SOME MOTOR ASPECTS OF SCHIZOPHRENIA: AN EMG STUDY

GEORGE B. WHATMORE, M.D., PH.D.,[1] AND RICHARD M. ELLIS, JR., B.S. IN E.E.[2]

We cannot get an accurate picture of the neurophysiology of neurosis and psychosis unless we include in our efforts a study of their motor components. A fact often overlooked is that motor activity within the central nervous system is intimately tied up with sensation, emotion, and thinking. There are no boundaries to separate these activities. An understanding of the neurophysiology of any of these, as well as their deviations in illness, must therefore include a description of the motor component. Present evidence and current thinking of numerous investigators favor the view that motor activity is more than just an end-product of psychic activity (1, 2, 4, 12, 18, 19, 20, 25, 26, 27, 28, 30, 31, 32, 33).

Compared to the neuroses and psychosomatic disorders, schizophrenia has received little study from a motor standpoint. Measurements of overt movement as well as of electromyographic activity during subjection of patients to certain stress situations have demonstrated greater degrees of motor reaction in schizophrenics than in control subjects (22, 24). EMG studies of the speech musculature in schizophrenics, combined with other observations, led Gould to the conclusion that auditory hallucinations were due to a motor disturbance of the speech mechanism rather than to a disturbance of perception (8, 9).

The studies to be reported in this paper[3] deal with electromyographic measurements of the low levels of residual motor activity found in persons endeavoring to rest and relax as completely as possible in a quiet comfortable environment. Residual motor activity is for the most part invisible to the naked eye.

APPARATUS

In order to study these low levels of resting activity in the motor portion of the nervous system by electromyographic methods, it was necessary to design and construct an instrument that would give suitable readings. Jacobson and associates have previously described an integrating neurovoltmeter for recording such activity (13, 14). Our instrument was patterned after theirs but is different in certain significant respects.

The instrumentation requirements of this investigation were primarily to pick up, amplify, and indicate the muscle action potentials located on the surface of the skin. One read-out was to be an indication of the instantaneous level of activity. In addition, an integrator must accumulate the potential for a definite period of time so that it may be recorded periodically.

The weakest electrical signal that can be detected is always limited by the noise level in the first stage of the amplifier. As we were interested in measuring voltages at as low a level as possible, it was of the greatest importance to design a pre-amplifier with great care.

Thermal noise in the first vacuum tube may be considered the only source of noise over which we have no control. Other sources of noise are, of course, the same as experienced in all EEG and EMG recording, such as 60 cycle power line pick-up. Standard techniques for eliminating this interference were carefully observed.

To maintain the most favorable signal to noise ratio, the frequency response of the amplifier was restricted to what was found to be an optimum bandwidth. This is indicated in the block diagram of Figure 1 as a filter. The frequency response of the entire system is from 120 cps to 300 cps at the 3db points with a low end slope of 12 db per

[1] 509 Medical-Dental Bldg., Seattle, Wash. This study was conducted at Eastern State Hospital, Medical Lake, Wash.

[2] Electrical Engineer, Seattle Development Laboratory, Minneapolis Honeywell Regulator Co., Seattle, Washington.

[3] Gratitude is expressed to Mr. Jack Kirk for his careful work in processing the many graphs and carrying out the necessary calculations, and to Marga Burgoon, Mae Carey, Mildred Chitwood, Jessie Harris, and Ethel Sanders for their valuable assistance in preparing the subjects for EMGs. Thanks are also expressed to Mr. John Dudley and Mr. Dean Franklin for their assistance in the design and construction of equipment.

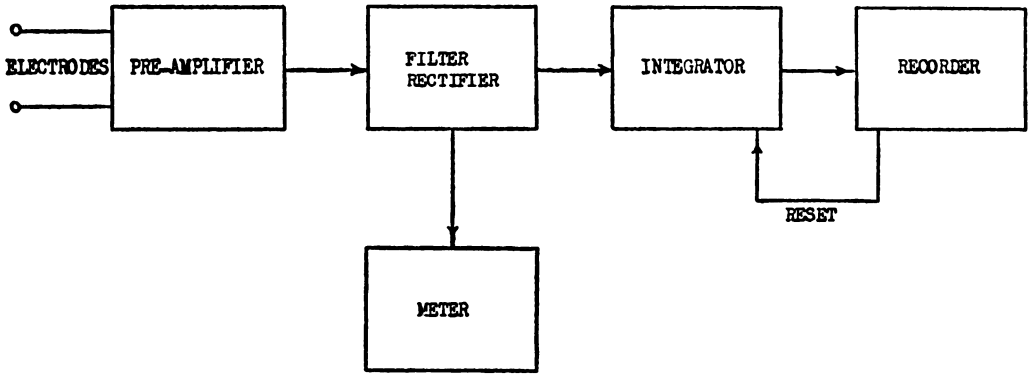

FIG. 1.—Block Diagram of Electromyograph.

