DISCUSSION ON PHYSIOLOGICAL MEASUREMENTS OF
"EMOTIONAL TENSION"

Dr. Brian Ackner (London):

In the everyday currency of our language we do not seem able to do without the coin
"emotion", but for scientific purposes it cannot be considered to be valid currency. There is
no such thing as emotion, it is an abstraction derived from certain subjective mental states
and associated behavioural and bodily changes. However, although the concept of emotion
as such cannot be the object of scientific investigation, reportable subjective mental states
and observable behavioural and bodily changes seem to be reasonable fields of enquiry.

James, in his "Principles of Psychology" in 1890 said:

"Our natural way of thinking about these coarser emotions (grief, fear, rage, love) is that
the mental perception of some fact excites the mental affection called the emotion and that
this latter state of mind gives rise to the bodily expression. My theory, on the contrary, is
that the bodily changes follow directly the perception of the exciting fact and that our feeling
of the same changes as they occur is the emotion."

The James-Lange theory is no longer acceptable but its predecessor is still implicit in much
of our everyday thinking. We still talk about anxiety causing bodily changes, anxiety
bringing on an attack of migraine and so on. Our tacit dualism still credits the inmaterial
with acting on the material. For the purpose of scientific investigation emotional states
should be regarded as subjective epiphenomena which may occur concomitantly with bodily
changes, both being accompaniments of stressful adaptations.

If both the subjective states and the observable changes arise from a common cause it
could be hoped that a full knowledge of one would provide us with some understanding of
the other two. However, we cannot satisfactorily quantify the subjective state, and the
behavioural expression of emotion is affected by cultural factors and influenced by the
immediate environmental situation. We do not even know to what extent the various bodily
changes are intercorrelated and whether changes in one field can be reliably accepted as
evidence of changes in others. Nor can we quantify "emotional stress" which is dependent
on factors both without and within the individual. We can expose subjects to comparable
stimuli or environmental factors, but it is the significance of the latter to the individual which
determines whether at any particular time they will be stressful or not.

In a study which has previously been reported, an attempt was made to overcome some of
these difficulties in the following way. A group of patients was selected on the basis of
subjective complaints, appearance and behaviour, all of which left no doubt that the term
"severe anxiety", as generally used, appropriately described an aspect of their emotional
state. In other words, this was a markedly anxious group as judged by both the common
internal and external criteria. This group was compared for experimental purposes with
a normal control group and another patient group, in both of which it was considered that
the internal and external criteria of anxiety were absent. Instead of trying to induce and
quantify stress, the opposite was attempted, namely the absence of stress by means of
barbiturate-induced sleep. A number of physiological variables were recorded but particular
attention was paid to changes in the amplitude of the pulse volume wave as recorded by the
finger plethysmograph. Anxious subjects tend to manifest a relative peripheral vasocon-
striction but the tendency is only a statistical one. It seemed possible that sleep might
release a stress vasocostricctor factor and that under suitable experimental conditions this
would be manifested by the occurrence of a greater degree of vasodilatation in the anxiety
group than in the other two groups. Thus, rather than vasoconstriction, perhaps the
tendency to vasodilatation during sleep might be found, under suitable conditions, to
differentiate between markedly anxious and non-anxious subjects.

The technique and findings of this work have been described in detail elsewhere (Ackner,
1956). The results revealed that the three groups differed significantly in the size of the
pulse volume during the resting state and in the increase occurring during induced sleep. The
marked pulse volume increase in the anxiety group distinguished the subjects in this group
from those of the other two groups.

It seemed profitable to attempt to correlate these findings with the findings of another test
which also used barbiturate sedation in the study of anxiety. Shagass (1954) has described
a test which he named the "sedation threshold". Starting from the observation that the more
tense a patient the greater the quantity of sedation required to produce a relaxing effect, he
deduced that it would follow that the amount of sedative required to attain a specified end-
point of sedation could be taken as a quantitative index of degree of tension. His method
was to inject intravenously Sodium Amytal at the rate of 0.5 mg. per kg. body weight every 40 sec., the subject being asked 25 seconds after each injection to repeat 5-6-7, 77, Massachusetts, &c. The sedation threshold was defined as the amount of Sodium Amytal in mg./kg. body weight required to produce an inflection point in amplitude curve of the EEG frontal 15-30 c/s activity; the inflection point must occur within 80 seconds of the clinical observation of slurred speech. In a series of publications, Shagass has developed and expanded the application of the sedation threshold test and I shall describe briefly the way in which he has successively revised the interpretation of his findings.

The sedation threshold was first found to be correlated with "tension" as judged clinically, using neurotic and control subjects. Subsequently the term "manifest anxiety" replaced "tension" and it was considered that the greatest amount should be found in anxiety states, the least in conversion hysteries with the mixed neuroses intermediate. This expectation was found to conform to the sedation threshold results for different neurotic groups (Shagass and Naiman, 1956).

The majority of the anxiety states were found to have obsessional personalities whilst hysterical personalities, in common with patients having hysterical conversion symptoms, tended to have a low threshold. It was therefore considered that the sedation threshold was correlated not only with the degree of manifest anxiety but also with the hystero-obsessional personality continuum. This was thought to be in line with Eysenck's introversion-extraversion dimension of personality.

