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# SENSORIMOTOR CONTROL OVER FUSIMOTOR NEURONS OF THE TENUISSIMUS MUSCLE IN THE ANESTHETIZED CAT: A QUALITATIVE PRIMARY AFFERENT RECORDING

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**ABSTRACT**

Cortical control of the sensory output of muscle spindles was studied in thirteen anesthetized cats in the present experiment. Gamma motoneuron activity was monitored during electrical stimulation of the sensorimotor cortex while recording from single primary afferents from the tenuissimus muscle. Findings are as follows:

1. The state of anesthesia is crucial in obtaining reproducible results and variation in the state of anesthesia can alter the fusimotor effect from static to dynamic or even from excitation to inhibition, a finding consistent with those of Vedel and Mouillac-Baudevin.[30] The anesthetic agent used was also important in determining the magnitude and types of responses to cortical stimulation. the initial burst of the primary afferent in response to passive stretch was by far greater with chloralose than with barbiturate anesthesia in the tenuissimus muscle, suggesting that there may be a tonic low-level dynamic gamma excitation in chloralose anesthesia.

2. The state of the sensorimotor cortex is another determinant factor. Prevention of $CO_2$ escape from the surface of the cortex in the present experiments, by covering the cortex with 1cm of mineral oil, is thought to be the sole factor which made these results different from those obtained by Gladden and McWilliam.[11,12]

3. Different types of static gamma motoneurons could be recruited from the sensorimotor cortex independently.

4. The topographical mapping of the sensorimotor cortex in relation to the type of recruited gamma motoneurons, static or dynamic, was as follows: a) A "dynamic area" was identified from which dynamic effects were clearly elicited during stimulation. b) Static effects were elicited following stimulation of a much wider area across the sensorimotor cortex, the postcruciate dimple being almost at the center.



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5. The sensorimotor cortex was not only capable of controlling static gamma motoneurons independently from dynamic ones, but also capable of simultaneously inhibiting static gamma motoneurons and exciting others, lending support to the idea put forward by others.[6]

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**Keywords**: Fusimotor, Gamma motoneuron, Sensorimotor, Primary afferent, Tenuissimus.

## INTRODUCTION

The central influence on the gamma system was studied for the first time by Granit and Kaada.[15] They found that the discharge of both the gamma (Γ) axons and the muscle spindle afferents were facilitated or inhibited by stimulation of various parts of the central nervous system, including the cerebral cortex. The earliest indication of independent control of static and dynamic properties of muscle spindles by the anterior lobe of the cerebellum in decerebrate cats came in 1962,[21] and has since gained support by other observations reported.[1,2,7,14,18,19,23,25]

Vedel and his colleagues provided various examples of a rather selective central activation of either static or dynamic fusimotor neurons recording from primary afferents in cats under halothane anesthesia.[30] With regard to the possible existence of higher centers other than the red nucleus in the mesencephalon having descending systems which influence fusimotor neurons, the work of Vedel is of considerable interest. This author obtained contralateral spindle effects. Their results strongly indicate pyramidal tract control over the dynamic fusimotor neurons. In subsequent work, Vedel and his colleagues found that responses produced by stimulation of other areas of the central nervous system tended to be rather labile and at some sites the effect might change from predominantly static to predominantly dynamic on repeated application of the stimulus.[28,29] A more thorough investigation of the effects of cortical stimulation showed that on varying the depth of anesthesia, static effects could be obtained of the more usual dynamic effect.[30] The findings of Wand and Schwarz is consistent with the idea that the central nervous system can control the activity of static gamma ($\Gamma_s$) motoneurons innervating static bag ($Sb_2$) fibers (whose contraction excites primary endings, with little or no effect on secondary endings) independently of those innervating nuclear chain (Nc) fibers (whose contraction excites both primary and secondary endings).[31]

The purpose of the present work was firstly to investigate whether different types of fusimotor neurons can be independently recruited from specific areas within the sensorimotor cortex of the cat, and secondly to map the sensorimotor cortex topographically.

## MATERIALS AND METHODS

Thirteen cats of either sex weighing between 2.2 to 3.8 kg were used for the present investigation. Anesthesia was induced by barbiturates injected intra-peritoneally (45 mg/kg Sagatal, May and Baker). The last three cats were anesthetized with chloralose (40 mg/kg). Trachea was intubated after a pretracheal midline incision. A nutritive solution (Dextran or a mixture of D-glucose and sodium bicarbonate, 10-20 mL) was given through the intravenous

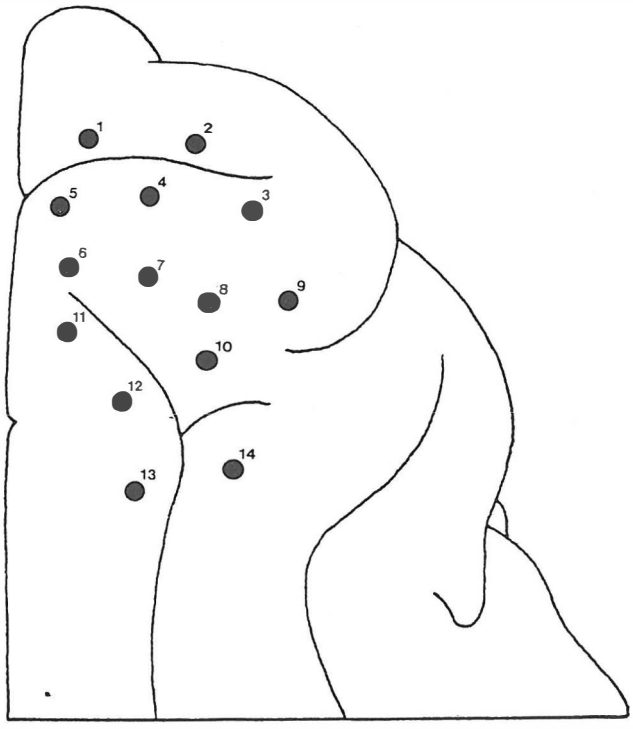


**Fig. 1.** Diagram of the right frontal cortex of the cat. The numbers show the main points of stimulation. The cruciate sulcus lies behind points 1 and 2 and the ansate sulcus almost ends at point 6. Point 8 represents the postcruciate dimple (Pcd).



