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42 Electrodermal Measures of Arousal in Humans with Cortical or Subcortical Brain Damage

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In seeking to understand the relationship between central nervous system (CNS) structures and human information-processing potential, numerous theories of attention have been proposed (e.g., see Broadbent, 1958; Kahneman, 1973; McGhie, 1969; Mostofsky, 1970; Pribram & McGuinness, 1975; Sutherland & Mackintosh, 1971; Swets & Kristofferson, 1970; Zeaman & House, 1963). In support of these theories, most work has centered around normal performance on a variety of tasks, but several applications to brain-damaged populations also have been made (Luria, 1966; McGhie, 1969; Pribram & McGuinness, 1975; Sokolov, 1960; Sutherland & Mackintosh, 1971; Zeaman & House, 1963). Because a number of transformations of stimulus input occur in the CNS during the processing of information, it has been reasoned that selective impairment of one aspect or another following damage to different areas of the brain can help to clarify the nature of these transformations as well as their possible cerebral loci (Kinsbourne, 1971). However, brain-damaged subjects may fail to show "normal attention" for numerous reasons, including perceptual deficiencies, motivational deficits, and emotional changes. Thus any demonstration of abnormal attention following brain damage would have to discriminate between primary etiology and etiology secondary to other causes (Kinsbourne, 1971; McGhie, 1969). In an effort to deal with this difficult problem, we have examined a relatively elementary aspect of attention, electrodermal indices of

arousal (e.g., Lader & Wing, 1966) in several groups of patients with contrasting regions of cerebral damage. These groups include patients with Korsakoff's disease, Huntington's chorea, Parkinson's disease, or aphasia. The groups represent individuals with different and relatively well-defined areas of cerebral damage and behavioral impairments, thereby allowing interesting and meaningful comparisons to be made.

For example, Korsakoff patients with damage to certain diencephalic structures (Brion, 1969; Victor, Adams, & Collins, 1971) are described as evidencing severe anterograde amnesia (Talland, 1965); patients with basal ganglia disease or expressive aphasia have been described as having various cognitive deficits in addition to their more obvious symptoms of motoric abnormalities or language impairments, respectively (Riklan, 1973; Piercy, 1964). Implicit in these descriptions is the assumption that the patient groups have intact capacities for attending to the information to be processed. Yet considerable evidence from behavioral experiments suggests that this assumption may not be warranted (Boll, Heaton, & Reitan, 1974; Goodman, Hall, Terrango, Perrine, & Roberts, 1966; Oscar-Berman, 1973; Oscar-Berman & Samuels, 1977; Oscar-Berman, Sax, & Opoliner, 1973; Riklan, 1973). Despite the behavioral evidence, surprisingly few studies have used physiological measures of arousal with human brain-damaged subjects (for reviews see Holloway & Parsons, 1978, and Stern & Janes, 1973). Results of the present study were expected to provide a first step toward testing existing assumptions about cognitive impairments in the disorders mentioned and the role of possible attentional deficits. In addition, such information would contribute toward localizing deficiencies in arousal within specific CNS structures or systems.

METHOD

Subjects

Fifty-eight subjects participated in the experiment (see Table 42.1). The subjects comprised the following five groups: (1) 18 normal controls (N) recruited from medical wards of the Boston V.A. Hospital, the hospital volunteer service, and a local senior citizens' group; (2) 15 parkinsonians (P) recruited from the Neurology Service of Boston University Hospital. They all were receiving placebo medicine at the time of testing but no other medication. Ten had previously been treated unsuccessfully with L-dopa, and all were hospitalized for trial treatment with a new antiparkinson drug (see Oscar-Berman, Gade, Feldman, & Saavedra, in press); (3) 10 aphasic patients (A) from the Neurology Service of the Boston V.A. Hospital. They had varying degrees of right-sided hemiplegia and Broca's aphasia; (4) 8 Korsakoff patients (K), also from the V.A. Neurology Service. They all had a history of chronic alcoholism and showed

TABLE 42.1
Comparison of the Five Groups of Subjects Tested in This Study

Group	Total No. Subjects	Males	Females	Age		Duration of Illness (years)		Education (years)	
				Mean	Range	Mean	Range	Mean	Range
N	18	11	7	54	42–72	–	–	11.1	8–17
P	15	7	8	64	55–74	7.9	0.5–27	13.0	7–20
A	10	10	0	52	33–69	0.6	0.1–2	13.0	8–19
K	8	8	0	54	47–66	?	?	10.3	7–12
HC	7	5	2	47	33–59	7.6	3–12.5	11.4	6–16

clinically significant memory deficits, with a memory quotient (Wechsler's Memory Scale) at least 20 points lower than their intelligence quotient (Wechsler's Adult Intelligence Scale); (5) 7 Huntington's chorea patients (HC) from the Boston V.A. Hospital and Boston University Hospital. In all cases but one the disease had progressed far enough to force the patients into retirement. Table 42.1 offers further descriptions of the five groups.

