J. Physiol. (1967), **189**, *pp.* 101–117
With 6 text-figures
Printed in Great Britain

SHORT LATENCY ACTIVATION OF PYRAMIDAL TRACT CELLS BY GROUP I AFFERENT VOLLEYS IN THE CAT

By JOHN E. SWETT,* and CHARLES M. BOURASSA

From the Laboratory of Neurophysiology Good Samaritan Hospital, Portland, Oregon, U.S.A.

(*Received* 25 *July* 1966)

SUMMARY

1. The contralateral bulbar pyramids were explored with low impedance micro-electrodes in cats anaesthetized with chloralose to reveal the effect of Group I afferent volleys (deep radial nerve of the forelimb) on pyramidal tract (Pt) cells.

2. Low rate (0·5/sec) stimulation of Group I afferents produced small responses (5–30 μV) in the bulbar pyramid which could be detected only with response averaging methods. The responses appeared with an initial latency of 7·0–11·2 msec and reached peak amplitude in 15·7 msec (mean latency). The pyramidal tract origin of the potential was demonstrated by its depression at stimulus rates above 1–2 sec and its disappearance at rates above 4/sec.

3. Recordings of neurones in the Group I cortical projection zone of the posterior sigmoid gyrus revealed that several types of cells, including Pt cells, were activated by Group I afferent volleys.

4. Pt cells responding to Group I afferent volleys frequently received convergent actions from low threshold cutaneous nerve volleys.

5. Averaged response recordings from electrodes positioned in the medial portions of the lateral funiculus of the spinal cord at the level of C_2, revealed a response to Group I afferent volleys as early as 7·4 msec which possessed the same characteristics as the relayed response to Group I in the bulbar pyramids. Some Pt cells, activated by Group I volleys orthodromically, could also be antidromically activated by stimulation of the recording site in C_2.

6. It was concluded that group I afferent volleys can influence, after short latencies, Pt and non-Pt cells and that some of these Pt cells gave rise to axons incorporated in the corticospinal tract.

* Present address: Department of Anatomy Upstate Medical Center, State University of New York, Syracuse, New York, U.S.A.

INTRODUCTION

Pyramidal tract (Pt) cells in the pericruciate cortex of the cat are capable of being influenced from a number of different sensory modalities and exhibit peripheral receptive fields of varying sizes (Adrian & Moruzzi, 1939; Buser & Ascher, 1960; Kennedy, Towe & Patton, 1960; Brooks, Rudomin & Slayman, 1961; Patton, Towe & Kennedy, 1961).

A Group I afferent volley from the deep radial nerve evokes a cortical response in a small region of the contralateral posterior sigmoid gyrus situated along the anterolateral margin of the postcruciate dimple (Amassian & Berlin, 1958; Oscarsson & Rosen, 1963, 1966; Swett & Bourassa, 1965, 1966; Bourassa & Swett, 1966). There is a smaller area in S II which is also affected by Group I volleys (Landgren & Wolsk, 1966; Andersson, Landgren & Wolsk, 1966). The neurone populations within or immediately adjacent to the post-cruciate dimple exhibit characteristics that are intermediate between those of the motor cortex anterior to the dimple and S I posterior to the dimple (Kennedy & Towe, 1962; Oscarsson & Rosen, 1963; Morse & Towe, 1964). On the basis of cytoarchitectonic studies, the Group I cortical projection zone can be considered to lie on the posterior boundary of the motor cortex (Campbell, 1905; Hassler & Muhs-Clement, 1964).

Data presented in this report show that some Pt cells receive powerful facilitatory synaptic actions in response to Group I afferent volleys and that a proportion of these cells give rise to axons which project into the spinal cord. Some of these findings have been reported in brief (Swett & Bourassa, 1966).

METHODS

These observations are based on data from thirty-five cats. Twenty-two of the animals were anaesthetized with sodium pentobarbital (Abbott, 35 mg/kg I.P.) and maintained at a level just adequate to block segmental reflex contractions.

The remaining thirteen animals were anaesthetized with chloralose (K & K Laboratories, 60 mg/kg I.P.). Sodium brevital (Lilly, 15 mg/kg I.V.) was used in conjunction with chloralose throughout the initial surgical stages but was discontinued 30–60 min before recording. To prevent reflexly induced or spontaneous movements, flaxedil (Davis & Geck, 20 mg/kg I.V.) was administered and artificial respiration was instituted.

Stimulation and recording. The superficial and deep radial nerves were the only somatic nerves used for testing. Stimulus intensities were controlled through a specially designed system capable of giving a direct reading of intensity in values relative to peripheral nerve or evoked response thresholds (Mills & Swett, 1965). Threshold is designated as 1·00*T*.

An intensity of 2·5*T* was usually required to maximally activate Group I afferent components (Ia and Ib) in the deep radial nerve. Threshold for Group II muscle afferent fibres occurred between 1·7*T* and 2·0*T*. In order to insure delivery of Group I volleys, uncontaminated by group II components, stimulus intensities for the deep radial nerve were maintained at 1·7*T* or below. No distinction will be made between Ia and Ib unless specified. The term Group I is used to indicate that both afferent types were stimulated in most

instances (Eccles & Lundberg, 1959; Eccles, Oscarsson & Willis, 1961; Magni & Oscarsson, 1962; Pompeiano & Swett, 1962).

Voltages required to reach threshold activation of the peripheral nerves ranged between 0·08 and 0·12 V. Rectangular pulses of 100 μsec duration were used exclusively and the proximal lead of the stimulating electrode was always the cathode. The deep radial nerve was always ligated about 10 mm distal to the stimulating electrode to prevent muscle contractions.

A sharpened stainless-steel electrode, 20–40 μ tip diameter, insulated to the tip, was placed under direct visual control into the exposed ventral surface of the bulbar pyramid contralateral to peripheral nerve stimulus sites. This electrode was used for monopolar recording or stimulation. The reference electrode was imbedded in a pad of saline soaked cotton immediately adjacent to the exposed pyramids. Rectangular cathodal pulses (50–200 μsec duration) were used for stimulating this tract.

