A Method for the Investigation of Somatic Response Mechanisms in Psychoneurosis[1]

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THE PSYCHONEUROSES are characterized by states, such as anxiety, which appear to be emotional responses of pathologically increased intensity and duration. Little is known concerning the neural processes that underlie these disorders of emotional experience in psychoneurosis, although recent investigations have indicated that they are accompanied by abnormally increased excitability in both the autonomic and the central nervous systems (*8*). This paper is concerned with the development of a method that could be used for experimental investigation of basic central nervous system processes involved in psychoneurosis.

Largely in response to the influence of Cannon and his school (*1*), physiological studies of emotion have centered upon the increased activity in the sympathetic division of the autonomic nervous system, for example, increased heart rate and blood pressure. That increased parasympathetic excitation may also occur during strong emotion, however, is clearly indicated by such phenomena as involuntary evacuation of the bowel and fainting. Further, although seldom the subject of experimental investigation, the important role of the central nervous system in states of emotional excitement has long been recognized from clinical observations of hyperreflexia; and the electromyographic studies of Jacobson (*3*) have provided objective evidence of increased muscular tension in emotional disturbance. Consequently, any concept that attempts to integrate the facts concerning the physiology of emotion must take into account the events occurring in all main divisions of the nervous system.

The most widely accepted integrating principles concerning the physiological aspects of emotion are Cannon's concepts of emergency reaction and homeostasis. From the standpoint of these concepts some emotions are viewed as states of preparation for fight or flight; and the autonomic changes that accompany these emotions have been interpreted as events designed to ensure appropriate bodily conditions during such activity as, for example, greater blood flow to the muscles provided by increased blood pressure. The attention directed toward the homeostatic role of the autonomic nervous system in emotions has unfortunately led to neglect of the part played by the central nervous system. Muscular tension changes in emotion cannot be fully understood in terms of increased supportive function by the autonomic nervous system. The somatic motor system is characterized by shorter latencies of reaction than the autonomic system. In some emotion-producing situations skeletal muscle tension may complete a cycle of rise and return to prestimulation level, well before autonomic homeostatic mechanisms have had time enough to complete their cycles. This makes it appear necessary to seek a homeostatic mechanism with direct control over *somatic* (skeletal) motor activities by the *central* nervous system.

Recent discoveries in the physiology of brain-stem, thalamic, and cortical interrelationships have resulted in the description of the thalamic and brain-stem reticular systems, which may provide the necessary mechanisms for "somatic" homeostasis (*4, 7, 9*). Jasper (*4*) has found that stimulation of the thalamic reticular system effectively eliminates cortical afterdischarge resulting from sensory stimulation, and muscular afterdischarge produced by direct stimulation of the motor cortex. Moruzzi and Magoun (*9*) have shown that stimulation of the brain-stem reticular system will produce similar effects. From this recent work Jasper has concluded as follows:

> It seems, therefore, that there exists a separate regulatory system involving thalamic and other brain stem structures which acts upon the cortex, controlling the form and rhythm of the background of cortical activity upon which afferent impulses must act, and regulating local and generalized excitatory states of the cortex as a whole (*4*, p. 418).

The reticular system thus seems to exert a regulatory (homeostatic) effect upon cortical and motor activity. Since pathological anxiety appears to involve an excess of excitation in the somatic motor

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mechanisms (*8*), it is conceivable that this could be due to defective regulatory action (inhibitory) of some somatic homeostatic mechanism, such as the thalamic or brain-stem reticular system.

The method described in this paper was designed to bring experimental data to bear upon the question of defective somatic regulatory action in pathological anxiety. The following features were sought in devising the method: (1) The physical characteristics of external stimulation should be subject to exact measurement and control. (2) Stimuli should be of nonpainful intensity so that avoidance movements would not be produced. (3) In order to simulate the simple conditions of neurophysiological stimulation experiments, no voluntary reaction to stimulation should be required. (4) Recordings should be made from the somatic motor system with an apparatus capable of following the rapid changes in this system and of providing exact measurements of activity. (5) Given all of these features, the method, to be useful for studies of pathological anxiety, must distinguish clearly between the physiological activity of patients with pathological anxiety, and that of normal subjects.