Apparatus

Physiological measures consisted of thumb electrodermal activity and earlobe pulse volume during basal rest conditions and during a series of auditory stimuli. Electrodermal activity and pulse volume were continuously recorded on a three-channel Grass Model 79D polygraph. For the measurement of electrodermal activity, a constant current of 10 microamperes was passed through silver–silver chloride electrodes with an inner (active) diameter of 15 mm (Lexington Instruments, Type C-1) attached to the palmar surface of the distal phalanx of the thumb and to the medial side of the upper arm about 10 cm from the elbow. The electrodes were fixed with adhesive rings, filled with jelly paste, and secured with tape. For pulse-volume measurements, a plethysmographic (photoelectric) transducer (Grass Instruments, Model RPT 1) was placed on the subject's earlobe, secured with tape, and shielded from light by a black cloth.

Procedure

After reviewing the human-consent form and the general nature of the experimental setup, the subjects were seated in a comfortable reclining armchair in an air-conditioned recording room. The general procedures and experimental paradigm were similar to those of Horvath and Mears (1974). Electrodes and the transducer were attached to the subject in that order, and after a calibration period of 1 to 3 min, the room lights were extinguished, leaving only a dim illumination at the polygraph. After another 2 min, a 10-min resting period began, followed by a series of 20 auditory stimuli (buzzer tone) of 100-db./SPL intensity and 1-sec duration played on a tape recorder located behind the subject. The stimuli were randomly separated by 30 to 80 sec of silence.

Derivation of measures. In addition to measuring skin conductance responses across the 20 stimulus presentations, we derived four overall mean measures from the electrodermal activity and one from the plethysmographic recordings:

1. Resting level of log skin conductance. Levels of skin resistance were determined at 30-sec intervals during the 10-min resting period; they were transformed into log (normal) skin conductance levels according to convention (Lader & Wing, 1966), and the readings were averaged.

2. Number of spontaneous fluctuations of skin conductance. Fluctuations greater than 0.003 log μmho (the smallest reliably measured change) were counted during each minute of the resting period and averaged.

3. Orienting response (OR), defined as the first skin conductance response to the first or second auditory stimulus. Skin conductance responses were calculated as change in log conductance initiated within the first 5 sec after the onset of the stimulus.

4. Habituation rate. A regression of the values of the 20 skin conductance responses to the log number of stimuli was carried out and the habituation rate determined as the resulting slope m of the regression line, $y = b - mx$, where the y coordinate is the log number of stimuli.

5. Pulse volume responses to the 20 auditory stimuli. These responses were measured from 2 to 8 sec after stimulus onset, and the adopted definition (Furedy, 1968) required response intiation and termination to occur for longer than two pulse periods. The amount of change was expressed as a percentage of the base pulse. Pulse volume could not be measured reliably in many records or portions of records because of movement artifacts, heart arrhythmia, and poor transducer placement.

RESULTS

Results can be seen in Figs. 42.1 and 42.2. A groups × blocks analysis of variance was performed on skin conductance responses in 10 blocks of two stimulus presentations each (see Fig. 42.1). There was a significant main effect of blocks—$F(9, 441) = 20.82$, $p < 0.001$—indicating that skin conductance responses decreased over trials. In addition there was a significant groups × blocks interaction—$F(36, 441) = 2.21$, $p < 0.001$—indicating that the groups differed in their responses over blocks. Subsequent t tests were performed in order to assess the nature of the group differences. On the first block of stimulus presentations, Korsakoffs were significantly less responsive than normal controls—$t(22) = 2.64$, $p < 0.05$—and aphasics—$t(16) = 2.76$, $p < 0.05$. Patients with Huntington's disease were also less responsive than normals—$t(21) = 2.96$, $p < 0.01$—and aphasics—$t(15) = 3.06$, $p < 0.01$—and were lower in responsiveness than Parkinson patients—$t(20) = 2.13$, $p < 0.05$. The Parkinson–Korsakoff difference did not reach significance—$t(21) = 1.96$, $0.10 < p > 0.05$. On subsequent blocks of stimulus presentations, the only individual group differences to reach significance ($p < 0.05$) were between the Huntington patients as compared to the aphasic and/or the Parkinson patients; these differences disappeared after the sixth stimulus block.

