J. Physiol. (1965), **180**, *pp.* 864–870
With 2 text-figures
Printed in Great Britain

SILENT PERIOD PRODUCED BY UNLOADING OF MUSCLE DURING VOLUNTARY CONTRACTION

BY R. W. ANGEL, W. EPPLER AND A. IANNONE

From the Clinical Neurophysiology Laboratory, Department of Neurology, Veterans Administration Hospital and the Division of Neurology, Stanford University School of Medicine, Palo Alto, California, U.S.A. and the Electronics Sciences Laboratory, Lockheed Missiles and Space Company, Palo Alto, California, U.S.A.

(*Received* 19 *March* 1965)

Merton (1950, 1951, 1953) has proposed that the fusimotor fibres are a normal pathway for initiating muscular contractions. Through its effect upon the muscle spindle, fusimotor discharge tends to increase the firing rate of the primary endings, thus causing reflex activation of the alpha motoneurones. On Merton's view, this mechanism provides a servo-control by which the muscle can be set reflexly to any desired length. Because of its great theoretical importance, the servo theory of contraction has attracted widespread attention (Roberts, 1952; Eldred, Granit & Merton, 1953; Granit, Holmgren & Merton, 1955; Hammond, Merton & Sutton, 1956; Adolph, 1959; Partridge & Glaser, 1960).

Evidence for this proposal first came from investigation of the 'silent period' which occurs in the electromyogram of a muscle contracting voluntarily when its nerve is stimulated electrically to produce a twitch. The suggested interpretation is that a twitch contraction unloads the spindles, reduces their rate of discharge and thus withdraws excitation from the motoneurones. However, the production of a silent period by electrical stimulation of the motor nerve does not imply that muscular contraction is initiated or sustained by the gamma route. As Matthews (1964) has pointed out, the silent period may result from several factors, of which the spindle pause is only one. Under the conditions of Merton's experiment, a synchronous discharge of motor axons is directed not only to the muscle, but also through recurrent collaterals to the Renshaw cells, which in turn deliver inhibitory impulses to the motoneurones. Another source of inhibition is the group IB fibres supplying the Golgi tendon organs. These would be activated by electrical stimulation and also by the tension developed in muscular contraction.

In order to establish the role of spindle silence, it is necessary to demon-

strate the silent period by some method which does not produce a synchronous discharge of motoneurones, Renshaw cells or Golgi tendon afferents. This requirement is not met by use of the tendon jerk. Although animal experiments of Jansen & Rujord (1963) have shown that the Golgi tendon organs do not play an essential part in determining the duration of the silent period, it is still necessary to exclude other possible sources of inhibition.

In the present investigation, we have produced the silent period by suddenly unloading a muscle during voluntary contraction. This method avoids the objections that have been raised against the use of electrical stimulation and the tendon jerk. It also permits one to study the 'check reflex' activity in the antagonistic muscle and the time course of the resulting limb displacement.

METHOD

Fifteen human subjects without neurological disease were used for the experiments. In eleven, electromyograms were obtained from the right biceps and triceps brachii; in four, from the right pectoralis major and infraspinatus together with posterior fibres of deltoideus. Muscle action potentials were detected by means of silver-disk or suction-cup electrodes, two of which were applied to the skin over each muscle being studied. After amplification, the action potentials were displayed on two beams of a storage oscilloscope.

Velocity of the hand was measured by means of special equipment that operates as follows: a metal rod is suspended vertically in front of the subject, who grasps the lower end with his right hand. This end is free to swing right or left, and a gimbal permits antero-posterior motion. The upper end of the rod transmits the right-left component of motion to a variable capacitor, which is wired in series with a battery and a resistor. When the rod swings to right or left, there is a change in the variable capacitance and current flows through the resistor, causing a voltage drop which is proportional to the velocity of the swinging rod. The scale factor of the equipment was determined by measuring the voltage produced by moving the rod at known velocities.

When movements at the elbow joint were studied, the subject was seated facing the rod with the shoulder abducted 90 degrees and the elbow flexed 90 degrees. The elbow was supported on a smooth platform, and the forearm extended horizontally forward. The volar surface of the wrist joint was taped firmly to the lower end of the rod. Lateral force was applied to the rod by means of a cord, pulley and measured weight. This force was counteracted by active contraction of the biceps. When movements at the shoulder joint were studied, the subject reached directly forward to grasp the rod, which he held in the median plane, using his pectoralis to counteract the lateral force.

While the subject held his hand stationary against the external force, he was asked to shut his eyes, and, without warning, the experimenter cut the wire that supported the weight. This broke an electrical circuit, and the oscilloscope was triggered at the instant the weight was released.