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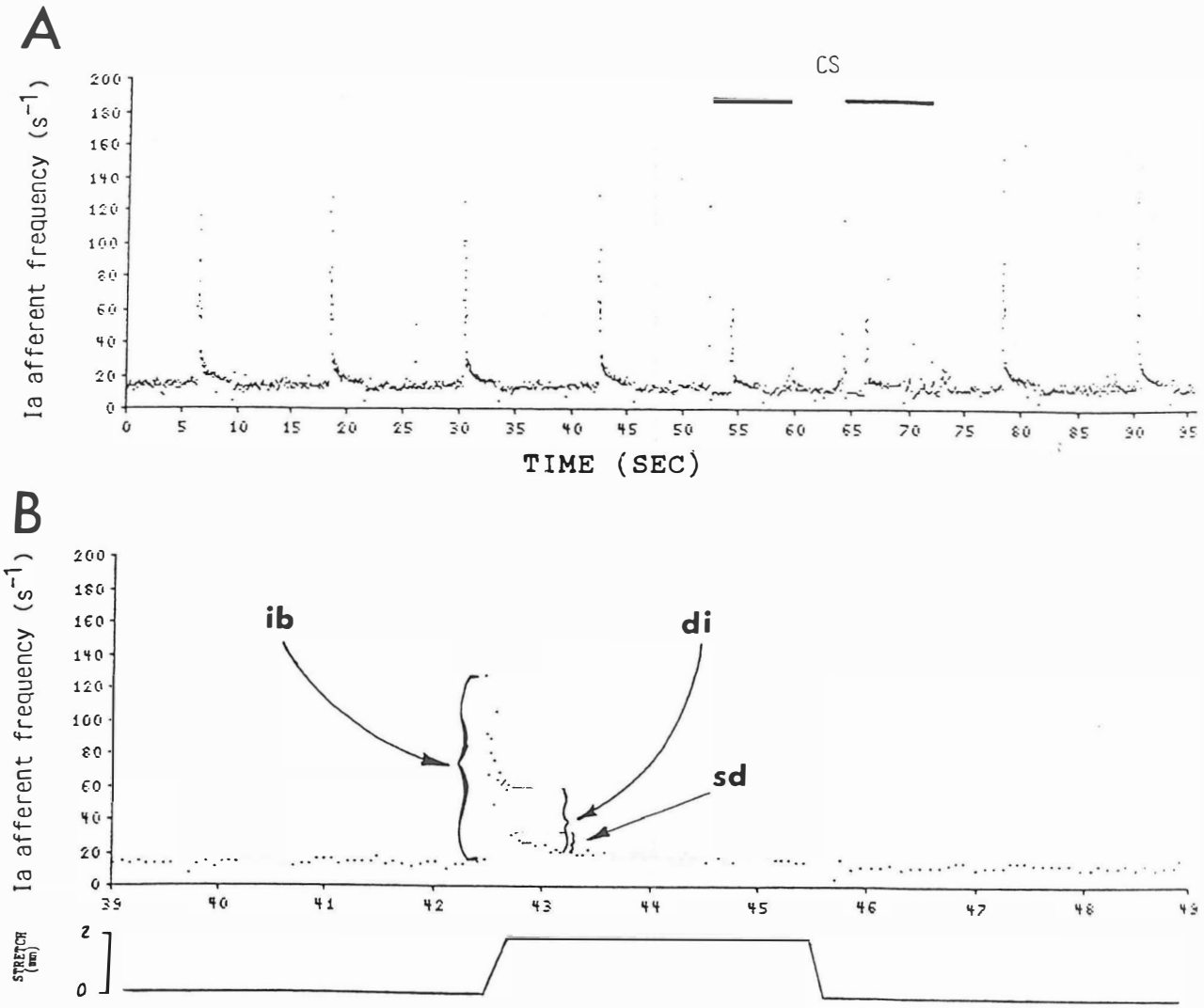


**Fig. 2.** Display of a computer digitized file whose time axis is partially extended in B in order to make measurements of the dynamic index, slow decay and initial burst possible. The lower trace illustrates how the initial burst (ib), the dynamic index (di) and the slow decay (sd) were measured.

cannula inserted into a superficial branch of the cephalic vein of the right forelimb. The right femoral artery was also cannulated; the cannula was passed up the artery until the tip was estimated to be at or near the bifurcation of the aorta in order to monitor blood pressure through a pressure transducer (Elcomatic EM752). The femoral and obturator nerves were cut. Laminectomy at the level of L5 to L7 was carried out. Hindlimb muscles apart from the tenuissimus needed to be denervated.

After preparatory surgery, the animal was transferred to the experimental frame. The stimulating electrodes, and later on, the muscle puller could be used. Following the construction of 37°C parrafin pool in the laminectomized area, the L7 and S1 dorsal roots were cut as close to their entry into the spinal cord as possible. L6 was also cut to prevent any reflex activity taking place. It was essential to keep the ventral roots intact.

Asingle pair of silver electrodes were lowered into the spinal pool and a pair of stimulating electrodes were placed on the tenuissimus nerve. The tenuissimus nerve was stimulated directly and evoked action potentials were sought for in all naturally occurring filaments of L7 and S1 dorsal roots. A stimulus pulse (usually of 0.02 msec duration) was generated through an isolated stimulator (Devices Sales) which was controlled by a digitimer (D4030 Devices Sales). The digitimer also provided trigger and gated pulses for the muscle puller, pulse generator (Neurolog) and also trigger pulse for the oscilloscope (Tektronix). Rootlets which showed regular evoked potentials 1 to 5 msec following the muscle nerve shock were isolated. These rootlets were then



