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AFFERENT CONNEXIONS TO GROUP I ACTIVATED CELLS IN THE MAIN CUNEATE NUCLEUS OF THE CAT

BY INGMAR ROSÉN

From the Institute of Physiology, University of Lund, Lund, Sweden

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SUMMARY

1. Extracellular recordings were made from a total of 240 group I activated cells in the main cuneate nucleus. Cuneothalamic relay neurones (128) were identified by antidromic stimulation of the medial lemniscus in the ventrobasal thalamic complex.

2. A majority of the relay neurones were activated by afferents in only one of six dissected forelimb nerves innervating muscle groups at various joints. Even among afferents from adjacent synergistic muscles, convergence to individual neurones was infrequent.

3. Some of the relay neurones received excitation from group II muscle afferents in the same nerve that provided group I excitation. Excitation from group II muscle afferents in other nerves was uncommon. Some neurones were weakly excited by cutaneous volleys.

4. Inhibition of group I relay cells was produced from cutaneous afferents and group II muscle afferents. Weak inhibition was sometimes observed from group I afferents. The relay cells were also inhibited by stimulation of the cerebral cortex with a focus around the lateral end of the cruciate sulcus. A good correspondence was found between the inhibition and the depolarization of group I afferent terminals in the cuneate nucleus.

5. A majority of the group I activated cells not antidromically activated from the ventrobasal complex ('non-relay cells') were excited by cortical stimulation. Excitation from cutaneous afferents and group II muscle afferents was frequently found among these cells.

6. The group I activated cells were found almost exclusively in the ventral part of the nucleus.

7. The pattern of convergence found in eleven group I activated cells in the dorsal horn of the spinal cord from C 2 to C 4 is described.

INTRODUCTION

It was recently demonstrated that group I forelimb muscle afferents ascending in the dorsal funiculus evoke monosynaptic activity in the main and external cuneate nuclei and in the dorsal horn of the rostral part of the cervical cord (Rosén, 1969*a*). The group I activated region of the main cuneate nucleus was localized to the ventral part of the nucleus and contains the second order neurones of the group I pathway to the cerebral cortex (Oscarsson & Rosén, 1963). The patterns of convergence of excitation and inhibition to neurones in one of the cortical projection areas of the group I afferent pathway have been studied (Oscarsson, Rosén & Sulg, 1966). Many neurones showed extensive convergence: they were influenced by group I afferents from many muscles of different function and also by cutaneous afferents. This pattern is different from what has been observed in other ascending group I activated pathways (Oscarsson, 1965). The thalamocortical neurones of the pathway were shown to receive convergence from adjacent synergistic muscles and from cutaneous afferents (Andersson, Landgren & Wolsk, 1966). One aim of the present investigation has been to study the patterns of convergence among the cuneothalamic relay neurones. A similar detailed study has also been made on the thalamocortical relay cells and will be presented in a subsequent paper (Rosén, 1969*b*), which will contain a discussion of the changes in convergence patterns at different levels of the group I afferent pathway to the cortex.

The transmission in the dorsal funiculus nuclei is under control from the cerebral cortex (Hernàndez-Peón, Scherrer & Velasco, 1956; Magni Melzack, Moruzzi & Smith, 1959; Jabbur & Towe, 1961; Towe & Jabbur 1961; Guzmán-Flores, Buendia, Anderson & Lindsley, 1962; Satterfield, 1962; Chambers, Liu & McCouch, 1963; Dawson, Podachin & Schatz, 1963; Andersen, Eccles, Schmidt & Yokota, 1964*b*, *c*, *d*; Andersen, Eccles, Oshima & Schmidt, 1964*a*; Gordon & Jukes, 1964; Levitt, Carreras, Liu & Chambers, 1964; Winter, 1965). Previous investigations have been concerned with the control of the cutaneous relay in these nuclei. A second aim of this investigation has been to establish if similar corticofugal mechanisms are active in the group I afferent pathway, which has different cortical projection areas (Oscarsson & Rosén, 1963, 1966; Landgren, Silfvenius & Wolsk, 1967) and which is not concerned with conscious perception (Giaquinto, Pompeiano & Swett, 1963; Merton, 1964; Gelfan & Carter, 1967; Swett & Bourassa, 1967). Some of the results have been reported in brief (Rosén, 1967, 1968).

METHODS

The experiments were performed on cats anaesthetized with pentobarbitone sodium (initial dose 40 mg/kg intraperitoneally and later intravenous doses of 10 mg). The animals were paralysed with gallamine triethiodide and artificially ventilated. Bilateral pneumothorax was performed in order to reduce movements of the brain stem. The body temperature and the mineral oil covering the wounds were kept at 36–38° C. The animals were continuously infused with a glucose-dextran-saline solution. Aramine was given when necessary to keep the blood pressure above 80 mm Hg.

The left dorsal column nuclei and the right frontoparietal cerebral cortex were exposed. The dorsal surface of the thalamus was exposed by sucking away the overlying parietal cortex and hippocampal fimbriae. After the cat had been fixed in a stereotactic frame with the head ventroflexed a silver wire electrode (0·5 mm thick and isolated to the tip) mounted on a micromanipulator was inserted vertically into the thalamus 6 mm lateral to the median plane and 1 mm caudal to the fissure separating the thalamus from the striatum. The electrode was used for recording during the penetration and large short-latency positive potentials on forelimb nerve stimulation could be recorded when it reached the ventrobasal thalamic complex (VPL). The electrode was then left in this position and used as a cathode to stimulate the medial lemniscus at its termination in the VPL (Andersen *et al.* 1964*d*). The following nerves in the left forelimb were cut and dissected for stimulation in different experiments: the muscle nerve component of the suprascapular nerve (SSC), the axillary nerve (Ax), the nerves to the long (LHT) and lateral (LAHT) heads of the triceps muscle, the nerve to the biceps muscle (B), the deep radial nerve (DR) which in some experiments was divided into the nerves to the extensor carpi radialis (ECR), the extensor digitorum communis (EDC), and the remaining part of the deep radial nerve (DRR). In addition the cutaneous nerve, superficial radial (SR), and the mixed ulnar nerve (U) were dissected. The incoming volleys were monitored by recording from the dorsal surface of the cuneate nucleus with a spring mounted ball electrode or a wire attached to a glass plate used for stabilizing the brain stem. The strength of peripheral stimulation is given as multiples of the strength evoking a just visible volley (T).

The stimuli to the peripheral nerves were condenser discharges of short duration (time constant about 40 μsec). The cerebral cortex was stimulated with a set of eight needle electrodes isolated to within 0·5 mm of their tips and inserted to about 1 mm below the cortical surface at fixed spots within the sensorimotor cortex. The stimuli through these electrodes and through the thalamic electrode were negative rectangular pulses of 0·2 msec duration. The positive electrode was placed in the temporal muscle and on the dorsal surface of the thalamus on cortical and thalamic stimulation, respectively. In some experiments the cerebral cortex was stimulated with bipolar surface electrodes (interelectrode distance about 1 mm). The current passing through the electrodes was calibrated by measuring the voltage difference across a 100 Ω series resistance. The antidromic thalamo-cuneate volley was monitored by recording from the dorsal surface of the cuneate nucleus.

Unitary discharges in the cuneate nucleus were recorded with capillary glass electrodes, filled with 2 M potassium citrate solution and with a resistance of about 7 MΩ, connected to a cathode follower. The micro-electrode and surface recordings were displayed on a dual beam oscilloscope.

The mass activity in the medial lemniscus 10 mm rostral to the obex was recorded in five experiments designed to study the inhibition exerted upon cuneothalamic relay neurones from peripheral nerves and the cerebral cortex. The amplitudes of the

lemniscal responses were measured after averaging of 40–100 single sweeps in a CAT 1000 computer. A small electrolytic lesion was made in the medial lemniscus by passing a current through the recording electrode in order to make the responses monophasic. In some experiments capillary micro-electrodes were used for recording from single fibres in the medial lemniscus 5 mm rostral to the obex.

The excitability of the cuneate group I afferent terminals after peripheral or cortical stimulation was tested in eleven experiments by using the method described by Wall (1958) and used in the cuneate nucleus by Andersen *et al.* (1964*c*) and by Carli, Diete-Spiff & Pompeiano (1967*a*).