Recording electrodes were positioned in the spinal cord between C_1 and C_2 on the side ipsilateral to the stimulated peripheral nerves. Electrodes were also positioned stereotaxically in the Group I relay nucleus of the ventrobasal complex (co-ordinates A 11, L 6, H+2) (Jasper & Ajmone-Marsan, 1955; Mallart, 1964; Andersson *et al.* 1966; J. E. Swett & C. M. Bourassa, personal observations)

Single unit recordings from the cerebral cortex were obtained with glass micro-electrodes filled with 3 M-KCL or 2 M-K-citrate (impedances 2·0–30 MΩ). For recording evoked responses or laminar field potentials in the cortex, low impedance (1–6 MΩ) electrodes (4 M-NaCl) were used. All unit recordings with high impedance electrodes were obtained in the conventional manner with the aid of a negative capacity electrometer (BAK) and DC coupled amplification.

Cortical pulsation was minimized by cisternal drainage, suspension of the animal by vertebral clamps, and application of 3 % agar in a solution of 8·5 % sucrose over the exposed cortex. The agar could be penetrated without causing damage to the electrode tips. Occasionally, a pressure plate, imbedded in the agar was also used to control cortical pulsations. This technique permitted visual control of the pial vascular bed.

Pyramidal tract potentials evoked by Group I volleys are usually too small to observe with standard recording methods (Fig. 3*c*). They were revealed by using an Enhancetron 1024, a response averaging system (Nuclear Data, Inc.). At least 127 sweeps and a sweep time of 63 msec were the customary parameters for accumulation of each averaged response. The amplitudes of the averaged responses were calibrated by feeding a 10 μV calibration pulse into the inputs of the preamplifier after a repeatable delay and averaging the response for the same number of sweeps used in accumulating the biological potential. Except for cortical recording sites all electrode placements in the spinal cord, brain stem and thalamus were verified by histology methods.

Identification of cortical neurone. Cortical units which responded in all-or-none fashion with unvarying latencies of 2·0 msec or less, following near threshold stimulation of the bulbar pyramids, and which faithfully responded to stimulus rates of 200/sec or more, were considered to be Pt cells although it is recognized that Pt cells can have much slower conduction rates (Kennedy & Towe, 1962). Units not meeting these criteria were classified as non-Pt cells. Cells not influenced from the pyramid or the ventrobasal complex and those cells not subjected to sufficiently detailed tests were classified as unidentified units.

RESULTS

Cortical evoked responses. Recordings from the region anterior to the post-cruciate dimple reveal a response to low threshold stimulation of the deep radial nerve (Amassian & Berlin, 1958; Oscarsson & Rosen, 1963; Bourassa & Swett, 1966). In agreement with Oscarsson & Rosen (1963)

this response differs from the cortical primary evoked by a cutaneous volley from the superficial radial nerve. The principle differences are illustrated in surface records in Fig. 1*a* and *b*. The response to a Group I volley at 1·7*T* (*a*) normally exhibits a positive phase of longer duration than that produced by a cutaneous volley (*b*) observed from the same recording site. The Group I response also usually lacks the prominent late surface negative phase typical of the cutaneous response.

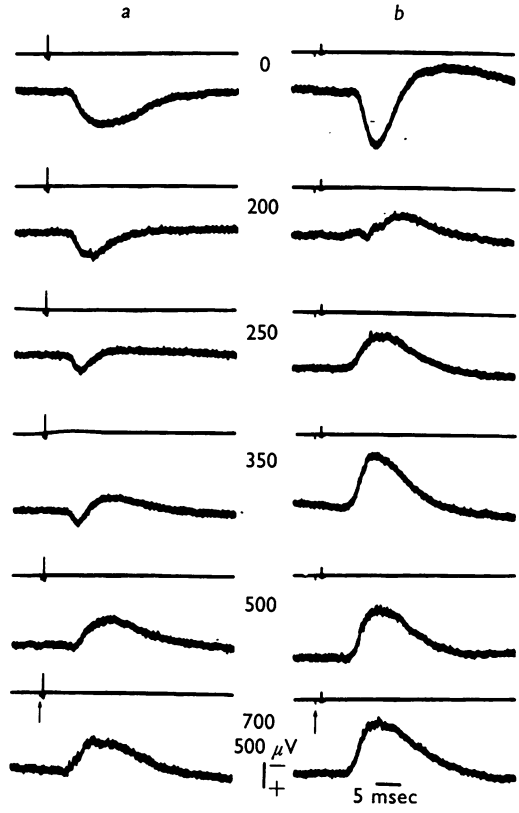

Fig. 1. Comparison of responses evoked by Group I volleys (*a*) and cutaneous volleys (*b*) recorded with the same electrode at indicated depths (micrometer readings) within the contralateral Group I projection zone of the cerebral cortex. Upper traces in *a* show the Group I afferent volley (1·7*T*) recorded from the brachial plexus. Arrows mark shock artifacts. Chloralose anaesthesia.

The Group I cortical primary has a shorter latency than the cutaneous response. Out of eleven subjects, the earliest suggestion of arrival of relayed Group I actions in the cerebral cortex occurred between 5·0 and 7·0 msec (mean 5·9 msec), whereas responses to cutaneous shocks in the Group I receiving area occurred between 6·2 and 10·0 msec (7·9 msec). The latency

difference can be largely explained by the relative conduction rates of the two primary afferent fibre groups between the site of stimulation and the relay with second order neurones of the dorsal column nuclei. The Group I volley produces a response in the juxtaolivary portion of the lemniscus in 2·5–3·0 msec, whereas the cutaneous volleys appear in 4·0–5·0 msec (see also Oscarsson, 1966).

Potentials below the cortical surface. When exploring the Group I response at varying depths below the cortical surface other differences between the evoked Group I response and the cutaneous response became apparent. Figure 1*a* illustrates a series of these potentials. The responses are due to a single shock to the contralateral deep radial nerve at 1·7*T*. Except for the region of reversal point at approximately 400 μ below the surface, the Group I evoked response maintained a constant latency of 5·5 msec. At depths between 700 and 1000 μ the negative potential reached maximum amplitude and slowly declined with further advances of the electrode until it almost vanished at depths below 2·0 mm. The reversal point for the Group I response generally occurred between depths of 400 and 500 μ if the cortex was in good condition and cortical dimpling by the electrode was minimized.

The reversal point for the potentials evoked by a cutaneous volley (Fig. 1*b*) consistently appeared in more superficial layers of the cortex relative to the Group I potential reversal point. As a rule, the two reversal points were separated by 250–300 μ, which suggests that there may be fundamental differences in the modes of termination of the two specific thalamocortical relays in respect to the distribution of their terminals within the cortical layers and the neurone populations in their respective spheres of influence.

