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CUTANEOUS FACILITATION OF TRANSMISSION IN REFLEX PATHWAYS FROM Ib AFFERENTS TO MOTONEURONES

BY A. LUNDBERG, KRISTINA MALMGREN AND E. D. SCHOMBURG[1]

From the Department of Physiology, University of Göteborg, Sweden

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SUMMARY

1. The effect of volleys in low threshold cutaneous afferents upon transmission of synaptic action from Ib afferents to motoneurones has been investigated with intracellular recording from alpha motoneurones to hind limb muscles.

2. There was facilitation from cutaneous afferents of transmission in excitatory and inhibitory reflex pathways from Ib afferents without any evidence for difference in effect on di- and trisynaptic pathways. It is postulated that volleys in cutaneous afferents evoke excitatory action in interneurones of these reflex pathways.

3. The time course of the facilitation suggests that cutaneous afferents have disynaptic excitatory connexions with the interneurones intercalated in the disynaptic Ib inhibitory pathways to motoneurones.

4. Some observations are reported suggesting that interneuronal transmission in Ib inhibitory pathways to motoneurones might be facilitated from Ia afferents.

5. The findings are discussed in relation to the presumed role of Ib reflex action in regulating muscle tension.

INTRODUCTION

It is now generally accepted that impulses in Ib afferents from Golgi tendon organs may contribute to reflex regulation of normal movements and not only provide protection against excessive tension. This view has gained strength from the findings that these receptors are very sensitive provided the appropriate motor units are activated (Houk & Henneman, 1967). Houk, Singer & Goldman (1970) have discussed Ib inhibition in terms of a

* Present address: Physiologisches Institut der Universität, 34 Göttingen, Humboldtallee 7, GFR.

force feed-back which may be employed to keep tension constant. In this connexion it is of interest that there is an intricate control from higher centres of interneuronal transmission of Ib reflex pathways. These interneurones can be inhibited by two systems originating from the brain stem, the dorsal reticulospinal system and the noradrenergic reticulospinal tract (Andén, Jukes, Lundberg & Vyklický 1966; Engberg, Lundberg & Ryall, 1968). Activity in the former system provides the tonic inhibition of transmission in the Ib reflex pathways found in the decerebrate state (R. M. Eccles & Lundberg, 1959*a*). Facilitation of reflex transmission from Ib afferents is evoked from the corticospinal and rubrospinal tracts, in both cases probably by monosynaptic excitatory action on the interneurones (Lundberg & Voorhoeve, 1962; Hongo, Jankowska & Lundberg, 1969; Illert, Lundberg & Tanaka, 1976). All these findings suggest that the brain has ample possibilities for regulating transmission in the Ib reflex pathways, and this setting of interneuronal transmittability may be an important part of a brain command for muscular force. In fact the original suggestion that the Ib reflex actions function to regulate tension in normal movements was partly based on findings showing that interneuronal transmission in the Ib reflex pathway was controlled from higher centres (Lundberg, 1959). In the present investigation it will be shown that interneuronal transmission in the Ib reflex pathways is facilitated at a segmental level by impulses in cutaneous afferents. This effect is taken to indicate the existence of a segmental regulatory mechanism allowing adjustment of Ib tension control during movement. A preliminary report has been issued (Lundberg, Malmgren & Schomburg, 1975).

METHODS

The experiments were performed on fourteen low spinal cats anaesthetized with chloralose (50–60 mg/kg). Small doses of pentobarbitone sodium (Nembutal; 2–5 mg/kg) were added during most of the experiments. The animals were immobilized with gallamine triethiodide (Flaxedil) and artificially respired. The expiratory CO_2 concentration, arterial blood pressure and rectal temperature were monitored continuously.

The ventral roots L7–S1 and L6 were cut and mounted for stimulation. The following hind limb nerves were dissected and stimulated: quadriceps (Q), posterior biceps and semitendinosus (PBSt), anterior biceps and semimembranosus (ABSm), gastrocnemius and soleus (G-S), flexor digitorum and hallucis longus (FDL), plantaris (Pl), the plantar section of the tibial nerve (Tib), tibialis anterior and extensor digitorum longus (DP), sural (Sur), saphenous (Saph), skin division of the superficial peroneal nerve (SP). The stimulus strength is expressed in multiples of threshold for the nerve ($\times T$). The strength for maximal group I and, when present, for the slow and fast components of the group I volley (only in Q, PBSt and ABSm) was determined by the double volley technique (Bradley & Eccles, 1953). Rectangular pulses of 100 μsec duration were used for stimulation.

Intracellular recording from motoneurones in L6 and L7 was made with 2 M-K-

citrate micro-electrodes. In order to investigate the convergence from cutaneous and Ib afferents at an interneuronal level the occurrence of spatial facilitation was tested. This technique has been employed to demonstrate convergence from different muscle afferents on to interneurones in the Ia reflex pathway (Fedina & Hultborn, 1972).

Averaging was performed in a sequential mode for conditioned and unconditioned responses (10–50 sweeps each). A computer with an integrating analog to digital converter was used with a time resolution of 0·125–0·25 msec/address.

RESULTS

Ib i.p.s.p.s. In the description of our results we will adhere to the generally accepted hypothesis that inhibitory actions from group I muscle afferents on motoneurones, other than the Ia reciprocal disynaptic action from antagonists, are evoked from Ib afferents (cf. Eccles, Eccles & Lundberg, 1957*a*). However, at the end of this section some results are reported which suggest that this assumption should be reconsidered.

