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GSR AND CORTICOSTEROID RESPONSE IN MONKEYS WITH FRONTAL ABLATIONS

W. E. GRUENINGER, D. P. KIMBLE, J. GRUENINGER and S. LEVINE

Dept. of Psychiatry, Stanford University School of Medicine, Palo Alto, California, U.S.A.

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Abstract—The GSR deficit produced by lesions of the dorsolateral frontal cortex was examined under several conditions. Except in presence of movement no GSR was recorded from "frontal" animals in response to a novel stimulus, to one presented in a shock associated environment, or to a stimulus repeatedly paired with shock. This absence of response contrasted with apparently normal EEG desynchronization to the novel stimulus and a resistance change of normal amplitude in response to electric shock.

The corticosteroid response to shock and to shock associated environment was measured. Under all conditions the total change in steroid level was the same for both groups. However, the lesioned animals did show markedly elevated steroid levels two weeks after the initial shock session, and demonstrated a different pattern of response within the individual sessions.

1. INTRODUCTION

THE recent volume *The Frontal Granular Cortex and Behavior* [1] attests to the widespread current interest in this cortical area as well as to the wealth of information already available. Although the behavioral studies have concentrated on the dorsolateral portion of this region, the physiological information which has appeared concerns, for the most part, the orbitofrontal cortex. Suprisingly little is known about the physiological concomitants of lesions of the dorsolatoral frontal cortex.

Some very recent studies have found that humans with frontal lesions [1, 2] and rhesus monkeys with dorsolateral frontal ablation [3] show a marked reduction in the galvanic skin response normally elicited by the presentation of a novel stimulus such as a tone. This marked diminution of the orienting GSR is a most intriguing finding in the light of the "frontal" monkeys demonstrated overresponsiveness to novel stimulus objects on behavioral tests [4, 5].

Another adaptive mechanism in which the dorsolateral frontal cortex has been vaguely implicated is the release of 17-OH steroids from the adrenal cortex. PORTER [6] reported that stimulating this area of frontal lobe caused an eosinopenia in monkeys, and MASON [7] demonstrated that portions of the limbic system (amygdaloid complex and hippocampus) modulate the release of adrenal corticosteroids. On the other hand, STORY *et al.* [8] reported that a corticosteroid stress response occurs in the complete absence of frontal cortex.

This study was designed to examine the effects of removal of dorsolateral granular isocortex upon some aspects of the galvanic skin response and of the corticosteroid response.

With regard to the galvanic skin response, the basic question asked was: Is the deficit in the orienting GSR symptomatic of an abnormal response to novelty or is it something more general? More specifically:

(1) Is the orienting response of the entire nervous system grossly altered?
(2) Will frontally lesioned animals give GSR's under conditions of intense noxious stimulation or is there some gross impairment of the peripheral mechanism?
(3) Will the frontally lesioned animals which fail to respond to a tone when it is a novel stimulus, respond to the same tone when it is presented in a shock associated environment or when the tone itself is paired with shock?

With regard to the corticosteroids, the basic question asked was: Does the removal of this cortex affect the release of corticosteroids? More specifically:

(1) Does the absence of dorsolateral frontal granular isocortex affect the steroid response to electric shock?
(2) Does the absence of this cortex affect the response to an environment previously associated with electric shock?

2. METHOD

2.1. *Subjects*

The test group originally consisted of eight adolescent rhesus monkeys (Nos. 101–108). Four animals, (Nos. 101–104) had undergone bilateral removal of dorsolateral frontal cortex 12 months prior to the beginning of this experiment. The lesions were made by subpial suction under aseptic conditions and consisted of the removal of the gray matter bounded by the arculate sulcus and the anterior tip of the hemisphere. The anterior bank of the arcuate sulcus and the banks and depths of the sulcus principalis were included*†. Post mortem examination showed animal No. 103 of this group to have incomplete degeneration of the parvo cellular portion of nucleus medialis dorsalis thalami in the left hemisphere, despite an apparently successful removal of the appropriate cortex. For this reason animal No. 103 will not be considered as part of the "frontal" group. Animals Nos. 105–108 served as unoperated controls. Subjects Nos. 101–108 had formed a group from the time the operated animals had recovered from surgery. All had been used in a number of behavioral experiments as well as in the GSR habituation study of KIMBLE *et al.* [3].

