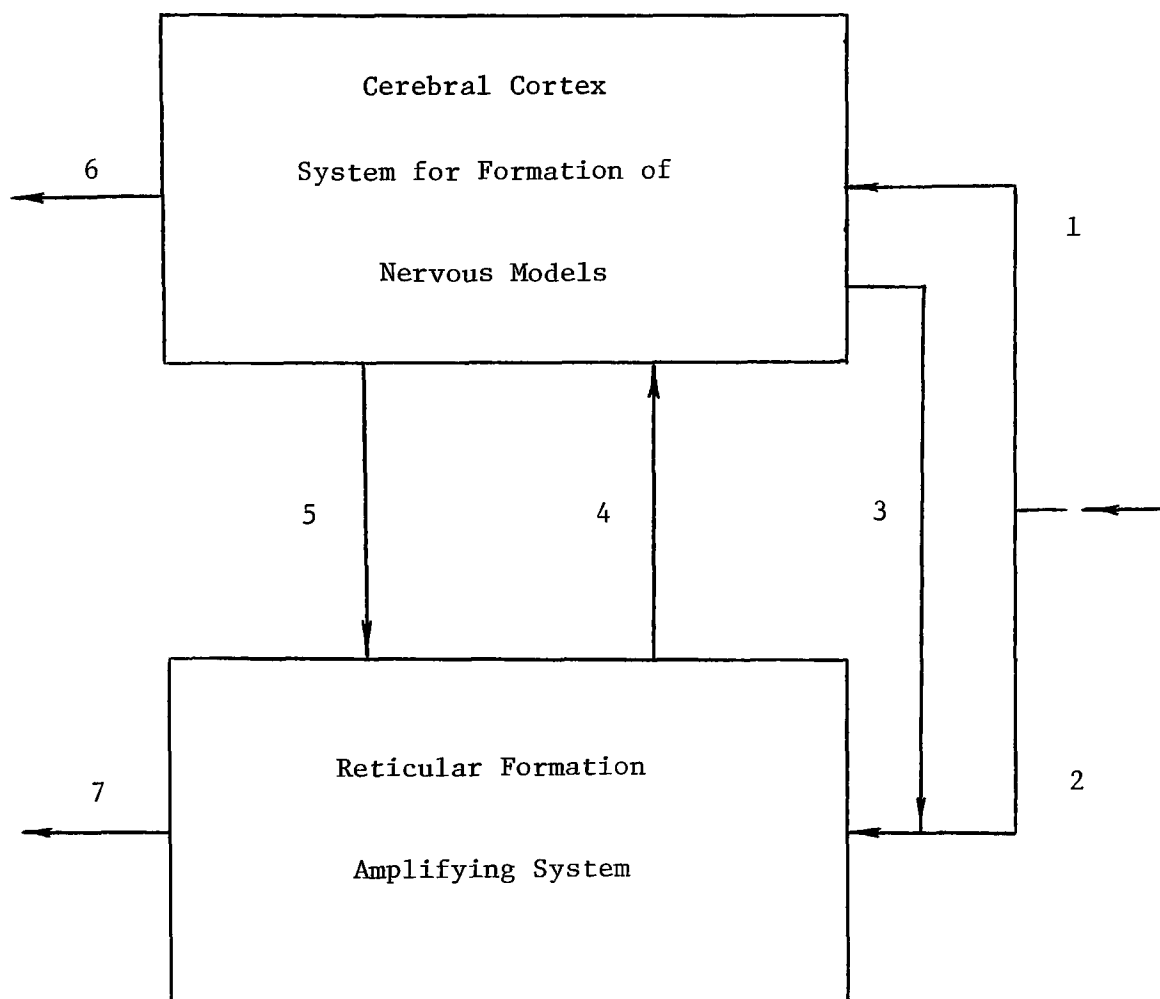


Figure 3. Sokolov's model for the orienting reflex. 1: sensory afferent to cortex. 2: collateral to reticular formation. 3: negative feedback from cortex to afferent input. 4: reticular input to cortex. 5: cortical input to reticular formation. 6: cortical output to habitual response mechanisms. 7: reticular output to somatic and vegetative components of the orienting reflex. (from Sokolov, 1960).

which occur rapidly if the stimuli are not meaningful and not intense. It occurs more slowly or not at all under conditions of increased alertness (Scholander, 1960), sleep (Johnson & Lubin, 1967), and central nervous system dysfunction (Bruillova, 1965; Davidoff & McDonald, 1964; Holloway & Parsons, 1971).

It has been suggested that the OR functions as a prerequisite for learning, and that habituation functions to free the higher processes of the brain from an overload of irrelevant sensory input (Lynn, 1966). Zeiner and Schell (1971) have demonstrated that subjects giving large ORs displayed significantly faster and better discrimination conditioning than did subjects giving small ORs. Cowles (1973) demonstrated that the OR is related to reaction time. Subjects with longer mean skin resistance response latencies tended to show slower performance on a simple reaction time task. The author suggested that there may be central nervous system mechanisms common to both processes.

It would appear that the OR is intimately associated with higher-order mental processes in humans. Evidence for this is found in the fact that cortical lesions in the frontal lobes affect both the capacity for higher-order problem solving and the components of the OR in humans (Luria, Pribram, & Homskava, 1964; Pribram, 1969).

Davidoff and McDonald (1964) compared the habituation of the OR in brain-damaged and non-brain-damaged patients, finding no differences in the habituation of the electrodermal (skin resistance responses) and alpha blocking components. However, the brain-damaged group failed to habituate the heart rate and vasomotor components. Similarly, Holloway and Parsons (1971) compared the habituation of electrodermal (skin conductance responses), cardiovascular, and electroencephalographic components of the OR in brain-

damaged and non-brain-damaged patients. Results indicated that, while the non-brain-damaged patients showed the expected habituation for all of these response variables, the brain-damaged patients displayed evidence of habituation only for the skin conductance measure. They also displayed higher initial skin conductance response amplitudes than did the non-brain-damaged group.

Bilateral differences in the orienting reflex

The only evidence for bilateral differences in the OR has been reported by Crocco (1974). For 82 subjects consistently higher response amplitudes were recorded from the left body side, the average left-right difference being approximately 570 ohms. The ORs were in response to verbal stimuli, and differences in response resistance demonstrated no relationship to handedness.

Investigating the habituation of the OR in patients with unilateral and bilateral brain damage, Holloway and Parsons (1971) found no consistent left-right differences in response latency, response amplitude, or habituation rate for the electrodermal component.

Electrodermal activity as an index of cortical plasticity

From the foregoing review of the literature on bilateral differences in electrodermal activity and in the OR, the following general statements can be drawn:

- (1) In the normal individual, significant bilateral differences in measures of electrodermal activity (basal skin resistance, skin conductance levels, skin potential levels, response resistance for spontaneous-GSRs) have been observed with some degree of consistency (Baitsch, 1954; Crocco, 1974; Fisher, 1958; Galbrecht, et al., 1965; Obrist, 1963; Varni, et al.,

- 1971; Wyatt & Tursky, 1969).
- (2) In the normal individual, these left-right differences bear no relationship to handedness or to cerebral dominance for language, and subjects often show reversals of left-right differences within and between recording sessions (Baitsch, 1954; Crocco, 1974; Fisher, 1958; Galbrecht, et al., 1965; Obrist, 1963; Varni, et al., 1971; Wyatt & Tursky, 1969).
 - (3) At least one study (Crocco, 1974) has demonstrated left-right differences in the amplitude of response resistance of the OR in normal subjects, with all subjects showing greater response amplitude on the left side (no relationship to handedness).
 - (4) In subjects with unilateral brain damage, left-right differences in electrodermal measures have been shown to be significantly greater than similar differences in subjects with bilateral brain damage and in subjects with no brain damage (Holloway & Parsons, 1969).
 - (5) In subjects with unilateral brain damage, these greater left-right differences are a result of significantly lower basal skin resistance (higher skin conductance) on the side of the body contralateral to the damaged hemisphere (Holloway & Parsons, 1969).
 - (6) No bilateral differences in the latency or amplitude of the electrodermal component of the OR have been demonstrated for subjects with either unilateral or bilateral brain damage.
 - (7) In brain damaged subjects, habituation of the OR has been seen consistently for the electrodermal component only (Davidoff &

McDonald, 1964; Holloway & Parsons, 1971).

- (8) With regard to the electrodermal component of the OR, the only consistent difference seen between brain-damaged and non-brain-damaged subjects is in the response amplitude on the first habituation trial, brain-damaged subjects having a significantly greater response amplitude to the first stimulus presentation (Holloway & Parsons, 1971).

With regard to the physiological mechanisms controlling these phenomena, the following hypotheses have been generated in the current literature:

- (1) The cerebral cortex, while exhibiting both excitatory and inhibitory effects, is primarily inhibitory in the frontal lobes (Wang & Brown, 1956).
- (2) These inhibitory effects are distributed contralaterally at the periphery (Wang, 1964).
- (3) The lower resistance (higher conductance) encountered in subjects with unilateral brain damage on the side contralateral to the damaged hemisphere is believed to be due to a cortical-release mechanism, that cortical lesions release reticular and autonomic networks from tonic inhibition to produce increased sweating and lower resistance on the contralateral body side (see Holloway & Parsons, 1969, 1971).
- (4) Two such cortical release mechanisms have been proposed:
 - (a) the premotor cortex-- lateral reticular formation of the midbrain-- spinal sympathetic motoneuron circuit, and
 - (b) the limbic cortex (anterior parahippocampal and cingulate gyri)-- anterior hypothalamus-- spinal sympathetic motoneuron

circuit.

The purpose of the present study was to examine the feasibility of using bilateral measures of electrodermal activity as an index of cortical recovery in patients with unilateral brain damage.

One of the most common symptoms of unilateral brain damage (especially in frontal lobe damage, as in carotid artery syndrome) is the occurrence of hemiplegia or hemiparesis, the paralysis or weakness, respectively, encountered in the musculature of the body side contralateral to the damaged hemisphere. Other symptoms may include language disturbances (aphasia) and perceptual disturbances (headache, hemianesthesia or hemihyperesthesia). These symptoms may have a sudden onset, a step-like onset, or a gradually progressive onset.

The extreme variability between patients in the clinical picture of recovery from these deficits warrants research into the predictors of cortical recovery and rehabilitative success. Patients may show complete recovery from these deficits, they may show no recovery, continuing at the same level of impairment, or they may show progressive deterioration. In the case of hemiplegia, Toole and Patel (1974) maintain that, if recovery is to occur, spontaneous improvement is usually discernible within a few days of the cerebrovascular episode. Rapid initial recovery will eventually be more complete than slow progressive recovery. If there has been no recovery of volitional activity after 6-12 weeks, it can be assumed that little will occur. Even after motor power begins to reappear, recovery may cease at any time and further deterioration is not impossible.

With the increasing cost of rehabilitation, it is increasingly important that objective predictors of the results to be expected from rehabilitative programs be developed. Gullickson (1970) has reviewed the

recovery predictors developed by the Kenny Rehabilitation Institute. Of the original 250 variables studied which related to the patient's physical capabilities, behavioral adjustment, and the etiological factors surrounding the cerebrovascular episode, the following variables have been shown to be most predictive of therapeutic recovery: (1) age, (2) duration since onset of episode, (3) level of consciousness during episode, (4) diastolic blood pressure, (5) degree of bladder continence versus incontinence, (6) self-care score at admission, (7) neurological involvement, left- or right-sided, (8) severity of visumotor disturbances, (9) impairment of voice quality, (10) MMPI scale score, and (11) score on the Picture Arrangement Subtest of the Wechsler Adult Intelligence Scale. From these variables, predictions can be made related to the patient's success in ambulation, required length of rehabilitation, and final disposition. Diller (1970) has reviewed some of the studies that have attempted to relate different predictors to different kinds of outcomes, concluding that further research is needed to understand mechanisms of dysfunction and recovery.

Bilateral differences in electrodermal activity could prove to be an objective index of cortical recovery and behavioral improvement in patients with unilateral brain damage. The large left-right differences in basal skin resistance and skin conductance levels seen in patients with unilateral brain damage could decrease, approaching normal bilateral difference values, as the associated cortical areas recovered from the trauma. It would be possible to monitor central nervous system recovery through bilateral electrodermal records, and behavioral improvement could be predicted from such estimates. Since the cortical areas controlling electrodermal activity (premotor and limbic cortex) also control functions of behavioral interest

(motor behavior and emotional responsiveness, respectively), recovery of electrodermal measures could serve as an index of recovery in the cerebral cortex or as a predictor of rehabilitative success. In addition, bilateral differences in the electrodermal component of the OR (differences in the latency or amplitude of responses and in the rate of habituation) could serve as an index for the behavioral concomitants of the OR (discrimination of stimuli, reaction time, higher-order problem solving).

A number of models are currently used to account for the actual recovery of the cerebral cortex after a trauma. These models include one form of regeneration, resolution of edema, and relief of intracranial pressure.

While observations of the phenomenon of regeneration in the nervous system have classically been limited to peripheral nerves, direct evidence has been reported for one type of regeneration in the central nervous system (Illis, 1973a, 1973b). The concept of the central nervous system as a fixed network is not tenable in view of these findings. Synaptic sprouting in response to injury, the development of new synaptic contacts between cells in the vicinity of an injured neuron, appears to be a general property of the nervous system, not confined to the periphery. Assuming that new synaptic contacts are being formed in response to lesions of the cerebral cortex, the full or partial recovery of a particular area of cortex should result in a corresponding recovery of the behavioral functions subserved by that cortical area.

Recovery in the cerebral cortex could also result from a resolution of the edema produced by injury. Tissue damage is often characterized by the presence of abnormally large amounts of fluid in the intercellular tissue spaces. In the central nervous system the presence of such fluid masses often exerts considerable pressure on the underlying tissue,

tending to disrupt the electrophysiological functions of that tissue. This fluid is eventually reabsorbed into the blood supply after trauma and the consequent reduction in pressure would be followed by a return to normal electrophysiological activity. Similarly, the relief of abnormally high pressure caused by other insults (subdural hematoma, cerebrovascular aneurisms) would be followed by a recovery of function. The full or partial recovery of a particular area of cortex by means of resolution of edema and pressure relief would also result in a corresponding recovery of the behavioral functions subserved by the particular cortical area.

Using bilateral differences in electrodermal activity to monitor the recovery of premotor and limbic cortex, one could presumably predict the recovery of those functions, other than electrodermal, influenced by these cortical areas. The premotor cortex influences, in addition to electrodermal activity, fine motor movements. One form of apraxia (impairment in the performance of learned movements without voluntary motor paralysis) is known to result from lesions of the premotor cortex (Barr, 1972). If the recovery of function in a particular area of cortex can be generalized to the surrounding cortical areas, the recovery of premotor cortex could be an indication of recovery in the surrounding areas, including the primary motor area (Brodmann's area 4), the prefrontal cortex (Brodmann's areas 9, 10, 11, and 12), and the motor speech area of Broca (in the left hemisphere). Limbic cortex has been implicated in the control of emotional behaviors and in the brain mechanisms for memory, in addition to influencing electrodermal activity (Barr, 1972).

Quantification and transformation of electrodermal data

The majority of studies in the current literature on electrodermal

research record basal skin resistance and response resistance, using a constant current technique. The data were kept in resistance units or transformed into units of conductance or log conductance. The transformation into conductance units was recommended by several authors (Darrow, 1964; Lykken & Venables, 1971) who maintained that, at high and low levels of bodily excitation, measurements of sweating, resistance, and conductance may not always be equivalent. Two distinctly different processes occurring respectively at high and low levels of resistance were seen to overlap in the middle range. The differing magnitudes of the measures of these two reactive phenomena were found to be combined in and quantitatively represented on a single scale when units of conductance were used. The transformation into log conductance units was recommended by Darrow (reported in Montagu & Coles, 1966) who suggested that biological responses were generally more logarithmic than linear in function.

Resistance, conductance, and log conductance data have been shown to demonstrate different experimental results when the same resistance data were used (Crocco, 1974; Haggard, 1949b), indicating that the measures may not always be equivalent. Haggard (1949a) has shown that a logarithmic transformation of data more properly meets the assumptions of the analysis of variance statistical procedure.

There is a tendency in physiological measures for response amplitudes to correlate with basal levels. This observation was formalized by Wilder (1957) as the Law of Initial Values (LIV), which stated:

Not only the intensity but also the direction of a response of a body function to any agent depends to a large degree on the initial level of that function at the start of the experiment. The higher this "initial level", the smaller is the response to function-raising, the greater is the response to function depressing agents. At more extreme initial levels there is a progressive tendency to "no response" and to "paradox reactions",

i.e., a reversal of the usual direction of the response. This rule holds true for 75-85 percent of all experiments (p. 73).

Even small differences in basal levels definitely influence the response. This is due to the fact that if basal levels are raised in arithmetic progression, the response amplitudes increase in logarithmic progression. Since the documentation of the LIV, a controversy has developed over various methods of quantifying and transforming response measures and over the actual validity of the LIV in electrodermal research (Germana, 1968; Lykken & Venables, 1971). No single method of handling data has been generally accepted to control for LIV effects, although a logarithmic transformation of response conductance data has been shown to be relatively independent of initial skin conductance levels (Raskin, 1969).

In the present study basal skin resistance and response resistance were recorded directly using a constant current technique. Response resistance data were transformed into conductance units and into log conductance units. Analyses were carried out using all three sets of data in an attempt to investigate the equivalence of these three measures.

Summary and statement of hypotheses

In an attempt to examine the feasibility of using bilateral differences in measures of electrodermal activity as an index of cortical recovery and behavioral improvement in patients with unilateral brain damage, the present study examined the relationship of bilateral measures of basal skin resistance and response resistance to the recovery of motor functions in left- and right-hemiplegic outpatients. Bilateral resistance measures were recorded during conditions of rest and passive auditory stimulation and these measures were related to performance on tests designed to measure strength, speed, and agility of motor behavior. In view of the

foregoing review of the literature, the following hypotheses were generated:

- (1) Left-right differences in basal skin resistance will be significantly larger for brain-damaged subjects than for non-brain-damaged controls.
- (2) The magnitude of left-right differences in basal skin resistance for all subjects will be positively correlated with left-right differences in the scores of motor tests of strength, speed, and agility.
- (3) With regard to the OR, left-right differences in the amplitude of response resistance will be compared between brain-damaged and non-brain-damaged subjects, although no hypothesis has been generated concerning this comparison.
- (4) Similarly, left-right differences in the rate of habituation of the electrodermal component of the OR will be compared between brain-damaged and non-brain-damaged subjects, although no hypothesis has been generated concerning this comparison.

