

RECORD GROUP

29 227

81-83/087

VOLUME

B01 34 3769

FILE

604-5-69

000001

Founded in 1887 by STANLEY HALL

OFFPRINTED FROM

THE AMERICAN JOURNAL OF PSYCHOLOGY

EDITED BY

KARL M. DALLENBACH
UNIVERSITY OF TEXAS

AND

M. E. BITTERMAN
BRYN MAWR COLLEGE

E. B. NEWMAN
HARVARD UNIVERSITY

LEO POSTMAN
UNIVERSITY OF CALIFORNIA, BERKELEY

WITH THE COÖPERATION OF

E. G. BORING, Harvard University; W. K. ESTES, Stanford University; J. P. GUILFORD, University of Southern California; HARRY HELSON, Kansas State University; E. R. HILGARD, Stanford University; FRANCIS W. IRWIN, University of Pennsylvania; F. NOWELL JONES, University of California, Los Angeles; G. L. KREEZER, Washington University; D. G. MARQUIS, Massachusetts Institute of Technology; ARTHUR W. MELTON, University of Michigan; W. C. H. PRENTICE, Wheaton College; T. A. RYAN, Cornell University

A VISUAL RATING SCALE FOR PRINTS OF FINGER-SWEAT

JOHN KENYON and HERBERT F. MÜLLER, McGill University

March, 1963, Vol. LXXVI
pp. 140-142

Published by The American Journal of Psychology, Department of
Psychology, University of Texas, Austin, Tex.

Founded in 1887 by STANLEY HALL

OFFPRINTED FROM

THE AMERICAN JOURNAL OF PSYCHOLOGY

EDITED BY

KARL M. DALLENBACH
UNIVERSITY OF TEXAS

AND

M. E. BITTERMAN
BRYN MAWR COLLEGE

E. B. NEWMAN
HARVARD UNIVERSITY

LEO POSTMAN
UNIVERSITY OF CALIFORNIA, BERKELEY

WITH THE COÖPERATION OF

E. G. BORING, Harvard University; W. K. ESTES, Stanford University; J. P. GUILFORD, University of Southern California; HARRY HELSON, Kansas State University; E. R. HILGARD, Stanford University; FRANCIS W. IRWIN, University of Pennsylvania; F. NOWELL JONES, University of California, Los Angeles; G. L. KREEZER, Washington University; D. G. MARQUIS, Massachusetts Institute of Technology; ARTHUR W. MELTON, University of Michigan; W. C. H. PRENTICE, Wheaton College; T. A. RYAN, Cornell University

A VISUAL RATING SCALE FOR PRINTS OF FINGER-SWEAT

JOHN KENYON and HERBERT F. MÜLLER, McGill University

March, 1963, Vol. LXXXVI
pp. 140-142

Published by The American Journal of Psychology, Department of
Psychology, University of Texas, Austin, Tex.

A VISUAL RATING SCALE FOR PRINTS OF FINGER-SWEAT

An index of finger-sweat has within recent years been widely used in work dealing with physiological measures of activation.¹ This method recommends itself for reasons of ease and convenience, particularly since the introduction of finger clamps for the purpose of taking such measures.² In a recent study, a visual rating scale for

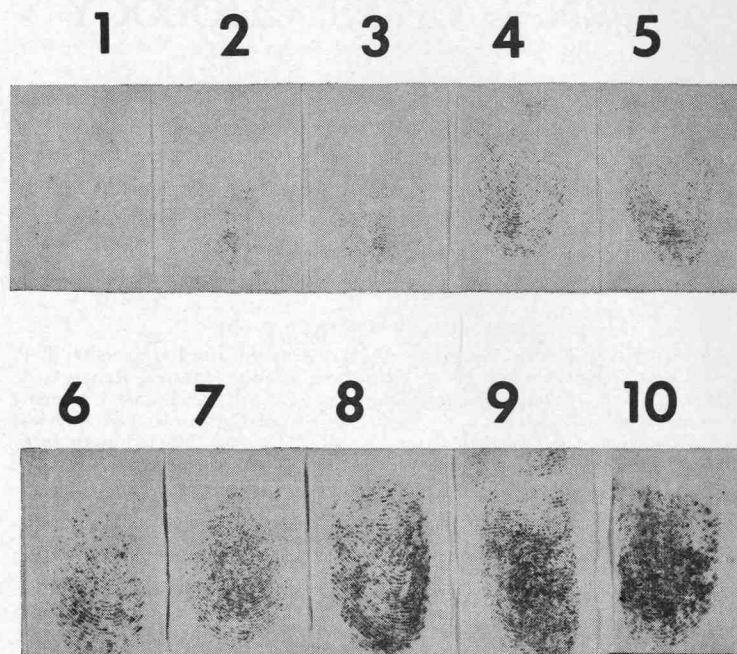


FIG. 1. GRADED SERIES OF PRINTS USED IN THE RATINGS

prints of finger-sweat was used, representing a further simplification of the method.³ This scale is the subject of the present paper.

* From the Allan Memorial Institute, McGill University. This research was supported in part by the Department of National Health and Welfare, Defence Research Board (Canada), and the United States Public Health Service.

¹ J. J. Silverman and V. E. Powell, Studies on palmar sweating: I. *Amer. J. Med. Sci.*, 208, 1944, 297-305; Studies on palmar sweating: II *Psychosom. Med.*, 6, 1944, 243-249; Roy Gladstone, *An Investigation of Certain Variables Affecting the Results of a Group-Test of Palmar Sweating*, Final report, Sub. Contract No. HumRRo 650-005 with George Washington University, 1954; R. C. Wilcott, Silverman-Powell index of sweating vs. skin conductance and a humidity index of surface moisture, *J. comp. physiol. Psychol.*, 62, 1959, 33-36.

² D. A. Chambers, Rowena Pasternak, and H. F. Müller, A clamp for finger-sweat prints, *Perceptual and Motor Skills*, 11, 1960, 35-38.

³ H. F. Müller "An exploratory investigation of 'clinical-type' physiological indicators in the measurement of arousal," Thesis for the Diploma in Psychiatry, McGill University, 1959.

The procedures for obtaining the sample of prints used in this study were the same as those previously described by Chambers *et al.*⁴ The samples included a wide range of prints differing in darkness. A selection of prints was made for a visual 10-pt. scale with subjectively equal darkness intervals, including the lightest (No. 1) and the darkest (No. 10) prints available. Densitometric values, obtained for the prints, were plotted against the visual rank-order. Photographic copies were then made of this visual scale.

A new sample of 100 prints was taken, and each print was classified according to its correspondence with the darkness of the scale-prints. For each print, the values obtained from the densitometric readings, the visual scale, and the photographic copies of the scale were compared. Test-retest and inter-judge reliabilities were computed.

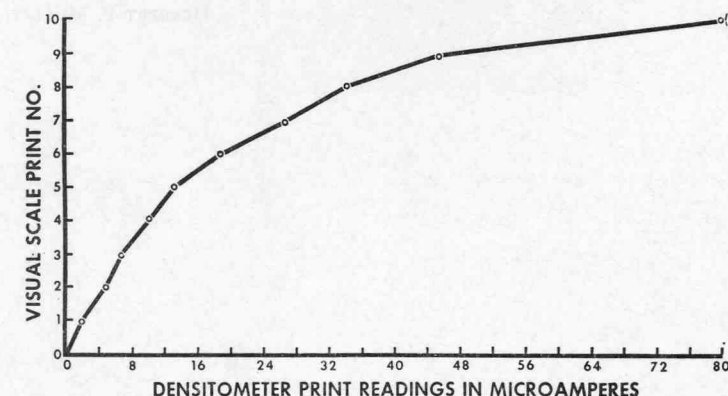


FIG. 2. VISUAL SCALE-RATINGS AS A FUNCTION OF THE DENSITOMETRIC READINGS

(The last point on the curve is probably beyond 80 microamperes; uncertain due to limitation of the densitometric scale.)

The 10-pt. scale resulting from the selection of prints is shown in Fig. 1. The plot of ranks on this scale against corresponding readings obtained with the densitometer is presented in Fig. 2. The relation is curvilinear and proved to be logarithmic. This was evident from a plot of the logarithms of the densitometric readings against the corresponding visual ranks, which resulted in a straight line for the range between Ranks 2 and 9. Obviously it is a Weber-Fechner function.

The various analyses for estimating validity and reliability yielded high correlation coefficients. The correlations between the densitometric values and ratings on both the visual scale and the photographic copy of the visual scale were respectively $r = 0.97$ and $r = 0.92$. The ratings on the photographic copy and on the visual scale correlated $r = 0.97$, and the test-retest correlation for the photographic copy was $r = 0.99$. The coefficients of inter-judge reliability (two judges) for the densitometer, the visual scale, and the photographic copy of the visual scale were $r = 0.96$, $r = 0.97$, and $r = 0.95$ respectively. (The N of prints was 100 in every case.) In

⁴ Chambers, Pasternak, and Müller, *op. cit.*, 36-37.



addition, a reliability rating was obtained for three judges using the photographic scale. An analysis of variance technique was applied to the ratings and yielded a value of $r = 0.93$ for the reliability of ratings for one rater and $r = 0.97$ for the reliability of ratings for three raters ($N = 100$).

Discussion. The results of this study show that finger sweating may be measured as reliably by a visual scale as by a densitometer. Such a scale has obvious advantages: It requires little equipment and is considerably faster. In addition, as many photographic copies of the visual scale can be reproduced as desired. The information obtained with the visual scale is adequate for most testing situations. As may be judged from Fig. 2, quite fine discriminations may be made with the visual scale, particularly at its lower end. This is an advantage in practice, because the majority of ratings fall in the range of light prints.

McGill University

JOHN KENYON
HERBERT F. MÜLLER

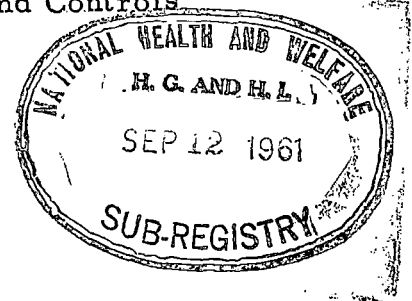
Re
1962-63
C.S.

Dominion-Provincial Mental Health Grant 604-5-69

Physiological Studies of Psychiatric Patients and Controls

Progress Report for 1960-1961

Robert B. Malmö



Publications

Seven recent publications (and papers read) are listed at the end of this report. As may be seen from reference to these publications, our experimental programme is progressing very well, and the findings are being published.

Effect of Induced Tension on Pain Perception

Experimentation on this project has consisted of an extensive study of 3 young male subjects who have served during many sessions. The psychological judgment required is difficult and a rather long training period is required before experimental conditions may be introduced. Furthermore, there were many technical difficulties to overcome in the pilot studies. The statistical analysis of the data from the main study are now underway. Although it is too early for final conclusions it appears likely from inspection of the data that all 3 subjects may show lowered pain threshold with induced tension. It also appears that the threshold lowering effect is produced by a rather wide range of induced tensions.

Large-scale Study of Psychiatric Patients

This study has yielded one publication (Chambers, D.A., Pasternak, R., and Mueller, H.F. A clamp for finger-sweat prints. Percept. Mot. Skills, 1960, 11, 35-38), and a further manuscript is being prepared by Mueller and Kenyon. This paper will deal with the rating scale for finger-sweat prints, and it will also present results from the longitudinal study of a number of psychiatric patients observed over long periods (months) of time.

Physiological and Behavioural Study of Children

Normative psychophysiological data on children are very much needed. This has been a badly neglected area for some three decades. Now underway is a comprehensive study of nursery school children, applying our new electroencephalographic (EEG) measures, in conjunction with physiological techniques, to behavioural studies in these young children. From quick inspection of the data it is clear that there are some very striking (and surprising) differences shown by the children. But these data require much further analysis before conclusions may be drawn.

Changes in EEG and Other Physiological Measures during Serial Mental Performance

In this study by MacNeilage, the general relation between EEG alpha amplitude and mental effort was investigated to determine whether EEG was distinctively related to variations in task complexity and other "mental" aspects of performance, or whether EEG varied concordantly

with other physiological variables, merely as one of several indicants of activation.

Results clearly showed a concordant variation of EEG alpha and other physiological measures in all cases except one, that of improvement in performance following a brief rest (the so-called "reminiscence" phenomenon). Here EEG alpha appeared to indicate a distinctive neural (probably cortical) shift not accompanied by autonomic changes of the same magnitude.

Unexpected findings during serial performance were that EEG alpha and beta usually covaried in amplitude and were dominated by effects of motor function rather than by more "mental" factors. These discoveries appear very important for our understanding of brain function, especially in relation to the EEG.

PUBLICATIONS & REPORTS

- Feldman, S.M. Differential effect of shock as a function of intensity and cue factors in maze learning. J. exp. Psychol., 1961, (in press).
- MacNeilage, P.F. Activation correlates of performance on a simple repetitive mental task. Paper read at Eastern Psychol. Ass., Philadelphia, April 1961. (a)
- MacNeilage, P.F. Changes in EEG and other physiological measures during serial mental performance. Unpublished doctoral dissertation, McGill Univer., 1961. (b)

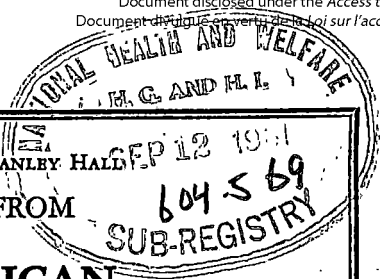
Malmo, R.B. Activation. In A.J. Bachrach (Ed.), Experimental foundations of clinical psychology, New York: Basic Books, 1961 (in press).

Malmo, R.B. Cognitive factors in impairment: a neuropsychological study of divided set. Amer. Psychologist, 1961, 16 (in press).

Malmo, R.B., & Davis, J.F. A monopolar method of measuring palmar conductance. Amer. J. Psychol., 1961, 74, 106-113.

Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and on performance. J. exp. Psychol., 1961, (in press).

A list of Laboratory Publications, the manuscripts by Feldman and Pinneo, and the reprint of the article on recording palmar conductance are attached hereto.



Founded in 1887 by G. STANLEY HALL

OFFPRINTED FROM

THE AMERICAN JOURNAL OF PSYCHOLOGY

EDITED BY

KARL M. DALLENBACH
UNIVERSITY OF TEXAS

AND

M. E. BITTERMAN
BRYN MAWR COLLEGE

E. B. NEWMAN
HARVARD UNIVERSITY

LEO POSTMAN
UNIVERSITY OF CALIFORNIA, BERKELEY

WITH THE COÖPERATION OF

E. G. BORING, Harvard University; W. K. ESTES, Indiana University, J. P. GUILFORD, University of Southern California; HARRY HELSON, University of Texas; E. R. HILGARD, Stanford University; FRANCES W. IRWIN, University of Pennsylvania; G. L. KREEZER, Washington University; D. G. MARQUIS, Social Science Research Council; GEORGE A. MILLER, Harvard University; W. C. H. PRENTICE, Swarthmore College; T. A. RYAN, Cornell University.

A MONOPOLAR METHOD OF MEASURING PALMAR CONDUCTANCE

By ROBERT B. MALMO and JOHN F. DAVIS, McGill University

March, 1961, Vol. LXXIV
pp. 106-113

Published by The American Journal of Psychology, Department of
Psychology, University of Texas, Austin, Tex.

Founded in 1887 by G. STANLEY HALL

OFFPRINTED FROM

THE AMERICAN JOURNAL OF PSYCHOLOGY

EDITED BY

KARL M. DALLENBACH
UNIVERSITY OF TEXAS

AND

M. E. BITTERMAN
BRYN MAWR COLLEGE

E. B. NEWMAN
HARVARD UNIVERSITY

LEO POSTMAN
UNIVERSITY OF CALIFORNIA, BERKELEY

WITH THE COÖPERATION OF

E. G. BORING, Harvard University; W. K. ESTES, Indiana University; J. P. GUILFORD, University of Southern California; HARRY HELSON, University of Texas; E. R. HILGARD, Stanford University; FRANCES W. IRWIN, University of Pennsylvania; G. L. KREEZER, Washington University; D. G. MARQUIS, Social Science Research Council; GEORGE A. MILLER, Harvard University; W. C. H. PRENTICE, Swarthmore College; T. A. RYAN, Cornell University.

A MONOPOLAR METHOD OF MEASURING PALMAR CONDUCTANCE

By ROBERT B. MALMO and JOHN F. DAVIS, McGill University

March, 1961, Vol. LXXIV
pp. 106-113

Published by The American Journal of Psychology, Department of
Psychology, University of Texas, Austin, Tex.



A MONOPOLAR METHOD OF MEASURING PALMAR CONDUCTANCE

BY ROBERT B. MALMO and JOHN F. DAVIS, McGill University

The apparatus and method described in this paper have been successfully used in a number of studies conducted in our laboratory, and we wish to make them available to our co-workers.¹ The monopolar method (which makes use of an indifferent or reference electrode and an active electrode) was selected because we wished to study the variations of skin-conductance at a single site.

Fig. 1 presents the circuit diagram for the apparatus. It is convenient to work with resistance units during recording, converting to conductance-units later on. A subject's (*S*'s) resistance is the unknown factor in the Wheatstone bridge that is approximately balanced by means of a General Radio Type 1432 Decade Resistance Unit, the unbalanced portion being recorded by the direct coupled (*DC*) amplifier and recording galvanometer. Three decades, in units of 1,000, 10,000, and 100,000 Ω will serve all requirements for recording from *S* (a separate millivoltmeter for checking the electrodes will be described later).

For purposes of recording the palmar conductance (*PC*), the direct coupled (*DC*) amplifier should have maximal available gain of 200 (al-

* Support for the development of this method has come from the following sources: Medical Research and Development Division, Office of the Surgeon General, Department of the U.S. Army: Contract Number DA-49-007-MD-626; National Institute of Mental Health, National Institutes of Health, U.S. Public Health Service: Grant Number M-1475; the Department of National Health and Welfare (Canada); Defence Research Board, Department of National Defence, Canada: Grant Number 19425-04; and the National Research Council of Canada: Grant Number A.P. 29.

Grateful acknowledgment is made to Mr. W. Mundl for technical assistance.

¹S. M. Feldman, Differential effect of shock as a function of intensity and cue factors in maze learning. Unpublished Doctoral dissertation, McGill Univer., 1958; R. B. Malm, Measurement of drive: An unsolved problem in psychology, in M. R. Jones (ed.), *Nebraska Symposium on Motivation*, 1958, 229-265; Activation: A neuropsychological dimension, *Psychol. Rev.*, 66, 1959, 367-386; M. M. Schnore, Individual patterns of physiological activity as a function of task differences and degree of arousal, *J. exp. Psychol.*, 58, 1959, 117-128; R. G. Stennett, The relationship of performance level to level of arousal, *J. exp. Psychol.*, 54, 1957, 54-61; R. G. Stennett, The relationship of alpha amplitude to the level of palmar conductance, *EEG Clin. Neurophysiol.*, 9, 1957, 131-138.

APPARATUS

107

though gain used at the sensitivity that we generally employ is only 33), and its drift should be no more than 2 mv. per hr. Since such amplifiers are not readily available from commercial sources (whose amplifiers have too much or too little gain), one must either be specially constructed or else converted from one of the models available. At present, we use an Epsco-type 8105 DC amplifier in which we have reduced the gain, by a factor of 10 or 15 to 1, by eliminating one of the stages.

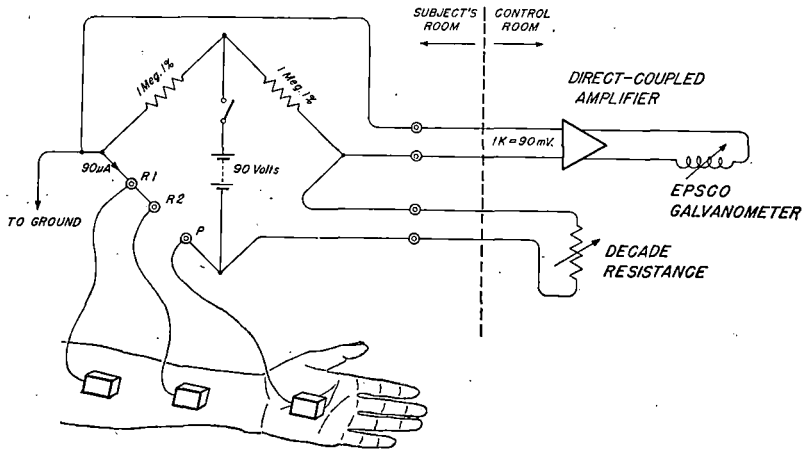


FIG. 1. CIRCUIT DIAGRAM FOR SKIN-RESISTANCE RECORDING.
R1 and R2 designate the two reference electrodes. See text for explanation.

If the current through *S* is too small, then the voltage drop through the skin resistance might be of the same order of magnitude as the skin voltage (*i.e.* the more or less steady biological potential which is always present across the skin). This makes it difficult to differentiate between skin voltage (Tarchanov) effects and skin-resistance (Fere) effects, and we wished to study the latter effects exclusively. On the other hand, if the current is too large, there is always the danger of polarization of the electrodes. We selected 90 μ A. as a compromise which avoids both difficulties. Since our bridge circuit is symmetrical, the current is the same in each leg.

In arranging the apparatus, if two adjoining rooms are available, it is preferable (less distracting for *S*) to install the Decade Resistance Unit, DC amplifier, and chart drive in a control room away from *S*.

Connecting PC reference leads to ground. With multiple-channel recording, especially when electromyograms (EMGs) are included, it is desirable



to ground *S* to prevent 60-cycle interference from various sources. In the absence of this ground, it is often impossible to secure *EMGs* at high gain that are free from artifacts, and sometimes the 60-cycle artifact is present even in the *PC*-tracing. Fig. 1 shows the reference leads going to ground (there are two ground electrodes connected in parallel).

It should be noted that grounding the body at more than one point is decidedly unwise. With more than one ground, and with the *PC*-bridge grounded at some point, the current that is being passed through *S* will be

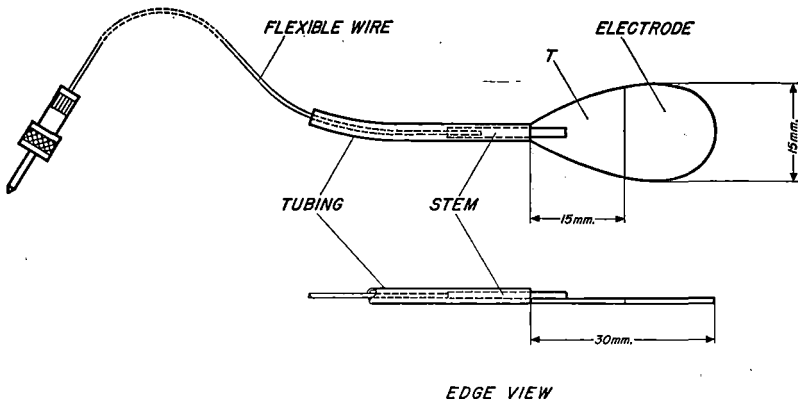


FIG. 2. DIAGRAMMATIC SKETCH OF *PC* ELECTRODE.
T represents area that is covered by Tygon primer and cement.

divided between grounds, and it is uncertain how the current will be divided.

Some of the unfavorable consequences of grounding *S* at two points on his body are as follows. The difference in resistance obtained from reversing the leads to the bridge (forward-reverse difference) is increased over the single-ground; voltage measurements on the body are disturbed, and *EKG* artifacts may appear in *EMG* tracings that had previously been free of them. Although we employ two ground electrodes, the body may be considered (for all practical purposes) to be grounded at only one point because of the small distance between electrodes.

Construction of the electrodes. Fig. 2 illustrates the *PC*-electrode which is cut from 1/32 in., fine grade silver, and is then filed and polished with steel wool until smooth. The lead wire (10 ft. of Belden 8014) is soldered to a stem which, in turn, is soldered to the small end of this electrode. After the electrode is cleaned with carbon tetrachloride, the solder joint is covered with one coat of Tygon primer and three thin coats of Tygon



paint, leaving a length of 15 mm. of uncovered silver. A solderless plug is attached to the other end of the lead for insertion in the input box of the bridge.

The purpose of the stem shown in Fig. 2 is to facilitate cleaning of electrodes. It serves as a handle while holding them. To provide this stem, solid tinned copper wire (No. 18 AWG) is doubled into the shape of a U, is soldered to the silver at one end, and to the lead wire at the other end. Actually, the solder extends along the whole length of the stem to give

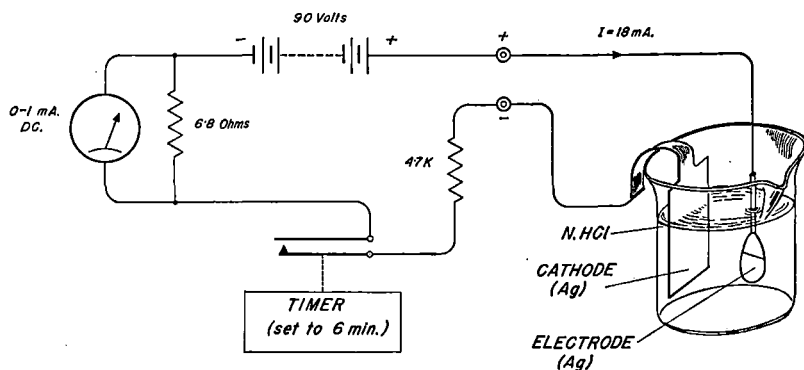


FIG. 3. CIRCUIT FOR CHLORIDING PROCESS.
See text for explanation.

stiffness to the wire. For strain relief, to prevent the wire from breaking at the point where it is soldered, a two-inch length of tubing is placed around the wire as illustrated in Fig. 2. We use Birnbach Biraco Tubing (no. 14 B & S).

Chloriding the electrodes. A diagram of the chloriding apparatus is presented in Fig. 3. The chloriding current of approximately 18 mA. is determined primarily by the value of the series resistor (4,700 Ω). Since the resistance of the chloriding bath is always a small fraction of this value, the current remains rather constant (within approximately 10%). Furthermore, the resistance of the meter-shunt is negligible as compared with the other resistors in the circuit, and hence this shunt has no effect on the operation of the circuit.

The electrode is cleaned, connected to the positive pole of the chloriding power supply, and immersed in the chloriding bath of normal hydrochloric acid. Next, the cleaned silver cathode is placed in the chloriding bath and connected to the negative pole of the chloriding power supply.

A Gralab timer is then set for a chloriding time of exactly 6 min., after which time the chlorided electrode is removed from the HCl bath, rinsed with water, and blotted dry with a soft, clean cloth. Darkening on the surface of the electrode should appear uniform. Specks on the surface of the electrode usually signify insufficient care in cleaning, and it is likely that such an electrode will not meet the voltage- and resistance-tests, and will have to be rechlorided. These tests, rather than the appearance of the electrode, determine, however, whether the electrodes may be used in

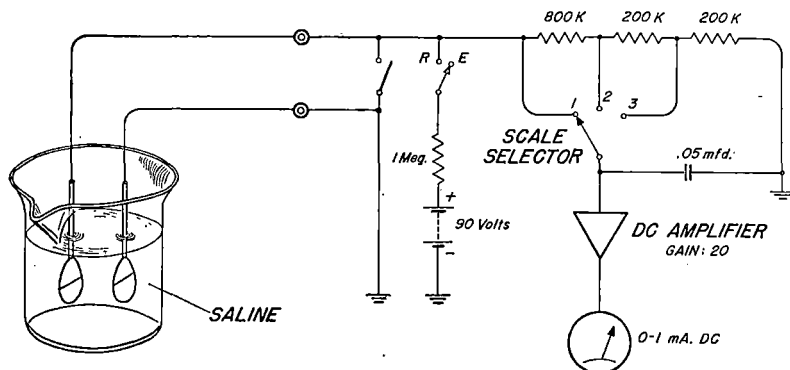


FIG. 4. CIRCUIT FOR VTVM THAT IS USED FOR CHECKING VOLTAGE AND RESISTANCE OF ELECTRODES IN SALINE.
"DC Amplifier" refers to direct-coupled amplifier.

Scale selection	E Millivolt range	R Range of resistance in ohms
1	0- 10	0- 100
2	0- 50	0- 500
3	0-100	0-1000

recording or must be rechlorided. If the electrode fails to meet the tests, it is best to remove the chlorided surface completely (with steel wool), and to repeat the entire chloriding process. The electrode is stored dry with protection against abrasion and mechanical damage.

Voltage measurements. Chloriding should bring the leads 'in balance,' which means that the electromotive force generated by the pair of electrodes placed in a physiological (0.9%) saline solution should be close to zero. Fig. 4 illustrates the method that we use to check electrodes for voltage and resistance. The DC amplifier for the vacuum tube voltmeter (VTVM) may be of low gain (e.g. 20) because it is used with a 1- mA. DC meter instead of with a recording galvanometer. The amplifier, how-



ever, should be so designed that the grid current is close to zero at the input. In practice, we have found that it is very desirable to have two DC amplifiers, one for measuring *PC* (illustrated in Fig. 1), and one for the purpose of taking measurements from the electrodes in saline (illustrated in Fig. 4).

One should strive to produce electrodes with no more than 1 mv. of unbalance. This requirement may seem over-strict since 1 mv. represents only 10 Ω in this circuit. Experience has demonstrated, however, that with an initial reading as low as 1 mv., the balance will usually hold during the session, whereas with higher initial readings (of the order of 5 mv.), electrode voltage may drift up to 15 mv. or more during the session, thus introducing a progressive voltage error and possible electrode-resistance increase.

Resistance measurements. After the voltage measurement, using the same electrode-checking instrument (Fig. 4), a current is passed through the electrodes and the ohm-scale is read for the two resistance values of the pair of leads, plugged in first left-to-right, and then right-to-left. A good pair of electrodes usually yields a resistance value in the neighborhood of 100-150 Ω . Reversing the electrodes will produce a different reading, but with good electrodes, the two resistance values should be within approximately 50 Ω of each other.

Electrode placement: Active lead. The electrode placed on the palm of the hand (or sole of the foot) is the active lead, and care must therefore be taken to avoid abrading the skin in the area where this lead will be placed. Gentle stroking with absorbent cotton saturated with ether cleans effectively without abrading the skin. A cellulose sponge (a block, 1 in. \times $\frac{3}{4}$ in. \times $\frac{5}{8}$ in.) serves to hold the electrode (in a slot that is cut in the sponge). After saturating the sponge with physiological saline (not the more concentrated conducting jelly), the sponge is placed on the *S's* palm, is covered with a thin piece of rubber sheeting to prevent loss of fluid, and sponge and sheeting are held securely in place by a band of elastic material (Lastonet). It is a wise practice to apply the saturated sponge to the palm as early as possible in the session that physical saturation of the palmar tissue with saline may reach its peak before the recording session commences, thus eliminating artifacts produced by the gradual soaking of the skin with saline. That is, by allowing the saline from the sponge to soak into the palm during the remainder of the preparation, any fall in resistance due merely to penetration of the salt solution into the skin should be practically complete by the time the experimental session commences.

Reference leads. In treating the skin for placement of the two reference

electrodes (see R_1 and R_2 in Fig. 1), there are two chief goals: (a) to bring resistance down as low as possible with no more than minor discomfort for S during the process, and (b) to bring the resistance of these leads to the same constant value in each S . One purpose of (a) is to prevent 60-cycle interference from various sources. In the absence of such a low-resistance ground lead, it is often impossible to secure artifact-free EMGs at high gain, and sometimes the 60-cycle artifact is present even in the PC tracing. Another purpose of bringing the reference resistance down to a low value is to minimize the contribution from reference areas to the total recorded resistance. The purpose of (b) is to minimize the undesired variation due to individual differences in resistance of the skin lying under reference electrodes. Experience in our laboratory has demonstrated that these two goals may be achieved by treating the skin of the forearm in such a way as to bring the contribution of the two reference leads, when connected in parallel, down to approximately 500 Ω . For example, with a total resistance of 10,000 Ω , approximately 9,500 Ω may be attributed to the palmar resistance and only about 500 Ω to the reference resistance.

In practice, the skin is treated in such a way as to yield a reading of 2,000 Ω (1,000 Ω from each electrode) between the two reference electrodes when they are plugged into the ohmmeter together (*i.e.* not in parallel). The final value of 500 Ω is approximated by connecting the two 1000- Ω resistances in parallel. To produce this value (approximately 500 Ω), it is essential to strive for equality in resistance from one reference lead to the other. In practice, this means that during the time that the skin is being treated, each reference electrode should be plugged in the ohmmeter with a third electrode from time to time in order to ensure that the resistance value for reference-1 to the third electrode is equivalent to the value for reference-2 to the third electrode at all stages of the resistance-reduction procedure. Unless shifts in baseline of palmar conductance are very rapid, the palmar electrode is a convenient one to employ as the third electrode.

The ventral (relatively hairless) surface of the left arm accommodates two electrodes nicely, leaving the right arm free for performance. Reference electrodes may be placed one on each arm, but this placement introduces a large EKG-artifact in EMGs from the left arm. The two reference electrodes are placed about midway between wrist and elbow on the left arm, spacing them at least 2 in. apart (and at least the same distance from other leads).

Treatment of the skin on which the reference leads are to be placed



proceeds as follows: Absorbent cotton, saturated with a grease-dissolving detergent, such as 'phisoderm,' is used to clean the skin and to reduce its resistance. Rubbing with the saturated cotton for about a minute and a half is advised. After thorough rubbing and cleaning, the electrodes are placed in the same way as that described for the palmar electrode.

At this point in the procedure, each reference electrode in turn is plugged in the ohmmeter with the third electrode in order to determine whether one reference electrode has an appreciably higher resistance than the other. In that event, further treatment is applied first to the skin under the high-resistance electrode. This treatment consists in careful application of the pointed tip of a wooden applicator (a thin wooden stick, 1-2 mm. in diameter) to the skin immediately under each electrode. The end of the applicator should be sharpened to a point, and this point dipped in electrode jelly before being applied to the skin. Although it is sometimes difficult to bring the resistance between reference electrodes down to 2,000 Ω , experience has shown that with skill and persistence, this may be achieved without vigorous scraping or injury of the skin, and with relatively little discomfort to S.²

In a recent experimental series, it was found that even after intervals of 2 hr. or more between pre- and post-calibration checks, the resistances of reference leads held very close to original values. Such findings indicate that purely physical factors such as evaporation of the saline solution in the sponge (that might increase resistance) or deeper penetration of saline into the skin (that might lower resistance) were well controlled.