The purpose of the present preliminary study was to test the differentiative capacity of a method that satisfies the first four criteria. If the method were shown to discriminate effectively between psychoneurotics and normals, it would be useful as a basic tool for further investigations of somatic response mechanisms in psychoneurosis.

As a technique that appeared likely to satisfy the criteria outlined above, we selected a procedure similar to the one used by Davis (*2*) in his studies of electromyographic response to strong auditory stimuli. Davis showed, with normal subjects, that auditory stimulation produces measurable electromyographic responses, even when subjects are instructed not to respond to the stimulus.

The subjects were 10 psychiatric patients, 5 of each sex, and 10 controls drawn from the medical and secretarial staffs of the hospital and matched with the patients for age and sex. The patients were all psychoneurotics in whom severe pathological anxiety was a prominent symptom. In one case of severe anxiety there were also symptoms suggesting early schizophrenia.

During the experiment the subject lay on a hospital bed. Action potentials from the extensor muscles of the right forearm were recorded by one channel of an Offner (Type D) EEG from silver leads, one placed over the extensor crest and the other at the wrist. The right forearm was placed in the prone position, and the subject was instructed to hold a rubber bulb in the right hand and to maintain a constant pressure on it throughout the experiment. Apart from this, he was asked to relax as much as possible. Davis (*2*) showed that induced tension increases the electromyographic response to sound. (In preliminary experiments we found that the induced tension provided by the rubber bulb was necessary for responses of sufficient magnitude to be easily recorded by our equipment.)

The auditory stimulus was a 1,000-cycle tone of 3 seconds' duration which was kept at an electrically constant intensity. This intensity was approximately 80 decibels above threshold, as determined by having 8 subjects compare the tone with an audiometer standard. Stimuli were 90 seconds apart, and there were 10 stimuli during the test proper. These were transmitted to the subject through binaural earphones. It should be mentioned that a constant feature of the stimulus was a sharp "on-effect," which gave the impression of a click of very brief duration.

The subject was instructed that he would hear a tone in the earphones. He was asked to disregard this tone and to make no response of any kind. Reassurance regarding the experimental situation was given as needed before and during the test. In a trial period before the test proper, 3 stimuli of increasing intensity were administered, the last one being of the same intensity as that used in the test. The subject was told that the last tone was what he would hear from then on, and that no louder tones would come.

Analysis of the electromyographic tracings was carried out by a method similar to that of Davis. The time periods chosen for measurement were as follows: (*a*) 1 second preceding the stimulus; (*b*) the 3-second stimulus period; (*c*) the 1-second period 12–13 seconds after the start of stimulation. These periods were divided off into fifths of seconds. For each fifth second the largest muscle potential spike was selected. This spike was measured in millimeters, and the millimeter measurements were converted to microvolts by reference to d-c calibrations.[3] This phase of the analysis required 5,000 individual measurements.

In statistical treatment of the data for each subject the 10 stimuli were averaged for each fifth second, and the mean amplitude of the muscle spikes determined. For group averages the medians of these means were calculated, because means would have been unduly influenced by the extremely high potentials of 2 subjects in the patient group. The chi-square test

[3] The largest spike in a given time period was selected for measurement in order to reduce the number of measurements required and to facilitate the actual measuring. The assumption was that total potential would parallel the largest spike. Frequency or spike duration was not measured because of methodological difficulties, and because previous studies with present recording methods have shown spike amplitude alone to be an adequate indicator of degree of muscular activity.

was used for determination of statistical reliability.