The threshold was not found to be significantly correlated with degree of tension in schizophrenics but the recent acute cases were found to have lower thresholds than equally tense chronic ones. Considering that in "acute psychoses, ego functions are thought to be more impaired than in non-deteriorated psychoses of long duration, in which reparative processes have taken place" the hypothesis was formulated that the sedation threshold was negatively correlated with degree of impairment of ego functions. According to this hypothesis, the greater the degree of ego impairment, the lower the threshold (Shagass and Naiman, 1955). The sedation threshold in various psychotic categories was again found to conform to expectation, the organic cases having the lowest thresholds, followed by the acute schizophrenics, the agitated depressives and the chronic schizophrenics, with the "borderline states" having the highest threshold (Shagass, 1956).

This concept of impairment of ego function is used to explain why very agitated depressives, who are very tense, have low thresholds. Such depressions, which are also named psychotic depressions, are considered to have impaired ego functions whereas "neurotic depressions, providing they do not occur in severe hysterics, should have high ones". The sedation threshold is claimed to distinguish psychotic depressions from neurotic depressions and anxiety states, and to be able to give a good indication of the "immediate response" to E.C.T. (Shagass et al., 1956).

Finally, in one of his latest papers, Shagass (1957) reports that whereas the sedation threshold was thought to be very stable on repeat testing, it is in fact found to decrease in some subjects where the degree of affective disturbance diminishes. Accepting that this must limit its value in the field of diagnosis and personality Shagass states that the sedation threshold "may now be proposed as an objective index of therapeutic change".

In view of the claims made for the value of the sedation threshold and the amount of attention it has attracted, Dr. Pampiglione and I decided to collaborate in an investigation of this test, our aim being to study the relationship of the sedation threshold to:

1. The maximum vasodilatation occurring during barbiturate-induced sleep.
2. The affective state of the individual at the time of the test.
3. The diagnostic category.

Certain modifications of the procedure as described by Shagass were introduced to achieve a greater reliability and objectivity. To familiarize the subjects with the test procedure and reduce test anxiety, a preliminary EEG and plethysmographic test (without barbiturate injection) was carried out within a few days prior to the actual test. To minimize the disturbance due to the actual injection a Gordh's needle was used. This is an intravenous needle with a rubber diaphragm at the end, which after insertion into the vein can be left strapped to the arm. By this means it is possible to wait until the patient is suitably settled and then the injection is begun, without discomfort to the patient, by means of another needle inserted through the rubber diaphragm.

The course of injection was not influenced by the time at which the subject's speech might appear to slur. Instead his verbal responses were registered by means of a tape recorder synchronized with the EEG and plethysmographic record, and the injection was continued until after the patient had ceased to give any verbal responses.

The test procedure was carried out in one room with the majority of the recording apparatus housed in an adjoining room. After the EEG electrodes and finger plethysmographic
cup had been applied, the patient was allowed to relax for a period and then was interviewed by myself and by the doctor assisting with the injection procedure. As judged by his appearance, behaviour and statements about his own feelings at the time, he was rated for "manifest anxiety" on a 0-5 scale (this was not a very satisfactory procedure, but corresponded to the method employed by Shagass).

Subsequently the needle was inserted and at a suitable time Sodium Amytal was injected at a rate of 0·5 mg./kg. body weight every 40 seconds. Dr. Pampiglione was in the adjoining room supervising the recording and being able through earphones to monitor the injection proceedings and I was responsible for the timing of the injections and for making the patient repeat, 25 seconds after each injection, 57, 5-6-7, British Constitution (the latter was considered to be more suitable to our subjects than Massachusetts!). When the patient finally ceased to respond, one further injection was given and then we waited until there was no further increase in the pulse volume or until 3 minutes had elapsed after the last injection. Consciousness was then restored by means of an intravenous injection of Megimide.

Immediately afterwards we attempted to determine the time at which the patient had slurred. Starting at various points, the tape recording was played back on a number of occasions until the decision was jointly agreed by Dr. Pampiglione, the doctor giving the injection, our recordist and myself. The figure on the dial of the tape recorder was then noted and the corresponding figure on the continuous paper recording was found. From this the amount of Sodium Amytal injected at the time of slurring could also be derived.

This determination of the slur point, although made as objectively as possible, was not without some difficulty. Sometimes the speech became thicker, sometimes slower and sometimes hesitant. But we finally decided to define slurring at the first time a definite disturbance of articulation was noted. However, some patients, having slurred, afterwards articulated quite clearly until later they began to slur again. Furthermore, we noted that at times I repeated the words louder and more firmly, and yet at other times my speech actually appeared to slur!

I shall describe our plethysmographic results and Dr. Pampiglione will subsequently report our findings in respect of the sedation threshold. The test procedure was carried out on 56 psychiatric patients and in 51 of these there was a satisfactory plethysmographic record. The figure for the pulse volume increase was obtained by subtracting the maximum pulse volume occurring during the resting period prior to the test, from the maximum pulse volume occurring following the intravenous injection of Sodium Amytal. The resulting figure was then corrected for a standard 5 c.c. finger, the size of the finger tip enclosed within the plethysmographic cup having been measured following the test. In other words the results are expressed as the pulse volume increase in c.m.m./5 c.c. finger tip.