2.2. *Apparatus*

A Foringer primate chair was used to secure the subjects. EEG recordings were made on a 4 channel Grass Polygraph. Skin resistance was measured by means of a Fels Dermohmmeter and continuous records were made with an Esterline–Angus GSR inkwriter. The GSR electrodes were of zinc–zinc sulfate and were approximately 1 cm in diameter. Fels electrode paste (zinc sulfate in agar) coupled these electrodes to the skin. The sound deadened experimental chamber used was a box 2 ft square by 7 ft high. It was insulated with 2 layers of acoustic tile and an internal layer of fiberglass insulation.

* Surgery by KARL H. PRIBRAM.

† Reconstructions of the lesions are published elsewhere in KIMBLE, BAGSHAW and PRIBRAM [3]. "The GSR of monkeys during orienting and habitation after selective partial ablations of the cingulate and frontal cortex".

2.3. *Procedure*

2.3.1. *Galvanic skin response.* Since the basic procedure involved in each of the three experiments concerned with the GSR was the same, it will be set forth once and the modifications specific to the separate experiments will be described subsequently.

One subject was run per day, and the S to be run was selected from alternate groups on alternate days. Starting time for the day's run remained the same for any one experiment.

The experimental session consisted of a preparatory phase and a test phase.

2.3.2. *Preparatory phase.* The animal was removed from his home cage and immediately secured in the primate chair. The plantar surfaces of the feet were washed with phisohex and dried with paper towels. The GSR electrodes were then placed, and held in position with elastic adhesive bandage to minimize movement artifact. This sequence was usually accomplished within 30 min, after which at least 10 min were allowed for hydration of the skin under the GSR electrodes before moving to the second phase. When these preparations were complete, the animal was placed in the sound insulated chamber. The electrodes were connected to the apparatus and the skin resistance record was monitored until it appeared that a stable base line had been reached.

2.3.3. *Test phase.* The test phase began when the above preparations were complete.

The precise conditions which pertained during the time the animal was confined in the test chamber varied in each experiment and are described below.

(1) *Initial study: EEG desynchronization during GSR habituation.* The procedure was as set forth above except that the preparatory phase was lengthened by the depilitation of the S's scalp by means of electric clippers and depilitory cream, and the placement of three small disc-type EEG electrodes over the frontal, parietal and occipital regions of the left hemisphere. An indifferent electrode was placed on the right ear. All EEG electrodes were secured by a skull cap made from adhesive tape.

During the test phase the S was connected to both the Grass Polygraph (EEG leads plus signal maker) and to the Fels Dermohmmeter. EEG and GSR records were monitored. When the former showed slow waves, preferably alpha, and the latter was stable, the stimulus was presented. This procedure was repeated from 80 to 100 times in one session. Each subject was run once with a 1,000 c/s tone at 2 sec duration as the stimulus, and a second time with a bright flash of light serving this role.

(2) *Main experiments GSR to shock.* Preparation was as outlined above. No EEG electrodes were used. A braided copper wire was tied around each of the subject's wrists to serve as shock electrodes. The electrodes were connected to a d.c. constant current source when the animal was placed in the sound insulated chamber. The test period lasted 55 min during which time the chamber remained dark and five shocks were administered. The first occured as soon as the GSR record had stabilized and the test period had therefore, begun. The others followed at intervals of 20, 10, 15 and 10 min respectively. All shocks were of 3m A. intensity and of 2 sec duration. The skin resistance was recorded continuously during the test period. The run ended when the skin resistance had reached a maximum deflection following the fifth shock, and the record showed that the resistance was again climbing.

Each subject was run for a second session after an interval of two weeks, and a third session two weeks after the second. In the second and third sessions conditions were similar to those described above except that no shock was administered.

(3) *Supplementary study: GSR in shock conditioned situations.* Preparation followed the basic procedure above including the shock electrodes of the main experiment. For this experiment the test chamber was illuminated sufficiently to permit visual observation of the subject through a one way window in one side of the box.

A 4 sec 1,000 c/s tone was presented at least 10 times or until the skin resistance record clearly demonstrated that the subject was giving no response in the absence of movement (Phase 1). Next (Phase 2), 4 shocks of 2 mA. intensity and 0·5 sec duration were given unaccompanied by tone, after which at least 10 presentations of the tone alone were repeated until the record clearly demonstrated that no sensitization or pseudo conditioning had occurred with respect to the GSR. Thereafter (Phase 3) 50 pairings of tone and shock were administered. The shock, initially at 2 mA., was increased to 4 mA. for the last 25 pairings. After every fifth pairing a test presentation of tone alone was used. A final series of tone presentations followed the conditioning sequence.