octave and a high end slope of 8 db per octave.

In order to drive a d.c. meter the signal is full wave rectified. A common back-bias technique in the meter circuit enables the meter to be set at zero when only the amplifier noise is present, which is done with the electrode leads shorted together. This procedure amounts to a further reduction of the noise level. The noise level with no back-biasing is about $\frac{1}{3}$ microvolt equivalent at the input, and the effective noise level remaining after back-biasing of the meter is about 0.1 microvolt.

An R-C integrator was used with a linear range of more than a minute. The recorder, which is a multiple point printer, samples and records the potential on the integrator, and immediately thereafter trips a reset relay which shorts out the integrating capacitors.

Arbitrary periods of one minute were used as the integration time. Several signal channels may be used with one recorder, and the present work was done with 4 channels, one recording every 15 seconds.

Four amplifier channels and the recorder were mounted in a 19-inch relay rack, except for the 4 read-out meters which were in individual containers to be located in strategic places for monitoring purposes.

METHOD

All subjects lay in the supine position on a bed in a semi-darkened relatively quiet room. They were given the instruction "Rest and relax as completely as possible and leave your eyes closed."

Continuous electromyographic readings of motor activity were taken simultaneously for 30 minutes from leg, forearm, jaw, and forehead regions. Surface pick-up electrodes were arranged in pairs (since the amplifiers were double-ended) and were placed over antagonistic muscles. Sanborn EKG paste was worked into the skin until the d.c. resistance between electrodes was 2000 ohms or less, and this resistance was measured again at the end of each period to be sure it had not changed. A ground electrode was placed a few inches away from each pair of pick-up electrodes. For the leg one Sanborn EKG electrode (measuring 3.2 cm. by 5.1 cm.) was placed over the anterior tibial muscle and the other over the gastrocnemius of the left leg. For the forearm one EKG electrode was placed over the extensor surface and the other over the flexor surface of the right forearm. To obtain readings from antagonistic muscles acting on the mandible an EKG electrode was placed over the left masseter muscle and a silver electrode measuring 1.8 cm. in diameter was placed in the submental region. Anatomical considerations as well as experimental tests indicated that this pair of electrodes recorded not only from the jaw-closing and jaw-opening muscles but also from some of the muscles controlling the tongue. To obtain readings from the muscles involved in frowning and raising the eyebrows one of the small silver electrodes was placed over the left corrugator muscle and the other in the midline over the

frontalis muscle. With electrodes paired in this fashion, activity in one, the other, or both of a pair of antagonistic muscles would give readings and it was not considered important to know which of the two muscles had been active. At the end of the test period the subjects were questioned to determine various subjective experiences such as whether they slept, whether they were in any pain or discomfort, whether they felt calm and relaxed or were nervous and restless, whether they were fearful, and what they could recall having thought about during the test period.

A group of 21 schizophrenic patients was compared to a group of 10 control subjects. The requirements for the schizophrenic group were that they have a clear-cut unquestionable diagnosis and that they show little or no sign of deterioration. No selections or calculations on the basis of type of schizophrenia were made. The patients varied from 21 years to 49 years of age with a median age of 35 years. The control group was selected from hospital personnel and the only requirement was that each subject should not ever have had a mental illness (psychosis). The control subjects varied from 23 years to 42 years of age with a median age of 33 years. A supplementary subdivision of the control group into those persons relatively free from functional nervous symptoms and those troubled somewhat with functional complaints was also made. We say "relatively free from functional symptoms" for the one subdivision because it probably would not be possible to find a person completely free from functional complaints at all times.