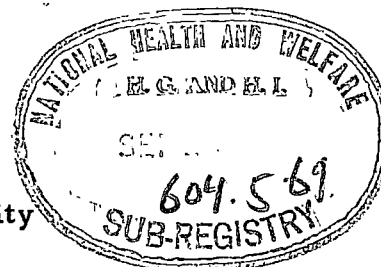
² Though we do not claim that the present monopolar method is necessarily superior to bipolar methods of recording *PC*, the success of the monopolar method in the studies cited encourages us to believe that it represents an advance over methods that we had tried previously. The work of one of us (J.F.D.) with measurement of DC potentials in the operating room has led to findings that have accelerated the present application of the monopolar method to *PC* recording.

The Effects of Induced Muscle Tension During Tracking on Level
of Activation and on Performance¹

Lawrence R. Pinneo²

Laboratory for Psychological Studies

Allan Memorial Institute, McGill University



Since Courts' (1942) review, several experiments have been reported on the effects of induced muscle tension on performance, but to the author's knowledge none have been concerned with the basis of the relationship. In a theoretical paper Meyer (1953) suggested that the effects of induced muscle tension on performance are the result of simultaneous responses occurring at the level of the motor cortex. Though a useful concept, Meyer fails to consider the involvement of the reticular activating system and level of arousal in muscle tension phenomena (Delafresnaye, Adrian, Bremer and Jasper, 1954).

In contrast to Meyer, Malmö (1959, p. 370) suggests that induced tension may be one of the ways in which activation level can be varied, and that activation level, in turn, is a function of the reticular activating system. If induced tension is indeed a reliable means of varying activation level, then in addition to behavioral effects, induced tension should also produce regular and consistent changes in the various physiological indicants of activation. Experiments by Freeman and Simpson (1938) encourage this idea. They have shown that palmar conductance increases as a function of muscle tension induced in the legs, and Freeman (1938) has shown that palmar resistance changes linearly with induced muscle tension during performance. However, these experiments were unfortunately limited to the one physiological measure of palmar conduct-

ance, while Malmo (1959) and Schnore (1959) have shown the need for a wide coverage of physiological functions in studies of activation. The purpose of this experiment was the exploration of the relation between induced muscle tension, activation, and level of performance.

Method

Subjects

A total of 38 McGill University undergraduate and graduate males, ranging in age from 18 to 30, served as Ss. All were paid \$5.00 for the three-hour session. Only right-handed Ss with normal hearing were used.

Apparatus³

Muscle Tension Induction System. A modified hand dynamometer was used for inducing tension. When the spring steel of the dynamometer was compressed, two strain gauges mounted on each side of the spring were deformed. This resulted in a change of the electrical resistance of the wire in the strain gauge proportional to the strain or the grip on the dynamometer. Each strain gauge was an arm of a balanced Wheatstone bridge. Changes in the resistance of either or both of the strain gauges upset the electrical balance of the bridge. The change in signal due to this imbalance was fed into a push-pull, strain gauge amplifier.

Part of the amplified signal was used to continuously monitor and permanently record the S's instantaneous performance, and part was fed back to the S to enable him to maintain his level of pull within prescribed limits. These limits were set by an a-c microammeter relay equipped with two movable contacts. Both contacts could be positioned anywhere on the meter face and

Pinneo

-3-

their relative positions could also be varied. By calibrating the meter deflection in kilograms, and using each of the meter-relay contacts as a switch to an audio oscillator, "low" and "high" tension limits could be determined. When the S did not squeeze the dynamometer sufficiently hard, he heard a 600-cycle tone via one contact, while if he squeezed too hard he heard an 800-cycle tone via the other contact. The desired pressure was obtained when S gripped the dynamometer such that he heard no tone at all.

Auditory Tracking System. This system was similar to that described by Davis, Stennett, and Quilter (1957), except that tracking was done with the right foot instead of with the hand. In addition, the function generator used in this system consisted of a potentiometer bridge circuit whose imbalance signified error in direction and intensity. The S, who was blindfolded for purposes of EEG recording, sat in a semi-reclining chair and performed a simple tracking task in the following manner. He first depressed the foot pedal until it came against a mechanical stop after a movement of eight degrees of arc. He next allowed the pedal to return to its normal position at the same rate at which the pedal was depressed. If the rate of pedal movement down and up was in exact conformance with that of the function generator, the S was "on target" and he heard no sound in his earphones. Failure to keep pace with the function generator was signaled to the S by means of a 1000 cps tone in his earphones. The loudness of this tone varied as a function of the S's "distance off target," and direction of error was cued by whether the tone appeared in the right or the left earphone. "Down" errors (i. e., pressing down on the pedal at too fast a rate or lagging behind on the way up) were indicated by

the tone appearing in the earphone on the right; conversely, "up" errors were indicated by the tone appearing in the earphone on the left.

The number of up and down errors made per trial and the total distance the S was off target (DOT) were the measures of performance. Number of errors were recorded on two electrical impulse counters, and DOT was recorded graphically by means of an electronic integrator with a four-second discharge rate (Davis, 1959). This latter system was calibrated such that a certain distance off target in degrees produced a given deflection on the ink writer, which was then measured with special scales.

Physiological Recording Apparatus. Three chart drives with ink-writing galvanometers were used for recording physiological tracings and error data. The primary record for electromyograms (EMG) from active and passive muscles, two EEG measures, and the heart rate (EKG), were recorded on a standard Grass IV A electroencephalograph (paper speed 25 mm/sec.). Signals taken from the output of the driver stages of the Grass were led into electronic integrators which summated the muscle potentials over successive four-second periods, and were recorded as deflections on a second chart drive. The EEG was quantified by passing these signals through band-pass filters (Ross and Davis, 1958) with cut-offs to provide bands of 8-12 cycles per second and 18-27 cycles per second. Outputs of the filters were also integrated and recorded on chart drive two (paper speed: 1 mm/sec.).

Chart drive three (paper speed: 2.5 mm/sec.) was used to record respiration (obtained with a Phipps and Bird pneumograph and recording

tambour), palmar conductance, DOT, and the output of the strain gauge amplifier.

Recording Electrodes. All electrodes except those used for palmar conductance were made of cellulose sponge of one cubic inch. Each had been dipped in normal saline and electrode jelly and attached to the skin of the S by means of elastic lastonet bands or elastoplast tape. Heart rate was recorded from electrodes placed on the chest wall immediately below the heart, and on the right shoulder. Electrodes for EMG recording were placed on the extensor muscles of the right and left forearms and the calves of the right and left legs in accordance with Davis' Manual of Surface Electromyography (1959). Two bipolar EEG leads were taken from the nondominant hemisphere from frontal and occipital positions equivalent to placements C2-F2 and P2-O2 of the 10/20 system used at the Montreal Neurological Institute (Jasper, 1941).

Palmar conductance (PC) was recorded from the left palm by means of a monopolar method described by Malmö and Davis (1959). Three silver, silver-chloride electrodes were employed: one in the palm and two parallel reference electrodes on the ventral surface of the left forearm.

Other Apparatus. The S's chair and tracking apparatus were located in a separate room from that of the amplifying and recording devices. It was a relatively soundproof room, entirely shielded with copper screening to reduce electrical interference, and was supplied with an air conditioning plant for the maintenance of constant temperature. Separating the S's room from the experimenter's control room was a double wall with a screened obser-

Pinneo

-6-

vation window. Junction boxes and cables provided connection between the S's electrodes and the instrumentation in the control room. A two-way inter-communication system was employed for instructing the Ss and answering questions.

Line voltage for critical instruments such as d-c amplifiers, integrators and the electroencephalograph, was regulated with a Sorenson Model 3000S regulator. The temperature in the control room was maintained at about 72 degrees Fahrenheit to prevent voltage drifts from excessive heat.

Procedure

All instructions pertaining directly to the experiment were read to each S. When a S first arrived he was informed that the experiment was designed to study the effects of induced muscular tension on his ability to perform a simple psychomotor task, and that the effects of this tension would be determined by simultaneously measuring certain physiological variables and the muscle tension directly. An assistant then prepared the S by placing the electrodes as discussed above.

S was then instructed by the experimenter in the method of inducing tension and in the method of maintaining a given level of tension. Subjective levels of tension were then determined by having the S squeeze the dynamometer very lightly for 30 seconds. After a rest period, this procedure was repeated twice more. The average reading of his pull in kilograms at the end of the three, 30-second periods was designated the Ss Very Light (VL) tension. After another rest period, S was asked to squeeze the dynamometer as tightly as he could and to hold this for 30 seconds. The reading at the end of the 30

Pinneo

-7-

seconds was designated his Maximum (Max.) pull. Following another rest period, the S was required to subjectively bisect the apparent pull on the dynamometer of the previous two instances (Very Light and Maximum), and to hold this for 30 seconds. This also was repeated twice more, and the average of the three dynamometer pulls in kilograms at the end of the 30-second periods designated as S's Very Heavy (VH) tension. Similarly, a Medium (M) tension was determined by bisecting the apparent pull between the Very Light and the Very Heavy, as determined by the average of three readings. Light (L) and Heavy (H) tension conditions of pull were then arbitrarily chosen as the physical mid-points, in kilograms, between the Very Light and Medium, and the Medium and Very Heavy, respectively.

A headset with independent earphones for tracking was placed on the S's head and adjusted for comfort. S was then instructed by the experimenter in the tracking procedure as described above. Each trial consisted of two complete cycles of the tracking pedal being depressed and released. This required 96 seconds; 96 seconds' rest was also given between each trial. One practice trial was followed by the learning trials to criterion. The criterion was the tracking of three successive trials without improvement, but never less than six nor more than ten.

A ten-minute rest period and a glass of water were given between learning and tension trials. No smoking was allowed. During the rest period all equipment was again checked to conform with calibration standards.

The five tension conditions of Very Light, Light, Medium, Heavy, and Very Heavy were given once only to each S in an order determined by a

table of random numbers. The only difference between the tension trials and the learning trials was that during the entire trial S was required to maintain the particular tension on the dynamometer in addition to tracking. After two practice trials, Ss had no difficulty in simultaneously tracking and maintaining a steady level of dynamometer pull.

Two control conditions of No Tension and Exertion (EX) were also randomly presented with the tension conditions. The No Tension control did not differ from the learning trials. Its purpose was to gauge the effects of performance with no tension. The Exertion condition was introduced in an attempt to determine the effect of induced tension alone (without tracking) on the various physiological measures. This condition required each of eight Ss to squeeze the dynamometer at a pressure equivalent to his Very Heavy pull, and each of 25 Ss to squeeze the dynamometer at a pressure equivalent to his Maximum pull. Five Ss did not have an exertion condition at all. Besides squeezing the dynamometer, in the Exertion condition each S was required to push the foot pedal down and keep it there for the length of the trial. He also heard a tone in both ears equal in intensity to what he would have heard had he been off target two degrees. Physical exertion was thus at a high level. The primary difference between this and other trials was that there was no actual tracking performance.

To increase the rate of learning during the early part of the experiment, Ss were told their score in terms of time off target at the end of each trial. This was not continued during the tension trials, however, in order to prevent associations of a particular score with a given level of tension

and consequent excitement or activation if the score were relatively poor.

The mean tolerance allowed for the maintenance of tension was about 1.5 kilograms. In the pilot experiments and the main study, it was found that after a little practice this tolerance was more than sufficient to enable Ss to maintain tension at quite steady levels over an entire trial.

Treatment of Data

EEG. The raw record of the electroencephalogram was recorded on the Grass chart drive. This record was used primarily to monitor artifacts that might appear in the EEG from movement, muscle, or bad electrodes. Signals from the channel on the Grass recording the occipital EEG were led to an 8-12 cycles-per-second band-pass filter; signals from the channel recording the frontal EEG were led to the 18-27 cps band-pass filter. Outputs from these two instruments were integrated with four-second discharge rates and recorded as deflections on chart drive two. During the 96-second trial, 24 of these deflections were recorded, the height of each one representing the summated potentials of the EEG over that four seconds for the frequencies studied. When artifact was present in a particular portion of the raw record, all of the four-second deflections that included these artifacts were eliminated from analysis of the integrated record. Each of the integrated EEG deflections was measured with special scales calibrated in microvolts. The level of EEG activity for a given trial was then defined as the mean voltage of the 24 integrated deflections.

EKG. EKG was obtained by counting the beats in each successive 12-second period and multiplying by five to get the mean number of beats per minute for that 12-second period. The eight values thus calculated were

averaged for the mean number of heart beats per minute for the trial.

EMG. The muscle potentials of the two active and two passive limbs were recorded on the Grass for monitoring purposes, and the four-second integrated deflection recorded on chart drive two for measurement. EMG was not as variable within a trial as EEG so that it was satisfactory to sample only eight peaks during a trial. The measured peaks for the right leg coincided with the deflection occurring approximately midway up and down the rises and falls in muscle tension due to tracking (the 3rd, 4th, 9th, 10th, 15th, 16th, 21st and 22nd deflections were measured). Like the EEG, these deflections were measured with special scales calibrated in microvolts. The level of muscle activity for a trial was then defined as the mean voltage of the eight measured deflections.

Respiration and Palmar Conductance. These measures were recorded on chart drive three and were obtained by taking a reading every 12 seconds. Respiration rate was found by counting the number of complete inspiration-expiration cycles from one 12-second line to the next, correct to the nearest 1/4 cycle. The mean rate for the trial was obtained by multiplying this value by five to get the number of cycles per minute for that period, and then obtaining the mean of the eight values. Palmar resistance was measured at the 12-second line with a special scale calibrated in ohms. This resistance value was converted to its reciprocal, conductance. The average conductance value in micromhos for the trial was obtained by calculating the mean of the eight conductance measures.

DOT and Strain Gauge. The integrated deflections of Distance

Off Target and the pull on the Strain Gauge Dynamometer were measured in the same way as the EEG. That is, each of the 24 deflections was measured using a special plastic scale, calibrated in degrees for DOT and kilograms for SG. The average of the 24 deflections was taken as the mean DOT or grip pressure for the trial.

Errors. The number of "up" and "down" errors per trial was obtained directly by means of two electromechanical counters.

Results

Figure 1A presents the graph for continuous rise in force as measured by the strain-gauge dynamometer through the graded series of induced tensions. It will be noted that the rise is approximately linear. Parts B and C of Figure 1 show that the effect of induced tension in this experiment was to impair tracking performance, with the higher tensions having a more detrimental effect than the lower ones. A two-way analysis of variance of these data shows that the between-trials variance of errors as measured by Distance Off Target was significant ($p = .05$) for the induced tension conditions.

Figure 2 presents the EMG data. As in the case of the strain-gauge, the curve for muscle tension in the right arm as a function of the amount of tension induced is approximately linear. The other three curves, for muscles not directly involved in pulling on the dynamometer, likewise show incremental changes corresponding to increasing values of induced tension. In Figure 2B the right leg is designated as active because it was used in tracking, but the load on the leg muscle was constant, of course, from one induced tension condition to the next. The two left limbs (Figure 2C and 2D) were not engaged

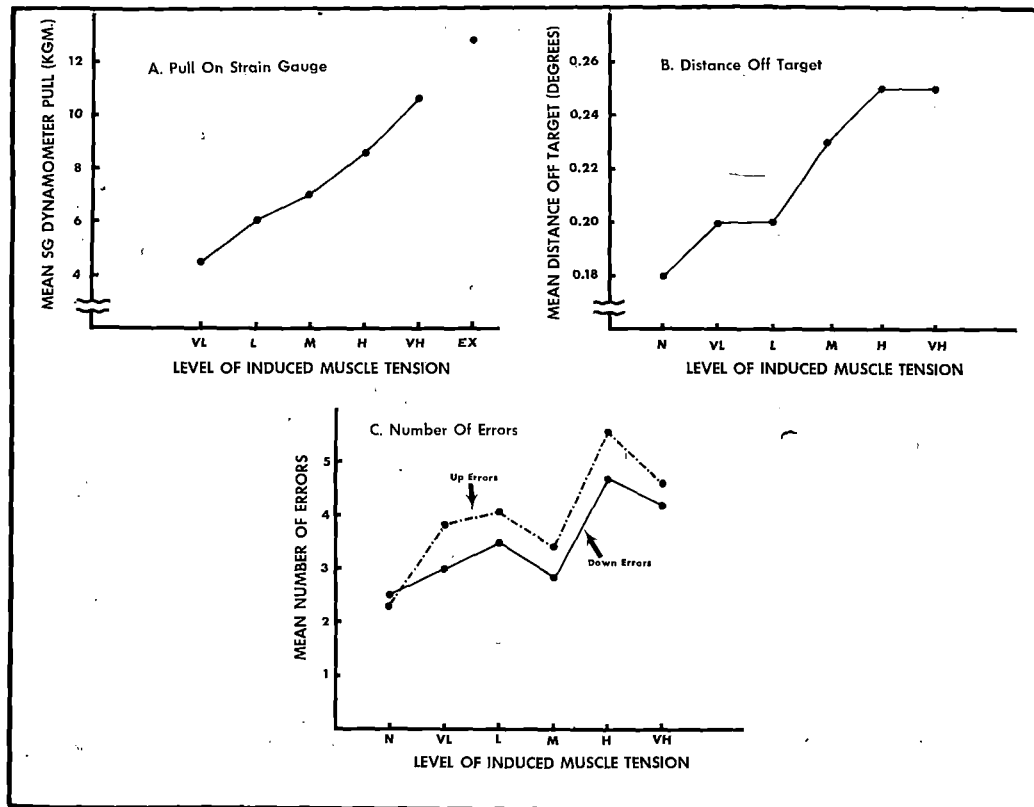


Figure 1. Performance measures as a function of induced muscle tension.

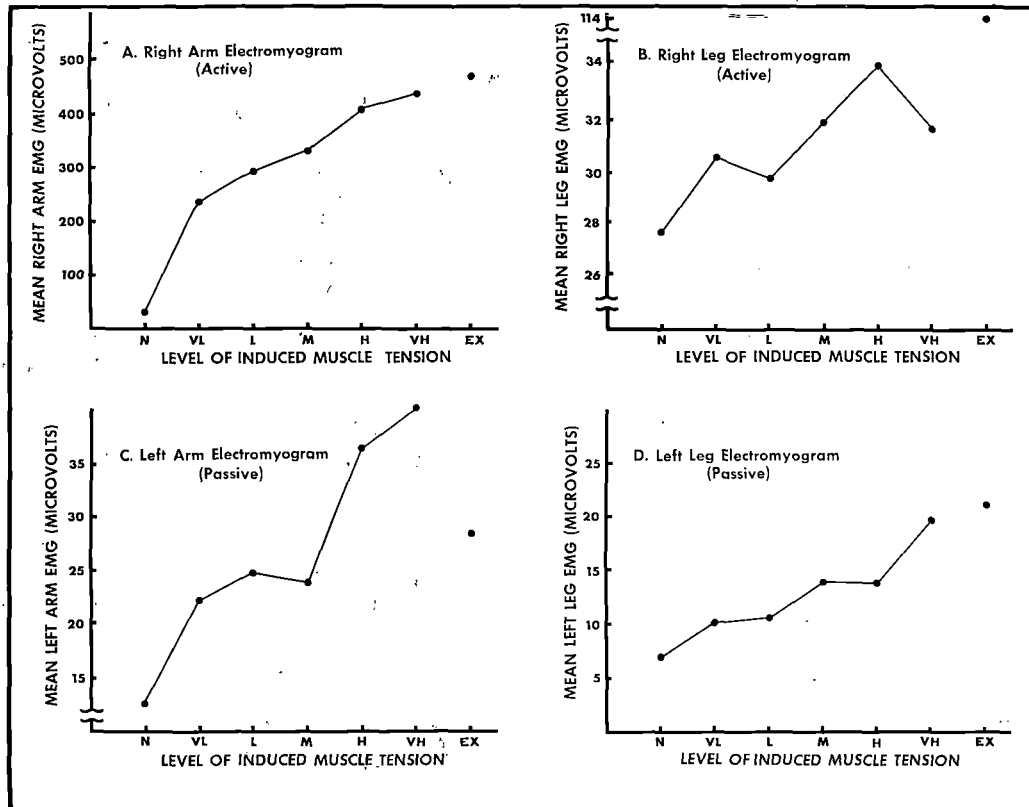


Figure 2. Muscle tension of active and passive limbs as a function of induced muscle tension.

in any activity. Despite this passivity, however, their curves show steady rise through the ascending series of induced tension values up to VH (i. e., through the series of tensions that were induced during tracking). Analysis of variance for all four muscles also showed the between-trials variance to be significant ($p < .01$) over the range of tension conditions with tracking.

The drop in the left arm in tension from the VH condition during tracking to the EX condition (no tracking) should be especially noted in Figure 2C. This is shown as a point on the far right. The drop was significant ($p < .01$). It will be recalled that 33 of the 38 Ss were included in the EX condition. Twenty-five of these Ss pulled on the dynamometer, their maximum amount during this trial, while eight Ss gripped with the same pressure as they used on the Very Heavy condition. With this amount of exertion, the right leg EMG (Figure 2B) showed a significant rise from VH to EX (the right leg held the tracking pedal all the way down during this condition, exerting the maximum leg tension for a trial). Yet, the right arm EMG (Figure 2A) which was squeezing the dynamometer, shows only a slight and very nonsignificant rise. In fact, 48 per cent of the 33 Ss actually showed a fall in the right arm EMG from the VH tracking condition to the EX non-tracking condition. Seventy-five per cent of the 33 Ss showed a fall in the left arm EMG from VH to EX. The slight rise shown in Figure 2D is not significant, of course. A summary of the percentage of Ss whose measures of activation fell for the EX condition as compared with the VH tracking condition is shown in Table 1.

Similar curves for palmar conductance, heart rate, and respiration are presented in Figure 3. It is noteworthy that there are no reversals in the curves which all show a regular progressive rise through the increasing series

Table 1

Per Cent of Subjects with Lower Values for Physiological Recordings Under
the Exertion Condition than Under the Very Heavy
Tracking Condition

S's Pull on EX	N	Right Arm	Right Leg	Left Arm	Left Leg	Palmar Cond.	Heart Rate	Resp.	EEG 8-12	EEG 18-27
Max.	25	48.00	32.00	72.00	40.00	16.00	0.00	56.00	52.00	60.00
VH*	8	50.00	12.50	87.50	62.50	62.50	62.50	75.00	62.50	12.50
Comb.	33	48.48	27.27	75.75	45.45	27.27	15.15	60.60	54.54	48.48

*This VH condition is not to be confused with the VH condition during tracking. These 8 Ss took their Exertion (i.e., no tracking) condition by pulling the same kgm. values on the dynamometer as they had pulled in the VH tracking condition.

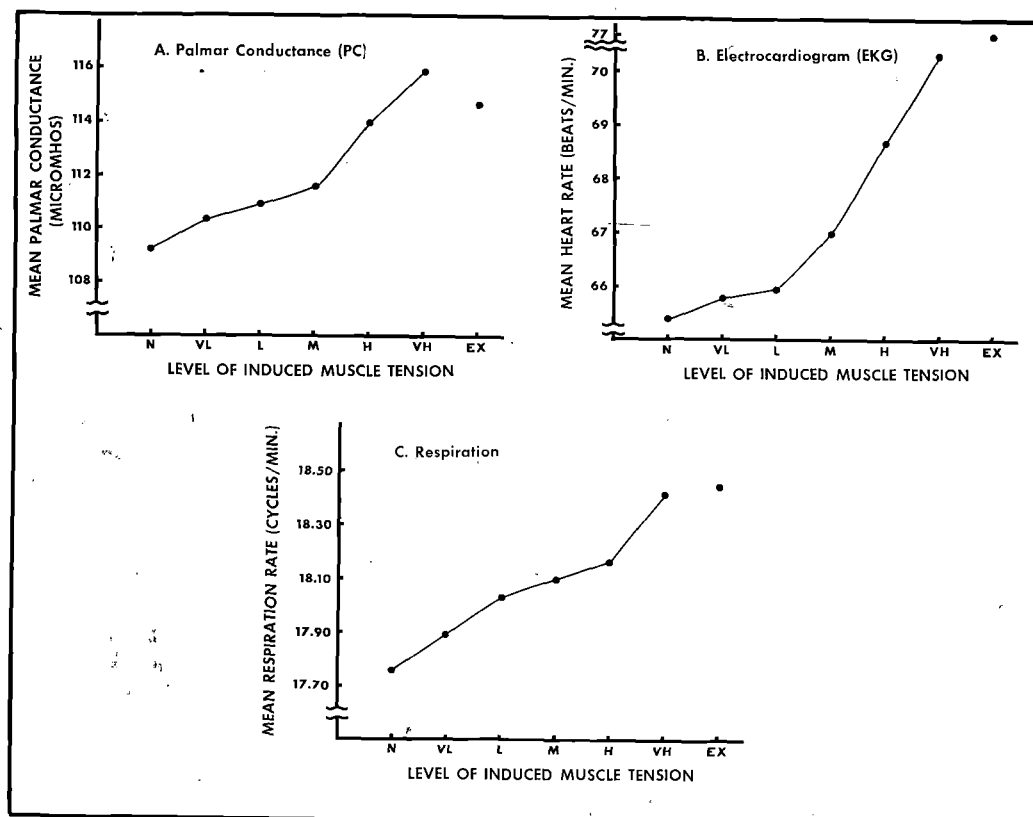


Figure 3. Peripheral measures of autonomic activity as a function of induced muscle tension.

of induced tensions. Again, a two-way analysis of variance revealed the between-trials variance of all three measures to be significant ($p < .01$) over the range of induced tension. The fall in palmar conductance from VH to EX in Figure 3A was significant ($p < .05$) as was the rise in heart rate ($p < .01$). The slight change in respiration was not significant.

Finally, Figure 4 presents the EEG data which again show remarkable correspondence with the increasing levels of induced tension. Again, both measures showed significant between-trials variance over the range of tension conditions by a two-way analysis of variance (8-12 cps, $p = .05$; 18-27 cps, $p = .01$). The large reversal between VH and EX is especially to be noted (i.e., falling 8-12 cps amplitude, significant with $p < .01$, compared with rising 18-27 cps amplitude almost significant at the .05 level).

Discussion

The physiological data were highly consistent in showing regular and continuous rise in level as a function of increments in tension induced in the right arm during tracking. Freeman's (1938) and Freeman and Simpson's (1938) results with palmar conductance were thus confirmed, but the additional data from muscle potentials, respiration, heart rate, and especially EEG considerably extend the possibilities of interpretation. The demonstration of such widespread changes, however, would appear to cast strong doubt on any rationale limited to the skeletal-motor (or somatic) system. It appears that Meyer's (1953) interpretation suffers from this limitation. The results of this experiment definitely encourage the idea that the proprioceptive return from induced muscular tension produces widely generalized physiological effects. It therefore appears that Malmo (1959) was correct when he suggested that

induced tension is one of the ways in which level of activation can be varied.

The EEG data require some special explanation. Generally, the terms "arousal" and "activation" have been used to refer to desynchronization in the EEG tracing in association with stimulation. This is what Lindsley refers to as "activation pattern" (1951, p. 505). It is well known, however, that this reaction does not inevitably follow an alerting stimulus. The 8-12 cps component of the human EEG may, in fact, be augmented by an alerting stimulus if the S is sufficiently drowsy. Stennett (1957) has systematically investigated the direction of the 8-12 cps change as a function of the S's prior level of activation showing it to be an inverted-U function.

In the present experiment, Stennett's (1957) practice of obtaining average voltage by means of band-pass filters was employed. This method permitted independent measurements of 8-12 cps EEG amplitude and 18-27 cps EEG amplitude. While the overall change in 8-12 cps EEG might have been expected to occur in the downward direction because of the generally activating effects of induced tension, finding a change in the upward direction was by no means unprecedented. In addition to Stennett's results there are recent (unpublished) findings of Malmo showing a significant overall rise in 8-12 cps amplitude in going from a condition of lower, to one of higher, activation level.⁴ Malmo and Surwillo (in press) have also shown that under appropriate conditions an overall fall of 8-12 cps EEG can occur with a rise in activation level.

The evidence from the EX condition (Table 1) quite clearly suggests an interaction between tension induction and tracking. That is, if induced tension were the sole factor, the points on the curves for the EX condition

Pinneo

-20-

should be more continuous with the points for the VH condition than they in fact are. These results support the previous contention of Malmo (1959, p. 373) that activation level is invariably determined by an interaction between internal factors (mainly those affecting the "tonic baseline" activity of the ARAS) and external (cue) factors.

Performance per se in this experiment was also affected by induced muscle tension but not in the expected direction. If it is assumed that Ss were relatively relaxed at the lower levels of muscle tension, then one would expect performance to be enhanced with induced muscle tension. On the contrary, however, there was a decrement in performance, and scores rose to pre-learning levels. One possible explanation for this is that Ss were overactivated at even the lowest tension level. However, the marked increase in physiological measures of activation with increased muscle tension above the Very Light condition would suggest that Ss were quite relaxed at the lower levels. Another possible explanation is that the necessity of squeezing the dynamometer while tracking represented a sufficiently difficult task to require divided attention. For this explanation to be adequate, it must be supposed that the difficulty associated with maintaining tension increased with tension level. However, the strain gauge integrated output actually showed amount of tension to be constant over the length of a given trial, regardless of the amount of tension that was induced. The most likely state of affairs is that the increase in errors with induced muscle tension involved a complex interaction of set, divided attention, activation, and possibly other factors.

Summary

Induced muscle tension has been demonstrated to have significant effects in relation to a wide range of behavioral phenomena. From the behavioral evidence, it appeared reasonable to consider that induced tension was only one of the many ways in which activation level can be varied. It followed that if induced tension were a reliable means of varying activation level, then in addition to the behavioral effects which have already been demonstrated, induced tension should also produce regular and consistent changes in the various physiological indicants of activation. The purpose of this experiment was to investigate such physiological changes.

Thirty-eight male college students were trained in an auditory tracking task. Physiological indicants of activation included heart rate, respiration rate, palmar conductance, frontal and occipital EEG, and EMGs from active and passive limbs. Following learning trials, Ss were required to track while squeezing a hand dynamometer in order to maintain one of five predetermined levels of muscle tension.

Results clearly showed close agreement between amount of tension induced and the level of activity in all physiological measures. These results were considered in support of a theory that the proprioceptive return from the induced muscle tension produces generalized behavioral and physiological effects indirectly by increasing activity in the reticular activating system.

References

- Courts, F.A. Relations between muscular tension and performance.
Psychol. Bull., 1942, 39, 347-367.
- Davis, J.F. Manual of surface electromyography. WADC Technical
Report 59-184, Wright Air Development Center, Ohio,
December, 1959.
- Davis, J.F., Stennett, R.G. & Quilter, R.E. An auditory tracking device
designed for use in conjunction with continuous EEG recording.
Percept. mot. Skills, 1957, 7, 239-244.
- Delafresnaye, J.F., Adrian, E.D., Bremer, F., & Jasper, H.H. (Eds.)
Brain mechanisms and consciousness. Springfield, Ill.:
Charles C Thomas, 1954.
- Freeman, G.L. The optimal muscular tensions for various performances.
Amer. J. Psychol., 1938, 51, 146-150.
- Freeman, G.L. & Simpson, R.M. The effect of experimentally induced tension
upon palmar skin resistance. J. gen. Psychol., 1938, 18, 319-326.
- French, J.D. Corticifugal connections with the reticular formation. In
H.H. Jasper, L.D. Proctor, R.S. Knighton, W.C. Noshay &
R.T. Costello (Eds.), Reticular formation of the brain.
Boston: Little, Brown, 1958. Pp. 491-505.
- Jasper, H.H. Electroencephalography. In W. Penfield & T.C. Erickson,
Epilepsy and cerebral localization. Springfield, Ill.:
Charles C Thomas, 1941. Pp. 380-454.
- Lindsley, D.B. Emotion. In S.S. Stevens (Ed.), Handbook of experimental
psychology. New York: Wiley, 1951, Pp. 473-516.

- Malmo, R.B. Activation: A neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
- Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Amer. J. Psychol. (in press).
- Malmo, R.B. & Surwillo, W.W. Sleep deprivation: Changes in performance and physiological indicants of activation. Psychol. Monogr., (in press).
- Meyer, D.R. On the interaction of simultaneous responses. Psychol. Bull., 1953, 50, 204-220.
- Ross, W.R. & Davis, J.F. Stable band-pass filters for electroencephalography. IRE Canadian Convention Record, 1958, paper No. 860, 202-206.
- Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. J. exp. Psychol., 1959, 58, 117-128.
- Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.

Footnotes

1. This paper is part of a thesis presented to the Faculty of Graduate Studies of McGill University for a Ph.D. degree. It was supported in part by the Medical Research and Development Division, Office of the Surgeon General, Department of the United States Army: Contract Number DA-49-007-MD-626; Defence Research Board, Department of National Defence, Canada: Grant Number 9425-04; National Institute of Mental Health, National Institutes of Health, U.S. Public Health Service: Grant Number M-1475; National Research Council of Canada: Grant Number A.P. 29; and Department of National Health & Welfare (Canada): Grant Number 604-5-69.

The author expresses his thanks to Dr. Robert B. Malmo for his continuing interest, guidance, and support, Messrs. D.J. Ehrlich, P.F. MacNeilage, and L.D. Rust for their discussion and criticism of the experiment, Mr. W. Mundl and Dr. J.F. Davis for assistance with the apparatus, and Jeanne M. Pinneo for her assistance in carrying out the experiment.

2. Now at the Institute of Physiology, University of Pisa, Italy.

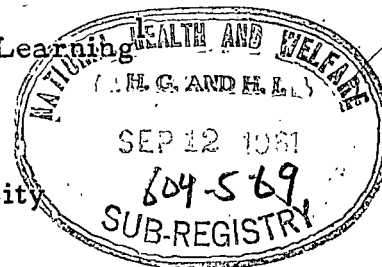
3. Much of the apparatus used in this experiment was designed and built in this laboratory. Detailed specifications and circuit diagrams of the tracking apparatus, the strain gauge tension induction system, the EEG band-pass filters, and the electronic integrators are on file at the Laboratory for Psychological Studies, Allan Memorial Institute, McGill University, Montreal, Canada.

