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ACTIVATION OF THE GAMMA MOTOR SYSTEM IN THE  
CAT FOLLOWING SELECTIVE STIMULATION OF THE  
MOTOR CORTEX AND THE PYRAMIDAL TRACT

by

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B.S., Old Dominion University, 1965

M.S., University of Richmond, 1967

Thesis

submitted in partial fulfillment of the requirements for the

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This thesis by James Elliott Forbes is accepted in its present form as satisfying the thesis requirement for the degree of Doctor of Philosophy.

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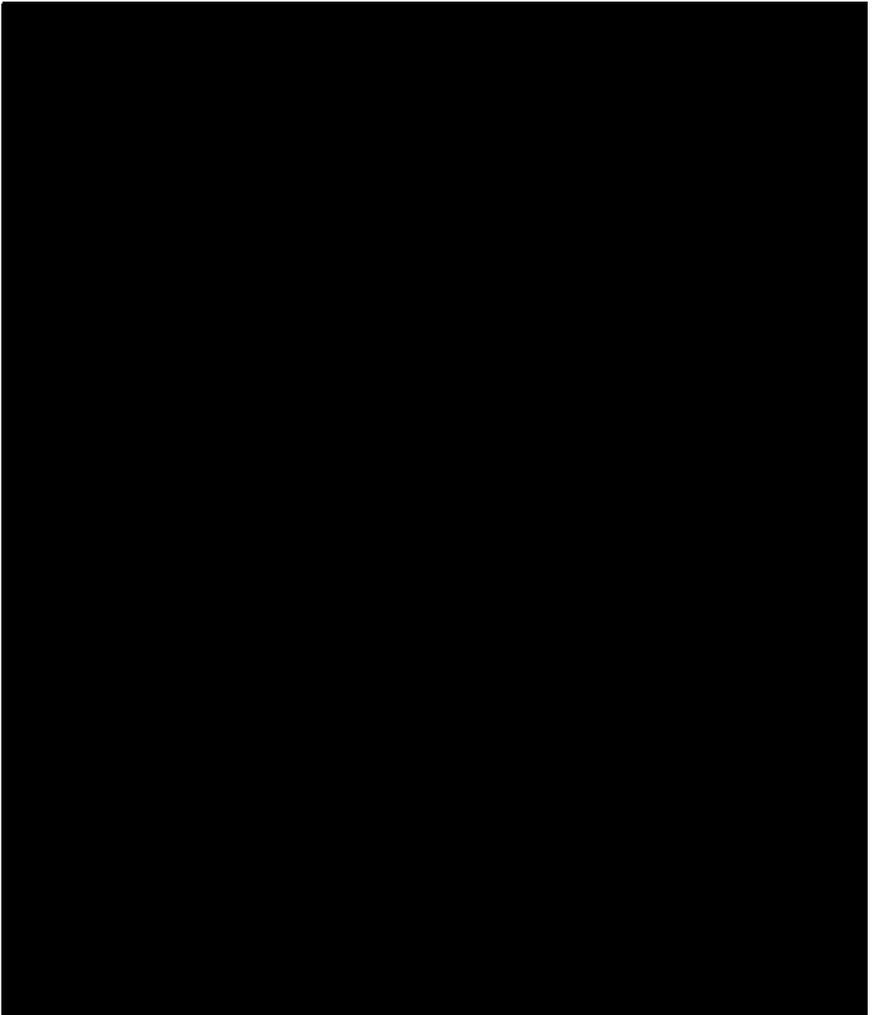
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CURRICULUM



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## TABLE OF CONTENTS

	Page
Introduction	I
Review of the Literature	3
A. Direct Evidence for Cortical and Pyramidal Control of the Gamma Motor System	3
B. Indirect Evidence for Cortical and Pyramidal Control of the Gamma Motor System	5
1. Lesion of the Pyramidal Tract	5
2. Lesion of the Cerebral Cortex	9
3. Electroencephalograph Studies	16
C. Morphology of the Muscle Spindle	16
1. Intrafusal Muscle Fibers	16
2. Sensory Innervation	18
3. Motor Innervation	19
D. Physiology of the Muscle Spindle	20
1. Afferent Innervation	20
2. Motor Innervation	23
E. Anatomy of the Cerebral Cortex	24
F. Anatomy of the Pyramidal Tract	25
1. Definition	25
2. Origin	27
3. Anatomical Path	30
4. Termination	31
5. Betz Cells	35
Method and Materials	36
A. Anesthesia	36
B. Pyramidal Tract Exposure	36
C. Exposure of the Spinal Cord	37
D. Exposure of the Cerebral Cortex	38
E. Denervation of the Hindlimb	38

F. Recording From $I_a$ Afferent Fibers	39
G. EMG Recording	40
H. Stimulation of the Pyramids	41
I. Stimulation of the Motor Cortex	41
J. Parameters of Electrical Stimulation	42
K. Deafferentation	42
L. Reproduction of Data	43
Results	44
A. Alpha and Gamma Motor Maps	44
B. Anterior Tibialis	44
C. Soleus	67
D. Gastrocnemius	69
Discussion	80
A. Anesthesia	80
B. Motor Map	81
C. Flexor Facilitation Versus Extensor Inhibition	83
D. Servo-Control Theory	88
Summary	93
Bibliography	94

## LIST OF FIGURES

	Page
Figure 1. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	47
Figure 2. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	49
Figure 3. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Pyramidal Stimulation	51
Figure 4. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Pyramidal Stimulation	53
Figure 5. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Pyramidal Stimulation	55
Figure 6. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	57
Figure 7. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	59
Figure 8. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	61
Figure 9. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Pyramidal Stimulation	63
Figure 10. Response of Anterior Tibialis I <sub>a</sub> Afferents and EMG to Cortical Stimulation	65
Figure 11. Response of Class I Soleus I <sub>a</sub> Afferents and EMG to Cortical and Pyramidal Stimulation	70
Figure 12. Response of Class II Soleus I <sub>a</sub> Afferents and EMG to Cortical Stimulation	72
Figure 13. Response of Class II Soleus I <sub>a</sub> Afferents and EMG to Cortical Stimulation	
Figure 14. Response of Class II Soleus I <sub>a</sub> Afferents and EMG to Pyramidal Stimulation	76

Figure 15. Response of Class III Soleus  $I_a$  Afferents and EMG to Cortical and Pyramidal Stimulation

## INTRODUCTION

Until the mid to late 1800's, the cerebral hemispheres were considered to be only the sites of mentation and intellectual activity. On the basis of his observations of epileptic patients, Hughlings Jackson (cited from Walshe, 1935a) theorized that the cerebral hemispheres were also sites of motor control and sensory experience. The electrical excitability of the cerebrum was first demonstrated by Fritsch and Hitzig in 1870. Since then the motor representation of the various muscles has been mapped out and the types of movement in response to stimulation of the motor cortex and the pyramidal tract have been thoroughly studied.

Mammalian muscles contain sensory receptors which supply the central nervous system information on muscle length and rate of change of muscle length. One important feature of muscle spindle receptors is that they receive an efferent (gamma) innervation from the central nervous system through which their sensitivity to stretch can be changed. There is little direct evidence for pyramidal and cortical control of the gamma motor system on the basis of experiments using electrical stimulation. Most of the evidence for the control of the gamma motor system by the cortex and pyramidal tract stems from studies in which the motor cortex or pyramidal tract are ablated or lesioned and the resulting changes in posture are observed. The basis of these posture changes is the stretch or myotatic reflex (Liddell and Sherrington, 1924, 1925). When the muscle belly or tendon is lengthened, the receptor region of the muscle spindle is stretched. This results in a volley of  $I_a$  impulses being sent into the spinal cord to the motoneurons of the stretched muscle. As a result of the  $I_a$  excitation, the motoneurons discharge causing the stretched muscle to contract, tending to return to its original

length .

Sherrington (1898, 1915, 1924) demonstrated the stretch reflex to be the basis of posture and resistance to gravity. Here the stretch is due to gravity and the resulting contraction of the extensor muscles results in the maintenance of an upright posture. The sensitivity of the spindle to stretch, hence the response of the muscles to stretch, can be influenced by the presence or absence of gamma motor activity. An increase of gamma motor activity can result in an increased stretch reflex and pathologically to rigidity or spasticity, while a decrease in gamma activity can result in hypotonus. The literature in this area is confusing. The clinical data suffers from the lack of precision in lesion localization and absence of detailed histological followup of the patients. Though the ability to produce more discrete lesions is greater in experimental animals, damage to areas peripheral to the intended lesion site cannot be prevented. Thus, these lesions are not truly restricted to a well-defined site. Furthermore, in many studies there have not been adequate descriptions of the areas lesioned or ablated (Fulton, 1935).

The purpose of the present experiments was to study the effect of electrical stimulation of the motor cortex and pyramidal tract on the gamma motor system of the cat .

## REVIEW OF LITERATURE

### Direct Evidence for Cortical and Pyramidal Control of the Gamma Motor System

Granit and Kaada (1953) first demonstrated cortical and pyramidal influence on the gamma motor system in cats by using electrical stimulation. They reported that a brief initial inhibition of gastrocnemius muscle spindles was followed by facilitation as a result of cortical stimulation. In one experiment the stimulating electrode was placed in the medullary pyramid, stimulation of which produced a transient facilitation of spindle discharge in the gastrocnemius muscle. Even after bilateral frontal cortex ablation pyramidal tract stimulation produced the same results. Gamma activity in ventral root filaments was also facilitated by stimulation of the pyramidal tract.

Mortimer and Akert (1961) studied the cortical influence on gamma (fusimotor) motoneurons in cats by recording fusimotor units from L<sub>7</sub> ventral root filaments but the muscles innervated by the fusimotoneurons were not identified. A facilitation of gamma activity was reported as a result of stimulation of the motor cortex. The cortical localization for a given fusimotor neuron was reported as being discrete. The same area was also an area of representation for the alpha motoneuron fibers in the same ventral root filament.

In a similar study (Kato et al. 1964), the effect of pyramidal tract stimulation on gamma motoneurons was studied in pyramidal cats in which all of the structures of the pons and medulla and the cerebellum were removed. Inhibition was found in 10 gamma motoneurons, facilitation in 4 and no response in 16. Again the muscles in-

nervated by the gamma motoneurons were not identified.

Fidone and Preston (1967, 1969) studied fusimotor neurons in which the muscle innervation of the gamma motor units was known. The pericruciate cortex of cats was stimulated and the resulting effects observed in the muscles of the ankle. In the flexors,  $1/3$  of the fusimotor neurons were inhibited and  $2/3$  were facilitated. With the extensors the results were just the opposite,  $1/3$  of the fusimotor neurons were facilitated and  $2/3$  were inhibited. In general, gamma motoneurons with higher spontaneous firing rates were found to be inhibited by cortical stimulation while units which had lower spontaneous firing rates were facilitated by cortical stimulation. Spontaneous changes in firing rate did not alter the response to cortical stimulation.

Pyramidal tract control of fusimotor neurons of cat forelimb extensors was studied by Yokata and Voorhoeve (1969), who monitored the gamma motor system via  $I_a$  afferents in teased dorsal root filaments. In all of the  $I_a$  afferents studied, there was an increase in the spontaneous firing rate upon stimulation of the motor cortex.

