8792 C

Modification of the Electromyogram of the Simple Reaction by Cerebellar Lesions.

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When simple quick voluntary movements are studied by a combination of action potential and gross movement technics, the contraction of the muscles, and the gross movements occur in a fairly definite sequence. Though finer details vary widely among normals, there are fairly constant gross characteristics.[1] If antagonistic striated muscles are recorded, it will be seen that they will both show potentials within an average time of about 9σ from one another. The contraction potentials of the muscle which will shorten in the response occur slightly ahead of its antagonist. The acting muscle will also contract more strongly than the antagonistic muscle though this difference is not always easily observable on action potential records. At a period of about 60 to 100σ after the appearance of the initial contraction in the muscle, the gross movement begins. (Fig. 1.) These statements are not in accord with the

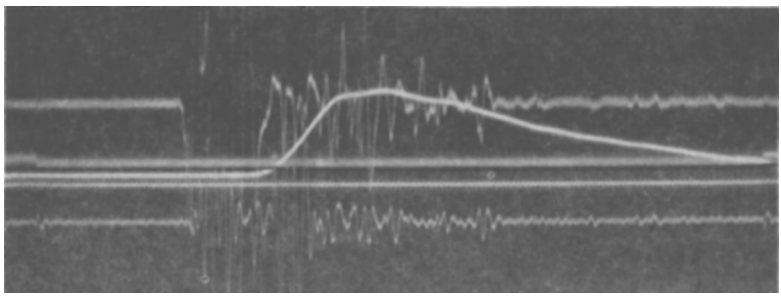

FIG. 1.

Simple extension of the left elbow in a normal. From above downward: Triceps lead, signal line showing stimulus point, gross movement record, 2σ time line, and biceps lead at the bottom.

current concepts of the lengthening reaction in antagonistic muscles, but they do substantiate the results of Tilney and Pike[2] who studied this question mainly from the standpoint of muscle volume changes

1 Hathaway, S. R., and Sisson, E. D., *J. Exp. Psycho.* (in press).

2 Tilney, F., and Pike, F. H., *Arch. Neur. and Psychiat.*, 1925, **13**, 289.

in voluntary movement. Weisenburg[3] has said, "If there is a lesion in any part of the cerebellar apparatus there is necessarily produced a disturbance or destruction of the association between agonists and antagonists, and the resulting movement must necessarily be incoordinate or asynergic."

Since we had studied the coordination of simple extension or flexion by the paired action of the triceps and biceps brachii, and to a lesser degree the quadriceps femoris and hamstring group, it seemed advisable to study this response in cases with cerebellar lesions.

The subject was placed in a comfortable position with the forearm supported so that it could rotate freely in a horizontal plane about the elbow. He was instructed to extend (or flex) the forearm as quickly as he could after the appearance of a light which was used for a stimulus. The same technic was used for the extension of the lower leg at the knee joint except that in this case no support was used, the lower leg being simply kicked forward. These movements were practiced before the pictures were actually taken. The records show the action potentials in the muscles involved, the gross movement, the point of appearance of the signal, and a time line for reading the time differences involved. The slope of the gross movement line is an approximate value for the force of the movement.

We also made several records of the cerebellar rebound phenomenon. The recording technics were essentially the same in this case, the subject being instructed to hold with considerable force against

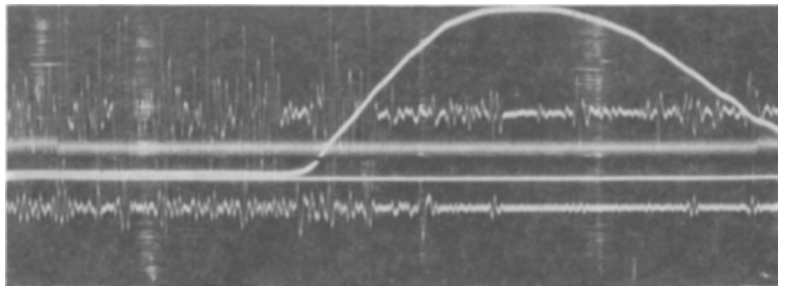

FIG. 2.

Cerebellar rebound in a normal. First pull against triceps. From above downward: Triceps lead, 5σ time line, gross movement record, and biceps lead at bottom.

[3] Proc. of the Assn. for Research in Nervous and Ment. Disease. VI. The Cerebellum, 1929, Baltimore.

a constant pull on the forearm. This pull was released with the appearance of the signal, allowing the subject's forearm to swing suddenly outward or inward as the case might be. (Fig. 2.)

We are reporting at this time the findings in two cases of cerebellar involvement. The first was a fairly advanced case of Marie's cerebellar ataxia in a man, age 35. He is at present unable to walk without support. The cerebellar symptoms are marked, and earlier in our observations were the only symptoms and signs from the nervous system. During the past year, there have been superimposed on the original syndrome both increasing deep reflexes and decreasing deep sensibility in the extremities. This subject's records show several definite abnormalities. The most obvious difference from the normal occurs in the antagonistic muscle which, in contradistinction to the normal, is but little involved in the reaction. This subject, then, shows in these movements a practically unopposed contraction of the acting muscle (Fig. 3). This is true both

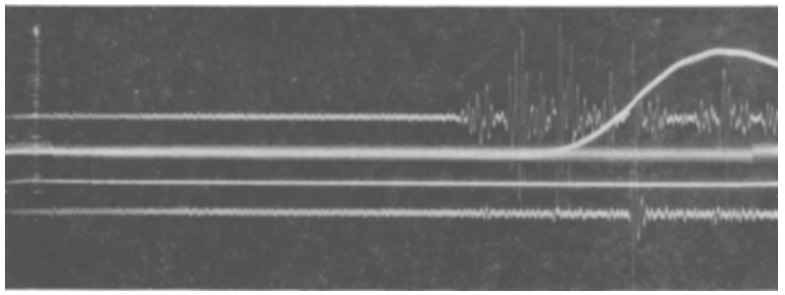

FIG. 3.