4. Personal communication.

Differential Effects of Shock in Human Maze Learning

Samuel M. Feldman²

Allan Memorial Institute, McGill University



The early observation of Tolman, Hall, and Brethall (1932) that electric shock facilitates learning, even when the shocks follow correct responses, has been confirmed by an impressive number of subsequent investigations (e. g., Feldman, 1954; Freeburne & Schneider, 1955; Freeburne & Taylor, 1952; Muenzinger, 1934; Muenzinger, Bernstone, & Richards, 1938; Muenzinger, Brown, Crow, & Powloski, 1952; Muenzinger & Newcomb, 1935; Muenzinger & Powloski, 1951; Prince, 1956). Muenzinger & Baxter (1957), on the basis of an investigation of the perceptual properties of shocks following correct (shock-right) and incorrect (shock-wrong) responses, have suggested that two factors operate when Ss are shocked. First, shock operates as a general facilitating mechanism, regardless of whether administered for correct or for incorrect responses. This is opposed to the common assumption that the only effect of shock is to produce avoidance. In addition to this, according to Muenzinger & Baxter, the shock takes on cue properties, leading to approach behavior in shock-right groups and to aversive behavior in shock-wrong groups. The shock-right groups, however, must overcome an initial aversion to shock. It is this initial aversion that accounts for the ultimate superiority of shock-wrong over shock-right in Muenzinger's data.

In this explanation, Muenzinger & Baxter make two assumptions. The first is that shock-wrong groups always learn faster than do shock-right groups, an assumption which is not borne out by the available evidence. While

this is usually the case, several experiments have resulted in shock-right being the superior condition for learning (Feldman, 1954; Tolman, Hall & Bretnall, 1932), and in still others, no difference has been observed between shock-right and shock-wrong groups (e.g., Freeburne & Schneider, 1955). A review of earlier studies suggests that intensity of the noxious stimulus employed may be the basis of these conflicting findings (see e.g. Muenzinger & Newcomb, 1935). It seems that when shock has been held to a relatively mild intensity (e.g., by allowing human Ss to adjust the intensity until they can "take no more"), shock-right has been the superior condition; but when the intensity of the shock has been relatively great (as in animal studies in which, e.g., E adjusts the current until S "jumps"), shock-wrong groups tended to perform better.

The second assumption of Muenzinger & Baxter is that cue properties alone differentiate the two shock conditions (i.e., shock-right vs. shock-wrong). If intensity is a critical factor in determining the relative effects of the two shock conditions, then the arousing properties of the shock stimulus bear investigation as well as the cue properties.

The present study was designed to obtain data bearing on questions concerning the interaction between shock intensity and shock condition in learning. To this end, an objective criterion of the arousing properties of the shock stimulus was required. Several authors (e.g., Duffy, 1957; Malmö, 1958; Schlosberg, 1954) have recently suggested that the measurement of the level of activity in certain physiological systems might provide meaningful indicants of arousal. Experiments carried out on human and animal Ss at

McGill University and at the University of Montreal have lent concrete support to this suggestion (see Malmö, 1959). The measurement of respiration rate, palmar conductance, and heart rate was therefore undertaken in the present investigation in order to study the arousing properties of shock intensity and shock condition in relation to the cue properties of these variables in a learning situation.

Method

Design

The Ss were randomly assigned to one of four groups, 15 to a group, in a 2 x 2 factorial design. All Ss received 15 trials on a 25-choice point "nailhead" finger maze. Half of the Ss received shocks for incorrect responses while the other half were shocked for correct responses. Each of these two shock conditions were divided into a "high" intensity and a "low" intensity group. The four groups were thus high shock-right (HSR), low shock-right (LSR), high shock-wrong (HSW) and low shock-wrong (LSW).

Subjects

Sixty male undergraduates at McGill University served as Ss. They were paid for the services at the rate of \$1.00 per hour.

Apparatus

"Nailhead" maze. A 25-choice point nailhead maze was constructed by imbedding fifty copper nails in a plywood board. They formed two adjacent columns of 25 nails each, which appeared to S as a mounted board containing a single vertical column of 25 horizontal pairs of nailheads. The correct and incorrect sequences of nailheads were wired in parallel, independently of

Feldman

-4-

each other. This made possible shock administration and the recording of all responses. Each adjacent pair of nailheads thus constituted a single choice point. The correct sequence, for all groups, was

L L R L R L L R R R L R R L L L R R L R R L L R L.

Shocking apparatus. When S touched a nailhead which was in the shocking circuit a condensor was discharged to his body through a large series resistance, which served to minimize individual differences in resistance. A shielded cable extended the shocking circuit from a control chamber to S's room, where one side was connected to the maze (either to the correct or to the incorrect sequence) and the other side was connected to a strip of brass, which was wrapped around the proximal phalanx of S's right index finger. A metal thimble, filled with electrode jelly, was placed on this finger. When the thimble was brought into contact with the shock-wired nailheads, the circuit was completed through S's finger. Current flow, at the moment of discharge of the condenser was 3 ma. for the low shock groups and 9 ma. for the high shock groups.

Testing procedure

After a brief interview in E's office, S was taken into the testing chamber and seated in a chair with adjustable armrests. All electrodes and attachments were applied, except for the shocking electrodes. The testing room was a shielded chamber which contained a table, adjustable chair, and electrodes, lead wires, and chemicals necessary for electrode attachment. An adjacent room, with a window looking into the testing chamber, contained all of the recording equipment. During the experiment, S was observed by E through the window, and S could be heard through an intercommunication

Feldman

-5-

system.

In order to get an estimate of S's level of physiological activity in a relatively relaxed condition, S was initially told that the study was a comparison of physiological activity during reading and during relaxation. After a 25-min. period of magazine reading, with recording apparatus attached, and equipment turned on, S was asked to relax. A 5-min. record of palmar resistance, respiration rate, and heart rate recorded during this session was used as a personal baseline for S. It was after this record had been taken that the learning task was introduced. Instructions for learning were given, the maze was placed on the table before S, and shocking leads were applied. Each S had 15 trials, with a 1-min. interval between trials. A trial consisted of a single traversal of the complete maze, choosing one nailhead in each of the 25 pairs. No practice trials were given.

Recording technique³

Heart rate. Heart rate was recorded by means of a continuous electrocardiographic tracing taken on an Offner Type D electroencephalograph. A chest placement was used for the electrodes, which were made of cellulose sponge, saturated with normal saline and electrode jelly. This Offner output signal was recorded on a chart drive which contained tracings of this and other systems which follow, as well as the responses made in learning.

The period selected for sampling physiological activity was the pre-trial period. The experiment required an estimate of S's level of arousal produced by the total situation. Since a specific reaction-to-shock measure was not desired, the post-trial period was not selected for study. The pre-trial measurements were samples of activity occurring during the period

Feldman

-6-

roughly 10-sec. prior to each trial. Since the four groups were independent, absolute level of activity could not be used. An estimate of S's resting level of activity had to be taken in order that S's pre-trial activity could be expressed in terms of a personal baseline. The resting level was estimated on the basis of pre-experimental samples, taken during the 5-min. resting period prior to the introduction of the learning task.

In the case of heart rate, the pre-experimental measure was an average of five 10-cycle (i. e. 11 beats) samples taken at the end of each of the five minutes during the pre-experimental "rest" period. The pre-trial measure was an average of the 10 cycles preceding the beginning of each trial. These averages were based on conversions to beats per minute (b/m).

Palmar conductance. Palmar conductance was calculated from a continuous recording of palmar resistance. The circuit used was a standard comparative bridge, with a current varying no more than 2% when S's resistance was in the range of 0 to 80,000 ohms. The monopolar technique employed for recording resistance has been reported in detail by Malmö & Davis (1960).

Palmar resistance was measured in ohms and then converted to conductance in micromhos. The measurements were made at the beginning and end of each of the 10-cycle/^{heart}rate samples. There were thus 10 measurements made during the pre-experimental period and two prior to each trial.

Respiration rate. Respiration rate was determined from a continuous recording taken with a tambour and pneumograph. An abdominal placement was used for the accordion pneumograph. The method used for sampling respiration was the same as that used for sampling heart rate,

except that three complete inspiration-expiration samples were measured rather than the 10-cycle sample used in the case of heart rate. These measurements were converted to respirations per minute (r/m).

Reliability of measurements. In order to check on the reliability of these measurements, odd-even rank order correlations were calculated for each physiological measure, in each experimental condition. These were based on an average of each S's activity on odd trials with activity on even trials. In all cases except one, the resultant Rho was greater than +.95. The one exception was respiration rate in the HSR group, in which case Rho was +.88.

Treatment of data

Learning. Since Ss received no practice trials, the number of errors made on trial 1 was determined by chance alone. The error score used, therefore, was total errors/errors on trial 1. In this way, each S's score was a function of an individual baseline. The same technique was employed in calculating time scores, i. e. , total time/time on trial 1.

Physiological Activity. Each measure for every S was expressed as mean pre-trial activity divided by mean pre-experimental activity. Absolute level scores were not appropriate since the groups were independent. The mean pre-experimental scores were based on the mean of 5 samples taken during the "resting" period.

It was felt that a measure of arousal should be able to differentiate between the high shock Ss and low shock Ss. Since this has not been demonstrated for the measures employed in the present study, the criterion for the

adoption of measures of arousal was their ability to provide this differentiation.

Results

Learning

As can be seen in Fig. 1, the HSW group made the least errors, the LSW group the most, while the two shock-right groups fell in between. This was the case for the time data as well, as is illustrated in Fig. 2. Table 1 presents a 2 x 2 Chi-square test of analysis of variance hypotheses (Wilson, 1956) for these data. In the case of both time and errors, neither shock intensity nor shock condition was seen to have a statistically significant effect; only the interaction term was significant. Mann-Whitney U tests (Table 2) showed that the difference between the LSR and LSW groups was significant for time and errors, beyond the .05 level of confidence, LSR being the condition associated with superior performance. Differences between the HSR and HSW groups were not statistically significant. When the mean time and error scores on trials 11 to 15 were analyzed (i. e., trials 11-15/trial 1), the time data showed a clear reversal as a function of shock intensity, LSR and HSW being significantly faster than LSW and HSR respectively. Differences in this analysis for errors were in the same direction, but failed to meet statistical significance.

In looking at the effect of increasing shock intensity, no significant differences were observed for time or for errors between the two shock-right groups. The LSW group, however, was consistently a poorer condition for learning than the HSW; all differences, for time and for errors, were statistically significant,

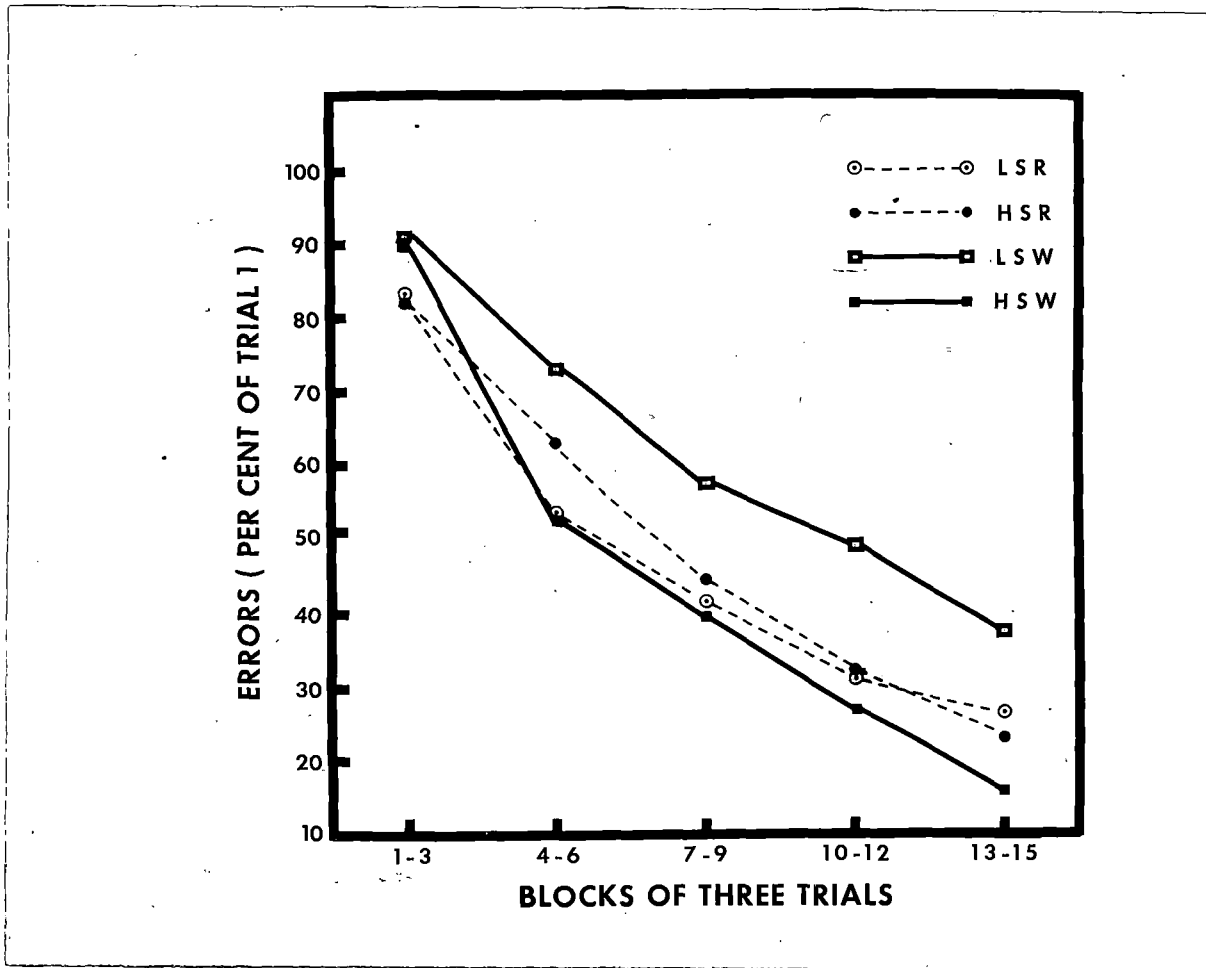


Figure 1. Errors (per cent of trial 1) as a function of practice.

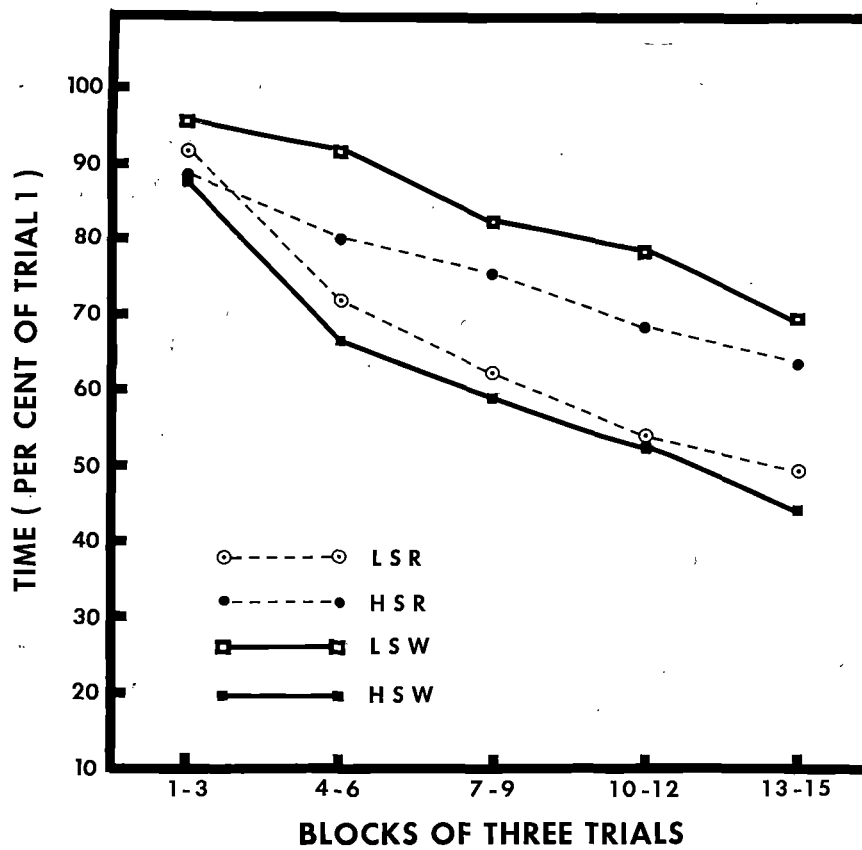


Figure 2. Time per trial as a function of practice.

Feldman

-11-

Table 1

2 x 2 Chi-Square Analysis of the Effects of Shock Intensity

and Shock Condition on Learning (Trials 1-15/Trial 1)

(N = 15 Ss per group)

Source of Chi-square	Errors		Time	
	X ²	p	X ²	p
Total	11.7	<.001	8.8	<.01
Shock Condition	2.4	n. s.	1.1	n. s.
Shock Intensity	0.0	n. s.	1.1	n. s.
Interaction	9.3	<.01	6.7	<.01

Table 2

U's Based on Differences between Groups in Learning

(N = 15 Ss per group)

Groups Compared	Time		Errors	
	U	p	U	p
LSR - LSW (trials 1-15)	64.0	<.05	63.5	<.05
HSR - HS ^W (trials 1-15)	70.0	n. s.	89.0	n. s.
LSR - LSW (trials 11-15)	46.0	<.01	74.0	n. s.
HSR - HSW (trials 11-15)	62.0	<.05	74.0	n. s.

Table 3

Mean Changes in Level of Physiological Activity

([Mean pre-trial/mean pre-experimental] x 100)

(N = 15 Ss per group)

Experimental Group	Respiration	Palmar	Heart
	Rate	Conductance	Rate
LSR	103	105	102
LSW	113	103	104
HSR	113	118	105
HSW	117	130	101

Physiological activity

Table 3 contains the mean changes in physiological activity resulting from the experimental conditions. Each entry represents a mean for 15 Ss of mean pre-trial/pre-experimental samples. It was pointed out earlier that any measure of physiological activity which is to distinguish between levels of arousal was required to differentiate the high shock Ss from the low shock Ss. As can be seen in Table 4, two of the three measures employed met this criterion; in the case of heart rate, there was no difference between the high shock and low shock groups.⁴ In the case of respiration rate the difference between the high shock and low shock groups was significant beyond the .01 level of confidence, high shock being associated with faster respiration than low shock. It can be seen in Table 4 that the interaction term for respiration rate was also statistically significant; this is due to the fact that the low-high difference was much greater in the case of shock-right than in the case of shock-wrong (Table 3).

Palmar conductance also met the above cited criterion. High shock was associated with greater conductance than was low shock. Table 4 shows this difference to be significant beyond the .05 level. Only heart rate failed to meet this criterion, and was therefore rejected as a measure of level of arousal in studying the shock conditions.

Wilson's 2 x 2 Chi-square test of analysis of variance hypotheses showed that the differences between shock conditions failed to meet statistical significance. The direction of the differences showed shock-wrong to be associated with higher levels of palmar conductance and respiration rate than shock-right. But these differences were not significant.

Table 4

2 x 2 Chi-Square Analysis of Effects of Shock Intensity and Shock

Condition on Changes in Level of Physiological Activity

(N = 15 Ss per group)

Source of Chi-Square	Respiration		Palmar		Heart	
	Rate		Conductance		Rate	
	X ²	p	X ²	p	X ²	p
Total	12.00	<.001	5.60	<.05	1.33	n. s.
Shock Condition	1.07	n. s.	1.07	n. s.	0.27	n. s.
Shock Intensity	6.67	<.01	4.27	<.05	0.00	n. s.
Interaction	4.26	<.05	0.99	n. s.	1.06	n. s.

Discussion

The many experiments in which humans and rats have been shocked for correct responses in learning have demonstrated that shock can act as a general facilitating mechanism. This is consistent with recent statements on the role of arousal in behavior (see Schlosberg, 1954; Hebb, 1955; Malmö, 1959). Muenzinger & Baxter (1957), in their investigation of the perceptual properties of shock, argued that shock-right learning could not be accounted for simply in terms of anxiety reduction (Mowrer, 1950; Prince, 1956). The present experiment represents an investigation of the arousing properties of the shock stimulus, in relation to the work of Muenzinger and his colleagues.

In an early paper, Muenzinger, Bernstone, & Richards (1938) spoke of the operation of both general and specific mechanisms in the use of shock. They assumed that more than one mechanism was operating, and that the specific mechanism (which Muenzinger & Baxter later concluded was primarily perceptual) did not contribute to the general one in an additive fashion.

The data of the present experiment are consistent with this view, in that shock intensity was seen to be accompanied by differences in level of physiological activity, while differences in shock condition were not.⁵

In their report, however, Muenzinger & Baxter assumed that shock-wrong was consistently a better condition for learning than was shock-right. The data of this experiment showed, on the contrary, that shock-right was the superior condition for learning when the shock intensity was relatively mild. When shock intensity was relatively high, this was not the case; some measures of learning showed no significant difference, and others showed

shock-wrong to be the superior condition. In all cases, the direction of difference favored shock-right at low intensity and shock-wrong at high intensity.

Muenzinger & Baxter further assumed that once the initial aversion to shock was overcome by shock-right Ss, they would then regard the shock, and all cues associated with it, as cues for approach behavior. In addition to this, the authors felt that this would serve to equalize the two conditions, and no differences would then be observed between shock-right and shock-wrong groups. The data of the present experiment suggest that once the initial aversion to shock is overcome, shock-right, having the advantage of the shock and cues associated with it as signals for approach behavior, becomes the better condition for learning. This happened early in the case of the LSR group, but late in the case of the HSR group, resulting in the interaction between shock condition and shock intensity in learning. (Actually this failure to overcome the initial aversion to shock was quite varied in the HSR group, as was evidenced by the fact that the variance in this group, both for time and for errors, was significantly greater than for any other group).

The data suggest, in summary, that the increased facilitating effect of increased shock, evident in the shock-wrong groups, was not evident in the shock-right groups due to the fact that the initial aversion to shock was overcome sooner in the LSR group than in the HSR group.

The present study thus supports the suggestions of Muenzinger & Baxter on the dual function of the shock stimulus. The interaction in the learning data, however, leads to a further condition; namely, that shock-

right is the superior condition for learning after the initial avoidance tendencies are overcome, and it is perceived as a signal for approach behavior. This occurs sooner in the case of the low shock Ss than in the case of the high shock Ss.

Summary

The main purpose of the present study was to investigate the role of shock in learning with reference to the problem of shock-right vs. shock-wrong. Four independent groups of 15 Ss each learned a "nailhead" finger maze. Shock intensity ("low" and "high") and shock condition ("right" and "wrong") were varied in a 2 x 2 factorial design. The results were consistent with previous findings in showing that shock-right was the superior condition when the intensity was low, but that shock-wrong tended to be the superior condition when the intensity of the shock was relatively high.

Continuous recordings of respiration rate and palmar conductance served to gauge the level of arousal produced by the experimental conditions. There was a significant difference in level of physiological activity accompanying the different intensities of shock in the present study, high shock being associated with higher levels of activity than low shock. No significant differences were found between physiological activity associated with shock-right (shock-intensity constant) and that associated with shock-wrong. It appears, therefore, that the differential effects of shock on these two conditions cannot be accounted for in terms of an inherent difference in arousal level between the two conditions. Rather, it is suggested that the differential effects

Feldman

-19-

be interpreted in terms of Muenzinger's and Baxter's suggestion that shock-right and shock-wrong are accompanied by differences in the perceptual role of shock.

References

- Duffy, E. The psychological significance of the concept of "arousal" or "activation." Psychol. Rev., 1957, 64, 265-275.
- Feldman, S.M. Escape from punishment and motivation in maze learning. Unpublished honor's thesis, Univer. of Penna., 1954.
- Freeburne, C.M. & Schneider, M. Shock for right and wrong responses during learning and extinction in human subjects. J. exp. Psychol., 1955, 49, 181-186.
- Freeburne, C.M. & Taylor, J.E. Discrimination learning with shock for right and wrong responses in the same subjects. J. comp. physiol. Psychol., 1952, 45, 264-268.
- Hebb, D.O. Drives and the C. N. S. (Conceptual nervous system). Psychol. Rev., 1955, 62, 243-254.
- Malmo, R.B. Measurement of drive: an unsolved problem in psychology. In M.R. Jones (Ed.), Nebraska symposium on motivation. Lincoln: Univer. of Nebr. Press, 1958. Pp. 229-265.
- Malmo, R.B. Activation: A neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
- Malmo, R.B. & Davis, J.F. A monopolar method for recording palmar conductance. Amer. J. Psychol., 1960 (in press).
- Mowrer, O.H. Learning theory and personality dynamics. New York: Ronald, 1950.
- Muenzinger, K.F. Motivation in learning. I. Electric shock for correct responses in the visual discrimination habit. J. comp. Psychol., 1934, 17, 267-277.

Muenzinger, K.F. & Baxter, L.F. The effect of training to approach vs. training to escape from electric shock upon subsequent discrimination learning. J. comp. physiol. Psychol., 1957, 50, 252-257.

Muenzinger, K.F., Bernstein, A.H., & Richards, L. Motivation in learning. VIII. Equivalent amounts of electric shock for right and wrong responses in a visual discrimination habit. J. comp. Psychol., 1938, 26, 177-186.

Muenzinger, K.F., Brown, W.C., Crow, W.J., & Powloski, R.F. Motivation in learning. XI. An analysis of electric shock for correct responses into its avoidance and accelerating components. J. exp. Psychol., 1952, 43, 115-119.

Muenzinger, K.F. & Newcomb, H. Motivation in learning. III. A bell signal compared with electric shock for right and wrong responses in the visual discrimination habit. J. comp. Psychol., 1935, 20, 85-93.

Muenzinger, K.F., & Powloski, R.F. Motivation in learning. X. Comparison of electric shock for correct turns in a correction and non-correction situation. J. exp. Psychol., 1951, 42, 118-124.

Prince, A.I., Jr. Effect of punishment on visual discrimination learning. J. exp. Psychol., 1956, 52, 381-385.

Schlosberg, H. Three dimensions of emotion. Psychol. Rev., 1954, 61, 81-88.

Feldman

Tolman, E.C., Hall, C.S., & Bretnall, E.P. A disproof of the law of effect and a substitution of the laws of emphasis, motivation, and disruption. J. exp. Psychol., 1932, 15, 601-614.

Wilson, K.V. A distribution-free test of analysis of variance hypotheses. Psychol. Bull., 1956, 53, 96-101.

Footnotes

1. This paper is based on the experimental portion of a thesis submitted to the Faculty of Graduate Studies and Research, McGill University, in partial fulfilment of the requirements for the degree of Doctor of Philosophy. Support for the investigation has come from the following sources: Medical Research and Development Division, Office of the Surgeon General, Department of the U. S. Army; Contract Number DA-49-007-MD-626; National Institute of Mental Health, National Institutes of Health, U. S. Public Health Service: Grant Number M-1475; the Department of National Health and Welfare (Canada): Grant Number 604-5-69; Defence Research Board, Department of National Defence, Canada: Grant Number 9425-04; and the National Research Council of Canada: Grant Number A. P. 29.

The author is indebted to Dr. R. B. Malmo for his supervision throughout this investigation, and for his generous provision of laboratory facilities. The author also wishes to thank Mr. W. R. D. Røes for his technical assistance, and Dr. M. M. Schnore for his many constructive comments and criticism.

2. Present address: Department of Physiology & Biophysics, University of Washington, Seattle, Washington.

3. Physiological systems other than those reported here were measured during the course of this study. These data are not pertinent here, and will be reported in a later publication.

4. Malmo (personal communication), in a recent study of sleep deprivation, found that heart rate increased significantly over a three-day vigil in two Ss tested, but that heart rate decreased significantly in a third S. Activity

in other physiological measures suggested that all three Ss increased in overall level of arousal as a result of the vigil. Heart rate, thus, may be an indicant of arousal through an increase in some Ss, and through a decrease in others. Further study, both of Ss and experimental conditions in relation to heart rate, is needed.

5. A note of caution must be introduced with respect to the lack of significance of difference in respiration rate and palmar conductance between the shock-right and shock-wrong groups. All groups were independent, which is not an optimal condition for the study of differences in physiological activity due to experimental conditions, since the variance from S to S is considerable in these measures. The large variances in the present study could, conceivably, have led to a Type II error.

Allan Memorial Institute of Psychiatry

3. Malmo, R.B., Shagass, C., Davis, J.F., Cleghorn, R.A., and Goodman, A.J. Standardized pain stimulations as controlled stress in physiological studies of psychoneurosis. Science, 1948, 108, 509-511.
4. Malmo, R.B., and Shagass, C. Physiologic studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.
6. Malmo, R.B., Shagass, C., and Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
7. Malmo, R.B., Shagass, C., and Davis, F.H. Symptom specificity and bodily reactions during psychiatric interview. Psychosom. Med., 1950, 12, 362-376.
8. Malmo, R.B., and Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
9. Malmo, R.B., Shagass, C., and Heslam, R.M. Blood pressure responses to repeated brief stress in psychoneurosis: a study of adaptation. Canad. J. Psychol., 1951, 5, 167-179.
12. Malmo, R.B., Shagass, C., Belanger, D.J., and Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
13. Malmo, R.B., Shagass, C., and Smith, A.A. Responsiveness in chronic schizophrenia. J. Personality, 1951, 19, 359-375.
14. Malmo, R.B., Shagass, C., and Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
15. Davis, F.H. and Malmo, R.B. Electromyographic recording during interview. Amer. J. Psychiat., 1951, 107, 903-916.
16. Shagass, C. and Malmo, R.B. Psychodynamic themes and localized muscular tension during psychotherapy. Psychosom. Med., 1954, 16, 295-313.
17. Malmo, R.B., Davis, J.F., and Barza, S. Total hysterical deafness: an experimental case study. J. Personality, 1952, 21, 188-204.
18. Davis, J.F. Manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1952 (mimeo.)

19. Smith, A.A. An electromyographic study of tension in interrupted and completed tasks. J. exp. Psychol., 1953, 46, 32-36.
22. Malmo, R.B., Shagass, C., and Davis, F.H. Specificity of bodily reactions under stress. Res. Publ. Ass. nerv. ment. Dis., 1950, 29, 231-261.
26. Malmo, R.B. Higher functions of the nervous system. Ann. rev. Physiol., 1954, 16, 371-390.
27. Malmo, R.B. Eccles' neurophysiological model of the conditioned reflex. Canad. J. Psychol., 1954, 8, 125-129.
28. Malmo, R.B., Wallerstein, H., and Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. J. Personality, 1953, 22, 162-187.
29. Shagass, C. The sedation threshold. A method for estimating tension in psychiatric patients. EEG Clin. Neurophysiol., 1954, 6, 221-233.
30. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canad. J. Psychol., 1955, 9, 21-28.
31. Davis, J.F., Malmo, R.B., and Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canad. J. Psychol., 1954, 8, 177-186.
32. Malmo, R.B., and Wallerstein, H. Rigidity and reactive inhibition. J. abn. soc. Psychol., 1955, 50, 346-348.
33. Malmo, R.B., and Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Malmo, R.B., Boag, T.J., and Raginsky, B.B. Electromyographic study of hypnotic deafness. J. clin. exp. Hypnosis, 1954, 2, 305-317.
35. Smith, A.A., Malmo, R.B., and Shagass, C. An electromyographic study of listening and talking. Canadian J. Psychol., 1954, 8, 219-227.
36. Wallerstein, H. An electromyographic study of attentive listening. Canadian J. Psychol., 1954, 8, 228-238.
37. Surwillo, W.W. A device for recording variations in pressure of grip during tracking. Amer. J. Psychol., 1955, 68, 669-670.

38. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canadian J. Psychol., 1955, 9, 215-230.
39. Malmo, R.B., Kohlmeier, W., and Smith, A.A. Motor manifestation of conflict in interview. J. abn. soc. Psychol., 1956, 52, 268-271.
41. Bartoshuk, A.K. Electromyographic gradients and EEG amplitude during motivated listening. Canadian J. Psychol., 1956, 10, 156-164
42. Davis, J.F. Operator's Manual: A.M.I. Integrator system. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1956 (mimeo.)
43. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.
44. Malmo, R.B., and Davis, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.
45. Hecaen, H., Penfield, W., Bertrand, C., and Malmo, R.B. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. Arch. Neurol. Psychiat., 1956, 75, 400-434.
46. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.
47. Stennett, R.G. The relationship of performance level to level of arousal. J. exp. Psychol., 1957, 54, 54-61.
48. Malmo, R.B., Boag, T.J., and Smith, A.A. Physiological study of personal interaction. Psychosom. Med., 1957, 19, 105-119.
49. Belanger, D. "Gradients" musculaires et processus mentaux superieurs. Canad. J. Psychol., 1957, 11, 113-122.
51. Malmo, R.B. Anxiety and behavioral arousal. Psychol. Rev., 1957, 64, 276-287.
52. Davis, J.F., Stennett, R.G., & Quilter, R.E. An auditory tracking device designed for use in conjunction with continuous EEG recording. Percep. Mot. Skills, 1957, 7, 239-244.
54. Surwillo, W.W. A new method of motivating human behavior in laboratory investigations. Amer. J. Psychol., 1958, 71, 432-436.
56. Ross, W.R.D., and Davis, J.F. Stable band-pass filters for electroencephalography. IRE Canadian Convention Record, 1958, Paper No. 860 202-206.