It was found that the 0-5 rating scale for anxiety produced too many small groups and so this six-point scale was reduced by combining ratings 0-1, 2-3, 4-5, to give three groups which represented clinically minimal, moderate and marked anxiety. The marked anxiety group had a mean increase of 11 c.m.m. and this is statistically significantly greater than the increase in the other two groups. The moderate anxiety had a larger mean increase (5-9 c.m.m.) than the minimal anxiety group (4-8 c.m.m.) but this difference is not statistically significant.

In the previous study (Ackner, 1956) it was found that an increase of 6 c.m.m. or more distinguished a markedly anxious group from a non-anxious patient group. Using this figure in the present study it was found that 11 of the 13 (84-6%) in the severe anxiety group and 9 out of 21 (42-9%) in the moderately anxious group had an increase of 6 c.m.m. or more. In the minimally anxious group 12 out of 17 (70-6%) had an increase of less than 6 c.m.m.

The figures reveal a number of similarities to those in the previous study. For instance in the marked anxiety groups of both the earlier and the present study the mean initial resting pulse volumes and the mean maximum increase are within a c.m.m. of each other (4-8 : 4-9; 12-3 : 11). However, there are some differences between the minimally anxious groups of the two studies. In the present study the mean initial volume is smaller (5 : 6-3), the mean maximum increase is larger (4-8 : 2-3) and 5 out of 17 patients have a pulse volume increase of 6 c.m.m. or greater. These differences may be due to the different population studied but there is a possibility that the difference in the two test situations may to some extent be of importance. In the earlier study the patient, after a previous resting test, lay on a couch and gradually went to sleep, usually with the aid of an oral barbiturate. This was apparently a quite unstressful procedure, in fact many patients spontaneously reported themselves as having enjoyed their "rest period". By contrast the present procedure has an element of stress, occasioned by the presence of two doctors and the anticipation of an injection which will impair consciousness. Now it is possible to demonstrate that, even though there may be no subjective or behavioural evidence of anxiety, an anticipatory attitude can cause various bodily changes (e.g. P.G.R. (psycho-galvanic reflex), pulse-rate, respiration, vasoconstriction, &c.) all of which appear to be part of a general alerting reaction. Whilst it is
unlikely that what we call anxiety can occur without these alerting responses being present, such alerting responses need not necessarily be accompanied by the subjective or behavioural features of anxiety. The presence of bodily changes, therefore, does not necessarily record the presence of emotional disturbance.

Under suitable experimental conditions the bodily changes occurring in those whose clinical features fulfil all the subjective and behavioural criteria of severe anxiety, can validly be contrasted with those in whom such criteria are lacking. Anxiety is, however, a concept whose features can be qualified but not quantified. It is, therefore, not valid to attempt to measure anxiety by using objective measures of bodily change.

REFERENCES


Dr. G. Pampiglione (London):

It is commonly believed that emotion causes visceral symptoms and influences the manifestations and course of disease.

A few hundred years ago emotion, the spring of action and conduct, used to sit in the heart, or in the belly, according to either Racine or Rabelais. Nowadays emotion is considered an agitation of the mind (as in the Oxford Dictionary) involving somehow the brain.

Clinical and intuitive methods are the most important sources of hypotheses concerning physiological mechanisms but the establishment of validity of such mechanisms cannot be based solely on clinical examples. Several studies made by physiologists on bodily changes in emotion showed that there is not a rigid parallelism between the occurrence and degree of agitation of the mind that the subject shows or says that he experiences, and the occurrence and degree of measurable changes in the body.

When Dr. Ackner and I began a combined study of some physiological variables during alertness, drowsiness and sleep it was soon apparent that the questions we were trying to answer had in fact multiplied the number of problems to be faced. In our measurements of electroencephalographic, respiratory, vasomotor and dermo-electrical changes, the variability in patterns from one subject to another was very marked and often difficult to predict. There were no trends and marked individual differences but these did not appear to be clearly related to particular clinical features. Moreover, in some of our investigations the physiological changes we measured revealed no gross differences between patients and members of the staff (Ackner and Pampiglione, 1955, 1957).

The first reports of Dr. Shagass' work (1954) were therefore read with great interest. Utilizing electroencephalographic and clinical changes induced by barbiturates he described a method of obtaining a quantitative index of tension in various groups of patients. This contribution appeared of importance not only because an objective pharmacological test was introduced in the quantification of symptomatology and grouping of patients but also because Shagass suggested particular physiological substrata of emotions. Moreover, he indicated the possibility that "the sedation threshold" as he calls some implications of his test "may reflect some basic neurophysiological aspect of personality organization"; as "not only the order but also the direction of the sedation threshold differences agrees with Eysenck's data and theory on introversion-extroversion" (Shagass, 1956).

