

POST-SYNAPTIC EXCITATION AND INHIBITION FROM PRIMARY AFFERENTS IN NEURONES OF THE SPINOCERVICAL TRACT

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SUMMARY

1. Intra- and extracellular recordings were made from cells of the spinocervical tract in the lumbosacral spinal cord. A convergence of mono-synaptic excitatory post-synaptic potentials (EPSPs) and disynaptic inhibitory post-synaptic potentials (IPSPs) was a general pattern of effects from the low threshold cutaneous fibres. Unitary IPSPs, probably mediated via the same disynaptic path, were evoked by light touch of hairs, which was also the adequate stimulus for exciting the cells. The receptive field for unitary IPSPs was closely related to the excitatory receptive field but was eccentric, not of a surround type.

2. EPSPs, IPSPs, or both, were evoked from the flexor reflex afferents in the great majority of neurones. Disynaptic IPSPs may be evoked from the interosseous nerve. No effects were produced by volleys in group I muscle afferents.

3. It is suggested, on the basis of the spatial organization of the excitatory and inhibitory receptive skin fields, that the spinocervical tract may give information regarding the direction of tactile stimuli.

INTRODUCTION

Tactile information to the cerebral cortex is mediated in the spinal cord not only by the dorsal column but also, as was originally shown by Morin (1955), by a pathway in the dorsal part of the lateral funicle. The latter pathway has subsequently been identified as the spinocervical tract (Norrzell & Voorhoeve, 1962; Andersson, 1962). This tract originates in

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the dorsal horn close to the entry zone of the primary afferents giving monosynaptic excitation, ascends ipsilaterally in the dorsomedial part of the lateral funicle and terminates in the upper cervical segments (Eccles, Eccles & Lundberg, 1960; Lundberg & Oscarsson, 1961; Lundberg, 1964*a*; Taub & Bishop, 1965).

Afferent inhibition of a surround type is a characteristic feature in the classical pathway from the dorsal column (Gordon & Paine, 1960; Andersson, 1962; cf. also Mountcastle & Powell, 1959) but has not been observed in Morin's pathway either in the spinocervical tract (SCT) (Wall, 1960; Lundberg & Oscarsson, 1961) or in the subsequent relays from it to the cerebral cortex (Gordon & Jukes, 1963; Horrobin, 1966; Andersson, 1962). These findings suggested that the dorsal column pathway may be important in spatial discrimination and that the SCT relays a less integrated message with a high degree of synaptic security (Gordon & Paine, 1960; Andersson, 1962). The importance of both these pathways for tactile discrimination has been demonstrated in behavioural experiments (Norrzell, 1966).

The above findings have been based mainly on extracellular or axonal recording. In the present investigation intracellular recording from spinocervical tract cells has revealed that post-synaptic inhibition is evoked in these cells from various kinds of primary afferents. In this connexion it is recalled that Taub (1964) found that skin stimulation gives a labile inhibition in the SCT which he suggested was caused by presynaptic inhibition. A preliminary report of some of the present results has been published (Hongo, Jankowska & Lundberg, 1966*a*).

METHODS

The main series of experiments was performed on unanaesthetized spinal cats, decerebrated or anaemically decorticated as described by Andén, Jukes, Lundberg & Vyklický (1966). Some experiments were made on spinal cats anaesthetized with chloralose (60 mg/kg). A few neurones encountered in non-spinalized cats under chloralose (60 mg/kg) or pentobarbitone sodium (30 mg/kg) anaesthesia have also been included. No systematic differences were recognized among the different preparations. The dissection and maintenance of the preparation, arrangements of the experimental set-up, recording techniques including micro-electrodes, histological controls, etc., were essentially the same as described by Hongo, Jankowska & Lundberg (1966*b*). The cells supposed to give origin to the spinocervical tract (Eccles *et al.* 1960; Wall, 1960, 1965) were identified by the following three criteria: (1) antidromic invasion on stimulation in the lower thoracic region of the ipsilateral spinal half (except the dorsal column) (e.g. Fig. 1 A) or the dissected dorsal part of the ipsilateral lateral funicle, (2) monosynaptic excitation by large cutaneous afferents (e.g. Fig. 1 B-E), and (3) the location of the cell body in the dorsal horn or the intermediary region, 1.1-1.8 mm from the dorsal surface (cf. Fig. 13). With these criteria there is so far no evidence to suggest that other than SCT neurones are included (cf. Lundberg, 1964*a*; Taub & Bishop, 1965). In order to allow natural stimulation of the skin the superficial

peroneal and the tibial nerves were dissected for stimulation with intact peripheral connections (5 expts.).

The SCT cells are scattered in the dorsal horn and in order to impale them we developed the following procedure: after picking up an extracellular unit spike, systematic tracking (at 50 μ intervals) was made in a direction that depended on the change in size of the spike. When this technique was employed we almost invariably succeeded in impaling the cell once the unit spike had been encountered.

Abbreviations for nerves dissected and other terms employed are as follows: quadriceps (Q), posterior biceps-semitendinosus (PBSt), anterior biceps-semimembranosus (ABSm), gastrocnemius-soleus (G-S), plantaris (Pl), flexor digitorum and hallucis longus (FDL), deep peroneal (DP), sural (Sur), superficial peroneal (SP), tibial (Tib), posterior knee joint nerve (J), interosseous nerve (Io), contralateral hamstring (coH), contralateral sural (coSur) ipsilateral thoracic cord (icord), spinocervical tract (SCT), post-synaptic potential (PSP), excitatory post-synaptic potential (EPSP), inhibitory post-synaptic potential (IPSP), flexor reflex afferents (FRA).

RESULTS

Intracellular recording from the SCT neurones was comparatively more stable than from spinal interneurons (cf. Eccles *et al.* 1960; Hongo *et al.* 1966*b*) probably because of the large size of the neurone soma (Szentágothai, 1964; Wall, 1965). However, since our main purpose was to investigate the synaptic organization we often waited until the spike mechanism of the cell had deteriorated. Since at this stage the resting membrane potential was low, 30–50 mV, EPSPs should have been smaller and IPSPs larger than at the normal level of the resting membrane potential. When KCl electrodes were used for recording, IPSPs often reversed because of leakage of chloride ions.

The results presented are based mainly on intracellular records from twenty-five neurones in which recording was stable and effects from all or almost all dissected nerves were properly tested. Substantially the same results were obtained from fifteen supplementary neurones in which the analysis for some reason was incomplete.

Effects from cutaneous afferents

(a) *Monosynaptic EPSPs.* Volleys in low threshold cutaneous afferents evoked an EPSP which could be graded with stimuli of increasing strength (Fig. 1 *B–E*). The central latency of 0.5–0.9 msec, measured from the arrival of the afferent volley to the dorsal root entry zone, shows that the EPSP is produced by monosynaptic action of the large cutaneous afferents. This monosynaptic EPSP from one or two neighbouring cutaneous nerves was used as one of the criteria in identifying the SCT neurone (cf. Methods). Polysynaptic EPSPs from high threshold muscle afferents are shown in Fig. 1 *H–J* for comparison. Spike potentials were elicited by the EPSP provided the resting membrane potential was high enough. Wall's (1965) claim that spikes can be evoked in these neurones without any signs of prepotentials or EPSPs was not confirmed in any cell in the present study.

(b) *Disynaptic IPSPs*. Figure 1 *B-E* shows a faster decay of the mono-synaptic EPSP from its peak depolarization than is usually found in mammalian nerve cells (Eccles, 1957) and at the higher stimulus strengths in Fig. 1 *D, E* the EPSP is followed by a hyperpolarization. These features are well explained by the assumption that the decaying phase of the EPSP is curtailed by an IPSP. This interpretation is supported by findings such as those illustrated in Fig. 2. A KCl electrode was used for recording. Soon

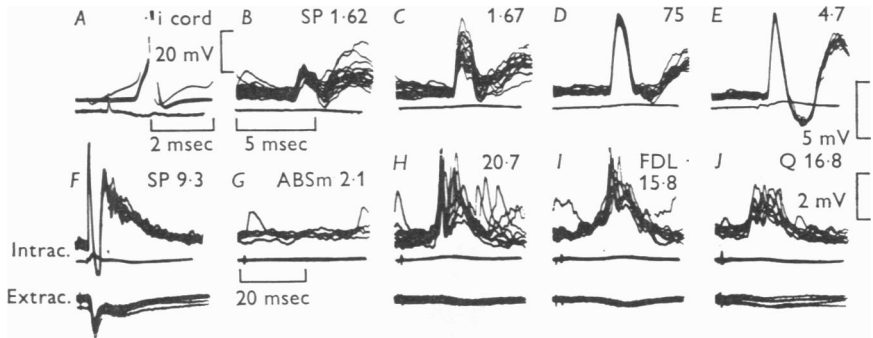


Fig. 1. Intracellular records from a SCT neurone showing EPSPs and IPSPs evoked from cutaneous and muscle afferents. The uppermost traces show intracellular potentials (citrate electrode) from a cell located 1.3 mm from the cord dorsum. The lower traces in *A-E* and *G* and middle traces in *F* and *H-J* are recorded with a silver ball electrode at the L7 dorsal root entry zone. The lowest traces in *F* and *H-J* show potentials recorded with the micro-electrode from a position just outside the cell. In this and subsequent figures upward deflexion indicates positivity in the micro-electrode records and negativity in the cord-surface recording. Stimulus strengths are indicated in multiples of the threshold strength for each nerve in all the figures. *A*: antidromic spike potential evoked from the ipsilateral thoracic cord. *B-F*: EPSPs and IPSPs from SP at five steps of stimulus strength. Note different sweep speed in *B-E* and *F*. *G-J*: response to muscle afferent stimulation. No PSPs are evoked by group I volleys in ABSm (*G*), but polysynaptic EPSPs appear from higher threshold afferents in ABSm (*H*), FDL (*I*) and Q (*J*) nerve. The voltage calibrations in *A, E* and *J* refer to the micro-electrode recording in *A, B-F* and *G-J* respectively. The time calibration in *B* is for the upper row (except *A*) and in *G* for the lower. All the records in this and subsequent figures consist of superimposed traces unless stated otherwise.