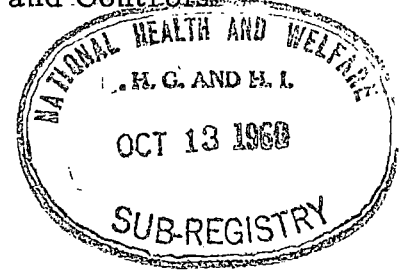
57. Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. J. exp. Psychol., 1959, 58, 117-128.
 59. Malmo, R.B. Activation: a neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
 60. Malmo, R.B., & Davis, J.F. A monopolar method of measuring palmar conductance. Amer. J. Psychol., 1961, 74, 106-113.
 61. Bartoshuk, A.K. Electromyographic reactions to strong auditory stimulation as a function of alpha amplitude. J. comp. physiol. Psychol., 1959, 52, 540-545.
 62. Malmo, R.B., & Surwillo, W.W. Sleep deprivation: Changes in performance and physiological indicants of activation. Psychol. Monogr., 1960, 74, (Whole No. 502), 1-24.
 63. Chambers, D.A., Pasternak, R., & Mueller, H.F. A clamp for finger-sweat prints. Percept. Mot. Skills, 1960, 11, 35-38.
 64. Malmo, R.B. Slowing of heart rate following septal self-stimulation in rats. Science, 1961, 133, 1128-1130.
 65. Feldman, S.M. Differential effects of shock in human maze learning. J. exp. Psychol. (in press).
 66. Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and on performance. J. exp. Psychol. (in press).
-
- A. Malmo, R.B. Interference factors in delayed response in monkeys after removal of frontal lobes. J. Neurophysiol., 1942, 5, 295-308.
 - B. Cohen, L.H., Malmo, R.B., & Thale, T. Measurement of chronic psychotic over-activity by the Norwith Rating Scale. J. gen. Psychol., 1944, 30, 65-74.
 - C. Malmo, R.B., & Shagass, C. Variability of heart rate in relation to age, sex and stress. J. appl. Psychol., 1949, 2, 181-184.
 - D. McMurray, G.A. Experimental study of a case of insensitivity to pain. Arch. Neurol. Psychiat., 1950, 64, 650-667.

Dominion-Provincial Mental Health Grant 604-5-69

Physiological Studies of Psychiatric Patients and Controls

Progress Report for 1959-1960

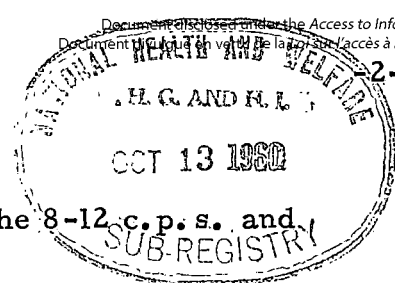
Robert B. Malmö



22 August 1960

Effects of Induced Muscle Tension

Muscle tension in psychiatric patients has been a subject for study in our laboratory over a 15-year period. We have recently become interested in securing more data on muscle tension from normal control subjects. It occurred to us that a study of induced muscle tension (in which subjects exert pressures of known amounts on a dynamometer) would be of particular value because induced muscle tension has been demonstrated to have significant general effects in relation to a wide range of behavioural phenomena (e.g., learning scores have been improved by induced tension). From the behavioural evidence, it appeared reasonable to consider that induced tension was only one of the many ways in which level of activation can be varied. It followed that if induced tension were a reliable means of varying activation level, then in addition to the behavioural effects which had already been demonstrated, induced tension should also produce regular and consistent changes in the various physiological indicants of activation. The indicants used were muscle tension from various muscles, heart rate, respiration, palmar conductance, and EEGs quantified by means of integrators, and



analyzed for frequency by means of band-pass filters in the 8-12 c.p.s. and 18-27 c.p.s. ranges. The main purpose of these experiments was the investigation of the physiological changes in 38 normal male McGill University students ranging in age from 18 to 30.

The results clearly showed close agreement between amount of muscle tension induced and changes in the wide range of physiological functions measured. It was thus clearly indicated (particularly by the EEG data) that induced muscle tension has very widespread and generalized effects and it appeared that general activation level could be altered in a regular and lawful way by induction of tension in local muscle groups (e.g., those involved in squeezing on a dynamometer).

Effect of Varied Incentive on Finger Sweating and on Palmar Conductance

Wilcott has recently suggested that the electrical measure of palmar conductance and the measure of sweat gland activity by the fingerprint stain technique are uncorrelated. It is true that with his method of recording palmar conductance, and with the experimental procedures he used, he did obtain an insignificant correlation between these two measures. However, it occurred to us that this low correlation may have been due to certain features of his method for recording palmar conductance, and it seemed important to investigate this question with methods that we have developed and found to be particularly sensitive and reliable. Furthermore, it seemed desirable not to limit the study to one of inter-individual correlations, but to look at intra-individual correlations as well.

OCT 13 1960

SUB-REGISTRY

With these points in mind an experiment was designed to study the physiological measures listed, under two distinctly different levels of incentive (one high, the other low, incentive being varied by means of instructions to the subject, and each subject serving once in the low-incentive condition and once in the other condition). Data have been collected from 60 normal males and these data are now being analyzed. Preliminary indications are that palmar conductance and finger sweating as measured by the stain technique, will turn out to be very significantly correlated, intra- and inter-individually.

Pain

Pain stimulation with the Hardy-Wolff-Goodell method has been used in this laboratory repeatedly as a means of providing mild stress under which to study the behavioural and physiological reactions of psychiatric patients. Again, careful investigations of the factors affecting pain threshold in normal subjects are very much needed. With this in mind, we have commenced to study relations between activation level and pain threshold. There are some indications from our work on sleep deprivation that pain thresholds are lowered by increasing activation level.

The first such pain study will be carried out with level of induced muscular tension as the independent variable, and the changes in tension level will be monitored by means of heart rate, respiration, and palmar conductance, continuously recorded. This study is in the pilot stage, and initial results appear encouraging.

Manuscripts and Publications

OCT 13

1. Chambers, D.A., Pasternak, Rowena, & Mueller, H.F. A clamp for finger-sweat prints. Percept. mot. Skills, 1960, 11, 35-38.
2. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Amer. J. Psychol., (in press).
3. Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and on performance. Unpublished Doctoral Dissertation, McGill University, 1960.

Meetings and Symposia (R.B. Malmo)

1. Chaired symposium on, "Experimental Foundations of Personality" at the VI Congress of the Interamerican Society of Psychology, Rio de Janeiro, Brazil, August, 1959.
2. Participated in a symposium on, "The Psychological Significance of Muscle Tension," at the Midwestern Psychological Association Meetings, St. Louis, Missouri, May, 1960.

Reprints of Publications from the Laboratory for Psychological Studies.

Allan Memorial Institute of Psychiatry

OCT 13 1960

3. Malmö, R.B., Shagass, C., Davis, J.F., Cleghorn, R.A., Graham, B.F., and Goodman, A.J. Standardized pain stimulations as controlled stress in physiological studies of psychoneurosis. Science, 1948, 108, 509-511.
4. Malmö, R.B. and Shagass, C. Physiologic Studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.
6. Malmö, R.B., Shagass, C., and Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
8. Malmö, R.B., and Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
9. Malmö, R.B., Shagass, C., and Heslam, R.M. Blood pressure responses to repeated brief stress in psychoneurosis: a study of adaptation. Canadian J. Psychol., 1951, 5, 167-179.
12. Malmö, R.B., Shagass, C., Belanger, D.J., and Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
13. Malmö, R.B., Shagass, C., and Smith, A.A. Responsiveness in chronic schizophrenia. J. Personality, 1951, 19, 359-375.
14. Malmö, R.B., Shagass, C., and Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
15. Davis, F.H. and Malmö, R.B. Electromyographic recording during interview. Amer. J. Psychiat., 1951, 107, 908-916.
16. Shagass, C. and Malmö, R.B. Psychodynamic themes and localized muscular tension during psychotherapy. Psychosom. Med., 1954, 16, 295-313.
17. Malmö, R.B., Davis, J.F., and Barza, S. Total hysterical deafness: an experimental case study. J. Personality, 1952, 21, 188-204.
18. Davis, J.F. Manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1952 (mime o.).

19. Smith, A.A. An electromyographic study of tension in interrupted and completed tasks. J. exp. Psychol., 1953, 46, 32-36
22. Malmö, R.B., Shagass, C., and Davis, F.H. Specificity of bodily reactions under stress. Res. Publ. Ass. nerv. ment. Dis., 1950, 29, 231-261.
26. Malmö, R.B. Higher functions of the nervous system. Ann. rev. Physiol., 1954, 16, 371-390.
27. Malmö, R.B. Eccles' neurophysiological model of the conditioned reflex. Canadian J. Psychol., 1954, 8, 125-129.
28. Malmö, R.B., Wallerstein, H., and Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. J. Personality, 1953, 22, 162-187.
29. Shagass, C. The sedation threshold. A method for estimating tension in psychiatric patients. EEG Clin. Neurophysiol., 1954, 6, 221-233.
30. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canadian J. Psychol., 1955, 9, 21-28.
31. Davis, J.F., Malmö, R.B., and Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canadian J. Psychol., 1954, 8, 177-186.
32. Malmö, R.B., and Wallerstein, H. Rigidity and reactive inhibition. J. abn. soc. Psychol., 1955, 50, 346-348.
33. Malmö, R.B., and Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Malmö, R.B., Boag, T.J., and Raginsky, B.B. Electromyographic study of hypnotic deafness. J. clin. exp. Hypnosis, 1954, 2, 305-317.
35. Smith, A.A., Malmö, R.B., and Shagass, C. An electromyographic study of listening and talking. Canadian J. Psychol., 1954, 8, 219-227.
36. Wallerstein, H. An electromyographic study of attentive listening. Canadian J. Psychol., 1954, 8, 228-238.
37. Surwillo, W.W. A device for recording variations in pressure of grip during tracking. Amer. J. Psychol., 1955, 68, 669-670.

38. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canadian J. Psychol., 1955, 9, 215-230.
39. Malmo, R.B. Kohlmeier, W., and Smith, A.A. Motor manifestation of conflict in interview. J. abn. soc. Psychol., 1956, 52, 268-271.
41. Bartoshuk, A.K. Electromyographic gradients and EEG amplitude during motivated listening. Canadian J. Psychol., 1956, 10, 156-164.
42. Davis, J.F. Operator's Manual: A. M. I. Integrator system. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1956 (mimeo.).
43. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.
44. Malmo, R.B. and Davis, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.
45. Hecaen, H., Penfield, W., Bertrand, C., and Malmo, R.B. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. Arch. Neurol. Psychiat., 1956, 75, 400-434.
46. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.
47. Stennett, R.G. The relationship of performance level to level of arousal. J. exp. Psychol., 1957, 54, 54-61.
48. Malmo, R.B., Boag, T.J., and Smith, A.A. Physiological study of personal interaction. Psychosom. Med., 1957, 19, 105-119.
49. Belanger, D. "Gradients" musculaires et processus mentaux superieurs. Canad. J. Psychol., 1957, 11, 113-122.
51. Malmo, R.B. Anxiety and behavioral arousal. Psychol. Rev., 1957, 64, 276-287.
52. Davis, J.F., Stennett, R.G., and Quilter, R.E. An auditory tracking device designed for use in conjunction with continuous EEG recording. Percep. Mot. Skills, 1957, 7, 239-244.
54. Surwillo, W.W. A new method of motivating human behavior in laboratory investigations. Amer. J. Psychol., 1958, 71, 432-436.

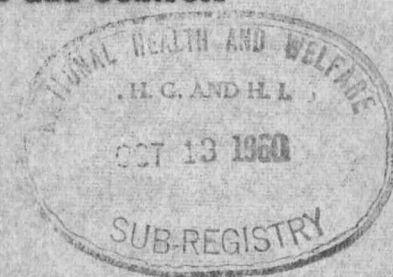
- 4-
- NATIONAL HEALTH AND WELFARE
H.C. AND H.L.
SUB-REGISTRY
56. Ross, W.R.D., and Davis, J.F. Stable band-pass filters for electroencephalography. IRE Canadian Convention Record, 1958, Paper No. 860, 202-206.
 57. Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. J. exp. Psychol., 1959, 58, 117-128.
 59. Malmo, R.B. Activation: a neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
 60. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute, 1959 (mimeo.)
 61. Bartoshuk, A.K. Electromyographic reactions to strong auditory stimulation as a function of alpha amplitude. J. comp. physiol. Psychol., 1959, 52, 540-545.
 62. Malmo, R.B., & Surwillo, W.W. Sleep deprivation: Changes in performance and physiological indicants of activation. (In press - Psychological Monographs, 1960).
-
7. Malmo, R.B., Shagass, C., and Davis, F.H. Symptom specificity and bodily reactions during psychiatric interview. Psychosom. Med., 1950, 12, 362-376.

Dominion-Provincial Mental Health Grant 604-5-69

Physiological Studies of Psychiatric Patients and Controls

Progress Report for 1959-1960

Robert B. Malmö



22 August 1960

Effects of Induced Muscle Tension

Muscle tension in psychiatric patients has been a subject for study in our laboratory over a 15-year period. We have recently become interested in securing more data on muscle tension from normal control subjects. It occurred to us that a study of induced muscle tension (in which subjects exert pressures of known amounts on a dynamometer) would be of particular value because induced muscle tension has been demonstrated to have significant general effects in relation to a wide range of behavioural phenomena (e.g., learning scores have been improved by induced tension). From the behavioural evidence, it appeared reasonable to consider that induced tension was only one of the many ways in which level of activation can be varied. It followed that if induced tension were a reliable means of varying activation level, then in addition to the behavioural effects which had already been demonstrated, induced tension should also produce regular and consistent changes in the various physiological indicants of activation. The indicants used were muscle tension from various muscles, heart rate, respiration, palmar conductance, and EEGs quantified by means of integrators, and

analyzed for frequency by means of band-pass filters in the 8-12 c.p.s. and 18-27 c.p.s. ranges. The main purpose of these experiments was the investigation of the physiological changes in 38 normal male McGill University students ranging in age from 18 to 30.

The results clearly showed close agreement between amount of muscle tension induced and changes in the wide range of physiological functions measured. It was thus clearly indicated (particularly by the EEG data) that induced muscle tension has very widespread and generalized effects and it appeared that general activation level could be altered in a regular and lawful way by induction of tension in local muscle groups (e.g., those involved in squeezing on a dynamometer).

Effect of Varied Incentive on Finger Sweating and on Palmar Conductance

Wilcott has recently suggested that the electrical measure of palmar conductance and the measure of sweat gland activity by the fingerprint stain technique are uncorrelated. It is true that with his method of recording palmar conductance, and with the experimental procedures he used, he did obtain an insignificant correlation between these two measures. However, it occurred to us that this low correlation may have been due to certain features of his method for recording palmar conductance, and it seemed important to investigate this question with methods that we have developed and found to be particularly sensitive and reliable. Furthermore, it seemed desirable not to limit the study to one of inter-individual correlations, but to look at intra-individual correlations as well.

With these points in mind an experiment was designed to study the physiological measures listed, under two distinctly different levels of incentive (one high, the other low, incentive being varied by means of instructions to the subject, and each subject serving once in the low-incentive condition and once in the other condition). Data have been collected from 60 normal males and these data are now being analyzed. Preliminary indications are that palmar conductance and finger sweating as measured by the stain technique, will turn out to be very significantly correlated, intra- and inter-individually.

Pain

Pain stimulation with the Hardy-Wolff-Goodell method has been used in this laboratory repeatedly as a means of providing mild stress under which to study the behavioural and physiological reactions of psychiatric patients. Again, careful investigations of the factors affecting pain threshold in normal subjects are very much needed. With this in mind, we have commenced to study relations between activation level and pain threshold. There are some indications from our work on sleep deprivation that pain thresholds are lowered by increasing activation level.

The first such pain study will be carried out with level of induced muscular tension as the independent variable, and the changes in tension level will be monitored by means of heart rate, respiration, and palmar conductance, continuously recorded. This study is in the pilot stage, and initial results appear encouraging.

Manuscripts and Publications

1. Chambers, D.A., Pasternak, Rowena, & Mueller, H.F. A clamp for finger-sweat prints. Percept. mot. Skills, 1960, 11, 35-38.
2. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Amer. J. Psychol., (in press).
3. Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and on performance. Unpublished Doctoral Dissertation, McGill University, 1960.

Meetings and Symposia (R.B. Malmo)

1. Chaired symposium on, "Experimental Foundations of Personality" at the VI Congress of the Interamerican Society of Psychology, Rio de Janeiro, Brazil, August, 1959.
2. Participated in a symposium on, "The Psychological Significance of Muscle Tension," at the Midwestern Psychological Association Meetings, St. Louis, Missouri, May, 1960.

Reprints of Publications from the Laboratory for Psychological Studies.

Allan Memorial Institute of Psychiatry

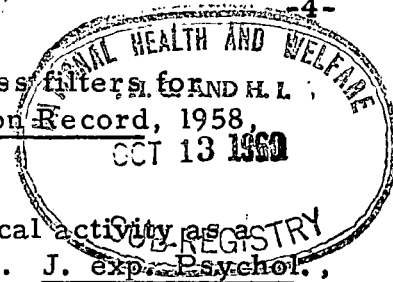
13 1360

3. Malmo, R.B., Shagass, C., Davis, J.F., Cleghorn, R.A., Graham, B.F., and Goodman, A.J. Standardized pain stimulations as controlled stress in physiological studies of psychoneurosis. Science, 1948, 108, 509-511.
4. Malmo, R.B. and Shagass, C. Physiologic Studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.
6. Malmo, R.B., Shagass, C., and Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
8. Malmo, R.B., and Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
9. Malmo, R.B., Shagass, C., and Heslam, R.M. Blood pressure responses to repeated brief stress in psychoneurosis: a study of adaptation. Canadian J. Psychol., 1951, 5, 167-179.
12. Malmo, R.B., Shagass, C., Belanger, D.J., and Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
13. Malmo, R.B., Shagass, C., and Smith, A.A. Responsiveness in chronic schizophrenia. J. Personality, 1951, 19, 359-375.
14. Malmo, R.B., Shagass, C., and Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
15. Davis, F.H. and Malmo, R.B. Electromyographic recording during interview. Amer. J. Psychiat., 1951, 107, 908-916.
16. Shagass, C. and Malmo, R.B. Psychodynamic themes and localized muscular tension during psychotherapy. Psychosom. Med., 1954, 16, 295-313.
17. Malmo, R.B., Davis, J.F., and Barza, S. Total hysterical deafness: an experimental case study. J. Personality, 1952, 21, 188-204.
18. Davis, J.F. Manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1952 (mime o.).

19. Smith, A.A. An electromyographic study of tension in interrupted and completed tasks. J. exp. Psychol., 1953, 46, 32-36.
22. Malmö, R.B., Shagass, C., and Davis, F.H. Specificity of bodily reactions under stress. Res. Publ. Ass. nerv. ment. Dis. 1950, 29, 231-261.
26. Malmö, R.B. Higher functions of the nervous system. Ann. rev. Physiol., 1954, 16, 371-390.
27. Malmö, R.B. Eccles' neurophysiological model of the conditioned reflex. Canadian J. Psychol., 1954, 8, 125-129.
28. Malmö, R.B., Wallerstein, H., and Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. J. Personality, 1953, 22, 162-187.
29. Shagass, C. The sedation threshold. A method for estimating tension in psychiatric patients. EEG Clin. Neurophysiol., 1954, 6, 221-233.
30. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canadian J. Psychol., 1955, 9, 21-28.
31. Davis, J.F., Malmö, R.B., and Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canadian J. Psychol., 1954, 8, 177-186.
32. Malmö, R.B., and Wallerstein, H. Rigidity and reactive inhibition. J. abn. soc. Psychol., 1955, 50, 346-348.
33. Malmö, R.B., and Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Malmö, R.B., Boag, T.J., and Raginsky, B.B. Electromyographic study of hypnotic deafness. J. clin. exp. Hypnosis, 1954, 2, 305-317.
35. Smith, A.A., Malmö, R.B., and Shagass, C. An electromyographic study of listening and talking. Canadian J. Psychol., 1954, 8, 219-227.
36. Wallerstein, H. An electromyographic study of attentive listening. Canadian J. Psychol., 1954, 8, 228-238.
37. Surwillo, W.W. A device for recording variations in pressure of grip during tracking. Amer. J. Psychol., 1955, 68, 669-670.

38. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canadian J. Psychol., 1955, 9, 215-230.
39. Malmo, R.B. Kohlmeier, W., and Smith, A.A. Motor manifestation of conflict in interview. J. abn. soc. Psychol., 1956, 52, 268-271.
41. Bartoshuk, A.K. Electromyographic gradients and EEG amplitude during motivated listening. Canadian J. Psychol., 1956, 10, 156-164.
42. Davis, J.F. Operator's Manual: A.M.I. Integrator system. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1956 (mimeo.).
43. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.
44. Malmo, R.B. and Davis, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.
45. Hecaen, H., Penfield, W., Bertrand, C., and Malmo, R.B. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. Arch. Neurol. Psychiat., 1956, 75, 400-434.
46. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.
47. Stennett, R.G. The relationship of performance level to level of arousal. J. exp. Psychol., 1957, 54, 54-61.
48. Malmo, R.B., Boag, T.J., and Smith, A.A. Physiological study of personal interaction. Psychosom. Med., 1957, 19, 105-119.
49. Belanger, D. "Gradients" musculaires et processus mentaux superieurs. Canad. J. Psychol., 1957, 11, 113-122.
51. Malmo, R.B. Anxiety and behavioral arousal. Psychol. Rev., 1957, 64, 276-287.
52. Davis, J.F., Stennett, R.G., and Quilter, R.E. An auditory tracking device designed for use in conjunction with continuous EEG recording. Percep. Mot. Skills, 1957, 7, 239-244.
54. Surwillo, W.W. A new method of motivating human behavior in laboratory investigations. Amer. J. Psychol., 1958, 71, 432-436.

56. Ross, W.R.D., and Davis, J.F. Stable band-pass filters for electroencephalography. IRE Canadian Convention Record, 1958, Paper No. 860, 202-206.
57. Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. J. exp. Psychol., 1959, 58, 117-128.
59. Malmo, R.B. Activation: a neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
60. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute, 1959 (mimeo.)
61. Bartoshuk, A.K. Electromyographic reactions to strong auditory stimulation as a function of alpha amplitude. J. comp. physiol. Psychol., 1959, 52, 540-545.
62. Malmo, R.B., & Surwillo, W.W. Sleep deprivation: Changes in performance and physiological indicants of activation. (In press - Psychological Monographs, 1960).
7. Malmo, R.B., Shagass, C., and Davis, F.H. Symptom specificity and bodily reactions during psychiatric interview. Psychosom. Med., 1950, 12, 362-376.

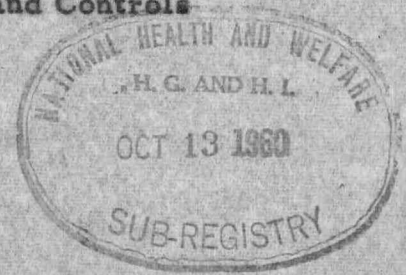


Dominion-Provincial Mental Health Grant 604-5-69

Physiological Studies of Psychiatric Patients and Controls

Progress Report for 1959-1960

Robert B. Malmö



22 August 1960

Effects of Induced Muscle Tension

Muscle tension in psychiatric patients has been a subject for study in our laboratory over a 15-year period. We have recently become interested in securing more data on muscle tension from normal control subjects. It occurred to us that a study of induced muscle tension (in which subjects exert pressures of known amounts on a dynamometer) would be of particular value because induced muscle tension has been demonstrated to have significant general effects in relation to a wide range of behavioural phenomena (e.g., learning scores have been improved by induced tension). From the behavioural evidence, it appeared reasonable to consider that induced tension was only one of the many ways in which level of activation can be varied. It followed that if induced tension were a reliable means of varying activation level, then in addition to the behavioural effects which had already been demonstrated, induced tension should also produce regular and consistent changes in the various physiological indicants of activation. The indicants used were muscle tension from various muscles, heart rate, respiration, palmar conductance, and EEGs quantified by means of integrators, and

analyzed for frequency by means of band-pass filters in the 8-12 c.p.s. and 18-27 c.p.s. ranges. The main purpose of these experiments was the investigation of the physiological changes in 38 normal male McGill University students ranging in age from 18 to 30.

The results clearly showed close agreement between amount of muscle tension induced and changes in the wide range of physiological functions measured. It was thus clearly indicated (particularly by the EEG data) that induced muscle tension has very widespread and generalized effects and it appeared that general activation level could be altered in a regular and lawful way by induction of tension in local muscle groups (e.g., those involved in squeezing on a dynamometer).

Effect of Varied Incentive on Finger Sweating and on Palmar Conductance

Wilcott has recently suggested that the electrical measure of palmar conductance and the measure of sweat gland activity by the fingerprint stain technique are uncorrelated. It is true that with his method of recording palmar conductance, and with the experimental procedures he used, he did obtain an insignificant correlation between these two measures. However, it occurred to us that this low correlation may have been due to certain features of his method for recording palmar conductance, and it seemed important to investigate this question with methods that we have developed and found to be particularly sensitive and reliable. Furthermore, it seemed desirable not to limit the study to one of inter-individual correlations, but to look at intra-individual correlations as well.

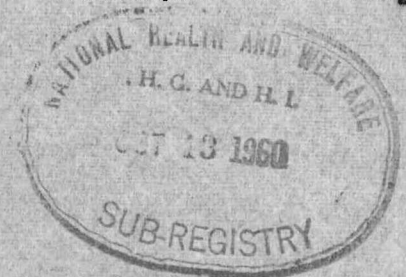
With these points in mind an experiment was designed to study the physiological measures listed, under two distinctly different levels of incentive (one high, the other low, incentive being varied by means of instructions to the subject, and each subject serving once in the low-incentive condition and once in the other condition). Data have been collected from 60 normal males and these data are now being analyzed. Preliminary indications are that palmar conductance and finger sweating as measured by the stain technique, will turn out to be very significantly correlated, intra- and inter-individually.

Pain

Pain stimulation with the Hardy-Wolff-Goodell method has been used in this laboratory repeatedly as a means of providing mild stress under which to study the behavioural and physiological reactions of psychiatric patients. Again, careful investigations of the factors affecting pain threshold in normal subjects are very much needed. With this in mind, we have commenced to study relations between activation level and pain threshold. There are some indications from our work on sleep deprivation that pain thresholds are lowered by increasing activation level.

The first such pain study will be carried out with level of induced muscular tension as the independent variable, and the changes in tension level will be monitored by means of heart rate, respiration, and palmar conductance, continuously recorded. This study is in the pilot stage, and initial results appear encouraging.

Manuscripts and Publications



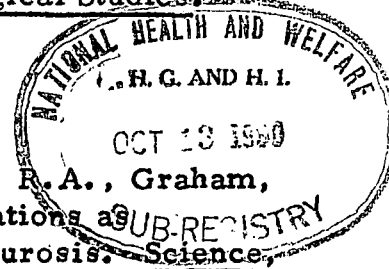
1. Chambers, D.A., Pasternak, Rowena, & Mueller, H.F. A clamp for finger-sweat prints. Percept. mot. Skills, 1960, 11, 35-38.
2. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Amer. J. Psychol., (in press).
3. Pinneo, L.R. The effects of induced muscle tension during tracking on level of activation and on performance. Unpublished Doctoral Dissertation, McGill University, 1960.

Meetings and Symposia (R.B. Malmo)

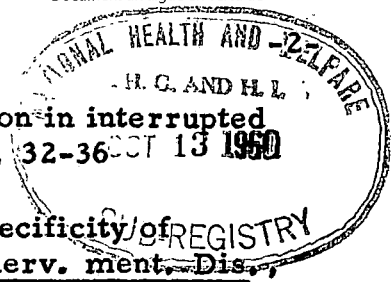
1. Chaired symposium on, "Experimental Foundations of Personality" at the VI Congress of the Interamerican Society of Psychology, Rio de Janeiro, Brazil, August, 1959.
2. Participated in a symposium on, "The Psychological Significance of Muscle Tension," at the Midwestern Psychological Association Meetings, St. Louis, Missouri, May, 1960.

Reprints of Publications from the Laboratory for Psychological Studies

Allan Memorial Institute of Psychiatry



3. Malmo, R.B., Shagass, C., Davis, J.F., Cleghorn, R.A., Graham, B.F., and Goodman, A.J. Standardized pain stimulations as controlled stress in physiological studies of psychoneurosis. Science, 1948, 108, 509-511.
4. Malmo, R.B. and Shagass, C. Physiologic Studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.
6. Malmo, R.B., Shagass, C., and Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
8. Malmo, R.B., and Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
9. Malmo, R.B., Shagass, C., and Heslam, R.M. Blood pressure responses to repeated brief stress in psychoneurosis: a study of adaptation. Canadian J. Psychol., 1951, 5, 167-179.
12. Malmo, R.B., Shagass, C., Belanger, D.J., and Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
13. Malmo, R.B., Shagass, C., and Smith, A.A. Responsiveness in chronic schizophrenia. J. Personality, 1951, 19, 359-375.
14. Malmo, R.B., Shagass, C., and Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
15. Davis, F.H. and Malmo, R.B. Electromyographic recording during interview. Amer. J. Psychiat., 1951, 107, 908-916
16. Shagass, C. and Malmo, R.B. Psychodynamic themes and localized muscular tension during psychotherapy. Psychosom. Med., 1954, 16, 295-313.
17. Malmo, R.B., Davis, J.F., and Barza, S. Total hysterical deafness: an experimental case study. J. Personality, 1952, 21, 188-204.
18. Davis, J.F. Manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1952 (mime o.).

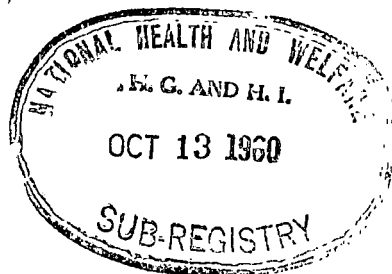


19. Smith, A.A. An electromyographic study of tension in interrupted and completed tasks. J. exp. Psychol., 1953, 46, 32-36.
22. Malmo, R.B., Shagass, C., and Davis, F.H. Specificity of bodily reactions under stress. Res. Publ. Ass. nerv. ment. Dis., 1950, 29, 231-261.
26. Malmo, R.B. Higher functions of the nervous system. Ann. rev. Physiol., 1954, 16, 371-390.
27. Malmo, R.B. Eccles' neurophysiological model of the conditioned reflex. Canadian J. Psychol., 1954, 8, 125-129.
28. Malmo, R.B., Wallerstein, H., and Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. J. Personality, 1953, 22, 162-187.
29. Shagass, C. The sedation threshold. A method for estimating tension in psychiatric patients. EEG Clin. Neurophysiol., 1954, 6, 221-233.
30. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canadian J. Psychol., 1955, 9, 21-28.
31. Davis, J.F., Malmo, R.B., and Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canadian J. Psychol., 1954, 8, 177-186.
32. Malmo, R.B., and Wallerstein, H. Rigidity and reactive inhibition. J. abn. soc. Psychol., 1955, 50, 346-348.
33. Malmo, R.B., and Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Malmo, R.B., Boag, T.J., and Raginsky, B.B. Electromyographic study of hypnotic deafness. J. clin. exp. Hypnosis, 1954, 2, 305-317.
35. Smith, A.A., Malmo, R.B., and Shagass, C. An electromyographic study of listening and talking. Canadian J. Psychol., 1954, 8, 219-227.
36. Wallerstein, H. An electromyographic study of attentive listening. Canadian J. Psychol., 1954, 8, 228-238.
37. Surwillo, W.W. A device for recording variations in pressure of grip during tracking. Amer. J. Psychol., 1955, 68, 669-670.

38. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canadian J. Psychol., 1955, 9, 215-230.
39. Malmo, R.B. Kohlmeyer, W., and Smith, A.A. Motor manifestation of conflict in interview. J. abn. soc. Psychol., 1956, 52, 268-271.
41. Bartoshuk, A.K. Electromyographic gradients and EEG amplitude during motivated listening. Canadian J. Psychol., 1956, 10, 156-164.
42. Davis, J.F. Operator's Manual: A.M.I. Integrator system. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1956 (mimeo.).
43. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.
44. Malmo, R.B. and Davis, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.
45. Hecaen, H., Penfield, W., Bertrand, C., and Malmo, R.B. The syndrome of apractognosia due to lesions of the minor cerebral hemisphere. Arch. Neurol. Psychiat., 1956, 75, 400-434.
46. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.
47. Stennett, R.G. The relationship of performance level to level of arousal. J. exp. Psychol., 1957, 54, 54-61.
48. Malmo, R.B., Boag, T.J., and Smith, A.A. Physiological study of personal interaction. Psychosom. Med., 1957, 19, 105-119.
49. Belanger, D. "Gradients" musculaires et processus mentaux supérieurs. Canad. J. Psychol., 1957, 11, 113-122.
51. Malmo, R.B. Anxiety and behavioral arousal. Psychol. Rev., 1957, 64, 276-287.
52. Davis, J.F., Stennett, R.G., and Quilter, R.E. An auditory tracking device designed for use in conjunction with continuous EEG recording. Percep. Mot. Skills, 1957, 7, 239-244.
54. Surwillo, W.W. A new method of motivating human behavior in laboratory investigations. Amer. J. Psychol., 1958, 71, 432-436.

56. Ross, W.R.D., and Davis, J.F. Stable band-pass filters for electroencephalography. IRE Canadian Convention Record, 1958, Paper No. 860, 202-206.
57. Schnore, M.M. Individual patterns of physiological activity as a function of task differences and degree of arousal. J. exp. Psychol., 1959, 58, 117-128.
59. Malmo, R.B. Activation: a neuropsychological dimension. Psychol. Rev., 1959, 66, 367-386.
60. Malmo, R.B. & Davis, J.F. A monopolar method for the measurement of palmar conductance. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute, 1959 (mimeo.)
61. Bartoshuk, A.K. Electromyographic reactions to strong auditory stimulation as a function of alpha amplitude. J. comp. physiol. Psychol., 1959, 52, 540-545.
62. Malmo, R.B., & Surwillo, W.W. Sleep deprivation: Changes in performance and physiological indicants of activation. (In press - Psychological Monographs, 1960).
7. Malmo, R.B., Shagass, C., and Davis, F.H. Symptom specificity and bodily reactions during psychiatric interview. Psychosom. Med., 1950, 12, 362-376.

ACTIVATION: A NEUROPSYCHOLOGICAL
DIMENSION¹



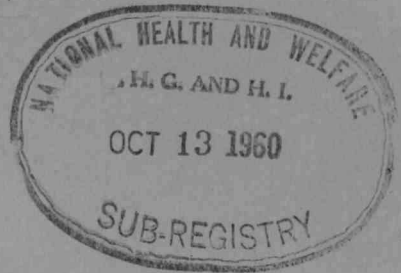
ROBERT B. MALMO

Allan Memorial Institute, McGill University

Reprinted from
PSYCHOLOGICAL REVIEW
Vol. 66 No. 6 November 1959

000096

PA 62
ACTIVATION: A NEUROPSYCHOLOGICAL
DIMENSION¹



ROBERT B. MALMO

Allan Memorial Institute, McGill University

Reprinted from
PSYCHOLOGICAL REVIEW
Vol. 66 No. 6 November 1959

000097

ACTIVATION: A NEUROPSYCHOLOGICAL DIMENSION¹

ROBERT B. MALMO

Allan Memorial Institute, McGill University

There have been three main lines of approach to the problem of activation: (a) through electroencephalography and neurophysiology, (b) through physiological studies of "behavioral energetics," and (c) through the learning theorists' search for a satisfactory measure of drive. Before attempting a formal definition of activation, I shall briefly describe these three different approaches to the concept.