Previous work has shown that the administration of barbiturates in man may evoke fairly constant and therefore predictable changes in the activity of the brain as measured electrically, particularly in the frontal regions. In addition when barbiturates such as Sodium Amytal are slowly injected into a vein a number of clinically observable changes may follow, such as drowsiness, difficulty in articulation, appearance of nystagmus, diminution in the speed and accuracy of movements, and later sleep. In normal subjects as well as in patients I had been unable to find any close correlation between the beginning of any of these phenomena and the development of the EEG changes in response to the drug. In another investigation with Dr. B. Mandelbrode and Dr. P. Sainsbury I was unable to establish any clear relationship between the time of appearance of the fast activity in the EEG and motor relaxation as recorded by a diminution in the action potentials from the muscles of the forehead and neck.
In the early days of electroencephalography it had been suggested that the records of anxious patients contained a great deal of fast activity. This is not so if we consider patients examined when no sedative has been given for many days and when no muscle action potentials mask the EEG.

The present investigation was undertaken both to establish the validity of Shagass’ test in our hands, and also to study, together with the EEG, other physiological variables such as those measured by finger plethysmography and chest pneumography, utilizing the same material and test procedure. From a pilot investigation a number of methodological problems arose. It was found that some patients were more apprehensive than others about the actual test and that the intravenous injection could not be carried out in several patients who had either inaccessible veins all the time or had marked vasoconstriction at the time of the test. Some patients were very restless with their arms and hands, increasing the difficulties of intravenous injection and of obtaining a reliable trace of the pulse volume waves from the finger.

Commonly when the doctor entered the room there was a change in the patient’s respiratory pattern and an increase in the muscle action potentials on the EEG and a moderate vaso-constriction. When the needle was inserted further vasoconstriction was seen as well as a marked increase in muscle activity which completely masked the EEG. A few minutes later the vasoconstriction diminished and the muscle action potentials became less prominent. Each subject was therefore tested twice within a few days and on the first occasion no Sodium Amytal was injected. This precaution demonstrated the variability in the autonomic reactions between subjects to the stress of the test situation.

It should be emphasized that the sedation threshold is based, according to Shagass, upon the combined occurrence of two phenomena, namely a change in speech (slur) and a change in the EEG (alteration in the gradient of the barbiturate-induced fast activity). In order to diminish the subjective element in the evaluation of “slurring” we defined slur as a disturbance of articulation, disregarding hesitation, slowness, and changes in phonation. In addition, as already described, the intravenous injection of Sodium Amytal at the appropriate dilution for the patient’s body weight continued until the patient became unresponsive even if one of us had the impression that slurring had occurred. Recently Thorpe and Barker (1957) demonstrated that 16 listeners were unable to agree regarding the onset of speech slurring in their seven patients examined with the technique described by Shagass. Our precaution of reassessing the patient’s disturbance of articulation a posteriori by repeating several times the run of tape recorder enabled us to check how often our preliminary judgment of slurring had been erroneous. In fact the assessment of a definite slurring point was doubtful in at least a third of our cases and many subjects appeared to speak normally for a time after a single occurrence of slur.

From the EEG aspect the methodology was more complex than at first pointed out by Shagass. In fact the fast activity response to barbiturates from different regions of the brain does not have the same growth curve in all patients though usually best seen in the frontal regions. Three EEG channels from 4 scalp electrodes (mid-frontal, right upper frontal, vertex, and left Sylvian) in standard position showed a considerable variability in the amplitude growth curve in each subject on each area of the brain. In view of previous personal experience patients suspected of cerebrovascular disease or other organic cerebral lesions were excluded from our series as their EEG response to barbiturates may be even more variable.

In order not to bias our impressions I did not begin to work out the measurements of the fast activity until 50 records had been collected. The correlations between the EEG and the clinical data were made later. The mean amplitude of the frontal barbiturate-induced fast activity (15–30 c/s) was calculated over a period of one second at regular intervals after each cubic centimetre of solution injected and a curve of the changes in the mean amplitude was built up. A special ruler had been designed by Shagass to count the waves and add up their amplitude. There is no difficulty in measuring the mean amplitude of the groups of waves with such a ruler but the measurement was very tedious and it may take up to two hours to plot the growth curve of one channel in each record. Moreover in 6 cases in which the count was repeated differences were common and often as large as 20%. This percentage of error may not appear very great to those working in the EEG field and is certainly not larger than the quantitative error of commercial wave analysers. Shagass (1954) compared his hand measurements with those of a wave analyser and found a close correlation between the two.

As a general trend the fast activity from the frontal regions is minimal at first but during the injection it tends to increase and eventually a maximum amplitude is reached. The curve obtained according to Shagass should be an S-shaped pharmacological curve, the final plateau being preceded by a fairly steep rise. The point at which the gradient changes is called by Shagass the “inflection point” and according to him the patient slurs within 80
seconds from this point. Such combination of events determines the "sedation threshold".

As previously described the intravenous injection in our study was not interrupted at the time of the "apparent" slurring, but was continued until the patient did not answer any longer. In this way the growth curve of the barbiturate-induced fast activity was not arbitrarily interrupted. It was found that although some of the curves obtained were  

shaped and slurring occurred near enough to the first inflexion, most of the other curves were complex with several inflexion points. In a few others the growth was slow but steady without definite inflexions until the patient was well asleep. Because of these various difficulties a definite sedation threshold as defined by Shagass could be determined in only a third of our cases. This determination was impossible in another third of the cases and appeared doubtful in the remaining third.