Pyramidal tract responses. The observation that the area from which the Group I response was recorded was located near the posterior limits of the motor cortex and that the reversal point for the Group I response was deeper than the reversal point for the cutaneous response suggested that depolarization of Pt neurones might contribute to the Group I cortical response wave form. To test this possibility the bulbar pyramids were explored with electrodes in animals anaesthetized with chloralose. Conventional recording methods, with display gains of 20–50 μV/cm, were not adequate to reveal relayed pyramidal responses to forelimb Group I volleys, but low amplitude responses may have been present and masked within the noise level. A signal averager was used to improve the signal-to-noise ratio and revealed that pyramidal discharges were evoked by Group I volleys. They normally could be detected only with such averaging techniques.

Group I relayed pyramidal responses are displayed in Fig. 2. The effect

of single Group I volleys is shown at decreasing stimulus repetition rates in Fig. 2*a–d*. At rates below 0·5/sec (*d*) the amplitude of the Group I pyramidal response was maximal; the duration of the response varied between 20–25 msec. Repetition rates of 4/sec (Fig. 2*a*) or more, generally abolished the responses. The amplitude of the response was increased to a variable degree with a brief train of Group I volleys (Fig. 2*e–h*), but responded to alterations in stimulus frequency as did responses to single shocks (Fig. 2*a–d*). Potentiation of the response by brief repetitive trains can be seen by comparison of traces *b* and *f* and also *j* and *l*. Even with a brief train the duration of the response was not greatly prolonged indicating that early facilitation was probably terminated by late powerful

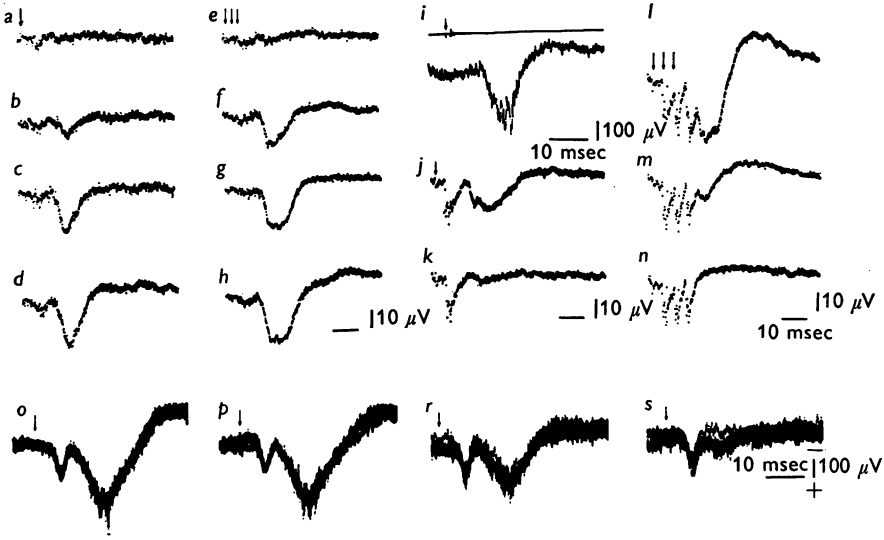

Fig. 2. Identification of the relayed Group I pyramidal response. The electrode tip was located within the bulbar pyramid at the level of the contralateral inferior olive in records *a–i*. Traces *a–h* and *j–n* are averaged responses of 127 sweeps each for a period of 63 msec. Stimulus to deep radial nerve at 1·7*T*; *a*, single shock at 4/sec; *b*, 2/sec; *c*, 1/sec; *d*, 0·5/sec; *e*, train of three Group I volleys with intershock intervals of 2·5 msec, train repetition rate was 4/sec; *f*, 2/sec; *g*, 1/sec; *h*, 0·5/sec; *i*, same recording site but showing a single sweep of a relayed pyramidal response to a cutaneous volley at 2·0*T* with conventional recording methods; *j–s*, responses recorded from another subject in which the electrode tip was positioned near the dorsal border of bulbar pyramid to show that the ascending lemniscal volley was not affected by repetitive stimulation. *j*, single Group I volleys (1·7*T*) at stimulus rate of 0·5/sec; *k*, 4/sec; *l*, three Group I volleys with 4 msec intershock interval with train repetition rate of 0·5/sec; *m*, 4/sec; *n*, 6·3/sec; *o–s*, same electrode location, conventional recording (five superimposed sweeps each) of relayed pyramidal responses to single cutaneous volleys at 2·0*T*. Repetition rates are: *o*, 0·5/sec; *p*, 1/sec; *r*, 2/sec; *s*, 4/sec. Chloralose anaesthesia. Arrows mark time of peripheral nerve stimulation.

inhibition of thalamic or cortical origin (Brooks, 1963; Andersen, Brooks, Eccles & Sears, 1964).

The amplitude of the relayed pyramidal Group I response was always extremely small compared to the relayed response to a cutaneous volley (Fig. 2*i*) recorded from the same site in the pyramid. This effect is probably a reflexion of the much larger neocortical projection zone receiving relayed cutaneous actions and the larger number of neurones responding in this zone.

Recognition of the relayed pyramidal responses to cutaneous and Group I afferent volleys was also supported by anatomical evidence showing that the electrode tips had to be placed within the bulbar pyramids to obtain records such as in Fig. 2*a–i* and that there was a depression of the response at stimulus rates exceeding 1–2/sec (Patton & Amassian, 1960). Additional evidence was found by advancing the pyramidal electrode dorsally 0·5–1·0 mm. This change placed the electrode tip in a region between the bulbar pyramid and the overlying medial lemniscus. With such an electrode placement, the early ascending lemniscal response was observed on the leading edge of the late pyramidal response (Fig. 2*j–s*) as was shown by Patton & Amassian (1960). Figure 2*j* and *k* shows examples of this type of experiment. The Group I relayed response, which is clearly visible at stimulus rates of 0·5/sec (*j*), is nearly abolished at a rate of 2/sec (*k*), but there was no alteration of the ascending lemniscal volley. A brief repetitive train of Group I volleys in Fig. 2*l* greatly facilitated the response as compared to the single shock in *j*; but a rate of 6·2/sec (Fig. 2*n*) totally abolished the relayed pyramidal response without modifying the ascending lemniscal volleys. The relayed pyramidal discharge to a Group I volley possessed the same general characteristics as the relayed response to a cutaneous volley. The latter also shows depression with an increase in stimulus rate above 1·0 sec as shown in Fig. 2*o–s* observed in the same location as *j* with conventional recording methods.

