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PRESYNAPTIC INHIBITION EVOKED BY MUSCLE CONTRACTION

BY M. S. DEVANANDAN, ROSAMOND M. ECCLES AND D. STENHOUSE

From the Department of Physiology, Australian National University, Canberra, Australia

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SUMMARY

1. Contractions of flexor and extensor muscles of the knee and ankle were used to investigate presynaptic inhibition at the spinal level.

2. Contractions evoked dorsal root potentials, and increased the excitability of the central terminals of group Ia, Ib and low threshold cutaneous primary afferent fibres.

3. The monosynaptic reflexes recorded in response to stimulation of flexor or extensor muscle nerves were depressed, in the presence of strychnine hydrochloride 0·1 mg/kg I.V., by the contractions.

4. It is suggested that these presynaptic inhibitory effects are largely due to the activation of Golgi tendon organs by contraction.

INTRODUCTION

In previous publications (Devanandan, Eccles & Yokota, 1965*a*, *b*), it was shown that brief (10 msec) stretches of muscle could evoke presynaptic inhibition of groups Ia, Ib and cutaneous pathways. Stretch was used to activate stretch receptors in the muscle (i.e. to produce impulses in Ia, Ib and group II afferent fibres), and presynaptic inhibition was demonstrated by a variety of techniques; dorsal root potentials, depression of the monosynaptic reflex, depression of the monosynaptic excitatory post-synaptic potential, and changes in excitability of the primary afferent fibres. While muscle stretch is a useful means of stimulating the primary and secondary endings and the Golgi tendon organs, it has certain disadvantages. The stretch required to stimulate the Golgi tendon organs is very much greater than that required to stimulate the primary endings (Matthews, 1933) or secondary endings (Hunt, 1954; Lundberg & Winsbury, 1960). Therefore a stretch that activates the tendon organs also causes the primary and secondary endings to discharge repetitively. This makes it difficult to estimate the contribution of active tendon organs to presynaptic inhibition of primary afferent pathways.

Though tendon organs are activated by stretch, they are very much more sensitive to contraction. Jansen & Rudjord (1964*a*, *b*) have shown that whereas a stretch of several hundred grams may be needed to activate tendon organs, a small contraction of a few grams can stimulate these endings. Therefore they suggested that Golgi tendon organs may be contraction receptors rather than stretch receptors. Since contraction releases any stretch on the muscle spindles, activity in group Ia and II fibres may cease during contraction, depending on such conditions as initial stretch of the muscle and the stimulus strength employed to evoke the muscle contraction (Hunt & Kuffler, 1951*b*). Therefore, it may be possible to obtain pure group Ib impulses in the muscle nerve during contraction. For these reasons, presynaptic inhibition of afferent pathways was examined using contraction to activate tendon organs.

METHODS

Adult cats anaesthetized with pentobarbital (Nembutal, Abbot Laboratories) 40 mg/kg intraperitoneally were used. The tail and right hind limb were completely denervated. In the left hind limb, all nerves were cut except those to the muscle prepared for contraction, i.e. semitendinosus (ST), medial gastrocnemius (MG), flexor digitorum longus (FDL) or peroneus brevis (P. Brev.) The particular muscle was freed from surrounding tissue as completely as possible and a brass hook tied to its tendon. In addition, the following nerves were cut and prepared for stimulating and/or recording: posterior biceps (PB) or in combination with semitendinosus as PBST if the ST muscle was not used for contraction, sural (Sur), lateral gastrocnemius and soleus (LGS) or combined with medial gastrocnemius as (GS) when medial gastrocnemius (MG) contractions were not used, plantaris (Pl).

After a laminectomy from L_1 to L_7 the cord was cut between the L_1 and L_2 segments. The ipsilateral S_1 and L_7 ventral roots were dissected out and severed in such a manner that the central part could be used for recording and the peripheral part for stimulation.

Recording and stimulating techniques are illustrated in Fig. 1. The peripheral end of the cut S_1 or L_7 ventral root was stimulated with a single shock or with four volleys at 250/sec. The stimulus strength was kept at twice threshold except for the experiment illustrated in Fig. 5. The resulting contraction of the muscle was recorded isometrically, using a strain gauge (Statham G1-80-35). The initial tension of the muscle was between 20 and 30 g.

To record the dorsal root potentials (DRPs) a filament of the dorsal root was dissected out from the ipsilateral lower L_6 or the upper L_7 dorsal root and mounted on platinum electrodes. The signal was amplified, using an amplifier system with a time constant of 1 sec and displayed on the cathode ray oscilloscope (Barron & Matthews, 1938).