A similar analysis was carried out on pulse volume data collected concurrently with the electrodermal data. We were able to obtain complete records from

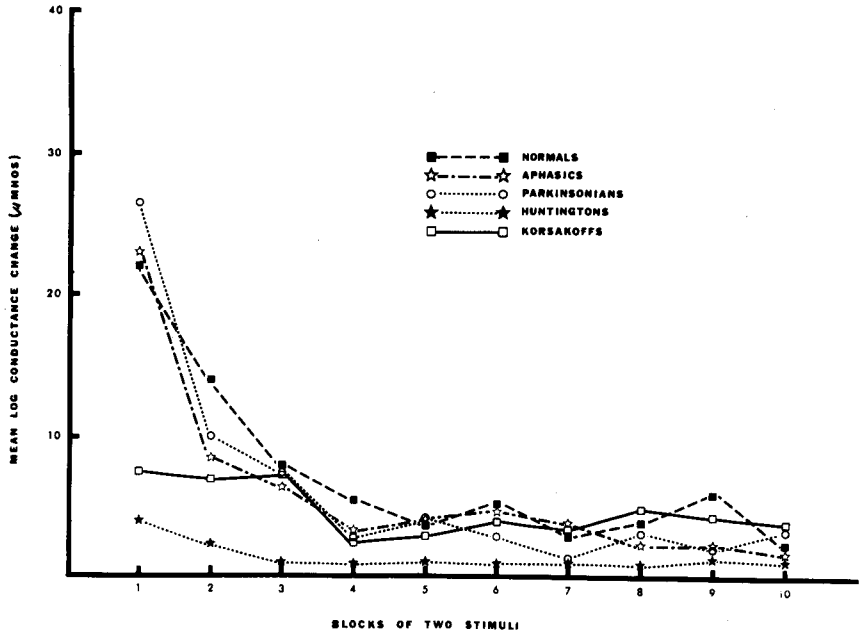

FIG. 42.1 Mean log skin conductance levels for the five groups of subjects as a function of the auditory stimulus presentations (10 two-trial stimulus blocks.) (From Oscar-Berman, 1978.)

only 11 normals, 6 aphasics, 7 Korsakoffs, 4 Parkinson patients, and 3 Huntington patients. Despite the reduction in sample size for this measure, results paralleled those obtained with electrodermal recordings. There was a significant groups × blocks interaction—$F(36, 234) = 1.46$, $p = 0.05$—and generally the Korsakoff patients were less responsive than normal subjects throughout the session; these differences reached significance ($p < 0.05$) on Stimulus Blocks 4, 6, 7, and 10. Likewise, Korsakoffs were significantly less responsive than Parkinson patients on Blocks 8 and 10 and the Huntington patients on Block 7. The only other significant differences among the groups occurred on Block 4, in which the aphasic and Parkinson patients evidenced lower responsiveness compared to neurologically intact subjects.

Results of the remaining measures are depicted in Fig. 42.2. The mean resting levels of conductance measured during the 10-min rest period preceding stimulus presentation are shown in the upper left quadrant of Fig. 42.2. There was no significant group main effect in the analysis of variance—$F(4, 52) = 1.53$, $p = 0.21$. However, we performed intergroups t tests in order to determine whether or not the suggestion of a low resting level by Korsakoffs approached significance. The only instances of significant differences from Korsakoff levels involved aphasics—$t(16) = 2.14$, $p < 0.05$—and Parkinson patients—$t(21) =$