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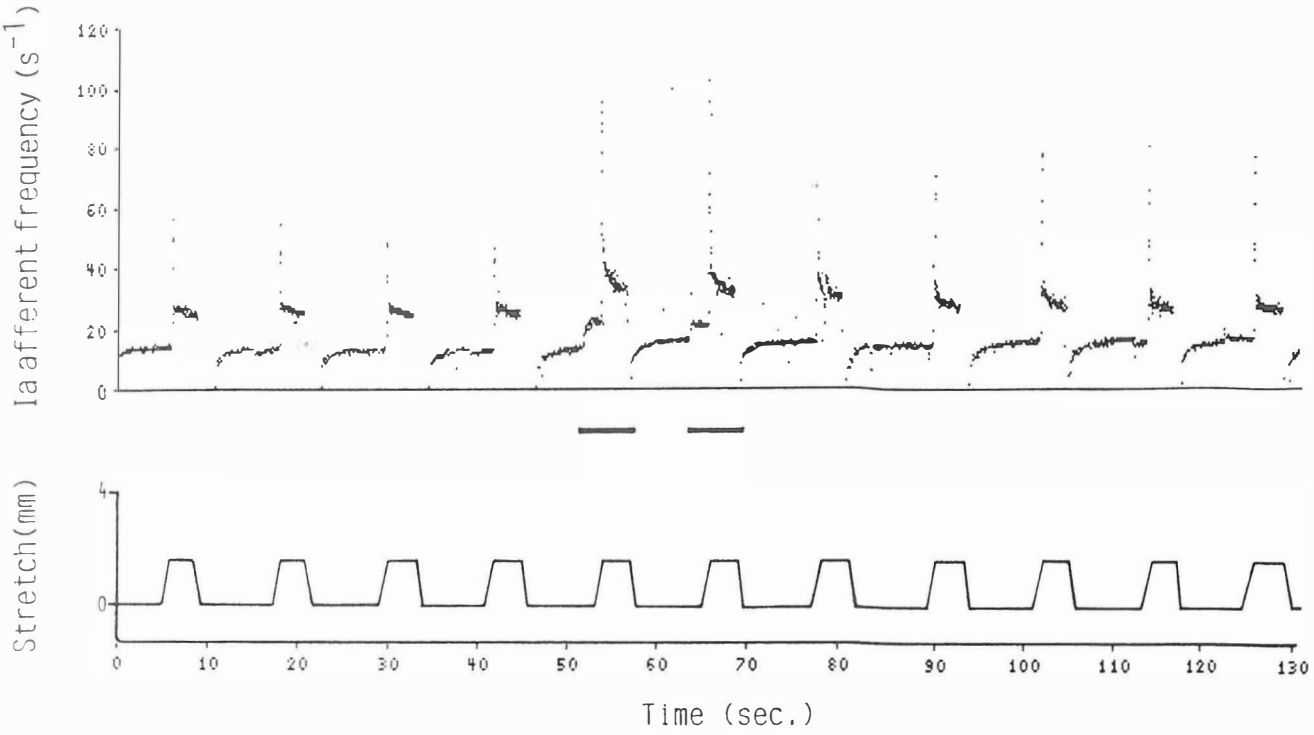


**Fig. 3.** A typical dynamic response obtained from tenuissimus primary afferents on cortical stimulation at an area 1 mm medial to point 5. Stretch: 2 mm. Cortical stimulation shown as solid bar.

subdivided to give filaments which contained only active afferents from the tenuissimus muscle. Occasionally some of these "single fiber" filaments contained other active fibers which were "separated" from the primary fiber by use of the window discriminator. When a single fiber filament was isolated, a tiny piece of coloured silk was tied to the end of it to act as a marker. Primary afferents from the left tenuissimus were distinguished from other afferent fibers on the basis of having conduction velocities over 70 m/sec. No Golgi tendon organ exists in the tenuissimus muscle!

After finding as many tenuissimus afferents as possible, the right sensorimotor cortex was exposed. To accomplish this, with the head of the animal in the head holder, a midline incision was made almost from the root of the nose to the lambdoidal ridge at the back of the head. The skin flaps were then retracted, revealing the temporalis muscle. The right temporalis muscle was dissected free of its origin, scraped from the skull using a scalpel, and retracted. Using a trephine, a 1 cm diameter hole was trephined in the skull parietal bone just lateral to the saggital suture. The disc of bone was removed, and the area of the hole enlarged forward up to the frontal sinus using bone nibblers. Bone wax was applied over the cut ends of the bone to stop bleeding and prevent air and paraffin embolism. A metal loop of 10 cm diameter was mounted on the body frame on top of the skull in order to construct the head parrafin pool. The retracted skin was tied round the metal loop using surgical thread. Liquid paraffin at 37°C was introduced to this pool before any attempts were made to remove the dura; the temperature of the pool was maintained by means of a thermometer and a heating lamp. Points of stimulation were marked with anatomical landmarks and numbered for easier communication. Fourteen points were chosen conventionally, the first two falling forward to the cruciate sulcus and the rest behind it. The postcruciate dimple (Pcd) was numbered 8 (Fig. 1).

Anodal or surface positive pulses of 0.3-3 mA current (rarely up to 4 mA) were applied through the spring-mounted platinum ball electrode, tip diameter 0.6 mm. The other pole of the stimulating electrode was attached to the bulk of the cut temporalis muscle using a pair of crocodile clips. A low stimulus current was chosen at the very beginning and increased if no fusimotor activity was seen. Most animals were responsive to a current of 2.0 mA strength. It was rarely necessary to increase the current to a level above 3.0 mA and usually no responses were obtained below 0.5 mA.

An IBM personal computer fitted with a digital input/output interface card was used to collect the data. The spike-trains to be analyzed were fed into Neurolog units. The time of occurrence of each spike was recorded and stored in a file on the computer's disk for offline analysis and graph prints. Each computer file consists of a response of an afferent to stimulation of a specific point on the sensorimotor cortex



