Methods of tendon jerk reinforcement

The role of muscle activity in reflex excitability

KENNETH H. OTT¹ AND M. MICHAEL GASSEL²

From the Department of Neurology, University of California School of Medicine, San Francisco, California, U.S.A.

This study was designed to reconcile the following clinical observations: subjects with anxiety represented in muscle tension generally have hyperactive tendon jerks. Yet, muscle tension evidenced by incomplete relaxation frequently results in reflex depression and the tendon reflex can only be elicited by having the subject 'relax'. Under consideration were the observations by Hoffmann (1922) and by Hagbarth (1962) that a moderate amount of voluntary activity facilitates the monosynaptic reflex in the muscle tested. A problem to be investigated was the relationship of the facilitation produced by the Jendrassik manoeuvre, during forceful contraction of distant muscles masses, to that produced by voluntary activity in the same muscle. Numerous other techniques have been reported to be useful in facilitating reflexes, ostensibly through an effect on muscle activity. Cutaneous stimuli, such as needleprick, cold or warm baths, pinching, electrical shock, or manipulation of muscles have been variously reported to reinforce the tendon jerks. It has been suggested that the effectiveness of these manoeuvres is related to the distraction of attention, assisting the relaxation of muscles held in high tension (Wartenberg, 1945).

METHODS

Fifteen normal subjects were examined. Subjects were placed in a prone position with legs extending over the end of the couch. The legs were supported firmly in a heavy metal frame which was fixed to a metal stand separate from the couch. The leg frame was adjustable in height and angle. An automatic solenoid hammer was attached to the leg frame by a metal supporting device with two complex joints. These joints permitted movement of the hammer in all directions and made it possible to position the hammer at a fixed distance (3 cm) directly over the Achilles tendon. The solenoid was triggered from the stimulator (DISA Multistim) and the delay to

the tendon tap was about 55 msec. This delay was measured before each experiment. The solenoid driven reflex hammer had a built-in microswitch which closed the circuit on percussion and triggered the sweep of the electromyograph. A regular and stabilized blow was delivered. A control series of maximal reflexes was elicited at a frequency of one every 10 seconds. Initially 20 reflexes were averaged by computer (CAT 1000) in establishing the control and test values. In later studies it was usually feasible to average just eight reflexes, when control variations were small. The controls were repeated at intervals during the investigation and are indicated in the graph at the appropriate test intervals.

The amplitude of a series of ankle jerk reflexes was measured during minimal, moderate, and intense degrees of isometric contraction of the ipsilateral and contralateral triceps surae, and ipsilateral pre-tibial groups. The electrical activity from these muscles was recorded with non-insulated subcutaneous needle electrodes. The potentials were amplified, displayed, and photographed on the DISA 3-channel electromyograph. The audio-amplifier of the electromyograph was utilized to allow the subject to maintain constancy of the desired degree of activity.

The ankle jerk was also conditioned by the Jendrassik manoeuvre. The manoeuvre consisted of brisk and strong contraction of the subject's interlocked fingers on the command 'Pull', and the quick relaxation with the command 'Release'. Duration of the manoeuvre was about two seconds, with the onset and termination marked on the photographic recording.

Finally, the recovery of the ankle jerk reflex after nociceptive cutaneous stimulation was measured. A DISA Multistim stimulator was utilized with a maximal voltage output, via transformer, of 250 V. Single 3 msec duration pulses were delivered to the skin through subcutaneous stainless steel needle electrodes placed 2 cm apart. The conditioning shock was followed at intervals ranging from 20 to 8,000 msec by the ankle jerk reflex. Skin sites stimulated in this investigation were the ipsilateral mid buttock and ipsilateral mid anterior arm.

RESULTS

The ankle jerk reflex was facilitated by mild degrees of activity in the ipsilateral triceps surae; moderate

¹Medical Student; USPHS Research Fellowships FR-05355-07 and NB-5101-13.

²This work was supported by NIH Grant NB-07562.