after impalement of the cell a diphasic PSP was evoked from cutaneous afferents (Fig. 2 *B*) similar to that in Fig. 1 *E*. As time elapsed, the hyperpolarization became smaller and reversed to a depolarizing potential (*F*) probably due to diffusion of chloride ions inside the cell. The potential was enhanced in *H* by injection of chloride ions, by the passage of a hyperpolarizing current through the recording electrode, but was again reversed during passage of a depolarizing current (*D*). The chloride dependency and the level of the equilibrium potential close to the resting membrane

potential shows that the hyperpolarizing response is an IPSP. The onset of the IPSP, indicated by the thickest arrow in the inset drawing in *A*, occurs 1.2 msec after the earliest incoming volley (first arrow) or 0.4 msec after the start of the initial EPSP (second arrow). IPSPs without pre-

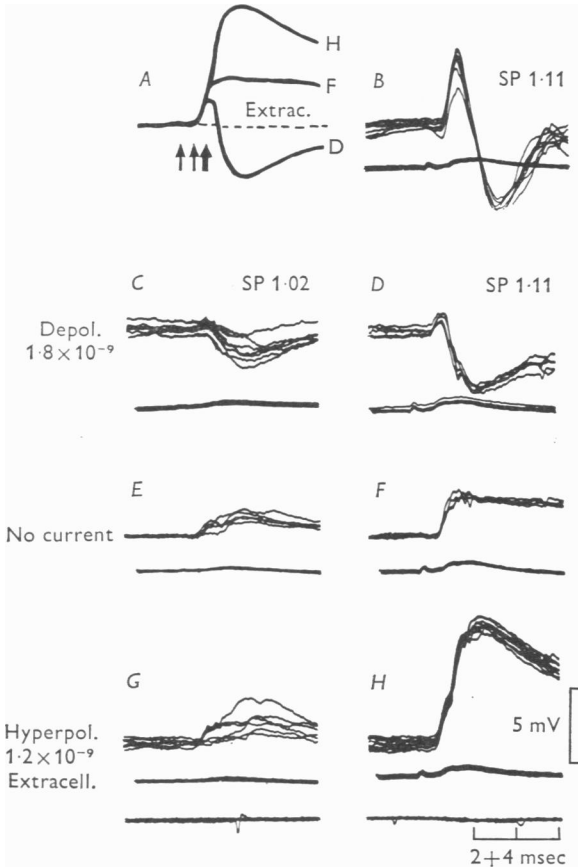


Fig. 2. Reversal of IPSPs from low threshold cutaneous afferents. Intracellular records (upper traces) with a KCl electrode from a cell located at a depth of 1.63 mm from the cord dorsum. The lowest traces in *G* and *H* are extracellular potentials recorded after withdrawal of the micro-electrode to a position just outside the cell. The lower traces in *B-F* and middle traces in *G* and *H* are from the L7 dorsal root entry zone. In the left row are shown PSPs evoked by a just threshold stimulation of SP nerve and in the right row those evoked by slightly increased stimuli. Record *B* was taken soon after penetration, the other records at a later stage during passage of current through the recording electrode. A depolarizing current (1.8×10^{-9} A) was passed in *B* and *C*, and a hyperpolarizing current (1.2×10^{-9} A) in *F* and *G*. The intracellular traces of *D*, *F* and *H* are superimposed in drawing *A*. The three arrows indicate: the arrival of the earliest incoming volley, the onset of the EPSP and of the IPSP (thick arrow). The dotted line gives the extracellular field potential.

ceding EPSPs were often observed when the stimulus strength was carefully graded near the threshold of the nerve (Fig. 2 *C*, *E* and *G*, Fig. 9 *C*, Fig. 11 *E* and Fig. 12 *B*) or when other cutaneous nerves were stimulated (Fig. 5 *B*, *F*, Fig. 9 *E*, *F* and Fig. 12 *G*). In these cases the onset of the IPSP was determined simply by comparing the intracellular and extracellular potentials. The central latencies of the IPSPs were 1.2–2.0 msec when measured from the earliest incoming volley detected in the cord dorsum. They were rather close to but, with the same nerve, consistently longer by at least 0.4 msec, than those of the monosynaptic EPSPs. Experiments with graded stimuli revealed no systematic difference between the thresholds for the EPSPs and IPSPs. They could both be evoked from the lowest threshold afferents and increased in parallel (Fig. 1 *B–E*) when the stimulus strength was raised. This shows that both the EPSPs and the IPSPs are evoked from cutaneous afferents in the same threshold range.

It is postulated that the synaptic linkage for the IPSP is disynaptic. It is true that the central latency of the IPSP in some SCT neurones is slightly shorter than is usually found for disynaptic IPSPs evoked from primary afferents. The explanation may be a relatively short intraspinal conduction distance in combination with a very strong synaptic linkage in the inhibitory pathway as suggested by the finding that even a minimal stimulus was sufficient to evoke an IPSP (Fig. 2 *C*, *E* and *G*). Hence our observations are not inconsistent with the hypothesis that primary afferents are excitatory (Eccles, 1957, cf. also Hongo *et al.* 1966*b*). The IPSPs are unlikely to be a consequence of recurrent activities since no effect was evoked by stimulation of the thoracic cord just subthreshold for the antidromic invasion of the impaled cell (cf. Fig. 6 *C*, Fig. 10 *B* and Fig. 11 *A*).

Furthermore, if it is assumed that the SCT neurones are excitatory, as in the lateral cervical nucleus (Gordon & Jukes, 1963; Horrobin, 1966; L. Fedina, G. Gordon & A. Lundberg, to be published) the shortest pathway for recurrent IPSPs evoked from the primary afferents should be trisynaptic. There is some evidence for recurrent inhibition via axon collaterals in the dorsal column nuclei (Gordon & Jukes, 1964). In the dorsal spinocerebellar tract, on the other hand, no such effect has been observed (Hongo & Okada, 1967).

The disynaptic IPSPs were found in all the twenty-five SCT neurones fully investigated. Therefore, a convergence of monosynaptic EPSPs from one source and disynaptic IPSPs from another (see later section 'Natural stimulation') may be regarded as a characteristic general pattern of synaptic linkage from the large cutaneous afferents to SCT neurones. It is particularly relevant that the disynaptic IPSP was produced independently

of whether the effect of volleys in high threshold muscle and joint afferents which are part of the FRA was excitatory (Figs. 1, 8), inhibitory (Figs. 9, 11) or lacking (Fig. 10). Moreover, the IPSPs from the FRA had longer latencies and a slower time course than the disynaptic IPSP (cf. below).

(c) *Inhibition of impulse discharge.* Several investigators have not observed inhibitory effects in Morin's pathway (Wall, 1960; Lundberg & Oscarsson, 1961; Gordon & Jukes, 1963; Andersson, 1962; however see Taub, 1964). It was therefore of interest to establish whether the IPSPs from cutaneous nerves are able to inhibit initiation of impulses in SCT neurones.

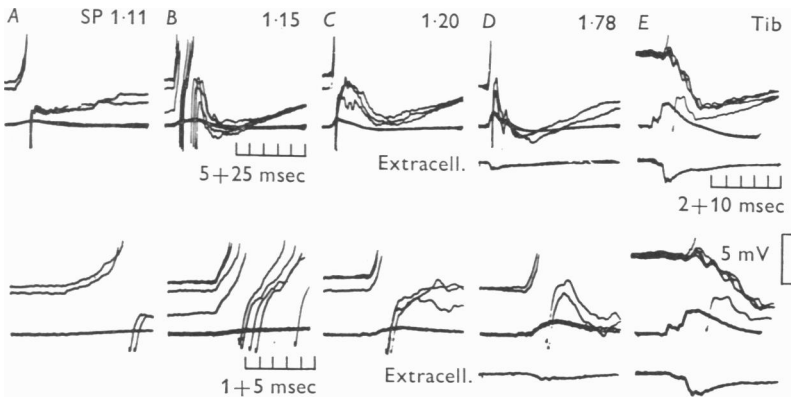


Fig. 3. Inhibition of discharge. Intracellular recording (upper traces) with a citrate electrode from a cell located at a depth of 1.1 mm from the cord dorsum. The lowermost traces in *D* and *E* (in both upper and lower set of records) are extracellular potentials recorded after withdrawal of the micro-electrode to a just extracellular position. Lower traces in *A-C* and middle traces in *D* and *E* are from the L7 dorsal root entry zone. The corresponding records in the upper and lower row were taken simultaneously at different sweep speeds. *A-D* show responses to graded stimulation of SP nerve. Observe that in *C* and *D* the second spike potential is inhibited by the hyperpolarizing potential. *E*, at threshold for the spike discharge, gives the after-hyperpolarization of the cell (see text). Voltage calibration in *E* applies to all micro-electrode records. Time calibrations for the upper (except in *E*) and for the lower row of records are given in *B* under the respective records. Observe that the upper records in *E* were taken at a slower speed.