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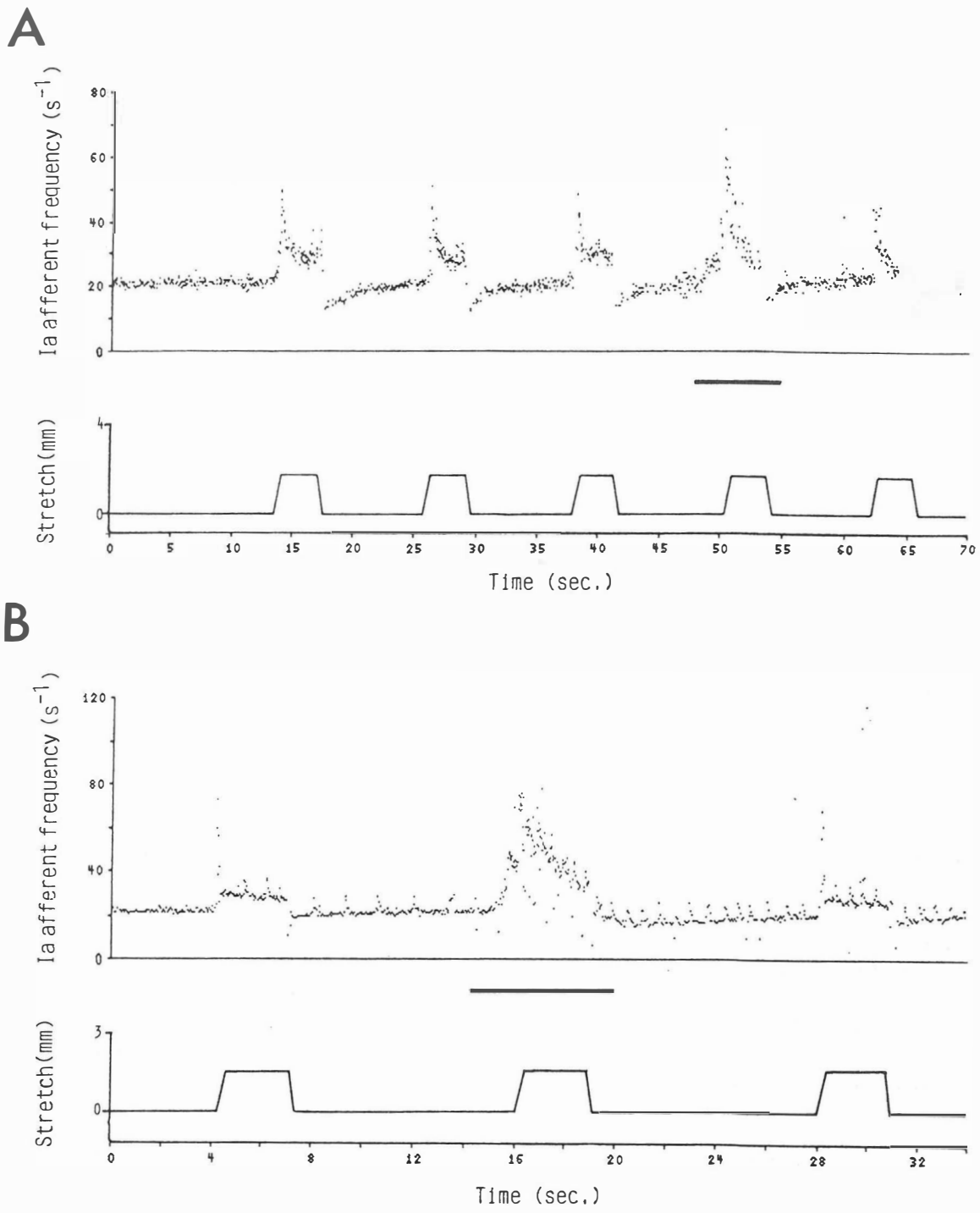


**Fig. 4.** A slight dynamic response of a tenuissimus primary afferent to cortical stimulation at points 6 (A) and 5 (B). Notice how the frequency of spontaneous regular bursting activity of the primary afferent in B increased due to cortical stimulation.

and the preceding and subsequent responses to stretches for comparison. To determine the character of an intrafusal effect (excitatory or inhibitory and if excitatory, whether dynamic or static), it was necessary to measure the "initial burst","dynamic index" and "slow decay" of the afferent response (on a ramp-hold stretch) before, during and after cortical stimulation. In order to measure them precisely, it was necessary to display each response to a ramp and hold stretch with an extended time scale. One example of such work is illustrated in Fig. 2.



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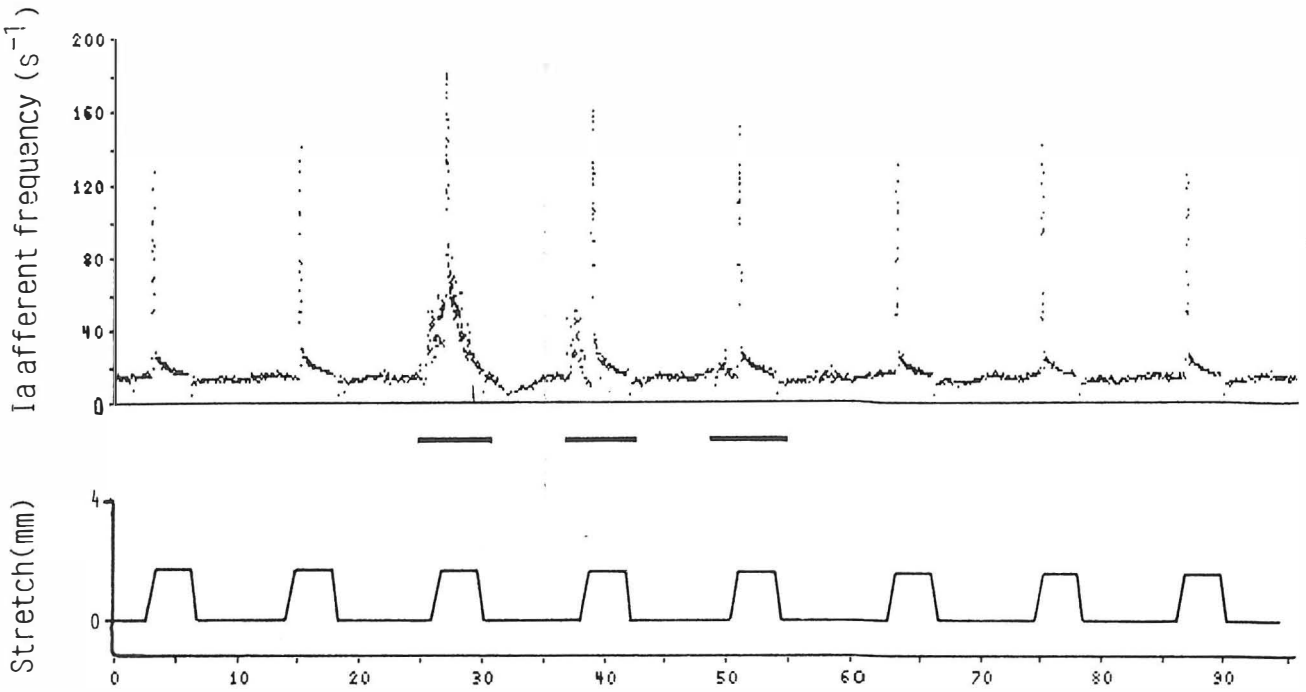


**Fig. 5.** The effect of three consecutive cycles of cortical stimulation of a tenuissimus primary afferent from a chloralose-anesthetized cat. Point of stimulation was on the cruciate sulcus anterior to point 5.