A sample EMG is given in Figure 2 and will be used to show the method for processing the data contained in each EMG. One was selected in which the various channels do not cross each other because it is easier to see. Each point on the graph is the integrated level of motor activity for a period of one minute, which is approximately the mean value of the motor activity for that minute. This value is expressed in "units of motor activity" although with extra calculations it could have been expressed in microvolts rms. The "unit of motor activity" is an arbitrary unit obtained by dividing the maximum range of the graph paper on the point printer into 100 subdivisions. For those readers who prefer these values expressed in microvolts the approximate conversion figures are 25 units of motor activity equal 1 microvolt rms., 55 units of motor activity equal 2 microvolts, and 85

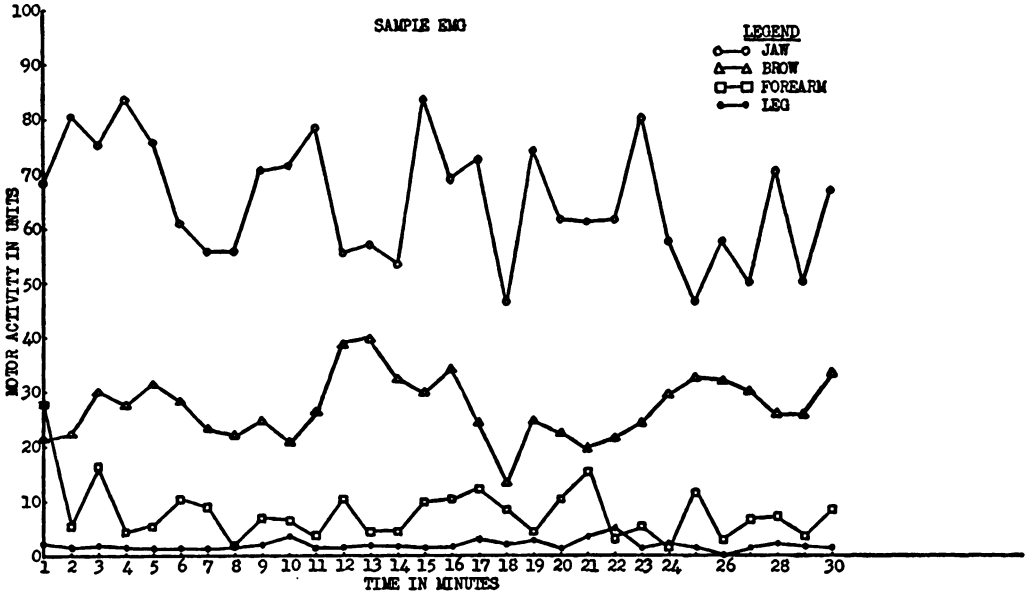

FIG. 2.—Sample EMG. One was selected in which the various channels do not cross each other. Each point on the graph is the integrated quantity of motor activity for a period of one minute.

units equal 3 microvolts. The instrument was calibrated both at the start of the period and at the end with a 1 microvolt rms signal at 200 cycles per second and at the end of the period with 2 and 3 microvolt signals as well. From the 30 points printed on the graph for each muscle area, a mean value was calculated. Values going off the top of the graph were assigned a value of 100 units since this is the highest point on the graph. From the mean values for each of the 4 muscle areas a grand mean was calculated. This grand mean is therefore a single number representing the mean quantity of motor activity for the entire 30 minute period.

For both the control group and the patient group, various mean values were calculated, as will be shown in detail in the section on results. The significance of each of these means was determined in accordance with the method described by Fisher(7). For the reader not familiar with the concept of the significance of a mean, this determination basically is answering the question "On the basis of the distribution of samples already obtained, what is the probability that a larger number of samples will give a different mean?". This probability can be calculated accurately and makes it possible to determine when a population sample is large enough to be representative of the population in question. In addition to the significance of means, the significance of the difference between the means of the control and patient groups was also calculated.

RESULTS

Table 1 shows the composition and various mean values for the control group. Table 2 shows corresponding information for the schizophrenic group. Figure 3 shows composite EMG's for the control group and the patient group. Each point on these graphs is the mean of the entire group for that particular minute and the points are plotted minute by minute for a 30 minute period. Table 3 gives a quantitative comparison of the control group and the patient group, and shows the significance of the differences between them. For both the control group and the patient group, the means of the grand means are highly significant ($P = <0.001$). The differences between the control group and the patient group with respect to the grand mean and the 4 individual muscle area means are highly significant ($P = <0.001$ for grand mean, forehead, and jaw; $P = <0.01$ for forearm and leg). It is remarkable to us that these 5 differences are quantitatively so nearly alike. They range from 33.9 to 36.4 units of motor activity. The difference in grand means was 35.0 units, the control group giving 19.9 units of motor activity and the patient group 54.9.