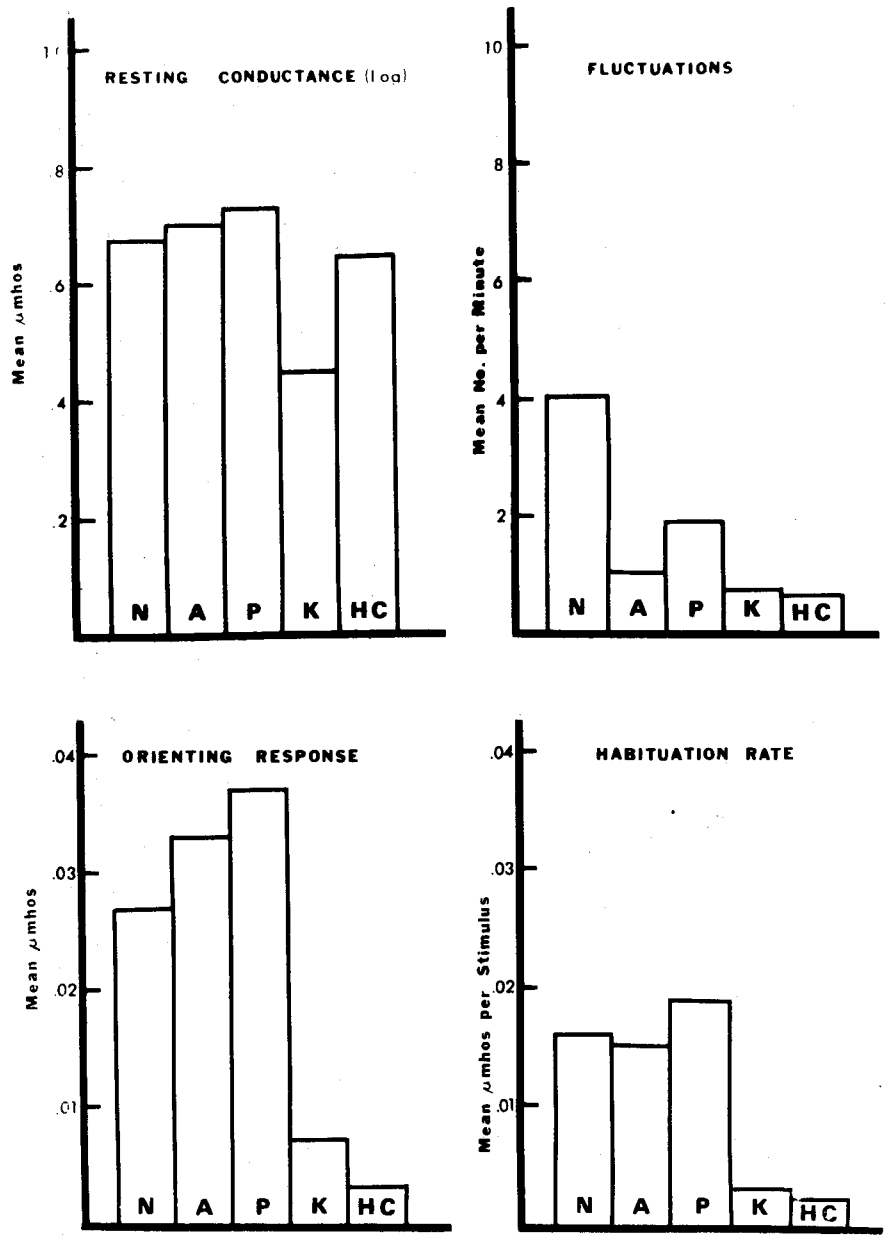

FIG. 42.2 Mean levels of responsiveness by the five groups of subjects during the rest interval (resting conductance and fluctuations) and during stimulus presentation (orienting response and habituation rate).

TABLE 42.2
Intergroup Comparisons of Orienting Response (OR) and Habituation Rate (HAB)

		Normals	*Aphasics*	*Parkinsons*	*Korsakoffs*	*Huntingtons*
Normals[a]	OR	—	NS	NS	$t(22) = 2.81$*	$t(21) = 3.19$**
	HAB	—	NS	NS	$t(23) = 2.65$*	$t(22) = 2.64$*
Aphasics	OR	NS	—	NS	$t(16) = 2.77$*	$t(15) = 3.02$**
	HAB	NS	—	NS	$t(16) = 2.60$*	$t(15) = 2.60$*
Parkinsons	OR	NS	NS	—	$t(21) = 1.90$[b]	$t(20) = 2.01$[b]
	HAB	NS	NS	—	$t(21) = 2.22$*	$t(20) = 2.17$*
Korsakoffs	OR	$t(22) = 2.81$*	$t(16) = 2.77$*	$t(21) = 1.90$[b]	—	NS
	HAB	$t(23) = 2.65$*	$t(16) = 2.60$*	$t(21) = 2.22$*	—	NS
Huntingtons	OR	$t(21) = 3.19$**	$t(15) = 3.02$**	$t(20) = 2.01$[b]	NS	—
	HAB	$t(22) = 2.64$*	$t(15) = 2.60$*	$t(20) = 2.17$*	NS	—

[a]The OR data from two normal subjects and the HAB data from one were not included.
[b]$0.10 < p > 0.05$.
*$p < 0.05$.
**$p < 0.01$.