## RESULTS

Most tenuissimus primary afferents were found in L7 and some in the S1 dorsal root. They were identified from other groups of afferents by their fast conduction velocities. If the tenuissimus nerve is stimulated just caudad to its emergence from the sciatic nerve, spikes are picked up in dorsal root filaments within a latency of 1.2-1.8 msec depending much on the distance between emergence of the tenuissimus nerve from the sciatic nerve and the spinal cord, which gets longer if the tenuissimus nerve emerges out low and vice versa. However, the mean latency value was around 1.4 msec for an average distance of 12 cm between the two stimulating and recording electrodes, which gives an average conduction velocity of 86 m/sec.

### A. Dynamic responses

A classic effect is characterized by a rise in static firing at the beginning of stimulation followed by an increase in both dynamic responsiveness to ramp stretch and dynamic index. However, not all afferents show all these criteria collectively and at the same time it would be possible for contraction of dynamic bag ($Db_1$) fibers to occur, the effect of which might be masked by simultaneous contraction of the intrafusal fibers. In other words, depending on the degree of activation of static bag ($Sb_2$) and nuclear chain (Nc) fibers, one or even two criteria might be masked. Stimulation of the contralateral cortex, points 4, 5 and 6, with anterior and medial borders being the ansate sulcus and longitudinal fissure respectively, gave rise to dynamic effects. No dynamic effects were attained on stimulation of other areas within the contralateral sensorimotor cortex except in one case which will be represented later.

In Fig. 3 where the cat was anesthetized with chloralose, the dynamic response is more of a classical type: arise in both static firing of the afferent and the dynamic index. In addition, the inital burst to the ramp stretch is considerably increased. The static firing level stayed high in the following cycles. The point stimulated was one centimeter medial to point 5 with 1.9 mA current strength.

A slight dynamic response was visible during stimulation of points 6 and 5 (Fig. 4). The dynamic index had risen by 5 and 13 ips in both A and B respectively. The initial bursts to ramp stretch and the decay were also increased. What is noticeable is the regular bursts of spontaneous activity in $\Gamma$ motoneurons giving rise to miniature bursts in the afferent firing of the primary. The spontaneous bursting activity seems to have been added to the slow decay, and their frequency increased even after stimulation had stopped. The shift to an increased bursting activity happened in less than



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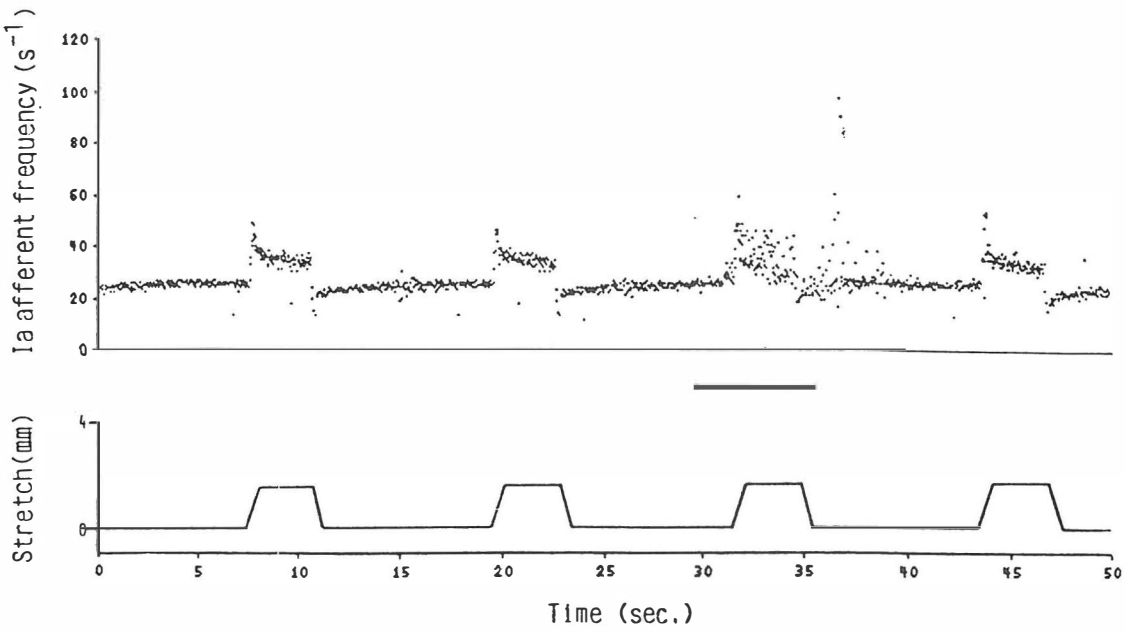


**Fig. 6.** The effect of cortical stimulation at point 5 on the response of a single tenuissimus primary afferent with 3.0 mA.

10 seconds, and that could not have occurred because of a change in anesthetic level.

Repeated cortical stimulation showed that the fusimotor system did not respond identically to the same stimuli in consecutive cycles of stimulation (Fig. 5). The same afferent showed a similar response to stimulation of point 1. It displayed the reduction in responsiveness to stimuli in successive stimulating cycles.

**B. Static responses**

Static fusimotor effects can be brought about by contraction of $Sb_2$ fibers alone, Nc fiber(s) alone or contraction of both. They are well characterized by an increase in mean frequency with reduction or even loss of length sensitivity of the primary ending. There may be an increase in the irregularity of the primary discharge to some degree depending on the recruitment of different types of intrafusal fibers. A high frequency scattery response is expected if $Sb_2$ and Nc fibers are recruited together.