Vedel and Mouilloc-Boudevin (1970) conducted similar studies on the soleus and anterior tibialis muscles of the cat. The results of both pyramidal and cortical stimulation were the same for the soleus muscle spindles. Light anesthesia resulted in a cessation in spindle firing while deep anesthesia resulted in increased firing. In recording from anterior tibialis spindles both cortical and pyramidal stimulation resulted in an increase in spindle firing rate at all levels of anesthesia. Similar results have reported for the ankle flexors and extensors of the baboon (Grigg and Preston, 1971).

## Indirect Evidence for Cortical and Pyramidal Control of the Gamma Motor System

### A. Lesions of the Pyramidal Tract

Some reports have indicated that pyramidal tract lesions resulted in initial decreases in muscle tone followed later by increased tone. Walker and Richter (1966) found in the monkey that struggling elicited hypotonus and decreased tendon jerks for the first two months. After this time the flexors of the hip and elbow became hyperactive with an increase in flexor resistance. One human case of vascular lesions was followed by Brown and Fang (1961). Initially the patient had a hypotonic paralysis of the arm and leg with the arm being more affected. Tendon and abdominal reflexes were absent on the side contralateral to the lesion. After two months the arms were reported to be held in flexion and two years later the patient was reported showing increased tendon reflexes and spastic paralysis. Meyer and Herndon (1962) reported similar results in a case of bilateral infarct of the pyramidal tract in man. Initially there was a decrease in tone and the tendon jerk was depressed, then absent. Four weeks later the reflexes began to appear, and hyperreflexia was present at six weeks.

Several studies report that pyramidal tract lesions result only in hypertonus. Ranson (1932) and Ranson et al. (1932) reported that cats suspended in a hammock held their limbs in an extended position. After sectioning the pyramidal tract the limbs contralateral to the lesion showed an increase in extensor tonus, the forelimbs being more affected than the hindlimbs. The rigidity was seen only when the cat was suspended in the hammock and no extensor tonus was present when the cat was in standing or walking positions. Marshall (1933, 1934) reported similar findings and further found

that the rigidity could be enhanced by ventral stimuli and depressed by dorsal stimuli. A decrease in flexor tone was reported. In time, the tone returned to normal but the flexor hypotonus outlasted the extensor hypertonus. There are several subsequent reports of similar tonus changes with the hindlimbs being affected more than the forelimbs, but a return to normal tone has not been observed (Walker and Fulton, 1938; Liddell and Phillips, 1944; Lance, 1954; and Chambers and Liu, 1957). In man, Langworthy (1932) reported the distal muscles to be affected more than proximal muscles, with hypertonus in the flexors of the arm and extensors of the leg. The tendon reflexes were increased, clonus was present and the hypertonus was of the clasp knife type.

As early as 1919, Walshe maintained that pure pyramidal tract lesions did not result in hypertonus. A decrease in flexor tonus was reported in the rat by Boron (1934) with the forelimbs being affected more than the hindlimbs and Hines (1937) claimed that pyramidal tract lesions resulted in the absence of clonus, hypotonus in all muscles, and reduced tendon reflexes. In Tower's (1935, 1940) classic studies the pyramidal tract was sectioned in the cat and monkey. Cats suspended in the air showed an extension of the limbs contralateral to the lesion while the normal limbs were slightly flexed. The basic postural mechanism of extension was intact but flexion as a part of preparation or adjustment was absent. In the scratch reflex, the postural component was intact but the phasic flexor component was diminished or absent. There was an increase in the threshold for the flexor reflex and in the extensors clonus, hypertonus and clasp knife phenomena were absent.

In the monkey, pyramidal tract section resulted in hypotonic paresis with

diminished muscle tone, diminished cutaneous reflexes, and tendon jerks that were full but slow and of high threshold. The hypotonus was present in all muscles except those of the neck and thorax and there was no condition of the animal in which the hypotonus was absent. Reflexes had increased thresholds, were sluggish, weak and unchecked by antagonist with clonus and clasp knife resistance absent. Bilateral pyramidal lesions produced similar results except that the effects were bilateral and the axial muscles were involved. All muscles were reported as being hypotonic with the flexors and extensors affected equally. Similar results were reported in the monkey by Liu and Chambers (1964).

Pedunclectomy in the monkey was reported (Cannon et al. 1944) to produce hypotonus with decreased tendon reflexes. Hypotonus as a result of pedunclectomy was also reported in several other studies, but tendon reflexes were reported to be initially depressed, then normal (Bucy and Keplinger, 1961; Bucy et al. 1966) or permanently depressed (Gilman and Marco, 1970). Gilman and Marco (1970) found the stretch reflexes to have higher thresholds and suggested the hypotonia was due to a decreased stretch reflex because of decreased fusimotor activity.

In man pedunclectomy was used by Putman (1938) to alleviate the rigidity of Parkinson's disease. The same method was used by Bucy and Keplinger (1961) for tremor reduction resulting in an initial flaccid paralysis followed by some increase in tone and tendon reflex activity. In man pedunclectomy does not result in hypertonus (Bucy and Keplinger, 1961; Bucy et al. 1964; Maspse and Pagni, 1964) or any clasp knife phenomena (Bucy et al. 1964).

Section of the corticospinal tract in monkeys by dorsal chordotomy in the cervi-

cal and thoracic regions (Cannon et al. 1943) has been reported to result in hypotonus and sluggish tendon reflexes.

Laursen and Wiesendanger have argued against the presence of tonus changes as a result of pyramidal tract lesions. Since Tower's studies (see page 6) caused considerable damage to the medial lemniscus, the experiments were repeated by Laursen and Wiesendanger in 1966. The limbs contralateral to the sectioned pyramidal tract were used less in grooming, play, feeding, struggling and fighting but when used, were used normally. Posture was reported to be normal except when the medial lemniscus was damaged and then the deficits lasted only a week. When the cats were suspended in a sling the normal limbs were flexed while the limbs contralateral to the lesion were extended. The flexor reflex of the affected limb was weak or absent but all of the extensors showed a normal stretch reflex. These abnormalities were the same if the pyramidal tract was uni- or bilaterally, partially or completely sectioned. Electromyogram (EMG) recording from the cats suspended in slings showed that EMG activity was present in the extensors of all limbs. EMG activity was present in the flexors of the normal side but was absent in the flexors contralateral to the lesion, suggesting that the extension of the limbs was due to a lack of flexor activity. The scratch and stretch reflexes were normal in all muscles. In later studies of cats (Laursen and Wiesendanger, 1967) and monkeys (Laursen, 1972) no abnormalities were reported after the first few post operative weeks. Tower (1942) proposed that pyramidal tract lesions which resulted in hypertonus, increased postural reflexes, increased tendon reflexes, clonus and clasp knife resistance were the result of the lesions extending into the pons at the midline. On the other hand, Laursen and Wiesendanger (1966) felt that damage

to the medial lemniscus, which had generally been extensive in the experiments of most investigators, had been responsible for the postural defects.

In summary, lesions of the pyramidal tract have been reported to produce hypotonus or no tonus changes, although there are a few reports of hypertonus resulting from this procedure.

#### B. Lesions of the Cerebral Cortex

In the opossum (Rogers, 1924) and rabbit (Swank, 1936) lesions of the motor cortex were reported to produce no hypertonus. In the monkey, Horsley and Schafer (1888) reported a decrease of reflexes with ablation of the motor cortex while King (1927) observed no tonic or postural changes in the cat. On the other hand, removal of the frontal area in the cat by Warner and Olmsted (1923) was reported to result in extensor rigidity. The responsible region of the frontal area was later reported to be the tissue about the cruciate sulcus (Olmsted and Logan, 1925). Initially there was a hypotonic paralysis, and in several days the extensors became hypertonic and the flexor reflex threshold was increased. Only during nervous agitation was the extensor hypertonus not evident. Ablation of the upper anterior margin of the cruciate sulcus produced similar results and if the cruciate sulcus was ablated toward the midline in the frontal area no paralysis was produced but the extensor rigidity was still produced. Extensor hypertonus was also reported in the rabbit, dog, and cat by Laughton (1928). In the rabbit the hypertonus was permanent, though the degree diminished with time. The hypertonus was present in the hip, knee and ankle in the dog, and the forelimbs were affected more than the hindlimbs in both the dog and cat. Removal of small bits

of the anterior sigmoid gyrus in the cat was reported to produce extensor hypertonus by McKibben (1929) and McKibben and Wheelis (1932) although the region responsible, the anterior sigmoid gyrus at the junction of the saggital sinus and the cruciate sulcus was better defined by Langworthy (1928) who also reported that ablation of other sigmoid areas did not produce hypertonus. Removal of the posterior sigmoid gyrus in the dog was reported by Woolsey (1933) to produce marked contralateral extensor hypertonus of a clasp knife type with equal fore- and hindlimb distribution. The hypertonus was especially pronounced when the animal was suspended in a hammock.

The numerous cytoarchitectonic maps of the primate cerebral cortex (Campbell, 1905; Brodman, 1909; Vogt and Vogt, 1911, von Economo, 1927) led to attempts to find special functions for the various areas of the cortex. Brodman's designations, the most commonly used in the literature, will be used in the present discussion. Of particular interest is area 4 (the motor area) and area 6 (the premotor area).

Lashley (1924) bilaterally removed the arm, leg and face areas from the monkey cortex and reported the production of hypertonus, while removal of the right motor area resulted in a hyperextension of the left limbs. Fulton (1932) reported that complete ablation of the frontal area produced no permanent alteration of posture nor did the addition of area 6 lesions or unitary area 6 lesions. However, Jacobsen (1933) reported hypertonic paresis with area 6 ablations and hypotonic paralysis with area 4 ablations. In 1933, Bucy and Fulton reported motor cortex ablation which produced hypotonic paralysis while ablation of the premotor cortex resulted in hypertonus. Combined bilateral motor and premotor ablations resulted in a thalamic preparation similar to that seen with decortication. Serial ablations of area 4 followed by abla-

tions of area 6 (Fulton and Kennard, 1933; Kennard and Fulton, 1933) resulted in an initial hypotonic paralysis followed by hypertonus after the removal of area 6. Ablation of area 6 in the left cortex resulted in a hypertonic paralysis of the right limbs which decrease as voluntary power returned. Subsequent ablation of the right premotor area resulted in the reappearance of hypertonus in the right limbs and hypotonus followed by hypertonus in the right limbs. After two weeks the hypertonus disappeared and it was concluded that area 4 lesions produced hypotonus and depressed tendon reflexes while area 6 lesions produced hypertonus and increased tendon reflexes of a transient nature, with the duration of the symptoms being greater with bilateral lesions. Further studies by Fulton (Fulton, 1934, 1935, 1936; Bieber and Fulton, 1938) confirmed that lesions of the premotor cortex resulted in hypertonus and increased tendon reflexes. In 1934, Fulton and Kennard again performed serial ablation experiments. Ablation of the motor cortex resulted in hypotonic paralysis which became hypertonic with subsequent premotor ablations. Forced grasping and restlessness was produced when the cortex anterior to the motor area was ablated. Only with subsequent motor lesions did hypertonus appear. In another series of experiments the left motor cortex was ablated, resulting in hypotonic paralysis and forced grasping in the right limbs. The left premotor cortex was then ablated resulting in the appearance of a mild hypertonus in the right limbs. Subsequent to these two ablations the right premotor cortex was ablated, resulting in enhancement of the grasp reflex in the right limb and the appearance of a temporary grasp reflex in the left limbs. After the final ablation, right motor area, hypertonus appeared in all limbs. In a third series, serial ablations of the left motor and premotor areas result-

ed in a slight temporary hypertonus in the right limbs. The subsequent removal of the right motor and premotor areas resulted in the appearance of a permanent hypertonus in all limbs. In 1935, Kennard again reported hypotonic paralysis after motor cortex ablations and hypertonus with combined motor and premotor ablations. There was no additional tone change if the cortex anterior to the premotor area (i.e. the frontal area) was subsequently ablated.