2.4. *Corticosteroids*

In the main experiment above, the adrenal steroid response was studied concurrently with the GSR. Three blood samples were obtained: the first as soon as the subject was secured in the primate chair, the second immediately before the animal and chair were placed in the test chamber, and the third upon removal of the subject from the sound insulated box at the end of the GSR measurement period.

The first sample was usually drawn within 2 or 3 min after the animal was first approached: the second, ½ hr later, and the third, about 1 hr after the second. The usual preparation procedure, with which the animal was quite familiar, occupied the interval between first and second samples, while the five shocks in the dark chamber were administered between the second and third samples. Each subject was run through this procedure in a second session which followed the first after an interval of two weeks, and through a third session two weeks after the second. On these last two sessions no shock was administered although everything else remained unchanged. In the second and third sessions a fourth blood sample was taken 1 hr after the third. During the interval between the third and fourth samples the animal remained confined in the chair but was removed from the experimental chamber to a relatively quiet room.

All blood samples were one or two ml and were drawn by venapuncture from a superficial leg vein. Steroid analysis was by the fluorometric method of SILBER, BUSH and OSLAPS [8]*.

3. RESULTS

3.1. *Galvanic skin response*

3.1.1. *Initial study.* Examination of the EEG records obtained in the habituation situation showed quite clearly that the frontal animals often responded to both tone and light with desynchronization of the EEG. The duration of such desynchronization varied from a few seconds to as long as a minute but in no case was a concomitant GSR observed in the absence of movement in any frontal animal.

*Corticosteroid analysis done by CAROLINE LOCKWOOD in the laboratory of Dr. SEYMOUR LEVINE.

3.1.2. *Main experiment.* The essential results of the analysis of the continuous record of skin resistance are as follows: It would appear that under the conditions of the experiment "frontal" and normal animals do not differ with respect to the amplitudes of the GSR's produced in response to each of the 5 shocks during the first session. This lack of between group difference is found whether the statistic compared is absolute resistance change, percentage change, change in the square root of the conductance, or change in log conductance. Whether these statistics are calculated from maximal deflection or from values of resistance 10 sec after shock, no differences are apparent.

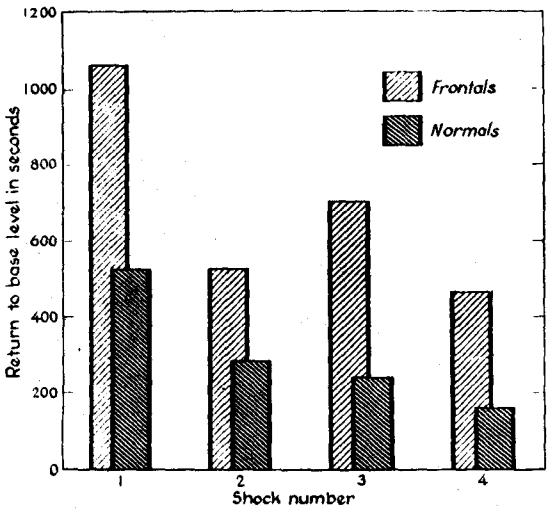

FIG. 1. Time for skin resistance to return to pre-shock level, expressed as group mean return time.

Figure 1 shows the results of an examination of a second parameter. That is, the length of time for skin resistance to return to its preshock level following the first four of the five shocks. Analysis of variance shows that the two groups do differ with respect to this index. ($F = 13{\cdot}18$ with 1 and 5 degrees of freedom, $P < 0{\cdot}025$). In fact, the return times for frontally lesioned animals are longer in every instance except one. Examination of the record indicated that even this one overlap was probably due to insufficient time for skin hydration before the first shock.

Figure 2 shows the results of the analysis of skin resistance records of sessions 1, 2 and 3 for yet another parameter—the number of GSR-like fluctuations per 50 min session in the experimental chamber. This was the length of time for which uniformly stable GSR records were available. All deflections in the resistance record which exceeded 200 Ω, and which resembled GSR's in the judgment of the skilled observer, were counted. Analysis of variance for the group difference yields an F of 43·73 with 1 and 5 degrees of freedom and a $P < 0{\cdot}005$. Even though movement-induced GSR's have been included in this statistic the "frontal" animals show so much less GSR-like activity that there is no overlap between the two groups.

