Unloading and shortening reactions in Parkinson's disease¹

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SUMMARY The unloading reflex was tested on both sides of a patient with asymmetrical Parkinsonism. The motor activity after the silent period was significantly greater on the more affected side. The findings support the hypothesis that the response to unloading and the shortening reaction share a common mechanism which is exaggerated in Parkinsonism.

More than a century ago, Westphal (1877, 1880) discovered that skeletal muscle will sometimes contract in response to passive shortening. This phenomenon, which he called the "paradoxical muscle reaction," has been confirmed by many observers (Förster, 1921; Delmas-Marsalet, 1927; Samojloff and Kisseleff, 1927; Broman, 1949; Denny-Brown, 1960; Rondot and Scherrer, 1966; Burke *et al.*, 1971; Andrews and Burke, 1972; Andrews *et al.*, 1972, 1973) but has not found a niche in the textbooks of neurology. The response to shortening might well be neglected if it were only a bedside curiosity. However, Rondot and Metral (1973) have recently suggested that the shortening reaction may be important in the pathophysiology of Parkinson's disease, athetosis, and dystonia. Furthermore, there is reason to believe that it may also take part in the control of normal posture and movement.

Broman (1949) has described the shortening reaction as follows: "When a passive movement is performed, the passively shortened muscles exhibit a contraction which tends to maintain the passively induced position." That description suggests that the shortening reaction may be related to another form of response to passive shortening—the motor activity that follows the unloading of an isometrically contracted muscle (Alston *et al.*, 1967). When the load is removed, the limb is accelerated by the unopposed force of the muscular contraction. As the muscle begins to shorten, there is an electrical

"silent period" followed by a burst of EMG activity. At the time of this renewed activity, the limb is moving by inertia, that is, passively. Hence, the motor activity after the silent period may be viewed as a muscular response elicited by passive shortening. The present study was designed to test the hypothesis that the contraction after unloading and the shortening reaction are both produced by the same neural mechanism, one which may have an important role in the normal and abnormal physiology of movement.

The common mechanism hypothesis can be tested by studying the effects of disease processes on the two phenomena. If the shortening reaction and the contraction after unloading are both mediated by the same neural mechanism, a disease process that increases the former will also increase the latter. Since the shortening reaction is known to be exaggerated in Parkinsonism (Broman, 1949; Andrews *et al.*, 1972), the hypothesis predicts that the contraction after unloading will also be exaggerated. However, there are no "normal standards" for the magnitude of the response to unloading. To meet this problem, we have studied a patient with asymmetrical Parkinsonism, comparing responses between the sides with and without clinically evident extrapyramidal disease. In such a patient, our hypothesis predicts that the contraction after unloading will be greater on the side with more severe Parkinsonism.

Subject and methods

The subject of the experiment was a 64 year old, right handed man with Parkinson's disease. He estimated the duration of illness to be three or

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four years, with no history of encephalitis or other health problems. His chief complaints were inability to use the left hand, tremor of the left hand, and difficulty in walking. The tremor was present at rest and much increased by fatigue or stress. The gait disorder was attributed to stiffness of the left leg and episodic, uncontrollable dorsiflexion of the left ankle and toes. These involuntary contractions had a tendency to occur during the weightbearing phase of gait, and sometimes caused him to stumble or fall. He denied any difficulty in using the right hand or any change of his handwriting. Although the patient felt that his right side was normal, his wife stated that he sometimes became weak "all over." She added that his left shoes and slippers were deformed by the repeated dorsiflexions. The patient showed good response to his medications without dyskinetic movements. At the time of testing, his regimen was carbidopa 10 mg with L-dopa 100 mg six times a day and trihexyphenidyl 6 mg per day.

On examination, the patient displayed typical features of Parkinsonism including facial seborrhoea and a paucity of expression. A quivering voice and mild instability of stance were present, but there was no difficulty in initiating or changing movements. A characteristic tremor and rigidity were found to a much greater degree on the left than on the right side. During walking, the left foot alone demonstrated the slow dorsiflexion described above.

Westphal's shortening reaction was demonstrable in muscles of the left arm, forearm, and calf. Passive dorsiflexion of the ankle, for example, was accompanied by a visible and palpable contraction of muscles in the anterior tibial compartment. The patient was not able to inhibit these contractions when instructed to do so. On the right side there was also a slight tendency for contraction of muscles that were shortened passively, but this was easily eliminated by instructing the patient to relax. Apart from bilateral impairment of hearing, the remainder of the neurological examination was normal. In particular, there was no sign of upper motor neurone involvement.