Our patients were divided into three groups according to the severity of their anxiety. There was no substantial difference in these three groups of patients in the amount of drug injected by the time the patient began to slur. Moreover the mean amount of drug necessary to render the patient verbally unresponsive was remarkably similar in each of the three groups suggesting that the epiphenomenon of anxiety does not bear recognizable relationship to the patient's resistance to a sedative of the kind employed. The amount of drug necessary to reach the maximum amplitude of induced fast activity was also similar in these three groups of patients. Where a "sedation threshold" could be determined there was no marked difference between patients with minimal anxiety and patients with severe anxiety.

In order to assess the relationship between the "sedation threshold" and the diagnostic categories studied by Shagass our patients were grouped as follows: (a) anxiety and neurotic depression; (b) obsessional states; (c) mixed neuroses; (d) endogenous depressions; (e) hysteria. The mean "sedation threshold" was found to be the same (6 mg./kg. body weight) both for the group of anxiety and neurotic depression and for the group of hysteria. This is in marked contrast with Shagass' findings and his theory of physio-pharmacological personality differences. The mean "sedation threshold" was slightly lower (5 mg./kg. b.w.) in the other three clinical groups. The amount of drug required to induce unresponsiveness was not substantially different for each of these diagnostic categories. There was no correlation between the sedation threshold and the time and amount of increase in the pulse volume waves in these groups, nor any constant correlation with the variations in pulse-rate.

When the injection of Sodium Amytal is continued at the same rate until the patient becomes unresponsive a single clear inflexion point in the curve of induced fast activity in the EEG is recognizable only in a limited number of cases. The slur therefore should be essential in deciding which of a number of inflexion points is to be used in determining the threshold. We cannot agree with Shagass that "slurring" roughly localizes the threshold while the EEG does it accurately.

It seems likely that a subjective judgment of "slurring" could unconsciously be influenced by a knowledge of the patient's emotional state (or diagnostic category) with the production of correlations not obtainable by more objective methods of investigation.

I wish to express my thanks to the Mental Health Research Fund for a personal grant in connexion with this investigation.

REFERENCES


Dr. Peter Sainsbury (Chichester):

Experimental studies which relate mental events and visceral changes due to autonomic activity are plentiful, but observations on changes in the activity of the skeletal muscles have not been numerous. This is surprising as changes in heart-rate or of the pupil, for example, have a less manifest relation to man's mental state than have the agitated movements of his hands or the tension in his facial muscles; and because the psychomotor behaviour of patients—their tense posture, their gestures and other expressive movements—are something we observe and avail ourselves of daily in the clinical examination and assessment of them. These muscular responses are the most conspicuous somatic manifestation of a patient's mental, and particularly his emotional, state.
The skeletal muscles can contract in only two ways: isometrically, that is, when tone increases without alteration in length; and isotonically, when the muscle shortens without change of tone and movement ensues. Ways must be found, therefore, to measure both tone and movements independently.

A. The Measurement of Tonic Activity

The patient is asked to lie down, relax and remain still. Bipolar surface electrodes are strapped over the muscles whose activity is to be sampled. The muscle potentials from any active motor units are amplified and fed to an oscilloscope and pen recorder to monitor the tracing. In a tense muscle the recorded tracing is even, continuously sustained, of relatively low amplitude and free from irregular bursts of higher amplitude activity produced by movements. This low amplitude activity may be due to reflex postural tone in the muscle, or it may arise centrally as when the individual is alerted or aroused emotionally. Next, the potentials are passed to an electronic integrator which operates a counter.

B. The Measurement of Movements

A movement appears on the record as a burst of activity of relatively higher amplitude, because in movement many motor units are rapidly recruited; and this type of activity is often superimposed on the sustained activity of lower amplitude due to reflex tone. To measure the movements, then, it is necessary to eliminate the effect of the tonic activity on the integrator. We have done this by placing a valve in the integrator circuit, the bias on the grid of which can be varied so that only those signals of a pre-selected amplitude are passed and counted. By inspecting the pen recording, a setting can be chosen at which all the low-amplitude, tonic activity is cut off: the larger spikes of movement remain to be counted.

When the integration reaches a certain value it triggers a valve which re-sets the integrator and passes a pulse to a pen which marks the paper record. The frequency of the marks varies with the integral (the area under the curve representing the potential); or, in practical terms, with the amount of movement the subject is making.

The procedure we use when recording movement is to have the patient sitting close to a table of a height such that he tends to rest his arms upon it: an arrangement which facilitates gestural movements of the arms. Small silver electrodes are placed over the maximum convexity of the forearm extensors and very light leads are passed to the back of the chair. The patient's clothes are then pulled over the electrodes and wires so that they are invisible to him and apparently they are soon forgotten. With this arrangement a record of shoulder elbow, wrist and finger movements is obtained. Should the patient rest his arm in a position involving continuous muscle strain—the chin supported in the cupped hand, for example—the tonic activity is usually still of a relatively lower amplitude and so not counted.