FIG. 1a. Average amplitude of 20 ankle jerk reflexes during minimal (+), moderate (++), and intense (+++) levels of voluntary activity on the ipsilateral (O) and contralateral (●) triceps surae in a representative subject. These averaged values have been normalized to control values. The insets are electromyograms (EMGs) of the three levels of activity.

levels of activity resulted in no change or mild depression, while more intense contraction markedly inhibited the ankle jerk.

Minimal activity in the contralateral triceps surae produced no change in the reflex, moderate activity evoked little or no facilitation, and a mild degree of facilitation occurred with intense activity in the contralateral triceps surae.

Figure 1 illustrates representative effects on the ankle jerk at each of three levels of activity in the ipsilateral and in the contralateral triceps surae. There was considerable variation in the extent of facilitation between subjects and to lesser extent in the same subject on different occasions. The conclusions are based upon investigative sessions lasting one and a half to two hours. Four of 15 volunteers were studied on two or more occasions.

Activity in antagonist pre-tibial muscle inhibited the reflex, and inhibition increased with increasing degrees of antagonist activity. Concurrent activity in the ipsilateral triceps surae and pre-tibial muscles usually resulted in inhibition, although of less marked degree than with antagonist action alone (Fig. 2).

The extent of facilitation produced by the Jendrassik manoeuvre was of about the same order as that evoked by mild activity in the agonist. There was occlusive interaction between the facilitation of the

Jendrassik manoeuvre and that of voluntary activity (see Fig. 1). Maximal facilitation of tendon jerk was achieved by simultaneous performance of the Jendrassik manoeuvre with maintained minimal voluntary activity in the muscle tested. Computer averages of the amplitudes of the reflexes during the combined manoeuvres were variable. It was therefore necessary to photograph long series of individual records, because of the considerable variability of the frequency of motor unit discharge during the Jendrassik manoeuvre (Fig. 3). The most regular change was the striking decrease in the ankle jerk with the decrease in motor unit activity during performance of the Jendrassik manoeuvre. The conclusions regarding occlusive interaction were based upon those studies in which there was no change in tonic motor unit firing.

It was possible to add facilitatory and inhibitory effects in some dexterous subjects. Simultaneous performance of the Jendrassik manoeuvre, maintained tonic activity of mild degree in the agonist, and activity in the antagonist resulted in mitigation of the facilitation. In a number of subjects inhibition by antagonist activity was prepotent.

The effect of cutaneous stimulation upon the ankle jerk was evaluated initially by pinprick of widespread skin areas. The ankle jerk was highly variable. Therefore, the skin was stimulated through

FIG. 1b. Computer (CAT 1000) average of 20 ankle jerk reflexes during minimal (upper trace), moderate (middle trace), and sub-maximal voluntary activity (lower trace) in the ipsilateral triceps surae.

subcutaneous electrodes fixed in place, which made it possible to deliver a regular and unvarying conditioning stimulus with a single shock to the skin. The stimulus was followed by an ankle jerk at regulated intervals. The motoneurone excitability changes were then evaluated by the classical recovery curve of the monosynaptic reflex.

There was striking facilitation of the ankle jerk with single-shock cutaneous stimulation of both the ipsilateral mid anterior arm and mid buttock. This began at 20 to 60 msec and ended at about 200 to 250 msec, when it was followed by a mild and variable degree of inhibition before the reflex returned to control levels. There was no muscle discharge evoked by the single-shock stimulus. Ten normal subjects were studied and representative recovery curves of the monosynaptic reflex are illustrated in Fig. 4.

DISCUSSION

There is evidence bearing on the mechanism by which tonic voluntary activity in the triceps surae

augmented the ankle jerk in the same muscle. The immediate consideration was to determine the extent to which the reflex enhancement was localized to the active muscle, or represented the local reflection of a more generalized facilitation with muscle activity as produced in the Jendrassik manoeuvre. It was found that the slight tonic activation of the triceps surae which facilitated the ankle jerk maximally produced no contralateral reflex reinforcement. Indeed, facilitation first appeared during maximal tonic contraction of the contralateral triceps surae at a level of activity which, in the ipsilateral muscle, produced depression of the reflex. The facilitation of the ankle jerk with intense activity only of the contralateral triceps surae could be related to the generalized facilitation of monosynaptic reflexes by distant muscle activity as in the Jendrassik manoeuvre; however, group 1B tendon afferents excited by the intense activity likewise facilitate contralateral triceps surae motoneurons (Perl, 1958). It would appear that in this large extensor muscle mass voluntary activation is manifested by an