Median amplitude curves for the patient and control groups are shown in Fig. 1. The median prestimulus tension level of the patient group (24.5 μv) was somewhat higher than that of the controls (15.9 μv); however, this difference was not statistically reliable. In the first 0.2 second after onset of the stimulus, both patient and control groups showed approximately equal rise in tension. In the next fifth of a second (0.2–0.4 second), the control subjects' tension fell to approximately prestimulus level, whereas the potentials of the patients continued to rise, so that their response to stimulation was now double that in the first 0.2 second. For the remainder of the stimulus period, EMG amplitude for the controls showed steady tension at about prestimulus level, with only a slight increase in tension at about the 1-second mark (probably the "b-response" of Davis). On the other hand, the patients' curve, after reaching peak amplitude from 0.2 to 0.4 second after start of stimulation, began to descend relatively slowly, and with numerous oscillations. At the end of the 3-second stimulus period, it was still well above the prestimulus level. The measurements for the period, 12–13 seconds after onset of stimulation, revealed that complete recovery had taken place, and both patients and controls were at about their prestimulus tension levels.

The data shown in Fig. 1 reveal a major difference

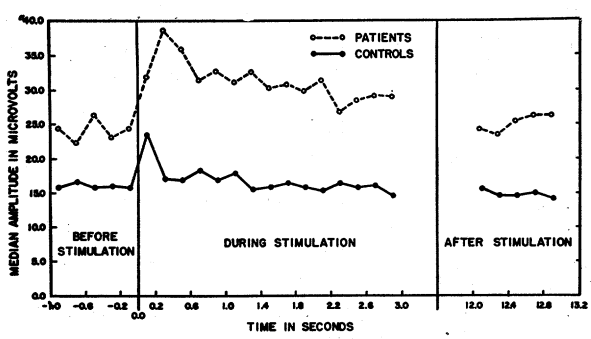

FIG. 1. Comparison of patients and controls with respect to median EMG amplitudes before, during, and after stimulation. Interval of measurement = 0.2 second.

in the response characteristic of the patients and controls. The initial tensional response (0–0.2 second) was equal in both groups, but, whereas the controls then returned to pre-existing tension levels, the patients showed further augmentation of response, and their response was prolonged over the entire period of auditory stimulation. Since the major qualitative and quantitative difference between the groups seemed to be evident particularly in the first second of stimulation, a more detailed analysis of this time period was carried out. The electromyograms for the half-second preceding stimulus onset and the first second of stimulation were remeasured for tenth-second intervals. These data for tenths of seconds were then averaged in the same way as those for fifths.

Fig. 2 shows response curves for patients and controls during the first second of stimulation. The curves are plotted in terms of median percent change from the average level preceding the stimulus. Percentages were used to avoid the possible influence of prestimulus tension differences on size of response. Fig. 2 shows clearly the identical electromyographic response (change) of patients and controls during the first 0.2 second of stimulation. From 0.2 to 0.3 second the control curve falls, and that of the patients continues to rise, reaching a peak at a time when the controls are almost back to prestimulus level.

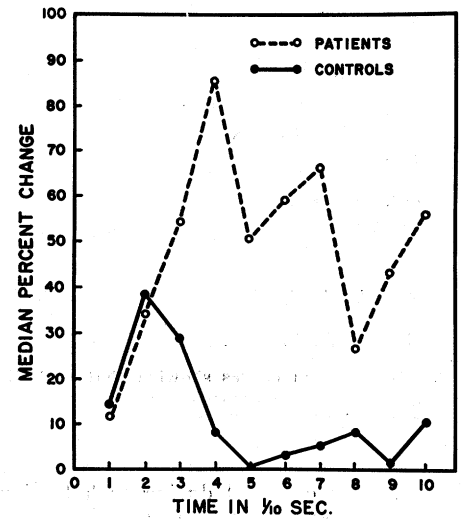

FIG. 2. Percent change in EMG response during first second of stimulation. Interval of measurement = 0.1 second.