Fig. 1 illustrates the principal findings of the present investigation. *A* and *B* show the synaptic effects in a G-S motoneurone evoked by separate volleys in cutaneous afferents (*A*) and in group I afferents from plantaris (*B*). The cutaneous SP nerve was stimulated at 1·2 times threshold at which strength there was only a liminal inhibitory post-synaptic potential, i.p.s.p. The stimulus strength for the plantaris nerve, 1·4 times threshold, was submaximal for group I afferents. The i.p.s.p. resulting from combined stimulation of the two nerves in *C* is considerably larger than the algebraic sum of the effects in *A* and *B* which indicates spatial facilitation in the interneuronal pathway mediating the i.p.s.p. Inspection of records *B* and *C* in addition reveals a latency shortening from 1·5 to 1·4 msec which also suggests interneuronal facilitation. A latency within this range is clearly indicative of a disynaptic action and our findings thus suggest that cutaneous impulses evoke excitatory action in Ib inhibitory interneurones projecting directly to motoneurones. The same spatial facilitation is shown in the averaged records in *D* and *E* employing a somewhat weaker plantaris test. The facilitation appears best from a comparison of the unconditioned group I i.p.s.p. from Pl (second trace in D) with the difference between the superimposed traces in *E*, which are the first and third traces in *D*. The time course in the graph (*J*) was obtained by varying the conditioning-test interval and refers to the disynaptic i.p.s.p. from Pl. Observe that there is virtually no facilitation when the conditioning and testing volleys arrive simultaneously to the spinal cord and that maximal facilitation seems to occur at an interval of about 2 msec. Accordingly, it is postulated that the cutaneous fibres do not act monosynaptically on the Ib inhibitory interneurones.

Some spatial facilitation was found also when the arrival of the Pl

group I volley preceded the SP volley or arrived simultaneously (I and *H*; see also Fig. 3*A* and *D*). However, latency measurements at different conditioning-testing intervals revealed that in this situation the onset of the i.p.s.p. was time-locked to the SP volley, the latency being 2·1 msec from the arrival of the SP volley to the spinal cord. This latency suggests

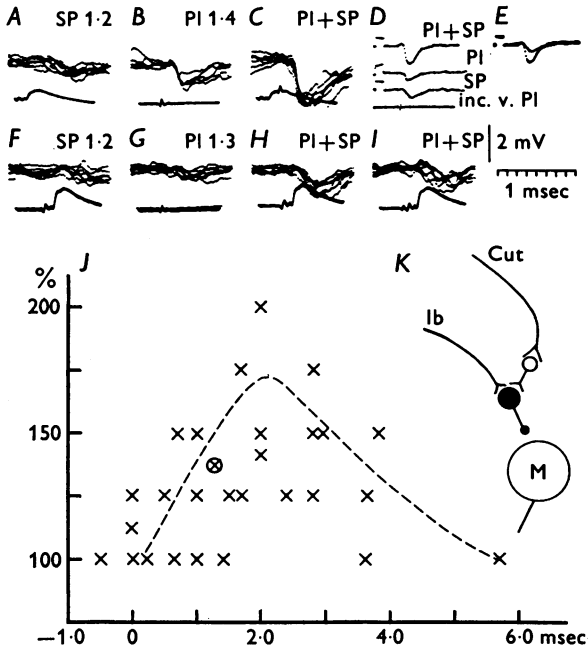

Fig. 1. Facilitatory interaction in inhibitory transmission to motoneurones from Ib muscle afferents (PI) and cutaneous afferents (SP). Upper traces, intracellular recording from a G-S motoneurone. Lower traces, incoming volleys recorded from the L7 root entry zone. *D*, averaged intracellular records (twenty-five responses each) taken in an alternating manner for combined and single nerve stimulation; fourth trace averaged incoming volley (inc. v.) from Pl. *E*, first (Pl + SP) and third (SP) trace from *D* superimposed: the difference between them compared with second trace in *D* gives the amount of facilitation. Calibration pulse in *D* and *E*, 0·5 mV amplitude and 2 msec duration. Stimulus strengths are indicated in multiples of threshold for the nerve. In *H–I* the conditioning-testing interval is changed with the Pl volley preceding the SP volley. *J*, time course of spatial facilitation of the Ib i.p.s.p. obtained by varying the conditioning-testing interval, averaged response in circle. Positive time means that stimulation of SP was preceding. *K*, wiring diagram of the presumed linkage, see text.

that the facilitated cutaneous i.p.s.p. was transmitted by a trisynaptic pathway (cf. Lundberg, 1975). Combining these results with the SP facilitation of the disynaptic i.p.s.p. in *A–E* we arrive at the tentative

wiring diagram shown in *K*, i.e. a convergence of monosynaptic Ib excitation and disynaptic cutaneous excitation on inhibitory interneurones projecting directly to motoneurones. The time course of the facilitation in graph *J* is compatible with the linkage suggested in *K*.