The intracellular records of Fig. 3 *A-D* show the effect of stimulation of a cutaneous nerve at four strengths. In *A* the stimulus strength was just above threshold for evoking a single spike. The subsequent hyperpolarization is probably not an IPSP but almost entirely an after-hyperpolarization, its size and time course being approximately indicated in *E* by the difference between the superimposed traces and the single trace in which

a spike was evoked. A slight increase of the stimulus strength in *B* brought about rebuilding of the EPSP and initiation of a second spike. With further increase of the stimulus strength the rebuilding of the EPSP was suppressed and substituted by a hyperpolarizing potential (*C* and *D*, see especially lower records). The difference between the hyperpolarization in *A*, on one hand, and in *C* and *D* on the other must be due to the addition of an IPSP to the after-hyperpolarization in the two latter records. It is postulated that this IPSP prevented the initiation of the second spike. The large hyperpolarization in *B* may in part be caused by a summation of after-hyperpolarizations (cf. Ito & Oshima, 1962).

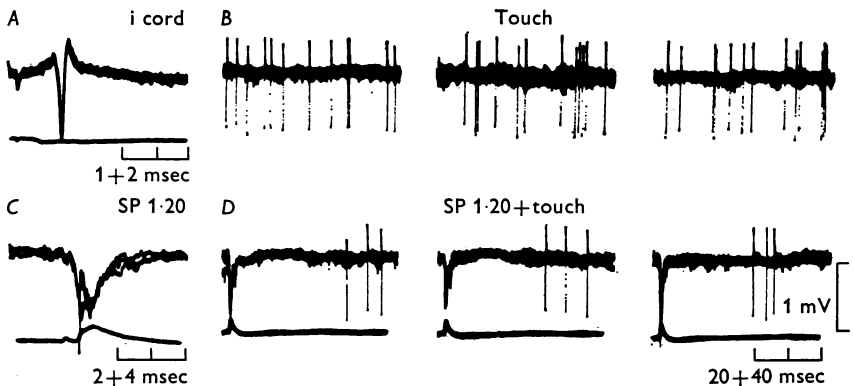


Fig. 4. Inhibition of naturally evoked discharges by a conditioning volley in low threshold cutaneous fibres. Extracellular records (*B* and upper traces in *A*, *C* and *D*) from a single unit at a depth of 1.5 mm from the cord dorsum. Lower traces in *A*, *C* and *D* show cord dorsum potentials near the L7 dorsal root entry zone. *A*: antidromic spike evoked by stimulation of the ipsilateral spinal half in thoracic region. *B*: spike activity during a continuous hair stimulation with a tuning fork (five superimposed traces). *C*: response to weak stimulation of the SP nerve; the neurone is occasionally fired by the stimulus. *D*: as in *B* but in addition a conditioning volley (the same stimulus as in *C*) was applied at the start of each sweep. Note that discharges are not evoked by hair stimulation during some 60 msec after the conditioning shock. The inhibition occurred independently of the presence or absence of the preceding discharges due to the conditioning shock. Voltage calibration applies to all the micro-electrode records.

Inhibition of naturally evoked discharges is shown in Fig. 4 with extracellularly recorded single unitary spikes. The three control records in *B* show the response to mechanical stimulation of hairs in the excitatory receptive field. A weak stimulus applied to a cutaneous nerve (SP) had a clear inhibitory effect (*D*). The conditioning volley did occasionally evoke firing of the cell as shown in *C* and by the right record in *D*. Refractoriness does not, however, explain the cessation of discharge, because the same effect occurred when the conditioning volley did not produce a spike (left

records in *D*). The inhibition seemed to start with a very short latency and it ended within 25–60 msec. It is possible that the inhibition of discharges is due to post-synaptic mechanisms although a participation of presynaptic inhibition cannot be excluded in this case (cf. Taub, 1964).

(*d*) *Natural stimulation*. For the analysis of the origin of the IPSPs natural stimulation of the skin was employed in five cats. The IPSPs

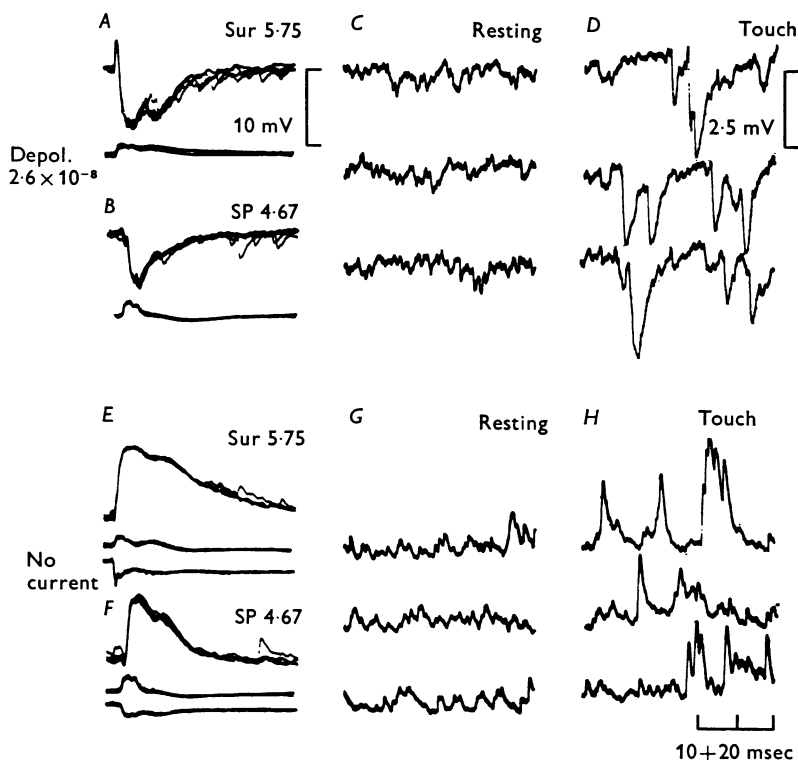


Fig. 5. IPSPs evoked by light touch of hairs. Intracellular records (*C*, *D*, *G*, *H* and upper traces in *A*, *B*, *E* and *F*) with a KCl electrode. The lowermost traces in *E* and *F* show extracellular potentials just outside the cell. Lower traces in *A* and *B* and middle traces in *E* and *F* are cord dorsum potentials. All records were taken after IPSPs had been reversed (due to chloride leakage): *A–D* were obtained during passage of a depolarizing current (2.6×10^{-8} A) and *E–H* in the absence of external current. IPSPs were in the hyperpolarizing direction (*A–D*) only when sufficient depolarizing currents were applied. *A* and *E* show monosynaptic EPSPs and disynaptic IPSPs evoked from the Sur nerve. There is an inflexion in the rising phase in *E* showing the onset of the reversed IPSP. *B* and *F* show disynaptic IPSPs from the SP nerve. *C* and *G* were obtained in the absence of hair stimulation and hence are control records for *D* and *H* where unitary IPSPs are evoked by light touch of hairs. Observe in *C* and *G* that the synaptic noise in the background has the same polarity as the IPSPs in *D* and *H*. Voltage calibration (for micro-electrode recording) is given in *A* for *A*, *B*, *E* and *F*, and in *D* for *C*, *D*, *G* and *H*. Time calibration below *H* is for all the records. *C*, *D*, *G* and *H* are single traces.

evoked by adequate stimulation were more easily recognizable during passage of a depolarizing current, under which condition the IPSPs were increased in amplitude, while the EPSPs were reduced and the spike mechanism inactivated. Records in Fig. 5 *A–D* were taken during passage of a depolarizing current through a KCl electrode, and show large unitary IPSPs induced by light touch of hairs (*D*) as well as synchronized IPSPs evoked by single shocks applied to cutaneous nerves (*A* and *B*). The EPSP from Sur was still in depolarizing direction as shown in *A*, but unitary EPSPs were hard to recognize under these conditions. Traces in *C* are control records obtained in the absence of hair stimulation. No large unitary IPSPs are present at rest though small IPSPs can be seen. It is not known whether these small IPSPs, like the miniature end-plate potentials, are due to spontaneous liberation of the transmitter substance (cf. Katz & Miledi, 1963), or appear as a consequence of impulses in inhibitory neurones. All these IPSPs reversed after withdrawal of the depolarizing current (*E–H*), an expected result of diffusion of chloride ions into the cell. The hyperpolarizing (*D*) and the depolarizing (*H*) unitary IPSPs were elicited from the same skin area, in both cases by touching hairs.

It was consistently found that touching of hairs evokes unitary IPSPs as in Fig. 5 *D, H*. The same type of natural stimulation is also effective in exciting SCT neurones. We did not measure exact threshold for these naturally produced effects, but no systematic differences were recognized between those for the post-synaptic excitation and inhibition. In some neurones slight touch of the pad cushion was effective in producing unitary IPSPs (Fig. 6 *K*). The latter were also elicited by pinching the skin and by pressing or squeezing deep tissue. It is probable that these unitary IPSPs are mediated by the polysynaptic inhibitory pathway from the FRA, which will be described below.