Relayed responses to Group I afferent volleys first appeared in the bulbar pyramids in 7·0–11·2 msec (mean 9·6 msec) and reached peak amplitudes having a mean latency of 15·7 msec.

Cortical units influenced by Group I afferent volleys. A total number of 57 units, influenced by Group I volleys, cutaneous volleys or both, were recorded in the Group I cortical projection zone. About half of these units were obtained from preparations anaesthetized with chloralose; the remainder were recorded with the animals under barbiturate anaesthesia. There was essentially no difference in the mean unit response latencies under these two conditions. Of the 57 units observed, 23 received convergent actions from low threshold deep radial nerve volleys and cutaneous afferent volleys. For these cells the mean latency to the first spike dis-

charge was 8·6 msec (range, 6·2–11·6 msec) with Group I volleys; the same cells responded to cutaneous volleys with a mean latency of 11·1 msec (range, 6·8–12·5 msec). Only 12 units responded exclusively to Group I volleys. Sixteen neurones responded to cutaneous volleys and to high threshold deep radial nerve volleys which indicated that cutaneous and high threshold muscle afferents had strong convergent actions (Oscarsson, Rosen & Sulg, 1966; Andersson *et al.* 1966).

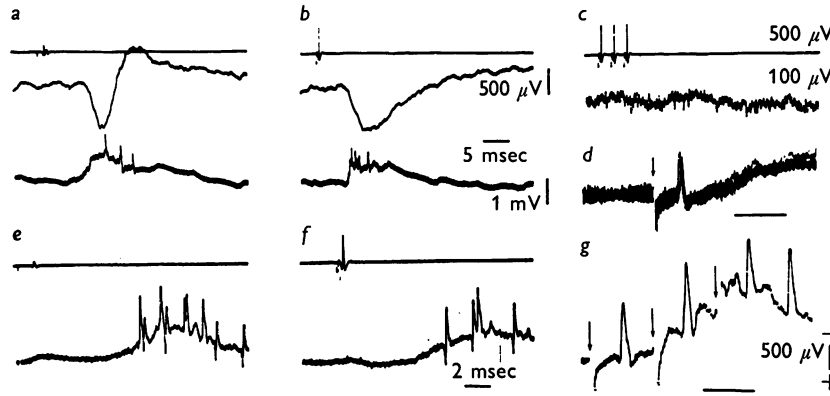

Fig. 3. *a* and *b*, Unit in the Group I cortical projection zone which responded to both superficial radial (2·0*T*) and deep radial nerve volleys (1·7*T*) respectively. The upper trace shows the peripheral nerve volley at the level of the brachial plexus. The middle traces show the surface responses. The lower traces show the extracellularly recorded response of the isolated unit. *c*, Single sweep recording from the pyramidal tract revealed no apparent response to a brief train of stimuli (intershock interval 2·5 msec) to the deep radial nerve (1·7*T*) with conventional recording methods. *d*, Stimulation of the bulbar pyramid at the same site as *c*, antidromically activated the unit. Another unit, *e–g*, also received convergent actions from superficial radial nerve volleys at 1·2*T* (*e*) and by deep radial volleys at 1·7*T* (*f*). Stimulation of the bulbar pyramid activated the unit at a short constant latency at a rate of 400/sec (*g*). Calibration: *a*, *b* and *c*. 5 msec, the remaining traces at 2 msec. Amplification in traces *d–g*, 500 μV. Chloralose anaesthesia.

Figure 3*a* and *b* shows a cortical neurone activated by a cutaneous volley and a Group I volley. The cortical surface (middle traces) was explored with a low impedance micro-electrode until the maximal response focus for the Group I volley was located. The surface electrode was kept in this location in order to monitor the condition of the cerebral cortex. A second, but higher impedance micro-electrode (2–30 MΩ) was positioned so that its tip could explore the deeper cortical layers directly under the surface lead. Figure 3*a* and *b* (lower traces) shows a unit responding on the crest of the deep negative field potential. The responding units almost always appeared at a time corresponding to the surface positive wave. The unit, located at a depth of 1100 μ, responded to a

cutaneous volley (*a*) with a mean latency of 13·0 msec and to a Group I volley (*b*) with a mean latency of 7·5 msec. Responses from single neurones revealed that actions by Group I and cutaneous volleys converging on the same cells were primarily excitatory and that Group I effects occurred at shorter latencies than cutaneous effects.

An unaveraged recording from the bulbar pyramid of this subject is shown in Fig. 3*c*. Even with the potentiating effect of a brief train of Group I volleys, no response was observed; however, averaging methods easily disclosed pyramidal responses similar to those seen in Fig. 2*e–h*. Weak stimulation of the bulbar pyramid at this recording site in Fig. 3*c* evoked an all-or-none, constant latency (0·95 msec) activation of the cortical cell (Fig. 3*d*). This unit was driven faithfully at a rate of 500/sec and was identified as a Pt neurone.

Figure 3*e–g* shows another Pt cell that received convergent actions from cutaneous and Group I afferent volleys. A weak cutaneous volley (1·2*T*) activated the cell after a delay of 9·5 msec (*e*); the Group I volley (1·7*T*) activated the cell in 8·4 msec (*f*). The unit was identified as a Pt cell by its ability to follow stimulation of the bulbar pyramid at a rate of 400/sec with a constant latency of 1·15 msec.

The destination of axons of Pt cells influenced by Group I volleys is only speculative, but it is known that many Pt cells give rise to axons which enter in the corticospinal tract system (Chambers & Liu, 1957). Figure 4*a* and *b* shows an extracellularly recorded response of a cortical neurone activated by a Group I volley (1·7*T*) in 9·5 msec (*a*) and a cutaneous volley (2·0*T*) in 10·5 msec. The cutaneous effect was more powerful and consistently evoked a burst of 1–4 spikes from the unit, whereas the Group I volley produced only 1–2 spikes. The unit was identified as a Pt cell because it responded after an invariable short latency (0·9 msec) to brief repetitive stimulation of the contralateral bulbar pyramid at 625/sec (Fig. 4*c*, *d*). Stimulation of the spinal cord in the ipsilateral medial portion of the dorsolateral quadrant of C_2 (Fig. 5) at 500/sec faithfully evoked an all-or-none response of the same Pt cell recorded in Fig. 4*a–d*, with a constant latency of 1·25 msec (Fig. 4*e*).