FIG. 2. The averaged amplitude of 20 ankle jerk reflexes conditioned by various means are plotted serially with corresponding controls. The effect on reflex amplitude is illustrated with increasing levels of voluntary activity in the ipsilateral triceps surae, Jendrassik manoeuvre, simultaneous ipsilateral agonist and antagonist activity, and minimal agonist activity and the Jendrassik manoeuvre concurrently performed.

increase in the subliminal fringe of the motoneurone pool innervated, as tested by the amplitude of the monosynaptic reflex, as well as by motoneurone discharge. Not only is voluntary activation associated with an increase in motoneurone excitability, but conversely changes in motoneurone excitability produce a modulation of motor unit firing during tonic activation (Gassel and Ott, 1969). This suggests that voluntary activation is effected through changes in excitability of the motoneurone pool. Evidence of the reactivity of tonic motor unit discharge in the triceps surae to changes in motoneurone excitability was obtained by comparing the effects upon motoneurone discharge, to the recovery curve of monosynaptic reflex, which had earlier been established by the same segmental stimulus. In general, it was found that conditioning stimuli which inhibited the monosynaptic reflex also depressed tonic voluntary activity, and that stimuli which augmented the ankle jerk also facilitated tonic firing, although there was sometimes inhibition which preceded or followed facilitation (Gassel and Ott, 1969). It had earlier been established that slight or moderate voluntary activity decreases the threshold and augments the monosynaptic reflex from the same muscle (Hoffmann, 1922; Hagbarth, 1962). Ashworth, Grimby, and Kugelberg (1967) found that motor units were usually recruited in the same

order when activated by the polysynaptic flexion reflex, by voluntary activity, or with the monosynaptic reflex. They also reported interaction between voluntary and reflex activation. It does not, of course, follow that voluntary activation is produced solely by an increase in excitability of the motoneurone pool. The specific and discrete movements possible in muscles, such as those of the hands, presupposes this is a regular mechanism; specific patterns of inhibition and activation must be involved.

The inhibition of the ankle jerk with intense or sometimes with only moderate muscle activity could be related to a number of factors. There is occlusion of the reflex with active motor units and with those in the refractory phase after discharge. The increase of muscle tension with strong tonic activity provides a stimulus to group 1B tendon afferents, with resulting autogenic inhibition. Recurrent inhibition through recurrent collaterals is also undoubtedly operative. It is of interest that significant recurrent inhibition does not occur with single unit firing. Recurrent collaterals produce a widespread inhibition affecting flexors and extensors in local spinal segments. However, the inhibition does not spread to affect the contralateral motoneurons (Holmqvist, 1961); and this may account in part, for the facilitation with maximal activity in the contralateral triceps surae at a level of activity which produced pronounced depression of the ipsilateral ankle jerk.

The inhibition of the reflex with activity of the antagonist muscles was occasionally striking. In some subjects even slight activity in the antagonist overcame potent facilitation. This effect may be related to reciprocal innervation. It is also possible that group 1A fibres are excited indirectly through fusimotor activation, which could produce direct inhibition of the extensor motoneurons (Eccles, Fatt, and Landgren, 1956). Group 1B fibres from pre-tibial flexors produced no effect on ankle extensors (Eccles, Eccles, and Lundberg, 1957).

The extent of the facilitation by the Jendrassik manoeuvre was of about the same order as that evoked with slight activity of the muscle tested. There was occlusive interaction between the facilitation of the Jendrassik manoeuvre and that of voluntary activity. Maximal facilitation of tendon jerks was achieved by simultaneous performance of the Jendrassik manoeuvre with maintained minimal voluntary activity in the muscle tested. The occlusive interaction is not unexpected, as both the Jendrassik manoeuvre (Gassel and Diamantopoulos, 1964b) and voluntary activity in the muscle tested by the monosynaptic reflex (see above) produce a direct effect on the excitability of the motoneurone pool.