Welch and Kennard (1944) reported that lesions of area 4 in baboons resulted in hypertonus of the proximal joints and hypotonus in the distal joints. Combined area 4 and 6 lesions resulted in hypertonus and increased tendon reflexes and area 6 lesions produced a spastic paresis of the flexors. Lesions of areas 3, 1, 2, 5 and 7 did not result in hypertonus and may produce hypotonus (Kennard and Kessler, 1940).

While Fulton and his associates advocated the view that lesions in area 4 caused hypotonus and lesions in area 6 caused hypertonus, Walshe (1935a,b) felt that in man lesions of the motor cortex resulted in hypertonus and increased tendon reflexes and suggested that the terms motor and premotor were not valid since the motor area responsible for hand and finger movement was anterior to the Betz cell area and resembled premotor cortex. Further hypotonic paralysis and depressed tendon reflexes were present only in acute preparations and the presence or absence of hypertonus depended on the rate of its development and not on the location of the lesion site, and that the physiological motor cortex included much of Fulton's premotor area. Walshe also proposed that the only consistent symptom of premotor lesions was forced grasping.

Hines (1936) reported that ablation of a strip of tissue 3 mm wide at the center

of the precentral gyrus at the border of area 4 and 6 could result in brisk tendon reflexes, hypertonus especially in the extensors and clonus. This strip of tissue was called area 4s or the strip area (Hines, 1937).

Ablation of area 4 in the monkey by Denny-Brown and Botterell (1948) produced hypertonus in the flexors of the knee, hip, ankle and elbow with decreased tendon reflexes. By the fifth day the hypertonus of the legs was absent while the tendon reflexes became brisk. On the 12th day a clasp knife resistance developed in the finger extensors, but by the 54th day no abnormality in posture or tone was present. A more extensive area 4 lesion encroaching on the caudal border of area 6 produced hypertonus in the flexors of the toes, ankle, elbow and fingers accompanied by brisk tendon reflexes. By 21 days the hypertonus had extended to the knee, hip, and wrist and was greater in the flexors than the extensors. Ablation of area 4s was found to produce moderate spasticity in the ankle, knee and elbow for the first two days and by the third day the knee extensors and arm flexors were hypertonic, after which there was no abnormal posture, clonus or reflexes. An incision between area 4 and area 6 produced some slight hypertonus in the ankle flexors and the knee extensors along with increased tendon reflexes, but these symptoms lasted for only 11 days. Ablation of area 6 produced hypertonus of the ankle flexors and of the flexors and extensors of the knee and elbow, which lasted for 6 days. The hypertonus was reported to be of a plastic type and tendon reflexes were not increased.

Many of the early motor maps had the motor region closely identified with area 4. Woolsey and his associates (Woolsey and Settlege, 1950; Woolsey et al. 1952) produced a series of motor maps in which the motor area was greatly extended into the

premotor area of Fulton and there was actually no motor-premotor separation. This was in accordance with Walshe (1935a,b). Travis and Woolsey (1952) bilaterally ablated the precentral and supplementary (Woolsey et al. 1950) motor areas in the same monkey and reported hyperactive tendon reflexes and some clonus. Ablation of the precentral motor area by Travis (1955a) produced an initial hypotonus followed by a return to normal tone. Reflexes, initially depressed, became hyperactive with clonus in the forelimb while the initially depressed reflexes of the hindlimb returned to normal. Bilateral removal of area 4s produced no changes in tonus or posture. Bilateral ablation of the motor area (Travis, 1955b) produced no hypotonus, but did result in depressed tendon reflexes, while bilateral supplementary motor area ablations produced hypertonus in the flexors of the limbs. Ablation of the left precentral and supplementary motor areas initially produced hypotonus followed by hypertonus of the extensors, no clonus and tendon reflexes that were initially depressed became normal and then hyperexcitable (Travis and Woolsey, 1953). Subsequent ablation of the two motor areas for the right leg produced results similar to those seen in the left limbs. In another series, ablation of the precentral arm and leg areas alone was performed seriatum. In both cases there was a depression of tendon reflexes and the muscle tonus was initially hypotonic but became hypertonic, then normal. When area 6 was removed bilaterally, no change in tone was found while bilateral ablation of the supplementary motor area resulted in increased tendon reflexes and finger clonus. Ablation of areas 4 and 6 resulted in normal tonus after an initial period of hypertonus, no clonus, and depressed tendon reflexes. Subsequent removal of the contralateral areas 4 and 6 resulted in strong extensor hypertonus and decreased tendon reflexes followed

by hyperreflexia. Unilateral ablation of the precentral and supplementary arm and leg areas produced hypotonus followed by hypertonus in all muscles of the limbs and tendon reflexes, which were initially depressed, became strong and often resulted in clonus. Similar results were also produced by bilateral precentral and supplementary motor ablations and by ablations of the precentral arm and leg areas plus the entire supplementary motor area. Contradictory to the results of Travis and Woolsey (1952, 1955a,b, 1958) are the results of Denny-Brown (1963) who reported supplementary motor area ablations to produce a plastic type of hypertonus mainly affecting the flexors with clonus being absent.

In summary, there are several conflicting points of view as to which cortical area ablation results in hypertonus. Fulton, Bucy and Kennard (1933, 1934, 1935, 1936, 1944) reported area 6 ablation produces hypertonus and area 4 ablation results in hypotonus, though Kennard stressed the importance of combined area 4 and area 6 ablation in the production of hypertonus. In all cases permanent hypertonus was produced only with bilateral area 6 ablation. Walshe (1935a,b) thought that ablation of the physiological motor cortex was responsible for hypertonus and rejected the functional separation of areas 4 and 6, while Hines (1936) took the view that area 4s was responsible for hypertonus, though her results were not verified in later studies by Denny-Brown (1948, 1963) and Travis and Woolsey (1952, 1955a,b, 1958). Denny-Brown reported that both area 4 and area 6 produced hypertonus when ablated but that hypertonus due to area 4 lesions was a spastic type while the hypertonus due to area 6 lesions was a plastic type. Finally, Travis and Woolsey reported hypertonus to be produced as a result of lesions of the supplementary motor area.

### C. Electroencephalograph Studies

Another indication of the cerebral control of the gamma motor system is seen in the studies by von Euler and Södenberg (1956, 1957) who recorded  $I_{\alpha}$  afferent activity in dorsal root filaments while monitoring electroencephalograph (EEG) activity in cats. Although no correlation was reported between gamma activity and EEG spindles, arousing stimuli activated the cortical EEG and increased  $I_{\alpha}$  activity while reduction of body temperature and cutaneous reflexes synchronized the EEG and decreased the  $I_{\alpha}$  activity. In similar studies Buchwald and Eldred (1961) recorded gamma and alpha motoneuron activity from ventral root filaments during conditioned motor learning experiments. Gamma activity was found to be closely associated with the EEG. Low voltage fast EEG waves were associated with an increase in gamma activity and gamma activity always preceded alpha activity when the latter was present. A correlation between EEG spindles and  $I_{\alpha}$  activity was found in these studies but it was reported to be very sensitive to anesthesia levels with the EEG activity being more sensitive to anesthesia than the gamma activity.

Since any interpretation of the results depends on an understanding of the anatomy of the pyramidal tract and the morphology and physiology of muscle spindles, these topics will be briefly reviewed.

### Morphology of the Muscle Spindle

#### A. Intrafusal Muscle Fibers

Mammalian muscles contain special sensory receptors which, because of their spindle-like appearance, are called muscle spindles (Kuhne, cited from Granit, 1955). Muscle spindles consist of specialized striated muscle fibers, the intrafusal

muscle fibers (Sherrington, 1894) which are enclosed over a part of their length by a connective tissue capsule composed of collagen fibers (Merrillees, 1960). The capsule extends over only 35-50% of the spindle length (Swett and Eldred, 1960). There are two types of intrafusal muscle fibers which are distinguished on the basis of the arrangement of their nuclei. The first of these, the nuclear bag fibers (Barker, 1948; Boyd, 1960) run the full length of the capsule and extend beyond it. At the polar ends, these fibers are striated but as the fibers enter the capsule there is a reduction in the degree of striation and the number of nuclei which become centrally located in a single row. This transition zone is known as the myotube region. In the center of the capsule, known as the equatorial region, the striations are lost and the nuclei are increased in number and lie three to five abreast. The second intrafusal muscle fiber type, the nuclear chain fibers (Boyd, 1960) present a similar picture except that in the equatorial region the nuclei are arranged in a single row. The nuclear bag fibers have a length of 4-13 mm in the act (Boyd, 1962) and 5 mm in man (Cooper and Daniel, 1963). They run beyond the length of the capsule about 1-2 mm and insert on the tendon or perimysium of the extrafusal muscle fibers while the smaller nuclear chain fibers with a length of 1.5-6 mm (Boyd, 1962) in the cat may terminate either within the capsule attaching to its wall or may extend beyond the capsule and attach to the nuclear bag fibers (Swett and Eldred, 1960; Boyd, 1962; Cooper and Daniel, 1963; Bridgman et al. 1969). Nuclear bag fibers have diameters of 10-30 $\mu$ , while the diameter of nuclear chain fibers is about 4-8 $\mu$  (Barker and Gidumal, 1960, 1961; Boyd, 1962; Cooper and Daniel, 1963). Generally, there are more nuclear chain fibers (1-10) than nuclear bag fibers (1-4) with the ratio depending on the species

(Boyd, 1962; Cooper and Daniel, 1963). The position of the two fiber types relative to each other within the capsule varies from spindle to spindle. The nuclear bag fibers generally run through the spindle in a straight line while the nuclear chain fibers twist and run among themselves and the nuclear bag fibers. Light microscope studies have suggested branching of the nuclear chain fibers (Barker and Gidumal, 1960, 1961; Boyd, 1960; Barker, 1962), but this has not been verified by electronmicroscopy (Corvaja et al. 1967, 1969; Adal, 1969).

A third type of intrafusal muscle fiber has been reported by Barker and Gidumal (1960, 1961) and Barker and Stacy (1970). This fiber is reported to be smaller in diameter than the nuclear bag fibers and to have nuclear bags in the myotube region. The presence of these fibers has not been confirmed by the ultrastructural studies of Corvaja et al. (1969).