Figure 6 was obtained on stimulation at point 5 with 3.0 mA. The anesthesia level seemed to be the determinant factor in generation of the pattern of responses; for the deeper the anesthesia, the less pronounced the rebound activity. The mean frequency of the afferent response did not change, but the initial burst (from 46 ips to 60 ips) and the dynamic decay (from 3 ips to 7 ips) were slightly increased, although very scattery. The excitation in this pattern of response was reproducible in many experiments. It not only shows the scattery firing of the afferent, but also the fact that cortical stimulation desensitized the spindle to length changes throughout the course of stimulus. It seems that there was some habituation to the stimulus at some level, for the afferent discharge declined progressively throughout the course of stimulation, and even returned back to the previous static firing level during the very last second of stimulation. Scattery firing of the afferent after the stoppage of stimulus might be considered as a reboundactivity of a yet non-specific intrafusal fiber which was possibly inhibited during the course of stimulation.

**C. Inhibition of intrafusal muscle fibers**

Inhibition occurred reproducibly in only one chloralose-anesthetized cat. The cortex was anodally stimulated at point 4 in two consecutive cycles with 1.4 mA (Fig. 7). Afferent static firing dropped and the initial burst to the ramp stretch was reduced remarkably, from 135 ips in the preceding cycle to 60 ips and 52 ips. Dynamic indices were reduced from 38 ips to 14 and 13 ips respectively. The dynamic decay was also reduced from 13 ips to 2 ips in both cycles. In the absence of stimuli in the following cycles(s), the response to the ramp was as good as before. Note the rebound activity of the afferent on the "off" of the stimulus in both cycles. Since EMG electrodes did not pick up anything during the period of stimulation, it seems that some intrafusal fibers were inhibited and the reduced static firing did not occur as a result of unloading of the muscle spindle.

## DISCUSSION

In order to interpret fusimotor changes brought about by stimulating the sensorimotor cortex, one needs a reliable criterion. According to general belief, ramp responses are



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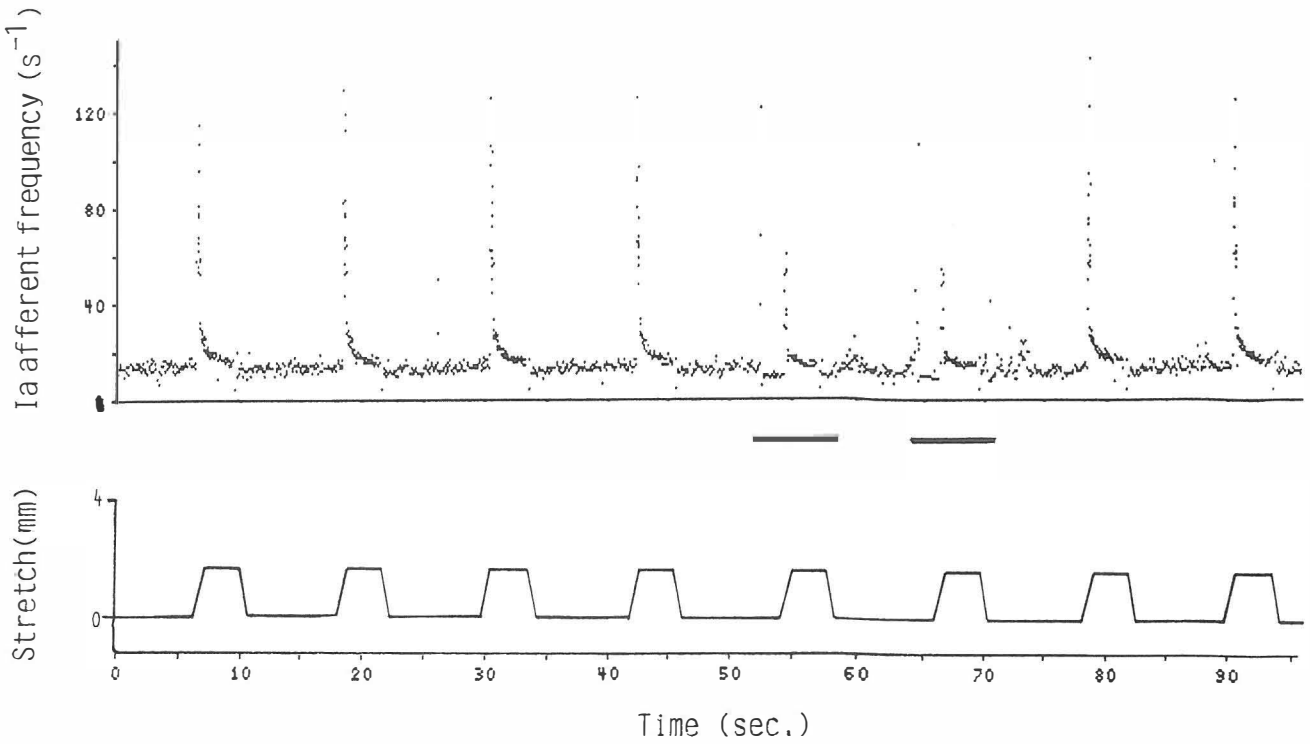


**Fig. 7.** The effect of two consecutive cycles of cortical stimulation at point 4 on the response of a tenuissimus primary afferent

reliable in interpreting fusimotor changes.[25] Therefore, eliciting ramp responses were the sole method used in present experiments. In addition, a one centimeter deep head parrafin pool was made in order to maintain the normal state of the cerebrum. In experiments carried out by Gladden and McWilliam, the cortex was simply kept moist while it was stimulated and at the same time they were not getting consistent fusimotor effects from the same cortical areas in different cats.[11,12] It is believed that $CO_2$ escape from the surface of the pia-covered cortex lowers the extracellular concentration of $pCO_2$ which elicits autoregulatory responses. Autoregulation, in turn, will be reflected as a reduction in the diameter of local arteries and finally deterioration of the cerebral blood flow. Therefore, it seems crucial to prevent $CO_2$ escape from the cortex. Kuschinsky and colleagues covered the cortex with a reasonable amount of warm mineral oil and demonstrated that it could stop much of the $CO_2$ escape.[22] With that simple technique, one can be fairly sure that the blood flow of the cerebrum stays reasonably good throughout the experiment.