CHAPTER III

METHOD

The design employed in the present study was the static group comparison (Campbell & Stanley, 1963). Subjects were assigned to either of two experimental groups on the basis of the laterality of unilateral brain damage resulting from a previous cerebrovascular accident. A third group of non-brain-damaged controls completed the design.

Subjects

Subjects were 14 hemiplegic outpatients (7 left-hemiplegics, 7 right-hemiplegics) contacted through the Royal Ottawa Hospital. Over 120 cases involving brain damage were reviewed before the final sample was selected. The diagnosis of hemiplegia was made by a qualified neurologist, based on a standard clinical examination and the results of cerebrovascular angiography. The diagnosis for all 14 subjects revealed a sudden occlusion of the internal carotid artery by an embolus of variable origin. Potential subjects were rejected if carotid angiograms were not available to confirm the diagnosis and if other pathologies were present (heart disease, epilepsy, etc.).

Figure 4 illustrates the area of cerebral cortex subject to vascular lesions when the internal carotid artery is occluded. It should be noted that those areas believed to influence electrodermal activity are affected. The internal carotid artery has a number of collateral branches before the terminal bifurcation into the anterior and middle cerebral arteries. These branches include (1) hypophyseal arteries, which supply the pituitary gland and hypothalamus, (2) the ophthalmic artery, which supplies the eye and orbital muscles, (3) the posterior communicating

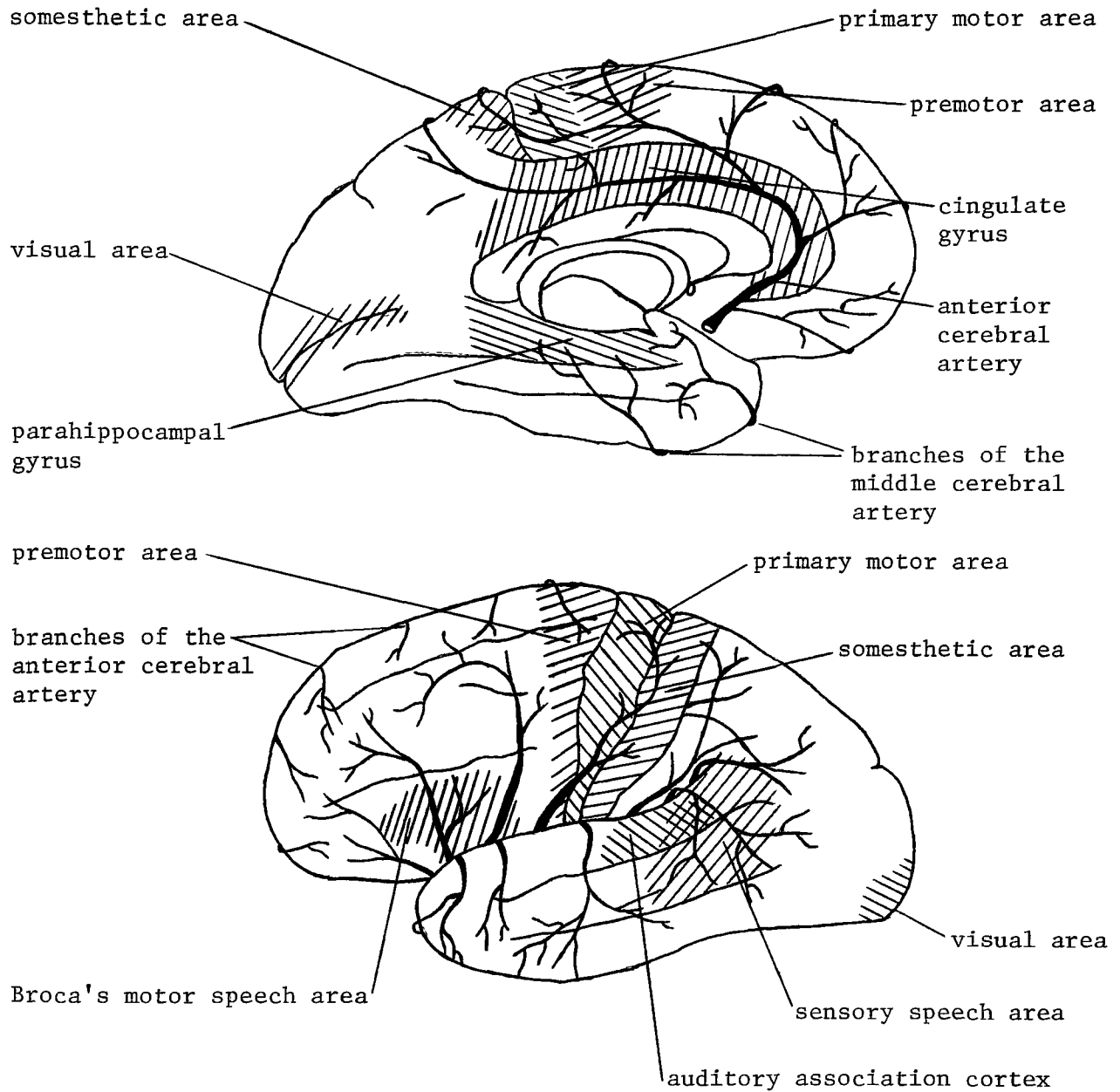


Figure 4. Areas of the cerebral cortex supplied by the internal carotid artery. Upper figure indicates the anterior cerebral artery on the medial aspect of the left cerebral hemisphere. Lower figure indicates the middle cerebral artery on the dorsolateral aspect of the left cerebral hemisphere.

artery, which is part of the circle of Willis, and (4) the anterior choroidal artery, which supplies the optic tract, amygdala, hippocampus, globus pallidus, lateral geniculate nucleus, and the ventral part of the internal capsule. The anterior cerebral artery supplies (1) the medial portion of the orbital surface of the frontal lobe, (2) the olfactory bulb and tract, (3) the medial portion of the frontal and parietal lobes, (4) the corpus callosum, (5) the somatosensory and motor areas for the contralateral leg, and (6) the head of the caudate nucleus and the anterior limb of the internal capsule. The middle cerebral artery supplies (1) the somatosensory and motor areas for the contralateral body side, (2) the auditory cortex, and (3) the dorsolateral aspect of both the frontal and parietal lobes.

In patients with symptoms of cerebrovascular disease, the incidence of carotid artery syndrome found at arteriography has been as high as 40% (Toole & Patel, 1974).

The symptom picture for the subject sample included a period of unconsciousness following the cerebrovascular episode, paralysis of the contralateral musculature, and symptoms of aphasia in the right-hemiplegics. All subjects had undergone some form of psychomotor rehabilitation with varying degrees of success. Subjects were assigned to one of two experimental groups on the basis of the damaged cerebral hemisphere; therefore, the left-hemisphere-damage group (LHD) included the 7 right-hemiplegics, and the right-hemisphere-damage group (RHD) included the 7 left-hemiplegics. In addition, a non-brain-damaged control group (NBD) was comprised of the 7 attendants and spouses who accompanied the patients to the recording sessions, bringing the total number of subjects in the study to 21.

Table 1 shows the demographic information for each of the three groups. All groups were equated for age and a comparison of the groups with the most extreme age differences (LHD, RHD) indicated that the mean difference was not significant ($t = .299$, $df = 12$, $p > .05$). All subjects were right-handed with the exception of one subject in the LHD group. All three groups contained equal numbers of male and female subjects (4:3, respectively). No significant difference was found between the two experimental groups with regard to the length of time since the onset of the cerebrovascular episode ($t = .153$, $df = 12$, $p > .05$).

The relatively strict criteria for choosing brain-damaged subjects resulted in two homogeneous experimental groups which were comparable in the location and extent of damage within the hemisphere.

Apparatus

Four silver-silver chloride electrodes were used for the active sites of the basal and response resistance measures. Electrodes were placed on the palmar sides of the first and third finger of each hand, on the middle phalange. Electrode sites were cleaned with acetone and allowed to dry before application. The electrode paste was a corn starch suspension containing 0.9% sodium chloride, isotonic to human sweat.

Basal and response resistances were recorded bilaterally using a constant current technique. A $10\mu\text{a}$. D.C. constant current was impressed across each of the left and right electrode pairs by a separate constant current generator. All measures were recorded on a six-channel Watanabe chart recorder, each pen having a 14-inch excursion. Two channels recorded left and right baselines, two channels recorded left and right responses, and a fifth channel was used as an event marker for the auditory stimuli. The differential input of the recorder kept the two

Table 1
Demographic Information for the
Three Groups of Subjects

Category	Group		
	LHD (N = 7)	RHD (N = 7)	NBD (N = 7)
Age (years) mean/standard deviation	52.0/6.16	47.3/13.15	47.9/9.35
Handedness (left:right)	1:6	0:7	0:7
Sex (male:female)	4:3	4:3	4:3
Duration since episode (months) mean/standard deviation	14.6/7.59	16.7/10.14	-----

electrical systems independent. Each system had its own independent power source and reference, such that the signal recorded by each system was the difference between its input and reference. This system eliminated the interfering currents that would result when two input systems have a single power source and a common ground.

All recording was conducted while the subject sat in a comfortable lounge chair in a temperature- and humidity-controlled, sound attenuated room. Lighting was maintained at two foot-lamberts and a background of white noise (55 db.), generated by an exhaust fan in the room, filtered out any extraneous noise. Auditory stimuli consisted of tones (80 db., 1000 Hz.) presented over a speaker centered immediately behind the subject's chair. Tone onset was controlled by the experimenter, and each stimulus presentation triggered the event marker on the chart recorder.

Motor tests were employed to assess the subject's strength, speed, and agility. Three standard tests from the Halstead-Reitan neuropsychological battery included (1) the dynamometer grip strength test, (2) the finger tapping test for speed, and (3) the grooved pegboard test for agility.

Procedure

The experiment was divided into three sessions: (1) an initial interview session for acquiring relevant subject data, (2) an electrodermal recording session, consisting of a 15-minute resting condition and a 15-minute passive auditory stimulation session, and (3) a motor testing session.

As each subject arrived for the experiment, he was seated at a table in the laboratory and interviewed with regard to the relevant aspects of his medical history (length of time since cerebrovascular episode, the

circumstances surrounding the accident, therapy since the accident) to corroborate the medical information already available. The subject was then tested to determine lateral dominance (a copy of the lateral dominance examination is presented in Appendix I). This initial interview session lasted approximately 15 minutes and allowed time for the subject to become acclimated to the temperature and humidity of the laboratory.

The subject was then seated in the lounge chair in the experimental room and instructed as to the general purpose of the study. As the electrodes were being attached, the subject was instructed to sit quietly and relax during the 30-minute recording session, to remain alert, and to move as little as possible. He was told that a series of tones would be presented over the loudspeaker and that he was only required to listen to these tones. After responding to questions, the experimenter closed the double doors of the experimental room and began the recording session.

During the 15-minute resting condition, the subject relaxed while measures of basal skin resistance were recorded from each hand. During the 15-minute passive auditory stimulation condition, a series of 30 auditory tones was presented to the subject. Each tone lasted 2 seconds with a variable inter-stimulus-interval of 20 to 40 seconds. Measures of basal resistance and response resistance (for evoked-GSRs) were recorded from both hands. The equipment used to record electrodermal activity was calibrated before each recording session and the two input channels were randomly varied between the left and right hands to eliminate any systematic machine bias.

At the conclusion of the recording session, the subject returned to the laboratory for motor testing. The dynamometer grip strength test was

administered before the subject was reseated. He was instructed to squeeze the dynamometer as hard as he could, and the procedure was repeated so that each hand had two trials, alternating each time between the dominant and non-dominant hands. The grip strength in kilograms was recorded after each trial. The subject was seated and the finger-tapping test was administered. The apparatus was presented to the subject and he was instructed to tap with his forefinger as fast as he could. The number of taps in a 10-second interval was recorded and the procedure was repeated five times for each hand, beginning with the dominant hand. The grooved pegboard test was the last test to be administered. The pegboard was presented to the subject and he was instructed to put each of the 25 grooved pegs into the holes in the board with his dominant as fast as he could. The time required to fill the board was recorded and the procedure was repeated for the non-dominant hand. The standardized instructions and procedures for each of these tests are presented in Appendix II.

Scoring procedure

All electrodermal records were scored using a double-blind procedure. All records were coded, and the experimenter did not know from which subject or group any given record had been obtained until all records were scored.

Basal resistance values were calculated for each 15-second interval in the 30-minute record; therefore, 120 basal resistance values from each hand were acquired for each subject from both resting and stimulus conditions.

A response was defined as a change in resistance of at least 200 ohms per square-centimeter, occurring between one and five seconds after the onset of the stimulus tone. For each of the 30 tones presented in the

stimulus condition, the amplitude (in kilohms) of the corresponding response was calculated. Amplitudes were quantified in resistance units and transformed into units of conductance and log conductance.

If R_b is the basal resistance at the point of response inflection and R_m is the resistance level at the point of maximum inflection, then the response amplitude in resistance units (R_r) was computed as follows:

$$R_b - R_m = R_r$$

The response amplitude as a change in conductance (C_r) was computed as follows:

$$1/R_b - 1/R_m = C_r$$

The response amplitude as a change in log conductance ($\log C_r$) was computed as follows:

$$\log_{10}(1/R_b) - \log_{10}(1/R_m) = \log C_r$$

After all 21 records were scored, 10 records were randomly selected to be scored a second time by another experimenter to determine the inter-scoring reliability. Separate product-moment correlation coefficients were calculated for basal and response measures. A correlation coefficient of $r = .96$ ($df = 2398$, $p < .01$) was calculated for the basal resistance data and a coefficient of $r = .92$ ($df = 298$, $p < .01$) was calculated for the response resistance data.

Data treatment

Physiological measures of basal skin resistance and response resistance were recorded from both left and right hands in all 21 subjects. The response resistance data were transformed into response conductance and response log conductance units. Similarly, motor performance measures were recorded from both left and right hands using the dynamometer grip-strength test,

the finger-tapping test, and the grooved pegboard test.

In order to directly assess the relationship between unilateral lesions of the cerebral cortex and possible bilateral differences in skin resistance and motor behavior, left-minus-right difference scores were computed for both basal skin resistance and response resistance, conductance, and log conductance. Similar difference scores were computed for the three motor measures. It should be noted that a positive difference score indicates that a particular left-hand measure is greater than the corresponding right-hand measure. A negative difference score indicates that a left-hand measure is less than the corresponding right-hand measure.

With regard to measures of basal skin resistance, the 30-minute recording session was divided into six trial blocks, each trial block having a duration of five minutes. With regard to measures of response amplitude, the series of 30 tones was divided into six trial blocks, each trial block consisting of the amplitudes recorded in response to five of the tones.

Four main hypotheses were stated at the end of chapter II. The first hypothesis stated that left-right differences in basal skin resistance will be significantly greater for brain-damaged subjects than for non-brain-damaged subjects. To test this hypothesis, a 3x6, groups by trial blocks, analysis of variance with repeated measures across trial blocks was applied to left-minus-right basal resistance data. Two similar analyses of variance were applied to left-hand and right-hand basal resistance data to determine whether or not these measures differentiated the three groups (LHD, RHD, NBD). If basal skin resistance for brain-damaged subjects is consistently lower on the body side contralateral to the damaged hemisphere,

the sign of the left-minus-right difference scores should also be consistent, LHD subjects having all positive scores and RHD subjects having all negative scores.

To test the second hypothesis, that the magnitude of left-right differences in basal skin resistance will be positively correlated with left-right differences in the scores of motor tests, left-minus-right scores for electrodermal measures were correlated with left-minus-right scores for motor tests using product-moment correlation coefficients. In addition, a correlation matrix was computed which correlated all electrodermal measures with all motor measures to determine whether or not the motor performance measures were in any way related to electrodermal baselines or response amplitudes.

The third hypothesis was concerned with differences in the amplitude of response resistance, conductance, and log conductance between groups, although no specific hypothesis was generated concerning the direction of these differences. Nine 3x6, groups by trial blocks, analyses of variance with repeated measures across trial blocks were applied to response measures of left-hand, right-hand, and left-minus-right data for each of the response resistance, conductance, and log conductance transformations. In other words, each set of response amplitude measures (resistance, conductance, and log conductance) was analyzed for left-hand data, right-hand data, and left-minus-right difference scores to determine whether or not these response measures could differentiate the subject groups.

The fourth hypothesis was concerned with differences in the rate of habituation of the OR between groups, although no specific hypothesis was generated concerning the direction of these differences. The 3x6, groups

by trial blocks, analyses of variance mentioned in connection with the third hypothesis were also used to determine differences in habituation rate.

In all analyses of variance, for those main effects and individual effects confounded by replication within subjects, the most conservative degrees of freedom were employed (after Greenhouse & Geisser, reported in Kirk, 1968). Tests on the difference between individual group or trial block means were accomplished with the Tukey post hoc procedure following analysis of variance.

Statement of null hypotheses

For the four main hypotheses stated at the end of chapter II, the following null hypotheses have been generated:

- (1) There is no difference between groups for left-minus-right difference scores for basal skin resistance.
- (2) There is no significant correlation between left-minus-right scores for basal skin resistance and left-minus-right scores for motor performance measures.
- (3) There is no difference between groups for left-minus-right difference scores for response resistance, conductance, or log conductance.
- (4) There is no difference between groups for the rate of habituation of the OR.

The level of significance sufficient to reject the null hypothesis was set at $p < .05$ for all statistical procedures.

CHAPTER IV

RESULTS

Basal resistance data

The first hypothesis in the present study predicted that bilateral differences in basal skin resistance would be significantly greater for brain-damaged subjects than for non-brain-damaged subjects. Table 2 is a summary of the 3x6, groups by trial blocks, analysis of variance for left-minus-right basal skin resistance. For basal resistance data, a trial block represents the mean resistance over a 5-minute recording epoch. There was no significant main effect for groups ($F = 0.19$, $df = 2/18$, $p > .05$); therefore, the first null hypothesis, that there is no difference between groups for left-minus-right scores of basal skin resistance, was not rejected. There was also no significant main effect for trial blocks ($F = 0.17$, $df = 1/18$, $p > .05$), indicating that differences in basal resistance levels did not change significantly over the 30-minute recording session.