FIG. 3. DISA recording of a single ankle jerk reflex with moderate voluntary activity in ipsilateral triceps surae and during simultaneous performance of the Jendrassik manoeuvre. Upper and middle traces are parallel EMGs from the triceps; the lower trace is the EMG from the ipsilateral pre-tibial muscles. The amplification of the middle and lower traces is $50 \times$ that in the upper trace. The Jendrassik manoeuvre is marked on the lower trace. Inset is a control reflex, unconditioned by voluntary activity or the Jendrassik manoeuvre.

FIG. 4. The recovery curve of the mono-synaptic reflex (ankle jerk) tested from a population of triceps surae motoneurons with conditioning stimulus to the ipsilateral mid anterior arm and mid buttock. Each graphic point represents the averaged value of eight reflexes normalized to controls.

The importance of muscle tension in reflex activity is well established and it has even been suggested that some level of muscle activity is a prerequisite for the evocation of a reflex. Certainly, monosynaptic reflexes are reduced or absent in the extreme relaxation of sleep; however, during the various stages of sleep, there are complex influences on spinal motoneurones excitability. There is a tonic inhibition of the monosynaptic reflex throughout desynchronized sleep and a phasic inhibition during rapid eye movements (REM) (Gassel, Marchiafava, and Pompeiano, 1964). The tonic inhibition is a result of descending impulses producing post-synaptic inhibition and the phasic change attributable to pre-synaptic inhibition (Gassel, Marchiafava, and Pompeiano, 1965). During REM myoclonic twitches occur predominantly and are related to descending facilitatory volleys which mitigate the phasic inhibition of the monosynaptic reflex. There is also a particular depression of fusimotor activity during desynchronized sleep and during spindle bursts of synchronized sleep (Gassel and Pompeiano, 1965). The antagonistic influences converge on the motoneurone pool where a quantitative interaction occurs, reflected in the response of the subliminal fringe whose excitability is evaluated by changes in the amplitude of the monosynaptic reflex.

The tendon jerks are evoked without preceding muscle activity in a vast majority of normal subjects, and it can be demonstrated that the augmentation of tendon jerks with the Jendrassik manoeuvre occurs without associated activity in the muscle tested (Gassel and Diamantopoulos, 1964a).

The primary response on stimulation of cutaneous afferents is ipsilateral withdrawal involving excitation of flexor and inhibition of extensor motoneurones. However, this response is not invariable and the occasional occurrence of extensor responses on stimulation of ipsilateral cutaneous nerves were early reported by Brown and Sherrington (1912), and Creed, Denny-Brown, Eccles, Liddell, and Sherrington (1932). Hagbarth (1952) found that extensor muscles were excited by stimulation of skin areas located directly over the muscle, in spinal and decerebrate cats. Kugelberg, Eklund, and Grimby (1960) studied the nociceptive reflexes of the lower limb in humans after delivering trains of cutaneous stimuli; reflex responses were sometimes recorded in the gastrocnemius on strong stimulation of the skin of the dorsum of the foot or of the buttock. Single-shock stimulation of areas of the skin in normal human subjects produces a reliable pattern of excitability change in the triceps surae motoneurones (Gassel and Ott, 1969). There was no muscle discharge in the course of the excitability changes evoked by the single-shock stimulus in the

present study. Patterns of excitability change, predominantly extensor facilitation, were evoked from widespread cutaneous areas of the upper and lower extremities, back, and face; that evoked on stimulation of the ipsilateral arm and buttock is illustrated in Fig. 4.

Clearly, stimulation of cutaneous afferents can facilitate monosynaptic reflexes without effecting discharge of motoneurones. The influence on motoneurone excitability of a cutaneous stimulus will depend upon the character of the stimulus, the timing of the ankle jerk after the conditioning stimulus, as well as the area of the skin stimulated.