The corticospinal tract response to Group I afferent volleys, similar to the pyramidal tract response at the level of the inferior olive, can be visualized only with the aid of a response averaging system. Figure 4*f* shows the response to a brief train of Group I volleys recorded at the same site from which the Pt cell in Fig. 4*e* was antidromically activated. The early complex disturbances are due to short latency Group I responses in fibres of the dorsal spinocerebellar tract and the neighbouring bundles of Group I primary afferent fibre collaterals. The late slow wave, the initial part of which is obscured by the fibre potentials of the third ascending

volley is due to cortical relayed activity descending in the corticospinal tract.

The corticospinal tract response was also subject to severe depression with stimulus repetition rates above 1/sec as would be expected from the fact that the pyramidal tract response at the level of the bulbar pyramid was also depressed at rates above 1/sec. At rates of 4/sec (Fig. 4*g*) a small portion of the relayed discharge was still present but at 6·3/sec (Fig. 4*h*) the Group I relayed response was almost totally abolished. The dotted line, in Fig. 4*f*, represents the base line profile of the relayed response

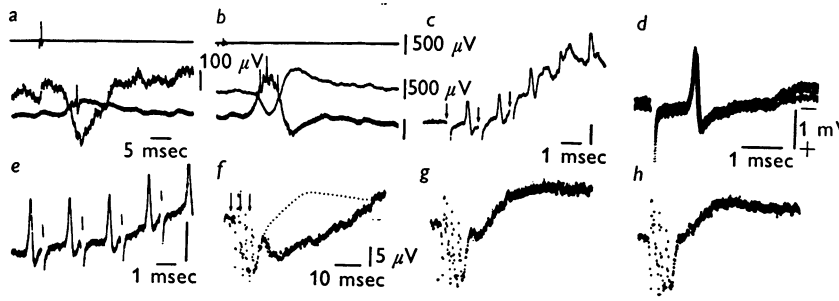

Fig. 4. Group I activation of the corticospinal tract system. *a*, deep radial nerve stimulation (1·7*T*). The upper trace shows the peripheral nerve volley, the middle trace shows the cortical surface response, and the bottom trace the unit response. *b*, The same unit responded to superficial radial nerve volleys (2·0*T*). *c–d*, The unit responded to repetitive stimulation (625/sec) of the bulbar pyramid with a uniformly short latency (0·9 msec, five superimposed sweeps). *e*, The same unit followed high rate (500/sec) stimulation of the spinal cord at C_2 at a latency of 1·25 msec. *f–h*, Averaged response recordings from the same position in the cord from which the Pt cell was antidromically driven. A brief train of Group I volleys (1·7*T*, intershock interval 4·0 msec) at a repetition rate of 0·5/sec revealed a late positive slow potential. An increase in the train repetition rates to 4/sec (*g*) and 6·3/sec (*h*) depressed the slow wave in the same manner that was observed for the relayed pyramidal tract responses in Fig. 2. The Group I relayed corticospinal tract response can be visualized by comparing the difference between the wave-forms of *f* and *h*, the latter represented by the dotted line in *f*. Chloralose anaesthesia.

recorded at a train repetition rate of 6·3/sec. The full extent of the depression of the corticospinal tract response can be compared with the maximum amplitude response accumulated at 0·5/sec. The location of the stimulating electrode in C_2 was verified histologically and is displayed in Fig. 5*a*.

Because of complications from fibre tract potentials from ascending Group I actions, the earliest measurable latency in Fig. 4*f*, for the cortically relayed Group I responses, was 12·5 msec. Corticospinal relayed responses to Group I volleys can arrive at C_2 much earlier. It should be recalled that

the mean latency for the relayed Group I pyramidal response at the level of the inferior olive was 9·5 msec. The Pt cell in Fig. 4*a* was activated at the same latency and would not have been detectable at C_2 much before 10·7 msec. Under optimum conditions it was possible to observe a relayed corticospinal tract response commencing as early as 7·4 msec. An example of this type of response is shown in Fig. 5*b*. The Pt cell origin of the response was confirmed by its disappearance with 4/sec stimulation of the deep radial nerve at 1·7*T* (Fig. 5*c*). Figure 5*d* shows a complex positive–negative slow wave at the same recording site with single cutaneous volleys at 2·0*T* and at a stimulus frequency of 0·5/sec. Both the initial

Fig. 5. *a*, Drawing of a spinal cross-section at the level of C_2 showing the area from which relayed corticospinal tract discharges could be recorded and from which cortical Pt neurones could be antidromically activated from the experiment in Fig. 4. The averaged responses in *b–d* were taken from another experiment, but electrode placement was essentially the same; *b*, Example of a corticospinal tract response to single Group I volleys (1·7*T*) with a 0·5/sec repetition rate showing a minimum latency of 7·4 msec. *c*, Same as *b*, except stimulus rate was 4/sec. *d*, The relayed response with single cutaneous volleys (2·0*T*) at a stimulus rate of 0·5/sec. Upward arrow indicated earliest relayed response with latency 9·6 msec. Downward arrows mark shock artifacts. Chloralose anaesthesia.

positive and the late, larger negative wave disappeared at frequencies above 6·3/sec (not shown). The early part of the positive deflexion of the relayed cutaneous response appeared after a delay of 9·6 msec (upward arrow). The Group I evoked corticospinal tract discharge, reached the C_2 spinal segment 2·2 msec earlier than the cutaneous response. It should be recalled that the mean latency difference between Group I and cutaneous primary evoked responses in the Group I projection zone of the neocortex was 2·0 msec.

Non-Pt cells and unidentified units. Many cortical neurones that responded at short latencies to either Group I or cutaneous afferent volleys could not be identified as Pt cells. Figure 6 shows a non-Pt cell located at a depth of 730 μ. It is shown responding to a Group I volley at 1·5*T* (*a*) and to a cutaneous volley at 1·2*T* (*b*). There was no clear-cut interaction between the two sensory inputs in this particular case. A conditioning volley to the deep radial nerve at 1·7*T* did not influence the unit response to a cutaneous volley (1·5*T*) delivered 25 msec later (Fig. 6*c*). When the bulbar pyramids were stimulated, the unit responded with a burst of

spikes but the earliest spike appeared after 4 msec. The latency of the initial spike was not constant indicating that the unit was not antidromically activated (Fig. 6*d*). Single shock or repetitive stimulation of the Group I thalamic relay nucleus (Fig. 6*e* and *f*) showed that only the initial orthodromic response of the cell occurred at predictable latencies and that the cell was unable to follow stimulus rates above 50/sec. Thalamic stimulation at 250/sec (Fig. 6*f*), showed that the second spike was already delayed and no further discharge could be evoked from the cell.