Volleys in cutaneous afferents also facilitate transmission in the autogenetic Ib inhibitory pathways and in Ib pathways from close synergists giving heteronymous Ia excitatory post-synaptic potentials, e.p.s.p.s, in the motoneurone. The records in Fig. 2 show the i.p.s.p.s evoked in a

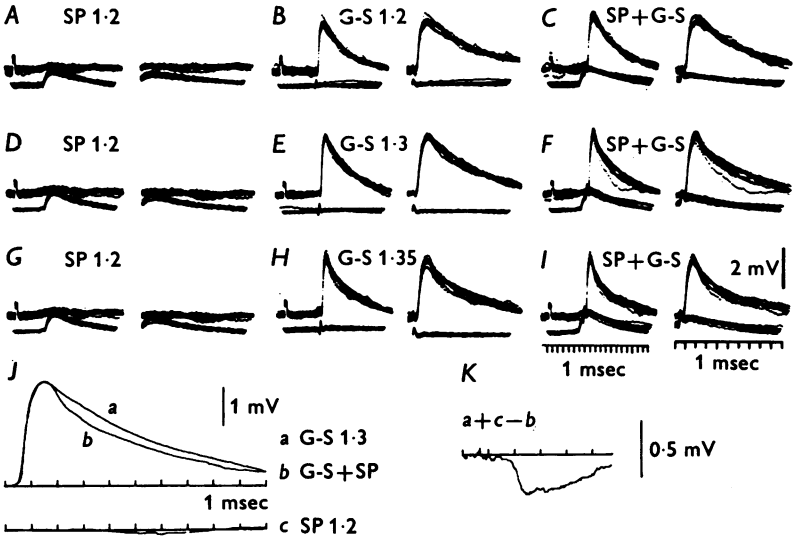

Fig. 2. Facilitation from cutaneous afferents (SP) of transmission in the autogenetic Ib inhibitory pathway. Intracellular recording from a G-S motoneurone. In *A–I* each record is displayed with two speeds (calibration in *I*) at the three testing stimulus strengths indicated. Stimulation of SP was liminal for evoking a p.s.p. Observe that there is a faster decay of the e.p.s.p. with combined stimulation in *F* than in *E* with unconditioned stimulation but no corresponding difference at the weaker testing strength in *B* and *C*. In *J* these p.s.p.s (*E* = *a* and *F* = *b*) are drawn superimposed and the liminal i.p.s.p. from SP (*D* = *c*) drawn below. In *K* the difference between the curves ($a+c-b$) is computed showing the time course of the facilitated i.p.s.p.; observe the different amplifications in *J* and *K*. Zero time in *J* and *K* is the incoming volley from G-S; the facilitated Ib i.p.s.p. has a central latency of 1·9 msec.

G-S motoneurone from the G-S nerve at three different group I strengths. The conditioning cutaneous SP volley was liminal for evoking a p.s.p. in the motoneurone. Observe in *F* that at 1·3 times threshold there is a faster decay of the e.p.s.p. during combined stimulation of the SP and G-S nerve

than of the unconditioned e.p.s.p. (*E*); the two traces are superimposed in *J* and the difference between them shown in *K* (cf. legend). This faster decay suggests the appearance of a superimposed Ib i.p.s.p brought about by interneuronal facilitation from SP. The alternative explanation that the SP volley produced 'remote' dendritic inhibition changing the time constant of the dendritic membrane to give a faster passive e.p.s.p. decay is virtually excluded by a comparison with the records in *B* and *C* obtained at weaker group I strength. In this case the SP volley does not give any change in the rate of decay of the e.p.s.p.s, the likely explanation

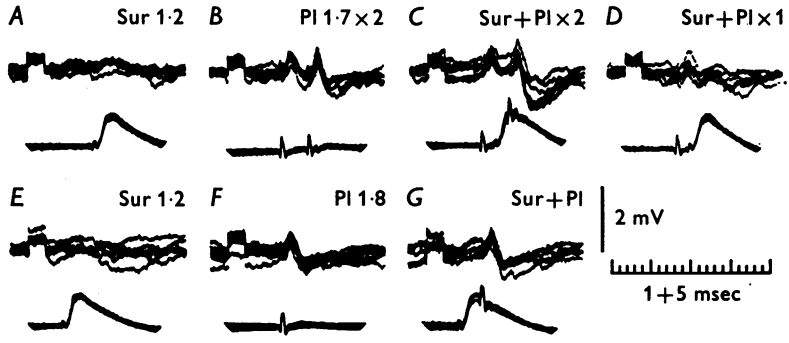

Fig. 3. Facilitatory interaction between i.p.s.p.s from Ib afferents of close synergists giving Ia e.p.s.p.s (Pl) and i.p.s.p.s from cutaneous afferents (Sur). Intracellular recording from a G-S motoneurone. In *B* and *C* Pl is stimulated with a double volley to enhance the test i.p.s.p. through temporal facilitation. The omission of the second stimulus in *D* shows that the i.p.s.p. in *C* is largely evoked by the second group I volley.

being that the stimulus strength now was subthreshold for evoking Ib i.p.s.p.s even during SP facilitation. At slightly higher I group strength, 1·35 times threshold (*H*), also the unconditioned Ia e.p.s.p. has a faster decay indicating the appearance of a Ib i.p.s.p. even without cutaneous facilitatory support. The presumed Ib effect is clearly enhanced by the preceding SP volley (*I*) again suggesting facilitation of Ib inhibitory transmission.

Since it is cumbersome to derive facilitation of Ib inhibition from the decay of the Ia e.p.s.p. we often used passage of depolarizing current through the recording electrode. With this procedure, decreasing the e.p.s.p. and increasing the i.p.s.p., there was no difficulty in demonstrating facilitation of Ib i.p.s.p.s from nerves giving homonymous and heteronymous Ia e.p.s.p.s.