Figure 5 illustrates the group means across trial blocks for left-minus-right basal resistance data. A positive difference score indicates that the left-hand values were larger than the corresponding right-hand values, while a negative difference score indicates that the right-hand values were larger than the corresponding left-hand values. The RHD group exhibited lower resistance on the right side across trial blocks, this finding being inconsistent with the prediction that brain-damaged subjects would show lower basal resistance on the side contralateral to the damaged hemisphere. The NBD group also showed lower resistance on the right side, although the left-right differences were less than those exhibited by RHD. The LHD group showed lower resistance on the right side for the first two trial

Table 2
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Minus-Right Basal Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	3225.277	2	1612.638	0.190
Subjects within groups	152533.240	18	8474.069	
<u>Within subjects</u>				
B (trial blocks)	63.045	1	12.609	0.174
AB	1870.151	2	187.015	2.580
Bx subjects within groups	6523.502	18	72.483	

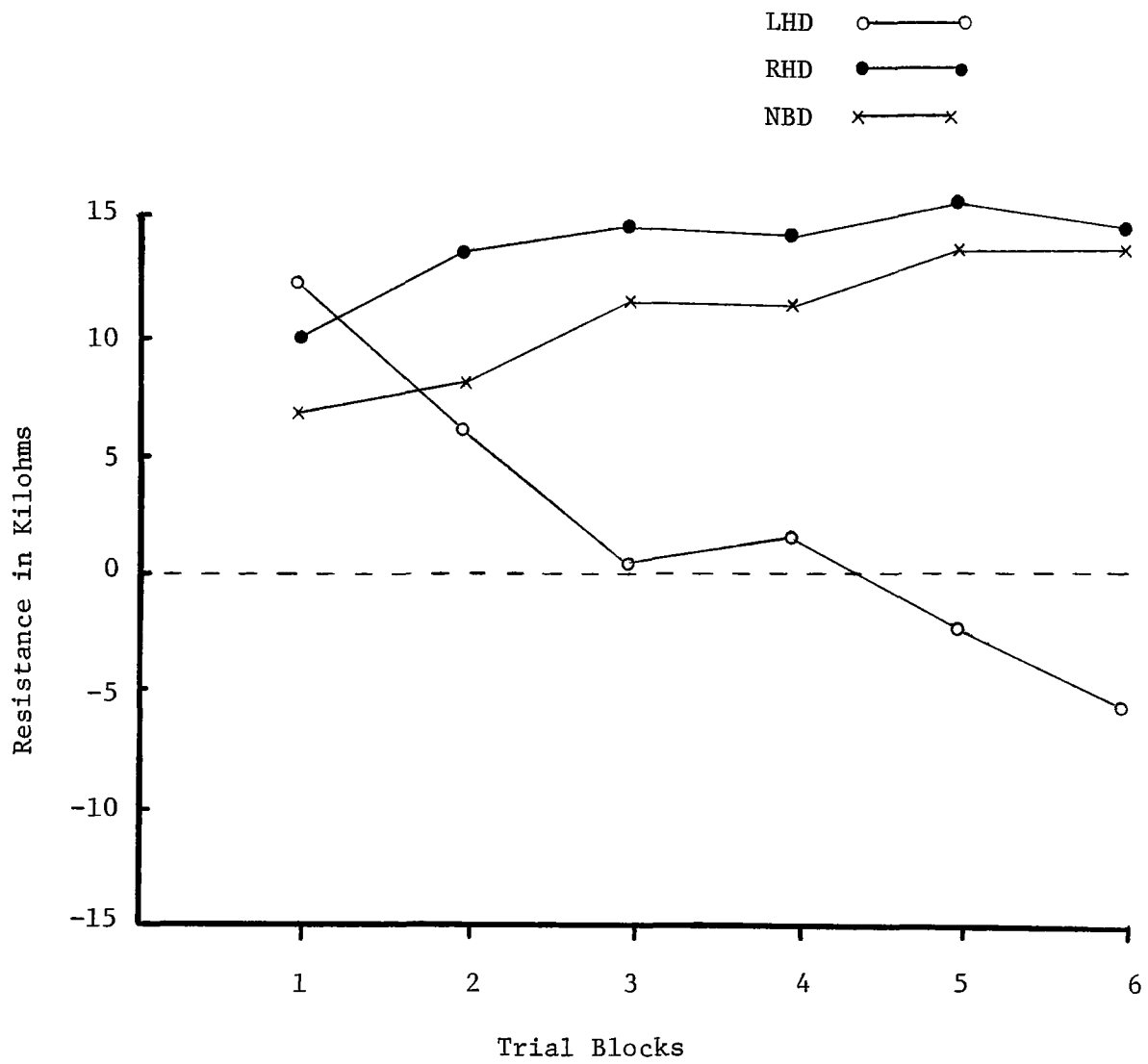


Figure 5. Trial blocks by left-minus-right basal resistance.

blocks, little difference between sides for the middle two trial blocks, and lower resistance on the left side for the final two trial blocks. Only the first two trial blocks are consistent with the prediction that resistance would be lower on the side contralateral to the damaged hemisphere.

The 3x6 analysis of variance was repeated for left-hand and right-hand basal resistances to determine whether or not the three groups could be differentiated on the basis of these data. Table 3 is a summary of the analysis of variance for basal resistance recorded from the left hand. There was no significant main effect for groups ($F = 0.16$, $df = 2/18$, $p > .05$), indicating that left-hand basal resistance could not differentiate the three groups. There was also no significant main effect for trial blocks ($F = 2.43$, $df = 1/18$, $p > .05$), indicating that resistance levels did not change significantly over the six trial blocks.

Figure 6 illustrates the group means across trial blocks for left-hand basal resistance. The two groups of brain-damaged subjects (LHD, RHD) exhibited similar resistance levels which were somewhat higher than the level of the control group (NBD). This finding was inconsistent with the prediction that resistance would be lower on the side contralateral to the damaged hemisphere for LHD and RHD subjects. For left-hand data, one would have expected similar basal resistance levels for LHD and NBD which would be higher than the basal level for RHD. One interesting finding was that all three groups showed a slow increase in basal resistance over the first three trial blocks, a drop in resistance between the third and fourth trial blocks, and another slow increase over the last three trial blocks. It should be noted that the series of 30 auditory tones began during the fourth trial block and continued to the end of the recording session. The slow increase in basal resistance over the first three trial blocks could

Table 3
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Hand Basal Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	3080.862	2	1540.431	0.158
Subjects within groups	175991.730	18	9777.319	
<u>Within subjects</u>				
B (trial blocks)	715.315	1	143.063	2.432
AB	191.860	2	19.186	0.326
Bx subjects within groups	5239.975	18	58.822	

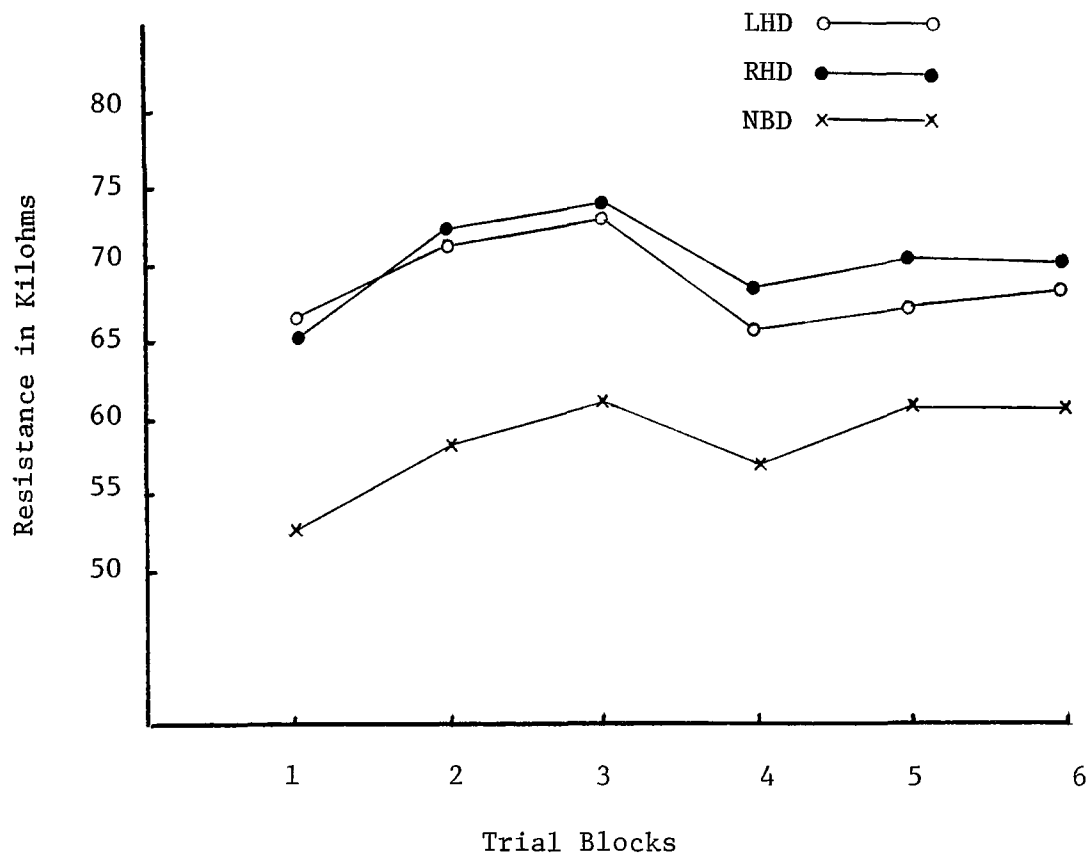


Figure 6. Trial blocks by basal resistance for left-hand basal resistance data.

be interpreted as a decrease in subject arousal during the resting condition of the experiment, the subject's becoming more relaxed in the experimental setting. The drop in resistance between the third and fourth trial blocks would result from an increase in arousal associated with the onset of the stimulus tones, while the slow increase over the last three trial blocks would result from the decrease in arousal that accompanies habituation to these stimuli.

The results of the analysis of variance for right-hand basal resistance, shown in Table 4, were similar to those for left-hand data. There was no significant main effect for either groups ($F=0.70$, $df=2/18$, $p > .05$) or trial blocks ($F=1.49$, $df=1/18$, $p > .05$). These results indicated that right-hand basal resistance data could not differentiate the three groups and that resistance levels did not change significantly over the six trial blocks.

Figure 7 illustrates the group means across trial blocks for right-hand basal resistance. The highest basal resistance level was recorded from LHD subjects with RHD subjects exhibiting a somewhat lower level. Subjects in the NBD group exhibited the lowest basal levels. This finding was inconsistent with the prediction that brain-damaged subjects would show lower basal resistance on the side contralateral to the damaged hemisphere. For right-hand data, one would have expected similar levels for RHD and NBD subjects which would be higher than the basal level for LHD subjects. All three groups showed fluctuations in right-hand basal resistance similar to those in the left-hand levels. There was a slow increase in resistance over the first three trial blocks, followed by a drop in resistance between the third and fourth trial blocks and a slow increase over the last three

Table 4
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Right-Hand Basal Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	7408.741	2	3704.370	0.704
Subjects within groups	94744.631	18	5263.591	
<u>Within subjects</u>				
B (trial blocks)	926.713	1	185.343	1.487
AB	1117.126	2	111.713	0.896
Bx subjects within groups	11217.447	18	124.638	

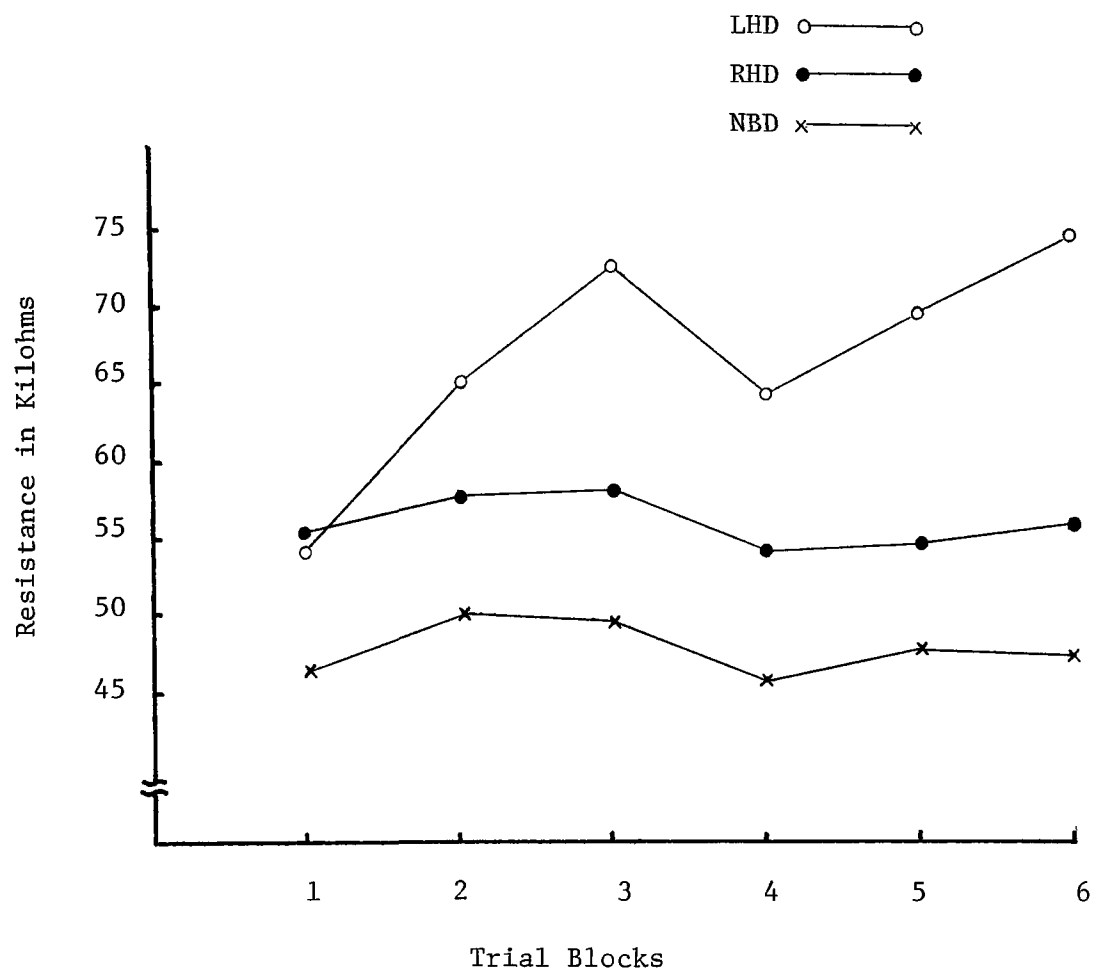


Figure 7. Trial blocks by basal resistance for right-hand basal resistance data.

trial blocks. While these changes in right-hand basal resistance levels were not significant, they were consistent for all three groups and their interpretation is the same as that for left-hand changes.

The second hypothesis issued in the present study predicted that the magnitude of left-right differences in basal skin resistance would be positively correlated with left-right differences in the scores of motor performance tests of strength, speed, and agility. It was predicted that asymmetry in electrodermal activity would be paralleled by a similar asymmetry in motor function because of the cortical areas common to both functions. Table 5 shows a correlation matrix between all electrodermal measures and all motor measures recorded in this study. Electrodermal measures are represented across rows and include left-hand, right-hand, and left-minus-right data for basal resistance, response resistance, response conductance, and response log conductance. Motor test measures are presented down the columns and include left-hand, right-hand, and left-minus-right data for the finger-tapping, dynamometer, and grooved pegboard tests. Both dimensions of the matrix include the variables of age and the length of time elapsed since the cerebrovascular accident to determine whether or not these variables correlated with either electrodermal measures or motor performance scores.

Results indicated that left-minus-right difference scores for basal resistance did not significantly correlate with any of the motor performance measures, including the left-minus-right difference scores for these measures; therefore, the second null hypothesis, that there is no significant correlation between left-minus-right scores for basal resistance and left-minus-right scores for motor performance, was not rejected. Similarly, left-hand and right-hand basal resistance measures did not significantly

Table 5

Correlation Matrix for Electrodermal
Measures and Motor Measures

	L-R Finger-Tapping	L Finger-Tapping	R Finger-Tapping	L-R Dynamometer	L Dynamometer	R Dynamometer	L-R Pegboard	L Pegboard	R Pegboard	Age	Time from CVA
L-R Basal R	-.07	-.11	.01	-.14	-.15	.04	.17	.08	-.16	-.35	.25
L Basal R	.02	-.13	-.10	-.01	-.22	-.18	-.01	.02	.02	-.17	.26
R Basal R	.11	-.04	-.15	.15	-.11	-.28	-.22	-.08	.23	.21	.03
L-R Resp. R	-.41	-.03	<u>.45</u>	-.41	-.11	.39	<u>.44</u>	.25	-.37	-.40	-.13
L Resp. R	-.04	.34	.27	-.02	.05	.07	.07	.14	.01	.01	-.14
R Resp. R	.35	.36	-.16	.36	.16	-.30	-.34	-.10	.36	.22	-.01
L-R Resp. C	-.34	-.11	.32	<u>-.44</u>	-.41	.18	.36	.28	-.25	-.04	.22
L Resp. C	.08	.14	.01	.07	-.06	-.13	-.03	.04	.06	-.28	.15
R Resp. C	.30	.15	-.25	.38	.29	-.20	-.30	-.20	.23	-.11	.10
L-R Resp. log C	-.27	.03	.32	.33	-.35	.09	.36	.32	-.22	-.04	-.03
L Resp. log C	.02	.35	.20	.03	.02	-.03	.03	.07	.01	-.06	-.13
R Resp. log C	.25	.41	-.01	.31	.31	-.10	-.25	-.17	.20	-.04	-.13
Age	.12	.05	-.10	.08	-.08	-.17	-.05	.31	.29	----	-.23
Time from CVA	.26	-.30	<u>-.54</u>	.14	-.31	<u>-.53</u>	-.10	.32	.37	-.23	----

_____ p < .05

correlate with any of the motor performance scores.

These results indicated that, contrary to the original hypothesis, subjects with larger bilateral asymmetry for basal resistance did not necessarily show larger asymmetry for motor performance.

Response resistance data

The third and fourth hypotheses predicted differences in the OR between the three groups, although the specific direction of these differences was not predicted. The third hypothesis predicted a difference between groups in the amplitude of responses to the 30 auditory stimuli. The fourth hypothesis predicted a difference in the rate of habituation to the series of stimuli. Response amplitudes were recorded in resistance units and converted into units of conductance and log conductance; therefore, separate analyses were conducted for these three sets of data.

Table 6 is a summary of the 3x6, groups by trial blocks, analysis of variance for left-minus-right response resistance. For response measures a trial block was defined as the mean amplitude of responses to a block of five tones. There was no main effect for groups ($F=1.64$, $df=2/18$, $p > .05$), indicating that the magnitude of bilateral asymmetry for response resistance was the same for all three groups. In addition, there was no main effect across trial blocks ($F=0.12$, $df=1/18$, $p > .05$), indicating that this asymmetry did not change significantly over the six trial blocks. The third null hypothesis, that there is no difference between groups for left-minus-right scores of response amplitude, was not rejected for resistance data.