*Neurophysiological approach: Lindsley's Activation Theory.*² The neuro-

¹ Support for some of the research reported herein has come from the following sources: National Institute of Mental Health, National Institutes of Health, United States Public Health Service: Grant Number M-1475; Medical Research and Development Division, Office of the Surgeon General, Department of the United States Army: Contract Number DA-49-007-MD-626; Defence Research Board, Department of National Defence, Canada: Grant Number 9425-04; and National Research Council of Canada: Grant Number A. P. 29.

Grateful acknowledgment is made to A. Amsel, R. C. Davis, S. M. Feldman, P. Milner, M. M. Schnore, R. G. Stennett, D. J. Ehrlich and L. R. Pinneo for constructive criticism of the manuscript.

The main parts of this paper were presented in a Symposium entitled, "Experimental Foundations of Clinical Psychology," under the chairmanship of Arthur J. Bachrach, at the University of Virginia, April 1-2, 1959. To Ian P. Stevenson, who was the discussant of my paper on that Symposium, I owe a debt of gratitude for his very helpful comments.

² I am using neuropsychology in a rather broad sense, meaning to include the work often referred to by the term "psychophysiology." This usage implies that the chief problems being studied are psychological ones, and it also stresses the importance of neurophysiological techniques. It is true

physiological approach to activation had its origin in electroencephalography (EEG). Early workers in the EEG field soon discovered that there were distinctive wave patterns characterizing the main levels of psychological functioning in the progression from deep sleep to highly alerted states of activity (Jasper, 1941). In deep sleep large low-frequency waves predominate. In light sleep and drowsy states the frequencies are not as low as in deep sleep, but there are more low-frequency waves than in the wakeful states. In relaxed wakefulness there is a predominance of waves in the alpha (8-12 c.p.s.) range that gives way to beta frequencies (approximately 18-30 c.p.s.) when the *S* is moderately alert. Under highly alerting and exciting conditions beta waves predominate. In addition to the increased frequency of the waves under these conditions of heightened alertness there is also a change from a regular synchronized appearance of the tracing to an irregular desynchronized tracing, usually of reduced amplitude.

that, strictly speaking, many of the physiological techniques in use are not neurophysiological ones; yet our main interest lies in the central neural control of the physiological functions under study rather than in the peripheral events themselves.

Later on in the paper I shall attempt a formal definition of activation. For the first section of the paper, I believe that it will be sufficient to say that in using the term "activation" I am referring to the intensive dimension of behavior. "Arousal" is often used interchangeably with activation; and level of drive is a very similar concept. For instance, a drowsy *S* is low, an alert *S* is high in activation.

For Lindsley's theory, desynchronization (called "activation pattern") became the single most important EEG phenomenon. My use of the term "desynchronization" is purely descriptive. Desynchronization or "flattening" in the EEG tracing was consistently found associated with increased alertness in a large variety of experiments with animal and human Ss. The consistency and generality of this phenomenon suggested the existence of mechanisms in the brain mediating behavioral functions having to do with levels of alertness, although at the time that the original observations were made it was not at all clear what these neural mechanisms were.

With the discovery of the ascending reticular activating system (ARAS), however, there was rapid and very significant advance in theory and experimentation. Some of the most important general findings have been as follows: (a) Lesions in the ARAS abolished "activation" of the EEG and produced a behavioral picture of lethargy and somnolence (Lindsley, 1957). (b) The "activation pattern" in the EEG was reproduced by electrical stimulation of the ARAS. Furthermore, in the monkey, Fuster (1958) recently found that concurrent ARAS stimulation of moderate intensity improved accuracy and speed of visual discrimination reaction. He also found that higher intensities had the opposite effect, producing diminution of correct responses and increase of reaction times. Interpretation of these latter findings is complicated by the fact that they were obtained with stimulation intensities higher than the threshold for the elicitation of observable motor effects such as generalized muscular jerks. It is not stated whether intensity of stimulation was systematically studied. In any event, these observations of deleterious effect from

high intensity stimulation are of considerable interest because they are what might be expected according to the activation theory.

The activation theory as first stated by Lindsley (1951)—although introduced in the handbook chapter on emotion—was, from the outset, conceived by him to be broader than an explanatory concept for emotional behavior. The theory was elaborated by Hebb (1955) in an attempt to solve the problem of drives. With the continuous flow of new experimental data on the ARAS (Lindsley, 1957), this area of neuropsychological investigation appears to be heading toward an important breakthrough. I shall attempt to state very briefly the main points of the current theory, drawing upon the ideas of several authors. According to this theory, the continuum extending from deep sleep at the low activation end to "excited states"³ at the high activation end is very largely a function of cortical bombardment by the ARAS, such that the greater the cortical bombardment the higher the activation. Further, the relation between activation and behavioral efficiency (cue function or level of performance) is described by an inverted U curve. That is, from low activation up to a point that is optimal for a given function, level of performance rises monotonically with increasing activation level, but beyond this optimal point the relation becomes non-monotonic: further increase in activation beyond this point produces a fall in performance level, this fall being

³ The expression "excited states" is frequently used to refer to the upper end of the activation continuum. In using this term I do not wish to imply increased overt activity. In fact, overt activity may be reduced to a very low level at the high end of the continuum, when—for example—a person is immobilized by terror.

directly related to the amount of the increase in level of activation.

Principles of neural action that could account for the reversal in the effects of nonspecific neural bombardment of the cortex by the ARAS have long been known (Lorente de Nó, 1939, p. 428). Circulation of neural impulses in a closed chain of neurons (or "cell assembly" to use Hebb's [1949] term) may be facilitated by impulses arriving outside the chain (e.g. from the ARAS). According to Lorente de Nó's schema, such extraneous impulses have the effect of stimulating certain neurons subliminally thus making it possible for an impulse from within the chain to finish the job, that is make it fire at the appropriate time in the sequence, when alone, without the prior hit, it would have failed to fire it.

Again, according to the same account by Lorente de Nó (1939, p. 428), the deleterious effects of overstimulation from impulses outside the chain can be explained. A neuron in the chain may fail to respond to stimulation if owing to repeated activity it acquires a high threshold, and this failure to transmit the circulating impulses would mean cessation of activity in a cell assembly. I proposed this kind of explanation previously (1958) to account for the downturn in the inverted U curve as an alternative to Hebb's suggestion that "the greater bombardment may interfere with the delicate adjustments involved in cue function, perhaps by facilitating irrelevant responses (a high D arouses conflicting sH_R 's?)" (Hebb, 1955, p. 250).

It seems reasonable to suppose that as diffuse bombardment from the ARAS greatly exceeds an amount that is optimal for some simple psychological function being mediated by a particular cell assembly, the operation

of that cell assembly will be impaired, and that the performance being mediated by it will suffer accordingly. This line of reasoning suggests that the inverted U relation should be found in quite simple psychological functions. Present evidence appears to support this suggestion. A recent (unpublished) experiment by Bélanger and Feldman, that I shall describe later in this paper, indicates that in rats the inverted U relation is found with simple bar pressing performance, and an experiment by Finch (1938) suggests that even such a simple response as the unconditioned salivary response yields the inverted U curve when plotted against activation level.

It may be noted that according to a response competition hypothesis, the inverted U relation should appear most prominently in complex functions where opportunities for habit interference are greater than they are in the case of simple functions. According to the response competition hypothesis, in the limiting case where response is so simple that habit interference is negligible, the relation between response strength and activation level should be monotonic. Therefore, finding the non-monotonic relation in such simple responses as bar pressing and salivation raises strong doubts that the habit interference explanation can account for the seemingly pervasive phenomenon of the inverted U curve.

Principle of activation growing out of work on behavioral intensity. Even before the EEG work on desynchronization, the behavioral evidence had suggested the existence of some brain mechanism like the ARAS. The writings of Duffy (1951, 1957), Freeman (1948), and others of the "energetics" group have long stressed the importance of an intensity dimension in behavior.

In an attempt to obtain a measure

of this intensity variable, Duffy relied mainly on records of muscular tension (1932) while Freeman's favorite indicator was palmar conductance (1948). These workers concluded from their experiments that there was a lawful relationship between a state of the organism, called "arousal," "energy mobilization," "activation," or simply "intensity" and level of performance. Moreover they suggested that the relationship might be described by an inverted U curve (Duffy, 1957). This suggestion has proved heuristic as indicated by the current experimental attack on the inverted U hypothesis (Stennett, 1957a; Bindra, 1959; Cofer, 1959; Kendler, 1959).

The inverted U shaped curve has been shown to hold in numerous learning and performance situations where the amount of induced muscle tension was varied systematically (Courts, 1942). It is tempting to conclude that tension induction is simply one of the many ways to increase activation level, but as Courts' (1942) discussion suggests this conclusion would be premature. It is possible that squeezing on a dynamometer, a typical means of inducing tension in these experiments, may produce generalized activation effects as some data from Freeman indicate (1948, p. 71). But Freeman's data are insufficient to establish this point, and there are alternative explanations for the relationship between the performance data and induced tension (Courts, 1942). By repeating the induced-tension experiments with simultaneous recordings of EEG and other physiological functions it would be possible to determine how general the effects of inducing tension actually are. Such direct tests of the activation hypothesis are very much needed.

Drive and activation. A third approach to the activation principle was

made by learning theorists, especially those of the Hull school. I have argued elsewhere (Malmo, 1958) that general drive (*D*), without the steering component, became identical in principle with activation or arousal. Set aside for the moment the attractive possibility of using ARAS as a neural model for mediation of *D*, and consider only the methodological advantages of physiological measures in the quantification of *D*. It seems that none of the other attempts to measure *D* have been really satisfactory, and that physiological indicants where applied have been surprisingly effective. Learning theorists up to the present time have made only very occasional use of physiological measures. For instance, in arguing that a previously painful stimulus had lost its drive properties, Brown (1955) cited the absence of physiological reaction when the stimulus was applied. More recently, Spence (1958) has reported some success with physiological measures in his studies of "emotionally-based" drive.

In keeping with traditional views concerning the place of physiological measures in psychology, on those few occasions that they were employed at all they were applied to aversive or emotionally based drive. According to the activation principle, however, it should be possible to use physiological measures to gauge appetitionally based as well as aversively based drive. This means, for instance, that in a water deprivation experiment there should be close correspondence between number of hours of deprivation and physiological level. That is, heart rate, for example, should be higher in an animal performing in a Skinner box after 36 hours of deprivation than after 24, higher still after 48 hours of deprivation and so on. In my Nebraska Symposium paper I stated that, as far

as I was aware, this kind of experiment had not been reported (Malmo, 1958, p. 236).

Bélanger and Feldman in Montreal have recently completed such an experiment, and, as can be seen by inspecting Fig. 1, the results were as predicted by the activation hypothesis. Heart rate in rats showed progressive change corresponding with increasing hours of water deprivation. Although there were only seven rats in the group, this change in heart rate was highly significant. Deprivations were carried out serially on the same group of animals, commencing at 12 hours and proceeding to 24, 48 hours and so on with sufficient hydration (four to seven days) between deprivation periods to prevent any cumulative effects from affecting the experiments. Heart rate was picked up by means of wire electrodes inserted in the skin of the animals and was amplified and registered graphically by means of a Sanborn electrocardiograph. Particular care was

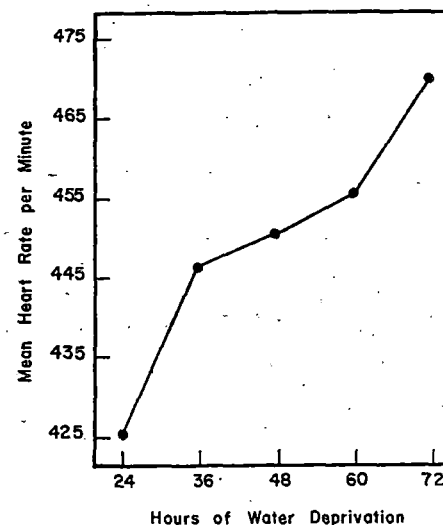


FIG. 1. Data from Bélanger and Feldman showing relation between water deprivation and heart rate in rats ($N=7$). See text for explanation.

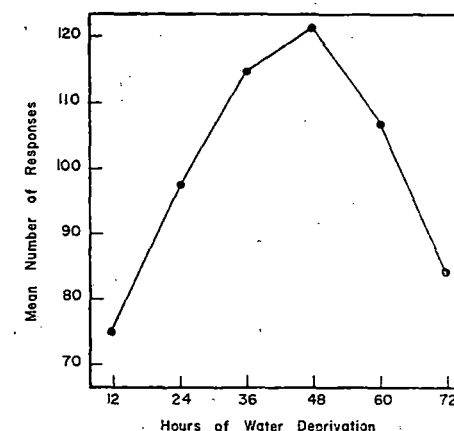


FIG. 2. Data from Bélanger and Feldman showing relation between water deprivation and Skinner box performance in rats ($N=7$). See text for explanation.

taken to record heart rate under nearly the same conditions of stimulation each time, that is, when the animal was pressing on the lever in the Skinner box or during drinking from the dispenser immediately after pressing. Under these conditions it was not possible to obtain sufficient heart-rate data at the 12-hour deprivation interval. Testing the animal under constant stimulating conditions is a very important methodological consideration. Some exploratory observations indicated that heart-rate measurements taken in a restraining compartment did not agree with those taken under the carefully controlled stimulus conditions provided by the Skinner box. I shall return to this finding later on because, aside from its methodological importance, I believe that it has considerable theoretical significance as well.

Figure 2 presents the behavioral data which are again in remarkably good agreement with prediction from the activation hypothesis. Up to the 48-hour deprivation interval there is an increasing monotonic relationship between number of bar presses and

hours of deprivation which is strictly in accordance with Hullian theory. The accompanying rise in heart rate suggests that for this part of the curve, hours of deprivation and the physiological indicant are roughly equivalent as measures of drive. But after the 48-hour point on the curves, the combined heart rate and behavioral data support predictions previously made from activation theory (Malmö, 1958) and suggest that the Hullian position requires revision. This kind of downward turn in the response curve has usually been attributed to a physical weakening of the animal due to the deprivation of food or water. In the absence of physiological data such an assumption appeared reasonable in many cases, although it did not account for response decrement in certain experiments where physical weakening seemed to be ruled out (Finan, 1940; Freeman, 1940; Fuster, 1958; Kaplan, 1952; Stennett, 1957a). Attack on this problem with physiological methods should soon provide a definitive answer concerning the main determinants of this response decrement. The present experiment represents an important first step in a program of animal studies that should go a long way towards solving this problem. It is not claimed that this one experiment demolishes the inanition hypothesis, but it does seem that the results are opposed to it. Heart rate in the Minnesota starvation experiments was found lowered in the weakened individuals (Malmö, 1958, p. 252) whereas heart rate in the present experiment was markedly increased during the period when number of responses was declining. Moreover, Bélanger was careful to record the weights of the animals all through the experiments, and he observed only very slight changes in weight, even at the 72-hour deprivation interval. Again, it should be

stressed that all through the experiment the animals received four to seven days of hydration between conditions. Furthermore, it is interesting to note that the animals continued to press the bar at fairly regular intervals in the high deprivation conditions (with response decrement). That is, their behavior did not appear as though they had "given up." The acts of pressing continued to occur regularly, only they were separated by longer temporal intervals than under more optimal conditions of deprivation.

The increasing monotonic curve for heart rate did not seem to be simply due to the physical conditions of exertion associated with the act of bar pressing. It is true that up to the peak of the performance curve increasing heart rate was accompanied by increasing frequency of bar pressing, but past this point, heart rate continued to show rise despite the decline in exertion due to bar pressing. One might conjecture that exercise may have had greater effect on heart rate under extreme deprivation, but this would be counterbalanced—to some extent, at least—by the reduced number of presses.

To control for possible serial effects in this experiment there were two checks. First, he obtained similar findings from a second group of rats in which the order of deprivation conditions was reversed, commencing with the 72-hour deprivation condition, and finishing with the 12-hour condition. Second, the group of rats that had the ascending order of deprivation intervals were tested one week after the end of the experiment under the 60-hour deprivation condition. Mean number of responses was 96.7 and mean heart rate was 458.9 beats per minute, thus providing good agreement with the results that were obtained in the main experiment.

Finally, it is possible to speculate along various lines about how the heart rate data could be accounted for without involving the concept of activation. Obviously, further experimentation is needed, but it is encouraging nonetheless that the first animal experimentation specifically designed to explore the relation between appetitional drive and activation turned out according to prediction.

CHARACTERISTICS OF ACTIVATION

The three approaches described in the previous section appear to lead to the same fundamental concept of activation. It will, of course, be difficult to state a precise definition of activation that will satisfy everyone. Neurophysiologically oriented workers will maintain a healthy scepticism concerning the so-called "peripheral" indicants of activation. The "energetics" group while welcoming the extended use of what is essentially their own methodology will in company with some learning theorists look askance at theoretical models that verge on neurologizing. Despite differences in point of view, however, it seems worthwhile to attempt to deal with certain major characteristics of activation on which we may expect a large measure of agreement.

Activation level a product of multiple factors. When a man is deprived of sleep for some 60 hours his activation level appears higher than it was before he had suffered sleep loss. Physiological indicants reveal an upward shift in activation level that is gradual and progressive throughout the vigil (Malmö, 1958). Having once demonstrated these physiological changes it is tempting to dispense with physiological recording in further work, assuming that 60 hours of deprivation will

invariably produce a heightened state of activation. Such an assumption, however, cannot be made. An example will make clear why this assumption is untenable. A sleep-deprived *S* requires constant stimulation to prevent him from going to sleep. It is a general finding in such studies that despite the best intentions of the *S* to remain awake he will "catnap" if left alone. When he is working at a task trying to keep his efficiency from falling, the effect of major sleep loss is to produce a large increase in activation level. The important point to see here, however, is that the higher activation level is a combined product of the stimuli and their demands on him plus the condition of sleep loss. Without such stimulation, the *S* would surely fall asleep and we know from our studies of sleep that physiological levels drop very rapidly as one drifts into sleep. It is obvious, therefore, that in the absence of the task, physiological indicants at 60 hours' deprivation would show lower, not higher, activation in comparison with the rested condition.

That the "drive state" is in large part determined by environmental stimulating factors is indicated also by the observations of Bélanger and Feldman in their water deprivation experiments. Incidental observations suggested that, in addition to being more variable, heart rates recorded from the animal in a restraining compartment seemed to be consistently lower than those that were recorded when the animal was pressing the lever or drinking. In the restraining compartment the animal could view the lever through glass so that apparently mere sight of the lever was insufficient stimulation to produce the full effect upon heart rate that was produced by the acts of pressing on the lever and drinking. It thus ap-

peared that, with deprivation time approximately the same, activation level differed appreciably depending upon the conditions of external stimulation. These observations were merely incidental ones in this experiment, and they should be repeated; but they encourage the point of view that activation level is in large part a function of environmental stimulating conditions. The experiments of Campbell and Sheffield (1953) seem to point in the same direction. In the absence of sufficient environmental stimulation, food deprived rats are no more active than satiated ones, but with stimulation they are much more active than the satiated controls.

Returning to the example of the water deprived rat in the Skinner box, the two major factors determining the level of activation in that situation are (a) the internal conditions produced by deprivation and (b) the environmental stimulating conditions. To restate a point previously made, level of activation does not seem to be simply determined by the condition of deprivation alone. This would mean that depriving an animal of water per se could not produce some direct effect on motor mechanisms such as a simple discharge into the cardiac accelerating mechanism, leading to increased heart rate. Instead of some direct effect of this kind leading immediately over to some observable effector action, deprivation appears to have a sensitizing effect that is undetectable (or latent). According to this view, when appropriate stimulation does occur, the previously latent effect of deprivation will show itself in the heart rate: within limits, the longer the period of deprivation the higher the heart rate. Furthermore, according to activation theory, the same central mechanism that increases heart rate also acts to increase bombardment of the cerebral cortex.

As previously stated, this central mechanism is presumed to be the ARAS.⁴

What could be the means of sensitizing cells in the ARAS by a condition such as deprivation of water or food? If some hormone like epinephrine were released by deprivation, it is conceivable that this hormone could act to sensitize the ARAS cells in degree proportional to the amount of time that the animal had been deprived. As a matter of fact, hormonal sensitization of neural mechanisms is a currently active area of research (Saffran, Schally, & Benfey, 1955; Dell, 1958).

There are some real difficulties in defending the position that the ARAS is a unitary intensity-mediating mechanism, because the ARAS does not appear to be a homogeneous anatomical system. Indeed, as Olszewski (1954) has shown, these central brain stem structures appear very complex and highly differentiated. This unreassuring fact must not be forgotten, but neither should it be accepted as precluding the unitary function. As Lashley points out in the discussion of Olszewski's paper, structural differences are not reliable indices of function when unsupported by other evidence.

As a matter of fact, there is some important functional evidence which encourages the unitary view despite the structural complexity of the ARAS. Dell (1958) has found that: "Epinephrine does not activate selectively mammillothalamocingular systems, . . . but instead activates the ascending reticular system *en masse*, thus leading to a generalized cortical arousal" (p. 370). Control experiments showed

⁴It is very likely that the descending reticular activating system is involved here too, but, at the present stage of knowledge in this field, it does not seem wise to introduce further complications into the neuropsychological model.

that the activation effect was due to a direct action of the epinephrine at the reticular level and not to an effect on the cerebral cortex. Similar results have been obtained by Rothballer (1956).

Another kind of difficulty for the quantitative view would be posed by showing that patterned discharge from the ARAS to the cortex (not merely total quantity of discharge) was the crucial factor in supporting some behavioral action. Don't the effector patterns of standing, walking, and righting pose just such a difficulty? The relation of midbrain mechanisms to posture seems to be clearly one in which patterns of discharge from the midbrain are important. But the decorticate mammal (guinea pig, rabbit, cat, dog) in which the cortex of both hemispheres has been removed shows approximately normal postural and progressional activities (Dusser de Barenne, 1934, p. 229). Since the activation concept under review deals with bombardment of the cerebral cortex, it appears that these non-cortically mediated response patterns fall outside of phenomena under present consideration.

I should add, finally, that my admittedly speculative suggestion concerning hormonal sensitization is by no means essential to the main point which is that the behavioral evidence clearly shows the effects of deprivation to be latent (i.e. unobservable) under certain conditions. Moreover, this stress placed on the latent effects of deprivation is not mere hairsplitting. In addition to being required for an explanation of the Montréal experiments, this concept of latent deprivation effects appears to account in large measure for the findings of Campbell and Sheffield (1953), and more generally for the failure of random activity

to adequately serve as a measure of drive or activation (Malmö, 1958).

Activation and the S-R framework. As the product of interaction between internal (perhaps hormonal) conditions and external stimulating ones, activation cannot be very reasonably classified as either stimulus or response. This means that the physiological measurements that are used to gauge level of activation do not fit very well into the S-R formula. It is perhaps useful to think of these physiological conditions as part of O in the S-O-R formula (Woodworth & Schlosberg, 1954, p. 2).

The momentary physiological reaction to a discrete stimulus like the sudden rise in palmar conductance accompanying pin-prick is not of primary concern to us in our study of activation. This kind of S-R reaction, important as it undoubtedly is for investigating other problems, is of little relevance for the study of activation, compared with the longer lasting changes. As Schlosberg has put it to me in personal communication, in employing skin conductance to gauge level of activation, one observes the "tides" and not the "ripples." I do not mean to disparage studies that use physiological reactions as R terms in the strict S-R sense. It is just that in this paper I am concerned with physiological functions only insofar as they are related to activation.

It may be queried whether we are dealing with a needless and hairsplitting distinction by saying that activation is not a response. However, the kind of difference I have in mind appears quite distinct and useful to keep in mind, though it should not be stressed unduly. Basically, it is the same distinction which Woodworth and Schlosberg (1956) make when they draw particular attention to the dif-

ference between slow and rapid changes in skin conductance. As examples of rapid changes in skin conductance, there are the "GSRs" as R terms in conditioned responses, and in free association tests. Examples of slow skin conductance changes, on the other hand, are the gradual downward drifts that occur over hours during sleep (see Fig. 4), the slow downward changes in skin conductance in Ss as they become gradually habituated to an experimental situation (Davis, 1934; Duffy & Lacey, 1946), and (going up the activation scale) the progressive upward changes in conductance during a vigil (Malmo, 1958).

I would not deny that there are stimuli and responses going on in the physiological systems, but at the present time I see no way of identifying and handling them. It should be added, however, that this does not give one license to completely disregard the antecedents of physiological changes. For instance, if the hand of a sleeping S becomes hot by being covered with heavy bedclothing the local thermal sweating induced thereby will bring about a sudden rise in palmar conductance which has nothing to do with activation. Or sleep may be induced by certain drugs which have a specific stimulating effect on respiration, such that respiration rate will not fall during sleep as it usually does (see Fig. 5 for curve obtained under nondrug conditions). Furthermore, artifacts due to movement and postural shifts may prevent muscle potentials from serving as reliable indicants of activation level.

Limitations of the activation concept. I am not attempting to solve the problem of selection, i.e., the problem of finding the neurophysiological mechanisms that determine which cues in the animal's environment are prepotent in the sense of winning out over other cues in triggering off a pattern of ef-

factor action. This point seems clear enough, especially when it is stressed that activation has no steering function; and yet there is still the risk that some critics may misunderstand and state as one shortcoming of this theory that it does not adequately handle the problem of selection. The theory may be open to criticism on the grounds that it is limited, but it should not be criticized for failing to do something which it was not intended to do.

It will be noted that in general an attempt is made to raise theoretical questions that stand a good chance of being answered by available experimental techniques. Schematically, the experimental paradigm is as follows:

Activation

level : Low Moderate High
Expected performance level : Low Optimal Low

It is important to stress that the measure denoted by "moderate activation level" has meaning only in relative (not in absolute) terms. That is, the level is "moderate" because it is higher than that of the low activation condition, and lower than the level of the high activation condition. Comparisons are invariably of the within-individual, within-task kind, which means that the level of activation which is found to be optimal for one task is not directly compared with the level of activation which is found to be optimal for a different task. Thus, at the present stage of theorizing, no attempt is made to deal with the question of whether tasks which differ in complexity, for example, also differ with respect to the precise level of activation which is optimal for each one. However, I have dealt elsewhere (Malmo, 1958) with the related question of response competition, suggesting an alternative to the response competition explana-

tion for decrement in performance with increased activation (or D).

Again, the theoretical formulations may be criticized for being too narrow. But it must be kept in mind that their narrowness is due to the close nexus between theory and experiment in this program. These formulations may also be criticized for an unjustifiable assumption in the postulation of a communal drive mechanism. One may well ask where the evidence is that proves the existence of a state of general drive. In dealing with this kind of question, it is essential to refer back to the outline of the experimental paradigm. The experimental induction of the three discriminable activation levels referred to in the outline depends upon the controlled variation of certain conditions in the S's environment. The fact that by varying conditions as dissimilar as appetitional deprivations and verbal incentives it is possible to produce similar shifts in physiological indicants provides a sound basis for introducing the operationally defined concept of activation level that cuts across traditional demarcation lines of specific drives. All this, of course, does not constitute final proof for a communal drive mechanism. Certainly further data are required before it is even safe to conclude equivalence of drive conditions in the alteration of physiological levels, to say nothing of proving the existence of a communal drive mechanism.

INTERRELATIONS BETWEEN PHYSIOLOGICAL INDICANTS OF ACTIVATION

Criticism directed against physiological measures as indicants of activation usually involves one or both of the following points. The first objection is that intercorrelations between physiological measures are so low that it is unreasonable to consider their use

for gauging a single dimension of behavior. A second objection is that activation properly refers to events in the brain and that the correspondence between these central events and what may be observed in such peripheral functions as heart rate, respiration, muscle tension and the like is not close enough to permit valid inferences from the peripheral events to the central ones. In the following section, I shall attempt to answer these criticisms.

Intra- and interindividual correlations among physiological indicants of activation. In an unpublished paper, Schnore and I have discussed certain misconceptions that have confused some critics of physiological methods. The most serious misunderstanding concerns correlations among physiological measures. It is true that *interindividual* correlations are low, but this fact is actually irrelevant insofar as using these measures to gauge activation is concerned. The important question is whether significant *intra-individual* correlations are found in a sufficiently high proportion of individuals, and the answer appears to be yes (Schnore, 1959).

What the low *interindividual* correlations mean, of course, is that an individual in any given situation may have a heart rate that is high relative to the mean heart rate for the group, and at the same time have a respiration rate or a blood pressure that is low relative to the group mean. These findings are in line with the principle of physiological specificity that is now supported by several lines of evidence.⁵

⁵ The general principle of physiological specificity states that under significantly different conditions of stimulation individuals exhibit idiosyncratic but highly stereotyped patterns of autonomic and somatic activation. I use the term *physiological specificity* as a generic reference to autonomic-response stereotypy (Lacey & Lacey, 1958) to symptom specificity (Malmo & Shagass,

Physiological specificity is a separate problem that is in no way crucial for the activation hypothesis. An illustration will make this clear. Take a rather extreme example of an individual with very *high* heart rate (say 95 when the mean for his group under specified conditions is 75) and very *low* palmar conductance (50 micromhos when the group mean is 100). In an experiment with varied incentive, in going from a low incentive to a high incentive condition this *S* will likely show an increase in heart rate from 95 to say 110 and an increase in palmar conductance from 50 to say 60 micromhos. The main point is that even though the *S*'s heart rate is already high compared with the mean for his group, it goes still higher (concordantly with palmar conductance) when the stimulating situation increases the level of activation. This is the kind of intraindividual correlation between physiological measures⁶ that is required for gauging the dimension of activation and, to repeat, the evidence strongly indicates that the intraindividual correlations are sufficiently high for this purpose.

RELATIONS BETWEEN CENTRAL AND PERIPHERAL INDICANTS OF ACTIVATION

As previously noted, the pioneer EEG workers observed definite changes in EEG pattern accompanying major shifts in the conscious state of the *S*. Moreover, they recognized a continuum

1949), and to stereotypy of somatic and autonomic activation patterns (Schnore, 1959).

⁶It is not claimed, however, that all physiological measures are equally useful for the purpose of gauging activation level. On the contrary, as Schnore's experiments have suggested, some measures appear superior to others, and eventually we may be able to select the most discriminating ones and thus improve our measurement (Schnore, 1959).

of increasing activation usually referred to as the sleep-waking-excitement continuum, just as other workers like Freeman (1948) and Duffy (1957) employing peripheral measures of palmar sweating and muscular tension recognized it. Among the early workers in this field, Darrow (1947) studied EEG and other measures simultaneously, but only very recently have techniques been made available that can provide the kind of quantitative EEG measurements required for critical comparisons along the activation continuum. That is, from simple inspection of the raw EEG tracing it is possible to see gross differences between sleeping and waking, or between a drowsy, relaxed state and one of extreme alertness. But for experiments on activation it is necessary to have an instrument that will reveal measureable differences for "points" lying closer to each other on the activation continuum. For example, it is essential to have a measure that will discriminate reliably between a moderately alert and a highly alert state. For such discriminations the method of inspection will not do, and a device for objective quantification of the wave forms is required.

Because of its complexity the EEG tracing has been difficult to quantify, and although gross differences in activation level could be detected by simple inspection of the tracing, this method was too crude for more detailed work. However, with the advent of EEG frequency analysers, quantification of the EEG looked promising because these analysers were designed to provide quantified EEG data for each of many different narrow frequency bands. Unfortunately, these instruments have not proved useful because of insufficient stability. In our laboratory we have been trying band-pass filters to provide stable quantification of various selected

frequency bands in which we are primarily interested (Ross & Davis, 1958). Results thus far appear highly encouraging.

Data indicating relationships between EEG and other physiological functions. In a recent sleep deprivation experiment, we found that palmar conductance and respiration showed progressive rise during the vigil, indicating increasing activation with deprivation of sleep. In the same experiment we recorded EEG and, by means of a band-pass filter, obtained a quantified write-out of frequencies from 8-12 per second, in the alpha range. It will be recalled that the classical picture of activation is reduction in the amount of alpha activity. Therefore, what we might expect to find in this experiment is progressive decrease in the amount of alpha activity. As a matter of fact, this is exactly what was found (Malmö, 1958, p. 237).

As Stennett (1957b) has shown, however, the relationship between EEG alpha activity and other physiological variables is sometimes curvilinear. In the sleep deprivation experiments physiological measurements were taken under highly activating conditions and at this high end of the continuum further increase in activation seems invariably to decrease the amount of alpha activity. But at the lower end of the continuum with the *S* in a drowsy state, increased activation has the opposite effect on alpha activity. An alerting stimulus, instead of producing a flattening of the EEG tracing, will actually produce an augmentation of the alpha activity. This has sometimes been referred to as a "paradoxical" reaction, although it seems paradoxical only when it is assumed that the relation between activation level and alpha amplitude is a decreasing monotonic one throughout the entire activation continuum. But Sten-

nett (1957b) has shown that the relationship is not monotonic. From his data he plotted a curve which has the shape of an inverted *U*. From this curve it would be predicted that with a drowsy *S*, stimulation should increase alpha amplitude. From the same inverted *U* curve it would also be predicted that an *S* whose activation level was sufficiently high (past the peak of the curve) before stimulation would show a decrease in alpha amplitude. Actually, some unpublished experiments on startle by Bartoshuk fit these predictions very well.

Recent data indicate the usefulness of a 2-4 c.p.s. band-pass filter in experiments on sleep. The data in the figures that follow represent mean values from three men who slept all night in our laboratory after serving as *S*s in our sleep deprivation experiments.

Bipolar sponge electrodes, soaked in electrode jelly and attached to the *S* by Lastonet bands, were used for the parietal EEG placement (two thirds of the distance from nasion toinion, and 3 cm. from the midline on each side). The primary tracing was recorded by an Edin Electroencephalograph, and the two secondary tracings were integrations of the EEG potentials that were passed through band-pass filters for selective amplification of signals in the 2-4 and 8-12 c.p.s. frequency bands. Measurements on the secondary tracings were carried out with special rulers, and these measurements were converted to microvolt values by reference to calibration standards.