Sometimes it turned out to be an advantage to use double group I volleys thus enhancing the test p.s.p. through temporal facilitation as shown in Fig. 3. *E–G* show only a very slight facilitation of the Ib i.p.s.p.

produced by a single maximal Pl group I volley. On the other hand, with a double submaximal test (*B*, *C*) the facilitatory effect by the same conditioning Sur volley is much more pronounced. The withdrawal of the second test volley in *D* shows that the i.p.s.p. in *C* is very largely evoked by the second group I volley; although the first group I volley facilitates transmission from cutaneous afferents so that the Sur volley now evokes a slight i.p.s.p. (cf. Fig. 1), this i.p.s.p. is clearly much smaller than the difference between the test i.p.s.p.s in *B* and *C*.

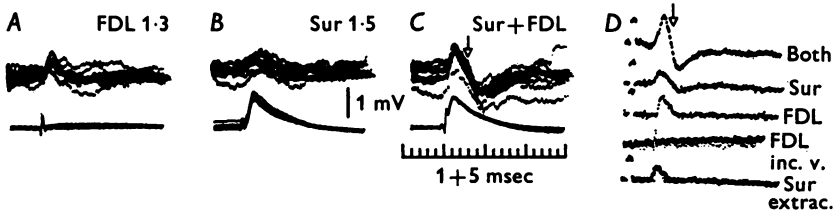

Fig. 4. Facilitation from cutaneous afferents (Sur) of a late presumably trisynaptic group Ib i.p.s.p. Intracellular recording from a Pl motoneurone. The facilitated i.p.s.p. in *C* and *D* (upper trace) is marked by an arrow. The initial deflexion in *B* and in the second trace in *D* is virtually identical with the extracellular field (see fifth trace in *D*). Fourth trace in *D* shows the incoming group I volley of FDL. Each averaged record consists of ten responses; calibration pulse 0·5 mV, 1 msec.

Eccles, Eccles & Lundberg (1957*b*) reported that the central latencies of Ib i.p.s.p.s distribute from 1·3 to 3·5 msec and postulated that transmission may be via di- or trisynaptic pathways. We have confirmed that Ib i.p.s.p.s with long latencies are common and have found facilitation by volleys in cutaneous afferents also of late, presumed trisynaptic, Ib i.p.s.p.s. In Fig. 4 there is considerable spatial facilitation between Sur afferents and group I afferents from FDL in inhibitory transmission to a Pl motoneurone. The facilitated i.p.s.p. in *C* (arrow) has a central latency of 2·8 msec which suggests transmission through a trisynaptic pathway (cf. Eccles *et al.* 1957*b*). From the averaged records in *D* it cannot be excluded that there is also some facilitation of more short-latency i.p.s.p.s but the facilitation of the late i.p.s.p. is certainly the most evident effect. Facilitation of di- and trisynaptic i.p.s.p.s to the same motoneurones was not uncommon. Altogether, we found a complete parallelism in effect to the di- and trisynaptic pathways. Both were facilitated from the cutaneous nerves tested and independently of whether or not the test i.p.s.p. was evoked from a nerve which gave a Ia e.p.s.p. in the motoneurones recorded from.

Even in nerves without separation of the group I volley in fast and slow

components Ib i.p.s.p.s appear at higher strengths than the Ia p.s.p.s (Eccles *et al.* 1957*a*), usually about 1·3 times threshold. It is noteworthy that during cutaneous facilitation presumed Ib i.p.s.p.s frequently were evoked at lower strengths, sometimes at 1·1–1·15 times threshold. The simplest explanation of this finding is that some Ib fibres have low threshold (cf. Fig. 7.11, Matthews, 1972) but normally fail to evoke i.p.s.p.s because activation of the interneurones requires summation. However, it is necessary to keep in mind the alternative possibility that the Ib inhibitory

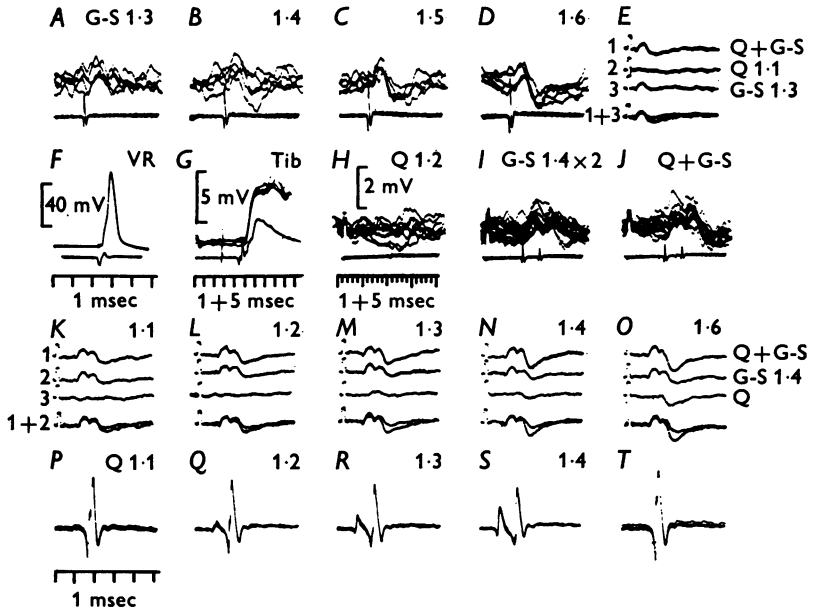

Fig. 5. Facilitation from low threshold Q afferents of Ib i.p.s.p.s evoked from G-S. Intracellular recording from a Tib motoneurone (*F* and *G*). Graded single volleys in G-S evoked a monosynaptic e.p.s.p. and a characteristic Ib i.p.s.p. (*A–D*). The averaged records in *D* show facilitatory effect of a synchronous weak Q volley; the amount of facilitation appears from a comparison of trace 2, Q volley alone, with the difference between the superimposed traces 1 + 3. *H–O* were obtained with a double volley test from G-S evoked at 1·4 times threshold. Averaged records in *K–O* were obtained with graded stimulation of Q at strengths indicated above each record. For comparison the double volley test at the same strength is shown in *P–T*; the second testing stimulus (alone in *T*) was supramaximal and kept constant throughout the series. Calibration pulse in averaged records 1 mV and 1 msec.