Simple extension of the left elbow in a case of Marie's cerebellar ataxia. From above downward: Triceps lead, superimposed gross movement and 5σ time lines with break in time line marking stimulus, and biceps lead at bottom.

in the arm and in the thigh. The reaction time is much longer than average and the gross movement tends to be somewhat wavering; the patient was nevertheless very cooperative and obviously doing his best.

Records of the cerebellar rebound show the antagonistic muscle coming in strongly as a reflex response, possibly myotatic, to the extension of that muscle which occurs when the arm is released. This action of the antagonist not only suddenly stops the outward movement of the released forearm, but brings it back so rapidly that the opposite type of contraction occurs in the other muscle producing a second extension and the typical rebound phenomena. Normal rec-

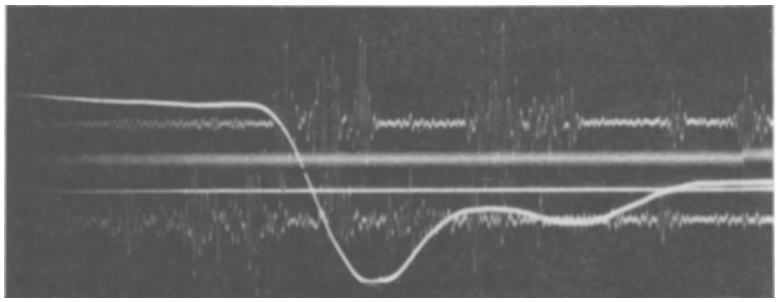

FIG. 4.
Cerebellar rebound in the left arm of a case of Marie's cerebellar ataxia. First pull against biceps. From above downward: Gross movement record, triceps lead, 5σ time line, and biceps lead at bottom.

ords of this same reaction do not show this interplay of antagonistic muscles (Figs. 2 and 4).

In view of the definite findings in this case, it seemed advisable to find a case in which the loss of cerebellar tissue would be much less marked and in which there would be no other demonstrable sensory and motor tract signs.

We selected from the clinic a 12-year-old girl, who, about 2 years previously had had an astrocytoma removed from the left lobe of the cerebellum. She was originally markedly ataxic but at present is neurologically negative. Records in this case (Fig. 5) were much more nearly normal. Reactions were within normal reaction time and in general appeared to the observer to be normal. When the records are closely examined, however, the antagonists are found to be involved in the reaction much less than the normal and the

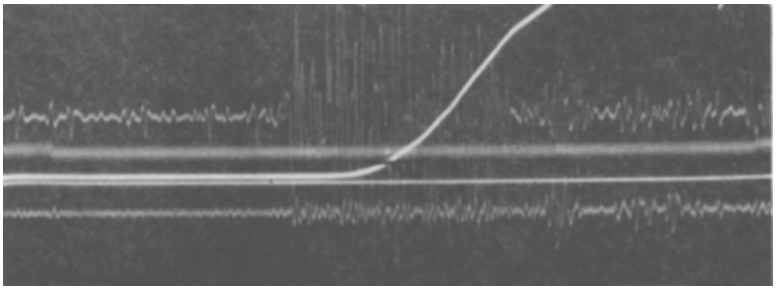

FIG. 5.
Simple extension of left elbow in a case with a small cerebellar lesion. From above downward: Triceps lead, 5σ time line with break showing stimulus point, gross movement record, and biceps lead at bottom. The preliminary tone in the triceps should be disregarded.

time between the appearance of contraction in the two muscles is usually about twice the normal time. This is particularly true in the left arm, which was originally the more ataxic. The records of cerebellar rebound were apparently normal.

As a check upon the possible sources of these disturbances especially regarding our first case, we obtained records from a moderately advanced uncomplicated tabetic patient who had complete loss of tendon pain, a much reduced muscle pain, and absent patellar reflexes. On this subject we obtained records from the left arm and leg. These records were not different from normal in any of the above points. This seems to eliminate the possibility that proprioceptors are determinants of activity of the antagonist in a simple reaction.

Though 2 cases of cerebellar disease do not constitute a satisfactory series, it is striking that they deviate in a similar way from the normal in recordings of simple reactions. Thus, instead of the nearly simultaneous contraction of agonist and antagonist which we have observed in normals, the cerebellar cases show a suppression of potentials in the antagonist. A tendency to increased reaction time is also notable. A generalization from these observations as to their applicability for the diagnosis of cerebellar involvement must obviously await the accumulation of more data.

8793 P

Rapid Procedure for the Quantitative Estimation of Antitrypsin.

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Gates,[1] Gilman and Cowgill,[2] and Pickford and Dorris,[3] have found that photographic film furnishes a useful substratum for the titration of proteolytic enzymes, since the gelatin layer, which is readily digested by pepsin or trypsin, is sufficiently uniform in thickness and consistency to permit quantitative tests. The release

1 Gates, F. L., PROC. SOC. EXP. BIOL. AND MED., 1926-27, **24**, 936.

2 Gilman, A., and Cowgill, G. R., *J. Biol. Chem.*, 1930, **88**, 743.

3 Pickford, G. E., and Dorris, F., *Science*, 1934, **80**, 317.