The intracapsular space about the intrafusal fibers is called the lymph space since this space can be injected via the lymph system (Sherrington, 1894).

## B. Sensory Innervation

The muscle spindle is innervated by a sensory nerve which enters the spindle near the equatorial region. Each spindle is innervated by one Group I myelinated fiber (diameter 8-12 $\mu$ ) which branches inside the capsule and surrounds each intrafusal muscle fiber (Barker, 1948; Boyd, 1962). This primary ending covers nearly all of the nuclear region of both fiber types. In some species (cat and rabbit) the endings are in the form of spirals surrounding the intrafusal fibers, but in man there is no regular spiral (Cooper and Daniel, 1963). In addition to the one primary ending,

each spindle may have up to five secondary sensory endings which terminate in the equatorial region on one or both sides of the primary ending in the nuclear chain fibers (Boyd, 1962; Cooper and Daniel, 1963). These Group II fibers have a diameter of  $6-9\mu$  (Boyd, 1962) and innervate the nuclear chain fibers and except for accessory fibers from the secondary endings to the nuclear chain fibers there are no Group II endings on the nuclear bag fibers (Barker and Ip, 1960; Boyd, 1962). There is no overlap between the regions of innervation of the two types of sensory fibers and there may be a space between the two in which there is no innervation at all (Corvaja et al. 1969).

### C. Motor Innervation

In addition to the sensory innervation of the muscle spindle there is also a motor innervation to the intrafusal muscle fibers. The most numerous type of ending is the gamma plate ending which is generally located outside the capsule (Barker, 1966a, 1967). These motor fibers terminate in motor endplates similar to those of the alpha motoneurons innervating the extrafusal muscle fibers and each motor endplate has its own gamma motor fiber (Barker et al 1970). The second type of motor innervation, the trail ending, terminates in the juxtaequatorial region just beyond the secondary afferents (Barker, 1966a). These gamma fibers terminate after diffuse branching into many scattered points. Histograms reveal two distinct peaks of gamma fiber diameters (Boyd, 1962), the larger fibers being referred to as  $\gamma_1$  and the smaller fibers as  $\gamma_2$ . Boyd further reported that  $\gamma_1$  fibers terminated as plate endings while  $\gamma_2$  fibers terminated as trail endings, however these re-

sults have been questioned by Barker and Cope (1962) and Adal and Barker (1965). Currently the terms gamma-plate and gamma-trail fibers are the most widely accepted (Granit, 1966) and Boyd now classes gamma fibers on the basis of conduction velocity with no attempt at a correlation with ending type or function (Boyd and Davey, 1968). Boyd has also claimed that the plate endings terminate solely on the nuclear bag fibers while the nuclear chain fibers have only trail endings (Boyd, 1962; Boyd and Davey, 1962, 1966). Again these results have been questioned by Barker and his associates (Barker, 1966a,b; Barker et al. 1970).

A third type of innervation via beta fibers (Adal and Barker, 1965), has been reported (Matthews, 1933; Leksell, 1945; Barker, 1948; Bessou et al. 1965). These fibers are actually alpha motor fibers which innervate both the extra- and intrafusal muscle fibers and terminate as endplates on both intrafusal fiber types (Barker, 1966a, 1967).

## Physiology of the Muscle Spindle

### A. Afferent Innervation

In 1928, Fulton and Pi-Suner noted that the muscle spindles appeared to be in parallel with the extrafusal muscle fibers while the Golgi tendon organs were in series with the extrafusal muscle fibers. On this basis it was predicted that stretch would cause an increase in the firing rate of both receptors. During contraction, the spindles would be shortened along with the rest of the extrafusal muscle fibers and would show a reduction in their firing rate while tendon organs, being in series with the extrafusal muscle fibers, would have the tension in the muscle transmitted to them

and would show an increase in firing rate. The predictions of this often overlooked paper were verified by Matthews (1933) who recorded the action potentials from single sensory nerve endings in the cat. Type A receptors (according to Matthews) were described as firing during passive stretch but reducing or ceasing their activity during a muscle twitch; that is, acting as if they were in parallel with the extrafusal muscle fibers. Type B endings were described as firing in response to the tension of the muscle whether passive or active, thus acting as if they were in series with the extrafusal muscle fibers. The rate of firing of the Type A afferents during stretch depended on the rate of stretch as well as the degree of stretch. Hence, they provided information on the rate of change of length as well as the length of the muscle. Hunt (1954) recorded from hindlimb afferents in the dorsal root filaments of cats and based on their response to stretch, twitch contraction and conduction velocity the Type A endings were classed as Group I<sub>a</sub> primary afferents to distinguish them from the Type B Golgi tendon organs which was classed as Group I<sub>b</sub> afferents. The secondary muscle afferents were classed as Group II fibers but no difference between the Group I<sub>a</sub> and Group II receptors was noted except for a higher threshold for the secondary fibers which was subsequently verified many times (Cooper, 1959, 1961; Harvey and Matthews, 1961; Bessou and Laporte, 1962; Renkin and Vallbo, 1964). The conduction velocity at which Group I and Group II fibers could be separated was originally placed at 72 M/sec (Hunt, 1954), however, it has been discovered that there is a considerable overlap in the 60–80 M/sec range (Matthews, 1963; Rack and Westbury, 1966). The phase of active stretch has been termed the dynamic phase while the term static phase has been used to denote the period of maintained or

constant stretch (Katz, 1950a,b). The secondary receptors have been considered to be relatively insensitive to the dynamic phase of stretch (Cooper, 1961) while the primary and secondary receptors are similar in their static properties (Jansen and Matthews, 1962). Goslow et al. (1973) have shown the Group II endings to be quite sensitive to the dynamic phase when the muscle is stretched over lengths found in normal movement and at rates similar to those in stepping, walking, trotting and running. Although both spindle afferents show a reduction in firing rate or pause during a twitch contraction (the silent period), secondary fibers require a greater degree of shortening to produce a pause or silent period (Bessou and Laporte, 1962). Static firing is directly proportional to the degree of stretch, although the variation of firing rate with varying stretch may differ between primary and secondary receptors (Eldred et al. 1953; Granit and Homma, 1959; Harvey and Matthews, 1961; Bessou and Laporte, 1962; Renkin and Vallbo, 1964).  $I_a$  afferents have been shown to respond to increments of stretch of less than  $20\mu$  while Group II afferents may have thresholds of  $500\mu$  (Lundberg and Winsburg, 1960). With vibration (Bianconi and van der Meulen, 1963) over a range of 50–500 cps, all primaries and half of the secondaries have been found to follow the vibratory rate. The remaining half of the secondaries showed an augmentation of their firing rate with the discharge independent of the phase and frequency of vibration. A further difference between primaries and secondaries is shown by their response to sinusoidal stretch (Stuart et al. 1965) where the primaries fire at a higher rate and at an earlier phase in the cycle than the secondaries.

## B. Motor Innervation

Before the details of the sensory and motor innervation of the muscle spindle had been worked out, it had been known that the silent period of the  $I_{\alpha}$  afferents during the contraction phase of the twitch could be reduced or eliminated by stimulation of the gamma motoneurons (Matthews, 1933; Leksell, 1945; Hunt and Kuffler, 1951a,b; Kuffler et al. 1951). Boyd (1966) observed under a microscope that the contraction of the intrafusal muscle fibers due to gamma stimulation stretched the sensory region of the muscle spindle in the same manner as if the spindle had been externally stretched. Further, in decerebrate cats the frequency of spindle firing at all degrees of muscle stretch is decreased when the ventral roots are sectioned (Eldred et al. 1963). Thus, the gamma activity or bias can alter the sensitivity of the spindle to stretch. Matthews (1962) described two types of fusimotor fibers. Dynamic fusimotor fibers were described as increasing the  $I_{\alpha}$  firing rate during dynamic and static phases of muscle stretch though the effect was greater during the dynamic phase. Static fusimotor fibers increased the  $I_{\alpha}$  firing rate only during the static phase, i.e., at the initial and final muscle lengths. Further, the effect of static fusimotor fibers on the firing rate of  $I_{\alpha}$  fibers during the static phase is greater than the effect of the dynamic fusimotor fibers during the static phase (Crowe and Matthews, 1964; Brown et al. 1965; Bessou et al. 1966). Studies on single gamma fibers teased from ventral roots (Crowe and Matthews, 1964a,b; Appelburg et al. 1965; Brown et al. 1965; Bessou et al. 1966; Emonet-Danand et al. 1966) have shown that the static fibers are more numerous (3:1) and have higher con-

duction velocities. The effect of the gamma bias can be seen in vibration where the gamma bias can increase the ability of spindle afferents to follow vibration (Granit and Henatsch, 1956; Bianconi and van der Meulin, 1963; Brown et al. 1967) with the effect being greatest on the primary endings. The primary response to sinusoidal stretch is also enhanced by the gamma bias (Crowe and Matthews, 1964b; Goodwin and Matthews, 1970). The spindle discharge during spindle shortening has been shown to be due to the activity of the static fusimotor fibers and not to the dynamic fibers (Crowe and Matthews, 1964a,b; Lennerstrand and Thoden, 1968).

#### Anatomy of the Cerebral Cortex

In the adult mammal the cerebral cortex is a multilayered structure consisting of six layers, each with its own characteristic cellular types, which may be characterized as follows: 1) the outermost layer, the molecular layer, consists of a dense tangential fiber mass (for which reason this layer is often called the plexiform layer) 2) the external granular layer consists of densely packed cells of a pyramidal, stellar or circular shape 3) the external pyramidal layer is characterized by the presence of medium sized pyramidal cells whose axons serve chiefly as association or commissural fibers 4) the internal granular layer consists of polygonal or triangular shaped cells 5) in the fifth layer are found the large pyramidal cells 6) the innermost layer of the cortex, the multiform layer consists of spindle shaped cells.

This general description with some modification is characteristic of all areas of the cortex. Different regions of the cortex show definite regional differences and there are several maps of the cortex based on the regional differences in the

cytoarchitecture (Campbell, 1905; Brodman, 1909; Vogt and Vogt, 1919; von Economo, 1927). Brodman's area 4 has been known as the motor area since Fritsch and Hitzig (1870) first demonstrated this area to be electrically excitable. This region is anatomically characterized by the presence of the giant pyramidal cells of Betz (1874) in the fifth layer. This region is located on the anterior wall of the central sulcus and occupies the adjacent regions of the precentral gyrus. Area 6, which lies immediately in front of the motor area, is known as the premotor area and is similar to the motor area except that it lacks the large Betz cells.

## Anatomy of the Pyramidal Tract

### A. Definition

The pyramidal or corticospinal tract is the most direct projection of the cerebral cortex to the spinal cord. Patton and Amassian (1960) defined the pyramidal tract as consisting of those neurons with descending axons passing longitudinally through the medullary pyramids. The pyramidal tract has several unique features (Lassek, 1948). Except for the cerebellum, the pyramidal tract is present in all major and minor subdivisions of the brain, having the longest uninterrupted path (up to 3 meters) deviating and transversing through a number of physically vulnerable areas where its location on the external surface of the central nervous system makes it subject to cerebrospinal fluid pressure changes, external medullary masses and birth damage. Because of its wide vascular supply it is subject to vascular accidents and its slow rate of maturation, up to two years in man, further adds to its sensitivity to injury. It has been suggested by Brouwer (1920) that the newness of the

pyramidal tract is also responsible for the susceptibility of the pyramidal tract to damage but Lassek (1945) was unable to find that the pyramidal tract was more susceptible to damage than other parts of the central nervous system.