Experiments were performed on fifty-three cats and recordings were made from 240 group I activated cells in the cuneate nucleus and from 23 group I activated axons in the medial lemniscus. In addition recordings were made from 178 cells activated from cutaneous and/or high threshold muscle afferents. A number of primary afferent fibres were also encountered in the penetrations through the nucleus. Eleven group I activated cells were isolated in penetrations through the cervical spinal cord caudal to the cuneate nucleus (cf. Rosén, 1969*a*). The vast majority of the unitary recordings in this investigation were extracellular. Intracellular recordings for short periods were made from a small number of cells. The cells activated from cutaneous nerves in the dorsal part of the nucleus were more easily impaled than the ventrally situated group I activated cells.

RESULTS

Types of responses. The majority of the unitary responses recorded in the cuneate nucleus were monophasic, negative and some of these became diphasic positive–negative after advancement of the recording electrode. Many of these neurones were spontaneously active and showed repetitive discharges on peripheral stimulation. The latencies of the discharges often shortened with increasing afferent volley. Responses of the type described have been considered to derive from cuneate cells. Monophasic positive unitary responses, often several millivolts in amplitude, have been interpreted as axonal recordings. Most of these responses were encountered in the dorsal part of the cuneate nucleus and in the dorsal funiculus at latencies corresponding to the arrival of the incoming afferent volley and were identified as recordings from primary afferents. A second group of fibres were found intermingled with the primary afferents. The pattern of convergence from cutaneous and high threshold muscle afferents in various forelimb nerves and the repetitive discharges found in this group of axons were similar to those described by Uddenberg (1968) for synaptically activated neurones ascending in the ventral part of the dorsal funiculus. A small group of axons were found in the ventral part of the cuneate nucleus and had the same properties as the cuneate cells. They could often be antidromically activated from the medial lemniscus and were interpreted as axons from cuneate cells. They were excluded from the section dealing with the depth distribution of cuneate neurones.

Identification of relay cells. Cells antidromically activated from the

ventrobasal thalamic complex were classified as cuneothalamic relay cells (CTR cells). The method of colliding the orthodromic and antidromic responses which was described by Darian-Smith, Phillips & Ryan (1963) was chosen as the routine for testing the antidromic nature of spikes evoked by stimulation of the ventrobasal thalamic complex. Of the 221 group I activated cuneate neurones tested 58% could be antidromically invaded. In addition twenty-three group I activated axons in the medial

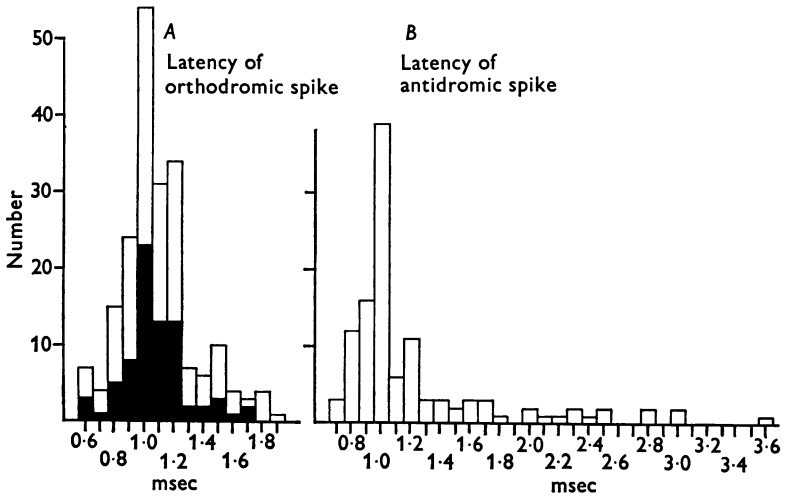

Fig. 1. *A*. Latency histogram of 203 group I evoked extracellularly recorded spike discharges in 136 neurones. White and black areas relate to CTR cells and non-CTR cells, respectively. The latencies were measured from the incoming afferent volley recorded at the level of the cuneate nucleus. *B*. Latency histogram of antidromic spikes evoked in 127 group I activated cells by stimulation of the contralateral ventrobasal thalamic complex.

lemniscus were included among the group I CTR neurones used in the analysis of convergence patterns. The majority of the antidromic spikes were evoked at stimulus strengths below 1 mA. The distribution of antidromic latencies on stimulation of the ventrobasal complex is shown for 127 group I CTR cells in Fig. 1*B*. The modal value was 1·0 msec which would imply a conduction velocity in the medial lemniscus of about 25 m/sec. The antidromic latencies for 75 cutaneous CTR cells were very similar (range 0·9–2·0 msec, mode 1·0 msec). Cells which could not be antidromically invaded are provisionally called non-relay cells (non-CTR cells).

Excitatory and inhibitory effects from the peripheral nerves

Group I excitation. Recordings from group I CTR cells are illustrated in Figs. 2, 3, 5 and 10. Spike discharges could often be evoked at stimulus

strengths at or just above the nerve threshold, indicating that only little spatial summation was required. As illustrated in Fig. 2 *A–D* the number of spikes could often be gradually increased by increasing the afferent volley. It can be concluded that group I afferents in the same nerve converge on single cuneate neurones. This is also shown in the intracellular recordings of Fig. 3 *A–E* and *H–L* where the evoked EPSPs could be gradually increased when increasing the group I afferent volley.

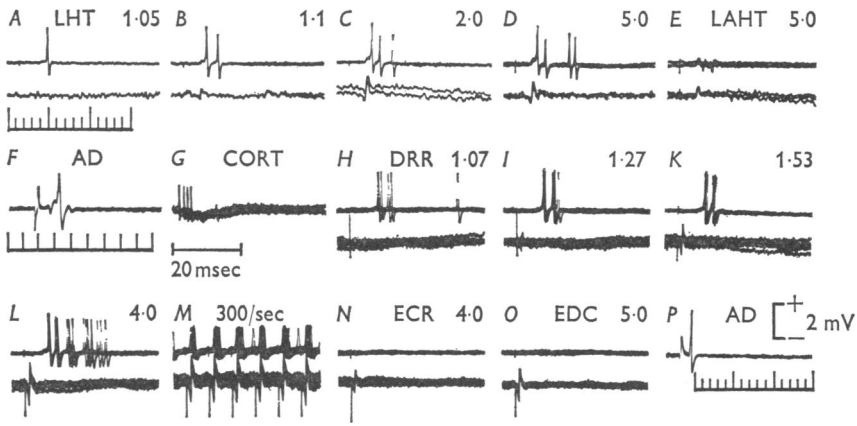

Fig. 2. Spike discharges in two group I CTR cells (*A–G* and *H–P*). Upper traces are micro-electrode recordings. Incoming volleys (lower traces) were recorded from the cuneate nucleus in *A–E* and from the brachial plexus in *H–O*. Note that antidromic volleys in alpha efferents are recorded together with afferent volleys in *H–O*; the DRR group I volley recorded from the cuneate nucleus (not shown) was maximal at 1·5 T. In addition to the large action potentials evoked from LHT in *A–D* small action potentials were recorded from a neighbouring cell activated from LAHT (*E*). A small antidromic spike from this cell is preceding the large antidromic spike in *F*. The two antidromic spikes could be selectively eliminated by colliding with volleys from LHT and LAHT, respectively (not shown). *H–O*: stimulation of three branches of DR, DRR (*H–M*), ECR (*N*) and EDC (*O*). *M*: stimulation of DRR at 300/sec and a stimulus strength of 4 T. The time scale in msec for *A–E* is indicated in *A*, for *H–P* in *P* and for *F* and *G* below each trace. The cortical stimulus strength (*G*) was 3 mA. The stimulus strengths for antidromic (AD) stimulation in *F* and *P* were 0·1 and 0·05 mA respectively. The stimulus strengths of peripheral stimulation are indicated above each trace in multiples of nerve thresholds. For abbreviations of peripheral nerves, see Methods.

In Fig. 4 the thresholds for the first (*A*), second (*B*), third (*C*), and further (*D*) spikes of discharges evoked in group I activated cells are given. The mean value and standard deviation of the stimulus strength necessary to evoke a just maximal group I afferent volley, calculated from forty-nine cases is indicated above the histogram in Fig. 4 *A*. Only those neurones in which action potentials were discharged at stimulus strengths below 1·5 *T*

were classified as activated by group I afferents. Excitatory effects from the mixed ulnar nerve with thresholds lower than 1·2T were assumed to be evoked by group I afferents (cf. Oscarsson & Uddenberg, 1965).