pathway may get facilitatory support from Ia afferents. In experiments of the present type the possibility of a convergence from Ia and Ib afferents can be experimentally approached only with respect to actions from nerves

in which the group I volley displays separation in fast and slow components (Bradley & Eccles, 1953), and even in this case uncertainty remains to which extent the two components represent activity in Ia and Ib fibres respectively; two investigations with recording from primary afferents have given somewhat different results (Laporte & Bessou, 1957; Coppin, Jack & McIntyre, 1969). The records in Fig. 5 are from a Tib motoneurone (innervating the intrinsic plantar muscles; identified in *F* and *G*). The G-S nerve evoked a typical Ib i.p.s.p. (*A–D*) appearing at a strength of 1·3–1·4 times threshold. The averaged records show the effect of combined stimulation of G-S at 1·3 and Q at 1·1 times threshold. Observe that the Q volley alone had no effect (second trace) but that the combined stimulation (first trace) gives spatial facilitation. The double volley test (P-T) revealed perfect separation of the Q group I volley in fast and slow components. Records *H–O* show the facilitatory action with graded stimulation of the Q nerve using double stimulation of G-S at 1·4 times threshold as test. The original records (*H–J*) also give the timing with the Q volley arriving 1 msec before the second G-S volley. The averaged records *K–O* show that facilitation (cf. legend) increased gradually with increasing Q stimulus strength: 1·1 (*K*), 1·2 (*L*) and 1·3 (*M*) times threshold. In record *N* the Q volley alone evokes an i.p.s.p. although the strength is subthreshold for the slow group I component (S), and in record *O* the i.p.s.p. increases when the stimulus strength is raised to 1·6 activating also fibres in the slow group I component. Observe also the declining spatial facilitation in *N* and *O*, presumably due to occlusion. Already Eccles *et al.* (1957*a*) noted that a volley in the fast group I component from Q might evoke small i.p.s.p.s in G-S motoneurones and suggested that this was due to Ib fibres in the fast component. We are now more hesitant about this interpretation. The Ia/Ib threshold separation in the Q nerve has not been investigated with primary afferent recording but Lindström and his collaborators investigated the effect of graded stimulation of the Q nerve in many VSCT cells. When the Q group I volley displayed separation in fast and slow components it was a characteristic finding in Ib VSCT cells that the monosynaptic e.p.s.p. was evoked only when fibres in the slow component were stimulated (cf. Fig. 4 in Lindström & Schomburg, 1974; S. Lindström, personal communication). Accordingly, it may now appear less likely that a facilitation contributed throughout the low threshold range as in Fig. 5 is due to stimulation of Ib fibres contaminating the fast component.

Ib e.p.s.p.s. We have confirmed that group Ib volleys from extensors predominantly evoke e.p.s.p.s in flexor motoneurones and that effects from flexors are rare (Eccles *et al.* 1957*b*). The Ib e.p.s.p.s have di- or trisynaptic central latencies ranging from 1·3 to 3·0 msec. Both kinds are facilitated by conditioning stimulation in low threshold cutaneous

afferents. Double volleys were often used to evoke the disynaptic test e.p.s.p. as is shown in Fig. 6*A–D* for a DP motoneurone. The single volley was without effect but a double volley evoked small e.p.s.p.s shown in *B* which were increased by preceding stimulation of the sural nerve. Observe that the distinct e.p.s.p. disappeared when the second test volley was withdrawn in *D* indicating that it was not caused by a group I facilitation of transmission from cutaneous afferents. *E–G* are from a PBSt motoneurone with facilitation of the e.p.s.p. evoked by a single volley. Observe

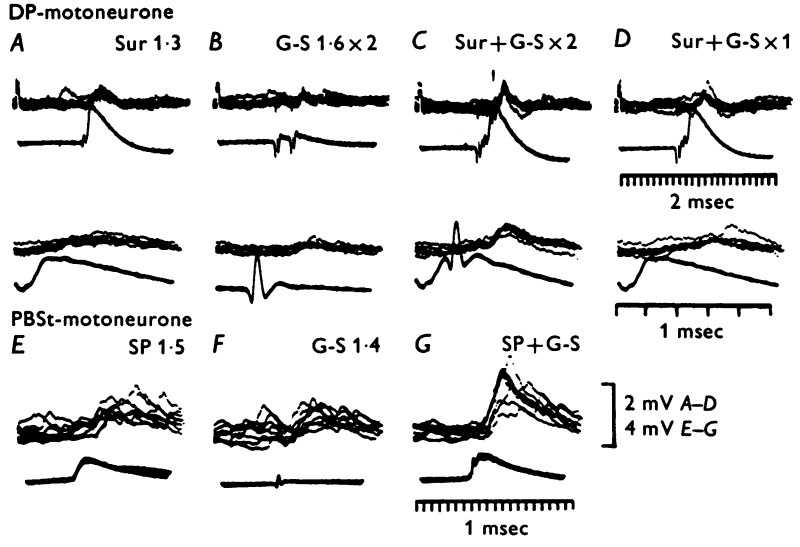

Fig. 6. Facilitation from cutaneous afferents (Sur and SP) of Ib e.p.s.p.s in flexor motoneurones. Intracellular recording from a DP (*A–D*) and a PBSt (*E–G*) motoneurone. The lower records in *A–D* are taken with expanded sweep. In *B* and *C* G-S was stimulated with a double volley. After withdrawal of the second stimulus (*D*) the facilitated distinct e.p.s.p. seen in *C* disappears. In the PBSt motoneurone already single G-S stimulation (*F*) revealed a small e.p.s.p., which was clearly facilitated by stimulation of SP (*G*).