Another point to be emphasized is that the rank order of the 4 muscle areas is the same for both control and patient groups but the patient group is set at a higher level. This can be seen most clearly in Figure 3 which gives composite EMG's for the control group and the patient group.

TABLE 1

MEAN VALUES OF MOTOR ACTIVITY AND SIGNIFICANCE OF THE MEANS FOR THE CONTROL GROUP

Subject	Brow mean (Units)	Jaw mean (Units)	Forearm mean (Units)	Leg mean (Units)	Grand mean (Units)
1. F.L.	17.39	35.47	1.33	0.40	13.6
2. P.A.	25.61	26.56	0.28	2.98	13.9
3. C.B.	23.58	24.17	0.38	2.66	12.7
4. R.P.	11.95	18.48	1.06	6.66	9.5
5. J.F.	11.92	28.61	1.74	3.13	11.4
6. F.W.	40.84	38.12	12.31	6.08	24.3
7. J.H.	35.70	38.37	13.66	6.80	23.6
8. M.H.	33.56	35.22	50.29	12.30	32.8
9. L.S.	15.07	72.99	2.75	10.74	25.4
10. N.B.	47.50	67.45	10.91	2.21	32.0
Group Mean =	26.3	38.5	9.5	5.4	19.9
P Value =	< 0.001	< 0.001	< 0.1	< 0.01	< 0.001

TABLE 2

MEAN VALUES OF MOTOR ACTIVITY AND SIGNIFICANCE OF THE MEANS FOR THE SCHIZOPHRENIC PATIENT GROUP

Patient	Brow mean (Units)	Jaw mean (Units)	Forearm mean (Units)	Leg mean (Units)	Grand mean (Units)
1. M.P.	88.02	92.83	49.45	49.56	70.0
2. E.M.	76.34	100.00	55.96	84.74	79.3
3. V.H.	58.15	39.03	5.38	2.18	26.2
4. G.S.	40.30	43.06	13.89	3.17	25.1
5. M.J.	59.87	54.71	3.48	0.06	29.5
6. W.N.	26.19	40.02	12.63	34.19	28.3
7. E.M.	28.49	63.10	99.00	57.79	62.1
8. M.B.	60.78	73.85	98.86	86.67	80.0
9. J.M.	61.91	93.08	94.30	96.95	86.6
10. L.N.	58.20	64.20	8.00	6.60	34.3
11. B.P.	50.40	74.87	54.29	30.26	52.5
12. V.R.	76.99	99.95	67.43	83.29	81.9
13. L.S.	41.86	75.73	3.85	18.68	35.0
14. B.N.	78.73	76.58	8.68	2.38	41.6
15. M.O.	93.30	60.16	10.35	10.36	43.5
16. A.C.	43.58	91.09	15.01	78.71	57.1
17. M.R.	38.33	57.61	29.85	3.01	32.2
18. M.M.	82.74	96.96	97.58	67.52	86.2
19. D.T.	53.78	100.00	59.06	13.00	56.5
20. R.R.	47.46	78.48	97.67	100.00	80.9
21. E.Z.	99.29	98.10	56.56	4.89	64.7
Group Mean =	60.2	74.9	44.8	39.7	54.9
P Value =	< 0.001	< 0.001	< 0.001	< 0.001	< 0.001

Only 5 grand means for the patient group overlap the range of the control group. They lie in the upper portion of the control range and none of them reach down to the mean value for the control group.

An interesting sidelight is the result obtained when the control group, which is composed of hospital personnel who have never had a mental illness, is broken down into two subgroups. One subgroup is made up of those persons relatively free from functional symptoms of any kind and the other subgroup is composed of those who are troubled somewhat with functional complaints. The former included 6 subjects and gave a grand mean of 15.5 units of motor activity. The latter was composed of 4 subjects and gave a grand mean of 26.5 units of motor activity. Even though there are only 6 subjects in one subgroup and 4 in the other, the difference between their means is significant ($P = < 0.05$).