It cannot be excluded that neurones with thresholds close to 1·5 T were activated from group II afferents instead of group I afferents. The identified group II activated cells in the present investigation differed from the group I activated cells in being regularly activated also from cutaneous afferents at short latencies (mean 3·2 msec, range 0·8–6·8 msec). Only three out of forty-six neurones tested could be activated exclusively from group II afferents. Possibly the cuneate nucleus has no separate channel for information from group II muscle afferents.

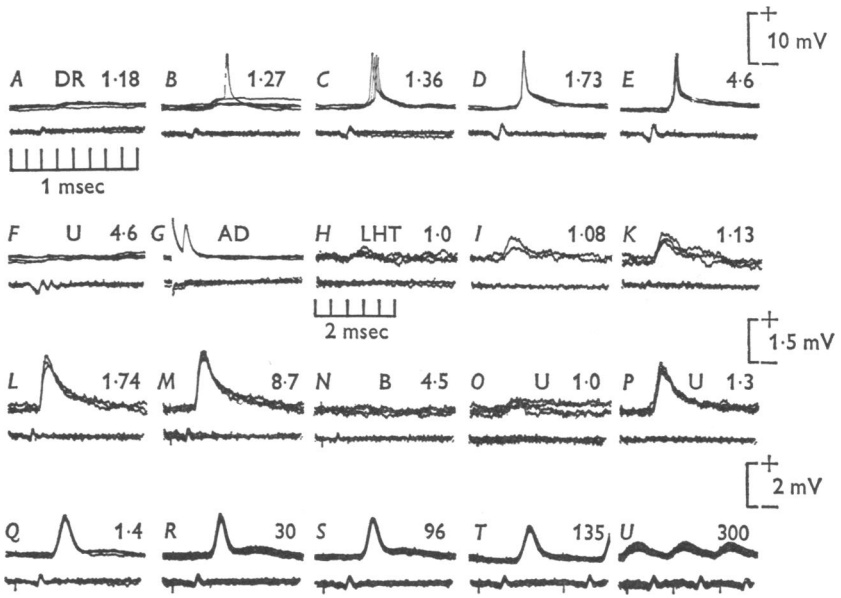

Fig. 3. Intracellular recording from two group I activated neurones, *A–G* and *H–U*. Citrate electrodes. Membrane potentials unknown. Upper traces record intracellular potentials. Lower traces record incoming volleys from the cuneate nucleus. DR (*A–E*), U (*F* and *O–P*), LHT (*H–M*) and B (*N*) were stimulated at strengths indicated in multiples of nerve thresholds. One of the cells could be antidromically stimulated from the contralateral medial lemniscus (*G*). LHT was stimulated in *Q–U* at indicated frequencies (stimuli/sec). The voltage calibration for the microelectrode recording in *A–G* is shown in *E*, for *H–P* in *P* and for *Q–U* in *U*. The time scale in *A* refers to *A–G* and in *H* for *H–U*. The spikes in *B–E* were slightly retouched.

Many of the neurones plotted in Fig. 4*A* had thresholds at the nerve threshold value. From Fig. 4*B* and *C* it can be concluded that many of the second and third spikes of the discharges were evoked by group I afferents. Some spikes had thresholds that indicate convergence from group II muscle afferents. Further comments on this convergence will be given in a later section.

The latencies of 203 group I evoked spike responses, measured from the arrival of the afferent volley, in 136 cells are given in Fig. 1*A*. The number of discharges exceeds the number of cells because in some of the cells group I responses were evoked from more than one nerve. Many of the

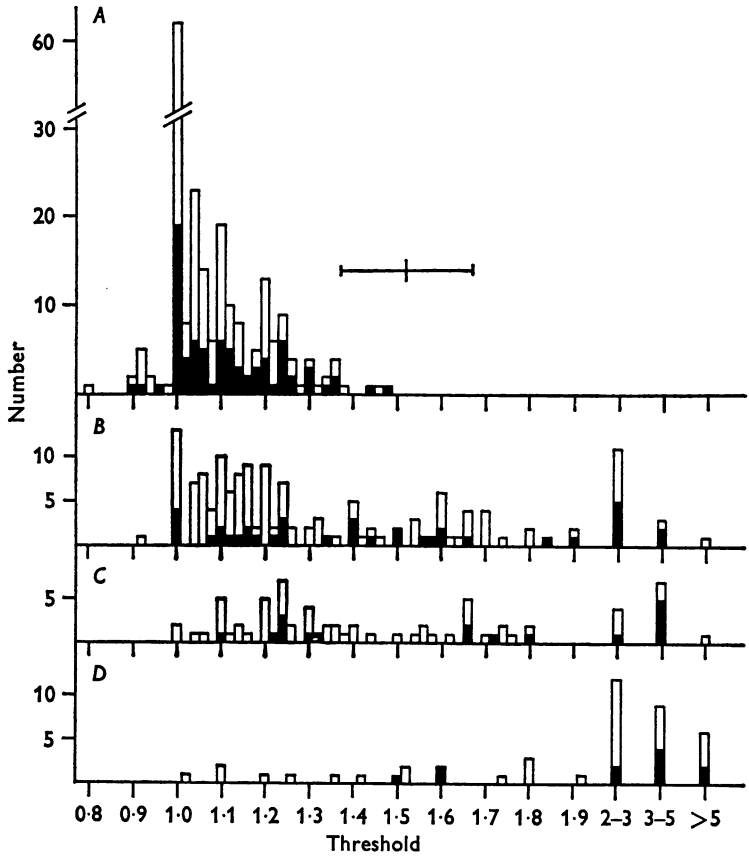

Fig. 4. Histogram showing the thresholds for the first (*A*), second (*B*), third (*C*) and further (*D*) spikes of 215 discharges evoked in 162 group I activated cells by stimulation of muscle nerves supplying group I excitation. The threshold values are expressed as multiples of the nerve thresholds. White and black areas refer to CTR cells and non-CTR cells, respectively. The mean value (1·52 T) and S.D. (± 0·15) of the stimulus strengths necessary to evoke a just maximal group I afferent volley from forty-nine muscle nerves is shown above the histogram in *A*.

latencies imply a monosynaptic connexion with the primary afferent fibres although some of the latencies would allow time for a disynaptic connexion. However, it seems more likely that most of the action potentials with a relatively long latency were evoked late on the slope of a mono-

synaptic EPSP. This is illustrated in Fig. 3*A–E*. The EPSPs in this cell had a latency of 0·95 msec and spikes were evoked 0·4 msec later. The latencies of 23 group I evoked EPSPs measured in sixteen neurones varied from 0·6 msec to 1·1 msec (mean 0·96 msec) indicating a monosynaptic connexion with the group I afferents.

Many of the cells followed high frequencies of group I afferent stimulation for long periods. High frequency stimulation was tested with 101 group I responses recorded in eighty-six group I CTR cells. In fifty-four cases the first spike of the group I response followed frequencies above 200/sec. Figure 2 *M* shows a cell which followed a frequency of 300/sec. In three cells the intracellular recording was stable enough to permit observations on the EPSP amplitudes at different stimulus frequencies. Recordings from one of these tests is illustrated in Fig. 3*Q–U*. The EPSPs were practically constant between 1 and 100/sec and gradually decreased at higher frequencies. A similar behaviour was reported for group I EPSPs in motoneurones (Curtis & Eccles, 1960) and in neurones of the dorsal spinocerebellar tract (Eccles, Oscarsson & Willis, 1961).

The spontaneous activity was studied in seventy-three group I CTR cells. Of these sixty-two had a background activity with frequencies between 2 and 100/sec (mean value 25/sec).