The pyramidal tract is considered to be present only in mammals (Kappers et al. 1967; Biedenback and Towe, 1970) although King (1911) reported the presence of a pyramidal tract in the parrot. The pyramidal tract shows some variation between different species and is quite variable in man (Brouwer, 1920; Nathan and Smith, 1955). The variability has been explained on the basis of the newness of the pyramidal tract's phylogenetic origin (Brouwer, 1920).

The number of fibers in the pyramidal tract depends on the species. The approximate number of fibers per pyramid based on studies by van Crevel and Verhaart (1962a,b), De Myer (1959), Jane et al. (1967), Lassek (1941), Lassek and Karlsberg (1956) and Lassek and Rasmussen (1940) are: mouse 32,000; rat and opossum 75,000; cat 80,000; dog 300,000; cow 500,000; monkey 550,000; seal 750,000; man 1,000,000. In man there is no dependence of fiber number on the basis of age, sex or race (De Myer, 1959) and in the cat there is no difference between right and left pyramids and no relation between fiber number and body weight or sex (van Crevel and Verhaart, 1963a,b). In the dog, a large animal may have as many as 100,000 more fibers than a small one, but it should be pointed out that this is a species in which there is a large variation in size among individuals (Lassek and Rasmussen, 1940). In most species, there is a mixture of large and small fibers although the opossum has only fine fibers (Biedenback and Towe, 1970). In most carnivores and primates only 3-4% of the fibers are large (10-15 $\mu$ ) while the remain-

der are less than  $10\mu$  with most being 1-5 $\mu$  (Lassek, 1941, 1948; Lassek and Rasmussen, 1939, 1940; Lassek and Karlsberg, 1956). The small fiber size makes the pyramidal tract a slow conductor (Lassek and Rasmussen, 1940; Lassek, 1948) and conduction velocities have been reported to range from 164 M/sec to 1.8 M/sec with most fibers conducting at 35-70 M/sec (Brookhart and Moris, 1953; Bernhard et al. 1953). There is no separation of fibers within the pyramidal tract on the basis of fiber size or conduction velocity. The degree of myelination within the pyramidal tract is also variable. In the monkey and seal, about 60% of the fibers are myelinated (Lassek and Karlsberg, 1956) and 94% of the fibers are myelinated in man (De Myer, 1959). However, the presence of unmyelinated fibers in the central nervous system has been questioned (van Crevel and Verhaart, 1963b).

## B. Origin

The origin of the pyramidal tract has been the subject of much debate and has resulted in several papers on the "exact origin of the pyramidal tract." Türk (see Marshall, 1936) observed that lesions of the cerebral hemispheres resulted in fiber degeneration in the pyramids. In 1909, Holmes and Page May sectioned the pyramidal tract by spinal cord hemisection and examined the cerebral cortex for signs of chromatolysis which was found to be present only in the giant cells of Betz which were coextensive with the excitable cortex. The species studied were cat, lemur, chimpanzee, monkey and man with all giving the same results. These results were later confirmed by Levin (1936). It was well established by several studies (Mott, 1893-94; Olmsted and Logan, 1925; McKibben, 1929; McKibben and Wheelis, 1932;

Kennard, 1935; Verhaart and Kennard, 1940; Barnard and Woolsey, 1956) that lesions of the motor cortex resulted in pyramidal tract fiber degeneration, as was the view that the Betz cells totally (Bucy et al. 1964, 1966) or partially (Walker and Richter, 1966) send their axons through the pyramidal tract. However, there has been considerable dispute over the issue of whether Betz cells are the sole source of the pyramidal tract fibers because of the large difference between the number of Betz cells in the cortex and the number of fibers in the pyramidal tract. In man, the number of Betz cells in the cortex has been estimated to be 35,000 to 40,000 (Lassek, 1940; Lassek and Rasmussen, 1940) while the number of pyramidal fibers is approximately 1,000,000. In a study of the spider monkey the number of Betz cells has been estimated to be 10,000 with 500,000 fibers present in the pyramidal tract (Lassek, 1943). In all cases the number of Betz cells in the cortex corresponds to the number of large fibers in the pyramidal tract suggesting that the Betz cells contribute the large diameter axons to the pyramidal tract. The reason the studies of Holmes and Page May showed only Betz cell degeneration is best explained by the observation that a considerable period of time is required for all of the pyramidal tract cells to degenerate. The experimental animals of Holmes and Page May were sacrificed 5-157 days after the lesions were placed in the pyramidal tract while the human cases were reported as having been examined 108 and 229 days after the damage to the pyramidal tract occurred. Since these studies, it has been shown that complete degeneration of pyramidal tract neurons requires 6-12 months (Lassek, 1946; Russell and De Myer, 1961; van Crevel and Verhaart, 1963a,b) with

the largest fibers being affected the earliest (Lassek, 1946; van Crevel and Verhaart, 1963) and to the greatest extent (Lassek, 1946).

Ablations of area 4 result in only partial pyramidal tract degeneration; 40% in the Macaca (Lassek, 1942; Russell and De Myer, 1961) and 81% in the bonnet monkey (Levin and Bradford, 1938). The observation by Levin and Bradford that lesions of the anterior border of area 4 cause less severe degeneration is consistent with the observation that area 4s does not contribute to the pyramidal tract (Verhaart and Kennard, 1940). Projections from the premotor cortex in man have been reported by Minckler et al. (1944) and projections from the parietal cortex have been reported by Lassek (1942). Twenty nine per cent of the pyramidal tract has been attributed to area 6 while 40% has been attributed to the parietal cortex (Russell and De Myer, 1961). Section of pyramidal tract by hemisection of the spinal cord has resulted in degeneration changes in the Betz cells in the transition zone between area 4 and 3 and in areas 3, 1, 2, and 5 (Levin and Bradford, 1938) while ablation of areas 3, 1, 2, 5, and 7 result in pyramidal tract degeneration (Peele, 1943; Spiegel et al. 1943). These anatomical studies have been verified electrophysiologically (Woolsey and Chang, 1947) with stimulation of the medullary pyramids resulting in antidromic response in areas 4, 6, 3, 1, 2, 5, and 7 in the monkey and in similar areas in the cat and rabbit. In the primate there appear to be no fibers originating from the temporal lobes (Rundles and Papey, 1938; Bucy and Kluver, 1955).

In the cat, fiber contributions from the temporal and occipital lobes (Walberg and Brodal, 1953) and from the parietal cortex (Gobbel and Liles, 1957) have been reported but these findings were not confirmed in later studies by van Crevel and Ver-

haart (1963a,b) who reported the following projections to the pyramidal tract; frontal and medial anterior sigmoid gyrus, 10%; remainder of the anterior sigmoid gyrus, 60%; post central (coronalis) gyrus, 30%.

### C. Anatomical Path

There is good agreement on the path of the pyramidal tract from its origin in the cortex to the level of the medullary pyramids. From the cortex the pyramidal tract descends into the internal capsule occupying the genu and posterior limb (Gobbel and Liles, 1945; Glee and Cole, 1950; Walberg and Brodal, 1953). From the internal capsule the fibers pass into the cerebral peduncles where they occupy the middle 3/4 to 3/5 of the peduncle (Turner, 1924; Levin, 1936; Bucy et al. 1964). In the monkey (Barnard and Woolsey, 1956) there is a somatotopic localization of the fibers in the pyramidal tract down to the level of the decussation, although there is extensive overlap even in the internal capsule and the cortex. The localization is due to the mechanical convenience of the descending fibers seeking the shortest route as they descend. In the pons the localization is lost as the continuity of the pyramidal tract is interrupted by the termination of the corticopontine fibers, the crossing of the pontocerebellar fibers and the subsequent rearrangement of the fibers in the pyramid.

From the cerebral peduncle, the pyramidal tract descends into the pons and at the caudal border of the pons the fibers unite to form a distinct bundle, the medullary pyramids. There is a high degree of variation as the pyramidal tract fibers descend from the pyramids into the spinal cord in various combinations of crossed and uncrossed

corticospinal tracts, with many differences between species and individuals of the same species. In the hedgehog and mole (King, 1911) none of the fibers decussate and all of the fibers descend in the ventral column of the spinal cord. In the opossum (Turner, 1924) the fibers cross in the ventral funiculus and descend in the dorsal funiculus. All of the fibers cross in the rat and descend in the dorsal funiculus (King, 1911; Barnard and Woolsey, 1956). In the cat there is a large contralateral corticospinal tract originating from the area about the cruciate sulcus (Olmsted and Logan, 1925; Tower, 1935; Chiarugi et al. 1955; Chambers and Liu, 1957). Fiber contributions to this tract have been reported to originate from the parietal (Gobbel and Liles, 1945) and temporal and occipital (Walberg and Brodal, 1953) areas. A small ipsilateral ventral corticospinal tract originates from the sigmoid gyrus (Chambers and Liu, 1957) and Walberg and Brodal (1953) report the presence of ipsilateral lateral and ventral ipsi- and contralateral corticospinal tracts originating from the temporal and occipital lobes.

The primate has a large crossed corticospinal tract consisting of fibers from the motor and premotor areas (Kennard, 1935; Levin, 1936; Chambers and Liu, 1958; Bucy et al. 1964) and the parietal areas (Peele, 1942). A small number of fibers descend as ipsilateral lateral and ipsilateral ventral tracts (Kennard, 1935; Levin, 1936; Peele, 1942; Chambers and Liu, 1958; Liu and Chambers, 1964) although the uncrossed tracts may be absent (Grünbaum and Sherrington, 1904; Weil and Lassek, 1929).

#### D. Termination

The termination of the pyramidal tract is also variable, again depending on the

species. The pyramidal tract does not descend below the decussation in the opossum and rabbit ending in the cervical cord (Turner, 1924; Lassek and Rasmussen, 1940; Biedenback and Towe, 1970). In the rat (King, 1911; Lassek and Rasmussen, 1940) the pyramidal tract descends to the level of the sacral cord. Anatomical and electrophysiological studies in the cat show the pyramidal tract to extend the full length of the spinal cord with the projections to the cervical cord being the greatest (Hoff, 1932; Tower, 1935; Lloyd, 1941; Brookhard and Moris, 1948; Walberg and Brodal, 1953; Chiarugi et al. 1955; Chambers and Liu, 1957) although the fibers from the parietal cortex extend only to the cervical cord (Gobbel and Liles, 1945) as do the ventral tracts (Chambers and Liu, 1957) after crossing in the ventral white commissure. Although there is much overlap, the forelimb fibers terminate mostly in C<sub>1</sub> to L<sub>9</sub> while the hindlimb fibers terminate in T<sub>7</sub> to S<sub>2</sub> (Chambers and Liu, 1957).