The excitability of the afferent nerve terminals was tested using the technique described by Wall (1958) and as used by Eccles, Schmidt & Willis (1963*a*, *b*). A low resistance, sodium chloride filled micro-electrode was inserted into the spinal cord, close to the terminals of the primary afferent fibres. A square pulse of 0·2 msec duration was passed through the micro-electrode, using a Grass stimulator (S4E) and its isolating unit (SIU-4B). The action potential was recorded antidromically in the peripheral nerve. This testing situation was conditioned by the muscle contraction evoked by stimulation of the ventral root. It is worth emphasizing that excitability changes of the magnitude illustrated in Fig. 6 were not always seen. It requires very careful tracking with the micro-electrode to find the position at which maximum excitability changes are observed. A small movement in any direction from this point will produce curves showing only small changes in excitability.

The PBST or GS monosynaptic reflex recorded in the central end of L_7 or S_1VR in response to stimulation of the PBST or GS nerves, was used as a second means of testing the presynaptic inhibition of group Ia afferents (Eccles, Schmidt & Willis 1962). When using this method intravenous strychnine hydrochloride 0·1 mg/kg was given to avoid any complication of the results from post-synaptic inhibition (Bradley, Easton & Eccles, 1953).

The temperature of the cat was between 36 and 38° C, and the back and leg oil pools were kept warm with heaters.

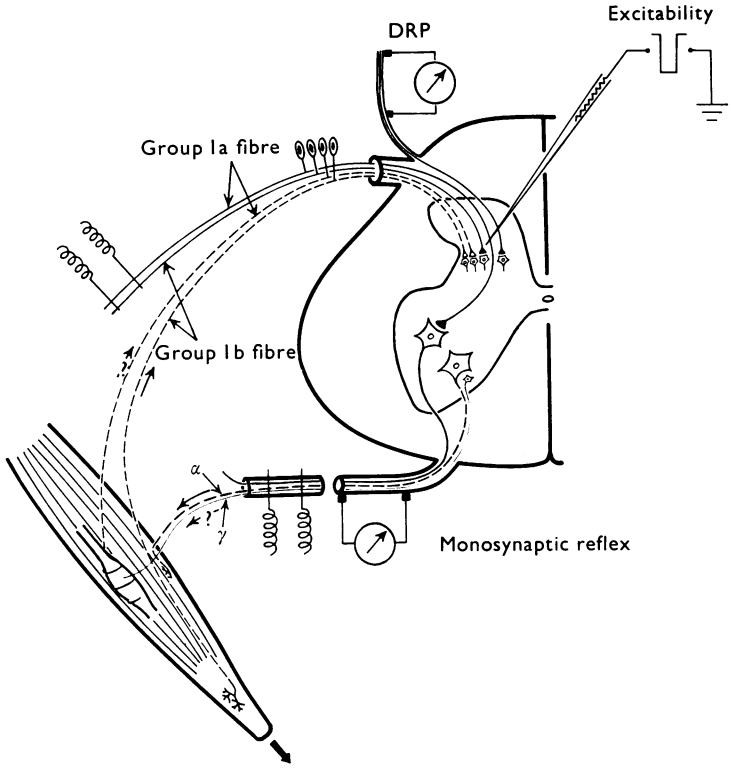

Fig. 1. Schematic figure illustrating the recording and stimulating techniques employed. Stimulation of the peripheral end of the cut ventral root causes the muscle to contract, thereby stimulating the Golgi tendon organs. The dorsal root potential (DRP) is recorded from the cut central end of a thin dorsal root filament. To measure the excitability of the central terminals of primary afferents, a micro-electrode is inserted near the endings of the required afferent pathway (here shown close to the group Ib endings). To record the monosynaptic reflex, the peripheral nerve is stimulated and the reflex is recorded from the central end of the ventral root. In the figure question marks are drawn against the fusimotor fibre to the muscle and the group Ia fibre from the muscle, since the stimulus strength (twice threshold) may have straddled the threshold for a few fusimotor fibres and therefore evoked some impulses from the spindle afferents during contraction.

RESULTS

When a muscle contracts, impulses will be generated by the Golgi tendon organs (Matthews, 1933; Hunt & Kuffler, 1951*b*). These impulses travelling centrally in the Ib fibres, depolarize the central terminals of group I and large cutaneous primary afferent fibres within the spinal cord (Eccles *et al.* 1963*a*, *b*). This depolarization can be recorded by electrotonic conduction in dorsal root filaments as the dorsal root potential (DRP) (Barron & Matthews, 1938) or by intra-fibre recording as primary afferent depolarization (Eccles *et al.* 1963*a*, *b*). Figure 2 illustrates DRPs recorded from a lower L_6 dorsal root filament following a contraction of the medial gastrocnemius muscle. The force of this contraction was altered in