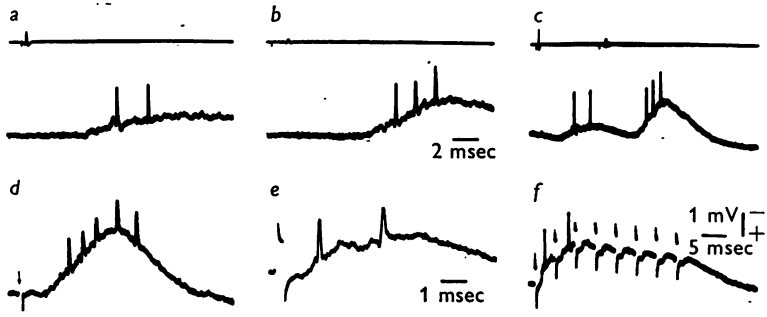

Fig. 6. A non-Pt cell with convergent facilitatory actions by Group I and cutaneous afferent volleys. *a*, Unit responded at mean latency of 7·5 msec to Group I volley at 1·5*T*. *b*, The same cell responded to a cutaneous volley at 1·2*T*. *c*, There was no clear interaction between the two sensory inputs. The Group I volley (1·7*T*) preceded the cutaneous shock (1·5*T*) by 25 msec but did not alter the cell's response to the cutaneous volley. *d*, Stimulation of the bulbar pyramid activated the cell at long variable latencies. The cell also failed to respond faithfully to repetitive stimulation. Single shock (*e*) and repetitive (*f*) stimulation of the Group I thalamic relay nucleus at 250/sec rate. Cell failed to respond at high rates orthodromically. Calibration of upper trace in *a*–*c*, 500 μV; *a*, *b* and *d*, 2 msec; *c* and *f*, 5 msec.

Some Pt cells, non-Pt cells and unidentified cells did not receive convergent actions from the two peripheral nerves. Some responded exclusively to Group I volleys, while others responded only to cutaneous or high threshold muscle afferent volleys. Of those cortical units driven by single Group I volleys, a few were activated at thresholds as low as 1·0*T* but were not influenced by cutaneous shocks as strong as 9·0*T*. Units that responded to low threshold cutaneous volleys also responded to high threshold deep radial nerve volleys.

Under Nembutal anaesthesia, efforts were made to obtain intracellular recordings of cells driven by Group I afferent volleys. Some unidentified cortical cells responded at extremely low thresholds to both cutaneous and Group I volleys. One cell was driven faithfully with Group I afferent volleys as low as 1·04*T*. The same neurone also responded equally well to cutaneous volleys at 1·05*T*. The low threshold actions of Group I

volleys clearly demonstrate that primary muscle spindle afferents (Ia) can evoke depolarizing actions in Pt cells but there is no clear evidence that Ib afferent volleys exert similar actions.

DISCUSSION

In agreement with Amassian, Patton, Woodbury, Towe & Schlag (1955) and Mountcastle, Davies & Berman (1957), all units described in this study were evoked on a negative slow potential, and the initial spike of the cell's discharge frequently occurred on the early portion of it. The reversal point of the Group I evoked potential was always 250–300 μ deeper than the reversal point of the cutaneous response. Potential reversals of evoked responses to superficial radial nerve volleys generally appeared at depths corresponding to layers II and III (Li, Cullen & Jasper, 1956). The depth at which the Group I cortical evoked response generally reversed was between 400–500 μ (layers III and IV) in agreement with Oscarsson's (1966) observations. The superficial location of reversal points was used as an indication that the cortex was in good condition (Amassian *et al.* 1955; Mountcastle *et al.* 1957).

The deep negative potential of the cortical primary is assumed to be largely due to post-synaptic potentials (PSPs) of cortical cells (Eccles, 1951; Perl & Whitlock, 1955; Mountcastle *et al.* 1957; Bremer, 1958; Chang, 1959; Amassian, 1961). Early potentials due to depolarization of presynaptic terminals were not seen in the present experiments, but they have been observed in response to Group I volleys by Oscarsson & Rosen (1963). Latency measurements of evoked response activity, therefore, probably represent the earliest post-synaptic activation of cortical neurones.

The late slow surface negative wave was usually absent from the Group I evoked response. At least two factors may contribute to this phenomenon. First, the cortical cells activated by a Group I afferent volley are far fewer in number and less widely distributed than those influenced by cutaneous volleys. As a result, relayed Group I afferent actions may have influenced such a limited population that late slow waves were attenuated in comparison to the early slow waves associated with early post-synaptic depolarizations. Secondly, because of the deep reversal point, the cell populations influenced by Group I volleys appeared to reside in deeper zones of the cortex. It is conceivable that these cells may have a less extensive array of cell processes extending into the more superficial cortical zones.

Recordings of relayed pyramidal tract responses to Group I volleys revealed variable latencies ranging between 7·0 and 11·2 msec (mean 9·5 msec). Cutaneous responses exhibited about the same mean latencies

(9·6 msec) but there was far less variability. The earliest latency observed for the Group I cortical evoked responses was 5·0 msec, consequently, a difference of only 2·0 msec appeared between the earliest appearance of a Group I evoked response in the cortex and the earliest appearance of the relayed response in the bulbar pyramid (7·0 msec). The shortest latency observed for a Group I driven Pt cell was 7·5 msec. Initiation of Pt cell soma spikes would be expected to occur at latencies slightly in excess of 5·0 msec, because the initial portion of the cortical response may have been extended slightly by depolarization in presynaptic terminals of thalamocortical fibres and because the soma spikes could be delayed by slowly rising excitatory post-synaptic potentials (EPSPs) (Oscarsson *et al.* 1966). There may be a delay of 1–2 msec between appearance of the EPSP and development of sufficient depolarization of the cell membrane to reach firing level (J. E. Swett & C. M. Bourassa, personal observations). The brief interval of 2·0 msec between arrival of the Group I volley in the cortex and appearance of the relayed pyramidal response suggests that some Pt cells may be monosynaptically activated by Group I thalamocortical relay cells (Amassian & Weiner, 1966).