Within the spinal gray matter of the cat, the lateral corticospinal tracts terminate in the base of the dorsal horn (Hoff, 1932; Lloyd, 1941; Chambers and Liu, 1957), in the apex of the dorsal horn and the intermediate gray (Chambers and Liu, 1957; Kuypers, 1963). Terminations in the ventral horn were reported by Hoff (1932) but these findings were not confirmed by Chambers and Liu (1957). Dyachkave et al. (1971) reported the major terminal site to be the lateral part of lamina V while Nyberg-Hansen and Brodal (1963) reported that the fibers from the motor cortex terminate ventrally in laminae IV, V, VI and VII and those fibers from the sensory cortex terminate dorsally in lamina IV, V and VI, although there is no complete separation. The pyramidal tract terminates on all cell types (Nyberg-Hansen and Brodal, 1963) synapsing on both dendrites and soma (Hoff, 1932; Nyberg-Hansen and Brodal, 1963).

There appears to be no anatomical evidence that the pyramidal tract fibers synapse directly on alpha motoneurons (Kuypers, 1963; Scheibel and Scheibel, 1966) though the dendrites of the alpha motoneurons may extend up into the region of pyramidal tract termination (Scheibel and Scheibel, 1966). Electrophysiological studies have shown no monosynaptic transmission to the alpha motoneurons in the cat (Lloyd, 1941; Bernhard and Bohm, 1954; Morrell, 1957; Hern et al. 1962; Vasilenko and Kostyuk, 1966; Preston et al. 1967).

In the primate the pyramidal tract also extends the full length of the spinal cord (Schäfer, 1883; Sherrington, 1889; Hoff and Hoff, 1934; Levin, 1936; Peele, 1942; Glees and Cole, 1950; Chambers and Liu, 1958; Kuypers, 1960; Liu and Chambers, 1964; Bucy et al. 1964, 1966). The ventral corticospinal tracts end in the upper thoracic cord (Weil and Lassek, 1929) while the ipsilateral fibers from the parietal area terminate in the cervical cord (Peele, 1942). As in the cat, the arm areas generally project to the cervical area while the leg areas project to the lumbar and sacral cord (Sherrington, 1889; Chambers and Liu, 1958; Liu and Chambers, 1964) although there is a great amount of overlap. In man (Weil and Lassek, 1929) 50% of the pyramidal tract fibers terminate in the cervical cord, 20% in the dorsal lumbar cord and 30% in the ventral lumbar cord with the projections from the cortical hand area having the least number of Betz cells (Weil and Lassek, 1929; Lassek, 1941; Walshe, 1935b, 1942). The distribution of the pyramidal tract fibers in the spinal gray matter of the primate is similar to the cat except for the presence of direct synapses on the ventral horn motoneurons (Hoff and Hoff, 1934; Bernhard et al. 1953; Bernhard and Bohm, 1954; Chambers and Liu, 1958; Kuypers,

1960; Preston and Whitlock, 1961; Landgren et al. 1962a,b; Liu and Chambers, 1964; Phillips and Porter, 1964; Uemura and Preston, 1965; Kernell and Wu, 1967; Preston et al. 1967; Clough et al. 1968; Koeze et al. 1968; Stewart and Preston, 1968).

Brodal and Walberg (1952) reported the presence of ascending fibers in the pyramidal tract of the cat as a result of lesions in the lateral funiculus of the spinal cord. These ascending fibers were estimated to represent only 1/25 of the total number of pyramidal tract fibers with most terminating in the sensory cortex. These results were reported to have been verified by electrophysiological studies (Brodal and Kaada, 1953) in which potential changes in the medullary pyramid were recorded in response to stimulation of cutaneous, muscle and mixed nerves. The greatest response was to stimulation of forelimb nerves. These results were disputed by Landau (1956) who repeated the experiments and reported that in all cases the area of the "pyramidal tract" responsive to cortical stimulation was superficial to the area responsive to peripheral nerve stimulation. Histological studies revealed the former area to be the pyramidal tract and the latter area to be the medial lemniscus. No response to nerve stimulation was reported to be present in the cerebral peduncles. It is now believed that there are no ascending fibers in the pyramidal tract.

In general, there is an increase in the size and extent of the pyramidal tract as one ascends phylogenetically and in going from the carnivores to primates the termination of the pyramidal tract shifts from interneurons to direct synapses on the ventral horn motoneurons. The development of the pyramidal tract has been linked to the increase in the use of the limbs for non-locomotor functions and the develop-

ment of more complex movements in the use of the limbs in climbing and grasping.

#### E. Betz Cells

Although those areas of the motor cortex associated with the most complex movements (the hands) have the least number of Betz cells, these cells have been of interest because of their size and presence in the motor cortex. Walshe (1942) considered the Betz cells simply to be the largest and most massive member of the large group of pyramidal cells, and that Betz cells were not to be regarded as specific morphological entities. Recent studies and the absence of the Betz cells in the hand area would seem to confirm Walshe's view. Electronmicroscopic studies (Kaiserman-Abramof and Peters, 1972) show the initial segment to have the same characteristics of other neurons and that Betz cells share other features in common with other neurons. One special feature of Betz cells is an early branching of the apical dendrite into 2-3 diverging branches of equal size. These spread quite far laterally so that, although Betz cells may be sparsely distributed, each cell receives a wide input. Functionally, Lux and Pollen (1966) were unable to find any difference between Betz cells and other excitable cells in regard to their action potentials.

## METHODS AND MATERIALS

### Anesthesia

Adult cats weighing 2.3-3.5 kg were anesthetized with Nembutal (30 mg/kg, ip). Tracheal and femoral vein cannulations were routinely performed. Dehydration was prevented by periodic injections of 5-10 ml of Ringer's i.v. every three hours (Stewart and Preston, 1968). Three to four hours elapsed from the time of initial surgical anesthesia to data collection. At the time of data collection the level of anesthesia was such that there was a flexor response to pinching of the paws, but no pupil dilation.

### Pyramidal Tract Exposure

The pyramidal tract at the level of the medullary pyramids was exposed through a ventral approach (Tower, 1935; Liddell and Phillips, 1944; Phillips, 1956). The tracheal cannula was placed as low in the neck as possible to facilitate the exposure. In order to expose the pyramidal tract the following structures were reflected and cut between double ties to prevent bleeding; cranial sternomodoideus m., sternohyoideus m., trachea, and esophagus. The basioccipital bone was exposed by removal of the preoccipital muscles. The pyramids were then exposed by making a small hole in the basioccipital bone with a pair of small rongeurs. The exposure allowed for a good view of the pyramids when the cat was prone in the stereotaxic frame. The dura over the pyramids was sectioned longitudinally to allow placement of the stimulating electrode directly on the pyramids. Two points of care in the operative procedure should be noted. First, cephalad reflection of trachea and

esophagus to expose the preoccipital muscles causes the base of the tongue to be pulled forward, often placing sufficient strain on the lingual arteries to rupture them. To prevent this, these arteries were ligated prior to removal of the preoccipital muscles. Second, sectioning of the dura over the pyramids resulted in extensive bleeding in about 80% of the cats. This bleeding was stopped with a cotton pad soaked in Ringer's solution. It is important that no clot be allowed to form over the surface of the pyramids since this will impair electrical stimulation. Attempting to remove the clot when the cat is prone in the stereotaxic frame can result in additional bleeding which is difficult to stop, since access to the pyramids is limited with the cat in this position. After the pyramids were exposed, the neck viscera were covered with cotton soaked in Ringer's solution and the neck was closed with a hemostat until the placement of stimulating electrodes. The head of the cat was then positioned in the stereotaxic frame.

#### Exposure of the Spinal Cord

The trunk of the cat was placed on a slanted operating board to facilitate the following procedures: the vertebrae from L<sub>4</sub> to S<sub>2</sub> were dissected free of the following muscles: interspinales, multifidus and intertransversori. The muscles were reflected from the midline with a blunt instrument with care being taken to thoroughly clean the muscles from the vertebrae. Hip pins were then screwed into the greater trochanter of each femur.

The lumbosacral vertebral column was then rigidly fixed in a spinal frame by:  
1) the hip pins in the greater trochanter; 2) by three spinal pins inserted into the

bodies of vertebrae L<sub>4</sub>, L<sub>5</sub> and L<sub>6</sub> , or L<sub>5</sub>, L<sub>6</sub> and L<sub>7</sub> . A laminectomy was performed from L<sub>4</sub> to L<sub>7</sub> to expose the spinal cord. The back musculature was reflected by ties and cotton packs between the vertebra and muscle to facilitate the laminectomy. The exposed spinal cord enclosed in its dura was covered by cotton soaked in Ringer's solution.

#### Exposure of the Cerebral Cortex

The pericruciate cortex was exposed with a trephine and a small rongeur was used to expose the cortex to the midline. Care was taken not to tear the superior sagittal sinus in order to prevent bleeding. The cortex was then covered with moist cotton.

#### Denervation of the Hindlimb

In the present study the muscles examined were the anterior tibialis, soleus and gastrocnemius. Except for the muscle under study, the hindlimb was completely denervated in order to facilitate recording from I<sub>a</sub> afferent fibers. The following nerves were routinely cut: the muscular branch of the sciatic, and the lateral and caudal surals. For study of the anterior tibialis muscle the following nerves were also cut: peroneus longus, deep peroneus, superficial peroneus and tibial. The following nerves were sectioned when the soleus muscle was studied: common peroneal, medial gastrocnemius, and the posterior tibial below the juncture of the medial gastrocnemius and the soleus-lateral gastrocnemius. To study the medial gastrocnemius muscle the common peroneus, soleus, lateral gastrocnemius and tibial nerve below the junction of the soleus-lateral gastrocnemius and the medial gastrocnemius were cut. Since a

branch of the tibial nerve supplies the soleus and lateral gastrocnemius the denervation was the same for these two muscles. Afferents from each of the muscles can be readily identified by discrete pulls of their tendons.

#### Recording From $I_a$ Afferent Fibers

The cotton was removed to re-expose the spinal cord. Bleeding from the muscle was stopped with oxycel and from bone with bone wax. The dura was sectioned with blunt scissors and reflected. The arachnoid pia was carefully dissected from the dorsal roots from the intervertebral foramen to their entry into the spinal cord.

Single  $I_a$  afferent units from the muscle under study were teased out from dorsal root filaments using fine jeweler's forceps. They were identified by their firing pattern during stretch and muscle twitch and by their conduction velocity (see below). A small dorsal root filament was carefully teased out and placed on the recording electrode and the muscle under study was then stretched. An increase in firing rate of the action potentials in the filament indicated the presence of stretch sensitive receptors. Group  $I_a$  muscle afferents were differentiated from Group  $I_b$  Golgi tendon organs on the basis of the fact that muscle spindles are in parallel with the extrafusal muscle fibers and show a decreased firing rate or silent period during a muscle twitch, while Golgi tendon organs show an increase in firing rate because they are in series with the extrafusal muscle fibers (Matthews, 1933; Granit and van der Meulen, 1962). Since the Group II muscles spindle afferents have smaller axon diameters, they have slower conduction velocities (Gasser and Grundfast, 1939;

Hursh, 1939) making it possible to separate the Group I and Group II fibers on this basis. However, there is some overlap of conduction velocity between these two groups and only those afferents with conduction velocities of 72 M/sec or more were accepted as  $I_a$  afferents (Hunt, 1954).