Method of recording and measuring palmar conductance was similar to that described by Stennett (1957a).

Electrocardiograms were picked up from electrodes placed on contralateral limbs, and heart rates were determined from measurements of electrocardiota-

chometric tracings. Respiration rates were obtained by means of a Phipps and Bird pneumograph.

All three Ss slept well throughout the night (approximately from 10 P.M. to 9 A.M. after some 60 hours without sleep). Physiological recordings were carried out continuously during the whole period of sleep in each case, and except for occasional attention to electrodes (e.g. application of electrode jelly and saline to electrodes) the Ss were undisturbed.

Four pairs of cellulose sponge electrodes were attached to the four limbs (to the pronator teres muscles of the arms and the peroneal muscles of the legs) for the purpose of recording muscle potentials. Primary muscle-potential tracings were recorded on the chart of a custom built Edin electromyograph (EMG). Electronic integrators (employing the condenser charge-discharge principle, like those used for the secondary EEG tracings), attached in parallel across the galvanometers of this EMG unit, integrated

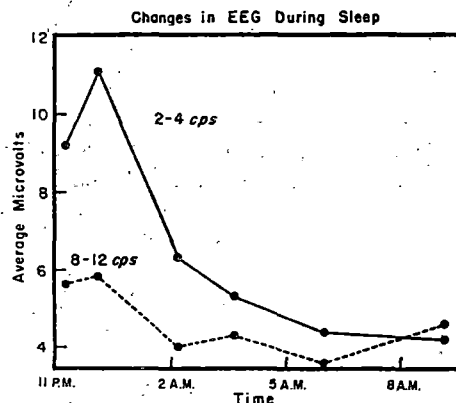


FIG. 3. Mean EEG values from three healthy young male Ss during a night's sleep. Subjects had been sleep-deprived. Band-pass filters were used in connection with electronic integrators to provide quantitative data in the two different frequency bands.

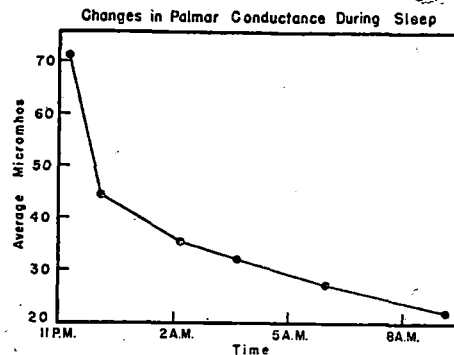


FIG. 4. Mean palmar conductance values from the same Ss, at the same times during sleep as in Fig. 3.

the muscle potentials over successive 4-second periods.

These muscle-potential tracings were used to record movements and periods of restlessness during sleep. Five-minute periods free from muscle-potential activity and preceded by at least 5 minutes of movement-free tracings were chosen for measurement in order to provide the values plotted in Fig. 3-5. The actual times plotted on the baseline represent the medians for the three Ss. In each instance the three times were close to one another.

In Fig. 3 observe that following a brief rise early in sleep the upper curve for 2-4 c.p.s. falls continuously during the entire period of sleep. This curve is consistent with published accounts of changes in EEG during sleep noted by inspection of the raw tracings (Lindsley, 1957, p. 68). Early in sleep there is an increase in slow waves around 2-4 cycles per second, but as sleep continues these waves are replaced by even slower ones. As far as I am aware, the data in Fig. 3 represent the first use of a 2-4 band-pass filter to quantify the EEG. The curve for 8-12 c.p.s. EEG also shows some fall, and the voltage is low in accordance with the well-known dis-

appearance of alpha waves from the raw tracings during sleep.

Figures 4 and 5 show data for palmar conductance, heart rate, and respiration, that were recorded at the same time as the EEG data. From the second plotted point on, there is rather close resemblance between these curves and the one for 2-4 c.p.s. EEG. It seems likely that a band-pass filter for fast frequencies in the beta range might yield a continuously falling curve commencing with drowsiness and continuing through the onset and early stages of sleep. There are serious technical difficulties in quantifying the next step of frequencies above the alpha band, but we are hopeful that a band-pass filter that has recently been constructed in our laboratory will overcome these difficulties.

Direct alteration of ARAS activity by means of electrical stimulation and related animal experimentation. The most relevant experiment on direct stimulation of the ARAS is, as far as I know, the one by Fuster (1958) that was mentioned earlier. By stimulating in the same part of the ARAS that produces the EEG picture of activation, Fuster was able to produce improved discrimination performance in the monkey. Presumably, this effect

was achieved by causing a larger number of impulses from the ARAS to bombard the cortex. The assumption would be that before the onset of electrical stimulation the cortex was not receiving sufficient bombardment for optimal performance (Hebb, 1955) and that ARAS stimulation brought total bombardment in the cortex closer to the optimal value. The situation may not be as simple as this, but the success of the Fuster experiment encourages further experimentation along these same lines. Finding that level of performance can be altered by electrical stimulation of the ARAS opens up the exciting possibility that if amount of neural activity in the ARAS can be measured, we might find a direct correlation between a central measure of activation and level of performance. For instance, the Bélanger and Feldman experiment described earlier might be repeated with the addition of recordings from the ARAS. The aim of such an experiment would be to determine whether the continuous rise in the heart rate curve with increasing deprivation times could be matched by a similar rise in amplitude of deflections from recording in the ARAS with implanted electrodes. Recent neurophysiological experiments appear encouraging with respect to the feasibility of such an approach (Li & Jasper, 1953, pp. 124-125; Magoun, 1958, p. 68).

EFFECTS OF INCREASED ACTIVATION ON LOCALIZED SKELETAL-MUSCLE TENSION IN PSYCHIATRIC PATIENTS

The implication of activation theory for various clinical phenomena might very well be the topic of a separate paper. Certainly there is not space to deal at length with the topic here. I have chosen, therefore, to present a

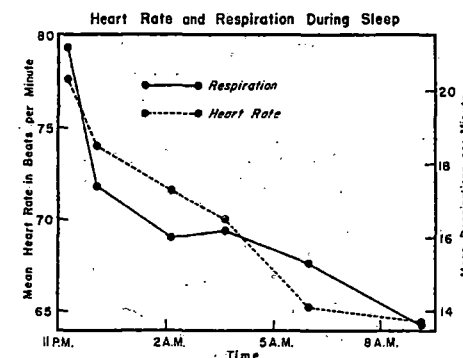


FIG. 5. Mean values for heart rate and respiration from the same Ss at the same times during sleep as in Fig. 3 and 4.

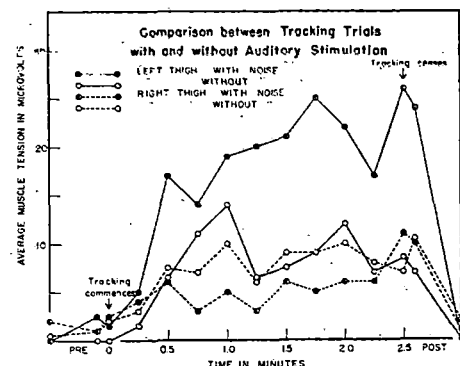


FIG. 6. Mean muscle tension from left thigh and right thigh from patient with complaint of tensional discomfort in the left thigh. Note that when patient was performing the tracking task under distraction (loud noise), tension rose in the left thigh but not in the right. See text for explanation.

few recent observations, chiefly in order to suggest how level of activation may be studied in relation to a clinical phenomenon.

The graph in Fig. 6 illustrates what appears to be a general finding in patients complaining of tensional discomfort in a localized muscular site. The data for the curves plotted in the figure were obtained from a psychiatric patient, a 42-year old woman who complained of muscular discomfort localized in the left thigh. In the session when these data were taken electromyograms (EMGs) were recorded from various muscles over the body; those from the left and right thighs are shown in the figure. The patient was engaged in pursuit tracking using an apparatus similar to the one employed by Surwillo (1955, 1956). Figure 6 shows that when a loud distracting noise, of the kind described by Schnore (1959), was presented during tracking, the tension in the left thigh was very much higher than that of the right thigh. When tracking was carried out under distraction free con-

ditions this tensional difference between thighs was not observed.

Interpretation of these data seems quite straightforward. When level of activation was increased by presenting a loud distracting noise the effect was shown entirely in one muscle group, the left thigh, which was the symptom area in this patient. Simultaneous recordings of tension from other parts of the body showed that the tension was specific to the left thigh and was not merely increased on the whole left side of the body.

The specificity of the left thigh in indicating the higher activation is quite clear. Observe that tension in the thigh muscles on the opposite side of the body actually fell slightly under the activating condition.

The same procedure was carried out with a second patient, a young girl of 28, who complained of a distressing feeling of tightness in the neck on the right side. Results were similar to the ones obtained in the previous case, with activation again showing its effect specifically in the symptom area. When the loud distracting noise was turned on during tracking, tension in this area showed marked increase whereas tension in the muscles on the left side of the neck showed no rise whatever.

Very similar results were obtained from two additional patients whose areas of tensional discomfort were localized in still different parts of the body. One woman with complaint of tension on the left side of her neck served as a useful control for the patient previously described with tension localized in the opposite side of the neck. No tracking experiment was carried out with this patient. Apparently the sight of the EMG recording room for the first time was itself sufficient to increase the amplitude of muscle potentials from the symptom area so that

it become appreciably higher than those on the opposite side of her neck. The other woman (fourth patient in this series) complained of tensional discomfort that appeared to originate in the left shoulder. EMGs were recorded from the left and right shoulders of this patient while she lay in bed listening to the playback of a recorded interview. During the first part of the playback, tension was about the same on the two sides of the body. But when the topic concerning her dead sister commenced to come over the speaker, tension in the left shoulder became much greater than that in the right.

As far as could be determined, the EMG data from all these patients were consistent in suggesting that for skeletal-muscle tension in patients with well-developed tensional symptoms, increasing the activation level up to a certain point has the effect of raising muscle tension in one localized muscle group, the one in which the patient complained of tensional discomfort. It was not necessary for the patient to actually feel the discomfort during the experimental session for this differential result to appear. I have been using the term "symptom area" to refer to the muscle group where the discomfort was localized when present.

Interesting findings that appear to parallel those from the patients were obtained from three young male non-patient Ss in our recent investigation of sleep deprivation. As previously mentioned, evidence from EEG, palmar conductance, and respiration indicated that activation during tracking increased progressively with hours of sleep deprivation. In addition to these other physiological tracings, EMGs from various areas over the body were also recorded. One muscle area, a different one for each S, showed significant rise in tension over the vigil. It

was the neck muscles in one S, the forehead in another, and the biceps muscle of the right arm in the third. In each case the one muscle showed statistically significant rise in tension, and in none of the Ss was there significant tensional rise in any other muscle. In fact, there was regularly progressive and very significant fall in the tension of the left forearm in all three Ss. As far as I know, none of the men actually complained of tensional discomfort in the areas showing rise in tension during the vigil.

Where high level activation is long continued as in a vigil or in certain psychoneurotic patients, it appears that skeletal tension may become localized to a single muscle group. The discomfort associated with this tension in some patients can become extremely severe. It should be noted that in one-session experiments, where rise in activation was for relatively short intervals of time, tensional rise occurred in more than one muscle group (Surwillo, 1956; Stennett, 1957a).

Methodologically, these results are important because they reveal a difference between EMGs and some other physiological measures with respect to gauging activation. Unlike heart rate or respiration rate that invariably yields one measure no matter how it is recorded, there are as many measures of muscle tension as there are muscles that can be recorded from. It appears that when sufficient care is taken, EMGs may be very valuable in helping to gauge activation, but that considerable caution is required in the interpretation of results, and especially in the interpretation of negative results.

From the clinical point of view it seems an interesting speculation that the patient's localized muscle tension may itself actually increase the general activation level. (I do not mean

the level of muscle tension all over the body.) Two main assumptions are involved in this suggestion. The first one is that the area of localized muscle tension in the patient acts like tension that is induced, for example, by having an S squeeze on a dynamometer. From the generalized effects of tension induction on learning and performance it is clear that the effects of increased muscle tension are quite general ones. Though crucial physiological data are missing in these experiments, as previously mentioned, one very likely explanation of these results is that the local increase in muscle tension somehow produces an increase in the general level of activation, with rise in heart rate and blood pressure, with fall in level of EEG alpha, and so on. This is the second assumption. The results of two recent experiments are in line with this assumption. Meyer and Noble (1958) found that induced tension interacted with "anxiety" in verbal-maze learning ("anxiety" measured by means of the MAS [Taylor, 1953]), while Kuethe and Eriksen (1957) in a study of stereotypy likewise reported a significant interaction between these two variables when "anxiety" was experimentally produced by means of electric shocks. The MAS appears to select individuals who are significantly above the mean in activation, and from the results of Schnore (1959) and Feldman (1958) it seems safe to conclude that anticipation of shock also leads to increased levels of physiological activity. In short, generalizing from the induced tension experiments, it seems reasonable to suppose that a patient's muscular tension in a small focal area might have the general effect of increasing activation. If such is the case symptomatic treatment might have significant general as well as specific effects. Although based on only one patient, Yates' (1958)

results from symptomatic treatment of tics seems encouraging with respect to the feasibility of research in this general area.

SUMMARY

The neuropsychological dimension of activation may be briefly described as follows. The continuum extending from deep sleep at the low activation end to "excited" states at the high activation end is a function of the amount of cortical bombardment by the ARAS, such that the greater the cortical bombardment the higher the activation. The shape of the curve relating level of performance to level of activation is that of an inverted U: from low activation up to a point that is optimal for a given performance or function, level of performance rises monotonically with increasing activation level; but past this optimal point the relation becomes nonmonotonic: further increase in activation beyond this point produces fall in performance level, this fall being directly related to the amount of the increase in level of activation.

Long before the discovery of the ARAS the behavioral evidence of Duffy, Freeman, and others of the "energetics" group had suggested the existence of some such brain mechanism. Moreover, learning theorists of the Hull school have in their concept of the general drive state come very close to the activation principle. Up to the present time they have employed physiological measures only sparingly and have restricted their use to the aversive aspects of drive. But with evidence that such measures may also be applied to nonaversive (appetitional) drive, it seems likely that the present rather unsatisfactory measures of drive may eventually be replaced by physiological indicants.

Activation has a number of main

characteristics that may be listed as follows: (a) Activation has no steering function in behavior. (b) It is considerably broader than emotion. (c) Activation is not a state that can be inferred from knowledge of antecedent conditions alone, because it is the product of an interaction between internal conditions such as hunger or thirst, and external cues. (d) Activation does not fit very well into the S-R formula. It is a phenomenon of slow changes, of drifts in level with a time order of minutes (even hours) not of seconds or fractions thereof. (e) Activation is a quantifiable dimension and the evidence indicates that physiological measures show a sufficiently high intraindividual concordance for quantifying this dimension.

It is suggested that activation is mediated chiefly through the ARAS which seems, in the main, to be an intensity system. Neurophysiological findings strongly suggest that it may be possible to achieve more precise measurement of activation through a direct recording of discharge by the ARAS into the cerebral cortex. Research on this problem is urgently needed.

The concept of activation appears to have wide application to phenomena in the field of clinical psychology. As one illustration, in this paper, activation was applied to clinical phenomena of tensional symptoms.

REFERENCES

- BINDRA, D. *Motivation. A systematic re-interpretation.* New York: Ronald, 1959.
- BROWN, J. S. Pleasure-seeking behavior and the drive-reduction hypothesis. *Psychol. Rev.*, 1955, 62, 169-179.
- CAMPBELL, B. A., & SHEFFIELD, F. D. Relation of random activity to food deprivation. *J. comp. physiol. Psychol.*, 1953, 46, 320-326.
- COPER, C. N. Motivation. *Annu. Rev. Psychol.*, 1959, 10, 173-202.
- COURTS, F. A. Relations between muscular tension and performance. *Psychol. Bull.*, 1942, 39, 347-367.
- DARROW, C. W. Psychological and psychophysiological significance of the electroencephalogram. *Psychol. Rev.*, 1947, 54, 157-168.
- DAVIS, R. C. Modification of the galvanic reflex by daily repetition of a stimulus. *J. exp. Psychol.*, 1934, 17, 504-535.
- DELL, P. C. Humoral effects on the brain stem reticular formations. In H. H. Jasper, L. D. Proctor, R. S. Knighton, W. C. Noshay, & R. T. Costello (Eds.), *Reticular formation of the brain.* Toronto: Little, Brown, 1958. Pp. 365-379.
- DUFFY, ELIZABETH. The measurement of muscular tension as a technique for the study of emotional tendencies. *Amer. J. Psychol.*, 1932, 44, 146-162.
- DUFFY, ELIZABETH. The concept of energy mobilization. *Psychol. Rev.*, 1951, 58, 30-40.
- DUFFY, ELIZABETH. The psychological significance of the concept of "arousal" or "activation." *Psychol. Rev.*, 1957, 64, 265-275.
- DUFFY, ELIZABETH, & LACEY, O. L. Adaptation in energy mobilization: changes in general level of palmar skin conductance. *J. exp. Psychol.*, 1946, 36, 437-452.
- DUSSER DE BARENNE, J. G. The labyrinthine and postural mechanisms. In C. Murchison (Ed.), *A handbook of general experimental psychology.* Worcester, Mass.: Clark Univer. Press, 1934. Pp. 204-246.
- FELDMAN, S. M. Differential effect of shock as a function of intensity and cue factors in maze learning. Unpublished doctoral dissertation, McGill Univer., 1958.
- FINAN, J. L. Quantitative studies of motivation. I. Strength of conditioning in rats under varying degrees of hunger. *J. comp. Psychol.*, 1940, 29, 119-134.
- FINCH, G. Hunger as a determinant of conditional and unconditional salivary response magnitude. *Amer. J. Physiol.*, 1938, 123, 379-382.
- FREEMAN, G. L. The relationship between performance level and bodily activity level. *J. exp. Psychol.*, 1940, 26, 602-608.
- FREEMAN, G. L. *The energetics of human behavior.* Ithaca, N. Y.: Cornell Univer. Press, 1948.
- FUSTER, J. M. Effects of stimulation of brain stem on tachistoscopic perception. *Science*, 1958, 127, 150.
- HEBB, D. O. *The organization of behavior.* New York: Wiley, 1949.

- HEBB, D. O. Drives and the C.N.S. (conceptual nervous system). *Psychol. Rev.*, 1955, 62, 243-254.
- JASPER, H. H. Electroencephalography. In W. Penfield & T. C. Erickson (Eds.), *Epilepsy and cerebral localization*. Springfield, Ill.: Charles C Thomas, 1941, 380-454.
- KAPLAN, M. The effects of noxious stimulus intensity and duration during intermittent reinforcement of escape behavior. *J. comp. physiol. Psychol.*, 1952, 45, 538-549.
- KENDLER, H. H. Learning. *Annu. Rev. Psychol.*, 1959, 10, 43-88.
- KUETHE, J. L., & ERIKSEN, C. W. Personality, anxiety, and muscle tension as determinants of response stereotypy. *J. abnorm. soc. Psychol.*, 1957, 54, 400-404.
- LACEY, J. I., & LACEY, BEATRICE C. Verification and extension of the principle of autonomic response-stereotypy. *Amer. J. Psychol.*, 1958, 71, 50-73.
- LI, C. L., & JASPER, H. H. Microelectrode studies of the electrical activity of the cerebral cortex in the cat. *J. Physiol.*, 1953, 121, 117-140.
- LINDSLEY, D. B. Emotion. In S. S. Stevens (Ed.), *Handbook of experimental psychology*. New York: Wiley, 1951. Pp. 473-516.
- LINDSLEY, D. B. Psychophysiology and motivation. In M. R. Jones (Ed.), *Nebraska symposium on motivation 1957*. Lincoln: Univer. Nebr. Press, 1957. Pp. 44-105.
- LORENTE DE NÓ, R. Transmission of impulses through cranial motor nuclei. *J. Neurophysiol.*, 1939, 2, 402-464.
- MAGOUN, H. W. *The waking brain*. Springfield, Ill.: Charles C Thomas, 1958.
- MALMO, R. B. Measurement of drive: An unsolved problem in psychology. In M. R. Jones (Ed.), *Nebraska symposium on motivation 1958*. Lincoln: Univer. Nebr. Press, 1958, 229-265.
- MALMO, R. B., & SHAGASS, C. Physiologic study of symptom mechanisms in psychiatric patients under stress. *Psychosom. Med.*, 1949, 11, 25-29.
- MEYER, D. R., & NOBLE, M. E. Summation of manifest anxiety and muscular tension. *J. exp. Psychol.*, 1958, 55, 599-602.
- OLSZEWski, J. The cytoarchitecture of the human reticular formation. In J. F. Delafresnaye (Ed.), *Brain mechanisms and consciousness*. Springfield, Ill.: Charles C Thomas, 1954. Pp. 54-76.
- ROSS, W. R. D., & DAVIS, J. F. Stable band-pass filters for electroencephalography. *IRE Canad. Convention Rec.* 1958, Paper No. 860, 202-206.
- ROTHBALLER, A. B. Studies on the adrenaline-sensitive component of the reticular activating system. *EEG Clin. Neurophysiol.*, 1956, 8, 603-621.
- SAFFRAN, M., SCHALLY, A. V., & BENFEY, B. G. Stimulation of the release of corticotropin from the adenohypophysis by a neurohypophysial factor. *Endocrinology*, 1955, 57, 439-444.
- SCHNORE, M. M. Individual patterns of physiological activity as a function of task differences and degree of arousal. *J. exp. Psychol.*, 1959, 58, 117-128.
- SPENCE, K. W. Theory of emotionally based drive (D) and its relation to performance in simple learning situations. *Amer. Psychologist*, 1958, 13, 131-141.
- STENNETT, R. G. The relationship of performance level to level of arousal. *J. exp. Psychol.*, 1957, 54, 54-61. (a)
- STENNETT, R. G. The relationship of alpha amplitude to the level of palmar conductance. *EEG Clin. Neurophysiol.*, 1957, 9, 131-138. (b)
- SURWILLO, W. W. A device for recording variations in pressure of grip during tracking. *Amer. J. Psychol.*, 1955, 68, 669-670.
- SURWILLO, W. W. Psychological factors in muscle-action potentials: EMG gradients. *J. exp. Psychol.*, 1956, 52, 263-272.
- TAYLOR, JANET A. A personality scale of manifest anxiety. *J. abnorm. soc. Psychol.*, 1953, 48, 285-290.
- WOODWORTH, R. S., & SCHLOSBERG, H. *Experimental psychology*. New York: Holt, 1954.
- YATES, A. J. The application of learning theory to the treatment of tics. *J. abnorm. soc. Psychol.*, 1958, 56, 175-182.

(Received May 4, 1959)

ANXIETY AND BEHAVIORAL AROUSAL¹

ROBERT B. MALMO²

Allan Memorial Institute of Psychiatry, McGill University

Reprinted from the Journal of Psychological Review
Vol. 64, No. 5, 1957

ANXIETY AND BEHAVIORAL AROUSAL¹

ROBERT B. MALMO²

Allan Memorial Institute of Psychiatry, McGill University

During the past two decades there has been a growing interest in objective physiological studies of psychiatric patients. In this work, one of the most prominent psychological concepts has been that of anxiety. Although there is general agreement that the areas denoted by the term "anxiety" are important ones for study, there is nonetheless considerable disagreement concerning what the term means. In large measure, this semantic difficulty is part of a larger problem facing psychology today, and that is to find a way out of the confusion surrounding the concepts of motivation and emotion. Duffy has cogently argued that these concepts are second-order ones which reduce to primary factors of intensity and direction, and that along the intensity dimension, at least, the distinction between motivation and emotion is unnecessary (9, 10, 11).³

This is not to say that the directional aspect is not important or to deny that,

in terms of direction, meaningful distinctions may be made between motivation and emotion, and indeed between different emotions. However, for present purposes it is essential to focus on the question of what these phenomena have in common rather than to consider their differences; in this paper, therefore, we shall be primarily concerned with the intensity dimension.

The main purpose of the present paper is to consider recent experimental data in an attempt to find a way out of the present confusion. I shall begin with a summary of two lines of investigation in our laboratory, dealing first with our discovery that certain physiological measures may serve as indicants of intensity or "behavioral arousal." These experiments were performed with nonpatient subjects. Second, in summarizing our investigations of pathological anxiety in psychiatric patients, I shall attempt to use the concept of behavioral arousal in an integrative way. Third, I shall draw on data from recent neurophysiological investigations to indicate possible mechanisms involved in the pathology and etiology of anxiety. Finally, on the basis of these theoretical considerations, I suggest problems requiring further experimental study.

PHYSIOLOGICAL INDICANTS OF BEHAVIORAL INTENSITY

In 1951 we (31) reported finding a gradient phenomenon from electromyographic (EMG) recording during mirror tracing. Since that time the phenomenon has been observed under various conditions in our laboratory. Figure 1 presents mirror-drawing data from a study by Bartoshuk (1). Note that the

¹ This paper reviews work which was supported by the Medical Research and Development Division, Office of the Surgeon General, Department of the U. S. Army, under Contract Number DA 49-007-MD-626, by Defence Research Board Grant Number 9425-04 (Canada), and by Grant Number A.P. 29 from the National Research Council of Canada.

² The author is indebted to Drs. A. K. Bartoshuk, D. Bindra, F. R. Brush, D. E. Cameron, D. O. Hebb, and R. G. Stennett for criticizing earlier drafts of this paper.

³ I do not wish to imply that this has been Duffy's only theoretical contribution. Her writings contain prior reference to a dimension of behavioral intensity (conceived as a continuum of "arousal," or "activation"); and she has previously cited evidence to support the argument that physiological measures may serve as the chief means of quantifying such a dimension or continuum.

ANXIETY AND BEHAVIORAL AROUSAL

chin lead (which taps the speech muscles) also shows a gradient—that is, progressively rising muscle potentials from the beginning to the end of the task. Bélanger (3) found similar gradients from the arm in a size-discrimination task. Wallerstein (42) found gradients in the frontalis muscle in a task about as completely devoid of motor components as one could possibly design. The subject, reclining on a comfortable bed, listened to verbal material (short detective story or essay) presented to him by a tape recorder. In Wallerstein's experiment, the gradients extended over ten minutes and their steepness was related to the subject's reported degree of interest in listening (2, p. 228 f.).

Bartoshuk (2) was the first to show that the fastest and most accurate subjects (i.e., superior performers on mirror tracing) produced the steepest muscle-potential gradients. Such a relationship of EMG gradients to motivation has

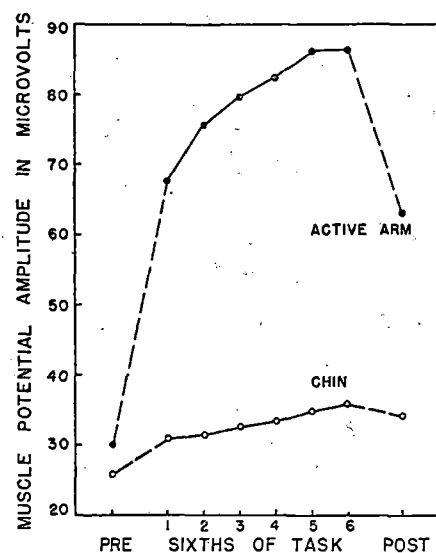


FIG. 1. Graphs showing mean EMG gradients in Bartoshuk's experiment (1). Note that gradient was also obtained from chin lead which records from muscles of speech. $N = 17$.

been confirmed by three subsequent studies, employing tracking tasks. Surwillo (39) demonstrated that raising incentive had the effect of increasing the steepness of EMG gradients in a visual tracking experiment. Figure 2 presents confirmatory data from a more recent experiment by Stennett (37) who employed auditory tracking under four conditions, with increasing degrees of incentive. Note that the muscle potentials were recorded from the nonactive, left arm. His "exertion" condition merely involved the subject's holding the tracking knob over at a fixed point in order to control for sheer physical work. Under the "calibration" condition the subject believed that he was just assisting with calibration of the apparatus, and that his tracking scores were not being recorded. The "optimal" condition was designed to motivate the subject sufficiently to elicit his most efficient performance, whereas the "incentive" condition was designed to "overmotivate" the subject by offering large bonuses for high-level performance and threatening with strong electric shock if performance did not reach this high level. The differences shown in the figure were statistically significant. In brief, Stennett's findings indicated that the most efficient tracking performance was associated with intermediate physiological levels (i.e., intermediate steepness of EMG gradients and intermediate levels of palmar skin conductance). With lower levels of physiological functioning (less steep gradients, lower levels of palmar skin conductance), performance on tracking was inferior. However, going now to the other extreme, performance on tracking associated with extremely high EMG gradients and extremely high palmar skin conductance was also inferior to tracking performance associated with moderately high levels of physiological functioning.

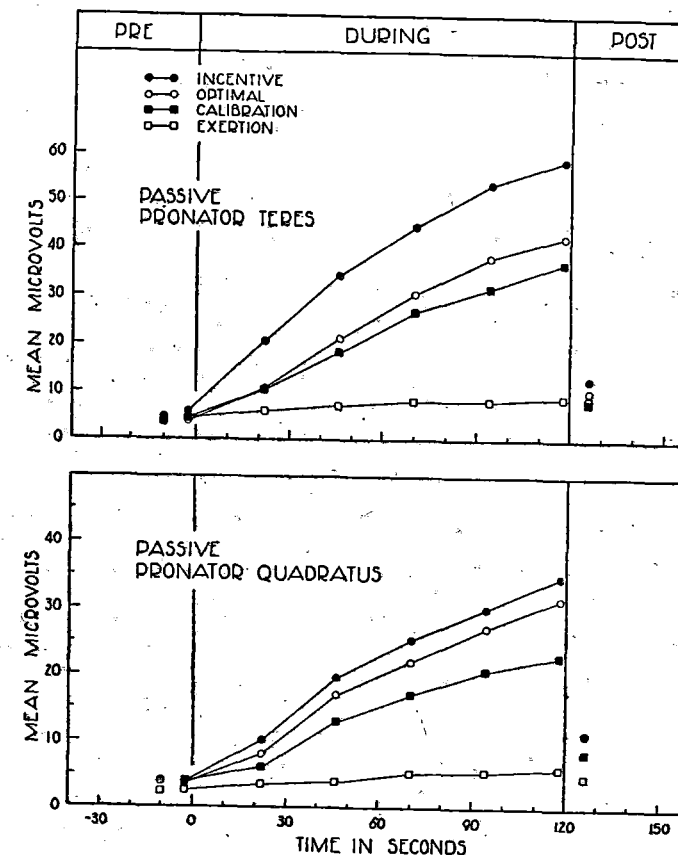


FIG. 2. Graphs from Stennett's experiment (37), showing mean EMG gradients obtained under conditions varying in degree of incentive. Steepness of gradient varies directly with degree of incentive. $N = 31$.

If we consider our physiological measures as indicants of arousal level, we may say that performance suffered in the first instance because of underarousal (or poor motivation), while in the second instance it suffered from overarousal (or emotional interference). In short, as Stennett has previously stated (37), we believe that the concept of arousal leads us in the direction of working out (empirically) a continuum of behavioral intensity which promises to have the very desirable feature of integrating the concepts of motivation and emotion. From available data it appears that physiological measures, such

as palmar skin conductance, EEG⁴ and gradients in skeletal muscle tension, heart rate, blood pressure and respiration (26) should provide reliable measures of the arousal variable. The objective nature of the physiological measures is a highly desirable feature which frees the worker from dependence upon merely manipulating situations in the

⁴ Stennett (38) has found that the relationship of alpha amplitude to arousal level is nonlinear. On the lower end of the arousal continuum the relationship is positive, such that raising arousal leads to increasing alpha amplitude; but past the middle range of arousal the relationship becomes inverse. This latter function is the better known one.

hope that he is producing intended changes in the arousal level of his subjects. Moreover, the physiological indicants have the further advantage that they may be applied to work with animals as well as with human subjects, and may thus serve usefully to bridge the gap, in the field of motivation, between work on human and on infrahuman subjects.

A word should be said concerning the different physiological measures which have served as indicants of behavioral arousal. Although gradient steepness has proved a very useful measure, level of palmar skin conductance seems equally promising. As a matter of fact, even with EMG, the correlation between average EMG level and gradient steepness is usually so high that it is meaningless to ask which is a better indicant. We still have much to learn concerning the application of physiological techniques to our problems. It may be that, as Lacey's work suggests (23), for most accurate assessment of arousal, special consideration should be given to individual differences in relative reactivity of different physiological systems.

Following the usage of Freeman (15) and Hebb (18), the term "arousal" is used to refer to the intensive dimension. I am aware that the term "arousal" is used by some EEG workers to denote flattening of an EEG tracing (e.g., 8, p. 132). When I use the term, I use it in a much broader sense, as a dimension of behavior, and I am not using this term to refer to the EEG phenomenon called "arousal" or "activation." It is for this reason that I specify *behavioral* arousal in the title of this paper. As investigative work proceeds, it may become heuristic to make a definite distinction between physiological arousal and behavioral intensity. Granting this possibility, I believe that for present purposes it may be preferable to accept a rather broad operational definition of the in-

tensity dimension, in which level of physiological activity, arousal, and intensive level are employed as roughly synonymous terms.

In short, the physiological measures appear to be useful tools in establishing and precisely quantifying a dimension of behavioral intensity. Indeed, I regard such objective measures as nearly indispensable to the achievement of a really satisfactory operational definition of behavioral intensity. In the absence of such objective measures, it is difficult to see how circularity can be avoided. Considerable work is required, of course, in working out the intensity dimension, and while present results are indeed encouraging, many further data are required. It may be helpful just here to relate the arousal continuum to the intensity dimension which Boring described (4). While Boring's main concern was with sensation, I believe that it is appropriate to consider that operations of measurement comparable in precision to those of psychophysics may be possible in the field of action.