**I. Topographical mapping of the sensorimotor cortex**

The fusimotor effects given in the results were obtained exclusively from the contralateral cortex in order to define the cortical regions particularly involved in the production of changes in the fusimotor system of the tenuissimus muscle. It seems that points 4, 5 and 6 fall in an area from where the $Db_1$ fiber can more likely be recruited. Therefore the area might be referred to as the "dynamic area". The boundaries were the cruciate sulcus (anteriorly), the ansate sulcus (posteriorly) and the saggital longitudinal fissure (medially). Laterally, the area extended half-way to the postcruciate dimple. One thing to notice is the small size of the dynamic area compared to the whole area of the sensorimotor cortex from which fusimotor effects can be elicited. Excitatory static effects, on the other hand, can be attained from almost all over the sensorimotor area! There are several reasons for this. One simply is the number of $\Gamma_d$ axons innervating $Db_1$ fibers compared to a larger number of $\Gamma_s$ axons innervating other intrafusal fibers. The ratio of $\Gamma_d$ axons over $\Gamma_s$ fibers is said to be 1/15 or even smaller.[6]

It was not possible to sharply map the areas of the sensorimotor cortex, from where different $\Gamma_s$ axons could be recruited. However, in a broad sense, taking into account the results of direct observation of intrafusal fibers (unpublished personal work), the $Sb_2$ fiber could be recruited from the whole area while chains could either be recruited from the same area or from a more confined area within it. Although Vedel and Mouillac-Baudevin did not give details about their points of stimulation on obtaining excitatory dynamic effects, nevertheless, going only by their graphs, the area seems to be coinciding exactly to our "dynamic area".[30] So does the cortical area of Gladden's experiments in 1982 (personal communication). Unfortunately, the area where different populations of $\Gamma_s$ axons could be selectively recruited could not be mapped with our results.



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**II. Effects of cortical stimulation on primary afferents**

According to our present knowledge, different patterns of influence on intrafusal fibers could be elicited on cortical stimulation: contraction of Nc fiber(s), $Sb_2$, one or more Nc with $Sb_2$, or $Db_1$ in combination with other static intrafusal fibers. Since primary afferents innervate all intrafusal fibers within muscle spindles, the interpretation is very complicated. The degree of contraction of each intrafusal fiber in each case is another variable that makes interpretation even more difficult. Another variable is concomitant contraction of both poles of a spindle or contraction in one pole and inhibition of the other.

Generally our results confirm the existence of a functional relationship between the sensorimotor cortex on the one hand and fusimotor neurons on the other. The study of changes in static discharge and dynamic sensitivity of primaries and their comparison with the effects obtained by Boyd and his colleagues on direct stimulation of fusimotor axons allows to define the action of the sensorimotor cortex on muscle proprioceptors and more particularly its role in modifying the activity of different types of Γ motoneurons, static and dynamic.[5] Almost all the present results obtained from the tenuissimus muscle were of an excitatory type, either static or dynamic. These results are consistent with results introduced by Vedel and Mouillac-Baudevin.[30] They were working on true physiological antagonists and reported different "facilitatory" and "depressor" effects on cortical stimulation depending on the level of anesthesia. They claimed that under "light" anesthesia (i.e., a persistently light level of EMG activity in the neck and shoulder muscles), stimulation of the sensorimotor cortex (and pyramidal tract) strongly reduced static discharge and dynamic sensitivity of soleus primary sensory endings.

However, in "deep" anesthesia (having very slight but permanent EMG activity in the neck and shoulder musculature), stimulation of the sensorimotor cortex produced a reinforcement of both facilitatory effects: one with a dynamic and the other with a static character.

Could primary afferent recordings reveal recruitment of the $Db_1$ fiber alone, without recruitment of static intrafusal fibers? The evidence shows no positive answer to the question. Although some of the afferents did not fire during the ramp release during the course of stimulation (e.g. Figs. 5 and 6), it does not necessarily mean that static intrafusal fibers were not recruited. Emonet-D'enand and colleagues working on peroneus brevis muscle afferents demonstrated that on combined stimulation of $\Gamma_d$ and $\Gamma_s$ axons, the primary discharge goes silent on ramp release.[8] Even on stimulation of a $\Gamma_s$ axon the primary can fall silent during the release. Therefore, if the anesthetic condition of the cat is light enough for $\Gamma_d$ motoneurons to be recruited, then the excitability of the cortex is definitely good enough for $\Gamma_s$ axons (of any type) to be jointly recruited as well.

Figure 6 demonstrates changes of afferent responses that are most probably a consequence of at least one type of static intrafusal fiber, either $Sb_2$ or Nc fibers. According to Boyd, if the afferent goes very irregular, Nc fibers must be mainly responsible with or without concomitant contraction of $Sb_2$ fibers.[6] In most cases recruitment was not very strong and it was mostly Nc and weak $Sb_2$ fiber contraction. As a result of the irregularity of the afferent firing due to static intrafusal fiber contraction, the ramp response is abolished too!

It is believed that cortical stimulation imposes some changes on the sarcomere length of intrafusal fibers during the course of stimulation which is sustained even after the stimulus is off.[9,24] In interpreting the results one should be aware that the after-effect phenomenon exists. It certainly is something that happens during movement in conscious man and is not an artifact that is being produced during laboratory experiments.[16] Gregory and coworkers, in a comparative study attributed the changes in the size of the stretch reflex in a conditioned muscle of the cat and man to the formation of stable cross-bridges between actin and myosin filaments when the muscle was shortened several seconds after a contraction at a long length.[16] Figure 6 may show examples of after-effects during our experiments. It seems that once the intrafusal fiber(s) is contracted and therefore shortens during the course of cortical stimulation, the myosin heads have a chance to form cross bridges and the fiber remains shortened after the stimulation ceases. The cross bridges would not break right away and as a result of their stiffness the primary afferent may have a higher static firing than previously recorded. On the next ramp stretch where the cross bridges are broken apart, they will be formed again at the release of the stretch but only at the usual length. As long as those changes usually disappear at the subseqent ramp stretch, it does not imply on sustained alterations of Γ axon discharge continuing after the cessation of cortical stimulation.