TABLE 3

COMPARISON OF CONTROL GROUP AND SCHIZOPHRENIC PATIENT GROUP SHOWING THE SIGNIFICANCE OF THE DIFFERENCES

	Control group (Units)	Schizophrenic patient group (Units)	Difference (Units)	Significance of difference
Brow mean ...	26.3	60.2	33.9	P = < 0.001
Jaw mean	38.5	74.9	36.4	P = < 0.001
Forearm mean..	9.5	44.8	35.3	P = < 0.01
Leg mean	5.4	39.7	34.3	P = < 0.01
Grand mean =	19.9	54.9	35.0	P = < 0.001

DISCUSSION

The activity we are recording in these experiments is most likely accompanied by increased activity in efferent neurons of the motor cortex and/or premotor cortex. Whether this hyperactivity of pyramidal cells plays an etiological role in the development of schizophrenia or is a consequence of the disorder is a very important question. Although we often think of motor activity in mental illness as being secondary to psychic processes we must not discard the reverse possibility prematurely. One reason for caution comes from research indicating the importance of motor states in the mainte-

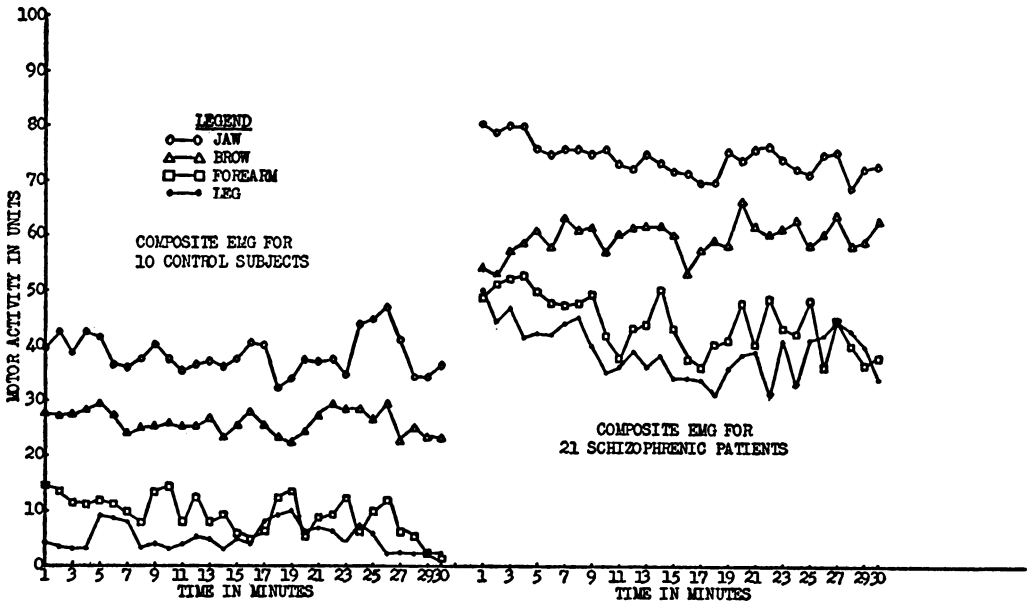

FIG. 3.—Composite EMG's for control group and patient group. Each point is the mean of the entire group for that particular minute.

nance of consciousness and the waking state (19, 28).

Hyperactivity of pyramidal cells can have widespread influence on the functioning of the cerebral cortex as well as subcortical structures. This influence would be exerted through the many collateral fibers given off at various levels along the descending axones and through afferent flow from proprioceptors activated when muscles contract. Many other neurons would thus become either hyperexcited or excessively inhibited depending on the nature of their connections with the hyperactive pyramidal cells.

There are indications from numerous sources that the act of thinking itself is in part a motor act involving pyramidal cells(1, 12, 26, 27, 30, 32, 33), and that feelings and emotion likewise are intimately tied up with motor states(3, 12). If this is the case it would not be hard to understand how hyperactivity of this motor system might lead to disturbances of thinking and of emotion. The point to be emphasized is that this possibility must be kept in mind and we must not jump to conclusions hastily.