Muscle action potentials were recorded by means of Beckman skin electrodes, used with Beckman electrode paste and adhesive collars. Paired electrodes were fastened over the right or left pectoral muscles, the interelectrode distance being about 50 mm. The action potentials were rectified and smoothed by means of a special filter with the period of integration set at 44 ms (Garland *et al.*, 1972).

Movement of the hand was recorded by means of a 750 mm aluminium rod which was hung at

arm's length in front of the shoulder being tested. The upper end of this rod was coupled to an axle which rotated as the rod swung to the right or left. The axle turned a variable capacitor, whose output was proportional to the angular velocity of the handle. The signal representing hand velocity was recorded on magnetic tape, simultaneously with the rectified and filtered EMG potentials.

The external load was supplied by a coil spring which extended horizontally from the handle toward a steel plate. At the start of each trial, the steel plate was held fast by an electromagnet. When the handle was pulled to the designated starting position, the spring exerted a force of about 4.5 kg (10 pounds), opposing the force of muscular contraction. Upon release of the magnet, the muscles were unopposed, and the arm swung medially.

For each test, the subject was seated facing the handle which he grasped firmly and pulled to the starting position. When he was ready, the magnet was released. The resulting movement and EMG events were displayed on an oscilloscope and recorded on three channels of magnetic tape at a speed of 381 mm (15 ins) per second. On the first day of testing, the unloading response was recorded 24 times with the right arm and 18 times with the left. To confirm the initial findings, the entire procedure was repeated on another day, 12 times with the right arm and 12 times with the left. Since the results were essentially the same on both occasions, the data were pooled for statistical analysis.

After each set of trials, the tape speed was changed to 95 mm/s for playback. The velocity and EMG signals were displayed by means of a Grass polygraph, with the paper speed set at 5 mm/s. The temporal resolution was thus five milliseconds per millimetre of the ink-written display.

The duration of the silent period was measured for each trial, according to an arbitrary rule: determine the maximal height of the rectified EMG signal during the interval before unloading, and divide by two; draw a horizontal line at the 50% level, as illustrated in Figs. 1 and 2. The silent period is defined as the interval during which the rectified EMG signal does not exceed the 50% line.

The amplitude of the EMG burst after the silent period was measured on the tracing of the filtered EMG and expressed as a fraction of the peak level before unloading (P1). For each trial, the value of P1 was taken as the highest point on the filtered EMG during the 250 ms just before unloading. The size of the volley after unloading

Fig. 1 Unloading reflex, right arm. Top line: velocity of right hand. Middle line: rectified EMG, right pectoral muscle. Bottom line: filtered EMG. As trace begins, patient is resisting a spring by contraction of arm adductors. At time shown by upward deflection of top line, spring is released. Duration of silent period is shown by line S.P. P1 is highest point on filtered EMG during 250 ms before unloading. Amplitude of the contraction after unloading is measured by ratio $P2 \div P1$.

was taken as the highest peak after the end of the silent period (P2): for each trial, the value of P2 was divided by the corresponding P1. Thirty-four such ratios were obtained for the right arm and 30 for the left.

Results

The differences between the right and left sides were apparent by inspection of individual tracings. The amplitude of the motor activity after the silent period was obviously greater on the left side (Figs. 1 and 2). Whereas the mean value of $P2/P1$ was 0.40 ± 0.03 (SE) on the right side, it was 0.86 ± 0.03 on the left. The difference was highly significant ($t=10.08$; $P<0.01$). The mean duration of the silent period was longer on the right side (183 ± 18 ms) than on the left (96 ± 3 ms). Again, the difference was significant ($t=4.84$; $P<0.01$). These results agreed with the prediction that the contraction after unloading would be greater on the side that was most affected by

Fig. 2 Unloading reflex, left arm. Top line: velocity of left hand. Middle line: rectified EMG from left pectoral muscle. Bottom line: filtered EMG. Unloading is followed by a relatively brief silent period (S.P.) and a large burst of EMG activity (P2). Ratio $P2 \div P1$ is larger than in Fig. 1.

Parkinson's disease. They showed, in addition, that the contraction began earlier, after a shorter silent period, on the more affected side.