The significant difference between patients and controls was shown in two ways: (1) Although immediate electromyographic response (change) upon stimulation was approximately the same in both patient and control groups, after the first 0.2 second of stimulation the change was decidedly greater in the patients. (2) The peak response of the patients occurred much later than the peak response of the controls. The statistical reliability of these differences was determined as follows: (*a*) The median response of the entire subject group for the period from 0.2 to 0.6 second was determined; 8 of the 10 patients showed responses greater than the median, and 8 of the 10 controls showed responses less than the median. The difference was reliable at the 1 percent level of confidence. (*b*) The tenth of a second during which the peak potential was reached during the first second of each stimulus was determined for each subject and for the group. Fig. 3 shows how

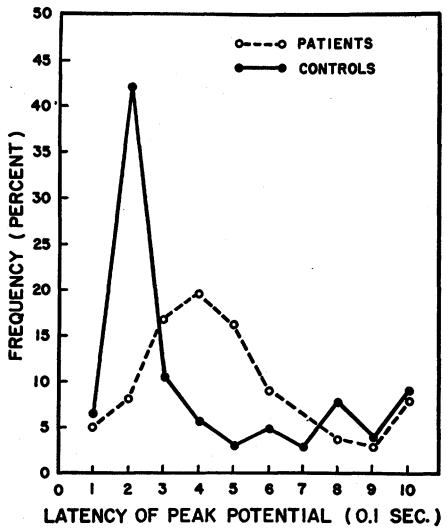

FIG. 3. Distribution curves showing that peak EMG responses in patients occurred most frequently after the normal latency for arm in the startle pattern (approximately 0.2 second).

often peaks were observed during each tenth-second interval in the patient and control groups. Whereas in the control group 50 percent of the peaks were found in the first 0.2 second, the patients showed only 13 percent of their peaks during this time. Moreover, the patients had 63 percent of their peaks from 0.2 to 0.6 second, whereas the controls had only 25 percent of their peaks during this period. These differences were highly reliable statistically. Individual comparisons between matched patients and controls also yielded a highly reliable difference with respect to the frequency with which peak responses occurred during the first 0.2 second of stimulation.

Onset of tonal stimulation at high intensity and the click ("on-effect") appeared to constitute an effective startle stimulus for the subjects. According to Landis and Hunt (*6*, p. 30) the latencies for the arm in the startle pattern range from 125 to 195 milliseconds. It was during this interval in our experiment that percent of change was approximately equal for patients and controls. This also agrees quite well with Davis' finding of what he calls the "a-response" (and which he relates to startle) with a peak at 0.2 second. It seems reasonable to assume, therefore, that the immediate startle reaction was approximately the same in patients and controls. The difference between the groups appeared *after* the 0.2-second period of reflex startle.

These results are what might have been expected from the hypothesis that, in anxiety, inhibition of cortical afterdischarge, through some regulatory mechanism, such as the reticular system (thalamic and/or brain-stem) is defective. The term afterdischarge is used on the assumption that *normally* the initial impact of click and tone has higher stimulating value (startle) than the continuation of the tone for the remainder of the 3-second stimulation interval. This assumption is based on our present finding with normal control subjects.

These positive findings make it seem worth while to proceed to a more detailed analysis of somatic response mechanisms in psychoneurosis. Kubie (*5*) has previously emphasized the importance of the factor of somatic overexcitation in anxiety. He has integrated observations from the clinical field, from Pavlovian conditioning, and from the work of Landis and Hunt on the startle pattern; and he has identified anxiety with the anticipation of explosive "irradiation" of excitation.

Present considerations suggest that the principal source of somatic overreaction in pathological states of anxiety may be defective somatic regulation by such a mechanism as the thalamocortical elaborative system. The present technique, modified according to the particular purpose of each separate experiment, appears a promising one for further investigations of somatic response mechanisms in psychoneurosis.

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