Figure 8 illustrates group means across trial blocks for left-minus-right response resistance. For RHD subjects, the response amplitude was higher on the left side for the first two trial blocks, with left-right

Table 6
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Minus-Right Response Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	8.077	2	4.038	1.635
Subjects within groups	44.463	18	2.470	
<u>Within subjects</u>				
B (trial blocks)	0.410	1	0.082	0.117
AB	7.240	2	0.724	1.033
Bx subjects within groups	63.106	18	0.701	

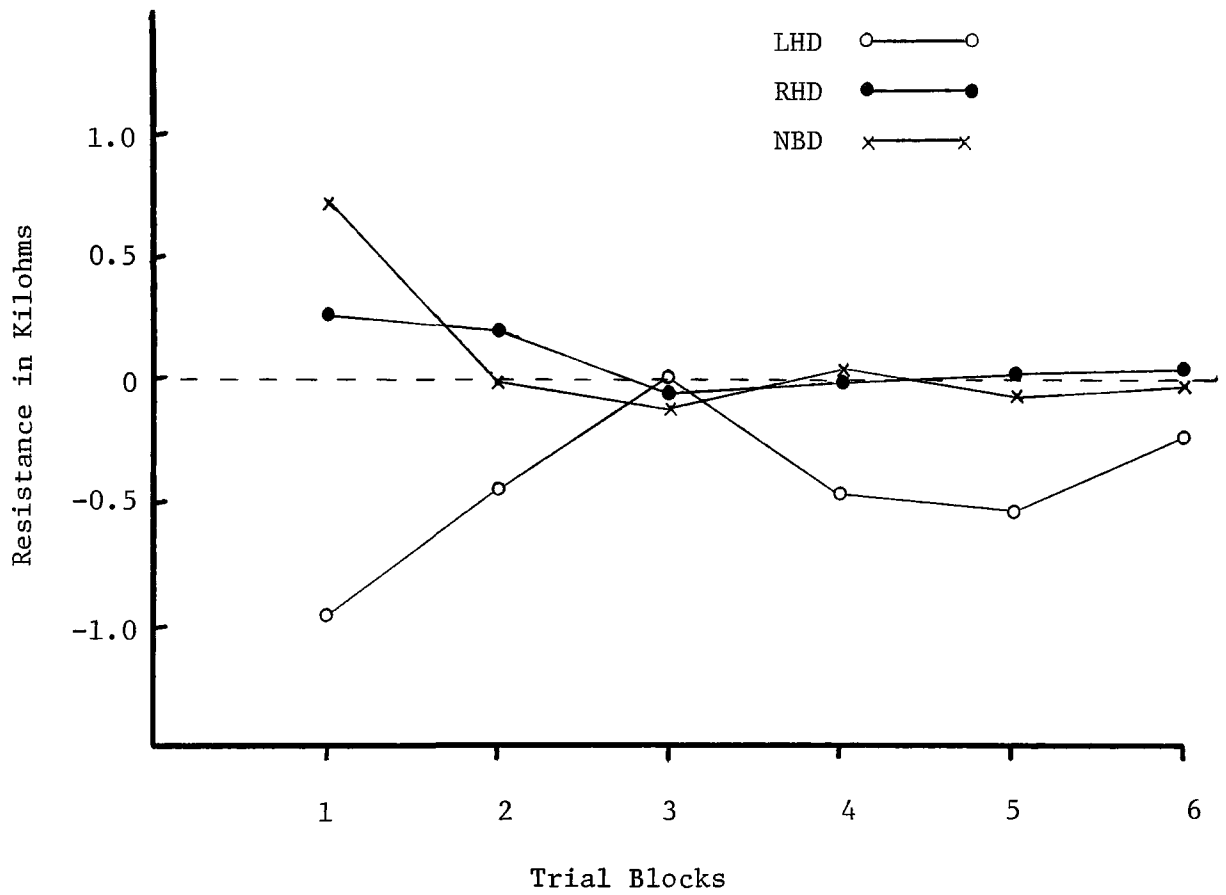


Figure 8. Trial blocks by left-minus-right response resistance.

differences approaching zero for the last four trial blocks. The NBD subjects had higher response amplitudes on the left side for the first trial block only, with left-right differences approaching zero over the last five trial blocks. With the exception of the third and sixth trial blocks, LHD subjects had higher response amplitudes on the right side. If one had predicted lower response resistance on the side contralateral to the damaged hemisphere, as in the case of basal resistances, the present findings would be inconsistent with that prediction.

The analysis of variance was repeated for left-hand and right-hand response resistance to determine whether or not these measures could differentiate the three groups. Table 7 is a summary of the 3x6 analysis of variance for left-hand response resistance. There was no significant main effect for groups ($F=0.08$, $df=2/18$, $p >.05$), indicating that the groups could not be differentiated by left-hand response resistance data. There was a significant main effect for trial blocks ($F=5.80$, $df=1/18$, $p <.05$), indicating a significant change in response amplitude over trial blocks. However, an F_{\max} statistic indicated that the assumption of homogeneity of variance had been violated for this analysis ($F_{\max}=15.6$, $k=6$, $df=20$, $p <.01$). The 3x6 analysis of variance was repeated on the same data after it had been normalized using a square-root transformation as recommended by Kirk (1968). Results indicated that the main effect for trial blocks remained significant ($F=5.82$, $df=1/18$, $p <.05$). Tukey post hoc tests indicated significant differences between the means of trial blocks one and two ($q=4.23$, $p <.05$), one and three ($q=6.34$, $p <.01$), one and four ($q=5.60$, $p <.01$), one and five ($q=5.42$, $p <.01$), and one and six ($q=6.11$, $p <.01$). The decrease in response resistance across trial blocks was attributed to habituation to the series of tones. Figure 9 illustrates this habituation

Table 7
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Hand Response Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	0.520	2	0.260	0.084
Subjects within groups	55.471	18	3.082	
<u>Within subjects</u>				
B (trial blocks)	31.281	1	6.256	5.799*
AB	6.478	2	0.648	0.600
Bx subjects within groups	97.102	18	1.079	

* p < .05

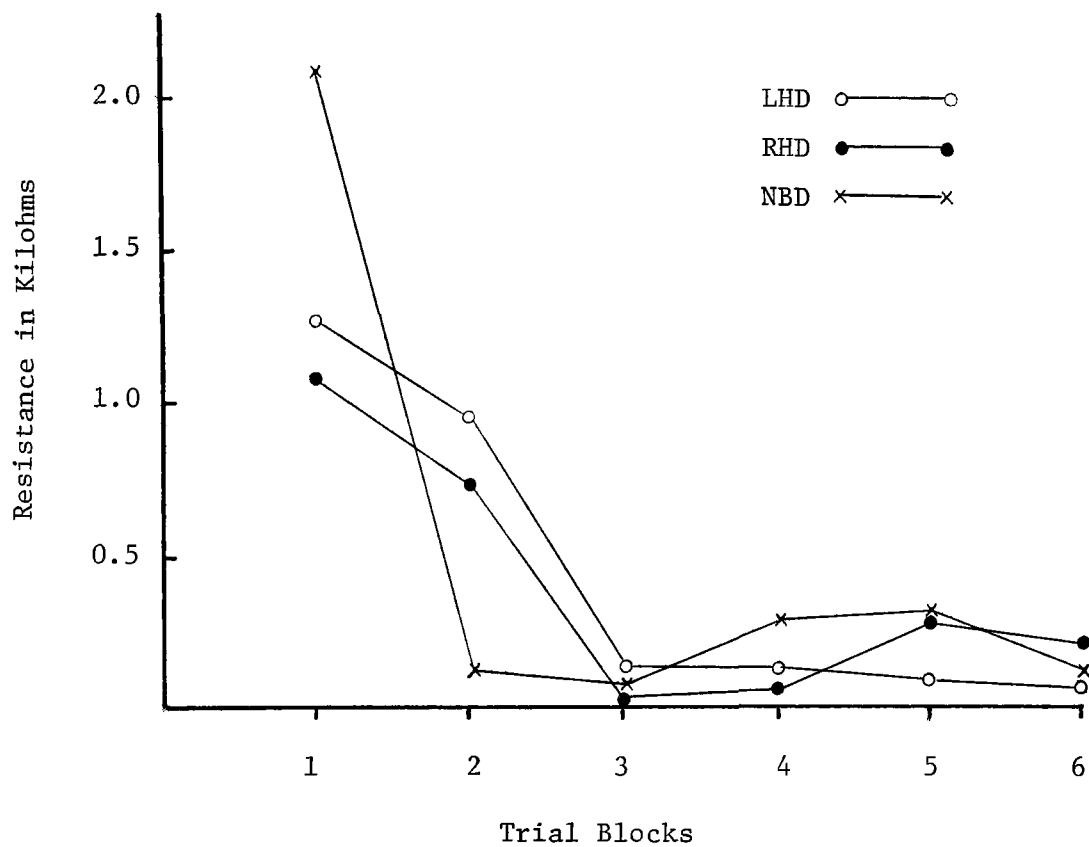


Figure 9. Trial blocks by response resistance for left-hand response resistance data.

of response resistance across trial blocks for left-hand data. Response amplitudes were highest in the first trial block, approaching zero by the third trial block (stimuli 10-15).

Table 8 is a summary of the 3x6 analysis of variance for right-hand response resistance. There was no significant main effect for groups ($F=1.47$, $df=2/18$, $p > .05$), indicating that the three groups could not be differentiated by right-hand response resistance. There was a significant main effect for trial blocks ($F=6.57$, $df=1/18$, $p < .05$). However, an F_{\max} statistic indicated that the assumption of homogeneity of variance had been violated for this analysis ($F_{\max}=19.1$, $k=6$, $df=20$, $p < .01$). The analysis of variance was repeated on the same data after it had been normalized using a square-root transformation. Results indicated that the main effect for trial blocks remained significant ($F=6.63$, $df=1/18$, $p < .05$). Tukey post hoc tests indicated significant differences between the means of trial blocks one and two ($q=4.41$, $p < .05$), one and three ($q=7.01$, $p < .01$), one and four ($q=5.93$, $p < .01$), one and five ($q=5.49$, $p < .01$), and one and six ($q=6.57$, $p < .01$). The decrease in response amplitude over trial blocks was again attributed to habituation to the series of stimuli. Figure 10 illustrates the habituation of response resistance across trial blocks for right-hand response resistance. Amplitudes were highest in the first trial block, approaching zero by the third trial block (stimuli 10-15).

The three groups did not differ significantly in the rate of habituation of the OR as indicated by both left- and right-hand response resistance. The groups by trial blocks interaction was significant for neither left-hand data ($F=0.60$, $df=2/18$, $p > .05$) nor right-hand data ($F=0.81$, $df=2/18$, $p > .05$). This finding indicated that groups did not differ significantly in response

Table 8
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Right-Hand Response Resistance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	8.407	2	4.203	1.474
Subjects within groups	51.315	18	2.851	
<u>Within subjects</u>				
B (trial blocks)	28.715	1	5.743	6.570*
AB	7.080	2	0.708	0.810
Bx subjects within groups	78.666	18	0.874	

*
 $P < .05$

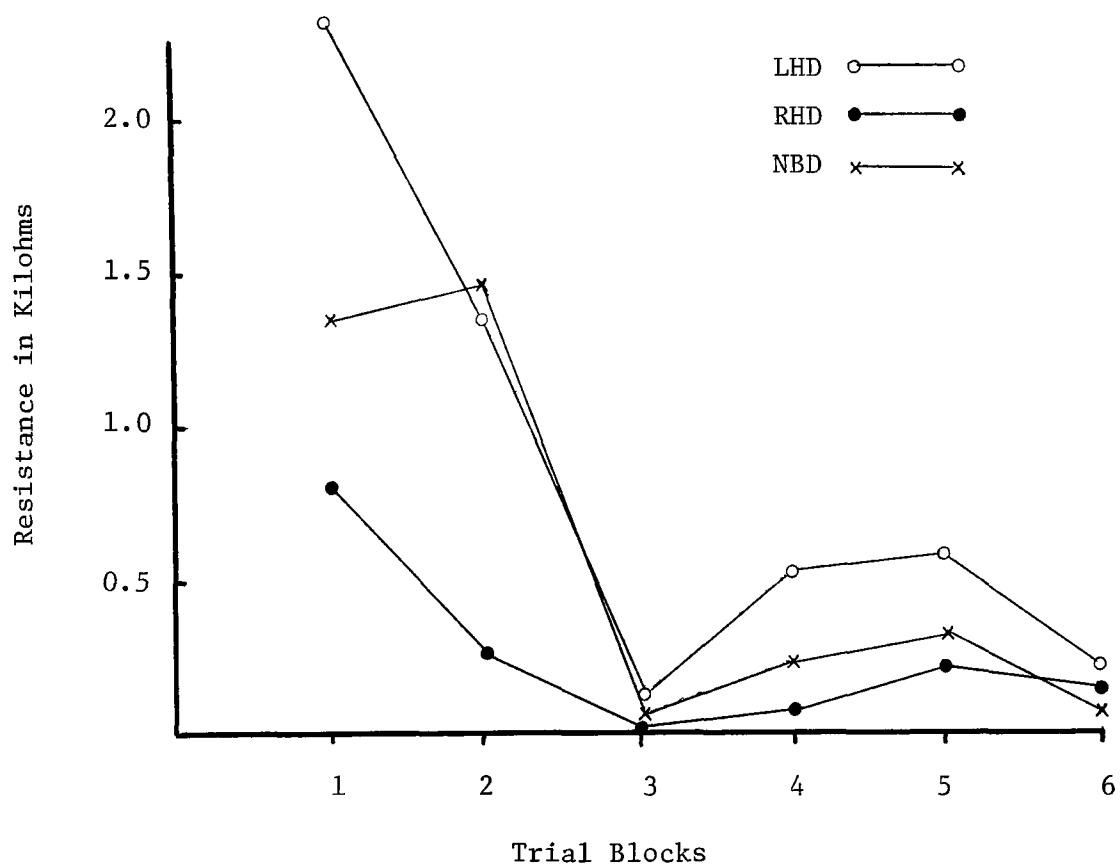


Figure 10. Trial blocks by response resistance for right-hand response resistance data.

resistance for any individual trial blocks; therefore, the fourth null hypothesis, that there is no difference between groups for rate of habituation of the OR, was not rejected for response resistance data.

Returning to the correlation matrix in Table 5, it was found that left-minus-right response resistance measures were positively correlated with both right-hand finger-tapping scores ($r = .45$, $df = 19$, $p < .05$) and left-minus-right pegboard scores ($r = .44$, $df = 19$, $p < .05$). The first correlation that, as the bilateral asymmetry increased for response resistance, the finger-tapping scores for the right hand also tended to increase, indicating a higher level of motor performance with greater electrodermal asymmetry. The second correlation indicated that, as the bilateral asymmetry increased for response resistance, the bilateral asymmetry for performance on the pegboard test also increased. The second correlation was consistent with the prediction that left-right asymmetry in electrodermal measures would follow that of motor performance measures. Left-hand and right-hand response resistance measures did not significantly correlate with any of the motor performance measures.

Response conductance data

Response amplitudes were quantified in conductance units and the various analyses for response data were repeated. Results for response conductance were essentially the same as for response resistance data.

Table 9 is a summary of the 3x6, groups by trial blocks, analysis of variance for left-minus-right response conductance data. There was no significant main effect for groups ($F = 1.33$, $df = 2/18$, $p > .05$); therefore, the third null hypothesis, that there is no difference between groups for left-minus-right scores of response amplitude, was not rejected for response

Table 9
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Minus-Right Response Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	6.122	2	3.061	1.330
Subjects within groups	41.418	18	2.301	
<u>Within subjects</u>				
B (trial blocks)	0.585	1	0.117	0.813
AB	1.115	2	0.112	0.775
Bx subjects within groups	12.950	18	0.144	

conductance data. There was no significant main effect across trial blocks ($F = 0.81$, $df = 1/18$, $p > .05$), indicating that the magnitude of asymmetry for all three groups did not change significantly over trial blocks.

Figure 11 illustrates group means across trial blocks for left-minus-right response conductance data. While the differences between groups were not found to be significant, the directions of these differences are consistent with the prediction that conductance would be higher (resistance would be lower) on the side contralateral to the damaged hemisphere.

Table 10 is a summary of the analysis of variance for left-hand response conductance. There was no main effect for groups ($F = 0.34$, $df = 2/18$, $p > .05$), indicating that left-hand response conductance could not differentiate the three groups. There was a significant main effect for trial blocks ($F = 6.66$, $df = 1/18$, $p < .05$), indicating a significant change in response amplitude across trial blocks. Tukey post hoc tests indicated a significant difference between the means of trial blocks one and three ($q = 6.27$, $p < .01$), one and four ($q = 5.88$, $p < .01$), one and five ($q = 5.68$, $p < .01$), and one and six ($q = 5.88$, $p < .01$). The decrease in response amplitude across was attributed to habituation to the series of stimuli. Figure 12 illustrates the habituation of response conductance for left-hand data. Response amplitudes were highest in the first trial block, approaching zero by the third trial block (stimuli 10-15).