EXPERIMENTAL STUDIES WITH PSYCHIATRIC PATIENTS

Having elucidated the concept of arousal with these reference experiments, we are now in a favorable position to take a fresh look at the data comparing patients and nonpatients with respect to level of physiological reaction under controlled stimulating conditions. At the outset, we may say that the chief impression which one gets in going over all of these data is that, under "stress," psychoneurotic patients appeared to show a higher level of physiological reaction than controls, and that level of reaction seemed particularly high in patients suffering mainly from pathological anxiety. By pathological anxiety, I mean a state of such severity that work efficiency is seriously affected over long periods of time, and a state which is

characterized by one or more of the following complaints: persistent feelings of "tension" or "strain," "irritability," "unremitting worry," "restlessness," "inability to concentrate," "feelings of panic in everyday-life situations." I should like to make it very clear that I do not employ the term "anxiety" to refer to transient affective states. When I use the term I am talking about a pathological condition which—as far as we can determine—develops slowly, over months or years, and from which recovery (when it occurs) is also slow and gradual. The experiments which we shall consider in this section employed patients suffering from "pathological anxiety," as we have just defined it. For the sake of convenience, these subjects will be called "anxiety patients."

In a study with pain as standard stimulus (27), the following physiological measures showed significantly greater reaction in anxiety patients than in other psychiatric patients: finger movement (and number of voluntary pressures to indicate pain), neck-muscle activity, deviation in amplitude and rate of respiration throughout the test, respiratory irregularities occurring at time of stimulation, and heart-rate variability. In a different study (29) with a perceptual test and a Luria-type recording from the left hand, finger movement was significantly more irregular in anxiety patients than in other psychiatric patients.

To repeat an earlier statement, these findings indicate that under standard conditions of stimulation psychoneurotics are more reactive than controls, and that patients with anxiety predominating in the symptom picture are the most responsive of all.

Need for "Standard Stress" in Demonstrating Differences Between Patients and Controls

Another question which we sought to answer was whether a certain level of

arousal must be reached in order to demonstrate differences between patients and nonpatients or whether such differences could be obtained under resting, "basal" conditions. From reviewing the literature prior to conducting our own experiments, we were led to suspect that some stimulation would be necessary because experiments which had been carried out under resting conditions had usually yielded negative or inconclusive results.

Our findings did indeed clearly show that, in differentiating between patients and controls, some form of stimulation was definitively superior to merely taking records under resting conditions. This has been demonstrated for blood pressure (28, p. 89), for muscle potentials in motor tasks (31, p. 54 and pp. 59 ff.), and again for muscle potentials in two separate investigations of startle (30, p. 327; 7, p. 181). The only measure which we have found to discriminate well between patients and controls under "resting" conditions was frontalis-muscle potentials (33). However, we know that "resting" conditions associated with a testing session are by no means basal, and that—for example—significantly lower blood-pressure readings may be obtained from patients resting quietly on the ward than in the so-called "resting" condition of our experiments (32).

"Specific" vs. "nonspecific" stimulating situations. In producing higher levels of arousal in patients, is it necessary to present material to which patients are specifically sensitized or is it possible to demonstrate the difference between patients and controls by employing the same standard stimulating situation for all subjects? Our experiments clearly show that the latter is true. It is not necessary to present the patients with words or situations which have special meanings for them in order to produce more arousal in them than in controls.

As an example of a "specific-con" 000112

plex" technique of producing high-level arousal, Luria (25) employed the method of controlled association in which he compared motor reaction to "critical" words (those which were especially arousing for the subject because of their association with specific life experiences) with reaction to indifferent words. Our situations, on the other hand, were chosen for their general arousal value, and we sought to avoid situations which would have special meaning for particular individuals.

With this point especially in mind we devised our standard situation of painful stimulation, because of the nearly universal avoidance reaction to pain. In order to permit more generalized conclusions, we also employed standard situations other than pain. One study is of especial interest because we reproduced the essential features of Luria's procedure, only substituting a series of size discriminations for the series of verbal stimulations which Luria employed (29). Conclusions from these experiments were as follows. All measures of motor activity recorded during performance of speeded size discrimination yielded reliable differences between patients and controls. In every instance there was evidence of greater physiological disturbance in the patients. The measures employed may be distinguished as skeletal-motor (motor control, muscular tension) and autonomic (systolic blood pressure). These differences in motor activity were manifested even though psychoneurotics, acute psychotics, and controls were practically identical with respect to perceptual performance.

These results led us to question certain views concerning determinants of higher arousal levels in psychoneurotics. In much current writing there is the underlying assumption that physiological disturbances in the psychoneurotic can be accounted for entirely in terms of situational explanations. These writers

assume that there is no need to look for pathology in central and motor mechanisms, because they believe that amount of physiological disturbance is commensurate with the special significance which the situation has for the patient. Implied in this view is the assumption that only those stimuli which, through learning, have acquired special meaning for the patient have the power to produce an "abnormal" level of arousal. It assumes that the patient may participate in many situations without showing abnormally high levels of physiological reaction.

However, this view may well be questioned because it does not appear to fit with clinical observations. Cameron has written as follows:

It will be noted that nearly all such patients [with anxiety states] complain that they cannot go into crowded places or into any situation where sustained efforts will be required of them. Their symptoms are made more severe by anything which elicits emotional reactions, such as altercations or participating in a discussion of illness. Nearly all find, at least at first, that their symptoms are increased by visiting their former places of employment or meeting fellow-workers. In other words, their symptoms are exacerbated by anything which serves to increase tension. *Emphasis should be placed upon the fact that their symptoms are elicited or intensified, not primarily by the reactivation of any conflict situation which may exist, but literally by everything in the course of the day which serves to increase tension* (5, pp. 56-57. Italics mine).

In therapy, relaxants of various kinds are devised to "damp" the "autonomous" reaction before proceeding with psychotherapy (41).

Strong auditory stimulation. Strong auditory stimulation served as another and very different kind of standard stimulating situation for comparing patients and controls. Two separate studies, the first one (30) with induced tension (produced by squeezing a rubber bulb), and the second (7) without induced tension and with a less intense stimulus, agreed in showing that the

most reliable difference between anxious patients and controls was in "after-response" following the period of primary reflex-startle reaction.

NEUROPHYSIOLOGICAL CONSIDERATIONS

In the interpretation of our findings in the experiments on strong auditory stimulation (7, 30), we cited the parallel between these observations on patients and findings in neurophysiological experiments on the reticular formation. In certain animal preparations, after-discharge in the cerebral cortex was abolished by stimulation in the reticular formation of thalamus (20) and brain stem (35). We believe that it is reasonable to suggest that some such inhibitory mechanism (as the one which abolished after-discharge) may be weakened in pathological anxiety.

Having implicated inhibition, we are required to examine this concept critically for a moment. Although there is by no means complete agreement on the matter of inhibitory mechanisms in the central nervous system, present evidence appears to point more and more in the direction of inhibition as a phenomenon in its own right, independent of excitation (i.e., not merely absence of excitation).

Of the current theories of inhibition known to me, Eccles' view seems most reasonable (12). Eccles and his co-workers developed a technique for placing a microelectrode within a single spinal motoneurone, and they were thus able to observe the electrical potential between the inside and the outside of the cell. They observed that when they stimulated an inhibitory nerve fiber it increased the polarization of the nerve cell on which it ended. Eccles called this effect "hyperpolarization," which, electrically is the opposite of what occurs when a nerve cell is fired (depolarized).

While Eccles' work was done on cells in the spinal cord, it nonetheless seems reasonable to suggest that the reticular formation could produce widespread inhibition in the cortex by hyperpolarizing cortical cells. Because the study of neuronal discharge in the cortex is a new field of research, sufficient data to decide this point are not at hand. But data which are presently available seem to be in line with the proposition that some impulse arriving in the cortex may have facilitatory effects, while others may produce opposite results (21, Fig. 19, p. 62).

If Eccles' theory is essentially correct,⁵ we may work with inhibition as an independent process, and seek to understand the pathology of anxiety in terms of weakened inhibition. To make matters more concrete, we may draw on Eccles' hypothesis of a chemical transmitter for inhibition (12, p. 163) and on the recent experimental work of Elliott and Florey (13) to suggest that, in anxiety, the effectiveness of this substance has been reduced.

THE PROBLEM OF ETIOLOGY

The disorder of pathological anxiety may be conceived of almost entirely in terms of constitutional factors. It is logical to consider that certain individuals may inherit a deficient inhibitory mechanism. Such a person would consistently suffer from inability to relax throughout life, and would be seriously limited in the amount of stimula-

⁵ Recent findings, although supporting Eccles' main conclusions, suggest that the phenomenon may be somewhat more complex than he originally supposed. The observations of Kuffler and Eyzaguirre (22) on inhibition of stretch receptor organs in crustaceans indicate that the polarity of the "inhibitory potential" varies with the state of the cell. When the cell is depolarized, an inhibitory volley causes polarization; when the cell is resting, an inhibitory volley causes depolarization.

tion that he could withstand. In such a case the constitutional weakness, rather than learning, would be the primary factor in etiology. While constitutional differences of genetic origin may account for degree of predisposition to the pathological condition of anxiety, clinical evidence stands against a purely genetic etiology. The fact that such a large number of patients recover from anxiety states (17, 34) argues against a purely genetic-constitutional explanation of pathological anxiety.

Declining the genetic-constitutional explanation of anxiety implies that learning mechanisms are somehow involved in the pathology. In order to understand the full implications of this point of view, it is helpful to consider that degree of arousal is not a "given" in the stimulating situation. The same stimulating situation may produce quite different levels of physiological reaction in different persons, depending upon the effects of past learning.⁶ We may compare individuals with respect to their physiological reactions in a large number of different situations. We may find, for example, that a certain person generally shows significantly higher levels of physiological reaction than most other individuals. If this person can avoid stimulating situations with high-arousal values he appears no different from others. However, in ordinary, everyday living, it is unlikely that he will be

⁶ The reader will recall that in our physiological studies of psychiatric patients we attempted to avoid experimental situations which had special meanings for particular individuals. In an earlier section of this paper we referred to these situations as "nonspecific." We assume that an anxiety-prone individual, before he actually develops the pathological state (and after he recovers from it), will not show higher arousal levels in such "nonspecific" stimulating situations. The stimulating situations referred to as producing quite different levels of physiological reaction in different persons are, of course, what we called "specific" in the earlier section of this paper.

able to avoid such situations, and he will, therefore, be more or less constantly operating at physiological levels which are higher than normal. We may conjecture that in such a case in which stimulation keeps physiological levels constantly very high, over a long period of time there will be a weakening of inhibitory mechanisms from overuse.

FURTHER CLINICAL-EXPERIMENTAL CONSIDERATIONS

Anxiety in combat. If our theory is correct, anxiety may be considered as a "disease of overarousal" (or in Selye's [36] terms, a disease of "adaptation"). That is, the critical neural change is thought of as being produced by a process of attrition from excessive and extended overarousal. It would not matter whether this overarousal were produced in an individual whose previous learning made him more prone to overarousal, or whether the individual were anxiety-resistant from past training, and was simply "overexposed" to situations (like battle) that everyone reacts to with extremely high physiological levels. With this view we can readily understand why under battle conditions each soldier would have his "breaking point," and why despite resistance to overarousal from constitution and previous learning, if situations of high-arousal level are repeated over a long enough time period, the critical change will finally occur. This seems to be the picture which emerges from studies of anxiety in combat (16, pp. 85 ff.).

Inhibitory Deficiency in Anxiety and in Manic States

From the clinical point of view, Cameron (6, p. 388) has drawn attention to the prominence of overactivity in the anxiety states. Cameron is inclined to believe, however, that the manic state best represents "pure overfacilitation," in comparison with anx-

iety, which he has described as "curbed overactivity." In drawing this comparison, Cameron was influenced by his careful observation of body movements. He found that the typical anxious patient was restless and in constant movement, but that he did not have the open, wide, flung-out movements of the manic. In general, the movements of the anxious patient remained within the body silhouette.⁷

The internally generated manic overactivity ("pure overfacilitation") could reasonably be accounted for by positing increased activity of facilitatory mechanisms.

PROBLEMS FOR FURTHER STUDY

The line of reasoning followed in the present paper suggests certain hypotheses which might be put to experimental test. In the first place, longitudinal physiological study of patients suffering severe states of anxiety should reveal changed physiological reaction under conditions of standard stimulation. That is, during performance of a motor task—for example, palmar skin conductance—electromyographic gradients and other physiological indicants of arousal should show decline when the patient is in remission, and should show increase again with relapse and return of the anxiety. This is a straightforward kind of investigation which one might suppose had already been under-

⁷ On the surface, this appears incongruous with the notion of weakened inhibition. However, we may account for this constrained appearance of inhibition by suggesting the substitution of less efficient mechanisms of inhibition for the one which has suffered impairment. It may be, for example, that anxiety patients compensate for weakened autonomous mechanisms by calling on voluntary motor mechanisms (i.e., the pyramidal motor system). For example, in the absence of sufficient control from autonomous inhibitory mechanisms, the anxiety patient may avoid loss of motor control through co-contraction of antagonistic muscles.

taken. However, as far as I am aware, the study has not been carried out with anxiety states in the way proposed.

Anxiety and Learning

Physiological measures of arousal should prove valuable in learning experiments in which anxiety has been studied as a variable (14). For example, workers have employed questionnaires and scales (e.g., the Taylor scale [40]), to select subjects high in "anxiety." The chief purpose of such experiments has been to compare the learning speed of subjects scoring high on such a scale with other subjects scoring lower on the scale. It would appear that physiological measures could be applied to such problems with considerable advantage. Subjects who would probably react at high physiological levels could still be selected with the scales as an initial screening device; but physiological measurements could then be applied to provide actual values to place each subject on a continuum. Such methodology would appear promising in providing a continuous variable (i.e., physiological intensity, or arousal) for study in place of the rather dubious anxiety-nonanxiety dichotomy, and would have other advantages. For example, a low scorer on the scale might be temporarily upset, and so be misclassified in an experiment unless his actual physiological measures were available on the day of the experiment.

Research with Reserpine and Chlorpromazine

Patients exhibiting anxiety as the predominant symptom have been reported to improve significantly following the administration of reserpine and chlorpromazine (19). It should prove illuminating to study the effects of such drugs on physiological reaction of anxiety patients under controlled stimulating conditions. For example, with administra-

tion of these drugs, would the electromyographic reaction of patients to strong auditory stimulation resemble the normal reaction more closely (show less after-response) than in the absence of the drugs?

It would likewise be of interest to determine the effect of such drugs on levels of physiological reaction in anxiety patients under conditions of moderate stimulation, such as those in our experiments with pain and with performance tasks. Would drug administration bring levels of physiological reaction down close to normal values under these conditions?

As a matter of fact, our experiments with psychiatric patients were performed prior to the full development of the concept of an intensity continuum in behavior, measured in terms of EMG gradients, level of palmar skin conductance, and other such physiological indicators. It would be highly desirable, therefore, to apply these more refined physiological measures to the study of anxiety patients. Do they, in fact, show steeper EMG gradients than normals in tracking, and are these gradients reduced in slope with administration of reserpine and chlorpromazine?

Proposed Animal Experiments

Certain aspects of these problems may be more advantageously studied with animal subjects. Studies of "experimental neuroses," as reviewed by Liddell (24), have shown that it is possible to produce chronic states characterized by physiological deviation. For present purposes it would be desirable to employ a form of stimulation which effectively maintains high levels of physiological reaction over long periods of time. For our purposes it would not matter particularly how the stimulation was produced; the main requisite is that high physiological levels be recorded continuously over days and weeks.

The main purpose of such an experiment would be to determine whether keeping physiological levels constantly high would finally produce "anxiety" in animals (i.e., animals with raised physiological levels in standard test situations). If such experiments did turn out positively, valuable animal "preparations" would be available for neurophysiological and pharmacological studies.

Such a "preparation" might be used, for example, to determine whether inhibitory effects from stimulation in the reticular formation are weaker than in normal animals. We might even conceive of an experiment paralleling the ones which we carried out with human subjects. It would seem possible to implant electrodes in the reticular formation to search for areas which fire inhibitory impulses to the cerebral cortex following strong auditory stimulation. Furthermore, pharmacological investigation (13) might be directed to the question whether there is an inhibitory substance in the brain which becomes dilute with long-continued overarousal.

SUMMARY

The main purpose of this paper is to consider some recent experimental data which suggest a way out of the present confusion surrounding the concepts of motivation, emotion, and anxiety. Two lines of investigation, each employing physiological methods, are examined. In one experimental program, measures such as steepness of muscle-potential gradients and level of palmar skin conductance were found to be useful indicators of arousal level. The results of several experiments demonstrated significant relationships between such physiological indicators and excellence of performance on various motor tasks, such as mirror tracing and tracking. In this empirical setting, problems of relationship between concepts of motivation and emotion are reconsidered.

The arousal concept is then applied to the problem of pathological anxiety in psychiatric patients. The earlier results from physiological studies carried out with psychiatric patients as subjects are reviewed in the light of the more recent work on physiological indicants of arousal. Considerable confusion has arisen because the term "anxiety" has been used to denote two quite different states of the organism: (a) any increase in level of arousal, however brief the rise (or however selective the stimulating condition); and (b) a pathological state in which the patient appears chronically overreactive (physiologically) to every stimulating situation.

It seems reasonable to restrict the term "anxiety" to the chronic pathological condition. Results from physiological studies carried out with patients suffering this pathological condition indicated that standard stimulation (or "stress") accentuated the differences in arousal between anxiety patients and controls. Under resting conditions such differences were usually insignificant. On the basis of the data reviewed, certain hypotheses concerning the nature and etiology of pathological anxiety are tentatively advanced. It is suggested that anxiety may be produced in an individual (in animal as well as in man) by keeping level of arousal very high over long periods of time. Finally, recent neurophysiological findings are cited in stating the hypothesis that such continuous overarousal may result in impairment of central inhibitory mechanisms.

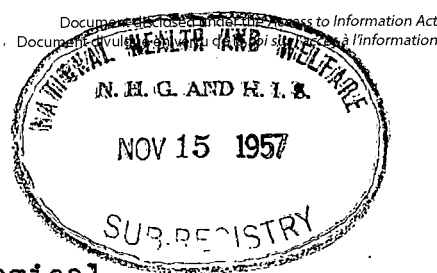
REFERENCES

1. BARTOSHUK, A. K. Electromyographic gradients in goal-directed activity. *Canad. J. Psychol.*, 1955, 9, 21-28.
2. BARTOSHUK, A. K. Electromyographic gradients as indicants of motivation. *Canad. J. Psychol.*, 1955, 9, 215-230.
3. BÉLANGER, D. J. "Gradients" musculaires et processus mentaux supérieurs. *Canad. J. Psychol.* (in press).

4. BORING, E. G. *The physical dimensions of consciousness*. New York: Century, 1933.
5. CAMERON, D. E. Autonomy in anxiety. *Psychiat. Quart.*, 1944, 18, 53-60.
6. CAMERON, D. E. Some relationships between excitement, depression, and anxiety. *Amer. J. Psychiat.*, 1945, 102, 385-394.
7. DAVIS, J. F., MALMO, R. B., & SHAGASS, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. *Canad. J. Psychol.*, 1954, 8, 177-186.
8. DELAFRESNAYE, J. F. (Ed.). *Brain mechanisms and consciousness*. Springfield, Ill.: Thomas, 1954. (See especially discussion by H. H. Jasper, p. 132.)
9. DUFFY, ELIZABETH. The conceptual categories of psychology: a suggestion for revision. *Psychol. Rev.*, 1941, 48, 177-203.
10. DUFFY, ELIZABETH. A systematic framework for the description of personality. *J. abnorm. soc. Psychol.*, 1949, 44, 175-190.
11. DUFFY, ELIZABETH. The concept of energy mobilization. *Psychol. Rev.*, 1951, 58, 30-40.
12. ECCLES, J. C. *The neurophysiological basis of mind*. Oxford: Clarendon, 1953.
13. ELLIOTT, K. A. C., & FLOREY, E. Factor I—Inhibitory factor from brain. Assay. Condition in brain. Stimulating and antagonizing substances. *J. Neurochem.*, 1956, 1, 181-192.
14. FARBER, I. E. Anxiety as a drive state. In M. R. Jones (Ed.), *Nebraska Symposium on Motivation*. Lincoln: Univ. of Nebraska Press, 1954.
15. FREEMAN, G. L. *The energetics of human behavior*. Ithaca, N. Y.: Cornell Univ. Press, 1948.
16. GRINKER, R. R., & SPIEGEL, J. P. *Men under stress*. Philadelphia: Blakiston, 1945.
17. HARRIS, A. The prognosis of anxiety states. *Brit. med. J.*, 1938, 2, 649-664.
18. HEBB, D. O. Drives and the CNS. (conceptual nervous system). *Psychol. Rev.*, 1955, 62, 243-254.
19. HOLLISTER, L. E., TRAUB, L., & BECKMAN, W. G. Psychiatric use of reserpine and chlorpromazine. Results of double-blind studies. In N. S. Kline (Ed.), *Psychopharmacology*. Washington, D. C.: Amer. Assoc. for Advancement of Science, 1956.
20. JASPER, H. H. Diffuse projection systems: the integrative action of the thalamic

- reticular system. *EEG Clin. Neurophysiol.*, 1949, 1, 405-420.
21. JUNG, R. Neuronal discharge. *EEG Clin. Neurophysiol.*, 1953, Suppl. No. 4, 57-71.
22. KUFFLER, S. W., & EYZAGUIRRE, C. Synaptic inhibition in an isolated nerve cell. *J. gen. Physiol.*, 1955, 39, 155-184.
23. LACEY, J. I. Individual differences in somatic response patterns. *J. comp. Physiol. Psychol.*, 1950, 43, 338-350.
24. LIDDELL, H. S. Conditioned reflex method and experimental neurosis. In J. McV. Hunt (Ed.), *Personality and the behavior disorders*. New York: Ronald, 1944. Vol. I, pp. 389-412.
25. LURIA, A. R. *The nature of human conflict*. New York: Liveright, 1932.
26. MALMO, R. B., & DAVIS, J. F. Physiological gradients as indicants of "arousal" in mirror tracing. *Canad. J. Psychol.*, 1956, 10, 231-238.
27. MALMO, R. B., & SHAGASS, C. Physiologic studies of reaction to stress in anxiety and early schizophrenia. *Psychosom. Med.*, 1949, 11, 9-24.
28. MALMO, R. B., & SHAGASS, C. Studies of blood pressure in psychiatric patients under stress. *Psychosom. Med.*, 1952, 14, 82-93.
29. MALMO, R. B., SHAGASS, C., BÉLANGER, D. J., & SMITH, A. A. Motor control in psychiatric patients under experimental stress. *J. abnorm. soc. Psychol.*, 1951, 46, 539-547.
30. MALMO, R. B., SHAGASS, C., & DAVIS, J. F. A method for the investigation of somatic response mechanisms in psychoneurosis. *Science*, 1950, 112, 325-328.
31. MALMO, R. B., SHAGASS, C., & DAVIS, J. F. Electromyographic studies of muscular tension in psychiatric patients under stress. *J. clin. exp. Psychopath.*, 1952, 12, 45-66.
32. MALMO, R. B., SHAGASS, C., & HESLAM, R. M. Blood pressure response to repeated brief stress in psychoneurosis: a study of adaptation. *Canad. J. Psychol.*, 1951, 5, 167-179.
33. MALMO, R. B., & SMITH, A. A. Forehead tension and motor irregularities in psychoneurotic patients under stress. *J. Personality*, 1955, 23, 391-406.
34. MILES, H. H. W., BARRABEE, EDNA L., & FINESINGER, J. E. Evaluation of psychotherapy. *Psychosom. Med.*, 1951, 13, 83-105.
35. MORUZZI, G., & MAGOUN, H. W. Brain stem reticular formation and activation of the EEG. *EEG Clin. Neurophysiol.*, 1949, 1, 455-473.
36. SELYE, H. *Stress*. Montreal: Acta, 1950.
37. STENNETT, R. G. The arousal continuum. *J. exp. Psychol.* (in press).
38. STENNETT, R. G. The relationship of alpha amplitude to the level of palmar conductance. *EEG Clin. Neurophysiol.*, 1957, 9, 131-138.
39. SURWILLO, W. W. Psychological factors in muscle-action potentials: EMG gradients. *J. exp. Psychol.*, 1956, 52, 263-272.
40. TAYLOR, JANET A. A personality scale of manifest anxiety. *J. abnorm. soc. Psychol.*, 1953, 48, 285-290.
41. TYHURST, J. S., & RICHMAN, A. Clinical experience with psychiatric patients on reserpine—preliminary impressions. *Canad. med. Assoc. J.*, 1955, 72, 458-459.
42. WALLERSTEIN, H. An electromyographic study of attentive listening. *Canad. J. Psychol.*, 1954, 8, 228-238.

(Received December 10, 1956)



Publications from The Laboratory for Psychological
Studies.

1. Malmo, R.B. and Amsel, A. Anxiety-produced interference in serial rote learning with observations on rote learning after partial frontal lobectomy. J. exp. Psychol., 1948, 38, 440-454.
2. Malmo, R.B., and Shagass, C. Behavioral and physiologic changes under stress after operations on the frontal lobes. Arch. Neurol. Psychiat., 1950, 63, 113-124.
3. Malmo, R.B., Shagass, C., Davis, J.F., Cleghorn, R.A., Graham, B.F., and Goodman, A.J. Standardized pain stimulations as controlled stress in physiological studies of psychoneurosis. Science, 1948, 108, 509-511.
4. Malmo, R.B. and Shagass, C. Physiologic studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.
5. Malmo, R.B. and Shagass, C. Physiologic study of symptom mechanisms in psychiatric patients under stress. Psychosom. Med., 1949, 11, 25-29.
6. Malmo, R.B., Shagass, C., and Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
7. Malmo, R.B., Shagass, C., and Davis, F.H. Symptom specificity and bodily reactions during psychiatric interview. Psychosom. Med., 1950, 12, 362-376.
8. Malmo, R.B. and Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
9. Malmo, R.B., Shagass, C., and Heslam, R.M. Blood pressure responses to repeated brief stress in psychoneurosis: a study of adaptation. Canadian J. Psychol., 1951, 5, 167-179.
10. Malmo, R.B. and Shagass, C. Variability of heart rate in relation to age, sex and stress. J. appl. Physiol., 1949, 2, 181-184.
11. McMurray, G.A. Experimental study of a case of insensitivity to pain. Arch. Neurol. Psychiat., 1950, 64, 650-667.
12. Malmo, R.B., Shagass, C., Belanger, D.J., and Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
13. Malmo, R.B., Shagass, C., and Smith, A.A. Responsiveness in chronic schizophrenia. J. Personality, 1951, 19, 359-375.

Publications from The Laboratory for Psychological Studies - Cont'd

14. Malmo, R.B., Shagass, C., and Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
15. Davis, F.H. and Malmo, R.B. Electromyographic recording during interview. Amer. J. Psychiat., 1951, 107, 908-916.
16. Shagass, C. and Malmo, R.B. Psychodynamic themes and localized muscular tension during psychotherapy. Psychosom. Med., 1954, 16, 295-313.
17. Malmo, R.B., Davis, J.F., and Barza, S. Total hysterical deafness; an experimental case study. J. Personality, 1952, 21, 188-204.
18. Davis, J.F. Manual of surface electromyography. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1952 (mimeo.).
19. Smith, A.A. An electromyographic study of tension in interrupted and completed tasks. J. exp. Psychol., 1953, 46, 32-36.
20. Malmo, R.B. Psychological aspects of frontal gyrectomy and frontal lobotomy in mental patients. Res. Publ. Ass. nerv. ment. Dis., 1947, 27, 537-564.
21. Malmo, R.B. Experimental studies of mental patients under stress. In Reymart, M.L. Feelings and emotions, New York: McGraw-Hill, 1950, P. 169.
22. Malmo, R.B., Shagass, C., and Davis, F.H. Specificity of bodily reactions under stress. Res. Publ. Ass. nerv. ment. Dis., 1950, 29, 231-261.
23. Malmo, R.B. The psychologist as a researcher. The Canadian Psychologist, 1952, 2, 19-21.
24. Malmo, R.B. Research: experimental and theoretical aspects. In Wittkower, E.D. and Cleghorn, R.A. Recent developments in psychosomatic medicine. London: Pitman, 1954. Pp. 84-100.
25. Malmo, R.B. Conversion hysteria. In Burton A. and Harris, R.E. (Eds.) Case histories in clinical and abnormal psychology. New York: Harper, 1955. Pp. 213-230.
26. Malmo, R.B. Higher functions of the nervous system. Ann. rev. Physiol., 1954, 16, 371-390.
27. Malmo, R.B. Eccles' neurophysiological model of the conditioned reflex. Canadian J. Psychol., 1954, 8, 125-129.

Publications from The Laboratory for Psychological Studies - Cont'd.

28. Malmo, R.B., Wallerstein, H., and Shagass, C. Headache proneness and mechanisms of motor conflict in psychiatric patients. J. Personality, 1953, 22, 162-187.
29. Shagass, C. The sedation threshold. A method for estimating tension in psychiatric patients. EEG Clin. Neurophysiol., 1954, 6, 221-233.
30. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canadian J. Psychol., 1955, 9, 21-28.
31. Davis, J.F., Malmo, R.B., and Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canadian J. Psychol., 1954, 8, 177-186.
32. Malmo, R.B. and Wallerstein, H. Rigidity and reactive inhibition. J. abn. soc. Psychol., 1955, 50, 346-348.
33. Malmo, R.B. and Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Malmo, R.B., Boag, T.J., and Raginsky, B.B. Electromyographic study of hypnotic deafness. J. clin. exp. Hypnosis, 1954, 2, 305-317.
35. Smith, A.A., Malmo, R.B., and Shagass, C. An electromyographic study of listening and talking. Canadian J. Psychol., 1954, 8, 219-227.
36. Wallerstein, H. An electromyographic study of attentive listening. Canadian J. Psychol., 1954, 8, 228-238.
37. Surwillo, W.W. A device for recording variations in pressure of grip during tracking. Amer. J. Psychol., 1955, 68, 669-670.
38. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canadian J. Psychol., 1955, 9, 215-230.
39. Malmo, R.B., Kohlmeyer, W., and Smith, A.A. Motor manifestation of conflict in interview. J. abn. soc. Psychol., 1956, 52, 268-271.
40. Malmo, R.B. Symptom mechanisms in psychiatric patients. Trans. N.Y. Acad. Sci., 1956, 18, 545-549.
41. Bartoshuk, A.K. EMG gradients and EEG amplitude during motivated listening. Canadian J. Psychol., 1956, 10, 156-164.
42. Davis, J.F. Operator's Manual: A.M.I. Integrator system. Montreal: Laboratory for Psychological Studies, Allan Memorial Institute of Psychiatry, 1956 (mimeo.).

Publications from The Laboratory for Psychological Studies - Cont'd.

43. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.

44. Malmo, R.B., and Davis, J.F. Physiological gradients as indicants of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.

45. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.

46. Stennett, R.G. The relationship of performance level to level of arousal. J. exp. Psychol., 1957, 54, 54-61.

47. Malmo, R.B., Boag, T.J., and Smith, A.A. Physiological study of personal interaction. Psychosom. Med., 1957, 19, 105-119.

48. Belanger, D. "Gradients" musculaires et processus mentaux supérieurs. Canad. J. Psychol., 1957, 11, 113-122.

49. Malmo, R.B. Experimental approach to symptom mechanisms in psychiatric patients. Psychiatric Research Reports 7, American Psychological Association, April 1957, 33-53.

50. Malmo, R.B. Anxiety and behavioral arousal. Psychol. Rev., 1957, (in press).

Anxiety and Behavioral Arousal¹

Robert B. Malmö²

Allan Memorial Institute of Psychiatry, McGill University

During the past two decades there has been a growing interest in objective physiological studies of psychiatric patients. In this work one of the most prominent psychological concepts has been that of "anxiety." Although there is general agreement that the areas denoted by the term "anxiety" are important ones for study, there is nonetheless considerable disagreement concerning what the term means. In large measure, this semantic difficulty is part of a larger problem facing psychology today, and that is to find a way out of the confusion surrounding the concepts of motivation and emotion. Duffy has cogently argued that these concepts are second-order ones which reduce to primary factors of intensity and direction, and that along the intensity dimension, at least, the distinction between motivation and emotion is unnecessary (9, 10, 11).

This is not to say that the directional aspect is not important nor to deny that, in terms of direction, meaningful distinctions may be made between motivation and emotion, and indeed between different emotions. However, for present purposes it is essential to focus on the question of what these phenomena have in common rather than to consider their differences; and, in this paper, therefore, we shall be primarily concerned with the intensity dimension.

The main purpose of the present paper is to consider recent experimental data in an attempt to find a way out of the present confusion. I shall begin with a summary of two lines of investigation in our laboratory, dealing first with our discovery that certain physiological measures may serve as indicants of intensity or "behavioral arousal." These experiments were performed with nonpatient subjects. Secondly, in summarizing our investigations of pathological anxiety in psychiatric patients, I shall attempt to use the concept of behavioral arousal in an integrative way. Thirdly, I shall draw on data from recent neurophysiological investigations to indicate possible mechanisms involved in the pathology and etiology of anxiety. Finally, on the basis of these theoretical considerations, I suggest problems requiring further experimental study.

Physiological Indicators of Behavioral Intensity

In 1951 we (31) reported finding a gradient phenomenon from electromyographic (EMG) recording during mirror tracing. Since that time the phenomenon has been observed under various conditions in our laboratory. Figure 1 presents mirror-drawing data from a study by Bartoshuk (1). Note that the chin lead (which taps the speech muscles) also shows a gradient, that is, progressively rising muscle potentials from the beginning to the end of the task. Bélanger (3) found similar gradients from the arm in a size discrimination task. Wallerstein (42) found gradients in

the frontalis muscle in a task about as completely devoid of motor components as one could possibly design. The subject, reclining on a comfortable bed, listened to verbal material (short detective story or essay) presented to him by a tape recorder. In Wallerstein's experiment, the gradients extended over ten minutes and their steepness was related to the subject's reported degree of interest in listening (2, p. 228f.).