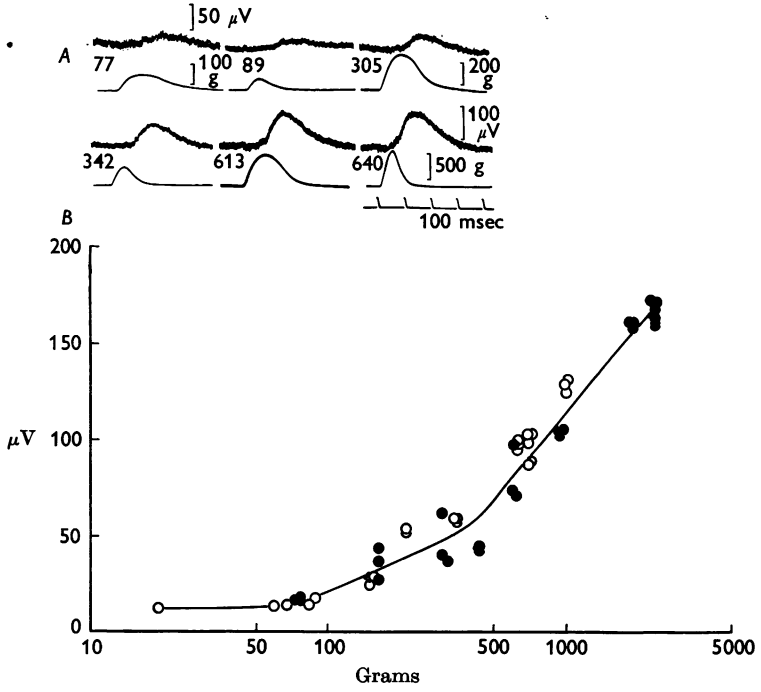

Fig. 2. The relation between the size of the dorsal root potential (DRP) and the force of muscle contraction. *A*. Specimen records of DRPs (top line) with their respective contractions (bottom line). The figures on the contraction records give the force of the contraction in grams. The DRPs of 89, 342 and 640 and open circles of *B* were in response to one volley to the ventral root while the rest of the records and the filled circles of *B* were evoked by four volleys (250 c/s). The DRPs were all recorded at 100 μV potential scale, with the exception of the first at 50 μV. The tension scale of 100 g applies to 77; 200 g to 89, and 305 and 500 g to the remainder. All records on the same time scale. *B*. Graph relating the size of the DRP (μV-ordinate) to the force of contraction (g-abscissa). Note the logarithmic scale of the abscissa.

two ways; (*a*) by stimulation of the S_1 ventral root with single or repetitive volleys (the tension developed during a contraction in response to four volleys at 250 c/s being greater than that due to a single shock), (*b*) by stimulating only a fraction of the ventral root so that fewer motor fibres are activated and there is therefore a corresponding reduction in tension developed by the muscle. In Fig. 2*A*, contractions are illustrated below the DRPs they evoke. Contractions of 77 or 89 g give small but definite DRPs. Increase in contraction to 305 g and later to 613 g led to larger DRPs. It may be noted in Fig. 2*B* that the size of the DRP is related to the tension developed by the muscle, irrespective of whether a single shock (open circles) or four volleys (filled circles) were applied to the S_1VR. It has already been noted that DRPs indicate the presence of presynaptic inhibition but give no indication of the type of afferent fibres depolarized, and other techniques will give more exact information (Eccles, Schmidt & Willis, 1962; 1963*a*, *b*).

Presynaptic inhibition of the group Ia pathway was examined by two methods: depression of the monosynaptic reflex, and the changes in excitability of the primary afferent fibres. The size of the monosynaptic reflex is a sensitive index of the state of the appropriate motoneurone pool and in the spinal cat any reduction of reflex amplitude could be due either to a reduction of the presynaptic input or to post-synaptic inhibition of motoneurones. If post-synaptic inhibitory changes are precluded by the use of adequate doses of strychnine, then any depression of the monosynaptic reflex may be attributable to presynaptic inhibition. Figure 3 illustrates the effect of contraction of the FDL muscle on the facilitated GS reflex (i.e. the effect of the contraction of an extensor muscle on an extensor reflex). The GS reflex to a single volley in the GS nerve was rather unstable, so it was facilitated by a slightly earlier (2 msec) stimulus to the same nerve which alone did not evoke a reflex. This stable reflex was then preceded by a contraction of the FDL muscle, stimulated by a single shock (Fig. 3*A*, *B* and *C*) or four volleys at 250/sec (Fig. 3*D*, *E* and *F*) applied to the L_7VR. It will be seen that contraction causes a long-lasting depression of the reflex. Since this depression persists in the presence of 0·1 mg/kg strychnine hydrochloride as seen by a comparison of open circles with filled circles in Fig. 3*C* and *F*, post-synaptic inhibition is not a major contributing factor (Bradley *et al.* 1953). Similarly, if the monosynaptic reflex of an extensor muscle was conditioned by the contraction of a flexor muscle, a large depression of the reflex was seen, with a peak at 20–30 msec, and recovery over a time course of 140 msec (strychnine being present). By reducing the force of the contraction the amount of reflex depression could be reduced.