3.1.3. *Supplementary study.* The results of the supplementary study are briefly summarized in Table 1. No tone evoked GSR was recorded from any of the frontal animals in

Table 1. Conditioning of the GSR shows the number of GSR's occurring for each subject under each phase of conditioning.

Subject No.		Tone alone	Elect. shocks	Tone alone	Shock tone pairs 1–10	2 test tones	Shock tone pairs 11–20	2 test tones	Shock tone pairs 21–30	2 test tones	Shock tone pairs 31–40	2 test tones	Shock tone pairs 41–50	Test tones	Tone alone
101	GSR *s* M	0	0	0	0	0	0	0	0	0	0	0	0	0	*
	GSR c M	1	0	0	0	0	0	0	0	0	0	0	1	0	*
	s GSR *s* M	9	3	9	6	2	6	2	3	2	0	2	0	0	*
	M *s* GSR	0	1	1	4	0	4	0	7	0	10	0	9	2	*
102	GSR *s* M	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	GSR c M	0	3	4	1	1	0	1	3	0	0	0	0	0	0
	s GRS *s* M	5	6	5	1	1	0	1	0	0	0	2	0	1	7
	M *s* GSR	6	1	1	8	0	10	0	7	2	10	0	10	1	3
104	GSR *s* M	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	GSR c M	10	4	1	3	1	3	1	2	1	4	1	2	2	17
	s GSR *s* M	0	0	9	4	1	2	1	7	1	1	1	6	0	14
	M *s* GSR	0	0	0	3	0	5	0	1	0	5	0	2	0	9
103	GSR *s* M	5	0	0	0	0									13
	GSR c M	12	0	26	8	2									79
	s GSR *s* M	9	0	1	0	0									3
	M *s* GSR	10	4	10	2	0									35
105	GSR *s* M	30													
	GSR c M	18													
	s GSR *s* M	34													
	M *s* GSR	23													

* Subject became untied.
GSR *s* M = GSR present without movement; GSR c M = GSR and movement present; *s* GSR *s* M = no GSR nor movement present; M *s* GSR = movement present without GSR.

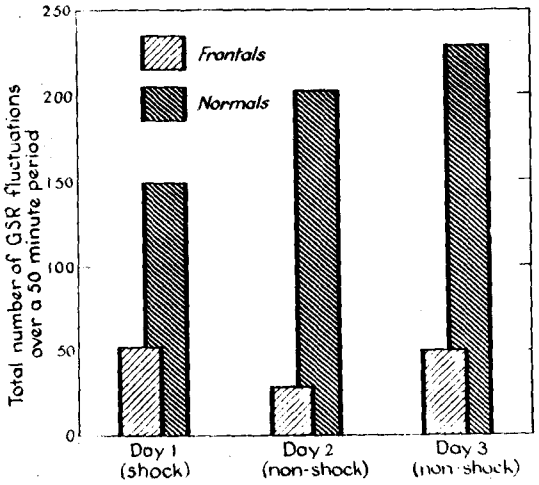

FIG. 2. Total GSR activity on shock and non-shock days in frontal and normal monkeys.

the absence of movement, although there were repeated occasions when movement did not occur and a GSR could have been expected, particularly during the conditioning procedure. In fact, many localized movements occured without concomitant GSR's. Further, no conditioning of the GSR was observed although behavioral conditioning could clearly be seen in the form of anticipatory movement. Since this experiment was intended to determine the extent of frontal GSR unresponsiveness there was no need to run a normal control group. Just to be sure, however, one normal S (No. 105) was submitted to the same procedure and the results also shown in Table 1. As expected, extreme GSR activity was recorded in Phase 1, making Phases 2 and 3 superfluous.

Of interest also is the record produced by the subtotally lesioned "frontal" subject (No. 103). This record is in marked contrast to those of the true "frontal" animals and is similar to that of normal animal No. 105.

While the "frontal" animals did not respond to any tone with an uncontaminated GSR, examination of the records did show spontaneous GSR-like fluctuations unaccompanied by observed movement. Such spontaneous fluctuations occurred at rare intervals throughout the entire procedure, and bore no apparent relationship to any tone or shock.

3.2. *Corticosteroids*

Figure 3 graphs the "frontal" and normal group averages for each sample in each of the three sessions*. Inspection of this graph immediately shows that the frontal animals are not unreactive. In general the two groups would appear quite similar. An analysis of variance yields an F of less than unity for the overall group difference. Thus one might safely conclude that there is no overall group difference in corticosteroid levels.