Bartoshuk (2) was the first to show that the fastest and most accurate subjects (i.e. superior performers on mirror tracing) produced the steepest muscle-potential gradients. Such a relationship of EMG gradients to motivation has been confirmed by three subsequent studies, employing tracking tasks. Surwillo (39) demonstrated that raising incentive had the effect of increasing the steepness of EMG gradients in a visual tracking experiment. Figure 2 presents confirmatory data from a more recent experiment by Stennett (37) who employed auditory tracking and four conditions with increasing degrees of incentive. Note that the muscle potentials were recorded from the nonactive, left arm. His "exertion" condition merely involved the subject's holding the tracking knob over at a fixed point in order to control for sheer physical work. Under the "calibration" condition the subject believed that he was just assisting with calibration of the apparatus, and that his tracking scores were not being recorded. The "optimal" condition was designed to motivate the subject sufficiently to elicit his most efficient

Robert B. Malmö

performance, whereas the "incentive" condition was designed to "overmotivate" the subject by offering large bonuses for high-level performance and threatening with strong electric shock if performance did not reach this high level. The differences shown in the figure were statistically significant. In brief, Stennett's findings indicated that the most efficient tracking performance was associated with intermediate physiological levels (i.e. intermediate steepness of EMG gradients and intermediate levels of palmar skin conductance). With lower levels of physiological functioning (less steep gradients, lower levels of palmar skin conductance) performance on tracking was inferior. However, going now to the other extreme, performance on tracking associated with extremely high EMG gradients and extremely high palmar skin conductance was also inferior to tracking performance associated with moderately high levels of physiological functioning.

If we consider our physiological measures as indicants of arousal level, we may say that performance suffered in the first instance because of underarousal (or poor motivation), while in the second instance that it suffered from overarousal (or emotional interference). In short, as Stennett has previously stated (37), we believe that the concept of arousal leads us in the direction of working out (empirically) a continuum of behavioral intensity which promises to have the very desirable feature of integrating the concepts of motivation and emotion. From available data it appears that physiological measures, such as palmar skin conductance, EEG³ and gradients in skeletal

muscle tension, heart rate, blood pressure and respiration (26) should provide reliable measures of the arousal variable. The objective nature of the physiological measures is a highly desirable feature which frees the worker from dependence upon merely manipulating situations in the hope that he is producing intended changes in the arousal level of his subjects. Moreover, the physiological indicants have the further advantage that they may be applied to work with animals as well as with human subjects, and may thus serve usefully to bridge the gap, in the field of motivation, between work on human and on infrahuman subjects.

A word should be said concerning the different physiological measures which have served as indicants of behavioral arousal. Although gradient steepness has proved a very useful measure, level of palmar skin conductance seems equally promising. As a matter of fact, even with EMG, the correlation between average EMG level and gradient steepness is usually so high that it is meaningless to ask which is a better indicant. We still have much to learn concerning the application of physiological techniques to our problems. It may be that, as Lacey's work suggests (23), for most accurate assessment of arousal, special consideration should be given to individual differences in relative reactivity of different physiological systems.

Following the usage of Freeman (15) and Hebb (18) the term "arousal" is used to refer to the intensive dimension. I am aware that the term "arousal" is used by some EEG workers to denote flattening of an EEG tracing (8, p. 132). When I

Robert B. Malmö

-6-

use the term, I use it in a much broader sense, as a dimension of behavior, and I am not using this term to refer to the EEG phenomenon called "arousal" or "activation." It is for this reason that I specify behavioral arousal in the title of this paper. As investigative work proceeds, it may become heuristic to make a definite distinction between physiological arousal and behavioral intensity. Granting this possibility, I believe that for present purposes it may be preferable to accept a rather broad operational definition of the intensity dimension, in which level of physiological activity, arousal, and intensive level are employed as roughly synonymous terms.

In short, the physiological measures appear useful tools in establishing and precisely quantifying a dimension of behavioral intensity. Indeed, I regard such objective measures as nearly indispensable to the achievement of a really satisfactory operational definition of behavioral intensity. In the absence of such objective measures, it is difficult to see how circularity can be avoided. Considerable work is required, of course, in working out the intensity dimension, and while present results are indeed encouraging, much further data are required. It may be helpful just here to relate the arousal continuum to the intensity dimension which Boring (4) described. While Boring's main concern was with sensation, I believe that it is appropriate to consider that operations of measurement comparable in precision to those of psychophysics may be possible in the field of action.

Experimental Studies with Psychiatric Patients

Having elucidated the concept of arousal with these reference experiments we are now in a favorable position to take a fresh look at the data comparing patients and nonpatients with respect to level of physiological reaction under controlled stimulating conditions. At the outset, we may say that the chief impression which one gets in going over all of these data is that, under "stress," psychoneurotic patients appeared to show a higher level of physiological reaction than controls, and that level of reaction seemed particularly high in patients suffering mainly from pathological anxiety. By pathological anxiety, I mean a state of such severity that work efficiency is seriously affected over long periods of time, and a state which is characterized by one or more of the following complaints: persistent feelings of "tension" or "strain," "irritability," "unremitting worry," "restlessness," "inability to concentrate," "feelings of panic in everyday-life situations." I should like to make it very clear that I do not employ the term "anxiety" to refer to transient affective states. When I use the term I am talking about a pathological condition which--as far as we can determine--develops slowly, over months or years, and from which recovery (when it occurs) is also slow and gradual. The experiments which we shall consider in this section employed patients suffering from "pathological anxiety," as we have just defined it. For the sake of convenience, these subjects will be called "anxiety-patients."

In a study with pain as standard stimulus (27) the following physiological measures showed significantly greater reaction in anxiety-patients than in other psychiatric patients: finger movement (and number of voluntary pressures to indicate pain), neck-muscle activity, deviation in amplitude and rate of respiration throughout the test, respiratory irregularities occurring at time of stimulation, and heart-rate variability. In a different study (29) with a perceptual test and a Luria-type recording from the left hand, finger movement was significantly more irregular in anxiety-patients than in other psychiatric patients.

To repeat an earlier statement, these findings indicate that under standard conditions of stimulation psychoneurotics are more reactive than controls, and that patients with anxiety predominating in the symptom picture are the most responsive of all.

Need for "standard stress" in demonstrating differences between patients and controls

Another question which we sought to answer was whether a certain level of arousal must be reached in order to demonstrate differences between patients and nonpatients or whether such differences could be obtained under resting, "basal" conditions. From reviewing the literature prior to conducting our own experiments we were led to suspect that some stimulation would be necessary because experiments which had been carried out under resting conditions had usually yielded negative or inconclusive results.

Our findings did indeed clearly show that, in differentiating between patients and controls, some form of stimulation was defini-

tively superior to merely taking records under resting conditions. This has been demonstrated for blood pressure (28, p. 89), for muscle potentials in motor tasks (31, p. 54 and p. 59ff.) and again for muscle potentials in two separate investigations of startle (30, p. 327, 7, p. 181). The only measure which we have found to discriminate well between patients and controls under "resting" conditions was frontalis-muscle potentials (33). However, we know that "resting" conditions associated with a testing session are by no means basal, and that--for example--significantly lower blood-pressure readings may be obtained from patients resting quietly on the ward, than in the so-called "resting" condition of our experiments (32).

"Specific" vs. "nonspecific" stimulating situations. In producing higher levels of arousal in patients is it necessary to present material to which patients are specifically sensitized or is it possible to demonstrate the difference between patients and controls by employing the same standard stimulating situation for all subjects? Our experiments clearly show that the latter is true. It is not necessary to present the patients with words or situations which have special meanings for them in order to produce more arousal in them than in controls.

As an example of a "specific-complex" technique of producing high-level arousal, Luria (25) employed the method of controlled association in which he compared motor reaction to "critical" words (those which were especially arousing for the subject because of their association with specific life experiences) with

reaction to indifferent words. Our situations, on the other hand, were chosen for their general arousal value, and we sought to avoid situations which would have special meaning for particular individuals.

With this point especially in mind we devised our standard situation of painful stimulation, because of the nearly universal avoidance reaction to pain. In order to permit more generalized conclusions, we also employed standard situations other than pain. One study is of especial interest because we reproduced the essential features of Luria's procedure, only substituting a series of size discriminations for the series of verbal stimulations which Luria employed (29). Conclusions from these experiments were as follows. All measures of motor activity recorded during performance of speeded size discrimination yielded reliable differences between patients and controls. In every instance there was evidence of greater physiological disturbance in the patients. The measures employed may be distinguished as skeletal-motor (motor control, muscular tension) and autonomic (systolic blood pressure). These differences in motor activity were manifested even though psychoneurotics, acute psychotics, and controls were practically identical with respect to perceptual performance.

These results led us to question certain views concerning determinants of higher arousal levels in psychoneurotics. In much current writing there is the underlying assumption that physiological disturbances in the psychoneurotic can be accounted for entirely in terms of situational explanations. These writers assume that there is no need to look for pathology in central and

motor mechanisms, because they believe that amount of physiological disturbance is commensurate with the special significance which the situation has for the patient. Implied in this view is the assumption that only those stimuli which through learning have acquired special meaning for the patient have the power to produce an "abnormal" level of arousal. It assumes that the patient may participate in many situations without showing abnormally high levels of physiological reaction.

However, this view may well be questioned because it does not appear to fit with clinical observations. Cameron has written as follows:

It will be noted that nearly all such patients [with anxiety states] complain that they cannot go into crowded places or into any situation where sustained efforts will be required of them. Their symptoms are made more severe by anything which elicits emotional reactions, such as altercations or participating in a discussion of illness. Nearly all find, at least at first, that their symptoms are increased by visiting their former places of employment or meeting fellow-workers. In other words, their symptoms are exacerbated by anything which serves to increase tension. Emphasis should be placed upon the fact that their symptoms are elicited or intensified, not primarily by the re-activation of any conflict situation which may exist, but literally by everything in the course of the day which serves to increase tension (5, pp. 56-57, italics mine).

In therapy, relaxants of various kinds are devised to "damp" the "autonomous" reaction before proceeding with psychotherapy (41).

Strong auditory stimulation. ~~Strong auditory stimulation~~ served as another and very different kind of standard stimulating situation for comparing patients and controls. Two separate studies, the first one (30) with induced tension (produced by squeezing a rubber bulb) and the second study (7) without induced tension and with a less intense stimulus, agreed in showing that the most reliable difference between anxious patients and controls was in "after-response" following the period of primary reflex-startle reaction.

Neurophysiological Considerations

In the interpretation of our findings in the experiments on strong auditory stimulation (30, 7) we cited the parallel between these observations on patients, and findings in neurophysiological experiments on the reticular formation. In certain animal preparations after-discharge in the cerebral cortex was abolished by stimulation in the reticular formation of thalamus (20) and brain stem (35). We believe that it is reasonable to suggest that some such inhibitory mechanism (as the one which abolished after-discharge) may be weakened in pathological anxiety.

Having implicated inhibition we are required to examine this concept critically for a moment. Although there is by no means complete agreement on the matter of inhibitory mechanisms in the central nervous system, present evidence appears to point more and more in the direction of inhibition as a phenomenon in its own right, independent on excitation (i.e. not merely absence of excitation).

Robert B. Malmö

Of the current theories of inhibition known to me, Eccles' view seems most reasonable (12). Eccles and his co-workers developed a technique for placing a microelectrode within a single spinal motoneurone, and they were thus able to observe the electrical potential between the inside and the outside of the cell. They observed that when they stimulated an inhibitory nerve fiber it increased the polarization of the nerve cell on which it ended. Eccles called this effect "hyperpolarization" which, electrically is the opposite of what occurs when a nerve cell is fired (depolarized).

While Eccles' work was done on cells in the spinal cord, it nonetheless seems reasonable to suggest that the reticular formation could produce widespread inhibition in the cortex by hyperpolarizing cortical cells. Because the study of neuronal discharge in the cortex is a new field of research, sufficient data to decide this point are not at hand. But data which are presently available seem to be in line with the proposition that some impulse arriving in the cortex may have facilitatory effects, while others may produce opposite results (21, Fig. 19, p. 62).

If Eccles' theory is essentially correct,⁴ we may work with inhibition as an independent process, and seek to understand the pathology of anxiety in terms of weakened inhibition. To make matters more concrete, we may draw on Eccles' hypothesis of a chemical transmitter for inhibition (12, p. 163) and on the recent experimental work of Elliott and Florey (13) to suggest that in anxiety, the effectiveness of this substance has been reduced.

The Problem of Etiology

The disorder of pathological anxiety may be conceived of almost entirely in terms of constitutional factors. It is logical to consider that certain individuals may inherit a deficient inhibitory mechanism. Such a person would consistently suffer from inability to relax throughout life, and would be seriously limited in the amount of stimulation that he could withstand. In such a case the constitutional weakness rather than learning would be the primary factor in etiology. While constitutional differences of genetic origin may account for degree of predisposition to the pathological condition of anxiety, clinical evidence stands against a purely genetic etiology. The fact that such a large number of patients recover from anxiety states (17, 34) argues against a purely genetic-constitutional explanation of pathological anxiety.

Declining the genetic-constitutional explanation of anxiety implies that learning mechanisms are somehow involved in the pathology. In order to understand the full implications of this point of view it is helpful to consider that degree of arousal is not a "given" in the stimulating situation. The same stimulating situation may produce quite different levels of physiological reaction in different persons, depending upon the effects of last learning.⁵ We may compare individuals with respect to their physiological reactions in a large number of different situations. We may find, for example, that a certain person generally shows significantly higher levels of physiological

reaction than most other individuals. If this person can avoid stimulating situations with high-arousal values he appears no different from others. However, in ordinary-everyday living, it is unlikely that he will be able to avoid such situations, and he will, therefore, be more or less constantly operating at physiological levels which are higher than normal. We may conjecture that in such a case, in which stimulation keeps physiological levels constantly very high, over a long period of time, there will be a weakening of inhibitory mechanisms from overuse.

Further Clinical-Experimental Considerations

Anxiety in combat. If our theory is correct, anxiety may be considered as a "disease of overarousal" (or in Selye's (36) terms, a disease of "adaptation"). That is, the critical neural change is thought of as being produced by a process of attrition from excessive and extended overarousal. It would not matter whether this overarousal were produced in an individual whose previous learning made him more prone to overarousal, or whether the individual were anxiety-resistant from past training, and was simply "overexposed" to situations (like battle) that everyone reacts to with extremely high physiological levels. With this view we can readily understand why under battle conditions each soldier would have his "breaking point," and why despite resistance to overarousal from constitution and previous learning, if situations of high-arousal level are repeated over a long enough time period, the critical change will finally occur. This

seems to be the picture which emerges from studies of anxiety in combat (16, p. 85ff.).

Inhibitory deficiency in anxiety and in manic states

From the clinical point of view, Cameron (6, p. 388) has drawn attention to the prominence of overactivity in the anxiety states. Cameron is inclined to believe, however, that the manic state best represents "pure overfacilitation", in comparison with anxiety which he has described as "curbed overactivity." In drawing this comparison, Cameron was influenced by his careful observation of body movements. He found that the typical anxious patient was restless and in constant movement, but that he did not have the open, wide, flung-out movements of the manic. In general, the movements of the anxious patient remained within the body silhouette.⁶

The internally generated manic overactivity ("pure overfacilitation") could reasonably be accounted for by positing increased activity of facilitatory mechanisms.

Problems for Further Study

The line of reasoning followed in the present paper suggests certain hypotheses which might be put to experimental test. In the first place, longitudinal physiological study of patients suffering severe states of anxiety should reveal changed physiological reaction under conditions of standard stimulation. That is, during performance of a motor task--for example--palmar skin conductance, electromyographic gradients and other physiological

Robert B. Malmö

-17-

indicants of arousal should show decline when the patient is in remission, and should show increase again with relapse and return of the anxiety. This is a straightforward kind of investigation which one might suppose had already been undertaken. However, as far as I am aware, the study has not been carried out with anxiety states in the way proposed.

Anxiety and Learning

Physiological measures of arousal should prove valuable in learning experiments in which anxiety has been studied as a variable (14). For example, workers have employed questionnaires and scales (e.g. the Taylor scale (40)), to select subjects high in "anxiety." The chief purpose of such experiments has been to compare the learning speed of subjects scoring high on such a scale with other subjects scoring lower on the scale. It would appear that physiological measures could be applied to such problems with considerable advantage. Subjects who would probably react at high physiological levels could still be selected with the scales as an initial screening device; but physiological measurements could then be applied to provide actual values to place each subject on a continuum. Such methodology would appear promising in providing a continuous variable (i.e. physiological intensity, or arousal) for study in place of the rather dubious anxiety-nonanxiety dichotomy, and would have other advantages. For example, a low scorer on the scale might be temporarily upset, and so be misclassified in an experiment unless his actual physiological measures were available on the day of the experiment.

Robert B. Malmo

Research with Reserpine and Chlorpromazine

Patients exhibiting anxiety as the predominant symptom have been reported to improve significantly following the administration of reserpine and chlorpromazine (19). It should prove illuminating to study the effects of such drugs on physiological reaction of anxiety-patients under controlled stimulating conditions.

For example, with administration of these drugs, would the electromyographic reaction of patients to strong auditory stimulation resemble the normal reaction more closely (show less after-response) than in the absence of the drugs?

It would likewise be of interest to determine the effect of such drugs on levels of physiological reaction in anxiety-patients under conditions of moderate stimulation, such as those in our experiments with pain and with performance tasks. Would drug administration bring levels of physiological reaction down close to normal values under these conditions?

As a matter of fact, our experiments with psychiatric patients were performed prior to the full development of the concept of an intensity continuum in behavior, measured in terms of EMG gradients, level of palmar skin conductance and other such physiological indicators. It would be highly desirable, therefore, to apply these more refined physiological measures to the study of anxiety-patients. Do they, in fact, show steeper EMG gradients than normals in tracking, and are these gradients reduced in slope with administration of reserpine and chlorpromazine?

Proposed Animal Experiments

Certain aspects of these problems may be more advantageously studied with animal subjects. Studies of "experimental neuroses" as reviewed by Liddell (24) have shown that it is possible to produce chronic states characterized by physiological deviation. For present purposes it would be desirable to employ a form of stimulation which effectively maintains high levels of physiological reaction over long periods of time. For our purposes it would not matter particularly how the stimulation was produced; the main requisite is that high physiological levels be recorded continuously over days and weeks.

The main purpose of such an experiment would be to determine whether keeping physiological levels constantly high would finally produce "anxiety" in animals (i.e. animals with raised physiological levels in standard test situations). If such experiments did turn out positively, valuable animal "preparations" would be available for neurophysiological and pharmacological studies.

Such a "preparation" might be used, for example, to determine whether inhibitory effects from stimulation in the reticular formation are weaker than in normal animals. We might even conceive of an experiment paralleling the ones which we carried out with human subjects. It would seem possible to implant electrodes in the reticular formation to search for areas which fire inhibitory impulses to the cerebral cortex following strong auditory stimulation. Furthermore, pharmacological investigation (13) might be directed to the question whether there is an inhibitory

substance in the brain which becomes dilute with long-continued overarousal.

Summary

The main purpose of this paper is to consider some recent experimental data which suggest a way out of the present confusion surrounding the concepts of motivation, emotion and anxiety. Two lines of investigation, each employing physiological methods, are examined. In one experimental program, measures such as steepness of muscle-potential gradients and level of palmar skin conductance were found to be useful indicants of arousal level. The results of several experiments demonstrated significant relationships between such physiological indicants and excellence of performance on various motor tasks such as mirror tracing and tracking. In this empirical setting, problems of relationship between concepts of motivation and emotion are reconsidered.

The arousal concept is then applied to the problem of pathological anxiety in psychiatric patients. The earlier results from physiological studies carried out with psychiatric patients as subjects are reviewed in the light of the more recent work on physiological indicants of arousal. Considerable confusion has arisen because the term anxiety has been used to denote two quite different states of the organism: (a) any increase in level of arousal, however brief the rise (or however selective the stimulating condition); and (b) a pathological state in which the

patient appears chronically over-reactive (physiologically) to every stimulating situation.

It seems reasonable to restrict the term "anxiety" to the chronic pathological condition. Results from physiological studies carried out with patients suffering this pathological condition indicated that standard stimulation (or "stress") accentuated the differences in arousal between anxiety-patients and controls. Under resting conditions such differences were usually insignificant. On the basis of the data reviewed, certain hypotheses concerning the nature and etiology of pathological anxiety are tentatively advanced. It is suggested that anxiety may be produced in an individual (in animal as well as in man) by keeping level of arousal very high over long periods of time. Finally, recent neurophysiological findings are cited in stating the hypothesis that such continuous overarousal may result in impairment of central inhibitory mechanisms.

References

1. Bartoshuk, A.K. Electromyographic gradients in goal-directed activity. Canad. J. Psychol., 1955, 9, 21-28.
2. Bartoshuk, A.K. Electromyographic gradients as indicants of motivation. Canad. J. Psychol., 1955, 9, 215-230.
3. Bélanger, D. "Gradients" musculaires et processus mentaux supérieur. Canad. J. Psychol. (in press).
4. Boring, E.G. The physical dimensions of consciousness. New York: Century, 1933.
5. Cameron, D.E. Autonomy in anxiety. Psychiat. Quart., 1944, 18, 53-60.
6. Cameron, D.E. Some relationships between excitement, depression, and anxiety. Amer. J. Psychiat., 1945, 102, 385-394.
7. Davis, J.F., Malmö, R.B. & Shagass, C. Electromyographic reaction to strong auditory stimulation in psychiatric patients. Canad. J. Psychol., 1954, 8, 177-186.
8. Delafresnaye, J.F. (Ed.) Brain mechanisms and consciousness. Springfield, Ill.: Thomas, 1954 (see esp. discussion by H.H. Jasper, p. 132).
9. Duffy, Elizabeth. The conceptual categories of psychology: A suggestion for revision. Psychol. Rev., 1941, 48, 177-203.

10. Duffy, Elizabeth. A systematic framework for the description of personality. J. abn. soc. Psychol., 1949, 44, 175-190.
11. Duffy, Elizabeth. The concept of energy mobilization. Psychol. Rev., 1951, 58, 30-40.
12. Eccles, J.C. The neurophysiological basis of mind. Oxford: Clarendon, 1953.
13. Elliott, K.A.C. & Florey, E. Factor I--Inhibitory factor from brain. Assay. Condition in brain. Stimulating and antagonizing substances. J. Neurochem., 1956, 1, 181-192.
14. Farber, I.E. Anxiety as a drive state. In M.R. Jones (Ed.) Nebraska Symposium on Motivation. Lincoln: Univer. of Nebraska Press, 1954.
15. Freeman, G.L. The energetics of human behavior. Ithica, N.Y.: Cornell Univer. Press, 1948.
16. Grinker, R.R. & Spiegel, J.P. Men under stress. Philadelphia: Blakiston, 1945.
17. Harris, A. The prognosis of anxiety states. Brit. Med. J., 1938, 2, 649-664.
18. Hebb, D.O. Drives and the C.N.S. (conceptual nervous system). Psychol. Rev., 1955, 62, 243-254.
19. Hollister, L.E., Traub, L. & Beckman, W.G. Psychiatric use of reserpine and chlorpromazine. Results of double-blind studies.

Robert B. Malmö

In N.S. Kline (Ed.) Psychopharmacology. Washington, D.C.: Amer. Assoc. for the Advancement of Science, 1956.

20. Jasper, H.H. Diffuse projection systems: the integrative action of the thalamic reticular system. EEG Clin. Neurophysiol., 1949, 1, 405-420.

21. Jung, R. Neuronal discharge. EEG Clin. Neurophysiol., 1953, Supplement No. 4, 57-71.

22. Kuffler, S.W. & Eyzaguirre, C. Synaptic inhibition in an isolated nerve cell. J. gen. Physiol., 1955, 39, 155-184.

23. Lacey, J.I. Individual differences in somatic response patterns. J. comp. physiol. Psychol., 1950, 43, 338-350.

24. Liddell, H.S. Conditioned reflex method and experimental neurosis. In J. McV. Hunt (Ed.) Personality and the behavior disorders. New York: Ronald, 1944.

25. Luria, A.R. The nature of human conflict. New York: Liveright, 1932.

26. Malmö, R.B. & Davis, J.F. Physiological gradients as indicators of "arousal" in mirror tracing. Canad. J. Psychol., 1956, 10, 231-238.

27. Malmö, R.B. & Shagass, C. Physiologic studies of reaction to stress in anxiety and early schizophrenia. Psychosom. Med., 1949, 11, 9-24.

28. Malmö, R.B. & Shagass, C. Studies of blood pressure in psychiatric patients under stress. Psychosom. Med., 1952, 14, 82-93.
29. Malmö, R.B., Shagass, C., Bélanger, D.J. & Smith, A.A. Motor control in psychiatric patients under experimental stress. J. abn. soc. Psychol., 1951, 46, 539-547.
30. Malmö, R.B., Shagass, C. & Davis, J.F. A method for the investigation of somatic response mechanisms in psychoneurosis. Science, 1950, 112, 325-328.
31. Malmö, R.B. Shagass, C. & Davis, J.F. Electromyographic studies of muscular tension in psychiatric patients under stress. J. clin. exp. Psychopath., 1951, 12, 45-66.
32. Malmö, R.B., Shagass, C. & Heslam, R.M. Blood pressure response to repeated brief stress in psychoneurosis: a study of adaptation. Canad. J. Psychol., 1951, 5, 167-179.
33. Malmö, R.B. & Smith, A.A. Forehead tension and motor irregularities in psychoneurotic patients under stress. J. Personality, 1955, 23, 391-406.
34. Miles, H.H.W., Barrabee, Edna L. & Finesinger, J.E. Evaluation of psychotherapy. Psychosom. Med., 1951, 13, 83-105.
35. Moruzzi, G. & Magoun, H.W. Brain stem reticular formation and activation of the EEG. EEG Clin. Neurophysiol., 1949, 1, 455-473.

36. Selye, H. Stress. Montreal: Acta, 1950.
37. Stennett, R.G. The arousal continuum. J. exp. Psychol. (in press).
38. Stennett, R.G. The relationship of alpha amplitude to the level of palmar conductance. EEG Clin. Neurophysiol., 1957, 9, 131-138.
39. Surwillo, W.W. Psychological factors in muscle-action potentials: EMG gradients. J. exp. Psychol., 1956, 52, 263-272.
40. Taylor, Janet, A. A personality scale of manifest anxiety. J. abn. soc. Psychol., 1953, 48, 285-290.
41. Tyhurst, J.S. & Richman, A. Clinical experience with psychiatric patients on reserpine--preliminary impressions. Canad. Med. Assoc. J., 1955, 72, 458-459.
42. Wallerstein, H. An electromyographic study of attentive listening. Canad. J. Psychol., 1954, 8, 228-238.

Footnotes

1. This paper reviews work which was supported by the Medical Research and Development Division, Office of the Surgeon General, Department of the U.S. Army, under Contract No. DA 49-007-MD-626, by Defence Research Board Grant No. 9425-04 (Canada), and by Grant No. A.P. 29 from the National Research Council of Canada.
2. The author is indebted to Drs. A.K. Bartoshuk, D. Bindra, F.R. Brush, D.E. Cameron, D.O. Hebb, and R.G. Stennett for criticizing earlier drafts of this paper.
3. Stennett (38) has found that the relationship of alpha amplitude to arousal level is nonlinear. On the lower end of the arousal continuum, the relationship is positive, such that raising arousal leads to increasing alpha amplitude; but past the middle range of arousal, the relationship becomes inverse. This latter function is the better known one.
4. Recent findings, although supporting Eccles' main conclusions, suggest that the phenomenon may be somewhat more complex than he originally supposed. The observations of Kuffler and Eyzaguirre (22) on inhibition of stretch receptor organs in crustaceans indicate that the polarity of the "inhibitory potential" varies with the state of the cell. When the cell is depolarized, an inhibitory volley causes polarization; when the cell is resting an inhibitory volley causes depolarization.

Robert B. Malmö

-28-

5. The reader will recall that in our physiological studies of psychiatric patients we attempted to avoid experimental situations which had special meanings for particular individuals. In an earlier section of this paper we referred to these situations as "nonspecific." We assume that an anxiety-prone individual, before he actually develops the pathological state (and after he recovers from it) will not show higher arousal levels in such "nonspecific" stimulating situations. The stimulating situations referred to as producing quite different levels of physiological reaction in different persons are, of course, what we called "specific" in the earlier section of this paper.

6. On the surface, this appears incongruous with the notion of weakened inhibition. However, we may account for this constrained appearance of inhibition by suggesting the substitution of less efficient mechanisms of inhibition for the one which has suffered impairment. It may be, for example, that anxiety-patients compensate for weakened autonomic mechanisms by calling on voluntary motor mechanisms (i.e. the pyramidal motor system). For example, in the absence of sufficient control from autonomic inhibitory mechanisms, the anxiety-patient may avoid loss of motor control through co-contraction of antagonistic muscles.

Robert B. Malmö

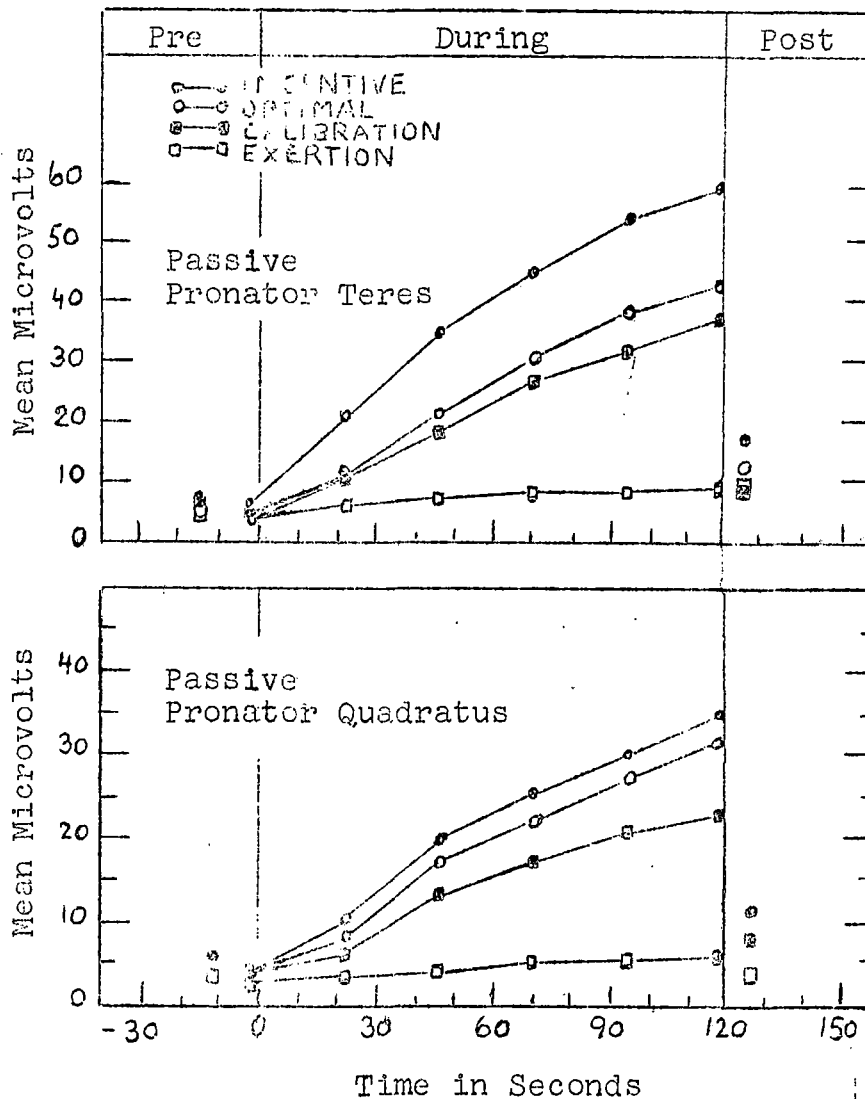


Fig. 2. Graphs from Stennett's experiment (37) showing mean EMG gradients obtained under conditions varying in degree of incentive. Steepness of gradient varies directly with degree of incentive. N = 31.

Robert B. Malmö

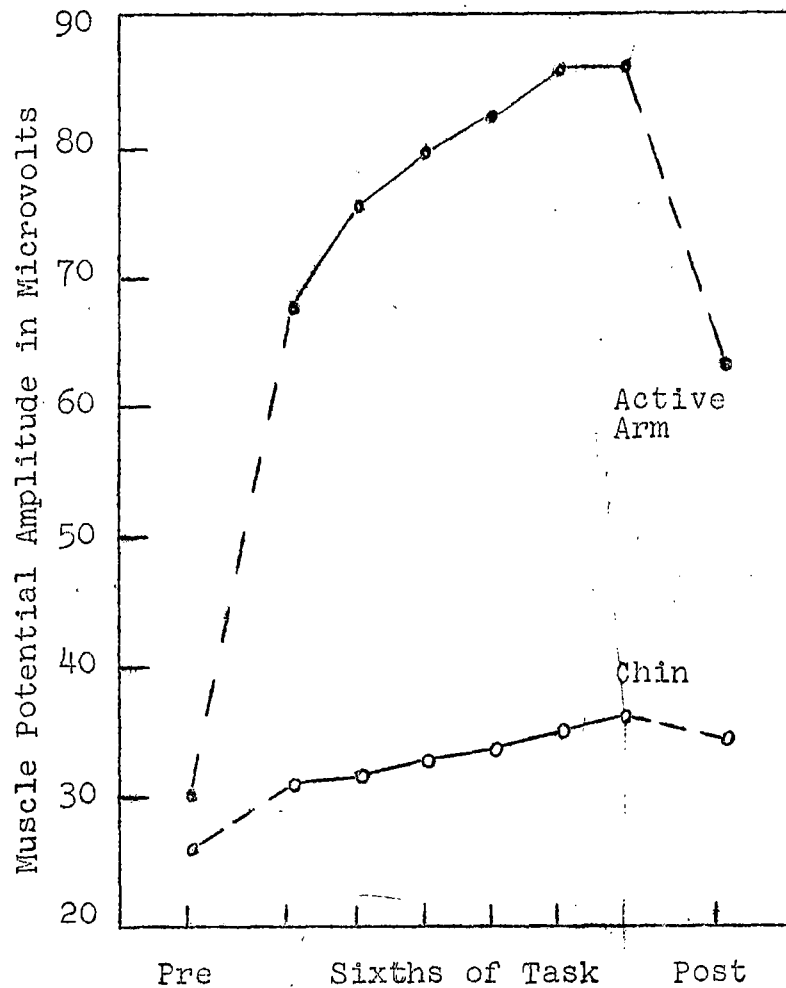


Fig. 1. Graphs showing mean EMG gradients in Bartoshuk's experiment (1). Note that gradient was also obtained from chin lead which records from muscles of speech. N=17.