The apparatus is made to respond to the differing levels of muscular activity present in different patients by adjusting on the integrator the gains and settings which determine the amplitude of signals which will be counted.

Mr. J. C. Shaw, our research physicist, designed and calibrated the apparatus I now use at Graylingwell. At the gain commonly used, 670 microvolts give a 5 mm. deflection, and there is a linear relation between voltage and the recorded deflection of the pen. Similarly, on calibrating the integrator, a linear relation is found between the number of counts and the amplitude of the EMG tracing in millimetres. This relationship holds when the integrator is set to respond to all signals, as when tone in the muscle is to be recorded; and when it is set to respond only to those above 1, 2 or 3 mm. when movement is under investigation.

Satisfactory data on the daily stability of the integrator was also obtained.

A. Findings on Muscle Tension

Jacobson (1938) was the first to record the action potentials in the muscles and to show they varied in a predictable way with mental activity and especially with feelings of tension. Next Malmo and Shagass (1949) showed in one of a number of valuable papers that anxious patients, on painful stimulation, had more activity in their neck muscles than did controls; and that in those patients with head complaints, presumed to be muscular, the neck muscle activity was significantly higher than in those whose symptoms were of a cardiovascular kind. Wolff (1948) also recorded electromyograms from the scalp and back muscles of anxious patients with symptoms referred to these areas and found that the activity in the muscle varied with the intensity of the symptoms.

Muscular relaxation has been developed as an effective therapy in tense patients by Jacobson and by Garmany (1952). These observations prompted Gibson and me to study, by a controlled experiment, the relation between the symptoms complained of by anxious
patients and the activity in their muscles (Sainsbury and Gibson, 1954). In order to limit the experimental conditions to the simplest and so avoid subjecting the patient to a stress situation which cannot be evaluated reliably, we measured the muscle activity occurring in our subjects when they were relaxing on a bed in a position which avoided reflex postural tone in the muscles being investigated. The sustained activity recorded under these conditions we called "muscle tension" to differentiate it from the activity due to postural tone, and we considered it to have a central origin.

The patient's complaints of anxiety and tension were scored by means of an inventory. The items were largely derived from a survey undertaken by Dr. Gibson, in which 29 senior psychiatrists at the Maudsley Hospital were questioned about the clinical observations by which they ascribed "tension" to a patient. Their answers clearly suggested that it is the general appearance of muscular over-activity, as judged from facial expression, posture, and so on, in the anxious patient which the majority of clinicians refer to when they describe their patients as "tense".

The patient's feelings and symptoms of anxiety and tension scored on the inventory were related to the counts of muscle activity. Records were obtained from the frontalis muscle of 30 anxious patients, and of 26 healthy soldiers, the controls. The muscle activity of each subject was counted for two 7-minute periods while he relaxed on a bed with eyes closed.

We found:

1. The product-moment correlation between the counts for the two periods of relaxation was 0.9: the test-retest reliability of the counts was therefore satisfactory.

2. The counts from the frontalis of the patients were significantly higher than from the controls (P > 0.001).

3. Patients whose scores for feeling tense and anxious were above the median had significantly higher counts in both frontalis and forearm extensor muscles than those patients whose scores were below the median.

4. A significant concordance was found between the measures of tension in four distinct muscle groups, which suggests that the body musculature as a whole received an increased innervation in patients who are anxious. This view was supported by another observation: that tense patients who complained of tremulousness or of starting unduly, symptoms which might be ascribed to a general increased innervation of muscles, had significantly more muscle tension in both their forearm and frontalis muscles than did patients without these complaints.

5. Anxious patients with the symptoms referred to a definite area, such as tightness at the back of the neck, or a band across the forehead, showed significantly more activity in the muscles of that area than did those patients without such muscular symptoms.

I have recently collected further data for the hypothesis that muscle tension is positively related to emotional disturbance by measuring the activity in muscles of depressed and anxious patients on admission and again on discharge from Graylingwell. A significant decrease at the 5% level has been found.

To summarize these observations on muscle tension: the physiological measurement of it correlates with patients' anxiety scores and particularly with those complaints which may be reasonably ascribed to generalized muscle activity; also that the muscle tension is greatest in the muscle area to which the symptom is referred.

B. Findings on Movement

An increase of spontaneous bodily movements might similarly be expected to form part of the picture of emotional disturbance. In fact, many of the spontaneous movements we make are commonly referred to as "nervous habits" and as "expressive movements", thereby acknowledging their relation to mental processes.

There are two categories of expressive movements we have measured. First, gestures, which accompany speech and so assist in communication; and secondly, those comparatively meaningless, self-directed fidgetings which Krout (1935) calls "autistic gestures". Everyday instances of autistic gestures are: unnecessary tie-adjusting, nail-biting, or the restlessness of the agitated patient.

A brief description of some of our experiments on these movements follows.

1. The validity of the scores of movement obtained: the counts on the EMG correlated 0.9 with those obtained by time-sampling the movements directly observed.