The receptive field from which the unitary IPSPs could be evoked by bending hairs was determined in several neurones. They appeared to be large in the proximal and small in the distal parts of the limb, as is also the case with the receptive fields for excitation (Lundberg & Oscarsson, 1961). In five cells the receptive fields were successfully mapped for both excitation and inhibition. This was accomplished by determining the IPSP field during intracellular recording and the excitatory field before penetration of or after withdrawal from the cell, using extracellular spikes as the indicator (Fig. 6). Care was taken to ensure that the extra- and intracellular records were obtained from the same cell. Criteria for this identity were not only the latency and the threshold of the response to antidromic (Fig. 6 *A, C*) and orthodromic (Fig. 6 *B, G*) stimulation but also the progressive growth of the extracellular spike before the cell was impaled.

Usually this identification offered no problem since the SCT cells are scattered in the dorsal horn. Most cells were impaled only after systematic tracking (cf. Methods) during the course of which responses from other SCT neurones were rarely detected at the depth of the cell aimed at. The excitatory receptive field of the cell in Fig. 6 was very small, being located on the medial surface of the second toe (see drawing). Unitary IPSPs (*K*), on the other hand, were evoked by touching hairs on the dorsal surface of the same toe (hatched region in *L*). The IPSPs were not fatigable and they were invariably produced from the same region (cf. Taub, 1964).

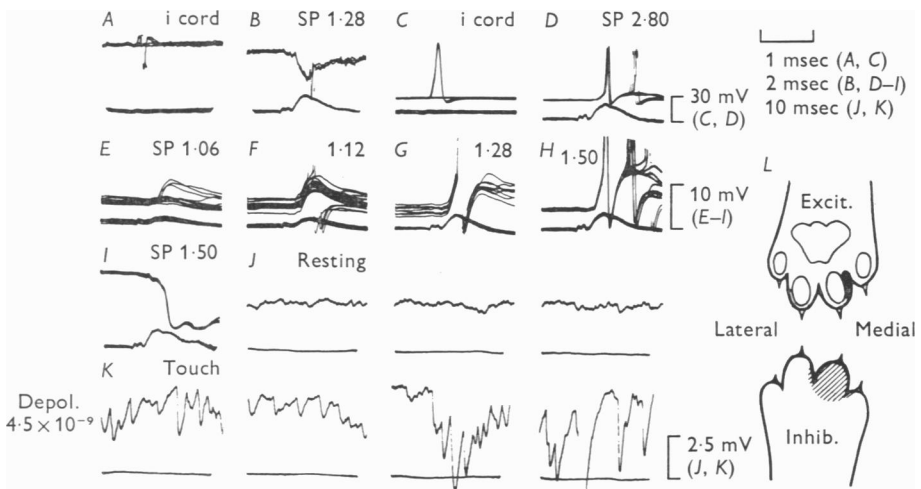


Fig. 6. Excitatory and inhibitory receptive fields. Extracellular (*A* and *B*, upper traces) and intracellular (*C*–*K*, upper traces) recording with a KCl electrode from a cell located 1.48 mm from the cord dorsum. Lower traces are from the L7 dorsal root entry zone. *A* and *C* show antidromic spike potentials evoked by stimulation of the ipsilateral spinal cord in the lower thoracic region. *B* and *D*–*I* show PSPs and spike potentials evoked from SP nerve at different stimulus strengths. *I*–*K* were obtained during passage of depolarizing current (intensity not measured). Since under these conditions the SP volley does not evoke an EPSP the membrane potential presumably was lowered to a level close to the equilibrium potential for the EPSP. The late depolarizing PSPs in *D* and *H* are probably reversed IPSPs. Records in *K* were obtained during light hair stimulation and should be compared with the control records in *J*, taken in the absence of hair touching. The region from which the unitary IPSPs are evoked from hairs is indicated by the hatched area in the schematic drawing (*L*). The black area shows the excitatory receptive field from which discharges (extracellularly recorded) could be evoked by hair stimulation. The excitatory field was mapped before impalement of the cell and also after withdrawal from the cell to a just extracellular position. Note the same latencies in the extra- and intracellular recording for both antidromic (*A* and *C*) and orthodromic (*B* and *G*) responses. Voltage calibration (for intracellular potentials) in *D* (30 mV) is for *C* and *D*, in *H* (10 mV) for *E*–*I* and in *K* (2.5 mV) for *J* and *K*.

Results from the other four neurones are given in Fig. 7. In all five cells the IPSP field is eccentric to the excitatory field and not of the surround type found for the afferent inhibition in the relays of the dorsal column lemniscal system (Gordon & Paine, 1960; Andersson, 1962).

Effects from the FRA

Polysynaptic effects common to cutaneous, high threshold joint and high threshold muscle afferents have been described as evoked from the FRA (R. M. Eccles & Lundberg, 1959). Stimulation of these afferents

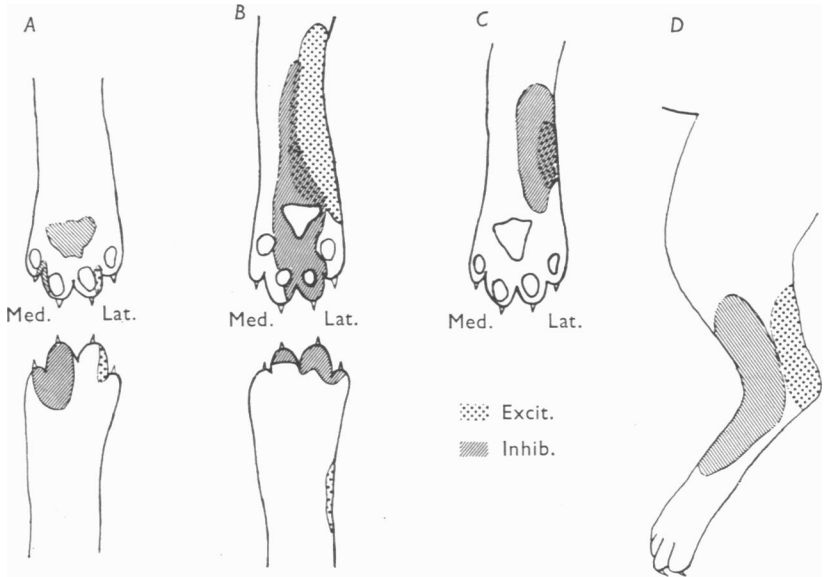


Fig. 7. Excitatory and inhibitory receptive fields of four SCT cells. The fields were determined as for the cell illustrated in Fig. 6.

evoked synaptic effects in the great majority of SCT neurones. The previous finding that some SCT neurones are excited by volleys in the FRA (Lundberg & Oscarsson, 1961; Lundberg, Norrsell & Voorhoeve, 1963) was confirmed as is shown in Fig. 8 (cf. also Fig. 1). The usual monosynaptic EPSPs and disynaptic IPSPs (*B* and *C*) were evoked from low threshold cutaneous afferents, while volleys in high threshold muscle and cutaneous afferents evoked longer lasting polysynaptic EPSPs (*D-F*). A train of volleys in the contralateral FRA (*G-H*) evoked the opposite effect, polysynaptic IPSPs. EPSPs from the ipsilateral FRA were observed only in the minority of neurones (7 of 22, Table 1) but small EPSPs may have been overlooked, in particular when the cells were depolarized and IPSPs concomitantly evoked.

IPSPs from the ipsilateral FRA were found in eighteen of twenty-two cells investigated (Table 1). Figure 9 illustrates such IPSPs from cutaneous (*E, F, H, I*), joint (*G, J*) and high threshold muscle (*K*) afferents. The IPSP from the joint nerve (*G, J*) has two components, the second with a time course resembling that of the IPSP evoked from high threshold muscle afferents (*K*). In many SCT cells only late IPSPs were evoked from joint afferents (cf. Fig. 11 *H*). It is possible that only the late wave should