Separate beams of the storage oscilloscope were used to display electrical activity of each muscle and the velocity of hand motion. Four quantities were measured: latency of relaxation of biceps (or pectoralis), latency of contraction of triceps (or posterior deltoid), latency of resumed contraction of biceps (or pectoralis) and maximal velocity of the hand. Weights of 2·25, 4·5 and 6·75 kg were used on different trials. For all subjects, the test was repeated several times to verify the presence of a silent period. In three of the 'biceps' group, the test was repeated a minimum of five times with each size of weight, to permit estimation of

the mean latencies and velocities. In four of the 'pectoralis' group, the test was repeated a minimum of five times with 4·5 kg and five times with 6·75 kg.

With two subjects in the 'biceps' group, a wire was attached to the metal rod so that elbow flexion could be stopped at any point, depending upon the amount of slack in the wire. The weight-dropping test was then repeated with various amounts of slack in the restraining wire. For each test, the duration of movement, maximal velocity, total displacement of hand, and length of silent period was determined.

RESULTS

As the subject held his limb stationary against the applied force, there was a steady background of electrical activity in the agonist, and the antagonist was electrically silent. Release of the external force was followed by a predictable series of events. The hand immediately showed a rapid acceleration, caused by the unopposed contraction of the agonist. This produced a maximal velocity of 76–152 cm per second, depending upon the size of the weight used. Within about 40 msec after release of the weight a silent period began in the agonist (Fig. 1). A burst of activity in the antagonist was followed in about 50 msec by deceleration of the limb. Action potentials were again seen in the agonist, 70–150 msec after release of the weight. This second burst of activity was often of greater amplitude than the previous background activity, but it had no apparent effect on the velocity curve. Velocity became zero in 150–300 msec.

Table 1 shows the average measurements obtained from four subjects using the pectoralis and deltoid muscles and 6·75 kg of force. In these cases, the mean duration of the silent period varied from about 69 to 114 msec. The maximal velocities attained were invariably greater with 15 than with 10 lb, but there was no significant difference in the latencies of the muscular responses. The figures in the table, therefore, may be taken to illustrate those obtained with either 4·5 or 6·75 kg of force.

When flexion of the elbow was prevented by the restraining wire, the silent period did not occur, and there was no discharge in the triceps. When the distal end of the radius was allowed to move 1·3 cm or more, the silent period was again seen, and the triceps showed a burst of action potentials. With increasing amounts of flexion up to 4 cm, there was a progressive increase in duration of the silent period. There was no further increase of the silent period when movements beyond 4·0 cm were allowed (Fig. 2).

DISCUSSION

These experiments obviate certain objections that have been raised against Merton's analysis of the silent period. Before release of the weight there was a steady, asynchronous discharge of motor units in the agonist muscle. Release of the weight could not be expected to produce a synchronous burst of activity in the motoneurones, either reflexly or by

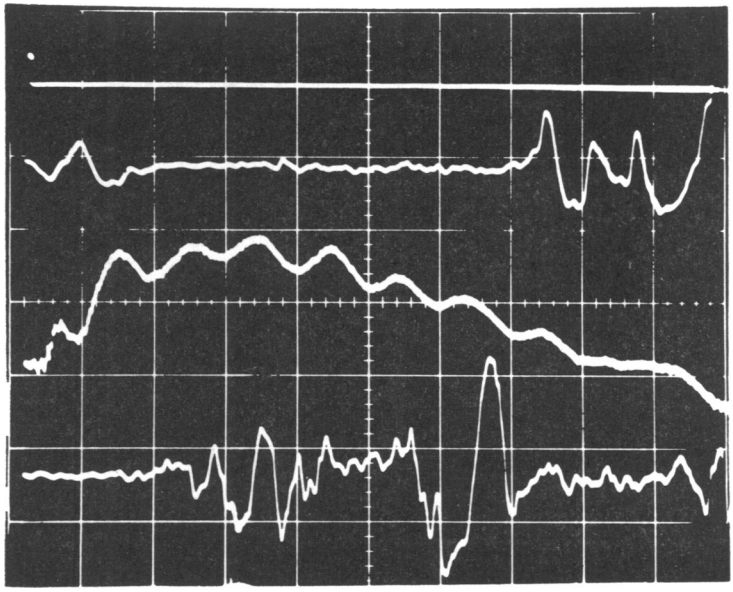

Fig. 1. Electromyograms and velocity of hand following release of 6·75 kg force. Subject held the hand in median plane, resisting a 6·75 kg lateral force. Top line: signal indicating the instant that force was released (discontinuity at extreme left). Second line: action potentials from pectoralis muscle. 2 mV per division. Third line: velocity of right hand. Upward deflexion represents movement to left. 91 cm per second per division. Fourth line: action potentials from infraspinatus and posterior fibres of deltoid muscle. 1 mV per division. Time: 20 msec per division.