Inspection of this graph also suggests that the "frontal" animals had abnormally high steroid levels on the second experimental session. Analysis of the group by day interaction shows an F of 4·12 with 2 and 10 degrees of freedom and a P of $<0{\cdot}05$. Thus the "frontal" animals show elevated steroid levels two weeks after the shock session.

* For simplicity, the values for the fourth sample on sessions 2 and 3 have been omitted from Figs. 3 and 4 and from the analysis of variance, since no comparable data exist for the first session. A separate analysis of these data alone yielded no significant group differences.

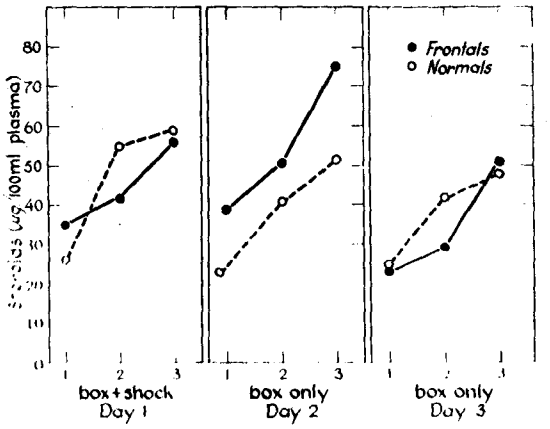

FIG. 3. Blood steroid concentrations under experimental conditions, expressed as group means.

Still another difference is suggested by the shapes of the curves in Figure 3. The "frontal" group seems to lag behind the normal group. This is more easily seen when presented in terms of change in steroid level as in Figure 4 which shows the average change in blood steroid level occurring between successive samples. This statistic corrects for discrepant initial levels and concentrates on the change produced in the experimental situation. This figure shows that the "frontal" steroid levels increase less between samples 1 and 2 and more between samples 2 and 3 than do the normals. When an analysis of variance is performed on these different scores this group by interval interaction is found to be significant at the $P<0{\cdot}025$ level ($F=15{\cdot}28$ with 1 and 5 degrees of freedom). As evident in Figures 3 and 4, the overall change in the steroid levels is very similar for the two groups. In fact, the group over individual F ratio equals 0·018 since the mean group change is almost identical for the two groups.

Thus while the overall reactivity of the "frontal" animals with respect to steroidogenesis is unimpaired, they differ from normals in two important respects. The "frontal" animals showed an elevated steroid level throughout the second session, which was separated from

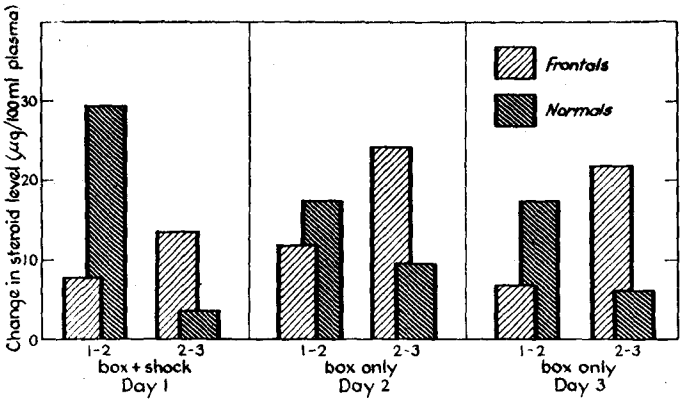

FIG. 4. Change in steroid levels between successive samples, expressed as group mean change.

the first session by an interval of two weeks. Further, within each session the "frontals" showed a different pattern of response with less of an increase evident between samples 1 and 2 (first ½ hr), and more of an increase between samples 2 and 3 (the hour of shock and/or confinement in the chamber).

4. DISCUSSION

4.1. *Galvanic skin response*

The "frontal" deficit in the orienting GSR reported by LURIA, PRIBRAM and HOMSKAYA [2] and by KIMBLE *et al.* [3] might have represented a deficiency in the orienting *per se*, or in the GSR mechanism. The apparently normal EEG desynchronization of the "frontal" animals which we observed in the habituation situation agrees with the behavioral evidence that frontals are not *un*responsive to novelty. The disturbance, therefore, must lie in the GSR mechanism itself, or in the coupling of this mechanism to the rest of the nervous system.