Convergence of excitation to CTR cells. The pattern of group I excitation obtained in sixty-one group I CTR cells in experiments with six forelimb nerves dissected for stimulation is presented in Table 1. The majority (thirty-five) of the cells were activated from one nerve only. No cell was activated from more than three of the six nerves. Many different combinations were found among the cells which showed convergence from different muscle nerves. Co-activation from the antagonistic biceps and triceps muscles was not observed. The convergence from the suprascapular and axillary nerves observed in seven cells does not necessarily imply co-activation from shoulder extensor and flexor muscles because these two nerves also innervate muscles rotating the shoulder outward. The convergence between the deep radial and ulnar nerves observed in six cells might be due to co-activation from wrist dorsiflexors (DR) and palm muscles (U), i.e. from muscle groups acting at different joints. Another possibility might be a co-activation from extensor (DR) and flexor (U) carpi ulnaris which together deviate the wrist to the ulnar side. It can be concluded that co-activation from antagonistic muscles acting at the same joint was not demonstrated with certainty among the group I CTR cells, whereas convergence of excitation from muscles acting at different joints was a relatively common finding. Figure 5 illustrates recordings from a relay cell which was activated by group I afferents in three different nerves (SSC, Ax, LHT). The intracellular recordings of Fig. 3

TABLE 1. Convergence of excitation from group I afferents in nerves to extensor (E) and flexor (F) groups at various joints. Abbreviations of nerves explained in Methods. Number of nerves supplying excitation is given in column at extreme right. Crosses indicate responses, dots lack of responses. Sixty-one group I CTR cells are represented

		Wrist		Elbow		Shoulder		
	No. of cells	F (DR)	E (U)	F (B)	E (LHT)	F (AX)	E (SSC)	Convergence
11	3	+	.	+	.	+	.	3
	2	+	+	+	.	.	.	3
	1	+	+	.	.	.	+	3
	1	+	.	+	.	+	.	3
	1	+	.	+	.	.	+	3
	1	+	.	.	.	+	+	3
	1	.	.	+	.	+	+	3
	1	.	.	.	+	+	+	3
15	4	.	.	.	.	+	+	2
	3	+	+	.	.	.	.	2
	3	+	.	+	.	.	.	2
	3	+	.	.	.	.	+	2
	1	.	+	+	.	.	.	2
	1	.	.	+	.	.	+	2
35	20	+	.	.	.	.	.	1
	9	.	.	.	.	.	+	1
	3	.	.	.	+	.	.	1
	2	.	.	.	.	+	.	1
	1	.	.	+	.	.	.	1

Total 61

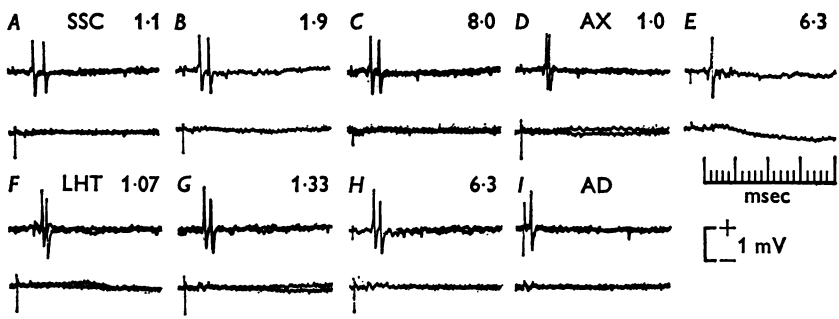

Fig. 5. Extracellular recording from group I CTR neurone showing convergence of excitation from group I afferents in SSC (*A–C*), Ax (*D* and *E*) and LHT (*F–H*). Antidromic spike shown in I. Voltage scale refers to microelectrode recordings (upper traces). Lower traces: potentials recorded from the dorsal surface of the cuneate nucleus. Stimulus strengths in multiples of nerve threshold.

H–P show convergence of excitation from an elbow extensor (LHT) and a wrist or digital ventroflexor (U), whereas afferents from the elbow flexor (B) did not influence the cell (Fig. 3*N*).

The pattern of convergence from adjacent and synergistic muscles is shown in Tables 2*A* and *B*. Two sets of muscle nerves were stimulated in these experiments: the three deep radial branches, ECR, EDC and DRR (cf. Methods) and the nerves to the long and lateral heads of triceps, LHT

TABLE 2*A*. Convergence of excitation from group I afferents in three branches of the deep radial nerve. Number of nerves supplying excitation is given at the right. Crosses indicate responses, dots lack of responses. Twenty-seven group I CTR cells are represented. *B*. Convergence of excitation from the nerves to the long and lateral heads of the triceps. Fourteen group I CTR cells are represented. For abbreviations, see Methods

2*A*

No. of cells	ECR	EDC	DRR	Convergence
1	+	+	+	3
8 (4)	.	+	+	2
8 (3)	+	+	.	2
8 (1)	+	.	+	2
18 (12)	.	.	+	1
18 (4)	.	+	.	1
18 (2)	+	.	.	1

Total 27

2*B*

No. of cells	LHT	LAHT	Convergence
1	+	+	2
13 (12)	+	.	1
13 (1)	.	+	1

Total 14

and LAHT. In only ten of forty-one cells co-activation from adjacent muscles was observed. Figure 2*A–G* illustrates one neurone that is activated from LHT but not from LAHT. (Small spikes from a neighbouring cell activated from LAHT are shown in Fig. 2*E*.) Another cell activated exclusively from one of the DR branches is shown in Fig. 2*H–P*. Co-activation from two of the DR branches is illustrated in Fig. 10*C–E*. It is concluded that the cuneate group I CTR cells show a high degree of spatial specificity also with respect to afferents from adjacent muscles of similar function. This has been confirmed in a recent study (I. Rosén & B. Sjölund, in preparation) using adequate stimulation of muscle receptors.

Excitatory convergence of group II muscle afferents to group I CTR cells was observed in forty-six (30 %) of the 151 neurones tested. In 70 %

of the cases the convergence was seen as an addition of late discharges to those evoked by group I afferents. This phenomenon is illustrated in Fig. 2*K* and *L*. At stimulus strength, maximal for group I afferents two spikes were evoked (*K*), and later spikes were added when also group II afferents were stimulated (*L*). Group III stimulation was tested with seventeen group I CTR cells receiving also group II excitation and was ineffective in all cases. No group III convergence was observed in the fifty-eight group I CTR cells tested.

In 24 % of the group I CTR cells stimulation of the cutaneous (SR) nerve evoked discharges at latencies which were usually much longer than the group I effects. The mean latency, measured from the incoming afferent volley, was 5·7 msec (range 2·0–12·0 msec, $n = 37$). In most of these cases only one spike was produced at varying latency and with low probability of firing. In three experiments (eighteen group I activated cells isolated) the SR nerve was left in continuity with the periphery permitting observations of effects on natural stimulation. No acceleration of the resting activity was found on touch, pressure or pinching the skin. The electrical thresholds for the nerve effects, tested in a few cases, were between 1·25 and 2 T. When tested at different stimulus frequencies the responses usually disappeared at frequencies above 10/sec.

As will be described in a later section group I afferent cuneate terminals were depolarized by single cutaneous volleys. Cutaneous nerve effects in group I CTR cells might possibly be explained by a dorsal root or dorsal funiculus reflex produced in group I primary afferents by cutaneous volleys. Spikes with latencies shorter than about 3·5 msec observed in twelve cells cannot be explained by this mechanism (Andersen *et al.* 1964*c*). Presynaptic depolarization of group I terminals could also be produced by high threshold muscle afferents but only when repetitive stimulation was used. The excitatory effects in group I CTR cells produced by single shocks to high threshold muscle afferents can presumably not be explained by a dorsal root or funiculus reflex.

Properties of group I non-CTR cells. Of the group I activated cuneate cells tested ninety-two (42 %) could not be antidromically invaded from the ventrobasal thalamic complex and were denoted non-CTR cells. Thresholds and latencies did not differ from those of the identified relay cells as shown in Figs. 4 and 1*A*. The patterns of convergence from group I afferents in functionally unrelated muscles (Ax, LHT, B, DR, U) did not differ significantly from that found among the CTR cells. The possibility must be considered that cells denoted non-CTR cells belong to the same group as the identified relay cells and that the stimulus to the medial lemniscus was too localized to stimulate all the fibres terminating in the ventrobasal complex. However, as will be described in a later section a majority (75 %) of the non-CTR cells but only few (7·5 %) of the CTR could be excited by stimulation of the cerebral cortex, suggesting that the

two groups are functionally different. The effect of cortical stimulation was tested in fifty-three cells. Forty of these were activated from the cortex, and differed in some respects from the CTR cells. Fifty-eight % of these cells were excited by volleys in the superficial radial nerve which should be compared with 24 % of the CTR cells. Bursts of spikes were