$I_a$  afferents from the anterior tibialis muscle were generally found to be located in the most caudal portion of  $L_6$  filaments. Frequently as the  $L_7$  roots were pushed up and pulled away from those of  $L_6$ , one could find a small filament which belonged to  $L_6$  but tended to be pulled up with  $L_7$ . This "straggler" from  $L_6$  usually contained the anterior tibialis afferents. The soleus and gastrocnemius afferents were found in the most caudal portion of  $L_7$  or the most cephalad portion of the  $S_1$  filaments.

Recordings were made with a bipolar silver-silver chloride electrode from which signals were fed into a Tektronix 122 preamplifier and observed on a Tektronix dual beam oscilloscope. To facilitate the isolation of  $I_a$  afferent fibers and their response to electrical stimulation, the  $I_a$  action potentials were also fed into a Grass AM-3 audio amplifier and speaker system.

#### EMG Recording

To determine the influence of cortical and pyramidal stimulation on motoneuron activity in the muscle under study, EMG activity was recorded with a bipolar needle electrode inserted into the belly of the muscle (Bosma and Gellhorn, 1947a). These electrodes prevent recording EMG activity from adjacent muscles while permitting the recording of the electrical activity in the muscle under study (Landau, 1952; Asanuma

et al. 1968; Basmajian, 1972). EMG potentials were fed into a Tektronix 122 pre-amplifier and observed on a Tektronix dual beam oscilloscope.

#### Stimulation of the Pyramids

After the first  $I_a$  fiber was teased out or a single unit was isolated, a bipolar ball tip stainless steel electrode (tip separation 1-3 mm and tip diameter 0.5 mm) was placed on the pyramid ipsilateral to the exposed cortex. Sufficient exposure of the pyramids for electrode placement could be obtained by reflecting the trachea and esophagus forward with ties and the neck viscera laterally with a large retractor. Care was taken not to constrict the carotid arteries.

#### Stimulation of the Motor Cortex

After all other procedures were completed the dura of the cerebral cortex was sectioned (Ward and Clark, 1936; Adrian, 1939) and dehydration of the cortex was prevented by keeping it covered with a pool of warm mineral oil for the remainder of the procedure. Because of the lack of agreement among other investigators on the location of the hindlimb motor area and its relation to topographical features of the cortical surface, it was necessary to map the hindlimb area for each cat. For mapping and subsequent stimulation of the cortex, bipolar stainless steel ball tip electrodes (tip separation 1-3 mm and tip diameter 0.5 mm) were placed on the cortical surface and the cortex was stimulated with a 60 cycle biphasic square wave current of 1.0 msec duration. Response of the muscle under study was observed visually and by EMG and  $I_a$  activity. Various points on the cortex were stimulated until the point of lowest threshold for activity of the muscle was located. The electrode was then held in place

at this point with an electrode holder. The mineral oil pool was then covered with Saran Wrap. The mapping procedure was done for both alpha and gamma motoneurons.

#### Parameters of Electrical Stimulation

The electrodes used in these experiments (bipolar stainless steel ball tip with tip separations of 1-3 mm and tip diameter of 0.5 mm) were selected because most of the current flows between the two electrode tips and there is little current spread to adjacent tissues (Murphy and Gellhorn, 1945; Cure and Rasmussen, 1954). The stimulating current was a biphasic square wave pulse from a Nuclear Chicago constant current stimulator. The duration (1-2 msec) and intensity (0.5-2.0 ma, depending on whether gamma or gamma plus alpha activity was being stimulated) were held constant and the frequency was varied since the pattern of muscle activity is dependent on the stimulus frequency (Hyde and Gellhorn, 1951; Cure and Rasmussen, 1954). Cortical stimulations were at intervals of at least three minutes to permit recovery from the previous stimulation period (Dusser de Barenne and McCulloch, 1932; Dusser de Barenne, 1936; Ward and Clark, 1936; Clark and Ward, 1937; Murphy and Gellhorn, 1945; Clark, 1948; Woolsey et al. 1952; Cure and Rasmussen, 1954).

#### Deafferentation

The appropriate dorsal roots for the muscle under study were sectioned at their entry into the spinal cord. Thirty minutes were then allowed for recovery from any

shock due to the deafferentation before recording data.

#### Reproduction of Data

All data was recorded on an Ampex 5700 FM magnetic tape recorder at the time of experiments. Data was later played back and filmed on a Grass C<sub>4</sub> oscilloscope kymograph camera using Kodak linograph paper.

## RESULTS

These results are based on studies from 44 cats and a total of 244  $I_{\alpha}$  afferents from the anterior tibialis muscle (40), medial gastrocnemius muscle (76), lateral gastrocnemius muscle (65) and soleus muscle (63).

### ALPHA AND GAMMA MOTOR MAPS

Prior to the actual experiment it was necessary to map out the motor cortex to locate the areas which activated or influenced the particular muscle to be studied. The optimal foci for the muscles under study were found to be along the cruciate sulcus, 3-5 mm from the midline, with the alpha and gamma foci being co-extensive. There was generally little or no separation of the foci for the various hindlimb muscles and a relatively large area of cortex was found to influence both the fore- and hindlimbs. Thus under the conditions of these experiments, there was no discrete separation of hindlimb from forelimb or discrete localization of the muscles under study, and there was no separation of inhibitory and facilitatory stimulation points.

### ANTERIOR TIBIALIS

Two patterns of  $I_{\alpha}$  facilitation and EMG activity were seen when recording from the anterior tibialis muscle. In one case cortical and pyramidal stimulation resulted in  $I_{\alpha}$  facilitation followed by a post-stimulus period in which the  $I_{\alpha}$  firing rate ceased or was reduced. Figure 1 is representative of the response of these anterior tibialis  $I_{\alpha}$  afferents to cortical stimulation at gamma threshold stimulus levels (0.5-1.0 ma). In all cases the results were the same: increased  $I_{\alpha}$  firing rate,

no EMG and a post-stimulus cessation of  $I_{\alpha}$  firing rate. The facilitation of the spindle was maintained throughout the stimulus period, but was greatest during the initial portion of the stimulus period, and the period of increased  $I_{\alpha}$  firing outlasted the stimulus.

When the strength of cortical stimulus was increased to activate alpha motoneurons (1.0-1.5 ma) a phasic EMG was produced which was preceded by an increase in  $I_{\alpha}$  firing rate (Fig. 2). The post-stimulus period was characterized by a reduction in  $I_{\alpha}$  firing rate followed by a return to pre-stimulus levels.

Both the pattern of  $I_{\alpha}$  firing and the latencies between the onset of the stimulus and the period of  $I_{\alpha}$  facilitation (200-700 msec) were independent of the stimulus frequency.

With pyramidal tract stimulation the threshold (0.05-1.0 ma) for alpha and gamma motoneurons was the same, and resulted in  $I_{\alpha}$  facilitation, EMG activity, and post-stimulation cessation of spindle firing. At a stimulus frequency of 60/sec (Fig. 3) there was no EMG during the first period of the  $I_{\alpha}$  facilitation. The initial period of  $I_{\alpha}$  facilitation was followed by a reduction in spindle firing which was then followed by  $I_{\alpha}$  facilitation and EMG activity. There was no silent period associated with the EMG activity. Stimulation at 80/sec (Fig. 4) resulted in  $I_{\alpha}$  facilitation, EMG activity of a phasic type with each EMG burst preceded by a period of  $I_{\alpha}$  facilitation and followed by a period of decreased  $I_{\alpha}$  firing. With 100/sec (Fig. 5) the EMG activity was of a phasic type but the  $I_{\alpha}$  facilitation was maintained through all phases of EMG activity.

In the second anterior tibialis response pattern there was a facilitation of  $I_{\alpha}$

activity at gamma threshold levels (0.5-1.0 ma) but no post-stimulus reduction in  $I_{\alpha}$  firing rate (Fig. 6).

When the stimulus strength of the cortical stimulus was increased to activate alpha motoneurons (1.0-1.5 ma), two types of responses were seen. In one response (Fig. 7) at 60/sec there was a period of  $I_{\alpha}$  facilitation preceding the onset of EMG activity. At this frequency the EMG was not continuous with the stimulation period while the  $I_{\alpha}$  facilitation was continued through the stimulation period and was also present during the period of EMG activity. At a higher frequency of 100/sec (Fig. 8) there was again  $I_{\alpha}$  facilitation preceding the EMG which, once initiated, was more intense and remained for the rest of the stimulation period.

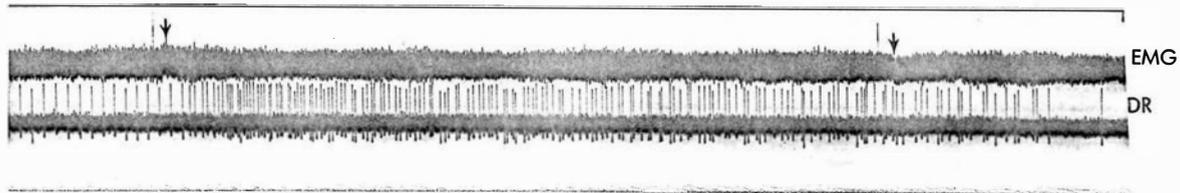
The EMG response to pyramidal tract stimulation was not phasic but was maintained throughout the stimulus period (Fig. 9). The record shows that as the frequency increased the EMG activity increased; the latency between  $I_{\alpha}$  facilitation and EMG activity decreased until they were co-activated; the latency between the onset of the stimulus and  $I_{\alpha}$  facilitation was constant; the  $I_{\alpha}$  facilitation was maintained during the period of EMG activity and there was no off stimulus cessation of spindle firing.

Figure 10 shows an unusual response seen in only four spindles in which there was a decrease of  $I_{\alpha}$  activity preceding the EMG activity. After the EMG had been initiated the  $I_{\alpha}$  activity increased. Although the EMG activity did not last for the duration of the stimulus period, the  $I_{\alpha}$  facilitation did and at the end of the stimulation there was no post-stimulation cessation of spindle firing.

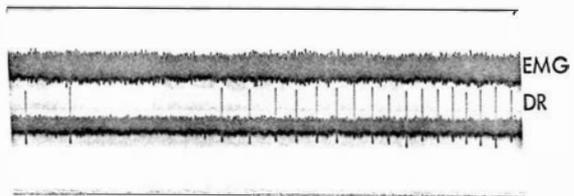
In order to determine what effect, if any, the  $I_{\alpha}$  facilitation which preceded the EMG had on the EMG, the dorsal roots were sectioned in several experiments.

Figure 1. A and B are continuous records of anterior tibialis  $I_a$  afferent and EMG response to cortical stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Stimulation resulted in an increase in  $I_a$  firing rate and no EMG. There was a transient decrease in  $I_a$  firing in the post-stimulus period.