2. This experiment was designed to study the reliability of the movement scores. The gestures of 16 patients at Graylingwell were measured while they were interviewed for ten minutes on two occasions, separated by at least twenty-four hours. In these sessions the interviewer took the role of an interested listener, his only contribution being to encourage the patient to continue. The scores for movement during the two interviews correlated highly (r = 0.87). Another 21 patients sat alone for ten minutes on two different days. The product-moment correlation for their autistic gestures' scores was 0.76. These figures, in
addition to establishing the reliability, also suggest that the level of gestural activity is a characteristic attribute of the personality. I sought confirmation of this by correlating the movement scores made by these patients, while filling up a questionnaire, when alone, and while being interviewed. The concordance between the movements made in these three situations was significant at the 1% level.

(3) To test the hypothesis that emotional disturbance is accompanied by increased psycho-motor activity: (i) The autistic gestures of 10 patients selected for leucotomy at the Maudsley and of 15 healthy controls, were counted by the EMG. The subjects in both groups were left sitting alone in a room for two periods of ten minutes each, on two separate occasions. The patients' mean movement score was significantly greater than the controls' (P > 0-025).

The 10 patients were re-examined three months following their leucotomies. In the 8 patients in whom the operation effected an improvement in anxiety, tension and depression, there was a significant decrease of both gesture during interview and of autistic gesture while alone. Changes in the patients' gestures were measurable, therefore, and were related to changes in their emotional state (Sainsbury, 1954).

(ii) I have recently correlated the movements made by 21 patients sitting alone (their autistic gestures) and their ratings on an anxiety scale (r=0.76); and patients diagnosed as depressive or agitated depressive made significantly fewer movements than those in whom anxiety was the predominant feature.

(4) I aimed in another experiment to relate the amount of gesture to the content of a psychiatric interview planned to include topics of a kind likely and unlikely to disturb the patient.

The interview was recorded on tape. The patient also wore a throat microphone and this was fed to a pen which marked the electromyogram and integrator record when he was speaking. In this way we were subsequently able to synchronize the content of the interview on the tape with the movement score by the pen recorder.

12 patients were given 16 interviews. The mean movement score for the half-minute intervals in which the topics discussed with the patient were those predicted as disturbing to him, were compared with the mean score during intervals in which his feelings were unlikely to have been disturbed. In all the interviews there was more gesture in the periods when disturbing topics were introduced: this increase was significant in 14 of them.

In three interviews every utterance of the patients was independently rated for the disturbance of affect manifested and the ratio of movements to words was scored for each utterance. It was found that the more affectively charged the utterance, the greater was the amount of gesture.

Finally, 4 patients were interviewed on a second occasion and it was found that those topics which had been accompanied by most gestures in the first interview were also those with most gesture in the second (Sainsbury, 1955).

Changes in the innervation of the voluntary muscles have, therefore, been found with emotion and they seem to be as much part of the physiology of emotion as are the changes in visceral innervation. In the last experiment, for example, the heart-rate was recorded concurrently with the gestures and was also found to increase with the disturbing topics. Evidence is accumulating that the hypothalamus and mid-brain mediate changes in the voluntary as well as in the autonomic pathways with emotional arousal or, indeed, with arousal of a less urgent kind such as when the individual simply attends to his surroundings.

The central discharge to muscles in these circumstances can be demonstrated as changes in spontaneous movement and in muscle tension. The former are evident clinically as restlessness, or as an increase in gesture; the latter as a stiff posture, or furrowed brow. Subjectively this increased muscular innervation is described and experienced, we found, as irritability, feeling on edge, as tightness or even pain in the muscle, or simply as feeling "tense" (a term 79% of our patients used to mean that they were unable to relax muscually).

It would be consistent with our findings, with the reports of the majority of our patients, and with what we found to be the practice of the majority of psychiatrists, if the term "tension" were limited to the muscular manifestations of the emotion.

REFERENCES


Dr. Denis Hill (London) said that while the recognition of stress was a relatively easy clinical task and its treatment, especially by the new tranquillizing drugs, had made some progress, its measurement had always proved a baffling problem. There were different ideas about the nature of emotional tension. Could it be described only as an over-readiness to respond to meaningful stimuli, or is it an enduring state of activity within the nervous system, or perhaps a biochemical lesion? If the first of these was correct then all evidence of tension should disappear in sleep when the meaning of stimuli ceases; on the other hand if the second alternative was correct then even in deep sleep there might be some positive evidence of abnormal function which could be related to the state called "tension". Clinical evidence concerning the sleep of tense patients favours the second hypothesis. The problem of understanding how emotional tension could persist as an organization within the nervous system as an enduring state is a problem common to all those issues concerning the relationship between neuronal and mental activity. To solve one such problem may be to solve all. For example, if we understood how memories were retained within the nervous system through waking and sleeping life, through the violent disturbances of concussion and E.C.T. and many other circumstances which profoundly alter cerebral activity, we might well understand how an emotional state such as tension could persist.

From the physiological point of view the probable mechanisms concerned with tension are largely inaccessible; only the effects are discernible. The speakers had shown how studies at two ends of the system could help in such measurement. Recent studies by Drs. Ackner and Pampiglione had shown that it was the same nervous mechanism which caused physiological arousal of the cortex and changes in the calibre of peripheral vessels. No doubt this applied too, as Dr. Sainsbury suggested, to the changes in muscular tone. It seemed highly probable that the mechanism involved was the much-discussed mid-brain reticular system.