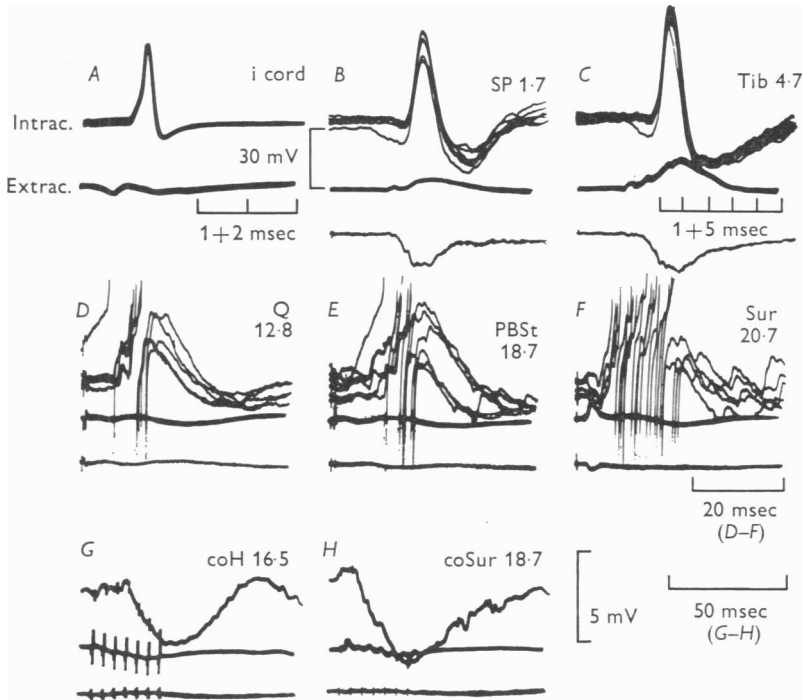


Fig. 8. An SCT neurone receiving polysynaptic EPSPs from the ipsilateral FRA. Intracellular records (upper traces) with a citrate electrode from a cell located at a depth of 1.85 mm from the cord dorsum. Extracellular records (lowermost traces in *B-H*) were taken from just outside of the cell. The lower trace in *A* and middle traces in *B-H* are from the L7 dorsal root entry zone. *A*: antidromically evoked spike potential to stimulation of the ipsilateral spinal cord in the lower thoracic region. *B* and *C*: EPSPs and subsequent IPSPs evoked from SP (*B*) and Tib (*C*). *D-F*: polysynaptic EPSPs from high threshold muscle afferents in Q (*D*) and PBSt (*E*) and high threshold cutaneous afferents in Sur (*F*). No PSPs are evoked from group I fibres in *D* and *E*. *G* and *H*: polysynaptic IPSPs from high threshold muscle (*G*) and cutaneous (*H*) afferents of the contralateral hindlimb. Observe reciprocal effects from the ipsilateral (*D-F*) and contralateral (*G-H*) FRA. Voltage calibration (for micro-electrode recording) in *A* is for *A* and in *H* is for *B-H*. Time calibration in *A* is for *A*, in *C* (1+5 msec) is for *B* and *C*, in *F* (20 msec) is for *D-F* and to the right of *H* (50 msec) is for *G* and *H*. *G* and *H* show single traces.

be classified as an FRA effect presumably mediated by the interneuronal pathway shared by cutaneous and high threshold muscle afferents. The same consideration holds for the late IPSP evoked from cutaneous afferents (Fig. 11 *G*).

In some neurones mixed excitatory and inhibitory actions were evoked from the FRA (Table 1). The neurone illustrated in Fig. 10 was exceptional in that it received almost no effects from the FRA (*I-L*), whereas as usual

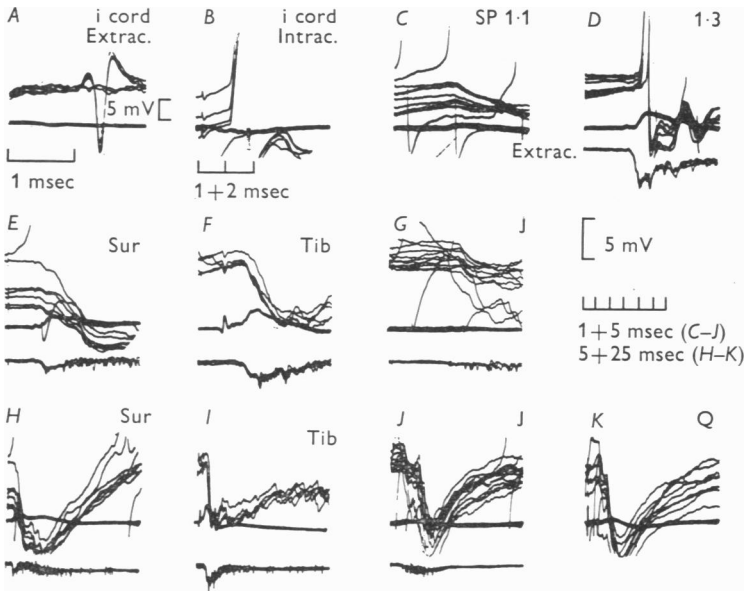


Fig. 9. An SCT neurone receiving polysynaptic IPSPs from FRA. Intracellular (except in *A*) records (upper traces) with a K-citrate electrode from a cell located at a depth of 1.45 mm from the cord dorsum. Lowermost traces in *D-J* are extracellular records from just outside the cell. Lower traces in *A-C* and *K* and middle traces in *D-J* are from the L7 dorsal root entry zone.

TABLE 1. Effects from ipsilateral and contralateral FRA in twenty-two SCT neurones. The left column gives numbers of cells in which stimulation of the FRA evoked IPSPs, EPSPs, mixed excitatory and inhibitory post-synaptic potentials or else had no detectable effect. In nine of these cells the effects from the coFRA were tested and are shown in the right column, separately for the cells with different PSPs from the iFRA

	iFRA	coFRA
IPSP	14/22	IPSP 5/6 No effect 1/6
EPSP	3/22	No effect 1/1
IPSP + EPSP	4/22	IPSP 1/2 No effect 1/2
No effect	1/22	

monosynaptic EPSPs and disynaptic IPSPs were evoked from the low threshold cutaneous afferents (*C-H*).

Effects from the contralateral FRA were examined in nine cells, IPSPs were evoked in six of them while no appreciable effects were detected in three cells. The IPSPs from the contralateral FRA were found in neurones receiving EPSPs as well as in those receiving IPSPs from the ipsilateral FRA. These crossed IPSPs may contribute to contralateral inhibition of discharges in SCT neurones (Taub, 1964) and of neurones in the lateral cervical nucleus (Gordon & Jukes, 1963).

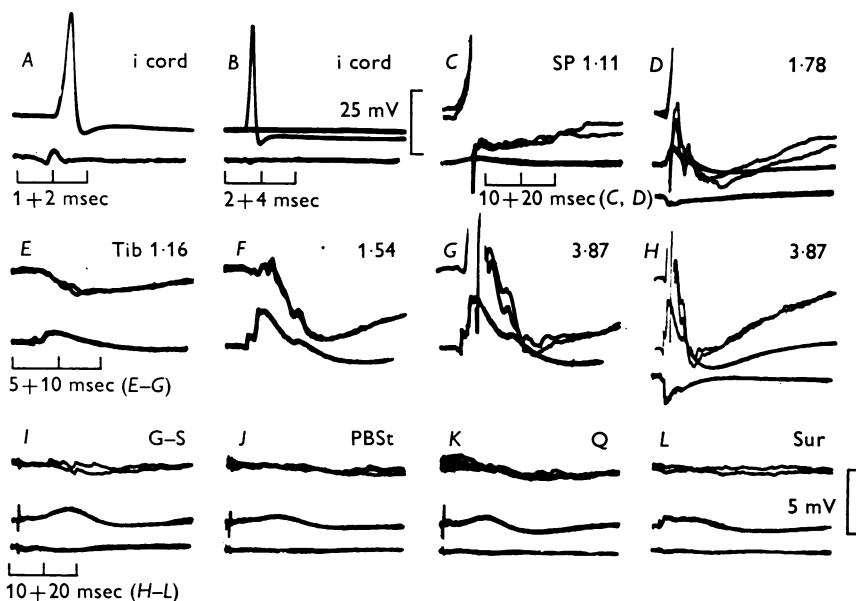


Fig. 10. An SCT neurone uninfluenced from FRA. Intracellular recording (upper traces) with a citrate electrode from a cell located at a depth of 1.1 mm from the cord dorsum (the same cell as in Fig. 3). The lowermost traces in *D*, *H* and *I-L* are extracellular field potentials recorded just outside the cell. Middle traces in *D*, *H* and *I-L* and lower traces in *A-C* and *E-G* are from the L7 dorsal root entry zone. *A* and *B*: antidromic spike potentials evoked by stimulation of the ipsilateral spinal cord in the lower thoracic region. In *B* the stimulation is at threshold for the axon of the neurone. *C-H*: PSPs and spike potentials evoked from SP (*C*, *D*) and Tib (*E-H*) nerves. Both EPSPs and IPSPs are evoked from these nerves. Spike potentials are initiated in *C*, *D*, *G* and *H* but the hyperpolarizations following the spikes in *D*, *G* and *H* are largely IPSPs as appears from a comparison with records *D* and *F*. The hyperpolarization in *C*, on the other hand, is almost entirely an after-hyperpolarization, the magnitude and time course being similar to that in *B* (see also Fig. 3). *I-J* show absence of effects from high threshold muscle afferents and cutaneous afferents. Voltage calibration (for micro-electrode recording) in *B* applies to *A* and *B* and in *L* to *C-L*. The time calibration below *C* (10+20 msec) is for *C*, *D* below *E* (5+10 msec) for *E-H* and below *I* (10+20 msec) for *I-L*.

Effects from other primary afferents

(a) *Interosseous and joint afferents.* IPSPs of simple configuration could be evoked from fibres in the group I range of the FDL nerve. Examples of such IPSPs are shown in Fig. 11 *B-D* together with PSPs from cutaneous (*E-G*) and joint afferents (*H*). IPSPs from FDL were found in four of sixteen neurones tested. In all the four cases the interosseous branch (cf. Hunt & McIntyre, 1960) was stimulated together with the FDL nerve.