The results reported here show that a galvanic skin response can be recorded from a "frontal" animal under the appropriate conditions. Those necessary conditions are electric shock or movement. In each "frontal" record there also appeared a few spontaneous fluctuation at times when movement was not noted. Although all galvanic skin responses share a final common path, the pseudomotor neurons, there at least three distinct ways in which this final path can be activated: (1) the GSR can be elicited from the "spinal" animal as a nocioceptive reflex; (2) it can be elicited by stimulation of the hypothalamus even in the decerebrate preparation and (3) by stimulation of the sensory-motor cortex or the pyramidal tract both before and after destruction of the hypothalamus [9, 10, 11, 12]. It is hardly surprising that shock evokes a GSR from the frontal animal since even a "spinal" animal responds to shock. Nor is it overly surprising that the frontal animal gives a GSR along with movement since stimulation of the sensory-motor cortex or pyramidal tract evoked both movement and the galvanic skin response. What is very surprising, however, is the complete absence of all other GSR's—the "frontal" animals did not even respond to a tone which had been repeatedly paired with shock. The functional peripheral mechanism appears to have been decoupled from the major GSR activating system.

It has been implied above that the GSR's which accompany movement in the "frontal" animals may reflect activity in the motor system. If this is the case, an appropriately placed lesion in the sensory-motor cortex should eliminate the movement associated GSR. SCHWARTZ [13] reported that a unilateral lesion of the medial portion of the anterior sigmoid gyrus and the medial third of gyrus poreus completely abolished the GSR, except as a segmental reflex, in the contralateral forepaw in cats. The lesion specified by SCHWARTZ [13] may be equivalent to a lesion in sensory-motor cortex plus ablation of dorsolateral frontal granular cortex in primates. Such a lesion might also serve to clarify the origin of the few spontaneous GSR-like fluctuations observed in the "frontal" records. These fluctuations may be the accompaniment of unobserved movements or, on the other hand, they may represent activity in some system which is responsive not to external events but to internal factors. This question could be partially answered by taking an electromyographic recording from the musculature near the GSR electrodes, or from the GSR electrodes themselves.

In this study the "frontal" GSR deficit has been examined under conditions involving negative reinforcement. KIMBLE *et al.* [3] also studied the orienting response in a situation which may involve a touch of anxiety or fear. It is still possible that the "frontal" animal may show GSR activity in response to positive reinforcers. This could be determined in a simple experiment using a "frontal" monkey, a Fels Dermohmmeter and a peanut.

The complete failure of the "frontal" animals to show conditioning of the GSR is not entirely in accord with the results obtained by ASHBY and BASSET [14] and by ELITHORN, PIERCY and CROSSKEY [15] in leucotomized humans. The discrepancy may be the result of species difference, or may be a reflection of the different type of lesion (leucotomy vs cortical ablation). However, the fact that the nearly total operation on animal 103 resulted in only a partial reduction of the GSR activity suggests that GSR conditioning might be accomplished if only a few fibers were spared in a leucotomized subject.

4.2. *Corticosteroids*

Although the overall average corticosteroid response of the "frontal" animals was equal to that of the normals, several striking differences were observed. The "frontal" animals showed less of a change in corticosteroid level between the first two samples on each of the three sessions. Also, two weeks after the shock session the "frontal" group showed elevated initial levels and retained the same relative elevation throughout this session.

The first of these facts lend itself to several possible interpretations. Two variables must be considered—intensity of stressor, and time. The interval between the first and second samples was filled by a relatively quiescent period of electrode placement and confinement to the chair; whereas that between the second and third samples represented confinement in a dark chamber in which shocks were administered during the first session. The drawing of the first blood sample and the confinement in the chair with its concomitant events seemed quite sufficient to elicit a considerable steroid response from the normal animals and only a lesser increase in the "frontal" animals. It might be said that the "frontal" animal is less responsive to *low* intensity stressors.

A second interpretation is one in terms of time. It is possible that the time course of the corticosteroid response is altered by frontal ablation and that extreme inertia of the "frontal" response produced the picture here observed. This would seem to be supported by the fact that the "frontals" showed elevated steroid levels in the second session two weeks after shock. The explanation may involve both time course and intensity variables. The simple expedient of using an indwelling intravenous catheter for drawing a number of samples during hours in the primate chair could provide a great step toward the unraveling of this problem. A more complete investigation of this question is now being undertaken.