Table 11 is a summary of the analysis of variance for right-hand response conductance data. There was no main effect for groups ($F = 0.69$, $df = 2/18$, $p > .05$), indicating that right-hand response conductance could not differentiate the three groups. There was a significant main effect for trial blocks ($F = 5.79$, $df = 1/18$, $p < .05$), although an F_{\max} statistic

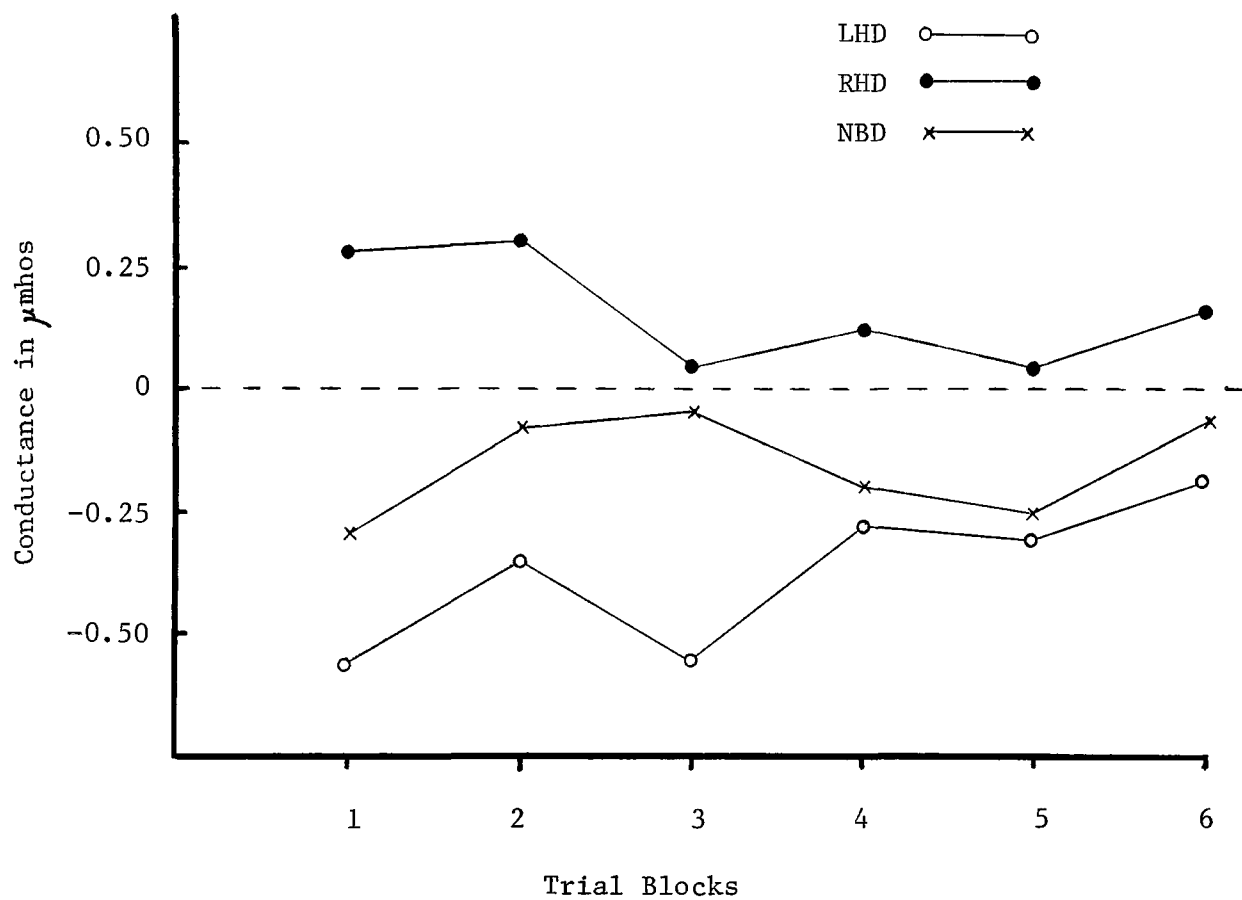


Figure 11. Trial blocks by left-minus-right response conductance.

Table 10
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Hand Response Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	0.700	2	0.350	0.342
Subjects within groups	18.403	18	1.022	
<u>Within subjects</u>				
B (trial blocks)	5.540	1	1.108	6.664*
AB	1.498	2	0.150	0.901
Bx subjects within groups	14.965	18	0.166	

*p < .05

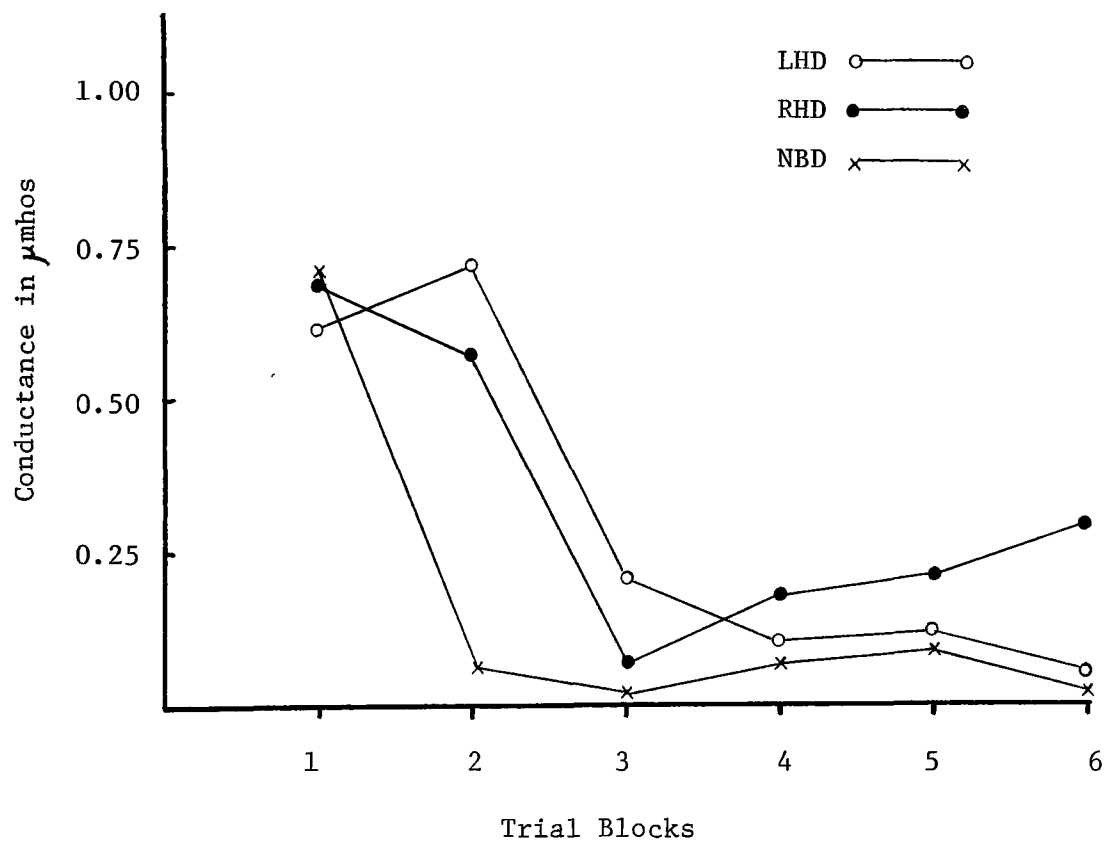


Figure 12. Trial blocks by response conductance for left-hand response conductance data.

Table 11
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Right-Hand Response Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	5.588	2	2.794	0.692
Subjects within groups	72.696	18	4.039	
<u>Within subjects</u>				
B (trial blocks)	6.620	1	1.324	5.792*
AB	3.310	2	0.331	1.448
Bx subjects within groups	20.574	18	0.229	

* $p < .05$

indicated that the assumption of homogeneity of variance had been violated for this analysis ($F_{\max} = 3.81$, $k = 6$, $df = 20$, $p < .05$). The analysis of variance was repeated on the same data after it had been normalized using a square-root transformation. Results indicated that the main effect for trial blocks remained significant ($F = 5.85$, $df = 2/18$, $p < .05$). Tukey post hoc tests indicated a significant difference between the means of trial blocks one and three ($q = 5.45$, $p < .01$), one and four ($q = 5.86$, $p < .01$), one and five ($q = 5.06$, $p < .01$), and one and six ($q = 6.63$, $p < .01$). The decrease in response amplitude across trial blocks was again attributed to habituation to the series of stimuli. Figure 13 illustrates the habituation of response conductance across trial blocks for right-hand data.

The three groups did not differ significantly in the rate of habituation to the OR as indicated by both left- and right-hand response conductance data. The groups by trial blocks interaction was significant for neither left-hand data ($F = 0.90$, $df = 2/18$, $p > .05$) nor right-hand data ($F = 1.45$, $df = 2/18$, $p > .05$). This finding indicated that groups did not differ significantly in response conductance for any individual trial block; therefore, the fourth null hypothesis, that there is no difference between groups in the rate of habituation to the OR, was not rejected for response conductance data.

Returning to the correlation matrix in Table 5, the only significant correlation for response conductance data was found between left-minus-right response conductance scores and left-minus-right dynamometer scores ($r = -.44$, $df = 19$, $p < .05$). This correlation indicated that, as bilateral differences in response conductance increased, bilateral differences in dynamometer scores decreased significantly. Subjects with large bilateral

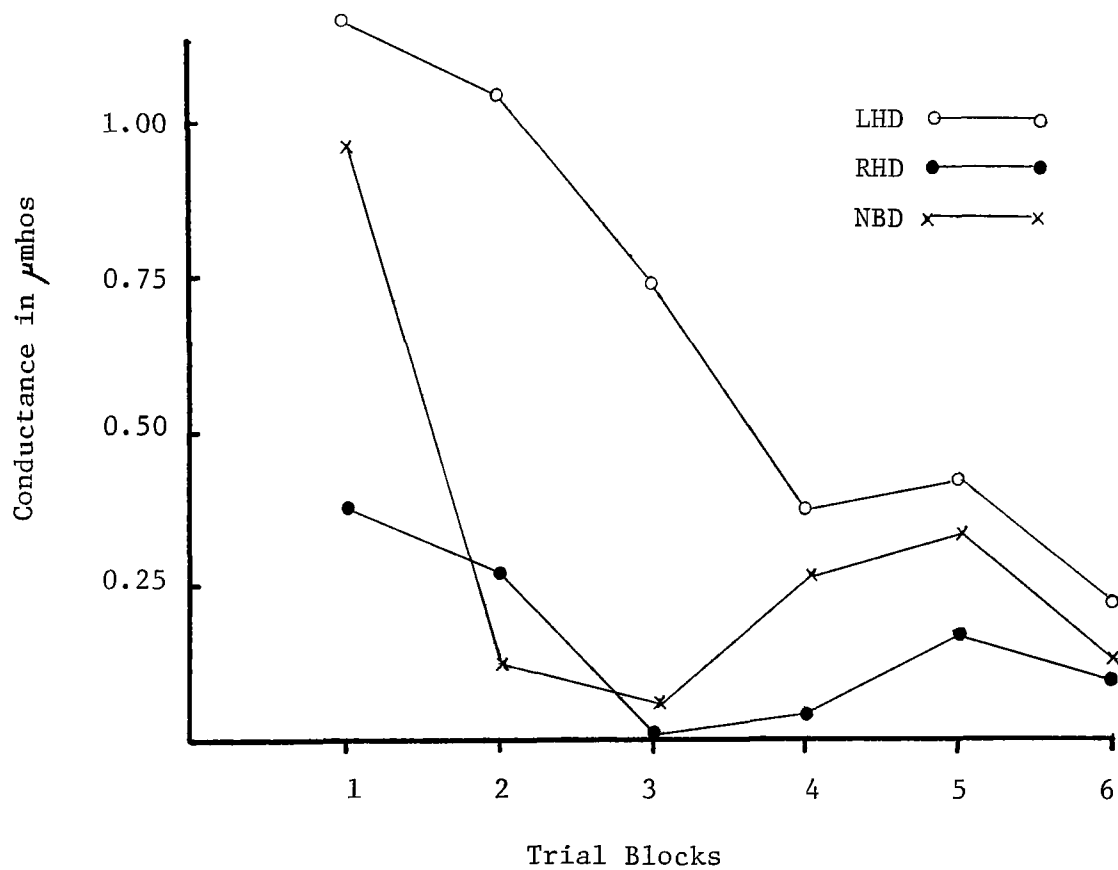


Figure 13. Trial blocks by response conductance for right-hand response conductance data.

differences in response conductance tended to have smaller bilateral differences in motor performance on the dynamometer grip-strength test. This finding was inconsistent with the prediction that electrodermal asymmetry would follow the asymmetry in motor performance.

Response log conductance data

Response amplitudes were quantified in response log conductance units and the various analyses for response data were repeated. Results for response log conductance were essentially the same as for response resistance.

Table 12 is a summary of the 3x6, groups by trial blocks, analysis of variance for left-minus-right response log conductance data. There was no significant main effect for groups ($F=0.72$, $df=2/18$, $p>.05$); therefore, the third null hypothesis, that there is no difference between groups for left-minus-right scores of response amplitude, was not rejected for response log conductance data. There was no significant main effect across trial blocks ($F=1.22$, $df=1/18$, $p>.05$), indicating that the magnitude of bilateral asymmetry did not change significantly across trial blocks. Figure 14 illustrates group means across trial blocks for left-minus-right response log conductance.

Table 13 is a summary of the analysis of variance for left-hand response log conductance data. There was no significant main effect for groups ($F=0.08$, $df=2/18$, $p>.05$), indicating that left-hand response log conductance could not differentiate the three groups. There was a significant main effect for trial blocks ($F=5.55$, $df=1/18$, $p<.05$), although an F_{\max} statistic indicated that the assumption of homogeneity of variance had been violated for this analysis. The analysis of variance was repeated on the same data after it had been normalized using a square-root transformation. Results indicated

Table 12
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Minus-Right Response Log Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	0.000139	2	0.0000696	0.718
Subjects within groups	0.001744	18	0.0000969	
<u>Within subjects</u>				
B (trial blocks)	0.000250	1	0.0000501	1.220
AB	0.000172	2	0.0000172	0.418
Bx subjects within groups	0.003694	18	0.0000410	

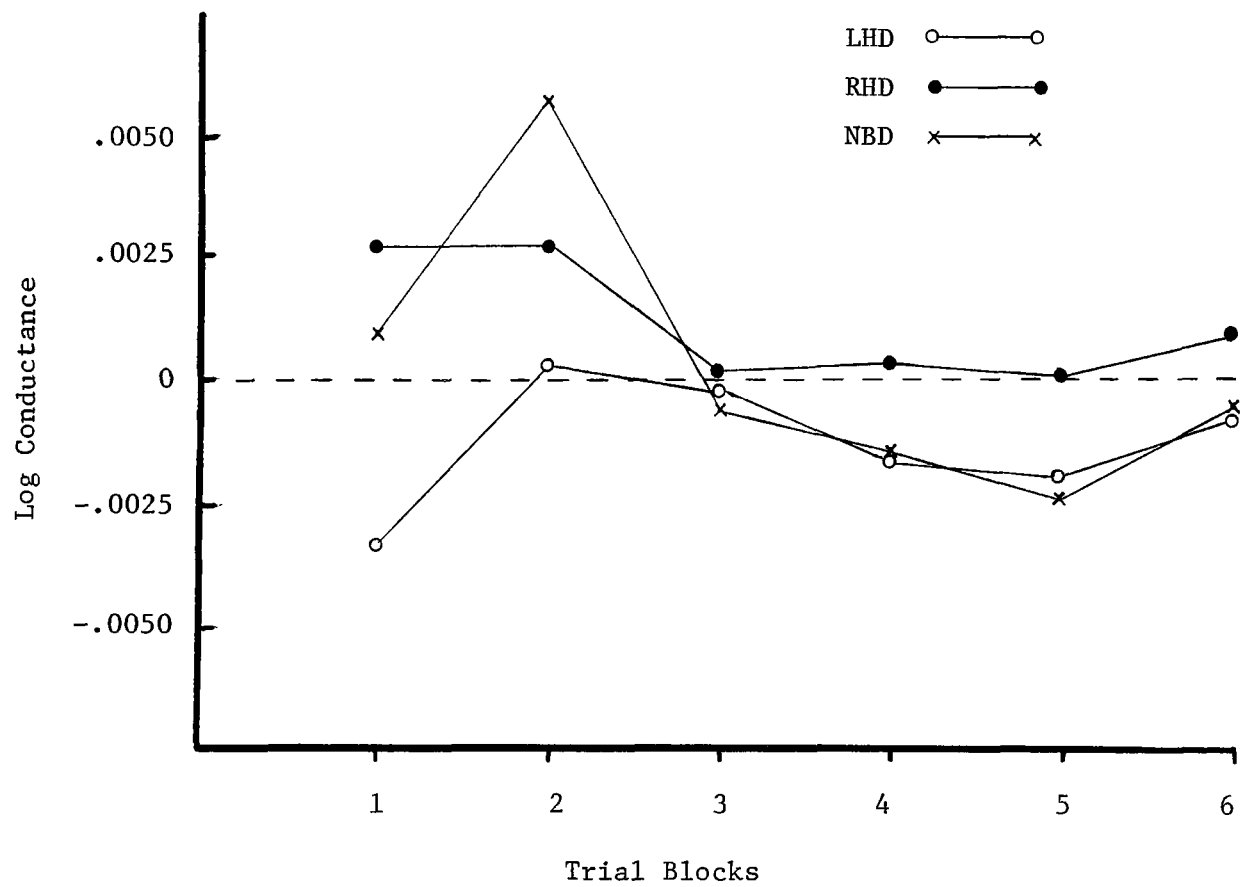


Figure 14. Trial blocks by left-minus-right response log conductance.

Table 13
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Left-Hand Response Log Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	0.0000372	2	0.0000186	0.077
Subjects within groups	0.0043474	18	0.0002415	
<u>Within subjects</u>				
B (trial blocks)	0.0023618	1	0.0004724	5.546*
AB	0.0002191	2	0.0000219	0.257
Bx subjects within groups	0.0076655	18	0.0000852	

*
 $p < .05$

that the main effect across trial blocks remained significant ($F=5.54$, $df=1/18$, $p < .05$). Tukey post hoc tests indicated a significant difference between the means of trial blocks one and three ($q=5.68$, $p < .01$), one and four ($q=5.31$, $p < .01$), one and five ($q=5.11$, $p < .01$), and one and six ($q=5.45$, $p < .01$). The decrease in response log conductance across trial blocks was attributed to habituation to the series of stimuli. Figure 15 illustrates the habituation of response log conductance across trial blocks for left-hand data.

Table 14 is a summary of the 3x6 analysis of variance for right-hand response log conductance data. There was no significant main effect for groups ($F=0.89$, $df=2/18$, $p > .05$), indicating that right-hand response log conductance data could not differentiate the three groups. There was a significant main effect for trial blocks ($F=9.55$, $df=1/18$, $p < .01$), although an F_{\max} statistic indicated that the assumption of homogeneity of variance had been violated for this analysis ($F_{\max}=4.02$, $k=6$, $df=20$, $p < .05$). The analysis of variance was repeated for the same data after it had been normalized using a square-root transformation. Results indicated that the main effect for trial blocks remained significant ($F=9.54$, $df=1/18$, $p < .01$). Tukey post hoc tests indicated a significant difference between the means of trial blocks one and two ($q=5.19$, $p < .01$), one and three ($q=8.27$, $p < .01$), one and four ($q=7.21$, $p < .01$), one and five ($q=6.61$, $p < .01$), and one and six ($q=8.04$, $p < .01$). The decrease in amplitude across trial blocks was again attributed to habituation to the series of stimuli. Figure 16 illustrates the habituation of response log conductance across trial blocks for right-hand data.