the short conditioning-testing interval employed in both tests illustrated in Fig. 6. For the excitatory pathway we have not obtained a time course as complete as that shown in Fig. 1. However, we observed maximal facilitation at an interval of 1–2 msec which suggests that the linkage is similar. Facilitation of trisynaptic Ib e.p.s.p.s has been illustrated previously (Lundberg, Malmgren & Schomburg, 1975).

As for the inhibition there was no difference between effects on e.p.s.p.s evoked from antagonist extensors and from other extensors. Fig. 7 from a PBSt motoneurone shows p.s.p.s evoked by double volley stimulation of

quadriceps at two strengths. Observe temporal facilitation in *D* with increment in the i.p.s.p. evoked by the second volley and that a conditioning volley in the sural nerve considerably reduces this increment in the i.p.s.p. (*F*). This reduction is taken to indicate the appearance of a Ib e.p.s.p. An alternative explanation might have been an inhibitory effect by cutaneous afferents on the Ia inhibitory interneurones which are known to receive a complex convergence from primary afferents. However, the results obtained with a weaker test stimulus illustrated in *A–C* are against this explanation since there is now hardly any reduction of the second i.p.s.p. by the same cutaneous conditioning volley.

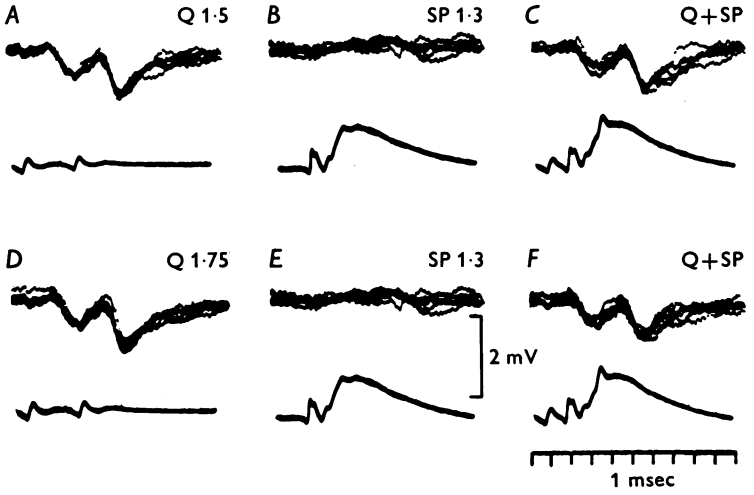

Fig. 7. Facilitation from cutaneous afferents of Ib e.p.s.p.s evoked in a flexor motoneurone (PBSt) from the antagonist extensor (Q). Intracellular recording from a PBSt motoneurone. Q is stimulated with a double volley. The conditioning volley in the sural nerve reduced the increment of the i.p.s.p. induced by the second volley from Q, when the Q stimulus strength was increased above group Ia strength (*D*, *F*).

Frequency of effects. Careful tests were made on 152 Ib p.s.p.s in eighty-three motoneurones, fifty-eight extensors and twenty-five flexors. Of the 119 Ib i.p.s.p.s, sixty-two were facilitated from low threshold cutaneous afferents. Correspondingly, of thirty-three tested Ib e.p.s.p.s, eighteen were facilitated. Virtually all the i.p.s.p.s were recorded from extensors (Q, Add fem, ABSm, G-S, Pl, FDL, Tib) and the e.p.s.p.s from flexors (PBSt, DP). There was no obvious difference in effect from the three cutaneous nerves used for conditioning (Sur, SP and Saph). With respect to the

negative tests it is worth noting that in many motoneurones large p.s.p.s were evoked from very low threshold cutaneous afferents. Accordingly, it was often difficult to investigate the effect by volleys in cutaneous nerves on Ib p.s.p.s.

Confirming previous observations (Eccles *et al.* 1957*b*) we found a considerable variation in Ib effects between different animals. In some cats we found both short-latency (1·3–2·1 msec) presumed disynaptic and long-latency (> 2·1 msec) presumed trisynaptic p.s.p.s while in others there was strong dominance of either of these. In a few cats hardly any Ib p.s.p.s were found despite the use of trains of stimuli.

DISCUSSION

For all reflex pathways but the one mediating reciprocal Ia inhibition (Jankowska & Roberts, 1972*a*, *b*; Hultborn, 1976) we are obliged to use indirect methods to investigate convergence on the interneurones. We have now used the spatial facilitation technique (cf. Lundberg, 1975) to show that volleys in cutaneous nerves facilitate interneuronal transmission in inhibitory and excitatory reflex pathways from Ib afferents to motoneurones. Similar facilitatory effects were found on autogenetic Ib i.p.s.p.s and those evoked from less closely related muscles operating at other joints. This parallelism was to be expected since there is Ib convergence on common interneurones (Lundberg *et al.* 1975). There was no difference with regard to di- and trisynaptic Ib reflex pathways. The present results refer entirely to the simple Ib pattern which is dominating in the low spinal cat, i.e. inhibition of extensors and excitation of flexors evoked from extensors (Eccles *et al.* 1957*b*). It remains to investigate whether more complex Ib patterns found in high spinal animals (Laporte & Lloyd, 1952) and during supraspinal facilitation (Hongo *et al.* 1969) are similarly controlled from cutaneous afferents.