SUMMARY

The role of muscle activity in reflex excitability was investigated in 15 normal subjects, utilizing a regulated system for eliciting the ankle jerk and recording muscle activity from agonist and antagonist. Minimal degrees of voluntary activity in the same muscle facilitated the ankle jerk; greater degrees produced inhibition. Facilitation first appeared during maximal contraction of the contralateral triceps surae at a level of activity which, in the ipsilateral muscle, produced depression of the reflex. Occlusion was demonstrated between facilitations by voluntary activity in the muscle tested and by the Jendrassik manoeuvre. Activity in antagonist muscles inhibited the reflex, overcoming potent facilitation. Maximal facilitation of tendon jerks is achieved by simultaneous performance of the Jendrassik manoeuvre with maintained minimal voluntary activity in the muscle tested. Single-shock stimulation of widespread areas of the skin can facilitate monosynaptic reflexes without effecting discharge of motoneurones. The precise pattern of excitability change with a cutaneous stimulus depends upon the character of the stimulus, the timing of the ankle jerk after the conditioning stimulus, as well as the area of the skin stimulated. The mechanisms of the various means of facilitation of tendon jerks were discussed as was the interaction of these augmentations with that produced by voluntary muscle activity.

REFERENCES

- Ashworth, B., Grimby, L., and Kugelberg, E. (1967). Comparison of voluntary and reflex activation of motor units; functional organization of motor neurones. *J. Neurol. Neurosurg. Psychiat.*, 30, 91-98.
- Brown, T. G., and Sherrington, C. S. (1912). The rule of reflex response in the limb reflexes of the mammal and its exceptions. *J. Physiol. (Lond.)*, 44, 125-130.
- Creed, R. S., Denny-Brown, D., Eccles, J. C., Liddell, E. G. T., and Sherrington, C. S. (1932). *Reflex Activity of the Spinal Cord*. Clarendon Press: Oxford.

- Eccles, J. C., Fatt, P., and Landgren, S. (1956). Central pathway for direct inhibitory action of impulses in largest afferent nerve fibres to muscle. *J. Neurophysiol.*, 19, 75-98.
- , Eccles, R. M., and Lundberg, A. (1957). Synaptic actions on motoneurons caused by impulses in Golgi tendon organ afferents. *J. Physiol. (Lond.)*, 138, 227-252.
- Gassel, M. M., and Diamantopoulos, E. (1964a). The Jendrassik maneuver. I. The pattern of reinforcement of monosynaptic reflexes in normal subjects and patients with spasticity or rigidity. *Neurology (Minneapolis)*, 14, 555-560.
- , — (1964b). The Jendrassik maneuver. II. An analysis of the mechanism. *Ibid.*, 14, 640-642.
- , Marchiafava, P. L., and Pompeiano, O. (1964). Tonic and phasic inhibition of spinal reflexes during deep, desynchronized sleep in unrestrained cats. *Arch. ital. Biol.*, 102, 471-499.
- , — (1965). Modulation of the recurrent discharge of alpha motoneurons in decerebrate and spinal cats. *Ibid.*, 103, 1-24.
- , and Pompeiano, O. (1965). Fusimotor function during sleep in unrestrained cats. An account of the modulation of the mechanically and electrically evoked monosynaptic reflexes. *Ibid.*, 103, 347-368.
- , and Ott, K. H. (1969). *Nature (Lond.)*, in press.
- Hagbarth, K.-E. (1952). Excitatory and inhibitory skin areas for flexor and extensor motoneurons. *Acta physiol. scand.*, 26, Suppl. no. 94.
- (1962). Post-tetanic potentiation of myotatic reflexes in man. *J. Neurol. Neurosurg. Psychiat.*, 25, 1-10.
- Hoffmann, P. (1922). *Untersuchungen über die Eigenreflexe (Sehnenreflexe) menschlicher Muskeln*. Springer: Berlin.
- Holmqvist, B. (1961). Crossed spinal reflex actions evoked by volleys in somatic afferents. *Acta physiol. scand.*, 52, Suppl. no. 181.
- Kugelberg, E., Eklund, K., and Grimby, L. (1960). An electromyographic study of the nociceptive reflexes of the lower limb. Mechanism of the plantar responses. *Brain*, 83, 394-410.
- Perl, E. R. (1958). Crossed reflex effects evoked by activity in myelinated afferent fibers of muscle. *J. Neurophysiol.*, 21, 101-112.
- Wartenberg, R. (1945). *The Examination of Reflexes*. Year Book Publishers: Chicago, Ill.