The averaged response recordings at the level of C_2 revealed that relayed Group I activity in the corticospinal tract appeared as early as 7·4 msec. This implies that a few cortical Pt cells may be activated by Group I volleys as early as 6·2 msec; some unidentified cortical cells did respond at these latencies. The areas in the spinal cord from which Group I corticospinal relayed responses could be recorded and from which Group I activated Pt cells could be antidromically activated corresponds closely with the anatomical location of the corticospinal tract (Chambers & Liu, 1957).

The results of the experiments described here do not fully agree with the conclusions of recent experiments by other investigators. Oscarsson *et al.* (1966) recorded from units in the neocortical Group I projection zone under experimental conditions similar to the ones used here. They were unable to identify Pt neurones that were activated by Group I afferent volleys. Except for this difference, other observations concerning sensory convergence between Group I and cutaneous volleys, the low threshold activation of cortical units by Group I and cutaneous volleys and the predominance of Group I evoked EPSPs in cortical units, are in agreement with their findings. Pt cell activation by Group I volleys may have been overlooked because of sampling error. It is also possible that only a fraction of the Pt cells driven by Group I volleys send their axons as far as the spinal cord to reach the point at which Oscarsson *et al.* (1966) applied their stimuli.

Previous work has shown that the activity produced by selective

activation of Group I fibres in peripheral nerve will not serve as a discriminative stimulus for the cat (Swett, Bourassa & Inoue, 1964). This is true even though Group I activation produced cortical potentials in the awake unrestrained subject (Bourassa & Swett, 1966).

Excluding cerebellar pathways, there is more than one route through which ascending cutaneous actions may be relayed to brain stem and diencephalic structures, but Group I afferent volleys appear to be conveyed only through the dorsal columns (Oscarsson & Rosen, 1966). While interruption of the dorsal column pathway may not effect sensory thresholds or sensory discrimination (Cook & Browder, 1965; Levitt & Schwartzman, 1966), it has been shown that the dorsal column system alone conveys sufficient sensory information to support normal thresholds for two-point and limb position discrimination (Levitt & Schwartzman, 1966; Vierck, 1966).

Andersson *et al.* (1966) have shown that some neurones of the ventrobasal complex which relay Group I activity to cortex also receive convergent actions from cutaneous volleys. Because Pt and non-Pt cells often respond to both Group I and weak cutaneous afferent volleys, it is evident that activity in some cortical neurones influenced by cutaneous stimulation is unrelated to discriminative behavioural processes.

The observation that Group I volleys do not support discrimination would be understandable if the thalamic relay nuclei and cortical cells, influenced by Group I volleys, were engaged solely in motor functions. The strong facilitatory actions of Group I volleys on Pt cells supports this point of view. On the other hand, it is probable that there are subtle forms of interaction so that Group I afferent volleys may effect excitability changes of cortical neurones outside of what can be considered motor cortex. Recent experiments by Andersson *et al.* (1966) and Landgren & Wolsk (1966) show that Group I effects can be detected in a small region of S II. It would appear, therefore, that the lack of discrimination to Group I afferent volleys may not be so much a function of the small cortical zones to which they project, but rather a function of additional cortical or subcortical systems which receive cutaneous actions to the exclusions of Group I actions. It is interesting to note in this regard that the brainstem reticular formation is almost devoid of Group I influences (Pompeiano & Swett, 1963*a*, *b*; Magni & Willis, 1964; Lymans' Kyi, 1966).

We would like to gratefully acknowledge the valuable assistance of Dr Robert S. Dow, Mrs L. Ronald, Mr Mills and Mr Lynch. This work was supported by USPHS Grants NB 05048 and NB 02289.

REFERENCES

ADRIAN, E. D. & MORUZZI, G. (1939). Impulses in the pyramidal tract. *J. Physiol.* **68**, 139–154.

AMASSIAN, V. E. (1961). Microelectrode studies of the cerebral cortex. *Int. Rev. Neurobiol.* **3**, 67–136.

AMASSIAN, V. E. & BERLIN, L. (1958). Early cortical projection of Group I afferents in forelimb muscle nerves of cat. *J. Physiol.* **143**, 61*P*.

AMASSIAN, V. E., PATTON, H. D., WOODBURY, J. W., TOWE, A. & SCHLAG, J. E. (1955). An interpretation of the surface response in somatosensory cortex to peripheral and interareal afferent stimulation. *Electronceph. clin. Neurophysiol.* **7**, 480–488.

AMASSIAN, V. E. & WEINER, H. (1966). Monosynaptic and polysynaptic activation of pyramidal tract neurones by thalmic stimulation. *The Thalamus*, ed. PURPURA, D. P. & YAHR, M. D., pp. 255–282. New York: Columbia University Press.

ANDERSON, P., BROOKS, C. M., ECCLES, J. C. & SEARS, T. A. (1964). The ventro-basal nucleus of the thalamus: potential fields, synaptic transmission and excitability of both presynaptic and postsynaptic components. *J. Physiol.* **174**, 348–369.

ANDERSSON, S. A., LANDGREN, S. & WOLSK, D. (1966). The thalamic relay and cortical projection of Group I muscle afferents from the forelimb of the cat. *J. Physiol.* **183**, 576–591.

BOURASSA, C. M. & SWETT, J. E. (1966). Cortical evoked potentials and detection of somatic sensory volleys in awake cats. *Proc. Am. psychol. Ass.* 74th Annual Convention, pp. 103–104.

BREMER, F. (1958). Cerebral and cerebral potentials. *Physiol. Rev.* **38**, 357–388.

BROOKS, V. B. (1963). Variability and redundancy in the cerebral cortex. *Electroenceph. clin. Neurophysiol.* **24**, 13–32.

BROOKS, V. B., RUDOMIN, P. & SLAYMAN, C. S. (1961). Sensory activation of neurons in the cat's cerebral cortex. *J. Neurophysiol.* **24**, 286–301.

BUSER, P. & ASCHER, P. (1960). Mise en jeu reflexe du système pyramidal chez le chat. *Archs ital. Biol.* **98**, 123–164.

CAMPBELL, A. W. (1905). *Histological Studies on the Localization of Cerebral Function*, 1st edn., pp. 276–279. Cambridge University Press.

CHAMBERS, W. W. & LIU, C-N. (1957). Cortico spinal tract of the cat. *J. comp. Neurol.* **108**, 23–55.