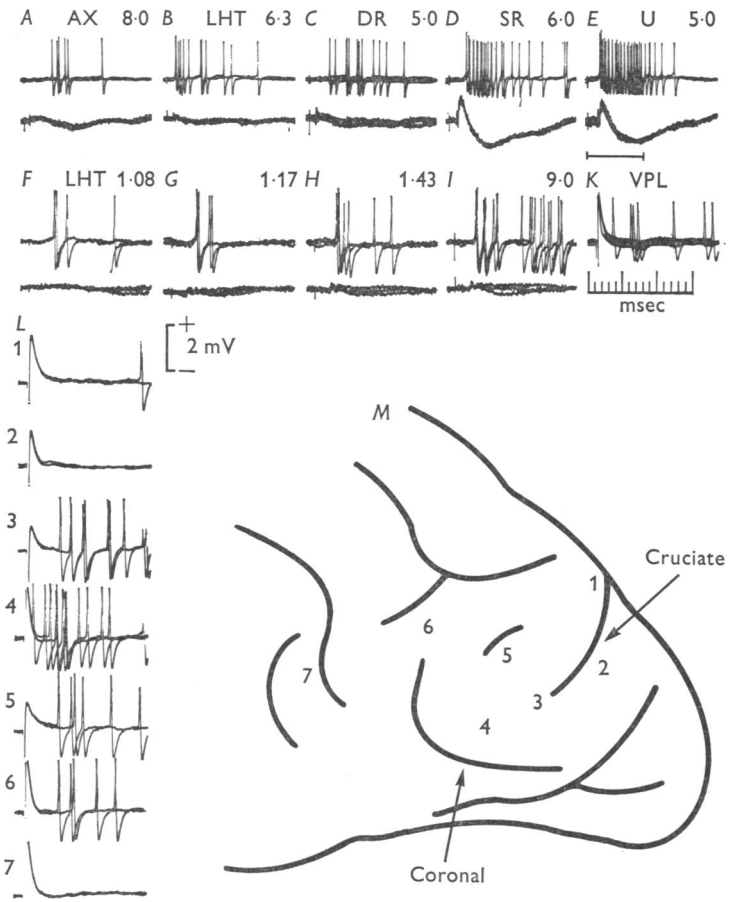

Fig. 6. Extracellular recording from a cortically activated non-CTR cell. Upper traces show micro-electrode recordings (voltage calibration in *L*); lower traces show incoming afferent volleys recorded from the cuneate nucleus. Note different time scales for *A–E* (horizontal bar in *E*, 20 msec) and for *F–L*. The thresholds for spike discharges evoked from the Ax, LHT and DR nerves were 1·8 T, 1·08 T and 2·0 T, respectively. Stimulation of LHT at increasing stimulus strengths is shown in *F–I*. Stimulation of the ventrobasal complex, shown in *K* (stimulus strength 1·0 mA) evoked late spikes at varying latency indicating a synaptic activation. The cerebral cortex was stimulated with needle electrodes in *L*: 1–7 (stimulus strength 3 mA). The cortical points stimulated are shown in *M*. The cruciate and coronal sulci are indicated by arrows.

often produced in these non-CTR neurones on stimulation of the cutaneous nerve (Fig. 6). The mean latency of the responses evoked from the skin nerve was 4·1 msec (range 0·8–7·8 msec, $n = 22$), suggesting a shorter latency than among the CTR cells. Forty-eight % of the cortically excited non-CTR cells were activated from group II muscle afferents. Convergence of excitation from group II afferents in muscle nerves that did not provide any group I excitation to the cell was more frequently found in this group of neurones than among the relay cells. Group III muscles afferents, tested in seven cases, were ineffective. A non-relay cell is illustrated in Fig. 6. This cell was activated from group I and II afferents in the triceps nerve, from group II afferents in the axillary and deep radial nerves, and from afferents in the superficial radial and ulnar nerves.

Inhibition from peripheral nerves. Conditioning volleys in cutaneous nerves regularly produced a depression of the group I evoked responses recorded from the medial lemniscus. The time course of the inhibition obtained in one of the four experiments is illustrated in Fig. 7*C*. Low threshold cutaneous afferents were effective because inhibition was observed at stimulus strengths as low as 1·2 times the nerve threshold. Stimulation of muscle nerves with a brief tetanus (five shocks at 300/sec) and at maximal group II strength produced inhibition which was always less than the inhibition produced by cutaneous volleys. In three of the four experiments short trains of conditioning volleys submaximal for group I afferents produced weak inhibition which increased when also high threshold muscle afferents were stimulated. Fig. 7*D* illustrates the inhibition of a DR group I evoked discharge in the medial lemniscus caused by repetitive stimulation of group I and group II afferents in the LHT nerve. In these experiments group I afferents from shoulder (SSC) and elbow (LHT) muscles were as effective as group I afferents in the ulnar nerve in producing an inhibition of the lemniscal test response evoked from the deep radial nerve.

Fifty-six group I CTR cells were tested for inhibition at conditioning-testing (*C–T*) intervals varying from 4 to 35 msec. In most of the cells only one or two intervals were tested. Conditioning stimulation was performed as described in the preceding paragraph. Fifty-one % of the cells were inhibited from cutaneous afferents and 33 % from high threshold muscle afferents. No clear-cut inhibition from group I afferents was observed in these experiments.

Presynaptic depolarization. Changes of excitability in group I afferent terminals after stimulation of various forelimb nerves was tested in eight experiments by measuring the amplitude of the antidromic volleys in the DR, LHT and B nerves after local stimulation in the deep part of the cuneate nucleus (Wall, 1958; Andersen *et al.* 1964*c*). The early antidromic

volleys recorded from the muscle nerves after such a stimulation could be completely eliminated by collision with just maximal group I volleys. No separation between antidromic Ia and Ib volleys could be seen. Results obtained in two different experiments are illustrated in Fig. 7*A* and *B*. Single conditioning cutaneous volleys caused a considerable excitability increase with a maximum at about 15 msec and a duration of at least

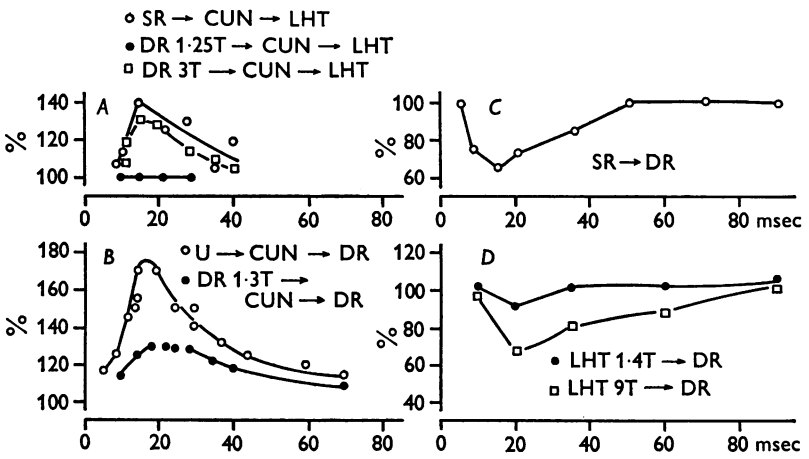

Fig. 7. Time course of excitability increase of group I terminals (*A* and *B*) in cuneate nucleus and of inhibition of the DR group I response in the medial lemniscus (*C* and *D*) produced by electrical stimulation of peripheral nerves. Results from four different experiments are presented. Repetitive conditioning stimuli (five shocks at 300/sec) were given to the muscle nerves at strengths indicated in multiples of nerve thresholds (T). The SR and U nerves were stimulated with single shocks at about 3 T. The values in *A* and *B* were obtained by measuring the amplitudes of the antidromic volleys recorded from the peripheral nerves (LHT in *A*, DR in *B*) after local stimulation in the ventral part of the cuneate nucleus. The amplitudes of the lemniscal responses in *C* and *D* were measured after computer averaging of sixty sweeps.

100 msec. A smaller excitability increase was sometimes observed after brief tetanic stimulation of group I afferents (Fig. 7*B*). Afferents from extensors were as effective as those from flexor muscles. When the conditioning stimulus strength was increased to stimulate also group II afferents the excitability was further increased in fourteen of thirty-two cases. In eight cases no further increase was produced and in eight cases high threshold muscle afferents were effective when group I afferents were ineffective (Fig. 7*A*). The time course of the excitability change produced by muscle afferents was usually slower than that produced from cutaneous afferents (Fig. 7*B*).

Micro-electrode recordings have been performed intra-axonally from three group I afferent terminals in the cuneate nucleus. Two of these were depolarized by trains of group I afferent volleys at latencies, measured from the first shock artifact, of 8 and 9 msec respectively and with a maximum at about 20 msec in both cases. Cutaneous volleys produced depolarization of all three terminals at the latencies of 4, 4·5 and 6 msec respectively and with maximum at 12, 13 and 17 msec.