4.3. *Summary*

Some of the questions this study sought to answer have been answered. The orienting response of the entire central nervous system is not altered after frontal ablation. EEG desynchronization still occurs. The peripheral mechanism needed for the production of a GSR is functional in frontally lesioned animals, and remains functional through repeated successive stimulation. A tone does not evoke GSR from the "frontal" animals even after it has been repeatedly paired with a strong shock. The corticosteroid response in the

"frontal" animal is of the same magnitude as that of the normal, both in the presence of shock and upon later exposure to the same environment in the absence of shock. There is, however, a marked difference between "frontal" and normal animals in the pattern of the steroid response.

It is tempting to seek a parsimonious explanation of both the GSR and corticosteroid results obtained in these experiments. An interpretation in terms of sluggishness or increased intertia of adaptive response was advanced in the discussion of corticosteroid data. Although this interpretation awaits the experimental verification outlined above, it finds unexpected corroboration in the slow return time found in the post shock skin resistance records of the "frontal" animals. These returns were not the result of an abnormally high peripheral reaction and, therefore, would seem to represent an abnormally slow reequilibration occuring centrally. If the GSR reflects only rapid changes in such central equilibrium, this interpretation might also account for the absence of the major portion of GSR activity following dorsolateral frontal ablation. The peripheral GSR would remain unreactive to the slower central change in the same fashion that a capacitively coupled amplifier would be unresponsive to a slowly occurring d.c. shift. Thus the functional decoupling would be the result of an altered central input to the GSR pathways not to a defect in the GSR mechanism itself.

If this tentative interpretation is correct it might be possible to find other homeostatic mechanisms which are similarly affected by frontal lesions. Slowness of change might be observable in blood pressure, heart rate, or adrenal medullary hormones. The autonomic measure mentioned could be studied in a situation similar to that of the main experiment reported here. The important variable would be the return of the system to pre-shock equilibrium. It is also possible that by varying the intensity and duration of the noxious stimulus a difference might be observed in the overall response. A short shock of low intensity might not have a chance to activate the system to the same extent in a "frontal" as in the faster reacting normal animal*.

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Résumé—Les déficits dans la réponse électrodermale déterminés par les lésions du cortex frontal dorsolatéral ont été examinés sous plusieurs conditions.

Sauf lorsqu'il y avait mouvement aucune réponse électrodermale n'était enregistrée chez les "animaux frontaux" en réponse à un stimulus nouveau, à un stimulus présenté dans une situation associée à un choc ou à un stimulus couplé de façon répétée avec un choc.

Cette absence de réponse contrastait avec la désynchronisation EEG apparamment normale se manifestant à un stimulus nouveau et avec la modification de la résistance d'amplitude normale dans la réponse au choc électrique.

La réponse des corticostéroïdes au choc et à la situation associée au choc fut mesurée. Sous toutes ces conditions le changement total du niveau des stéroïdes restait le même pour les deux groupes. Cependant les animaux avec lésions montraient des niveaux de stéroïdes élevés de façon notable deux semaines après la séance du choc initial et présentaient également un pattern différent des réponses dans certaines des séances.

Zusammenfassung—Die bei Affen mit fronto-dorso-lateraler Cortexläsion auftretende Abschwächung der galvanischen Hautreflexerregbarkeit wurde unter bestimmten Voraussetzungen geprüft. Falls keine Störung durch Bewegungsartefakte eintrat, konnte bei den Tieren keine Reflexantwort auf einen Neureiz registriert werden. Der galvanische Hautreflex fehlte sowohl auf Einzelreiz und Schock, als auch auf schockgekoppelte Serienreize. Dieses Ausbleiben einer Reflexantwort unterschied sich völlig von der reizbedingten, offensichtlich normalen EEG-Desynchronisation und der schocküberdauernden Amplitudenänderung im Hirnstrombild.

Die durch Schock und schockgekoppelte Reize gesetzten Corticosteroidreaktionen wurden einer quantitativen Analyse unterzogen. Die Gesamtverschiebung im Steroidspiegel war in beiden Gruppen gleich gross. Darüber hinaus war bei den cerebral geschädigten Tieren ein markanter Anstieg des Steroidgehaltes noch 2 Wochen nach dem Beginn der Schockperiode zu verfolgen, wobei eine unterschiedliche Menge an Steroiden zu verschiedenen Zeiten nach den Schocks nachgewiesen wurde.