Shagass' idea of a sedation threshold seemed at first sight a fairly simple thing. From his early work he had suggested that the sedation threshold is a constant for the individual, perhaps a constitutional character. Shagass' most recent paper, however, and the new evidence of Drs. Ackner and Pampiglione made it clear that the sedation threshold is not a constant for the individual, but varies with his degree of manifest tension and anxiety and with illness, and also can be altered when the techniques by which the threshold is measured are altered. Moreover it is for some patients a measurement which cannot be made because there is no clear end-point or because the two measurable factors—fast rhythm facilitation and slurring of speech—dissociate in some patients. It would seem that the sedation threshold does not vary directly with what is called clinically emotional tension, or rather that the method may point the way to differentiating various types of tension. The results of these studies on cortical function contrast strongly with those made on peripheral structures, described by Dr. Sainsbury and by Dr. Ackner. The ingenious sampling devices developed by Dr. Sainsbury enable him to observe a part of muscular functions—and he can vary his sampling mechanisms to observe any part of these functions which he wishes. Moreover this can be done with minimal stimulation or threat to the patient. The correlations with clinical tension which emerge seem quite clear, just as do those relating to vasomotor tonus which Dr. Ackner had described. These methods seem to hold great promise for future work.

The results on the sedation threshold, inconsistent with those of Shagass, which Drs. Ackner and Pampiglione had described, are not surprising. In the case of peripheral vasomotor tonus and in the case of muscular tone it might be surmised that the mechanisms by which the excitability of spinal motor neurones are influenced by the mid-brain reticular system are relatively simple, involving few neuronal pathways—simple that is, in comparison with the mechanisms involved when one thought of the action of the mid-brain system in the opposite direction, on the cerebral cortex.

Shagass himself evoked the idea of another variable, "impairment of ego functions", to explain the differences in threshold between anxious neurotics and anxious psychotics. It is surprising that these results place hysterics, psychotic agitated depressions and organic dementia in the same functional category.

There was little doubt that studies such as they had heard about held great promise for the future. The benefit to clinical practice and to therapeutics in psychiatry would be enormous if reliable measurement of emotional tension could be achieved. The research worker in this field is, however, always limited by the fact that the more he wishes to observe, the more he interferes with what he observes. The patient attached to a laboratory full of apparatus is liable to resemble the traveller prepared for a journey into outer space and experiences no doubt some of the apprehension which such travellers will experience. One could only hope that the ingenuity and resourcefulness which the speakers had obviously brought to bear on their work would continue to open the way for them in the future.
Dr. Murdo Mackenzie (London):
The opening papers have stressed the importance of measurement in the investigation of emotional tension: and it seems prudent to emphasize the need for uniformity in the use of words to denote the several entities which are being examined.
The word "depression" has been cited, but in such different contexts as to suggest a lack of precision in the employment of the term. The diagnosis covers, *inter alia*, simple depression, reactive depression, endogenous depression and agitated depression.
The word, in ordinary language, is used in two disparate ways: one connotes lowering, as for instance, in a barometer to indicate atmospheric change; the other depicts reduction in vigour, as for example, a recession in the trade cycle. The variant of meaning is rendered clear, in each instance, by the object to which it refers.
Depressive states, as observed in the clinic or consulting room, reveal similar differentiation. One patient affirms "I am all in and can face no more strain"; another declares "It has been the same all my life. I may appear, in a sort of way, successful; but, au fond, I am bogus and know it".
The first averment refers, manifestly, to a sense of loss of vitality; and the second to a lowering or denigration of the individual personality, by a psychological device. It would be of assistance, then, in the delineation of states of emotional tension if the distinctive uses of the word were preserved.
The term "personality" has been quoted in the discussion; and it, too, suffers from a general lack of consistency in application. Dr. Leopold Bellak, for instance (1956), writes of the "psychoanalytic theory of personality". Dr. Margaret Mead, on the other hand, in the same series (1956), employs "personality" to denote "... the total pattern of the individual's behaviour..." The other essays show a corresponding diversity of approach.
Personality, in everyday language, suggests the notion of a distinctive individual trait; and an interesting feature of great personalities is the way they show enduring contrast in presentation. Sir Winston Churchill, for instance, came to one of his peak points in oratory in the speech on "the beaches of Britain": in this the emphasis is on an accentuation of the significance of the "here and now". President Franklin Delano Roosevelt, on the other hand, in a similar situation, created the fireside chat on the wireless; in this the emphasis in presentation resides in a diminution of the significance of the "here and now".
If personality be defined as presentation with individual contrast, the concept might be used to further the observations on muscular contraction, in individuals under conditions of strain, described in one of the opening papers. Some patients asseverate that they, invariably, respond to stress by a manifest gesture: others affirm that they, automatically, assume the "poker face". The duality might be found to correspond with differing methods of presentation in personality contrast.
Precision in the use of terms can, at all events, only assist in promoting accuracy in the quest for "physiological measurements of 'emotional tension'".

REFERENCES