Discussion

From the observations published in the past 100 years, several properties of the shortening reaction have been documented: (1) it occurs in normal subjects (Andrews *et al.*, 1972; Walsh, 1975); (2) its amplitude can be increased by "reinforcement" or "preparatory set" (Andrews *et al.*, 1973; Walsh, 1975); (3) it is decreased or abolished by lesions of the upper motor neurone (Foix and Thevenard, 1923; Broman, 1949); and (4) it is exaggerated in certain patients with disease of the basal ganglia (Broman, 1949; Rondot and Scherrer, 1966; Andrews *et al.*, 1972). Some points have not been clarified. The shortening reaction has not been classified either as a "reflex" or as a "reaction-time movement" (in Houk's (1977) terminology); the receptors mediating the reaction have not been identified, in spite of studies using ischaemic nerve block and procaine infiltration of agonist or antagonist muscle (Denny-Brown, 1960; Andrews *et al.*, 1973); and the primary neural structures

that mediate the response to shortening have not been determined, although the "gain" of the system is evidently controlled by pyramidal and extrapyramidal structures. The present study was motivated by the need for a better understanding of the muscular responses elicited by passive shortening.

Both the shortening reaction and the contraction after unloading occur under conditions that decrease the load on a muscle, allowing it to shorten by its own elasticity. In each case, a burst of EMG activity appears in the muscle during passive shortening. This led us to suspect that a common mechanism may be responsible for both phenomena.

As noted above, the shortening reaction is known to be diminished by lesions of the upper motor neurone. If the contraction after unloading is produced by the same mechanism, then it should also be decreased by such lesions. In a previous study (Angel, 1968) the unloading reflex was elicited on both sides of patients with spastic hemiparesis. In each case tested, the contraction after unloading was found to be significantly smaller on the spastic side than on the unaffected side (Angel, 1973).

The present study was designed to test another prediction based on the hypothesis of a common mechanism. Since the shortening reaction is known to be exaggerated in Parkinson's disease, we predicted that the response to unloading would be affected in the same way. Since there are no "normal standards" for the size of the contraction after unloading, we chose to study a patient with asymmetrical Parkinsonism, using the less affected side as a control.

The patient tested in this study cannot be regarded as a case of hemi-Parkinsonism, because there were signs of the disease on both sides. The facies, the slight rigidity of the right wrist, and the history of generalised weakness would all suggest that the right side was affected to some degree. However, the patient denied any symptoms referable to the right side and stated that, if the left side were as well as the right, he would have no complaints whatever.

On neurological examination the asymmetry of the disease process was very striking. In particular, Westphal's phenomenon was readily demonstrated on the left side. The spontaneous dorsiflexion affecting the left ankle and hallux, which impaired his walking and occasionally caused him to stumble, was suspected to be an exaggerated form of the shortening reaction. This contraction tended to occur during the weightbearing phase of gait, when the ankle is dorsiflexed by forward

motion of the body, allowing the muscles of the anterior tibial compartment to shorten.

The asymmetry of the findings led us to predict that the contraction after unloading would be larger on the left side than on the right. That prediction was confirmed by the data, which showed that the mean value of P2/P1 was more than two times greater on the left side. The relatively short duration of the silent period on the left side shows that the response was not only larger but also tended to begin earlier on that side.

The exaggeration of the motor activity after the silent period resembles a finding obtained by Drechsler and Boshes (1970). They elicited a silent period by supramaximal stimulation of the ulnar nerve during voluntary contraction of the abductor digiti minimi. In patients with Parkinsonism, there was a "marked rebound phenomenon" immediately after the silent period.

The common mechanism hypothesis provides an explanation for the results obtained in our Parkinsonism patient, as well as those obtained in patients with spastic hemiparesis. However, it remains to be seen whether the parallel changes of the shortening reaction and the contraction after unloading in pyramidal and extrapyramidal disease are caused by involvement of a common mechanism.

Many observations have confirmed the fact that muscles tend to contract in response to passive shortening or unloading. One is inclined to ask whether these contractions have any functional significance. Sherrington (1951) has pointed out the value of such inquiry. "Yet every reflex is in its own measure an integral reaction, and is purposive in that it bears some biological purport for its organism. Every reflex can, therefore, be regarded from the point of view of what may be called its 'aim'. To glimpse at the aim of a reflex is to gain hints for further experimentation on it."

It is generally believed that one function of the stretch reflex is to compensate for an increase of load on the muscle. Quite clearly, the shortening reaction cannot serve this function, because it occurs when the load is decreased by passive movement. A possible function of the reaction might be to "take up the slack" in muscle. When a passive movement brings the origin and insertion of a muscle closer together, the force on the tendon is reduced, and the muscle is free to shorten. If the elastic shortening is not sufficient to remove the slack, the muscle will be unable to act efficiently as agonist or antagonist in the next voluntary movement. Hence, the function of the shortening reaction might be to prepare the

muscle for efficient contraction at the new, shorter length.