A comparison of Fig. 3*C* with *F* (open circles) shows that the pre-

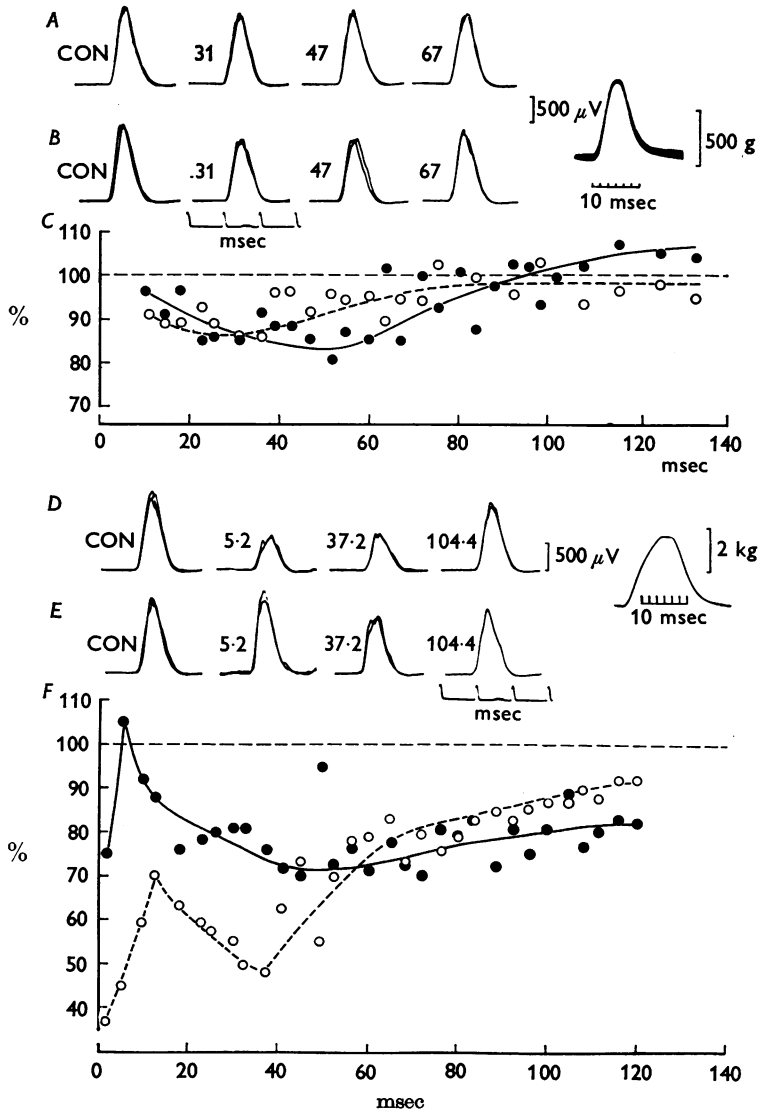

Fig. 3. The GS facilitated monosynaptic reflex in the S_1VR was conditioned by a contraction of the FDL muscle in response to one volley to the L_7VR (*A*,*B*,*C*) and to four volleys (*D*,*E*,*F*). *A*,*D* and open circles in *C* and *F* show the reflex depression before the administration of strychnine. *B*,*E* and filled circles in *C*,*F* show the same after strychnine. The first records in *A*,*B*,*D* and *E* are the control reflexes (CON); the numbers on the records give the time in msec between the conditioning and testing stimuli. The contraction to one volley is given between *A* and *B* 500 g scale; for four volleys between *D* and *E* (2 kg scale). All reflexes were recorded on the same time and potential scales.