Since corresponding facilitation is obtained with stimulation of the posterior joint nerve (Lundberg *et al.* 1975) the possibility should be considered that the effect may be caused by stimulation of joint afferents 'contaminating' the cutaneous nerves. Skoglund (1956) showed that some knee joint afferents join the saphenous nerve, and it is conceivable that the skin nerves from the distal part of the limb might receive afferents from the numerous joints in the foot. However, the sural nerve has been analysed in this respect by Hunt & McIntyre (1960) who found only two joint afferents among 421 fibres. Since pronounced effects on the Ib pathways were obtained also from the sural nerve it seems safe to conclude that receptors in the skin are responsible. Furthermore, as the facilitation was obtained at low strength of stimulation it is likely that these fibres have receptors activated by light mechanical stimuli (Hunt & McIntyre, 1960).

In the case of the simplest disynaptic Ib inhibitory pathway our time course measurements and other observations suggest that cutaneous afferents have disynaptic excitatory connexions with the Ib inhibitory interneurones as shown in the wiring diagram in Fig. 1. Other studies with the indirect technique (cf. Introduction) have shown that interneurones of Ib reflex pathways are controlled from different descending tracts; for example rubrospinal volleys evoke monosynaptic e.p.s.p.s in Ib inhibitory interneurones. Hongo, Jankowska & Lundberg (1972) did not record monosynaptic rubrospinal e.p.s.p.s in any of the interneurones in the intermediate region which receive monosynaptic excitation both from group I muscle afferents and from low threshold cutaneous afferents (cf. also Hongo, Jankowska & Lundberg 1966), while they were common in group I cells without monosynaptic cutaneous excitation. Some interneurones of the latter group did in fact receive disynaptic excitation from cutaneous afferents (unpublished observations by Hongo *et al.* 1972) and they may therefore qualify as candidates for interneurones intercalated in the disynaptic Ib inhibitory pathway to motoneurones. It would certainly be of interest to find out if interneurones belonging to this particular group receive convergence also from other descending systems which from studies with the indirect technique are known to project to the Ib inhibitory interneurones. It seems desirable to let such studies precede attempts at finding the projection of these interneurones with the technique so successfully used by Jankowska & Roberts (1972*a*, *b*) for identification of the Ia inhibitory interneurones.

Hongo *et al.* (1966) also reported that many of the group I excited interneurones in the intermediate region were excited (or inhibited) from the FRA, i.e. not only from cutaneous afferents but also from high threshold muscle and joint afferents. Since preliminary experiments with the indirect technique have failed to show clear facilitation of Ib i.p.s.p.s from high threshold muscle afferents (unpublished observations) and since the facilitation is evoked from very low threshold cutaneous afferents it is tempting to assume that private interneuronal pathways from cutaneous afferents contribute. It is important to bear in mind also other convergent actions found in interneurones. For example, Hongo *et al.* (1966, 1972) showed that convergence of monosynaptic e.p.s.p.s and disynaptic i.p.s.p.s from group I muscle afferents is very common in interneurones in the intermediate region, but so far the indirect technique has not revealed any corresponding finding for Ib pathways to motoneurones (unpublished observations). Many interneurones in the same region also seem to have convergence of Ia and Ib e.p.s.p.s (Hongo *et al.* 1966, 1972). Their function is unknown but it cannot be excluded that they are part of reflex pathways to motoneurones. Recent experiments with injection of

horseradish peroxidase have in fact revealed that some of them have axons projecting to the region of the motor nuclei (Czarkowska, Jankowska & Sybirska, 1976). Our present results rather suggest that interneurones of some Ib inhibitory pathways may be monosynaptically facilitated from Ia afferents. It should be emphasized that views on reflexes from tendon organs to a considerable extent are based on the *assumption* that all group I i.p.s.p.s which are not mediated by the reciprocal Ia inhibitory pathway are evoked from Ib afferents (Eccles *et al.* 1957*a*). Findings like those illustrated in Fig. 5 should incite critical tests of this assumption. It also deserves to be mentioned that recent experiments have revealed convergence of monosynaptic e.p.s.p.s from Ib afferents and group II muscle afferents in some more ventrally located interneurones (A. Lundberg, K. Malmgren & E. D. Schomburg, unpublished observations). The possibility should be kept in mind that the reflex pathways from Ib afferents may not be 'homogeneous' but that pathways might exist with different interneuronal convergence patterns not only for Ib connexions from different muscles (cf. Hongo *et al.* 1969) but also from other primary afferents. Traditionally one type of proprioceptive pathway has been believed to regulate a variety of motor acts, but we can by no means exclude the possibility of more differentiated control systems through reflex pathways in which afferents from diverse receptors act together in different combinations.