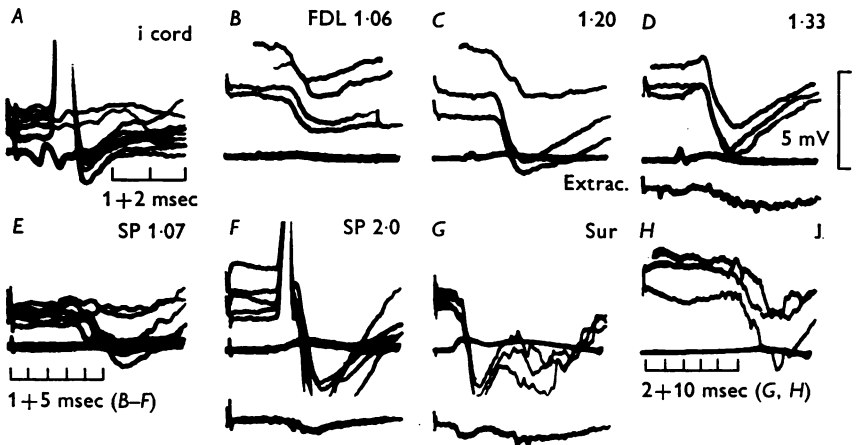


Fig. 11. Disynaptic IPSPs from low threshold fibres in the FDL nerve. Intracellular recording (upper traces) with a citrate electrode from a cell located at a depth of 1.35 mm from the cord dorsum. The lowermost traces in *D*, *F* and *G* are extracellular records taken after withdrawal of the electrode to a position just outside the cell. Note that in *D* the extracellular recording was done with a different sweep speed than the intracellular recording. Lower traces in *A-C*, *E* and *H* and middle traces in *D*, *F* and *G* are from the L7 dorsal root entry zone. *A*: antidromic invasion from the ipsilateral spinal cord. *B-D*: IPSPs evoked from fibres of group I range in the FDL nerve with the interosseous branch. The central latency is comparable to that of cutaneous IPSPs (cf. *E*) and suggests a disynaptic linkage. *E-G*: PSPs and spike potentials evoked from SP (*E* and *F*) and Sur (*G*) nerve. *H*: polysynaptic IPSPs from J nerve. Note different time courses of IPSPs from FDL (*B-D*) and the FRA (*G* and *H*). Voltage calibration in *D* applies to all the micro-electrode recordings. Time calibration below *A* (1+2 msec) is for *A*, below *E* (1+5 msec) for *B-F* and below *H* (2+10 msec) for *G* and *H* and the extracellular record in *D*.

In two experiments the interosseous nerve was dissected for separate stimulation. Figure 12 shows that an IPSP having the same time course as in Fig. 11 *B-D* was evoked from the interosseous branch (*E*) while stimulation of the FDL nerve at maximal group I strength (*F*) had no effect. It is suggested that the fast conducting fibres in the interosseous nerve, but not the group I muscle afferents from FDL, give rise to the IPSP under discussion. This IPSP differed from that evoked from the

FRA in that its time course was characteristically short and simple like the group Ia IPSP in alpha motoneurons (e.g. Coombs, Eccles & Fatt, 1955). The central latency was about 1.5 msec (Figs. 11 *B-D*, 12 *E*) which suggests a disynaptic linkage. The duration of this IPSP was much shorter than of the IPSPs from the FRA (cf. Fig. 11 *B-D* and *G-H*; note different sweep speeds). Thus it is postulated that the disynaptic IPSP from the

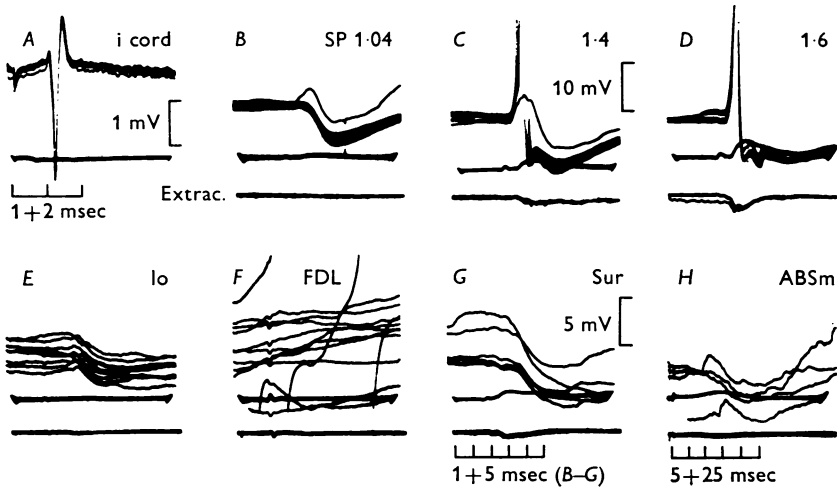


Fig. 12. Disynaptic IPSPs evoked by volleys in low threshold fibres in the interosseous nerve. Intracellular (upper traces in *B-H*) and extracellular records (*A*) with a KCl electrode from a cell located 1.5 mm from the cord dorsum. The lowest traces in *B-H* are extracellular records from just outside the cell. Lower trace in *A* and middle traces in *B-H* are from the L7 dorsal root entry zone. *A*: extracellular spike potential antidromically evoked by stimulation of ipsilateral spinal cord in the lower thoracic region just before penetration of the cell. *B-D*: disynaptic IPSPs and EPSPs with spikes on them, evoked from the SP nerve. *E*: IPSP (probably disynaptic) evoked by stimulating low threshold fibres in interosseous nerve (Io). The IPSP has a similar latency and configuration as that from FDL containing the interosseous branch (Fig. 11 *B-D*). The latency is comparable to that of IPSPs from cutaneous nerves (*B* and *G*). *F*: records showing no effect from group I FDL fibres. *G*: disynaptic IPSPs from Sur nerve. *H*: polysynaptic IPSPs from the FRA (high threshold afferents in ABSm). Observe the different time courses of the IPSPs from the Io nerve (*E*) and from the FRA (*H*) (different sweep speeds). Voltage calibration (for micro-electrode recording) in *A* is for *A*, in *C* for *B-D* and in *G* for *E-H*. Time calibration below *A* (1+2 msec) is for *A*, below *G* (1+5 msec) for *B-G* and below *H* (5+25 msec) for *H*.

interosseous nerve does not belong to the FRA response, but indicates a more specific action from the afferents connected with Pacinian corpuscles (Hunt & McIntyre, 1960) on SCT neurones.

On some occasions it was observed that volleys in the joint nerve evoked an early IPSP (Fig. 9 *G*). The stimulus strength was not graded in

these cases, and it is neither known if this early IPSP was evoked from the large joint afferents nor if the linkage was disynaptic since the arrival of the afferent volley could not be detected with certainty in the record from the dorsal root entry zone.

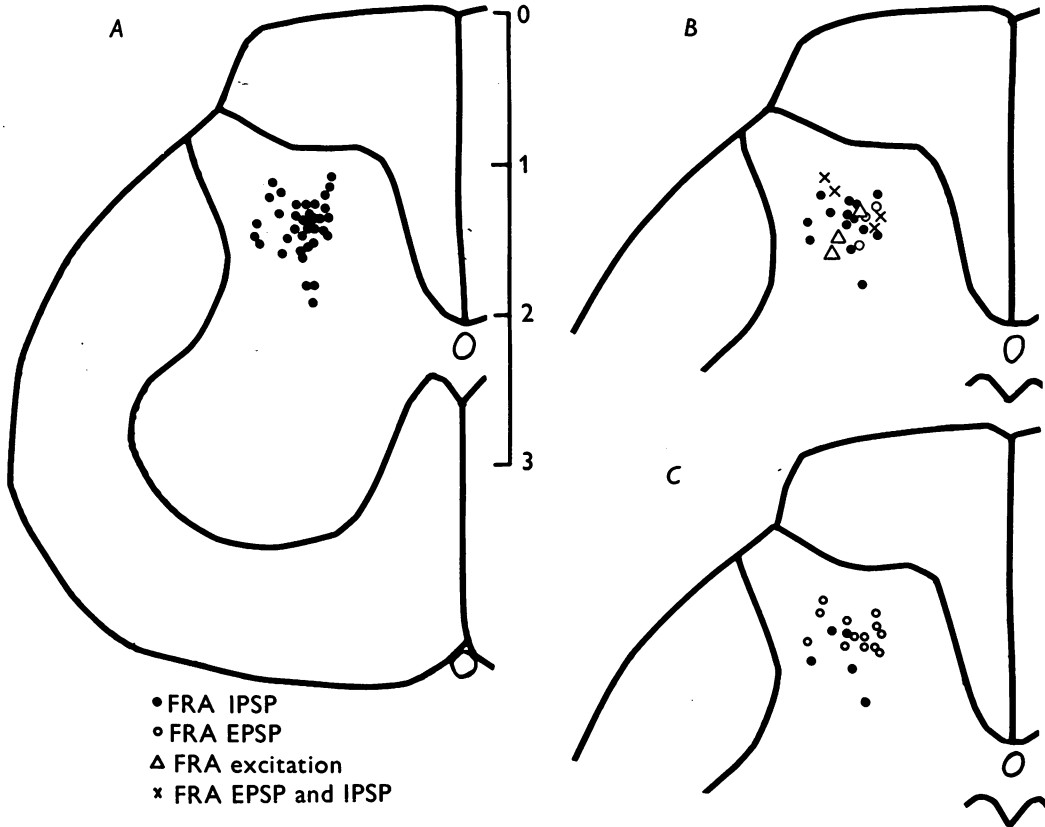


Fig. 13. Location of SCT neurones. The location of individual neurones was determined from the reading (on the micromanipulator scale) of the distance from the cord dorsum at which the cells were recorded from and by the identification of the electrode tracks in histological sections. Due correction was made for shrinkage during histological procedures which was about 15%. In the drawings of A-C the location of all the cells is projected on a representative transverse section of the L7 spinal cord. A shows location of thirty-six SCT neurones recorded either intracellularly or extracellularly. The drawings in B and C are made for the intracellularly recorded neurones analysed in detail. B shows the location of cells with polysynaptic EPSPs (open circles), IPSPs (filled circles), and both EPSPs and IPSPs (crosses) from the ipsilateral FRA (triangles) indicate location of units excited from FRA and recorded extracellularly). There is no evidence for a specific location depending on the difference in effect from FRA. C shows the location of cells with (filled circles) and without (open circles) disynaptic IPSPs from low threshold fibres in the I_o nerve or the FDL nerve containing the interosseous branch.