CHANG, H. T. (1959). The evoked potentials. In *Handbook of Physiology*, section 1, vol. I. ed. FIELD, J. Washington, D.C.: American Physiological Society.

COOK, A. W. & BROWDER, E. J. (1965). Function of posterior columns in man. *Archs Neurol.* **12**, 72–79.

ECCLES, J. C. (1951). Interpretation of action potentials evoked in the cerebral cortex. *Electroenceph. clin. Neurophysiol.* **3**, 449–464.

ECCLES, J. C., OSCARSSON, O. & WILLIS, W. D. (1961). Synaptic action of Group I and II afferent fibers of muscle on the cells of the dorsal spinocerebellar tract. *J. Physiol.* **158**, 517–543.

ECCLES, R. M. & LUNDBERG, A. (1959). Synaptic action in motoneurons by afferents which may evoke the flexion reflex. *Archs ital. Biol.* **97**, 199–221.

HASSLER, R. & MUHS-CLENENT, K. (1964). Architektonischer Aufbau des sensorimotorischen und parietalen Cortex der Katz. *J. Hirnforsch.* **6**, 377–420.

JASPER, H. H. & AJMONE-MARSAN, C. (1955). *Stereotaxic Atlas of the Diencephalon of the Cat.* Ottawa: Nat. Res. Council.

KENNEDY, T. T. & TOWE, A. L. (1962). Identification of a fast lemnisco-cortical system in the cat. *J. Physiol.* **160**, 535–547.

KENNEDY, T. T., TOWE, A. L. & PATTON, H. D. (1960). Contribution of pyramidal tract (PT) neurons to surface primary evoked response in cat. *Physiologist* **3**, 93.

LANDGREN, S. & WOLSK, D. (1966). A new cortical area receiving input from Group I muscle afferents. *Life Sci. Oxford* **5**, 75–79.

LEVITT, M. & SCHWARTZMAN, R. (1966). Spinal sensory tracts and two point tactile sensitivity. *Anat. Rec.* **154**, 377.

LI, C. L., CULLEN, C. & JASPER, H. H. (1956). Laminar microelectrode studies of specific somatosensory cortical potentials. *J. Neurophysiol.* **19**, 111–130.

LYMANS' KYI, YU. P. (1966). Responses of neurons of the medullary reticular formation to afferent impulses from cutaneous and muscle nerves. *Fedn Proc.* Translation supp. **25**, T15-T17.

MAGNI, F. & OSCARSSON, O. (1962). Comparison of ascending spinal tracts activated by Group I muscle afferents in the phalanger, rabbit and cat. *Acta physiol. scand.* **54**, 37–52.

MAGNI, F. & WILLIS, W. D. (1964). Subcortical and peripheral control of brainstem reticular neurons. *Archs. ital. Biol.* **102**, 434–448.

MALLART, A. (1964). Projections des afferences muscularies de la patte anterieure an nivean du thalamus chez le chat. *C. r. hebd. Séanc. Acad. Sci., Paris* **259**, 1215–1218.

MILLS, L. W. & SWETT, J. E. (1965). Precise control of stimulation parameters in low impedance conditions. *J. appl. Physiol.* **20**, 334–338.

MORSE, R. W. & TOWE, A. L. (1964). The dual nature of the lemnisco-cortical afferent system in the cat. *J. Physiol.* **171**, 231–246.

MOUNTCASTLE, V. B., DAVIES, P. W. & BERMAN, A. L. (1957). Response properties or neurons of cat's somatic sensory cortex to peripheral stimuli. *J. Neurophysiol.* **20**, 374–407.

OSCARSSON, O. (1966). The projection of Group I muscle afferents to the cat cerebral cortex. In *Muscle Afferents and Motor Control*, Nobel Symposium I, ed. GRANIT, R., pp. 307–316. New York: John Wiley and Sons.

OSCARSSON, O. & ROSEN, I. (1963). Projection to cerebral cortex of large muscle spindle afferents in forelimb nerves of the cat. *J. Physiol.* **169**, 924–945.

OSCARSSON, O. & ROSEN, I. (1966). Short latency projections to the cat's cerebral cortex from skin and muscle afferents in the contralateral forelimb. *J. Physiol.* **182**, 164–184.

OSCARRSON, O., ROSEN, I. & SULG, I. (1966). Organization of neurones in the cat cerebral cortex that are influenced from Group I muscle afferents. *J. Physiol.* **183**, 189–210.

PATTON, H. D. & AMASSIAN, V. E. (1960). The pyramidal tract: its excitation and function. In *Handbook of Physiology*, Section 1, vol. II, ed. FIELD, J., pp. 837–861. Washington, D.C.: American Physiological Society.

PATTON, H. D., TOWE, A. L. & KENNEDY, T. T. (1961). Activation of pyramidal tract neurones by ipsilateral cutaneous stimuli. *J. Neurophysiol.* **23**, 501–514.

PERL, E. R. & WHITLOCK, D. G. (1955). Potentials evoked in cerebral somatosensory region. *J. Neurophysiol.* **18**, 486–501.

POMPEIANO, O. & SWETT, J. E. (1962). Identification of cutaneous and muscular afferent fibers producing EEG synchronization or arousal in normal cats. *Archs ital. Biol.* **100**, 343–380.

POMPEIANO, O. & SWETT, J. E. (1963*a*). Actions of graded cutaneous and muscular afferent volleys on brainstem units in the decerebrate, cerebellectomized cat. *Archs ital. Biol.* **101**, 552–583.

POMPEIANO, O. & SWETT, J. E. (1963*b*). Cerebellar potentials and responses of reticular units evoked by muscular afferent volleys in the decerebrate cat. *Archs ital. Biol.* **101**, 584–613.

SWETT, J. E. & BOURASSA, C. M. (1965). Perception of somatic sensory volleys in awake cats. *Fedn Proc.* **24**, 140.

SWETT, J. E. & BOURASSA, C. M. (1966). Activation of corticospinal tract fibers by Group I afferent volleys. *Physiologist* **9**, 301.

SWETT, J. E., BOURASSA, C. M. & INOUE, S. (1964). Effects of cutaneous and muscle sensory nerve volleys in awake cats: a study in perception. *Science, N.Y.* **145**, 1071–1073.

VIERCK, C. J. (1966). Spinal pathways mediating limb position sense. *Anat. Rec.* **154**, 437