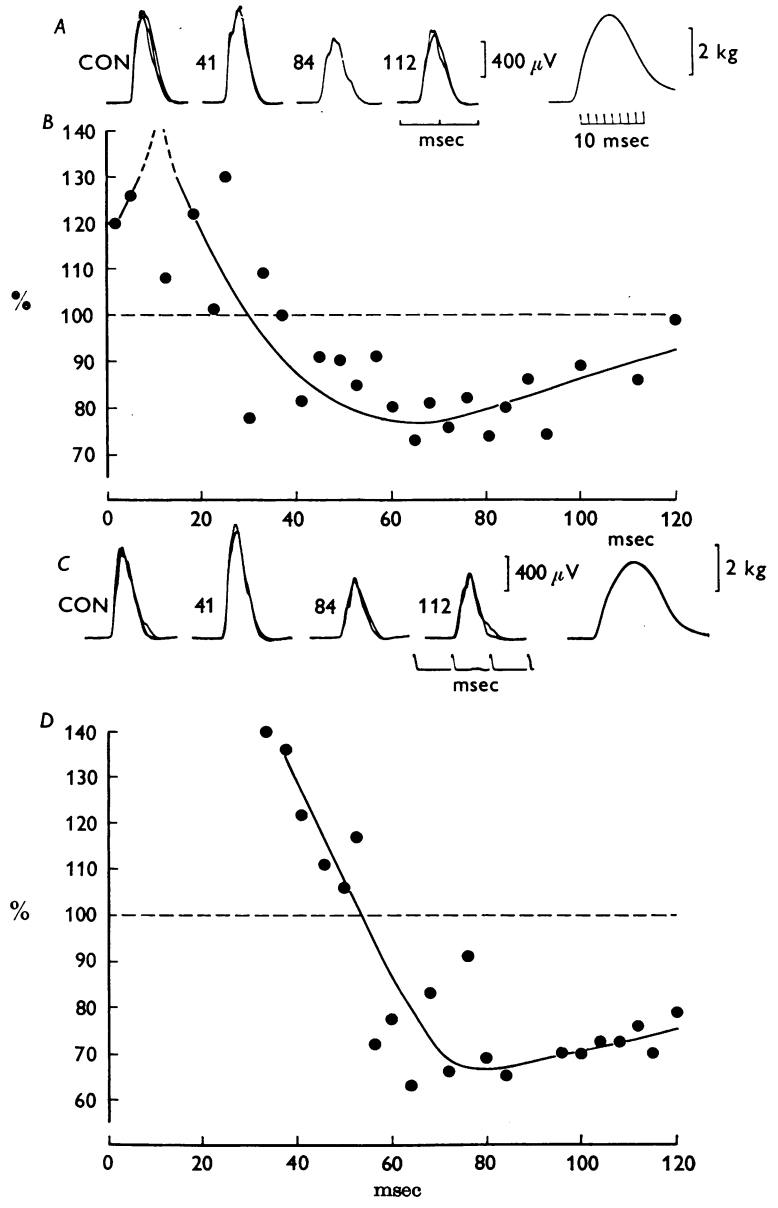

Fig. 4. The unfacilitated PBST monosynaptic reflex was conditioned by a contraction of the FDL muscle, the contraction being evoked by four volleys delivered to the L_7VR. *A* and *B* show the depression of the reflex before and *C* and *D* after the administration of strychnine. Reflexes recorded on the same potential and time scales; both contractions have the same tension and time scales. CON and numbers on the records—as for Fig. 3.

synaptic inhibition evoked by a tetanic contraction is greater than that produced by a twitch, and has also a later and more prolonged peak and a slower recovery phase. Since the contraction is more powerful when four volleys are used, the difference is presumably due to the increased frequency and duration of receptor firing (Matthews, 1933).

In Fig. 4, the effect of conditioning the monosynaptic reflex from a flexor muscle, posterior biceps-semitendinosus (PBST), by a contraction of an extensor (FDL) is illustrated. Impulses in group Ib afferents from the FDL have been shown to produce a post-synaptic excitation of the PBST motoneurones (Eccles & Lundberg, 1959). This can be seen by the early facilitation of the monosynaptic reflex in Fig. 4*B* and *D*. There is, however, a late depression starting with a latency of about 30 msec in Fig. 4*B*. After strychnine (Fig. 4*D*) it can be seen that the early facilitation is more prolonged, and depression now appears later at about 50 msec.

If the stimulus applied to the ventral root is increased so that the fusimotor fibres are excited, the spindle receptors in the muscle will fire during the contraction (Matthews, 1933; Leksell, 1945). In an animal given intravenous strychnine, the facilitated monosynaptic GS reflex was conditioned by a contraction of P. Brev. muscle evoked by a short repetitive stimulation of the peripheral end of the cut L_7VR. It can be seen from Fig. 5*A* that the depression resulting from a contraction in response to a stimulus, 20 times threshold, is greater than that produced by a contraction in response to a stimulus that is twice threshold at the early intervals; although after 10 msec the stronger contraction adds only 5% to that effected by the smaller contraction, and the time courses are identical. In Fig. 5*B* when the conditioning P. Brev. contraction occurred at a fixed interval of 27 msec before the test reflex, the effect of varying the stimulus which evoked the conditioning contraction from just threshold to 20 times threshold shows that almost maximum depression is obtained at twice threshold. At the end of the experiment, the nerve to P. Brev. was cut and mounted on electrodes and the compound action potential was recorded in the nerve in response to stimulation of the ventral root (Fig. 5*D*)—arrows indicate the start of the γ efferent component of the compound action potential (calculated on the basis of conduction velocity). The threshold for this γ component was twice that for α efferent fibres and a stimulus five times the α threshold stimulates virtually all the γ fibres (cf. Boyd & Eccles, 1963).