Thirty-six msec after the release of force, when the hand has moved only 1·5 cm, pectoralis becomes electrically inactive. At 50 msec, a burst of activity appears in infraspinatus. At 140 msec, pectoralis resumes activity. Velocity curve rises from zero to about 147 cm per second, then recrosses the zero line 190 msec after beginning of movement. The total displacement of the hand during this move is about 18 cm. (The oscillation superimposed on the velocity curve is due to mechanical vibration of the metal rod which transmits hand motion to a variable capacitor.)

TABLE 1. Events following sudden release of 6·75 kg weight

	Pectoralis becomes silent		Posterior deltoid fires		Pectoralis resumes firing		Maximal velocity of hand	
Subject	Mean (msec)	S.D.	Mean (msec)	S.D.	Mean (msec)	S.D.	Mean (in./sec)	S.D.
J.C.	41·0	2·6	37·1	7·9	151·0	9·9	37·8	1·5
E.M.	34·4	3·4	47·2	5·9	*	*	61·2	6·2
A.N.	28·4	7·5	41·2	8·7	142·4	26·6	51·0	6·0
J.Y.	43·8	7·3	43·3	8·1	112·3	26·1	58·0	13·5

* No clear resumption of firing.

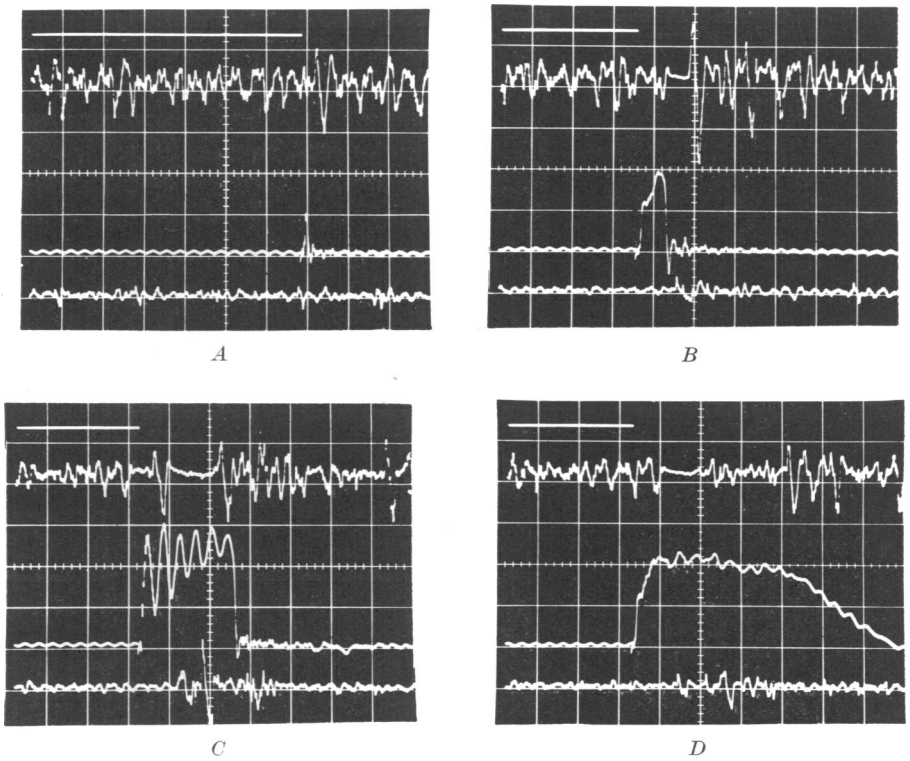

Fig. 2. Electromyograms and velocity of distal radius following release of 6·75 kg force. Subject holds right arm abducted 90 degrees and elbow flexed 90 degrees, resisting a 6·75 kg lateral force. Top line ends the instant that force is released. Second line: action potentials from biceps. 0·2 mV per division. Third line: velocity of distal end of radius. 36 cm per second per division. Fourth line: action potentials from triceps 0·2 mV per division. Time: 50 msec per division.

A. A taut wire prevents any flexion of forearm when the lateral force is released. No silent period is seen in biceps, and there is no reflex discharge of triceps. Velocity record shows that movement lasts only 10 msec.

B. The slack in restraining wire allows forearm to flex for 40 msec, and the distal radius moves about 2 cm. A silent period, lasting 25 msec, is seen in biceps, and there is a small discharge in triceps.

C. The slack allows forearm to flex for 120 msec, and the radius moves about 10 cm. The silent period now lasts 55 msec, and there is a larger response in triceps.