(b) *Group I muscle afferents.* No PSPs, excitatory or inhibitory, were evoked by volleys in group I muscle afferents (e.g. Figs. 1 *G*, 10 *I-K*, 12 *F*).

Location of SCT neurones

The location of thirty-six SCT cells was determined as described by Hongo *et al.* (1966*b*), and is shown in a representative transverse plane of the L7 spinal cord in Fig. 13 *A-C*. Each point does not necessarily indicate a relative position with respect to the lamination of the dorsal horn (Rexed, 1952) but should be referred to by the distance from the dorsal surface. Figure 13 *A* shows that all these neurones were found in the dorsal horn at depths from the cord dorsum, ranging between 1.1 and 1.8 mm. Figure 13 *B* shows the location of neurones with excitation (open circles), inhibition (filled circles) and mixed excitation and inhibition (crosses) from the FRA. There is no evidence for a specific location of cells with the different effects from the FRA. Data for neurones with (filled circle) and without (open circle) disynaptic IPSPs from the interosseous (or interosseous and the FDL) nerve are given in Fig. 13 *C*. The former group seems to be located more ventrally than the latter.

DISCUSSION

The combination of monosynaptic EPSPs and disynaptic IPSPs from cutaneous afferents is the principal pattern of synaptic actions on to the SCT cells and was observed in virtually all the neurones investigated. Similar findings were made in interneurons in the dorsal horn and the intermediary region (Hongo *et al.* 1966*b*). It is assumed that both these effects on the SCT neurones are specific actions of the cutaneous afferents and not part of an effect from the FRA: the reason is given under Results. It has also been shown above that the disynaptic IPSPs are not mediated by a recurrent inhibitory pathway. Both the monosynaptic EPSP and the disynaptic IPSP are evoked from large cutaneous afferents and experiments with adequate stimulation revealed that IPSPs as well as excitation can be produced by light touch of hairs. Most likely, the naturally produced effects are evoked by impulses conducted in the fibres which mediate the electrically evoked monosynaptic EPSPs and disynaptic IPSPs. In experiments with time-controlled mechanical stimulation it has recently been found that mechanical stimulation of hairs can evoke both EPSPs and IPSPs at latencies compatible with the velocity of fast conducting fibres (T. Hongo & H. Koike, unpublished). Taken together these findings show that the monosynaptic EPSPs and the disynaptic IPSPs are evoked from the same afferent system originating in hair receptors. It is not known if

hair receptors connected to slow conducting fibres (Brown & Iggo, 1964) also contribute.

Our finding that discharges in SCT cells can be inhibited by impulses in cutaneous afferents confirms the observation by Taub (1964) and is of interest since several investigators did not find inhibitory effects from hair receptors either in SCT or in its subsequent relays (Wall, 1960; Lundberg & Oscarsson, 1961; Andersson, 1962; Gordon & Jukes, 1963; Horrobin, 1966). The explanation may be that inhibition has been looked for against an excitation that was too powerful; recent experiments have disclosed a considerable convergence of primary afferents on to SCT neurones and also that impulses in single afferent fibres innervating the control region of the receptive field may evoke unitary EPSPs large enough to fire the neurones (Hongo & Koike, 1968). It may be that the IPSP can inhibit discharges evoked from the periphery but not the more potent excitation from the centre of the receptive field.

Surround inhibition is a characteristic feature in the dorsal column nuclei (Gordon & Paine, 1960). In SCT cells, on the other hand, the receptive field from which unitary IPSPs can be evoked by hair movement is not of a surround type but is eccentric to the excitatory field. This spatial arrangement of the inhibitory receptive field accords with Taub's description of the region from which SCT impulses could be inhibited as 'discrete, patchy and nonconcentric'. Since in both cases the inhibitory effects were evoked from hairs it is possible that the afferent inhibition described by Taub is post-synaptic. However, recent experiments have revealed a high degree of specificity also for the primary afferent depolarization evoked in the central terminals of cutaneous afferents (Jänig, Schmidt & Zimmermann, 1968). Hence it is at present difficult to draw any conclusion regarding the relative role of presynaptic and post-synaptic mechanisms for inhibition of transmission from tactile afferents.

It seems of special interest that tactile information is conducted by two ascending paths with different spatial organization of the excitatory and inhibitory receptive fields. The inhibition of a surround type in the path from the dorsal column may be of significance in increasing contrast in spatial discrimination as has been repeatedly pointed out both for this pathway (Gordon & Paine, 1960; Mountcastle & Powell, 1959; Andersson, 1962) and for other sensory systems (Kuffler, 1953; Hartline & Ratliff, 1957, 1958; von Bekesy & Rosenblith, 1951). Does the non-concentric distribution of the excitatory and inhibitory fields in SCT cells indicate that the SCT mediates information regarding other characteristics of the tactile stimuli, or is it merely an aberrant form of surround inhibition?

In this connexion the analysis of receptive fields and specific stimuli of cells in the visual cortex should be recalled. Most of the simple cells de-

scribed by Hubel & Wiesel (1959, 1962) have receptive fields consisting of an elongated centre with more or less symmetrical antagonistic flanks. This arrangement may permit discrimination of the orientation of elongated objects in the visual field. However, in some cells the flanking fields are not symmetrical, the extreme case being when one of the flanks is missing entirely so that the cells have an excitatory and an inhibitory field side by side. Some of the latter cells responded specifically to movement of a visual stimulus in one direction. Hubel & Wiesel (1959) discussed the mechanism by which these cells could have a unidirectional sensitivity.

The eccentric fields in the SCT could in a similar way provide a basis for information regarding the direction of a tactile stimulus. It should be noted, however, that the hypothesis of a directional sensitivity in the SCT is based entirely on the arrangement of the receptive fields from which the IPSPs are drawn; there is only very limited information from studies of the discharges in these neurones (cf. Taub, 1964). A further analysis of the directional sensitivity in SCT is likely to be difficult not only because tactile stimuli are much more difficult to handle and quantify than visual stimuli but also because we are dealing with second order neurones. It may well prove more favourable to test this hypothesis in the subsequent relays from SCT. To some extent this is suggested by the findings from the visual system, where in spite of the marked asymmetrical receptive field arrangement in some simple cells with unidirectional sensitivity in the visual cortex, no corresponding asymmetries have been found in the lower order visual neurones in the cat. In this connexion it is of great interest that Gordon & Manson (1967) have observed directional sensitivity in some 'touch-pressure' relay cells in the nucleus ventralis posterolateralis in the thalamus.

By contrast to the effects from tactile afferents the excitatory and inhibitory effects from the FRA on SCT neurones are drawn from large receptive fields often consisting of the entire hind limb and in the case of inhibition also from the contralateral hind limb. Large receptive fields are a characteristic feature of the effects from the FRA on ascending pathways (cf. Lundberg, 1964*a*), one exception being the recently discovered system which ascends in the dorsal column (Uddenberg, 1968). It has been suggested that ascending pathways influenced from the FRA may inform higher centres regarding interneuronal transmission in the flexor reflex pathways (R. M. Eccles & Lundberg, 1959). Support for this hypothesis has been obtained in investigations showing that a variety of descending systems converge on to interneurones of the flexor reflex pathways and of the spinobulbar pathways (Lundberg, 1964*a, b*). However, while this hypothesis may hold true for the ventral spinobulbar systems (cf. also

Oscarsson, 1967) and possibly also for some subdivisions of the dorsal spinocerebellar tract we do not favour it with regard to the FRA effects on the SCT. The inhibitory effects from the FRA may constitute an interactive mechanism between the SCT and other ascending pathways similar to those operating between spinal reflex pathways. If the limb is submitted to strong stimulation, the higher centres may not require detailed information from hairs and the IPSPs evoked from the FRA may assist in preventing this information from reaching higher centres (Fedina, Gordon & Lundberg, 1968).

Another way of looking at these FRA effects is by assuming that the SCT neurones may serve as afferents to a supraspinal reflex centre. On the basis of this hypothesis it is perhaps easier to understand the significance of the excitatory actions that volleys in the FRA evoke in some SCT neurones. If the reflex movements assumed to be evoked from the specific tactile afferents partly coincide with those produced from the FRA (for example in tactile placing which initially is dominated by flexion) a facilitatory convergence from the two afferent systems may be of some advantage. Also, it seems reasonable that motor adjustments produced by tactile stimuli should be inhibited by strong stimuli, which in themselves evoke large reflex movements.