Since recording DRPs as in Fig. 2 is not a very specific method of demonstrating primary afferent depolarization (Eccles, Magni & Willis, 1962) the method of excitability testing was used to demonstrate depolarization of the central terminals of various types of primary afferents (Wall, 1958; Eccles *et al.* 1963*a*, *b*). A pulse was passed through a low resistance

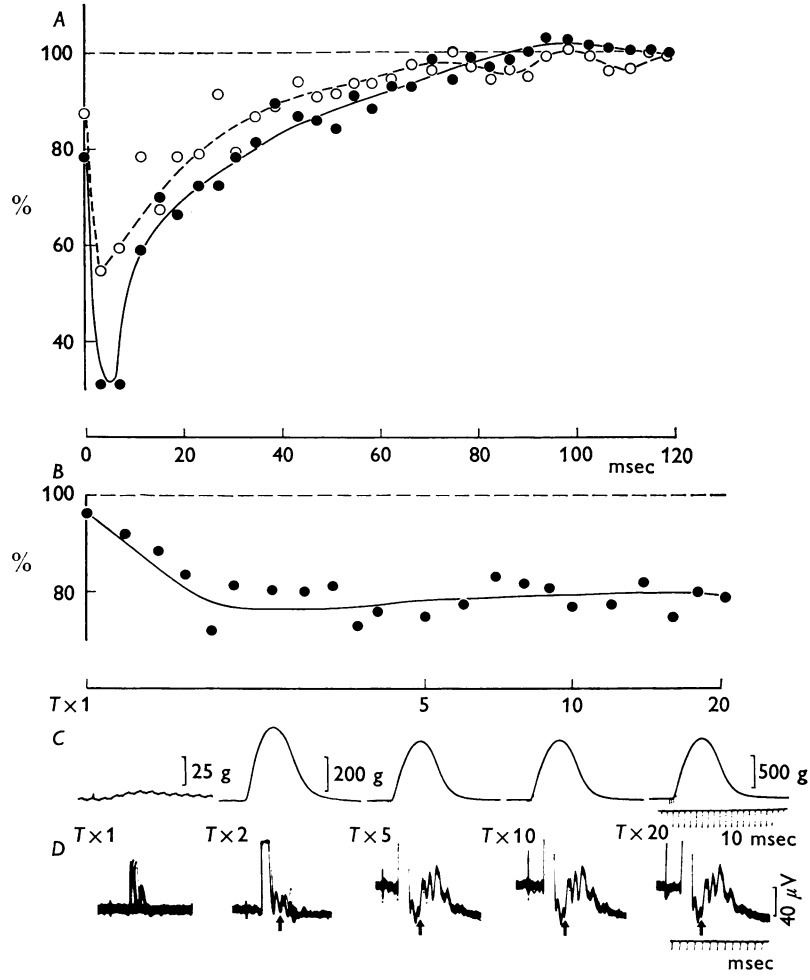

Fig. 5. *A*, shows the time course of the depression of a facilitated GS monosynaptic reflex (as a percentage of the control) when conditioned by contractions of the P. Brevis muscle evoked by repetitive stimulation (four volleys at 250/sec) of the L_7VR. Open circles show the depression when the contractions were evoked by stimuli of twice threshold and filled circles when they were 20 times threshold for the α motor fibres. In *B*, the L_7VR was stimulated at a fixed interval of 27 msec before the testing reflex. The reflex depression is plotted (as a percentage of the control) against the stimulus strength (given in times threshold) used to evoke the contraction of the P. Brevis. *C*, illustrates the size of the contractions in response to the various stimuli used in *B*; and *D*, the corresponding compound action potential recorded in the peripheral nerve. The stimulus strength in times threshold is marked above the records of the action potential. The arrows mark the elevation due to activity in γ motor fibres.

micro-electrode which was located at the point where focal synaptic potentials from the primary afferent fibres was greatest, and the resulting antidromic action potential was recorded in the peripheral nerve (all the ventral roots having been sectioned earlier). This was then conditioned by a contraction of a muscle and any change in the size of the action potential recorded. If depolarization occurs, the central terminals become more excitable and the recorded action potential will then be larger, provided all terminals near the electrode are not stimulated by a given pulse. Figure 6*C* and *F* show the excitability changes of the group Ia afferent terminals of LG (Fig. 6*C*) and PBST (Fig. 6*F*), evoked by contraction of the medial gastrocnemius muscle. These results indicate that the depression of the monosynaptic reflex when conditioned by a contraction, can