D. The restraining wire has been removed. The forearm flexes for 315 msec, and distal radius moves 17·5 cm. Silent period lasts 55 msec.

antidromic firing of the motor axons. Hence, there was no cause for synchronous activation of the Renshaw cells. The load on the tendons was constant until it was reduced by cutting loose the weight, so that there was no cause for increased firing of the Golgi tendon organs. When these two sources of inhibition are inoperative, the role of spindle silence assumes greater prominence.

Under the conditions of these experiments, the silent period might be attributed to reciprocal inhibition associated with stretch of the antagonist. While this possibility cannot be excluded, there were many tests in which the agonist fell silent before any potentials appeared in the antagonist. Figure 1 illustrates one such case. If the silent period were due to reciprocal inhibition, which requires more than one synapse, then we should not expect it to begin sooner than the monosynaptic reflex in the antagonist.

The experiments in which elbow flexion was restricted artificially show that sudden removal of force does not, in itself, produce a silent period. Unless the muscle is allowed to shorten, no silent period occurs, presumably because the spindles are not unloaded. When the distal radius is allowed to move about 1·3 cm, the silent period returns. A rough calculation shows that this movement corresponds to a 0·28 cm shortening of the biceps.

In correlating the velocity curve with the electromyograms, one must recognize that there is an appreciable lag between the electrical and mechanical events in muscular contraction (Wilkie, 1950; Hammond, 1954). The initial sharp rise in the velocity curve is obviously due to unopposed contraction of the agonist. The limb reaches a velocity at which the frictional resistance exactly counterbalances the force of the agonist and then moves with zero acceleration, i.e. constant velocity. Deceleration begins about 50 msec after the onset of firing in the antagonist.

The second burst of activity in the agonist is not followed by a positive acceleration of the arm. It appears to be an isotonic contraction which exerts no force on the limb but sets the muscle at a new length.

SUMMARY

1. The silent period has been demonstrated in fifteen human subjects, by sudden release of an external force against which a muscle was contracted voluntarily.

2. Under the conditions of these experiments, the silent period cannot be attributed to the discharge of Golgi tendon organs, inhibition through the Renshaw cells or the synchronized discharge of motoneurones.

3. Since the silent period often appeared before the onset of action potentials in the antagonist, it cannot be ascribed to reciprocal inhibition.

4. The silent period did not occur unless the agonist was allowed to shorten.

5. The time course of the velocity curve can be correlated with electrical events in the muscles.

REFERENCES

ADOLPH, A. F. (1959). Feedback in physiological systems: an application of feedback analysis and stochastic models to neurophysiology. *Bull. Math. Biophys.* **21**, 195–216.

ELDRED, E., GRANIT, R. & MERTON, P. A. (1953). Supraspinal control of the muscle spindles and its significance. *J. Physiol.* **122**, 498–523.

GRANIT, R., HOLMGREN, B. & MERTON, P. A. (1955). Two routes for excitation of muscle and their subservience to the cerebellum. *J. Physiol.* **130**, 213–224.

HAMMOND, P. H., MERTON, P. A. & SUTTON, G. G. (1956). Nervous gradation of muscular contraction. *Brit. med. Bull.* **12**, 214–218.

HAMMOND, P. H. (1954). Involuntary activity in biceps following the sudden application of velocity to the abducted forearm. *J. Physiol.* **127**, 23–25*P*.

JANSEN, J. K. S. & RUDJORD, T. (1963). The silent period during twitch contraction of the soleus of the decerebrate cat. *Acta physiol. scand.* Suppl. 213. **59**, 69–70.

MATTHEWS, P. B. C. (1964). Muscle spindles and their control. *Physiol. Rev.* **44**, 219–288.

MERTON, P. A. (1950). Significance of the silent period of muscles. *Nature, Lond.*, **166**, 733–734.

MERTON, P. A. (1951). The silent period in a muscle of the human hand. *J. Physiol.* **114**, 183–198.

MERTON, P. A. (1953). Speculations on the servo-control of movement. In *Ciba Foundation Symposium: The Spinal Cord*, pp. 247–260. London: Churchill; Boston: Little Brown and Co.

PARTRIDGE, L. D. & GLASER, G. H. (1960). Adaptation in regulation of movement and posture. A study of stretch reflexes in spastic animals. *J. Neurophysiol.* **23**, 257–268.

ROBERTS, T. D. M. (1952). The inclusion of the muscle-spindle afferent in the 'final common path'. *J. Physiol.* **118**, 8–9*P*.

WILKIE, D. R. (1950). The relation between force and velocity in human muscle. *J. Physiol.* **110**, 249–280.