Excitatory and inhibitory control from the cerebral cortex

Cortical excitation of group I CTR cells. Cortical stimulation by a set of eight needle electrodes inserted as indicated in Fig. 8 was tried when recording from sixty-seven group I CTR cells. Five of these cells (7·5 %) could be activated. The most effective part of the cerebral cortex was the lateral part of the post-cruciate gyrus between the coronal sulcus and the lateral part of the cruciate sulcus. In one of the cells the SII area was also effective. The latencies of the cortically evoked discharges were between 4·0 and 6·0 msec.

Among the cutaneous CTR cells encountered in the same experiments three cells out of seventy-four were excited from the cerebral cortex. No such excitation was found among the CTR cells described by Andersen *et al.* (1964*d*) in a similar investigation. In the gracile nucleus (Gordon & Jukes, 1964) only one of twenty-one cortically excited neurones was antidromically activated from the main body of the medial lemniscus. A considerably higher proportion (50 %) of trigeminothalamic relay cells was found to be excited from the cerebral cortex on chloralose animals (Darian-Smith & Yokota, 1966) as compared with the findings in the dorsal funiculus nuclei, which were all obtained under barbiturate anaesthesia.

Cortical excitation of group I non-CTR cells. Cortical stimulation evoked discharges in forty of the fifty-three group I non-CTR cells tested (75 %). The mean latency of the discharges was 6·5 msec (range 2·1–18 msec). Responses from a cortically excited non-relay cell are illustrated in Fig. 6. This cell could be excited from the cortical forelimb sensory and motor cortices (points 3–6) with an optimal point in the most lateral part of the post-cruciate gyrus (point 4). The distribution of the most effective cortical stimulation points for thirty-three of the cells is presented in Fig. 8. In six of the cells, two or three of the eight stimulating points were equally effective. It can be concluded that the cortical excitation of cuneate group I neurones originates in the forelimb area. The forelimb motor area rostrolateral to the post-cruciate dimple (Livingston & Phillips, 1957; Hassler & Muhs-Clement, 1964; Asanuma & Sakata, 1967) was more effective than the sensory area. It is obvious that the effective cortical area is not confined to the small cortical projection zone for group I afferents that is situated immediately rostral to the post-cruciate dimple.

Cortical inhibition of group I CTR cells. The group I response recorded

from the medial lemniscus could be depressed by cortical stimulation. This was demonstrated in five experiments using bipolar cortical stimulation in order to reduce the shock artifacts (inter-electrode distance 1 mm) and computer averaging of the lemniscal responses. Results from two of the experiments are illustrated in Fig. 9. The cortical focus for the inhibition was regularly found around the lateral end of the cruciate sulcus. Stimulation of the SII area produced an inhibition in two of the experiments

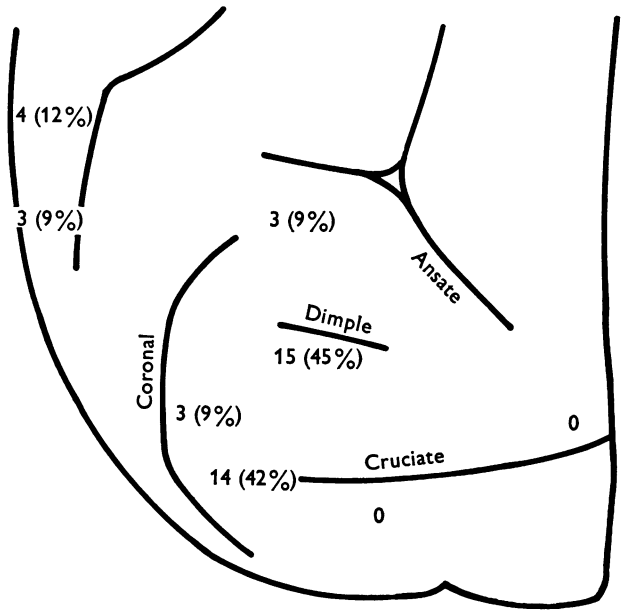

Fig. 8. Distribution of most effective points of stimulation for cortical excitation of thirty-three group I non-CTR cells tested with stimulating electrodes at eight fixed positions. The sum of the best points exceeds the number of cells because with six neurones, two or three of the stimulated points were equally effective. The ansate, coronal and cruciate sulci and the post-cruciate dimple are indicated.

although it was smaller than that evoked from the pericruciate area. The electrical threshold for the inhibition using stimulation with five shocks of 0·2 msec duration at 300/sec was about 0·4 mA. The time course of the inhibition illustrated in Fig. 9*F* was very similar to the time course of the cortical inhibition of cutaneous CTR cells, described by Andersen *et al.* (1964*a*) and confirmed in this investigation. In three of the experiments the inhibition was preceded by a facilitation (20–40 %) with a maximum at conditioning-testing (*C–T*) intervals of about 20 msec. The cortical best points for the facilitation obtained in these experiments were found

close to the coronal sulcus, slightly lateral and caudal to the best points of inhibition.

Cortical inhibition was also tested in forty-four group I CTR cells using brief repetitive stimulation of the cortex and *C–T* intervals between 12 and 100 msec. In twenty of the cells (45 %) the probability of firing was

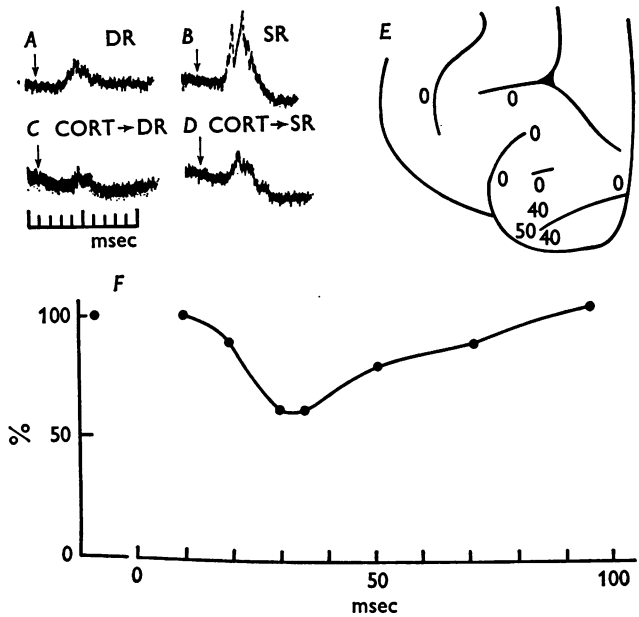

Fig. 9. Cortical inhibition of group I and cutaneous responses recorded from the medial lemniscus. *A* and *B* show unconditioned responses evoked by group I DR and cutaneous SR volleys, respectively. *C* and *D* show the responses after conditioning with brief repetitive cortical stimulation (C–T interval 50 msec; cortical stimulation with bipolar surface electrode, five shocks of 0·9 mA at 300/sec). The responses shown in *A–D* and the values in *E* refer to records obtained by averaging of forty-five sweeps. The per cent depression of the DR group I response obtained in the same experiment by stimulation of various cortical spots is given in *E*. The curve in *F* shows the time course of the cortical inhibition of the lemniscal DR group I response obtained in another experiment. Each point represents averaging of sixty sweeps. The cortical stimulation was the same as in *A–E*. Ordinate: per cent of unconditioned test response. Abscissa: conditioning-testing interval in msec.

decreased. The variability of the inhibitory effects is illustrated in Fig. 10 in which one of the two simultaneously recorded group I CTR cells was inhibited and the other cell was uninfluenced by cortical stimulation.