The three groups did not differ significantly in the rate of habituation

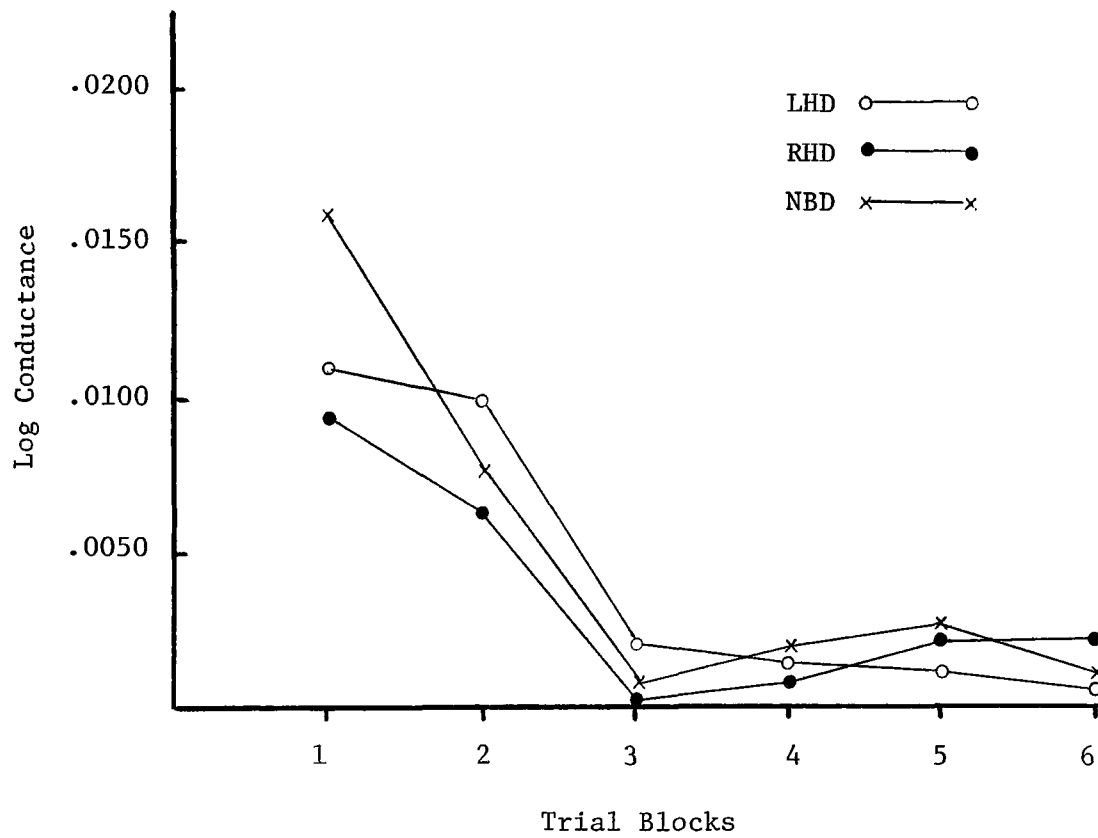


Figure 15. Trial blocks by response log conductance for left-hand response log conductance data.

Table 14
 Summary of the 3x6, Groups by Trial Blocks, Analysis
 of Variance for Right-Hand Response Log Conductance

Source of Variation	SS	df	MS	F
<u>Between subjects</u>				
A (groups)	0.0002534	2	0.0001267	0.888
Subjects within groups	0.0025687	18	0.0001427	
<u>Within subjects</u>				
B (trial blocks)	0.0017774	1	0.0003555	9.550**
AB	0.0004039	2	0.0000404	1.085
Bx subjects within groups	0.0033499	18	0.0000372	

**
 $p < .01$

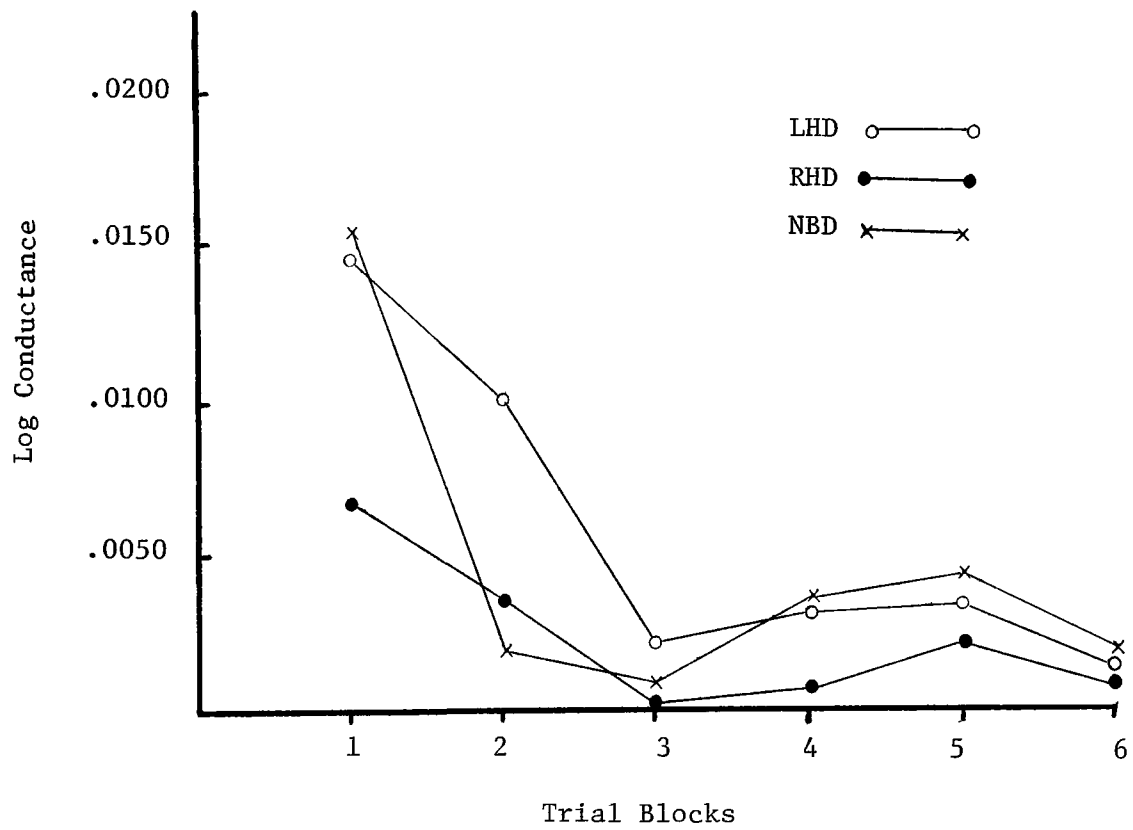


Figure 16. Trial blocks by response log conductance for right-hand response log conductance data.

to the OR as indicated by both left- and right-hand response log conductance measures. The groups by trial blocks interaction was significant for neither left-hand data ($F=0.26$, $df=2/18$, $p>.05$) nor right-hand data ($F=1.09$, $df=2/18$, $p>.05$). This finding indicated that groups did not differ significantly in response log conductance for any individual trial block; therefore, the fourth null hypothesis, that there is no difference between groups for rate of habituation to the OR, was not rejected for response log conductance data.

Returning to the correlation matrix in Table 5, no significant correlation was found between measures of response log conductance and any of the motor performance measures.

Age and time from CVA

The correlation matrix in Table 5 contained two variables in addition to those of electrodermal activity and motor performance. It was found that age, the first variable, did not correlate significantly with any of the electrodermal or motor performance variables. The second variable, time from CVA, was the length of time in months that had elapsed since the cerebrovascular accident. Time from CVA was found to correlate significantly with both right-hand finger-tapping scores ($r=-.54$, $df=12$, $p<.05$) and right-hand dynamometer scores ($r=-.53$, $df=12$, $p<.05$). These correlations indicated that right-hand performance on tests of speed and strength tended to be poorer for those subjects having longer durations since the cerebrovascular episode.

Law of Initial Values

To determine whether or not LIV effects were working in the present study, the response amplitude of the first OR was correlated with the basal

level preceding that response. The computation was done for response units of resistance, conductance, and log conductance. Results indicated that response resistance did not correlate significantly with basal resistance ($r = .12$, $df = 40$, $p > .05$), indicating that LIV effects did not influence measurements of response amplitude to a significant degree. Response conductance was found to be significantly correlated with basal conductance ($r = .74$, $df = 40$, $p < .01$), indicating that response amplitude tended to increase with basal conductance levels. The Law of Initial Values, however, would have predicted a negative correlation between response amplitude and basal level such that response conductance would decrease as basal levels increased. While the present finding was inconsistent with predictions based on LIV effects, basal conductance levels appear to have influenced response amplitudes in conductance units. Response log conductance did not correlate significantly with basal log conductance ($r = .09$, $df = 40$, $p > .05$), indicating no LIV effects for log conductance response measures.

Equivalence of transformation for response data

To determine the equivalence of the transformations of resistance units into conductance and log conductance units, correlations were computed between the three measures for left- and right-hand data. For left-hand data, resistance was found to be significantly correlated with conductance ($r = .56$, $df = 628$, $p < .01$) and with log conductance ($r = .91$, $df = 628$, $p < .01$). Conductance was found to be significantly correlated with log conductance ($r = .74$, $df = 628$, $p < .01$). For right-hand data, resistance was not significantly correlated with conductance ($r = .06$, $df = 628$, $p > .05$) although it was found to be significantly correlated with log

conductance ($r = .79$, $df = 628$, $p < .01$). Conductance was found to be significantly correlated with log conductance ($r = .52$, $df = 628$, $p < .01$). These correlations indicated that the application of various transformations in electrodermal research may not always produce equivalent measures, as in the case of the resistance to conductance transformation for right-hand data. The methodological significance of these findings is considered in more detail in chapter V.

Summary of results

The results of the present study indicated that the following null hypotheses cannot be rejected:

- (1) There is no difference between groups for left-minus-right difference scores for basal skin resistance.
- (2) There is no significant correlation between left-minus-right difference scores for basal skin resistance and left-minus-right difference scores for motor performance measures.
- (3) There is no difference between groups for left-minus-right difference scores for response resistance, conductance, or log conductance.
- (4) There is no difference between groups for the rate of habituation of the OR.

The three groups did not differ significantly for any electrodermal measure (left-hand, right-hand, or left-minus-right hand measures of basal skin resistance, response resistance, response conductance, or response log conductance).

From the correlation matrix between all electrodermal measures and all motor performance measures, the following correlations were found to be significant at $p < .05$:

- (1) L-R response resistance : R finger-tapping scores, $r = .45$.
- (2) L-R response resistance : L-R pegboard scores, $r = .44$.
- (3) L-R response conductance : L-R dynamometer scores, $r = -.44$.
- (4) Time from CVA : R finger-tapping scores, $r = -.54$.
- (5) Time from CVA : R dynamometer scores, $r = -.53$.

CHAPTER V

DISCUSSION

The results of the present study, in general, do not support the predictions stated in the second chapter and this discrepancy is considered, in detail, in this chapter. In addition, the bilateral electrodermal data obtained in the present study from normal subjects are compared with those of other studies. Finally, the methodological implications of results concerning the equivalence of transformations for response data are discussed.

Bilateral electrodermal activity and motor performance

The formal hypotheses of the present study were derived from the basic prediction that in unilaterally brain-damaged subjects the bilateral asymmetry of motor performance would be paralleled by a similar bilateral asymmetry in electrodermal activity. Unilateral brain damage, as in the case of hemiplegia, has been shown to produce partial or complete paralysis of the musculature on the body side contralateral to the damaged hemisphere, thereby producing a bilateral asymmetry in motor performance. Similarly, the damage has been shown to produce profuse sweating and lower basal skin resistance on the contralateral body side, thereby producing a bilateral asymmetry in an electrodermal measure (the literature is inconclusive concerning asymmetry of response amplitude). Since the premotor area of the cerebral cortex has been shown to subserve both functions, it was assumed that an improvement in one function (due to the recovery of the premotor cortex) would be paralleled by improvement in the other function. In other words, both degrees of pathological bilateral asymmetry should approach the limits of normal bilateral asymmetry in parallel. The results of the present study, however, did not support this general

prediction.

It was predicted that the two brain-damaged groups (LHD and RHD) would exhibit greater bilateral asymmetry in basal skin resistance than the control group (NBD), presumably because of lower resistance levels on the side contralateral to the damaged hemisphere. It was also suggested that these two groups could differ from the control group in response amplitude and rate of habituation for the OR, although the literature did not warrant a specific hypothesis concerning the direction of these differences. Results, however, indicated that there were no significant differences between groups for any of the electrodermal measures. These included left-hand, right-hand, and left-minus-right hand measures of basal skin resistance, response resistance, response conductance, and response log conductance.

The finding that the three groups did not differ significantly with respect to response amplitude (in resistance, conductance, and log conductance units) and rate of habituation for the electrodermal component of the OR were consistent with the findings of Holloway and Parsons (1971).

The finding that groups did not differ significantly in the asymmetry of basal skin resistance at first appears inconsistent with the findings of Holloway and Parsons (1969), who found greater asymmetry of skin conductance levels in brain-damaged subjects. In addition, the sodium amytal studies also demonstrated profuse sweating and lowered resistance levels on the body side contralateral to the deactivated cerebral hemisphere. However, Holloway and Parsons (1969) recorded skin conductance levels from patients who had suffered the cerebrovascular episode a relatively short time prior to the recording sessions. While figures concerning the exact length of time elapsed since the onset of the disorders

are not available, all subjects were still hospitalized and none had recovered sufficiently to permit rehabilitative therapy. Similarly, the results of the sodium amytal studies were obtained by means of an acute preparation. The effects of an injection of sodium amytal into an internal carotid artery were immediate and only lasted a matter of minutes. In the present study the time elapsed between the CVA and the recording session varied from 4 to 31 months, with a mean of 14.6 months for LHD and 16.7 months for RHD. Presumably, these subjects had recovered to some extent from the vascular lesions before the recording sessions.

The finding that both LHD and RHD subjects did not differ significantly from NBD subjects in the asymmetry of bilateral basal skin resistance suggests that, at least for electrodermal functions and for the OR, the brain-damaged subjects had recovered completely prior to the recording sessions. However, all brain-damaged subjects at the recording sessions showed some degree of motor impairment on the hemiplegic side, with some subjects exhibiting almost complete paralysis; therefore, it would appear that, while electrodermal functions appeared to be fully recovered, motor functions were still severely impaired. This finding tends to refute the proposal that electrodermal and motor functions recover in parallel following unilateral brain damage.

One possible alternative has been suggested to account for this discrepancy. McComas, Sica, Upton, Aguilera, and Currie (1971) have demonstrated that the nature of the motor dysfunction in hemiplegia changes markedly during the course of the affliction. In hemiplegic patients an estimate was made of the number of functioning motor units in a particular muscle group in the leg. A motor unit includes a

spinal motoneuron whose cell body is located in the anterior horn of the spinal cord, its peripheral axon, and the muscle fibers innervated by that axon. The technique involved the comparison of the muscle action potentials evoked by stimulating single motor units and then the whole muscle. Results indicated that in patients in whom the onset of the hemiplegia had occurred less than two months previously, there was a normal number of motor units in the muscle group of the hemiplegic leg. In patients with hemiplegia for three months or longer there was an abnormally small number of motor units for the muscle group in the affected leg as compared to the normal leg. The loss of motor units was found to have occurred rather abruptly, between two and three months after the CVA, but it did not progress further. The magnitude of the decrease in motor units was the same for patients with hemiplegia varying from four months to over ten years.

In the present study the time elapsed since the onset of the CVA was found to be negatively correlated with right-hand scores of both finger-tapping and dynamometer grip-strength. This indicated that, for subjects who had been afflicted for longer periods of time, the performance of the right hand tended to be poorer for these two tests. Since the number of motor units lost was shown to be relatively constant after four months, this progressive deterioration in performance was probably due to other factors (motivation, loss of strength and speed through disuse, etc.).

McComas et al. (1971) found that, within the anterior horn of the spinal cord, a number of motoneurons continued to function and their axons, through hypertrophy and/or collateral sprouting, reinnervated the muscle fibers of the degenerated motor units to some extent. The authors maintained that the motor units were lost as a result of transneuronal degeneration. The central nervous system lesion (in this case in the

cerebral cortex) deprived lower neurons and muscles of a trophic influence which would be normally exerted by motor cortex. This upper motoneuron lesion could cause secondary changes in lower motoneurons which, in turn, would affect muscles. This trophic influence could be either a particular pattern of electrochemical input from one neuron to another or a neurochemical substance transmitted via axoplasmic flow down the axon of one neuron to another.

These findings challenged the most widely held opinion that trans-neuronal degeneration and muscle wasting resulted from disuse only. That a number of cells in the spinal cord continued to function, even after three months of disuse, indicated that they were presumably less dependent on these trophic influences from cortical sources than were those neurons which degenerated.

According to this study (McComas, et al., 1971) the motor dysfunction seen in the first two months of hemiplegia is due to vascular lesions of the cerebral cortex and the subsequent interference with normal central nervous system control. After three or four months the dysfunction is presumably the result of lost motor units which, in turn, results from the termination of some trophic influence from higher up in the central nervous system. This would account for the discrepancy between Holloway and Parsons (1969) and the present study. Their findings of large bilateral differences in skin conductance levels in recently afflicted hemiplegic patients can be seen as a result of cortical dysfunction releasing reticular activating centers from normal tonic inhibition, thereby producing profuse sweating and higher conductance levels (lower resistance levels) on the contralateral body side. In the present study

it is conceivable that the normal bilateral differences in basal skin resistance seen in hemiplegic patients having experienced the CVA some 4 to 31 months previously reflects recovery of function in the cerebral motor cortex, but that the decrease in motor units resulting from transneuronal degeneration prevents this recovery of motor cortex from being expressed as the improvement of motor performance. In other words, the recovery of bilateral electrodermal measures may parallel recovery in motor cortex, but not in motor performance due to transneuronal degeneration in the periphery. McComas et al. (1971) maintained that, "in spite of attempts to re-educate patients in the use of hemiplegic limbs, the upper motoneurone lesion leaves a legacy in the cord such that any alternative descending motor pathways find fewer functioning lower motoneurons to play on."

It was assumed in the present study that transneuronal influences on the descending pathways were due to disuse; therefore, transneuronal degeneration would affect neurons in the anterior horn of the spinal cord somewhat indiscriminately. In other words, sympathetic neurons controlling electrodermal activity in the skin would be affected to the same degree as spinal motoneurons controlling motor performance. However, trophic influences which discriminate among different motoneurons could conceivably discriminate between spinal motoneurons and spinal sympathetic neurons. It is possible that spinal sympathetic neurons do not suffer any transneuronal degeneration due to continued input (and, presumably, continued trophic influences) from the hypothalamus.

This alternative explanation would account for the lack of proposed correlation between bilateral differences in basal skin resistance and

and differences in motor performance measures. It is apparent that the two functions did not recover concurrently.