The present results suggest that the SCT may have more than the two channels recognized by Lundberg & Oscarsson (1961). Only future experiments can show whether the effects from the ipsi- and contralateral FRA are significant enough to merit subdivision of the neurones according to their presence or absence and distribution. Neurones with disynaptic IPSPs from afferents of vibratory receptors (and possibly also those with short-latency IPSP from joint afferents) may also form a separate subgroup of SCT. It has already been suggested above that this effect is not part of the FRA response. It may constitute an inhibitory interactive mechanism similar to that discussed above for the FRA so that transmission from tactile receptors in some SCT neurones is inhibited from vibratory receptors (or by activation of the appropriate joint receptors). An alternative possibility is that the inhibitory effect in some way can be decoded at a cortical level and may have a more direct sensory significance. McIntyre (1962) has shown that afferents from vibratory receptors in the interosseous nerve give an evoked potential in the somatosensory cortical area and that this effect is mediated by the dorsal but not by the lateral column. If a difference of activity in the two ascending systems is utilized at higher levels of these sensory paths the reciprocal action at the first relays may amplify the effect from the vibratory afferents. In any case our results suggest that the function of the spinocervico-lemniscal system is not entirely limited to tactile events.

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REFERENCES

- ANDÉN, N.-E., JUKES, M. G. M., LUNDBERG, A. & VYKLYCKÝ, L. (1966). The effect of DOPA on the spinal cord. I. Influence on transmission from primary afferents. *Acta physiol. scand.* **67**, 373-386.
- ANDERSSON, S. A. (1962). Projection of different spinal pathways to the second somatic sensory area in cat. *Acta physiol. scand.* **56**, suppl. 194.
- BROWN, A. G. & IGGO, A. (1964). Hair follicle receptors with myelinated afferent nerve fibres. *J. Physiol.* **172**, 33P.
- COOMBS, J. S., ECCLES, J. C. & FATT, P. (1955). The specific ionic conductances and the ionic movements across the motoneuronal membrane that produce the inhibitory post-synaptic potential. *J. Physiol.* **130**, 326-373.
- ECCLES, J. C. (1957). *The Physiology of Nerve Cells*. Baltimore: The Johns Hopkins Press.
- ECCLES, J. C., ECCLES, R. M. & LUNDBERG, A. (1960). Types of neurone in and around the intermediate nucleus of the lumbosacral cord. *J. Physiol.* **154**, 89-114.
- ECCLES, R. M. & LUNDBERG, A. (1959). Synaptic actions in motoneurons by afferents which may evoke the flexion reflex. *Archs ital. Biol.* **97**, 199-221.
- FEDINA, L., GORDON, G. & LUNDBERG, A. (1968). The source and mechanisms of inhibition in the lateral cervical nucleus of the cat. *Brain Res.* (In the Press.)
- GORDON, G. & JUKES, M. G. M. (1963). An investigation of cells in the lateral cervical nucleus of the cat which respond to stimulation of the skin. *J. Physiol.* **169**, 28-29 P.
- GORDON, G. & JUKES, M. G. M. (1964). Descending influences on the exteroceptive organizations of the cat's gracile nucleus. *J. Physiol.* **173**, 291-319.
- GORDON, G. & MANSON, J. R. (1967). Cutaneous receptive fields of single nerve cells in the thalamus of the cat. *Nature, Lond.* **215**, 597-599.
- GORDON, G. & PAINE, C. H. (1960). Functional organization in nucleus gracilis of the cat. *J. Physiol.* **153**, 331-349.
- HARTLINE, H. K. & RATLIFF, F. (1957). Inhibitory interaction of receptor units in the eye of *Limulus*. *J. gen. Physiol.* **40**, 357-376.
- HARTLINE, H. K. & RATLIFF, F. (1958). Spatial summation of inhibitory influences in the eye of *Limulus*, and the mutual interaction of receptor units. *J. gen. Physiol.* **41**, 1049-1066.
- HONGO, T., JANKOWSKA, E. & LUNDBERG, A. (1966a). Post-synaptic inhibition evoked from primary afferents in neurones of the spinocervical tract. *J. Physiol.* **186**, 35-36 P.
- HONGO, T., JANKOWSKA, E. & LUNDBERG, A. (1966b). Convergence of excitatory and inhibitory action on interneurons in the lumbosacral cord. *Expl Brain Res.* **1**, 338-358.
- HONGO, T. & KOIKE, H. (1968). Large-sized EPSPs evoked from single cutaneous afferents in secondary ascending neurones in cats. *Proceedings of the International Union of Physiological Sciences*, vol. VII. XXIV International Congress, p. 198. Washington, D.C.
- HONGO, T. & OKADA, Y. (1967). Cortically evoked pre- and post-synaptic inhibition of impulse transmission to the dorsal spinocerebellar tract. *Expl Brain Res.* **3**, 163-177.
- HORROBIN, D. F. (1966). The lateral cervical nucleus of the cat; an electrophysiological study. *Q. Jl exp. Physiol.* **51**, 351-371.
- HUBEL, D. H. & WIESEL, T. N. (1959). Receptive fields of single neurones in the cat's striate cortex. *J. Physiol.* **148**, 574-591.
- HUBEL, D. H. & WIESEL, T. N. (1962). Receptive fields, binocular interaction and functional architecture in the cat's visual cortex. *J. Physiol.* **160**, 106-154.
- HUNT, C. C. & MCINTYRE, A. K. (1960). Characteristics of responses from receptors from the flexor longus digitorum muscle and the adjoining interosseous region of the cat. *J. Physiol.* **153**, 74-87.
- ITO, M. & OSHIMA, T. (1962). Temporal summation of afterhyperpolarization following a motoneurone spike. *Nature, Lond.* **195**, 910-911.
- JÄNG, W., SCHMIDT, R. F. & ZIMMERMANN, M. (1968). Two specific feedback pathways to the central afferent terminals of phasic and tonic mechanoreceptors. *Expl Brain Res.* **6**, 116-129.
- KATZ, B. & MILEDI, R. (1963). A study of spontaneous miniature potentials in spinal motoneurons. *J. Physiol.* **168**, 389-422.

- KUFFLER, S. W. (1953). Discharge patterns and functional organization of mammalian retina. *J. Neurophysiol.* **16**, 37-68.
- LUNDBERG, A. (1964a). Ascending spinal hindlimb pathways in the cat. *Prog. Brain Res.* **12**, 135-163.
- LUNDBERG, A. (1964b). Supraspinal control of transmission in reflex paths to motoneurons and primary afferents. *Prog. Brain Res.* **12**, 197-221.
- LUNDBERG, A., NORRELL, U. & VOORHOEVE, P. (1963). Effects from the sensorimotor cortex on ascending spinal pathways. *Acta physiol. scand.* **59**, 462-473.
- LUNDBERG, A. & OSCARSSON, O. (1961). Three ascending spinal pathways in the dorsal part of the lateral funiculus. *Acta physiol. scand.* **51**, 1-16.
- MCINTYRE, A. K. (1962). Cortical projection of impulses in the interosseous nerve of the cat's hind limb. *J. Physiol.* **163**, 46-60.
- MORIN, F. (1955). A new spinal pathway for cutaneous impulses. *Am. J. Physiol.* **183**, 245-252.
- MOUNTCASTLE, V. B. & POWELL, T. P. S. (1959). Neural mechanisms subserving cutaneous sensibility, with special reference to the role of afferent inhibition in sensory perception and discrimination. *Johns Hopkins Hosp. Bull.* **105**, 201-232.
- NORRELL, U. (1966). The spinal afferent pathways of conditional reflexes to cutaneous stimuli in the dog. *Expl Brain Res.* **2**, 269-282.
- NORRELL, U. & VOORHOEVE, P. (1962). Tactile pathways from the hindlimb to the cerebral cortex in cat. *Acta physiol. scand.* **54**, 9-17.
- OSCARSSON, O. (1967). Functional significance of information channels from the spinal cord to the cerebellum. In *Neurophysiological Basis of Normal and Abnormal Motor Activities*, ed. JAHR, M. D. & PURPURA, D. P. Hewlett, N.Y.: Raven Press.
- REXED, B. (1952). The cytoarchitectonic organization of the spinal cord in the cat. *J. comp. Neurol.* **96**, 415-496.
- SZENTÁGOTHAJ, J. (1964). Neuronal and synaptic arrangement in the substantia gelatinosa Rolandi. *J. comp. Neurol.* **122**, 219-230.
- TAUB, A. (1964). Local segmental and supraspinal interaction with a dorsolateral spinal cutaneous afferent system. *Expl Neurol.* **10**, 357-374.
- TAUB, A. & BISHOP, P. O. (1965). The spinocervical tract, dorsal column linkage, conduction velocity, primary afferent spectrum. *Expl Neurol.* **13**, 1-21.
- UDDENBERG, N. (1968). Functional organization of long, second order afferents in the dorsal funiculus. *Expl Brain Res.* **4**, 377-382.
- VON BEKESY, G. & ROSENBLITH, W. A. (1951). The mechanical properties of the ear. In *Handbook of Experimental Psychology*, pp. 1075-1115. New York: John Wiley and Sons, Inc.
- WALL, P. D. (1960). Cord cells responding to touch, damage and temperature stimuli. *J. Neurophysiol.* **23**, 197-210.
- WALL, P. D. (1965). Impulses originating in the region of dendrites. *J. Physiol.* **180**, 116-133.