Cortically evoked presynaptic depolarization of group I afferents. An excitability increase of the group I afferent terminals in the cuneate nucleus was demonstrated in seven experiments out of nine. The time

course was very similar to that described above for the cortical inhibition of group I CTR cells. The maximum excitability was reached at *C–T* intervals of about 35 msec and the duration was at least 100 msec. The optimal cortical point for this effect, localized in four experiments, lay close to the lateral end of the cruciate sulcus in two experiments. In two experiments the area between the post-cruciate dimple and the cruciate

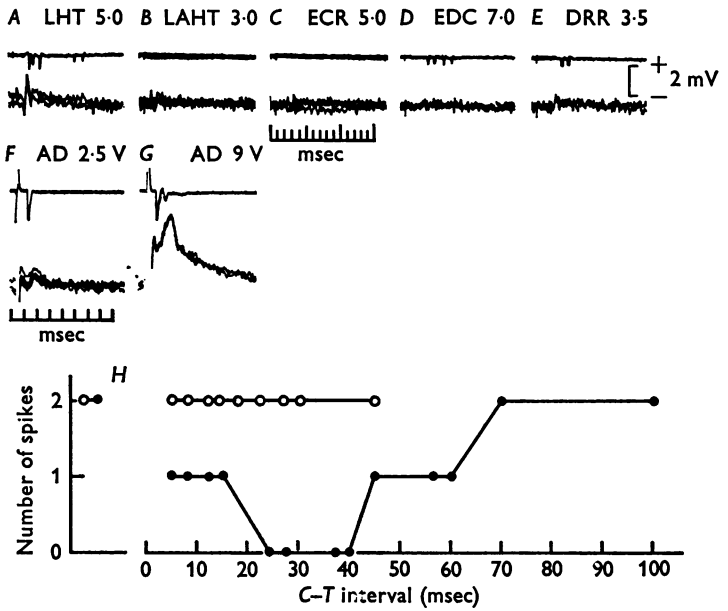

Fig. 10*A–G*. Simultaneous recordings from two group I CTR cells of which one was activated from LHT (*A*) and the other from EDC and DRR (*D*, *E*). One of the cells was antidromically activated at low stimulus strength (*F*). At higher strength both cells were invaded (*G*). The recording conditions for the first of these cells were improved in *F* and *G*. Voltage calibration refers to micro-electrode recordings (upper traces). Lower traces are potentials recorded from dorsal surface of cuneate nucleus. Time course of inhibition evoked by cortical bipolar stimulation (five shocks at 0·9 mA) close to the cruciate sulcus. The average number of spikes per sweep was calculated from five individual sweeps at each conditioning-testing (C–T) interval. In *H*, open circles, cell activated from LHT; filled circles, cell activated from EDC, DRR.

sulcus was equally effective. The forelimb somatosensory and hind limb sensorimotor cortical areas were ineffective. Stimulation of the SII area produced an excitability increase in one of three experiments.

It is concluded that there is a good correspondence between the cortical inhibitory effects on group I CTR cells and the presynaptic depolarization observed in the group I afferent terminals both in terms of the time course and effective cortical area.

Depth distribution of cuneate cells

In a previous study (Rosén, 1969*a*) it has been shown that group I afferent volleys evoke field potentials in the deep part of the cuneate nucleus. This has been confirmed in the present investigation in which the depth values of the isolated cells were obtained from the micromanipulator readings. The distribution of group I and cutaneous CTR as well as non-CTR cells are given in Fig. 11*A–D*. Cortically activated cells and cutaneous cells with convergence from group II muscle afferents are marked by

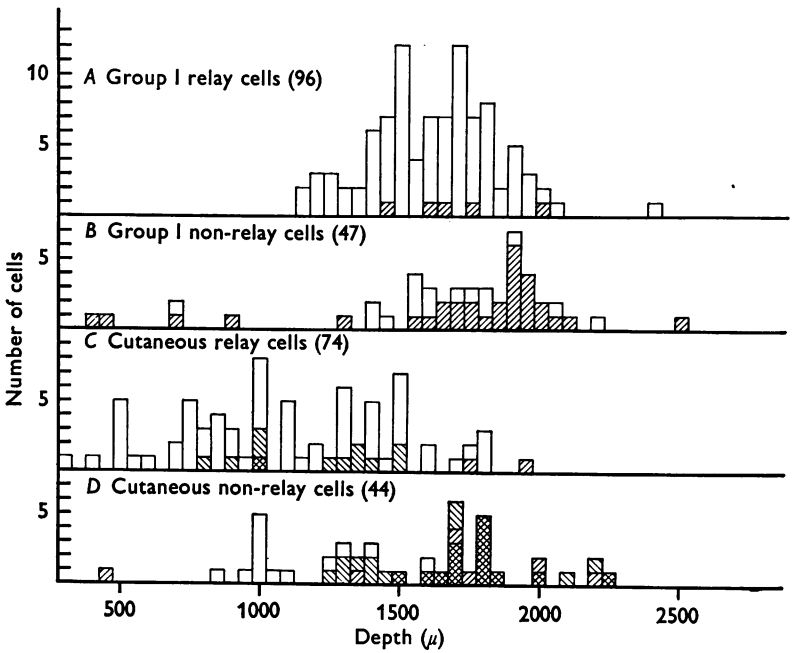

Fig. 11. Depth distribution of cells encountered in the main cuneate nucleus 0–3 mm caudal to the obex. Cortically excited cells (▨) and cutaneous cells (*C*, *D*, ▧) with excitatory convergence from high threshold muscle afferents are indicated by hatching.

different hatching as indicated in the Figure. Most of the group I activated cells, relay as well as non-relay cells, were encountered in the deep part of the nucleus although a few of the non-relay cells occurred more superficially. There was a slight tendency for non-relay cells to occur more deeply in the nucleus than the relay cells. The cutaneous relay cells had a more dorsal distribution but most of the cutaneous non-relay cells were encountered in the deep part of the nucleus together with group I activated cells. It should be noted that only three of seventy-four cutaneous CTR cells were cortically activated whereas twenty of the

forty-four cutaneous non-CTR cells were excited by cortical stimulation. This difference is in accord with previous observations on cuneate cells activated from cutaneous afferents (Andersen *et al.* 1964*d*). In the present investigation it has been demonstrated that many of the cutaneous non-relay cells also receive excitatory convergence from group II muscle afferents.

Group I activated neurones in the cervical dorsal horn. A group I activated zone in the base of the dorsal horn of the rostral cervical cord was described in a previous paper (Rosén, 1969*a*). Field potentials were recorded in this region after stimulation of group I afferents from distal muscle groups whereas afferents from elbow and shoulder muscles were almost completely ineffective. Cutaneous afferents also produced focal potentials in the same region. Recordings have now been made from eleven neurones,

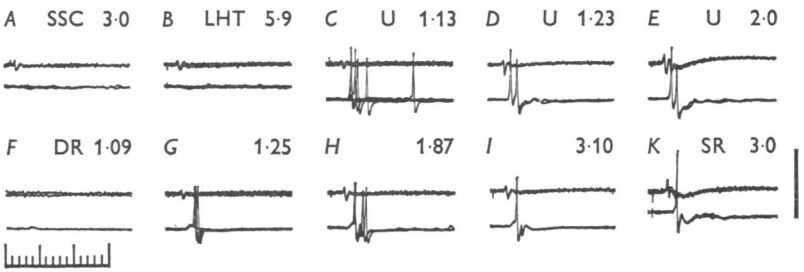

Fig. 12. Close contact recording from a group I activated cell in the base of the dorsal horn at C 3. The position of the micro-electrode tip was identified with fast green staining (illustrated in Rosén, 1969*a*, Fig. 10). Voltage calibration for micro-electrode recording (lower traces; positivity upwards) 10 mV. Time scale in msec. The incoming afferent volleys (upper traces) were recorded from the dorsal funiculus at C 3. Time scale in msec. Various forelimb nerves (abbreviations, see Methods) were stimulated at strengths indicated in multiples of nerve threshold.

monosynaptically activated from group I afferents in forelimb muscle nerves and encountered from C 2 to C 4. Recordings from one of these cells are illustrated in Fig. 12. The pattern of convergence among these cells was characteristically different from that described for group I activated cuneate neurones. Afferents from muscles acting at the shoulder (SSC, Ax) or elbow (LHT, B) joints were ineffective except in two cells which were activated from the suprascapular nerve and the triceps nerve, respectively. Group I afferents in the deep radial nerve evoked discharges in ten cells and in five of these cells group I ulnar afferents were also effective. All the cells were also activated by cutaneous volleys and in seven cases the latency was too short to allow more than one synapse. The cell in Fig. 12 was monosynaptically activated from the deep radial (group I), the ulnar and the superficial radial nerves. Presumably the effect from the ulnar nerve was evoked from group I afferents because of its low threshold and lack of additional activation when group II (cutaneous and muscle) afferents were stimulated (Fig. 12*E*).

DISCUSSION

Peripheral effects

Excitation. The cuneate neurones studied in this investigation were monosynaptically activated from group I afferents and often responded with repetitive spike discharges to afferent volleys. A similar behaviour was reported for group I activated neurones in the dorsal spinocerebellar tract, DSCT (Laporte, Lundberg & Oscarsson, 1956), and the cuneocerebellar tract (Holmqvist, Oscarsson & Rosén, 1963). Possible mechanisms for the repetitive discharge in DSCT neurones has recently been discussed (Kuno & Miyahara, 1968; Eide, Fedina, Jansen, Lundberg & Vyklicky, 1969).