Two correlations indicated that bilateral asymmetry in response resistance correlated positively with both bilateral differences in pegboard scores and right-hand finger-tapping scores. The first correlation indicates that, as the asymmetry of response resistance increases, the subjects tend to have a greater dissimilarity between the left and right hands for pegboard scores. This is consistent with the prediction that electrodermal asymmetry would correlate with motor performance asymmetry. The second correlation indicates that, as left-right differences in response resistance increase, subjects tend to have higher right-hand finger-tapping scores (improved performance). This is inconsistent with the original prediction. Bilateral asymmetry in response conductance was found to be negatively correlated with bilateral asymmetry in dynamometer scores. This indicates that, as left-right differences in response conductance increase, subjects tend to have less asymmetry for dynamometer scores. This is also inconsistent with the original prediction.

It is conceivable that all three correlations were spurious. In an 11x14 correlation matrix it is possible for 3 of the 154 resulting correlations to be significant by chance alone. In addition, if the significance of these correlations reflects some trend in the data, it is inconsistent for the first correlation to oppose the second and third in interpretation. Similarly, while the first correlation bears out the prediction that some electrodermal and motor performance asymmetries vary concurrently, the findings of the analyses of variance indicate that the brain-damaged groups did not differ significantly from the control

group in electrodermal measures. The correlation would then seem not to indicate that in hemiplegic patients electrodermal and motor functions recover from pathology concurrently, but that one set of measures can be used to predict the other in non-brain-damaged subjects as well as brain-damaged subjects. Finally, all three correlations were barely significant at the .05 level of significance. To rule out the possibility of spurious correlation, significance at a somewhat higher level of probability would be more desirable.

A second possible explanation for the failure to find significant differences between groups in electrodermal measures could involve the accuracy of the anatomical-physiological mechanisms proposed to account for the contralateral effects of unilateral cortical ablation and stimulation. The theoretical pathways illustrated in Figures 1 and 2 are rather oversimplified and attempt to account for the physiological and behavioral evidence cited by various authors. Furthermore, some of the suggested pathways have not been convincingly demonstrated anatomically while others have been shown to include components which would suggest that unilateral control by a cerebral hemisphere is exerted bilaterally rather than contralaterally.

In Figure 1, for example, fibers from the premotor cortex are shown descending through the brain stem either to synapse in the ipsilateral lateral reticular formation or to bypass this nucleus, to continue directly to the anterior horn of the spinal cord as corticospinal fibers. All fibers are shown crossing to the contralateral side in the decussation of the pyramids. This theoretical descending pathway accounts for the experimental findings of various authors cited in chapter II (Schwartz, 1937;

Wall & Davis, 1951; Wang, 1964; Wang & Lu, 1930; Wilcott, 1969).

Stimulation of premotor cortex would elicit electrodermal responses from the contralateral body side and ablation of this cortex would release the lateral reticular formation from tonic inhibitory input, thereby producing profuse sweating and lower basal resistance on the contralateral side. This would explain the findings of Holloway and Parsons (1969) and of the sodium amytal studies.

However, certain alternative anatomical models may be suggested. For example, while most corticospinal fibers cross to the contralateral body side in the decussation of the pyramids, about 15% of these fibers remain ipsilateral as the ventral corticospinal tract (Barr, 1972). Corticoreticular fibers may end in reticular nuclei other than the lateral reticular nuclei and some of these other nuclei (e.g. ventromedial reticular nuclei) are known to be inhibitory sweat centers (Wang, 1964). Finally, reticulospinal fibers have been shown to be uncrossed as well as crossed (Brodal, 1969). These findings suggest that the descending pathways from premotor cortex have bilateral as well as contralateral components and that inhibitory as well as excitatory sweat centers may be disinhibited following cortical lesions.

Similar anatomical alternatives can be suggested for the theoretical descending pathway illustrated in Figure 2. While there are direct corticohypothalamic fibers (as illustrated), most cortical input to the hypothalamus enters via the entorhinal area of the hippocampus in the case of limbic cortex and via the medial thalamic nucleus in the case of prefrontal cortex. These connections appear to be predominantly ipsilateral (Brodal, 1969). While the hypothalamus has been shown to be an excitatory

sweat center (Wang, 1964), its descending connections to the spinal cord are predominantly ipsilateral, no major contralateral projections having been documented (Brodal, 1969). While some degree of crossing could occur at the level of the cerebral cortex, the hypothalamus, and the spinal cord, there is still a major ipsilateral pathway (dorsal longitudinal fasciculus) with which to contend.

These alternative anatomical models suggest that one cerebral hemisphere, to some extent, exerts bilateral control over electrodermal functions and that this control is probably much more highly mediated than the mechanisms proposed in chapter II would indicate. If cortical control is bilateral, damage to one hemisphere would affect electrodermal activity on both sides; therefore, bilateral differences would not be expected to change significantly. This was the finding of the present study. However, the findings of Holloway and Parsons (1969) and of the sodium amytal studies suggest that the effects of unilateral dysfunction in the cerebral cortex are indeed contralateral. One can assume that, while anatomical evidence suggests bilateral control of electrodermal activity by either hemisphere, multiple interactions in the central nervous system in response to injury produce an overall effect on electrodermal activity which is seen on the contralateral body side. Therefore, if one accepts this inaccuracy in the anatomical models as the reason for the failure to find significant differences between groups in electrodermal activity in this study, then the findings of Holloway and Parsons (1969) and of the sodium amytal studies appear incongruent with the present findings. Since the first alternative explanation presented in this chapter (the reduction of peripheral motor units) encompasses the

findings of previous studies as well as those of the present study, it should be preferred to the second alternative.

Normal bilateral asymmetry in electrodermal activity

Studies concerned with the bilateral differences in basal skin resistance in normal subjects have reported predominantly higher levels of basal resistance (or the equivalent in skin conductance or potential) on the left side (Obrist, 1963; Wyatt & Tursky, 1969), on the right side (Baitsch, 1954; Fisher, 1958), and on the left and right sides with relatively equal frequency (Crocco, 1974; Galbrecht, et al., 1965; Varni, et al., 1971). In the present study, basal resistance levels were found to be greater on the left and right sides with relatively equal frequency in the seven normal subjects. These left-right differences were even seen to reverse themselves within the 30-minute recording session in some cases, a finding reported by other authors (Crocco, 1974; Obrist, 1963). However, mean values for left-minus-right scores of basal resistance were consistently positive across trial blocks for NBD subjects (see Figure 5), indicating that the bilateral differences were clearly larger when the left-hand levels were greater than the right-hand levels.

Similar results were obtained for response amplitudes. While response resistance was found to be greater on the left and right sides with relatively equal frequency, the mean left-minus-right score for the first trial block (stimuli 1-5) was positive (see Figure 8). This indicates that bilateral differences in response amplitude to the first five stimuli were larger when the left-hand amplitudes were greater than the right-hand amplitudes. The difference scores for the remaining trial blocks were close to zero, indicating roughly equal left and right amplitudes for

stimuli 6-30. Similarly, Crocco (1974) found higher response resistance on the left side.

A satisfactory explanation of normal bilateral differences in electrodermal activity has not yet been proposed. Wyatt and Tursky (1969) have suggested that higher left-hand resistances (lower conductance and potential) could result from differences in hemisphere dominance. In both right- and left-handed people, the left hemisphere may be dominant for speech in over 90% of the population (Penfield & Roberts, 1959); possibly, this dominance could reflect a lower right-hand resistance due to the inhibitory nature of cortical influences on subcortical structures and to the presumably contralateral expression of that control. This model, however, does not explain reversals of left-right differences within a recording session or instances where left-hand resistance levels are less than the right-hand levels.

It is possible that left-right differences in basal or response resistance and the occasional reversal of these differences may reflect changes in the level of activity between the two cerebral hemispheres. Goldstein, Stoltzfus, and Gardocki (1972) have demonstrated shifts between the left and right cerebral hemispheres in the overall amplitude of activity during various stages of sleep in man. Using the technique of integrative EEG analysis, they have shown that, with each occurrence of a shift from a period of slow wave sleep (NREM) to a period of rapid-eye movement sleep (REM), a similar shift occurs between the left and right hemispheres in the overall level of activity. A lower amplitude of activity in the left hemisphere (relative to the right hemisphere) was found during NREM sleep, while a higher amplitude was found during REM

sleep. The authors maintained that the difference in hemispheric activity relationships during NREM and REM sleep could prove to be a neurophysiological concomitant of the changes both in brain function and in the quality of dreams reported during these stages. Similarly, left-right differences in resistance levels could reflect differences in the amplitudes of activity between the two hemispheres. Shifts in left-right differences in resistance levels could also reflect shifts between hemispheres in the amplitudes of overall activity during the waking state.

Equivalence of transformations for response data

Response conductance and response log conductance were found to be significantly correlated with measures of response resistance for left-hand data. For right-hand data, conductance was not significantly correlated with resistance, although log conductance was significantly correlated with resistance. It would appear that, at least for right-hand data, the resistance-to-conductance transformation did not produce equivalent sets of data. While the two sets of data were related in direction, magnitude differences were not equivalent as indicated by non-significant correlations. This would appear to be due to the non-linear transformation required to convert resistance units into conductance units.

Using a constant-current technique, the change in resistance in response to a stimulus was measured directly. This change in resistance was converted into a change in conductance using the following formula:

$$1/R_b - 1/R_m \approx C_r$$

A lack of precision results from this transformation, especially when the basal resistances used in the formula are low. According to Montagu

and Coles (1966):

With the constant current method, conductance values are calculated from resistance measurements. The inverse relationship between these functions has certain effects which are worth noting. Since a small conductance is measured as a large resistance, the high resistance end of the scale becomes contracted during the process of reciprocation. Conversely, the low resistance end becomes expanded. As a result, a small error in the measurement of low resistances is magnified when these are expressed in terms of conductance (p. 267).

This error in transformation would explain the differences in the findings for LIV correlations. The amplitude of the first response was correlated with the basal level preceding that response. Results indicated that, for the resistance data and the log conductance data, response amplitude did not correlate significantly with the preceding basal level; therefore, LIV effects did not significantly influence response amplitudes. For response conductance, however, there was a significant correlation, indicating that the response amplitude was, in part, determined by the skin conductance level. Since this influence was not detected in the original data, one may assume that the significant correlation for response conductance data resulted from error introduced in the transformation from resistance units into conductance units.

The lack of equivalence for various transformations of electrodermal data suggests the need for standardized techniques of recording and quantification of electrodermal measures. Lykken and Venables (1971) have suggested that conductance be measured directly using a constant voltage technique, rather than computing conductance measures indirectly from resistance data. Different techniques of recording and quantifying electrodermal measures do not necessarily demonstrate certain phenomena to the same degree. A standardized technique would increase the extent

to which results could be compared between laboratories.

CHAPTER VI

CONCLUSION

The results of the present study did not support the original prediction that, for unilaterally brain-damaged subjects, the bilateral asymmetries in electrodermal measures would vary concurrently with the bilateral asymmetries in motor performance measures. In other words, bilateral differences in electrodermal measures cannot be used to predict the improvement in motor performance for brain-damaged patients.

Results failed to reject the following null hypotheses:

- (1) There is no difference between groups for left-minus-right scores of basal skin resistance.
- (2) There is no significant correlation between left-minus-right scores of basal skin resistance and left-minus-right scores of motor performance measures.
- (3) There is no difference between groups for left-minus-right scores of response resistance, response conductance, or response log conductance.
- (4) There is no difference between groups for the rate of habituation for the electrodermal component of the OR.

The three groups (left-hemiplegics, right-hemiplegics, normal controls) did not differ significantly for any electrodermal measure (left-hand, right-hand, and left-minus-right hand measures of basal skin resistance, response resistance, response conductance, and response log conductance).

The large bilateral differences in basal skin resistance, usually seen in patients who have recently suffered unilateral brain damage, recovered to normal levels of bilateral asymmetry within, at most, four

months following the CVA for the 14 hemiplegic patients in the present study. All 14 subjects showed varying degrees of motor impairment, the most severe cases exhibiting almost complete paralysis of the hemiplegic side; therefore, while electrodermal functions appeared to recover completely, motor performance was still severely impaired.

The conclusion of the present study was that bilateral differences in electrodermal measures cannot be used to predict the recovery of motor performance in patients with unilateral brain damage. While bilateral differences in these measures may parallel the recovery of premotor and motor cortex following injury, transneuronal degeneration occurring at about the third month after the CVA reduces the number of available motor units in the periphery, thereby further impairing motor performance.

REFERENCES

- Baitsch, H. Uber Geschlechts-und Sutendifferenzen im "Niveau"-
Elektrodermatogramm. (Sex and lateral differences in electrographic
levels) Confinia Neurologica, 1954, 14, 88-100.
- Barr, M.L. The human nervous system. New York: Harper & Row, 1972.
- Brodal, A. Neurological anatomy. New York: Oxford University Press, 1969.
- Bruillova, S.V. On some aspects of the orienting reflex in persons having
suffered a covert trauma of the brain and in neurotic persons. In
Voronin, L.G., Leontiev, A.N., Luria, A.R., Sokolov, Ye.N., &
Vinogradova, O.S. (Eds.), Orienting reflex and exploratory behavior.
Washington: American Institute of Biological Sciences, 1965, Pp. 343-350.
- Burch, N.R., & Greiner, T.H. Drugs and human fatigue: GSR parameters.
Journal of Psychology, 1958, 45, 3-10.
- Campbell, D.T., & Stanley, J.C. Experimental and quasiexperimental designs
for research. Chicago: Rand McNally, 1963.
- Cowles, M.P. The latency of the skin resistance response and reaction time.
Psychophysiology, 1973, 10, 177-183.
- Crocco, D. Bilingual memory and the orienting reflex. Unpublished doctoral
dissertation, University of Ottawa, 1974.
- Darrow, C. The rationale for treating the change in galvanic skin response
as a change in conductance. Psychophysiology, 1964, 1, 31-38.
- Davidoff, R.A., & McDonald, D.G. Alpha blocking and autonomic responses
in neurological patients. Archives of Neurology, 1964, 10, 283-292.
- Diller, L. Psychomotor and vocational rehabilitation: Presentation II.
In Benton, A.L. (Ed.) Behavioral change in cerebrovascular disease.
New York: Harper & Row, 1970, Pp. 81-105.

- Edelberg, R. Electrical activity of the skin: Its measurement and uses in psychophysiology. In Greenfield, N.S., & Sternbach, R.A. (Eds.) Handbook of psychophysiology. New York: Holt, Rinehart, & Winston, 1972.
- Fisher, S. Body image and asymmetry of body reactivity. Journal of Abnormal and Social Psychology, 1958, 57, 292-298.
- Galbrecht, C.R., Dykman, R.A., Reese, W.G., & Suzuki, T. Intrasession adaptation and intersession extinction of the components of the orienting response. Journal of Experimental Psychology, 1965, 70, 585-597.
- Gaviria, B., Coyne, L., & Thetford, P.E. Correlation of skin potential and skin resistance measures. Psychophysiology, 1969, 5, 465-477.
- Germana, J. Rate of habituation and the law of initial values. Psychophysiology, 1968, 5, 31-36.
- Goldstein, L., Stoltzfus, N.W., & Gardocki, J.F. Changes in interhemispheric amplitude relationships in the EEG during sleep. Physiology and Behavior, 1972, 8, 811-815.
- Gullickson, G. Psychomotor and vocational rehabilitation: Presentation XII. In Benton, A.L. (Ed.) Behavioral changes in cerebrovascular disease. New York: Harper & Row, 1970, Pp. 106-110.
- Guttman, L., & List, C.F. Die nervosen leitungsbahnen der schweissekretion beim menschen. Deutsch Ztschr f. Nervenheik, 1928, 167, 61-71.
- Haggard, E. On the application of analysis of variance to GSR data: I. The selection of an appropriate measure. Journal of Experimental Psychology, 1949a, 39, 378-392.
- Haggard, E. On the application of analysis of variance to GSR data: II. Some effects of the use of inappropriate measures. Journal of

Experimental Psychology, 1949b, 39, 861-867.

Hasama, B. Pharmakologische und physiologische Studien über die Schweisszentren:

II. Über den Einfluss der direkten, mechanischen thermischen und elektrischen Reizung auf die Schweiss- sowie Warmezentren. Archiv für experimentelle Pathologie und Pharmakologie, 1929, 146, 129-161.

Holloway, F.A., & Parsons, O.A. Unilateral brain damage and bilateral skin conductance levels in humans. Psychophysiology, 1969, 6, 138-148.

Holloway, F.A., & Parsons, O.A. Habituation of the orienting reflex in brain damaged patients. Psychophysiology, 1971, 8, 623-634.

Illis, L.S. Experimental model of regeneration in the central nervous system: I. Synaptic changes. Brain, 1973a, 96, 47-60.

Illis, L.S. Experimental model of regeneration in the central nervous system: II. The reaction of glia in the synaptic zone. Brain, 1973b, 96, 61-68.

Isamat, F. Galvanic skin responses from stimulation of limbic cortex. Journal of Neurophysiology, 1961, 24, 176-181.

Johnson, L.C., & Lubin, A. The orienting reflex during waking and sleeping. Electroencephalography and Clinical Neurophysiology, 1967, 22, 11-21.

Kirk, R.E. Experimental design: Procedures for the behavioral sciences. Belmont, California: Wadsworth Press, 1968.

Kuno, Y. Human perspiration. Springfield, Illinois: Charles C. Thomas, 1956.

Luria, A., Pribram, K., & Homskava, E. An experimental analysis of the behavior disturbances produced by the left frontal arachnoidal endothelioma. Neuropsychologica, 1964, 2, 257-280.

Lykken, D., & Venables, P. Direct measurement of skin conductance: A

- proposal for standardization. Psychophysiology, 1971, 8, 656-672.
- Lynn, R. Attention, arousal and the orienting reaction. Oxford: Pergamon Press, 1966.
- MacLean, P.D. Limbic system in relation to central gray and reticulum of brain stem: Evidence of interdependence in emotional process. Psychosomatic Medicine, 1955, 17, 355-366.
- McComas, A.J., Sica, R.E., Upton, A.R.M., Aguilera, N., & Currie, S. Motoneurone dysfunction in patients with hemiplegic atrophy. Nature (New Biology), 1971, 233, 21-23.
- Montagu, J., & Coles, E. Mechanism and measurement of the galvanic skin response. Psychological Bulletin, 1966, 65, 261-279.
- Neumann, E., & Blanton, R. The early history of electrodermal research. Psychophysiology, 1969, 6, 453-475.
- Obrist, P.A. Skin resistance levels and galvanic skin response: Unilateral differences. Science, 1963, 139, 227-228.
- Papez, J.W. A proposed mechanism of emotion. Archives of Neurology and Psychiatry, 1937, 38, 725-743.
- Parsons, O.A., & Chandler, P. Electrodermal indicants of arousal in brain damage: Cross validated findings. Psychophysiology, 1969, 5, 644-659.
- Pavlov, I.P. Conditioned reflexes. New York: Dover Publications, 1960.
- Penfield, W., & Roberts, L. Speech and brain mechanisms. Princeton: Princeton University Press, 1959.
- Pribram, K. The primate frontal cortex. Neuropsychologica, 1969, 7, 259-266.
- Raskin, D. Semantic conditioning and generalization of autonomic responses. Journal of Experimental Psychology, 1969, 79, 69-76.