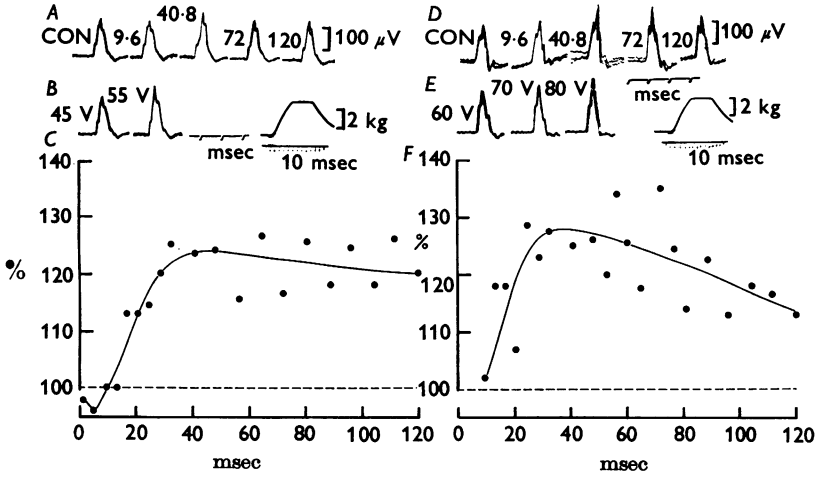

Fig. 6. The changes in excitability of the central terminals of the group Ia afferent fibres of the LG nerve, when conditioned by a contraction of the MG muscle is shown in *C* (specimen records in *A*). The control (CON in *A*) was evoked by a stimulus of 45 V through the micro-electrode. In remaining records of *A* numbers indicate the interval in msec between the conditioning contraction and the response to 45 V. The calibration records are given for this series in *B* (see Eccles, Schmidt & Willis, 1963*a* for full details). In the same experiment *D*–*F* give the excitability change of the PBST Ia primary afferent fibres to the same conditioning contraction. The potentials in *A*, *B* and *D*, *E* have their appropriate potential and time scales whilst the contractions (end of row *B* and *E*) their own tension and time scales.

be attributed to depolarization of the presynaptic terminals, which gives rise to presynaptic inhibition. The time course of the depression of the monosynaptic reflex approximates to the time course of the change in excitability of the terminals of the group I afferents, provided an extensor reflex is conditioned by a flexor or an extensor muscle (cf. Figs. 3 and 5 with Fig. 6*C*). But, on comparing Fig. 6*F* with Fig. 4*B* and *D*, it will be

seen that the time course of the change in excitability of the flexor group Ia fibres does not follow the time course of the reflex depression. This asymmetry was always found when testing flexor Ia pathways; the conditioning stimulus could be contractions or stretch of extensor or flexor muscles, or electrical stimulation of muscle nerves at group I strength. This increase in the flexor monosynaptic reflex is thought to be due to facilitation of the motoneurones of the flexor muscles by activity in the group Ib fibres (Eccles & Lundberg, 1959). To examine presynaptic inhibition in pathways other than the group Ia, the method of excitability testing was always used (Eccles *et al.* 1963*a*, *b*). The micro-electrode was placed in the region where focal synaptic potentials from group Ib primary afferents were greatest. The action potential produced in response to a stimulus through the micro-electrode was recorded in the peripheral nerve. Conditioning this potential by a contraction of a muscle (either flexor or extensor) resulted in an increase in excitability with a peak at 30 msec and a duration greater than 140 msec. Similarly the excitability of low threshold cutaneous afferents was tested. Contractions of both flexor and extensor muscles evoked an increase in excitability with a peak at 28–33 msec and a duration of more than 140 msec.

DISCUSSION

The sensory nerve fibres from mammalian muscle are classified into groups I, II and III (Lloyd, 1943). These fibres originate from various sensory receptors within the muscle. Group I fibres are subdivided into group Ia which originate from the primary endings in the spindle and group Ib from the tendon organs (Hunt, 1954). The group II fibres originate from the secondary endings in the spindle (Hunt, 1954). Our knowledge of the receptors from which the group III fibres originate is limited, though they are activated by pressure, painful stimulation and to some extent by contraction (Bessou & Laporte, 1960; Paintal, 1960).

When stimulation of motor fibres results in muscle contraction, different types of receptors will discharge, depending on such factors as initial tension of the muscle, and strength of the stimulus used (Matthews, 1933; Hunt & Kuffler, 1951*b*).