A majority of the group I CTR neurones were activated from afferents in a single muscle or occasionally a few adjacent muscles. The information signalled at the cuneate level of the group I afferent pathway to the cerebral cortex is thus spatially highly specific. Some of the neurones received a co-activation from functionally more unrelated muscle groups but convergence from pure antagonistic muscles was not demonstrated. The pattern encountered in the cuneate nucleus is very similar to that found in the dorsal spinocerebellar tract (Holmqvist, Lundberg & Oscarsson, 1956; Laporte *et al.* 1956; Eccles *et al.* 1961; Oscarsson, 1965). The forelimb equivalent of the DSCT, the cuneocerebellar tract (CCT) has been analysed in less detail but appears to be similarly organized (Holmqvist *et al.* 1963).

The ventral spinocerebellar tract (VSCT) and its forelimb equivalent, the rostral spinocerebellar tract (RSCT), both activated from tendon organ afferents (Oscarsson, 1965), have a more extensive convergence to individual neurones than the group I cuneothalamic pathway. It has been suggested that these pathways carry information concerning stages of movements or position of the whole limb rather than information about tension variations in individual muscles (Oscarsson, 1965). A similar function might be suggested for the group I CTR neurones with wide convergence. Another possibility would be that these effects represent aberrations in the synaptic connexions. In most of the neurones showing convergence one of the effective nerves had a more powerful synaptic effect than the others.

The DSCT contains separate groups of neurones activated from Ia and Ib afferents, respectively (Lundberg & Oscarsson, 1956; Lundberg & Winsbury, 1960; Eccles *et al.* 1961; Oscarsson, 1965). It was not possible with electrical stimulation of forelimb muscle nerves to separate Ia and Ib components. It is known that the group I afferent path to the cerebral cortex carries information from muscle spindles (Oscarsson & Rosén, 1963).

Experiments with natural stimulation of muscle receptors are in progress to find out if the cuneate nuclei in addition relay information from tendon organs.

Some of the group I CTR cells were activated from group II muscle afferents in the same nerve that contributed group I excitation. Excitation from group II afferents in other muscle nerves was very uncommon and excitation from group III muscle afferents was not observed. Excitation from cutaneous afferents was either weak or lacking. This is reminiscent of the pattern of convergence found in many DSCT cells (Laporte *et al.* 1956; Lundberg & Oscarsson, 1956; Eccles *et al.* 1961). The excitation from group I and II muscle afferents in these two pathways might represent a highly specific convergence from the two types of spindle afferents in the same muscle. In all other known pathways group II muscle afferents converge to produce synaptic effects together with the other components of the flexor reflex afferents (group III muscle, cutaneous, and high threshold joints afferents) (cf. Eccles & Lundberg, 1959; Lundberg, 1964; Oscarsson, 1967, and Discussion in the following paper).

Inhibition. The transmission in the cuneate group I relay was weakly inhibited by conditioning group I afferent volleys. A stronger inhibition was produced by high threshold muscle afferents and by cutaneous afferents (flexor reflex afferents). The inhibition from the peripheral nerves might be mainly presynaptic as depolarization of group I afferent terminals was observed to be produced by group I afferents in flexors and extensors, high threshold muscle afferents and cutaneous afferents. Of these three groups of afferents the cutaneous fibres were by far the most effective. This means that the presynaptic inhibitory pattern in the cuneate group I relay does not show the type of modality specificity that has been described for primary afferent terminals at the segmental level of the spinal cord (Eccles, Schmidt & Willis, 1963*a*, *c*; Jänig, Schmidt & Zimmermann, 1968). It is, however, reminiscent of the pattern of presynaptic inhibition of the group I relay of the DSCT (Eccles, Schmidt & Willis, 1963*b*; Jankowska, Jukes & Lund, 1965) in which the group I terminals are depolarized by flexor reflex afferents and group Ib afferents but not by group Ia afferents. The lack of Ia–Ib separation of volleys from forelimb muscle nerves made it impossible to decide if the group I evoked presynaptic depolarization of group I afferents in the cuneate nucleus also was exerted exclusively from tendon organ afferents.

Unfortunately it has not been possible in the present investigation to get enough intracellular recordings to discuss the occurrence of a postsynaptic inhibition in the group I relay cells, although IPSPs seemed to be rare in these cells as compared with the cutaneous relay cells. Stable intracellular recordings were obtained from three cells activated from LHT

group I afferents. In these cells stimulation of the antagonistic muscle nerve (B) did not produce any post-synaptic effects, neither inhibitory nor excitatory. A reciprocal type of post-synaptic inhibition corresponding to that described among spinal motoneurones (Lloyd, 1946*a*, *b*; Eccles & Lundberg, 1958) does not seem to occur regularly among the group I CTR cells.

Cortical control

The observations on effects in group I activated neurones evoked by stimulation of the cerebral cortex are similar to corresponding observations on cutaneous cells in the dorsal funiculus nuclei (Andersen *et al.* 1964*d*; Gordon & Jukes, 1964). With both groups of cells it has been found that neurones projecting into the medial lemniscus receive predominantly inhibition, whereas non-relay cells receive predominantly excitation from the cortex.

Relay cells. The inhibition of the group I relay cells was most effectively evoked from a cortical region at the lateral end of the cruciate sulcus, corresponding to the forelimb motor cortex (Livingston & Phillips, 1957; Hassler & Muhs-Clement, 1964; Asanuma & Sakata, 1967). Approximately the same area was reported by Levitt *et al.* (1964) to be the focus for inhibition of cutaneous cells in the cuneate nucleus. It is clearly separated from the main projection areas of the group I pathway and also from the caudal projection area for cutaneous forelimb afferents, situated caudal to the post-cruciate dimple (Oscarsson & Rosén, 1966). It is therefore not likely that the inhibitory path from the cerebral cortex to the cuneate group I relay represents a negative feed-back circuit through the primary group I projection area (cf. Andersen *et al.* 1964*a*). Possibly this inhibitory path is responsible for the phasic inhibition of both the cutaneous and group I relays in the cuneate nucleus during bursts of rapid eye movements (REM) in desynchronized sleep (Carli *et al.* 1967*a*, *b*). Ablation of the sensorimotor cortices abolished this inhibition without affecting the REM bursts.

A good agreement was found in the present investigation for the cortically evoked inhibition of the group I CTR cells and the presynaptic depolarization of the group I terminals in the cuneate nucleus. This does not exclude the possibility of a post-synaptic inhibition. Cortically evoked presynaptic as well as post-synaptic inhibition has been demonstrated among cutaneous relay cells in the cuneate nucleus (Andersen *et al.* 1964*a*, *d*) and among group I activated DSCT neurones (Hongo & Okada, 1967). A cortical control of transmission has also been demonstrated for the VSCT, which receives inhibition and facilitation from separate cortical areas (Magni & Oscarsson, 1961).

Non-relay cells. Cortical best points for excitation of non-relay group I cells were found within the region producing inhibition of the relay cells but also more caudally, including the group I projection area rostral to the post-cruciate dimple. The total excitatory region corresponds approximately to that described by Levitt *et al.* (1964) as most effective in evoking excitation of cutaneous cells. It is interesting that the group I projection area is situated in the cytoarchitectonic area 3a (cf. Hassler & Muhs-Clement, 1964), from which neurones were reported to project directly to the dorsal column nuclei (Gordon & Miller, 1969).

Cutaneous non-relay cells in the cuneate nucleus were usually excited by cortical stimulation (Andersen *et al.* 1964*d*, and present investigation). Andersen *et al.* (1964*a*, *d*) suggested these cells to be interneurones on the presynaptic and post-synaptic inhibitory pathways to the relay cells. Group I non-relay cells encountered in the present investigation might have a similar function. Many of these cells showed a convergence of excitation from cutaneous and group II muscle afferents and the cerebral cortex, which is in accord with their possible role as interneurones in the inhibitory pathways described in this paper. The separation between cutaneous and group I non-CTR cells is presumably not distinct. Neurones were encountered which received a strong excitation from the flexor reflex afferents and which were also weakly excited by trains of group I volleys. The partial discrepancy found between the cortical areas supplying excitation to non-relay cells and inhibition to relay cells, although tested with different cortical stimulation technique, may suggest that at least some of the non-CTR cells have a different function. Some of these cells may have ascending axons reaching other structures than the ventrobasal thalamic complex.

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