The present findings are but one example of convergence of afferents from different receptors on common interneurones in reflex pathways to motoneurones. For some time a very extensive convergence has been assumed to occur in the reflex pathway from the FRA comprising not only cutaneous and high threshold joint afferents but also high threshold muscle afferents from both flexors and extensors (cf. R. M. Eccles & Lundberg, 1959*b*). Results obtained with the indirect spatial facilitation technique have confirmed the existence of the FRA convergence (T.-C. Fu, E. Jankowska & A. Lundberg, unpublished results; cf. Lundberg, 1972). Recently, Hultborn and his collaborators have shown that the Ia inhibitory interneurones receive extensive convergence from different afferent systems actually corresponding to that found in agonist motoneurones (for reference see Hultborn, 1976). These findings have obvious implications when trying to understand how reflex actions are evoked in motoneurones from a given afferent system. It is important to realize that the same afferent volley may have several routes at its disposal; for example volleys in cutaneous afferents can probably evoke i.p.s.p.s in motoneurones via at least four different segmental reflex paths: (1) the Ia inhibitory pathway, (2) the Ib inhibitory pathway, (3) inhibitory pathways from the FRA, (4) private inhibitory pathways from cutaneous afferents. 'Private'

in this connexion implies that the interneurones do not receive excitation from other afferents – inhibitory effects are not excluded.

Conversely it is essential to obtain information about interneuronal convergence for the understanding of how a given reflex pathway functions. This problem has been extensively discussed for the Ia inhibitory pathway (Hultborn, 1972, 1976) and we shall now briefly consider the significance of the present results for the understanding of how reflexes from Ib afferents contribute to motor regulation. The convergence from descending pathways (cf. Introduction) has been taken to indicate that the brain can set the sensitivity of Ib tension regulation as part of a command for muscular force. To this descending control can now be added a segmental mechanism which may provide rapid adjustment of Ib tension regulation during the course of a movement. Activation of cutaneous receptors during an active movement may facilitate transmission in Ib inhibitory pathways and bring about a decreased muscular tension. It may be of some interest to consider some possibilities in relation to the models of Ib feed-back regulation presented by Houk *et al.* (1970) and Nichols & Houk (1976).

Houk *et al.* (1970) simplified their discussion of force feed-back with the suggestion that during certain motor tasks, e.g. exploratory movements, spindle feed-back may be inhibited and Ib feed-back facilitated, providing a system controlling tension rather than length. Let us assume that the facilitation of the Ib inhibitory pathway regulating such a movement is drawn from a skin field activated when the moving limb meets an obstacle. Then the result would be a decreased muscular tension not to force the obstacle. Post-synaptic inhibition of α-motoneurones directly from the skin might serve the same purpose, but a control of Ib inhibitory transmission increasing the gain in the Ib force loop provides an elegant solution since otherwise this feed-back mechanism would tend to maintain constant tension. This hypothesis has bearing also on reciprocal Ib excitation. All investigators seem to agree that inhibition is the primary Ib reflex effect but little attention has been given to the function of the reciprocal excitatory action. The parallel control of Ib inhibition and excitation not only from higher centres but also from the skin suggests that Ib excitation subserves Ib inhibition. In the above example of an exploratory movement meeting an obstacle, excitation of antagonist muscles would indeed supplement Ib inhibition in giving a purposeful brake on the movement.

Application of the servoconcept to motor control implies regulation keeping one function constant. It has thus been necessary to consider separately models for length servo and tension servo (Houk *et al.* 1970) although it seems more realistic to assume that movements are regulated both from muscle spindles and tendon organs. Nichols & Houk (1976) recently postulated that the regulated property is muscle stiffness and

that the main function of autogenetic reflexes may be to compensate for highly non-linear properties of skeletal muscles. While there is evidence that reflex actions may linearize components of the stretch reflex in the decerebrate cat it seems to us extremely unlikely that Ia and Ib reflexes in normal movements exclusively compensate for mechanical non-linearity – in this connexion it is important to keep in mind that only a minor part of the Ib inhibitory action is autogenetic. Perhaps the servo-focusing on one function at a time too much restricts our imagination. It is quite possible that different regulating functions are used in separate phases of a movement and that various control mechanisms, including segmental ones, govern the balance between them. These control mechanisms can be described as changing the gain of the servo, but how useful is the servo concept to describe regulation of movement if we have to add a continuous change in gain? Perhaps it is more convenient to view Ia and Ib actions in a more old-fashioned way merely as reflexes (cf. the load compensating reflex by C. von Euler, 1966) which regulate movements together, because then the apparent conflict between opposing length and tension servos does indeed vanish. For example, it has been suggested that during the stance phase of stepping there is a shift from initial dominance of Ib inhibition to a decreased Ib inhibition and increased stretch reflex activity later on during the stance phase (Bergmans, Burke, Fedina & Lundberg, 1974). Initial dominance of Ib inhibition may assure a long-lasting yield by counteracting the Ia stretch reflex mechanism which otherwise would come into operation because γ-operated spindles are stretched during the yield (cf. Lundberg, 1969). Later on during stance a decreasing Ib inhibition might allow the Ia stretch reflex to dominate and give load compensation when required for progression. Bergmans *et al.* (1974) discussed the possibility that presynaptic inhibition may bring about such a shift, but a control of Ib interneuronal transmission if evoked from the plantar surface of the foot would provide an interesting alternative. Similar considerations on the balance between the stretch reflex and autogenetic inhibition were presented by Newsom Davis & Sears (1970) who found that a sudden increase in load on human intercostal muscles gave short-latency inhibition followed by late excitation.

Clearly it is now required to find the skin region giving facilitation of Ib reflex pathways. For these experiments it may be an advantage to use the forelimb since recent findings suggest a more pronounced effect from cutaneous afferents on the Ib inhibitory pathway in forelimb segments (Illert *et al.* 1976).

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