Schachter, J. Pain, fear and anger in hypertensives and normotensives.

Psychosomatic Medicine, 1957, 19, 17-29.

Scholander, T. Habituation of autonomic response elements under two

conditions of alertness. Acta Physiologica Scandinavica, 1960, 50,

259-268.

Schwartz, H.G. Effect of experimental lesions of the cortex on the

"psychogalvanic reflex" in the cat. Archives of Neurology and Psychiatry,

1937, 38, 308-320.

Sokolov, Ye.N. Neuronal models and the orienting reflex. In Brazier, M.A.

(Ed.), The central nervous system and behavior. New York: Macy

Foundation, 1960.

Sokolov, Ye.N. Perception and the conditioned reflex. Oxford: Pergamon

Press, 1963.

Sourek, K. The nervous control of skin potentials in man. Pravda:

Nakladatelstvi Ceskoslovenski Academie Ved, 1965.

Stennett, R.G. The relationship of performance level to level of arousal.

Journal of Experimental Psychology, 1957, 54, 54-61.

Toole, J.F., & Patel, A.N. Cerebrovascular disorders. New York: McGraw-

Hill, 1974.

Varni, J.G., Doerr, H.O., & Franklin, J.R. Bilateral differences in skin

resistance and vasomotor activity. Psychophysiology, 1971, 8, 390-400.

Wall, P.D., & Davis, G.P. Three cerebral cortical systems affecting

autonomic function. Journal of Neurophysiology, 1951, 14, 507-517.

Wang, G.H. The neural control of sweating. Madison: University of

Wisconsin Press, 1964.

Wang, G.H., & Brown, V.W. Suprasegmental inhibition of an autonomic reflex.

- Journal of Neurophysiology, 1956, 19, 564-572.
- Wang, G.H., & Lu, T.W. Galvanic skin reflex induced in the cat by stimulation of the motor area of the cerebral cortex. Journal of Physiology, 1930, 4, 303-324.
- Wang, G.H., & Richter, C.P. Action currents from the pad of the cat's foot produced by stimulation of the tuber cinereum. Journal of Physiology, 1928, 2, 279-284.
- Wilcott, R.C. Electrical stimulation of the anterior cortex and skin-potential responses in the cat. Journal of Comparative and Physiological Psychology, 1969, 69, 465-472.
- Wilder, J. The law of initial values in neurology and psychiatry: Facts and problems. Journal of Nervous and Mental Disease, 1957, 125, 73-86.
- Wyatt, R., & Tursky, B. Skin potential levels in right- and left-handed males. Psychophysiology, 1969, 6, 133-137.
- Zeiner, A.R., & Schell, A.M. Individual differences in orienting, conditionability, and skin resistance responsivity. Psychophysiology, 1971, 8, 612-622.

FOOTNOTES

¹ Personal communication, Dr. Ronald Trites, Royal Ottawa Hospital,
1974.

APPENDIX I

LATERAL DOMINANCE EXAMINATION

Examiner requests that the subject show how he would do each of the following seven tasks (no actual test objects are presented). Right- and left-hand preferences are recorded for each task and the total number of right-hand and left-hand choices is recorded.

First, "SHOW ME HOW YOU WOULD THROW A BALL."

Second, "SHOW ME HOW YOU WOULD HAMMER A NAIL."

Third, "SHOW ME HOW YOU WOULD CUT WITH A KNIFE."

Fourth, "SHOW ME HOW YOU WOULD TURN A DOOR KNOB."

Fifth, "SHOW ME HOW YOU WOULD USE SCISSORS."

Sixth, "SHOW ME HOW YOU WOULD USE AN ERASER."

Seventh, "SHOW ME HOW YOU WOULD WRITE YOUR NAME."

For all motor tests, the dominant hand is tested first and then the non-dominant hand. The dominant hand is determined by the hand preference for writing. This means that although a person may perform all of the other lateral dominance tasks with the right hand but writes with the left hand, the dominant hand would be the left hand. If a subject's dominant hand is partially or completely paralyzed, that body side is used as the dominant side until two years following the onset of the paralysis, which means that if a person was right-handed prior to the paralysis and demonstrated all the lateral dominance items with the left hand, the dominant hand would be the right hand if the time elapsed was within two years. However, after the two years have elapsed, the dominant hand would be taken as the hand that was demonstrated.

APPENDIX II

INSTRUCTIONS AND PROCEDURES FOR MOTOR PERFORMANCE TESTS

Dynamometer grip-strength test

Present the dynamometer to the subject, dominant hand first, saying "WOULD YOU HOLD THIS IN YOUR (dominant) HAND, POINT IT STRAIGHT TO THE FLOOR LIKE THIS (demonstrate) AND SQUEEZE IT AS HARD AS YOU CAN. THIS TIME WOULD YOU HOLD IT IN (non-dominant) HAND AND POINT IT STRAIGHT TO THE FLOOR AND SQUEEZE IT AS HARD AS YOU CAN." Repeat the procedure so that each hand has two trials alternating each time between dominant and non-dominant hands. If the difference between the scores of each hand is within three kilograms, find the average of the two trials. If the difference between the scores of each hand is greater than three kilograms, re-administer the complete test for both hands. To score find the average of four trials for each hand. The handle of the dynamometer should be adjusted for the size of the fist of the patient, the average male is usually above five and the average female is usually between four and five. The handle should be held comfortably in the patient's hand.

Finger-tapping test

"NOW WE ARE GOING TO SEE HOW FAST YOU CAN TAP. WE WILL USE THIS LITTLE KEY HERE (present to subject). PLACE YOUR ARM AND HAND IN A COMFORTABLE POSITION LIKE THIS, AND WITHOUT MOVING YOUR WRIST OR ARM, I WANT YOU TO TAP AS FAST AS YOU CAN WITH YOUR FOREFINGER, LIKE THIS. YOU WILL HAVE TO REMEMBER TO LET THE KEY COME ALL THE WAY UP AND PUSH IT ALL THE WAY DOWN AND CLICK EACH TIME, OR ELSE IT WILL NOT CHANGE THE NUMBER ON THE DIAL." (Show the subject how the key works and how it should be allowed to click each time) "NOW, MOVE THE BOARD TO A COMFORTABLE POSITION FOR YOUR (dominant) HAND AND TRY IT FOR PRACTICE." (allow a few seconds for practice) "I WILL TELL YOU WHEN TO BEGIN AND

WHEN TO STOP EACH TIME. REMEMBER, TAP AS FAST AS YOU CAN. READY, GO."

The subject may rest his hand at any time, but insist that he rest after the third trial for each hand. Record the number of times the subject taps in a 10-second interval. Discontinue when five consecutive scores within a range of five points are obtained. If five consecutive scores within a range of five points cannot be obtained, discontinue after ten trials and compute the mean using the five best scores. Repeat the procedure for the non-dominant hand.

Grooved pegboard test

Place pegboard in the midline of the subject with the board at the edge of the table and the peg tray immediately above the board. Examiner says "WHAT I WANT YOU TO DO IS TO PUT THESE PEGS INTO THE HOLES IN THIS BOARD. NOTICE THAT EACH HOLE AND EACH PEG HAS A GROOVE ALONG ONE SIDE. TO GET THIS PEG INTO THE HOLE, YOU MUST TURN THE PEG SO THAT THE GROOVES WILL MATCH." Examiner demonstrates one row from left to right and then removes the pegs. "WHEN I SAY GO, BEGIN HERE (examiner points to the upper left corner for right-handed subjects, to the upper right corner for left-handed subjects) AND PUT THE PEGS INTO THE BOARD AS FAST AS YOU CAN, USING ONLY YOUR (dominant) HAND. FILL THE TOP ROW COMPLETELY, GOING FROM HERE TO HERE (examiner points left to right for right-handed subjects, right to left for left-handed subjects) BEFORE GOING ON TO THE NEXT ROW. IN EACH ROW, ALWAYS FILL THE BOARD THE SAME WAY YOU FILLED THE TOP ROW. READY, GO."

Record the time required to fill the board and, in parentheses, record the number of times the subject drops a peg. Repeat the procedure for the non-dominant hand, with the subject filling the rows in a direction opposite from trial one.

APPENDIX III

Means/Standard Deviations for the 3x6 Analysis
of Variance for Left-Minus-Right Basal Resistance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>12.457</u>	<u>6.141</u>	<u>0.440</u>	<u>1.533</u>	<u>-2.467</u>	<u>-5.983</u>	<u>2.020</u>
		54.030	64.507	64.929	58.138	59.648	58.305	56.589
	RHD	<u>10.034</u>	<u>13.649</u>	<u>14.886</u>	<u>14.420</u>	<u>15.889</u>	<u>14.597</u>	<u>13.912</u>
		25.900	28.086	27.228	27.474	26.513	26.225	25.291
	NBD	<u>6.676</u>	<u>8.299</u>	<u>11.795</u>	<u>11.539</u>	<u>13.793</u>	<u>13.817</u>	<u>10.986</u>
		7.163	6.487	6.719	8.538	11.664	13.474	9.203
	<u>9.723</u>	<u>9.363</u>	<u>9.040</u>	<u>9.164</u>	<u>9.071</u>	<u>7.477</u>	<u>8.973</u>	
	33.141	38.830	39.258	35.977	37.279	37.092	36.245	

Means/Standard Deviations for the 3x6 Analysis
of Variance for Right-Hand Basal Resistance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>54.250</u>	<u>65.463</u>	<u>72.646</u>	<u>64.223</u>	<u>69.546</u>	<u>74.210</u>	<u>66.722</u>
		47.090	46.098	42.337	35.379	36.536	36.296	38.910
	RHD	<u>55.530</u>	<u>58.067</u>	<u>58.387</u>	<u>54.464</u>	<u>54.694</u>	<u>55.777</u>	<u>56.153</u>
		29.922	29.993	31.161	28.826	27.714	26.116	27.221
	NBD	<u>46.664</u>	<u>50.391</u>	<u>49.863</u>	<u>46.099</u>	<u>47.636</u>	<u>47.293</u>	<u>47.991</u>
		20.128	17.747	20.839	19.118	21.257	24.548	19.476
		<u>52.147</u>	<u>57.974</u>	<u>60.299</u>	<u>54.929</u>	<u>57.291</u>	<u>59.093</u>	<u>56.956</u>
		32.733	32.275	32.435	28.143	29.223	30.219	30.386

Means/Standard Deviations for the 3x6 Analysis
of Variance for Left-Hand Response Resistance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>1.3180</u>	<u>0.9617</u>	<u>0.1274</u>	<u>0.1326</u>	<u>0.0971</u>	<u>0.0526</u>	<u>0.4483</u>
		2.580	1.342	0.260	0.237	0.144	0.077	1.231
	RHD	<u>1.0834</u>	<u>0.5257</u>	<u>0.0074</u>	<u>0.0977</u>	<u>0.2749</u>	<u>0.2211</u>	<u>0.3684</u>
		2.665	1.188	0.020	0.178	0.571	0.511	1.212
	NBD	<u>2.2140</u>	<u>0.1614</u>	<u>0.0806</u>	<u>0.3254</u>	<u>0.3383</u>	<u>0.1251</u>	<u>0.5258</u>
		2.588	0.283	0.108	0.766	0.535	0.151	1.288
		<u>1.5090</u>	<u>0.5496</u>	<u>0.0718</u>	<u>0.1852</u>	<u>0.2367</u>	<u>0.1330</u>	<u>0.4470</u>
		2.519	1.049	0.163	0.461	0.448	0.303	1.236

Means/Standard Deviations for the 3x6 Analysis
of Variance for Right-Hand Response Resistance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>2.3110</u>	<u>1.3628</u>	<u>0.1143</u>	<u>0.5417</u>	<u>0.5914</u>	<u>0.2354</u>	<u>0.8594</u>
		2.879	2.174	0.158	1.114	1.047	0.373	1.693
	RHD	<u>0.8143</u>	<u>0.2960</u>	<u>0.0063</u>	<u>0.0863</u>	<u>0.2303</u>	<u>0.1291</u>	<u>0.2604</u>
		1.764	0.492	0.017	0.124	0.372	0.271	0.772
	NBD	<u>1.3726</u>	<u>0.1489</u>	<u>0.0817</u>	<u>0.2457</u>	<u>0.3314</u>	<u>0.1211</u>	<u>0.3836</u>
		1.404	0.243	0.101	0.554	0.439	0.146	0.763
		<u>1.4990</u>	<u>0.6026</u>	<u>0.0674</u>	<u>0.2912</u>	<u>0.3844</u>	<u>0.1619</u>	<u>0.5010</u>
		2.101	1.347	0.113	0.712	0.673	0.270	1.180

Means/Standard Deviations for the 3x6 Analysis
of Variance for Right-Hand Response Conductance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>1.1898</u>	<u>1.0502</u>	<u>0.7565</u>	<u>0.3860</u>	<u>0.4313</u>	<u>0.2345</u>	<u>0.6747</u>
		1.928	1.814	1.975	0.856	0.971	0.550	1.419
	RHD	<u>0.3856</u>	<u>0.2723</u>	<u>0.0140</u>	<u>0.0560</u>	<u>0.1825</u>	<u>0.1130</u>	<u>0.1705</u>
		0.540	0.453	0.037	0.088	0.366	0.230	0.344
	NBD	<u>0.9838</u>	<u>0.1373</u>	<u>0.0793</u>	<u>0.2818</u>	<u>0.3587</u>	<u>0.1276</u>	<u>0.3281</u>
		1.104	0.224	0.104	0.662	0.474	0.147	0.621
		<u>0.8530</u>	<u>0.4866</u>	<u>0.2833</u>	<u>0.2412</u>	<u>0.3242</u>	<u>0.1584</u>	<u>0.3910</u>
		1.300	1.111	1.137	0.611	0.634	0.341	0.933

Means/Standard Deviations for the 3x6 Analysis
of Variance for Left-Hand Response Log Conductance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>0.0113</u>	<u>0.0108</u>	<u>0.0022</u>	<u>0.0016</u>	<u>0.0015</u>	<u>0.0008</u>	<u>0.0047</u>
		0.0203	0.0145	0.0051	0.0026	0.0029	0.0014	0.0109
	RHD	<u>0.0098</u>	<u>0.0066</u>	<u>0.0003</u>	<u>0.0014</u>	<u>0.0024</u>	<u>0.0024</u>	<u>0.0038</u>
		0.0205	0.0136	0.0009	0.0017	0.0040	0.0041	0.0102
	NBD	<u>0.0162</u>	<u>0.0079</u>	<u>0.0007</u>	<u>0.0023</u>	<u>0.0026</u>	<u>0.0011</u>	<u>0.0051</u>
		0.0177	0.0185	0.0009	0.0049	0.0033	0.0014	0.0115
<u>0.0124</u>		<u>0.0084</u>	<u>0.0011</u>	<u>0.0017</u>	<u>0.0021</u>	<u>0.0014</u>	<u>0.0045</u>	
0.0187		0.0150	0.0030	0.0032	0.0033	0.0026	0.0108	

Means/Standard Deviations for the 3x6 Analysis
of Variance for Right-Hand Response Log Conductance

		Trial Blocks						
		1	2	3	4	5	6	
Groups	LHD	<u>0.0147</u>	<u>0.0104</u>	<u>0.0023</u>	<u>0.0032</u>	<u>0.0034</u>	<u>0.0015</u>	<u>0.0059</u>
		<u>0.0141</u>	<u>0.0116</u>	<u>0.0050</u>	<u>0.0052</u>	<u>0.0044</u>	<u>0.0019</u>	<u>0.0092</u>
	RHD	<u>0.0068</u>	<u>0.0036</u>	<u>0.0001</u>	<u>0.0009</u>	<u>0.0022</u>	<u>0.0013</u>	<u>0.0025</u>
		<u>0.0124</u>	<u>0.0061</u>	<u>0.0003</u>	<u>0.0012</u>	<u>0.0033</u>	<u>0.0023</u>	<u>0.0060</u>
	NBD	<u>0.0151</u>	<u>0.0020</u>	<u>0.0011</u>	<u>0.0036</u>	<u>0.0047</u>	<u>0.0017</u>	<u>0.0047</u>
		<u>0.0157</u>	<u>0.0032</u>	<u>0.0014</u>	<u>0.0083</u>	<u>0.0062</u>	<u>0.0020</u>	<u>0.0088</u>
		<u>0.0122</u>	<u>0.0053</u>	<u>0.0012</u>	<u>0.0026</u>	<u>0.0034</u>	<u>0.0015</u>	<u>0.0044</u>
		<u>0.0140</u>	<u>0.0083</u>	<u>0.0030</u>	<u>0.0056</u>	<u>0.0046</u>	<u>0.0020</u>	<u>0.0082</u>

BILATERAL ELECTRODERMAL ACTIVITY
AND THE ORIENTING REFLEX IN
PATIENTS WITH UNILATERAL BRAIN DAMAGE

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Bilateral measures of five electrodermal variables (basal skin resistance, response resistance, response conductance, response log conductance, rate of habituation of the electrodermal component of the OR) were recorded from left- and right-hemiplegic patients (4-31 months following onset of CVA) and from normal controls. The 30-minute recording session consisted of a 15-minute rest condition and a 15-minute passive auditory stimulation condition during which a series of 30 auditory tones was presented to the subject. Bilateral measures of motor tests of strength, speed, and agility were also obtained from all subjects.

It was predicted that bilateral differences in basal skin resistance would be larger for brain-damaged subjects than for non-brain-damaged subjects due to a lowered basal resistance level on the hemiplegic side. It was also predicted that bilateral differences in basal skin resistance would correlate with bilateral differences in motor performance measures. Bilateral measures of electrodermal activity could then be used to predict the recovery of motor performance in subjects with unilateral brain damage.

Results indicated that the three groups did not differ significantly for any of the electrodermal measures. Since the hemiplegic patients exhibited varying degrees of motor impairment, it was concluded that, while

the bilateral differences in electrodermal activity had recovered to within normal limits, the subjects still showed large bilateral differences in motor performance. These findings did not warrant the use of bilateral measures of electrodermal activity as predictors of recovery in motor performance for patients with unilateral brain damage.