A disorder of the muscle shortening mechanism might add to the disability found in certain motor system diseases. Increased activity of the mechanism could play a part in the rigidity of Parkinson's disease, and decreased activity could impair the efficiency of muscles that are weakened by disease of the upper motor neurone. These considerations would suggest that the shortening reaction plays a part in both the physiology and the pathophysiology of the motor system.

References

- Alston, W., Angel, R. W., Fink, F. S., and Hofmann, W. W. (1967). Motor activity following the silent period in human muscle. *Journal of Physiology*, **190**, 189–202.
- Andrews, C. J., and Burke, D. (1972). A quantitative study of the effect of L-dopa and phenoxybenzamine on the rigidity of Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, **36**, 321–328.
- Andrews, C. J., Burke, D., and Lance, J. W. (1972). The response to muscle stretch and shortening in Parkinsonism rigidity. *Brain*, **95**, 795–812.
- Andrews, C. J., Neilson, P. D., and Lance, J. W. (1973). Comparison of stretch reflexes and shortening reactions in activated normal subjects with those in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, **36**, 329–333.
- Angel, R. W. (1968). Unloading reflex in patients with hemiparesis. *Neurology (Minneapolis)*, **18**, 497–503.
- Angel, R. W. (1973). Spasticity and tremor. Effects on the silent period and the after volley. In *New Developments in Electromyography and Clinical Neurophysiology*, vol. 3, pp. 618–624. Edited by J. E. Desmedt. Karger: Basel.
- Broman, T. (1949). Electromyo-mechanographic registration of passive movements in normal and pathological subjects. *Acta Psychiatrica Scandinavica*, Supplement **53**, 1–63.
- Burke, D., Andrews, C., and Gillies, J. D. (1971). The reflex response to sinusoidal stretching in spastic man. *Brain*, **94**, 455–470.
- Delmas-Marsalet, P. (1927). *Les réflexes de posture élémentaires*. Masson et Cie: Paris.
- Denny-Brown, D. (1960). Diseases of the basal ganglia. Their relationship to disorders of movement. *Lancet*, **2**, 1099–1105 and 1155–1162.
- Drechsler, B., and Boshes, B. (1970). Autogenic inhibition in patients with Parkinsonism: a neurophysiological study. *Transactions of the American Neurological Association*, **95**, 235–236.
- Foix, C., and Thevenard, A. (1923). Les réflexes de posture. *Revue Neurologique*, **39**, 449–468.
- Förster, O. (1921). Zur Analyse und Pathophysiologie der striären Bewegungsstörungen. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, **73**, 1–169.
- Garland, H., Angel, R. W., and Melen, R. D. (1972). A state variable averaging filter for electromyogram processing. *Medical and Biological Engineering*, **10**, 559–560.
- Houk, J. C. (1977). Participation of reflex mechanisms and reaction-time processes in the compensatory adjustments to mechanical disturbances. In *Cerebral Motor Control in Man: Long Loop Mechanisms*. *Progress in Clinical Neurophysiology*, vol. 4. Edited by J. E. Desmedt. Karger: Basel.
- Rondot, P., and Metral, S. (1973). Analysis of the shortening reaction in man. In *New Developments in Electromyography and Clinical Neurophysiology*, vol. 3, pp. 629–634. Edited by J. E. Desmedt. Karger: Basel.
- Rondot, P., and Scherrer, J. (1966). Contraction réflexe provoquée par le raccourcissement passif du muscle dans l'athétose et les dystonies d'attitude. *Revue Neurologique*, **114**, 329–337.
- Samojloff, A., and Kisseleff, M. (1927). Die Verkürzungs- und Verlängerungsreaktion des Knieextensors der decerebrierten Katze. *Pflügers Archiv für die gesamte Physiologie des Menschen und der Tiere*, **218**, 267–284.
- Sherrington, C. S. (1951). Postural activity of muscles and nerve. *Brain*, **38**, 193.
- Walsh, E. G. (1975). Shortening reactions in the human forearm. *Journal of Physiology*, **256**, 116–117P.
- Westphal, C. (1877). Unterschenkelphänomen und Nervendehnung. *Archiv für Psychiatrie und Nervenkrankheiten*, **7**, 666–670.
- Westphal, C. (1880). Über eine Art paradoxer Muskelcontraction. *Archiv für Psychiatrie und Nervenkrankheiten*, **10**, 243–248.