Different anesthetic agents, barbiturates or chloralose in our case, and halothane in other experiments,[30] might account for the great variation of results. In the case of Fig. 7 where some intrafusal fibers were inhibited, the dynamic area was stimulated. However, that does not mean the cats were too light at the time. In fact, in both cases, cats were not light enough to expect $Db_1$ fiber recruitment. Then, why was a clear-cut inhibitory response which was repetitively obtained in Vedel's experiments absent in the present study?[30] The controversy could be tackled by the anesthesia level. If one considers halothane to be one of those agents whose effects rapidly disappear, allowing the animal to become light quickly, then it may be rightly argued that in the "light anesthetic" state in Vedel's experiment, the cortex was actually not in a depressed condition, but rather in an excited one. Therefore, stimulation of the already excited cortex might be expected to have an opposite effect on volleys going corticofugally down to the Γ motoneuron population in the



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spinal cord. One possible mechanism at the central level might be due to the activation of a cortical inhibitory mechanism, which itself is one of the components of the cortex. It is worth noting that the evidence which illustrates that it does not take much time for the cat under halothane anesthesia to lose the desired and necessary surgical anesthetic state (stage III of Guedel[17]) has been given by Winters et al.[32] Halothane, according to them, is believed to be one of those anesthetic agents that induces stage III of anesthesia directly from stage I (excitation) without going to stage II (delirium).

Moreover, what is important to note is the ability of the sensorimotor cortex to inhibit as easily as to excite, providing that the cortex is in the proper state of excitation. This concept is confirmed both by their and my results as well as others.[11-13] Having in mind that only one cat showed inhibitory examples, one can assume that the "dynamic area" is not exclusively an excitatory area, and in more physiologic conditions, it might be able to excite one set of $\Gamma_s$ axons and inhibit others.

Linked to the consideration of inhibitory responses, the phenomenon of rebound activity should be discussed. This phenomenon is evident in some of the figures occurring as the "off" response. It certainly signifies inhibition of some intrafusal fibers within the corresponding muscle spindle; however, this inhibition is either not strong enough to reduce the efferent discharge drastically during the cortical stimulation, or is masked by powerful recruitment of other intrafusal fibers (e.g. $Sb_2$ fiber). It could even be caused by cortical inhibition of an active $\Gamma$ axon innervating one pole of the spindle in which another $\Gamma$ axon(s) innervating the opposite pole has been excited. However, it is thought that slight inhibition of a $\Gamma_s$ axon supplying Nc fibers may account for this type of change because the rebound activity results in very erratic discharges such as one might expect if chain fibers were contracting.

One limitation of the afferent recording technique is that it can not demonstrate any slight activation of bag fibers. Boyd claimed that because there are no action potentials in bag fibers, if they are stimulated below 10-15 impulses per second, they do not contract.[3] In other words, if by any means (e. g. cortical stimulation) fusimotor neurons become slightly active (i.e. with a frequency of less than 15), afferent recording will not show it.

**III. Physiological significance of central control**

It is well established by now that muscle receptors as well as joint and skin receptors are capable of producing a sense of kinesthesia. The cortex is one of the structures that is believed to have access to both incoming information from primary and secondary afferents, relaying information about the instantaneous length of the muscle, and the ability to adjust spindle afferent sensitivity via outgoing impulses through $\Gamma$ motoneurons to intrafusal fibers. However, it is much more complicated to use information from primary afferents compared to that of secondaries because firstly primary afferent discharges originate from all intrafusal fibers which are under the influence of at least two main $\Gamma$ populations, static and dynamic. Secondly, it is dependent on the velocity and the direction of movement. Thirdly and more importantly, is the fact that the primary afferent discharge is a nonlinear response to length changes. On the other hand secondaries give a much simpler signal to the brain and this has two advantages: firstly that it is much less affected by movement and secondly it is influenced only by $\Gamma_s$ axons. Moreover, it is established by now that Nc fibers are most effective in providing a large positive bias to the discharge of both primary and secondary endings. Therefore, having information about the ongoing activity of $\Gamma$ axons (innervating Nc fibers) and secondary's discharges of a muscle, it is fairly easy to work out the length of the muscle (which is the sense of kinesthesia). Although no particular structure in the central nervous system is known to do so, the role of the sensorimotor cortex may not be ruled out.

It has been shown that $\Gamma_d$ and $\Gamma_s$ axons affect different components of the primary and the secondary responses to stretch.[4,5] It is believed that $\Gamma_s$ axon stimulation increases the length sensitivity of the secondary ending,[20] while $\Gamma_d$ axon stimulation can increase that of the primaries. A recent collaborating work between Glasgow and Paris (personal communication) shows the same phenomenon on the primary afferent discharge in the tenuissimus muscle of the cat. Note that the increase in the length sensitivity of the primary afferent did not last to the end of the $\Gamma_d$ stimulation, apparently due to adaptation. If the matter of adjusting sensitivity to length changes is being taken care of at the level of the spinal cord (in spinal reflexes), one can not imagine why would there be a necessity for an exact duplication in the sensorimotor cortex rather than dedicate more sophisticated functions to the higher central nervous system. What then could be the role of the cortex in motor control? Is it capable of over-riding $\Gamma$ motoneuron activity in the spinal cord or can it only adjust the fusimotor balance as the cerebellum does for alpha motoneurons? Some of the results support the former idea and some the latter. For instance, on occasions where there was only facilitation of the response to the peripheral stimulus (e.g. ramp hold stretch) and also a simultaneous increase in static firing level of spindle primary afferents, the cortex is rather adjusting the activity of spinal $\Gamma$ motoneurons. However, when the usual response to the peripheral stimulus is abolished (the physiological importance of which is unknown to me), then the cortex is revealing its ability to override the ongoing activity of spinal $\Gamma$ motoneurons. With the above examples it seems that the cortex might be able to do both. However, the above statements can be criticized in that "in physiological conditions, are the cortical neurons subject to as strong a stimulus as we applied during the cortical stimulation (200 Hz and an average current of about



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2.0 mA), while at the same time the spinal Γ motoneurons are firing at a low level, with an average primary afferent frequency of about 25 Hz?" Whatever the real answer is, one can assume that with the presence of at least three types of Γ motoneurons in the spinal cord there should certainly be a center to "balance" the activity of the three, since an over-activity of any of them would bias wrongly the ascending information to the higher central nervous system. Why could the center not be the sensorimotor cortex, since it seems to be able to have influence on spinal Γ motoneurons and at the same time receives information from muscle spindle afferents?

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