$2.81, p < 0.05$. The upper right quadrant of Fig. 42.2 shows mean spontaneous levels of skin conductance during the 10-min rest period. Here the group effect in the analysis of variance was significant—$F(4, 48 = 2.71, p < 0.05$. Subsequent t tests revealed no significant differences among the individual groups. However, there was a general tendency for the normals to show a greater number of fluctuations when compared to all other subjects, and indeed, the normal versus patient comparison was significant—$t(51) = 3.20, p < 0.01$—as was virtually any comparison of normals with two or more groups of brain-damaged subjects (e.g., Parkinson and Huntington patients combined as a single group with basal ganglia damage; aphasic and Korsakoff patients combined as a single group with damage outside of the basal ganglia).

Responses during the stimulus presentation period are shown in the bottom two quadrants of Fig. 42.2. An analysis of variance on orienting responses by the five groups yielded a significant main effect of Groups—$F(4, 51) = 2.88$, $p < 0.05$—as did the analysis of habituation rate—$F(4, 52) = 3.02, p < 0.05$. Individual group comparisons by t tests are presented in Table 42.2. These comparisons revealed no differences between Korsakoff and Huntington patients. However, there were consistent differences between Korsakoff patients and each of the other three groups and between Huntington patients and each of the other three groups.

DISCUSSION

Results of the present study showed consistent hyporeactive arousal levels in Korsakoff and Huntington groups. In contrast, aphasic and Parkinson patients evidenced arousal levels within the range of neurologically intact subjects. These results support the views of Stern and Janes (1973) and Holloway and Parsons (1978) that there is no simple generalized autonomic change as a result of brain damage per se. Rather, damage to certain regions or systems within the human CNS will have drastic effects on activation levels, whereas damage elsewhere may have minimal effects.

Previous literature on psychophysiological measures with brain-damaged subjects is fraught with inconsistencies because of differences in recording methods, experimental paradigms, composition of patient groups, etiology and duration of the disorder within groups, nature of the control groups, and so forth. For example, in a series of very differently designed experiments, (a) Holloway and Parsons (1978) describe their findings of hyperreactivity in patients with diverse areas of cortical pathology; (b) Heilman, Schwartz, and Watson (1978) found hyperreactivity in left cortically damaged patients but hyporeactivity in right cortically damaged patients; (c) we found normal arousal levels in our left cortical subjects (aphasics). However, when similar paradigms are employed to study arousal, results can be strikingly parallel. Thus in most respects the

electrodermal measures obtained from Parkinson patients in the present study (modeled after Horvath & Mears, 1974) were consistent with similar pretreatment measures obtained on parkinsonians by Horvath and Mears (1974).

We cannot reconcile divergent results across divergent experiments, but we can examine the differences in the results of the present experiment between Korsakoffs and Huntington choreics on the one hand and the remaining three groups on the other hand. To our knowledge no study of this type has been done previously with Korsakoff or Huntington patients as separate groups.

Huntington patients have atrophy of the caudate nucleus, with some frontal cortical involvement as well (Barbeau, Chase, & Paulson, 1973). Korsakoff patients have primary lesions in the thalamic–limbic areas (Victor et al., 1971); these regions are part of a thalamic reticular system (Baker, 1978; Fuster, 1973; Truex, 1959) and have extensive projections to and from frontal cortex (Nauta, 1972). Baker (1978) and Rozin (1976) have suggested that the integrity of the thalamic reticular system and its frontal connections is essential to normal orienting responsivity; others (e.g., Pribram & McGuinness, 1975) stress the importance of limbic and frontal systems. Why, then, do Korsakoffs and Huntington patients show other (contrasting) behavioral symptoms? The cognitive deficits of Huntington patients appear to be diffuse and global, suggesting that their hyporeactive arousal may be a *primary* contribution to their intellectual impairment. Much empirical work is needed to test this assumption, because relatively little is known about the cognitive capabilities of these patients. Korsakoff patients, however, show very characteristic symptoms in the absence of generalized intellectual decline—for example, severe anterograde amnesia (Talland, 1965) and abnormally strong perseverative response tendencies (Oscar-Berman, 1978, in press; Oscar-Berman, Sahakian, & Wikmark, 1976; Oscar-Berman & Samuels, 1977; Talland, 1965). The answer may lie in the fact that CNS damage incurred by Korsakoff's disease due to alcoholism is not only diffuse but also involves extensive projections to and from the hippocampus and the frontal cortex (in subregions different from those involved in Huntington's disease) (Nauta, 1972). These are areas that when damaged in humans, lead to anterograde amnesia (Milner, Corkin, & Teuber, 1968) and to abnormal perseveration (Teuber, 1972). In short, neither Korsakoff's syndrome nor Huntington's chorea has a single deficit, and the damage causing the symptoms is not focal. Results of the present study underscore the necessity for identifying the contributions of deficits in arousal to other clinical symptoms and for evaluating the interaction of one type of deficit with another.

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