As noted in the introduction, contraction will consistently stimulate the tendon organs (Matthews, 1933; Hunt & Kuffler, 1951*b*; Jansen & Rudjord, 1964*a*, *b*). It can be assumed, therefore, that in the present experiments, impulses would always have been generated in tendon organs by the contractions of the muscles used. Receptors other than tendon organs may also have discharged during the contraction. If the stimulus strength is sufficient to stimulate the fusimotor fibres, the primary endings will respond with a discharge during contraction (Matthews, 1933; Leksell,

1945; Hunt & Kuffler, 1951*a*; Kuffler, Hunt & Quilliam, 1951). Boyd & Eccles (1963) have shown that the threshold for small motor fibres in the ventral root lies between 1·5 and 2·2 times the threshold for α motor fibres. Therefore, in our experiments even with stimuli of twice threshold, we may have been stimulating a few fusimotor fibres and thereby causing some discharge of spindle afferents during contraction. Further, Bessou, Emonet-Dénand & Laporte (1963) and Adal & Barker (1965) have shown that there is 'mixed motor innervation' in the deep lumbrical muscle of the cat, i.e. a motor axon innervating both extrafusal and intrafusal muscle fibres. Brown, Crowe & Matthews (1965) have provided physiological evidence that this may occur in the tibialis posterior muscle. Hence the possibility of spindle activation by mixed motor fibres cannot be excluded in the present series of experiments. Primary spindle afferents may have affected these experiments in another way. Hunt & Kuffler (1951*b*) described an early discharge in spindle afferents after stimulation of the ventral root or the whole nerve to muscles (see also Granit, Pompeiano & Waltman, 1959).

The possibility of secondary endings discharging during contraction will depend on (*a*) how isometric the contraction was, (*b*) initial tension, (*c*) stimulation of γ motor fibres (Harvey & Matthews, 1961; Bessou & Laporte, 1962). Early discharge from secondary endings, though present, is less common than from primary endings and from Golgi tendon organs. The greater the initial tension the more likely is this early discharge (Hunt & Kuffler, 1951*b*), whereas repetitive stimulation would reduce it (Granit *et al.* 1959). In these experiments the initial tension was kept between 20 and 30 g. It is likely therefore that a few group II endings discharged during the contraction. Similarly group III endings can sometimes be activated by contraction (Paintal, 1960).

Impulses in group Ia fibres produce presynaptic inhibition only of the group Ia pathway and have no effect on other afferent pathways (Eccles, Kostyuk & Schmidt, 1962; Eccles *et al.* 1963*a*, *b*). Therefore, in the present paper, the results illustrating depression of the monosynaptic reflex by contraction, and those showing changes in excitability of group Ia primary afferent fibres by contraction, may have been produced by activation of a few primary spindle endings, as well as tendon organs. However, in the experiment illustrated in Fig. 5, when the group Ia endings were deliberately activated by stimulating the fusimotor fibres there was an increase in reflex depression, particularly at the early intervals. At late intervals, this increase is no more than 5%. Since the stimulus used for muscle contraction was carefully kept at twice threshold, and since the amount of reflex depression is seen to vary with the force of the contraction (Fig. 3), which will affect tendon organs rather than spindle endings,

the depression of the monosynaptic reflex must be due largely to the presynaptic inhibitory effects evoked by active Golgi tendon organs. Impulses generated by group II and III endings will affect the results only with respect to the DRPs and the excitability of Ib afferents and skin afferents (Eccles *et al.* 1963*a*, *b*). It can be seen in Fig. 2 that the DRP keeps increasing in size, as the force of contraction is increased, suggesting that as the force of contraction increases a greater number of receptors are stimulated, and their rate and duration of firing increased, thereby producing more depolarization of primary afferent fibres. Unfortunately, tensions above 2 kg could not be accurately recorded and it was not possible to relate the size of the DRP to force of contraction above this value. It is probable that if only the Golgi tendon organs were stimulated by contraction, that the DRP would have kept increasing until the contraction reached a value where the optimum discharge of tendon organs resulted from each contraction and then remained at a constant value. The increase of the DRP with contractions greater than 2 kg may be due to stimulation of receptors other than tendon organs. Because the size of the DRP varies only with the force of the contraction between 20 g and 2 kg, and is independent of the number of stimuli, it is strong evidence that activity in tendon organs was mainly responsible for the DRP within this range.

It is noteworthy that, in this work, contractions of extensor muscles produce a considerable amount of presynaptic inhibition of the group Ia pathway (cf. Figs. 3, 4 and 6). This does not entirely agree with the results reported using electrical stimulation of muscle nerves (Eccles, Magni & Willis, 1962), although Eccles, Schmidt & Willis (1962) do illustrate graphs showing some presynaptic inhibition of monosynaptic reflexes, due to stimulation of extensor muscle nerves. (It should be noted that the quadriceps femoris has always been treated as an exception to the generalization that extensors do not produce presynaptic inhibition of Ia afferents.) However, in these experiments we used very powerful contractions of extensor muscles to show presynaptic inhibition of the Ia pathway. This suggests that powerful extensor muscle contraction causes presynaptic inhibition of the group Ia pathway, whereas electrical stimulation of the muscle nerve with four volleys may not do so to the same degree.

On the basis of the evidence presented in this paper, presynaptic inhibition of Ia, Ib and low-threshold cutaneous pathways results from muscle contraction and it is suggested that this is produced mainly by activation of Golgi tendon organs.

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