This state of hyperactivity of the motor portion of the nervous system, whether localized or generalized, intermittent or continuous, static or phasic, overt or invisible, has been referred to in the literature as neuromuscular hypertension and also as neuromuscular hyperkinesis(12, 15, 16). Perhaps the expression "hyperponesis" would be even more descriptive. This word comes from the Greek "hyper" meaning excessive, and "ponesis" meaning exertion.

There is growing interest in this condition and in determining its exact position in the neurophysiology of functional disorders. More attention has been given to its relation to the neuroses and psychosomatic disorders than to its relation to schizophrenia. Malmo, Shagass, and Davis(23, 24, 29), consider excessive muscular tension to be probably of considerable importance in the production of symptoms in patients with functional disorders. They have reported instances in which symptoms of a "tired feeling" in the head and head discomfort were preceded by a sustained burst of high-level electromyographic activity in the frontalis muscle or in neck muscles. Wolf(34) found sustained contraction of the diaphragm to underlie a common type of functional dyspnea and precordial pain. He found it could also produce occlusion of the lower end of the esophagus and could do this before the con-

tractile state of the diaphragm was sufficient to produce respiratory difficulty. Kaufman (17) refers to a similar mechanism in discussing the syndrome of spontaneous hypoventilation. Holmes and Wolff(11) refer to a pattern of motor behavior which they call the "on guard" pattern, and state that when it is utilized as a way of life it may place an intolerable burden on the individual's emotional and physical equipment. Haugen(10) referred to this same motor pattern calling it a bracing reaction and expressed the view that without this bracing reaction no neurosis can develop and unless this bracing reaction can be permanently decreased or eliminated the patient remains vulnerable to an exacerbation of his illness. There are many other such reports too numerous to mention(4, 5, 6, 20, 21, 35). Jacobson has proposed neuromuscular hypertension as a fundamental disorder underlying some of the psychoneuroses and certain psychosomatic disorders and he considers this neuromuscular hypertension to be so fundamental that unless it is overcome the patient tends to remain ill, no matter what the therapy(12, 15, 16). He has evolved methods of therapy which attack directly the exaggerated motor state by a re-educative process.

Studies of motor activity in schizophrenia reported to date have dealt with overt movement or the high levels of electromyographic activity present while patients are carrying out some prescribed activity or are being subjected to a stress situation(22, 24). It is important to emphasize the difference between these studies and the ones reported here. In our studies the motor activity measured was that present while the subjects were lying at rest and was for the most part invisible to the naked eye. Equipment capable of measuring reliably small differences at these low levels of activity had to be constructed before these measurements could be made.

Nevertheless our findings complement those of Malmo and coworkers(22, 24). Whereas their schizophrenic patients responded to certain types of stress with excessive motor activity, our patients showed excessive motor activity while at rest.

Summary

1. The value of studying motor activity in mental illness should not be overlooked. Motor activity within the central nervous system is intimately tied up with sensation, emotion, and thinking. An understanding of the neurophysiology of any one of these, as well as their deviations in illness, must include a description of the motor component.

2. Multi-channel electromyographic measurements on 21 schizophrenic patients and 10 control subjects are here reported. An electromyograph giving both integrated and instantaneous readings and capable of measuring minute amounts of motor activity was employed. Residual motor activity was recorded while the subjects endeavored to relax as completely as possible in the supine position. This residual motor activity is for the most part invisible to the naked eye.

Records were taken simultaneously from 4 muscle areas, namely forehead, jaw, forearm, and leg, for 30-minute periods. The patients exhibit higher levels of motor activity in all 4 muscle areas and the differences between these and the values for the control group are highly significant ($P = <0.001$ for forehead and jaw, and $P = <0.01$ for forearm and leg). Quantitatively these differences are remarkably similar for each muscle area. The grand mean for the patient group is 54.9 units of motor activity and for the control group 19.9 units.

3. This exaggerated motor activity is most likely accompanied by increased activity in efferent neurons of the motor cortex and/or premotor cortex. Such increased pyramidal cell activity could have widespread influence on the functioning of the cerebral cortex, as well as subcortical structures, through the agency of the many collateral fibers given off by these neurons. Since there is evidence that both thinking and emotion have motor components, it is reasonable that hyperactivity in the motor system might lead to disturbances of thinking and emotion. The possibility of motor system hyperactivity playing an etiological role of some type in the onset of schizophrenia should be kept in mind.

4. The term "hyperponesis" is suggested to refer to exaggerated activity within the motor portion of the central nervous system. This exaggerated activity may be localized to a portion of the motor system or generalized to include the whole motor system, it may be intermittent or continuous, static or phasic, overt or invisible.

BIBLIOGRAPHY

1. Allers, R., and Scheminzky, F.: Pflug. Arch. f. Physiologie, **212**: 169, 1926.
2. Bender, Lauretta: Psychopathology of children with organic brain disorders. Springfield, Ill.: Charles C Thomas, 1956.
3. Bull, Nina: The attitude theory of emotion. New York: Nerv. & Ment. Dis. Monographs, 1951.
4. Dixon, H. H., Dickel, H. A., Coen, R. A., and Haugen, G. B.: Western J. Surg., Obs., & Gyn., **58**: 667, 1950.
5. Dixon, H. H., Dickel, H. A., Shanklin, J. G., Peterson, R. D., and West, E. S.: Western J. Surg., Obs., & Gyn., **62**: 338, 1954.
6. Dixon, H. H., Peterson, R. D., Dickel, H. A., Jones, C. H., and West, E. S.: Western J. Surg., Obs., & Gyn., **60**: 327, 1952.
7. Fisher, R. A.: Statistical methods for research workers. New York: Hafner Publishing Co. Inc., 1954.
8. Gould, L. N.: J. Nerv. & Ment. Dis., **109**: 418, 1949.
9. Gould, L. N.: Am. J. Psychiat., **107**: 110, 1950.
10. Haugen, G. B.: A neurophysiologic theory of the neuroses. Paper presented at North Pacific Society of Neurology & Psychiatry. April, 1955.
11. Holmes, T. H., and Wolff, H. G.: Psychosom. Med., **14**: 18, 1952.
12. Jacobson, Edmund: Progressive relaxation. 2nd Ed., Chicago, Ill.: Univ. of Chicago Press, 1938.
13. Jacobson, Edmund: Rev. Scientific Instruments, **11**: 415, 1940.
14. Jacobson, Edmund: Am. J. Psychiat., **97**: 513, 1940.
15. Jacobson, Edmund: Am. J. Psychiat., **98**: 219, 1941.
16. Jacobson, Edmund: Am. J. Psychol., **68**: 549, 1955.
17. Kaufman, William: Mississippi Valley Med. J., **73**: 1951.
18. King, H. E: Psychomotor aspects of mental disease. Cambridge, Mass.: Harvard University Press. 1954.
19. Kleitman, Nathaniel: Sleep and wakefulness. Chicago, Ill.: Univ. of Chicago Press, 1939.
20. Kretschmer, Ernst: Psychotherapeutische Studien. Stuttgart, Georg Thieme Verlag., 1949.
21. Lundervold, Arne: J. Nerv. & Ment. Dis., **115**: 512, 1952.
22. Malmo, R. B., and Shagass, C.: Psychosom. Med., **11**: 9, 1949.
23. Malmo, R. B., Shagass, C., and Davis, F. H.: Res. Publ. Ass. Nerv. & Ment. Dis., **29**: 231, 1950.
24. Malmo, R. B., Shagass, C., and Davis, J. F.: J. Clin. & Exper. Psychopath., **12**: 45, 1951.
25. Max, L. W.: J. Gen. Psychol., **11**: 112, 1934.
26. Max, L. W.: J. Comp. Psychol., **19**: 469, 1935.
27. Max, L. W.: J. Comp. Psychol., **24**: 301, 1937.
28. Roger, A., Rossi, G. F., and Zirondoli, A.: EEG & Clin. Neurophysiol., **8**: 1, 1956.
29. Shagass, C., and Malmo, R. B.: Psychosom. Med., **16**: 295, 1954.
30. Shaw, W. A.: Arch. Psychol., **35**: 5, 1940.
31. Sperry, R. W.: Am. Scientist, **40**: 291, 1952.
32. Totten, E.: Compar. Psychol. Monographs, **11**: No. 3, Serial No. 53, 1935.
33. Vinacke, W. E.: The Psychology of Thinking. New York: McGraw-Hill Book Company, Inc., 1952.
34. Wolf, S.: J. Clin. Investig., **26**: 1201, 1947.
35. Wolpe, Joseph: Arch. Neurol. & Psychiat., **72**: 205, 1954.