A



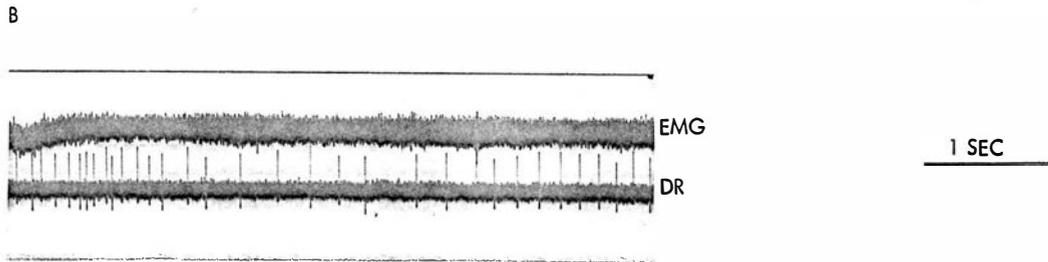
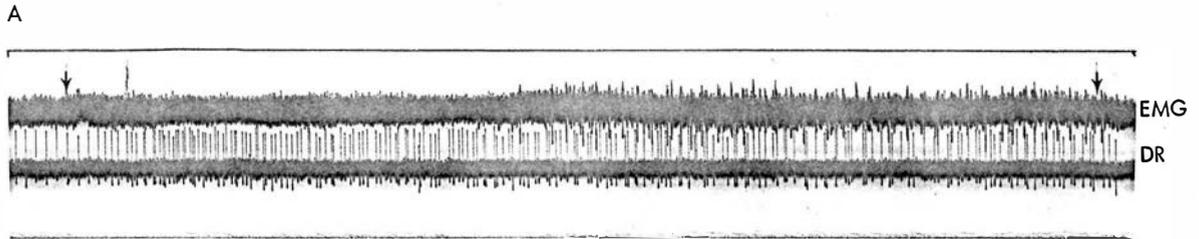
B



1 SEC

RESPONSE OF ANTERIOR TIBIALIS 1<sub>A</sub> AFFERENTS AND EMG TO CORTICAL STIMULATION

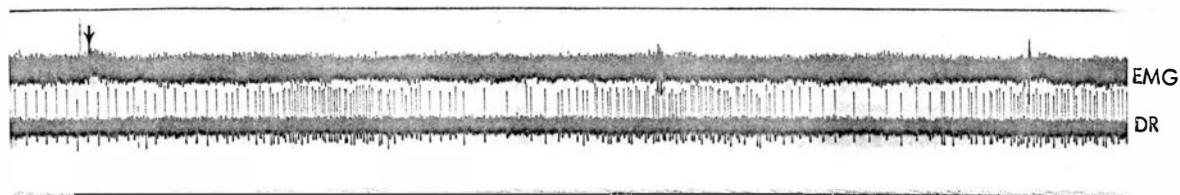
Figure 2. A and B are continuous records of anterior tibialis  $I_a$  afferent and EMG response to cortical stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Stimulation resulted in phasic EMG preceded by an increase in  $I_a$  firing rate. The post-stimulus period was characterized by a reduction of  $I_a$  firing rate.



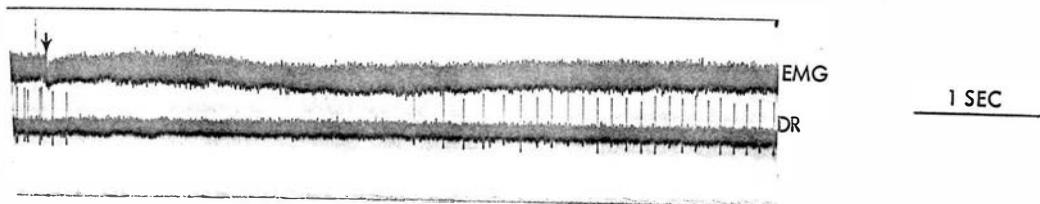
RESPONSE OF ANTERIOR TIBIALIS  $1_A$  AFFERENTS AND EMG TO CORTICAL STIMULATION

Figure 3. A and B are continuous records of anterior tibialis  $I_a$  afferent and EMG response to pyramidal stimulation at 60/sec which resulted in phasic EMG activity preceded by an increase in  $I_a$  firing rate and post-stimulus reduction of  $I_a$  firing. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.

A



B



RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENTS AND EMG TO PYRAMIDAL STIMULATION

Figure 4. A and B are continuous records of anterior tibialis  $I_a$  and EMG response to pyramidal stimulation at 80/sec which resulted in phasic EMG preceded by an increase in  $I_a$  firing rate and post-stimulus cessation of firing. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.

A



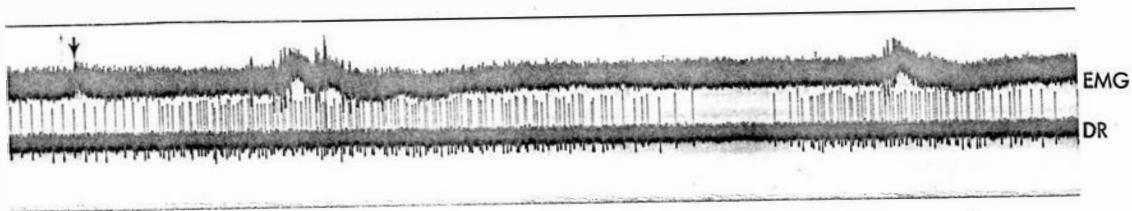
B



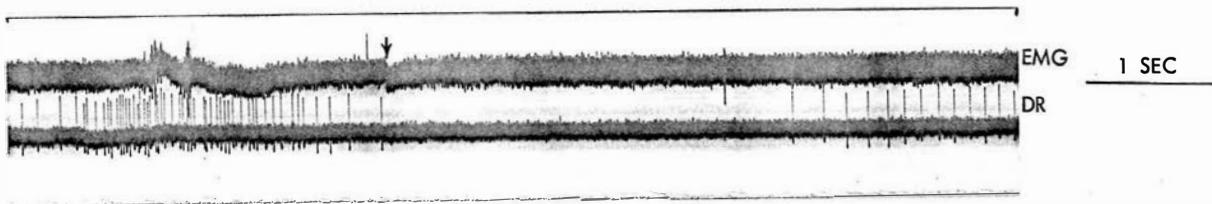
RESPONSE OF ANTERIOR TIBIALIS  $1_A$  AFFERENTS AND EMG TO PYRAMIDAL STIMULATION

Figure 5. A and B are continuous records of anterior tibialis  $I_a$  afferents and EMG response to pyramidal stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Although the EMG was phasic, the  $I_a$  facilitation was maintained through all phases of the EMG.

A



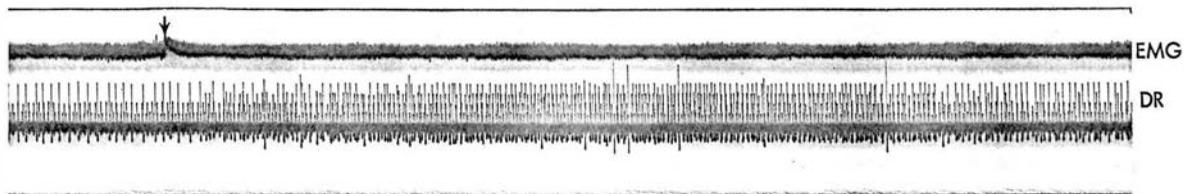
B



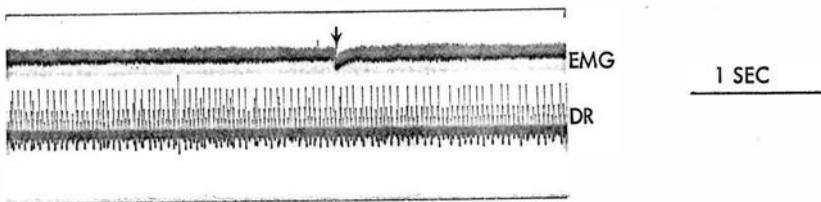
RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENTS AND EMG TO PYRAMIDAL STIMULATION

Figure 6. A and B are continuous records of anterior tibialis  $I_a$  afferents and EMG response to cortical stimulation (60/sec) which resulted in  $I_a$  facilitation and no EMG. There was no post-stimulus  $I_a$  reduction in firing rate. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.

A



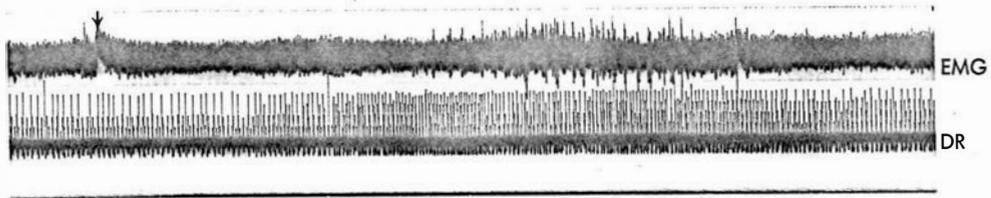
B



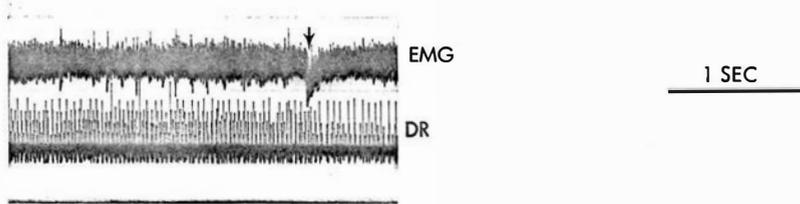
RESPONSE OF ANTERIOR TIBIALIS  $1_A$  AFFERENT AND EMG TO CORTICAL STIMULATION

Figure 7. A and B are continuous records of anterior tibialis  $I_{\alpha}$  afferents and EMG response to cortical stimulation (60/sec) which resulted in EMG preceded by  $I_{\alpha}$  facilitation. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.

A



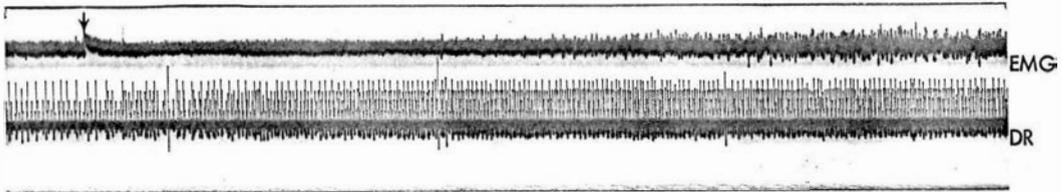
B



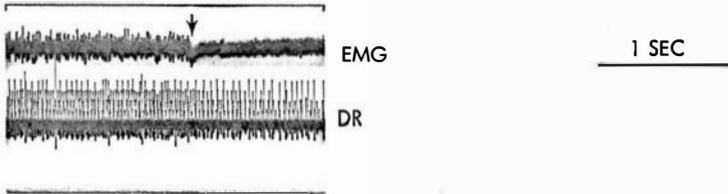
RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENT AND EMG TO CORTICAL STIMULATION

Figure 8. A and B are continuous records of anterior tibialis  $I_a$  afferents and EMG response to cortical stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.  $I_a$  facilitation preceded onset of EMG which once initiated continued for the rest of the stimulus period.

A



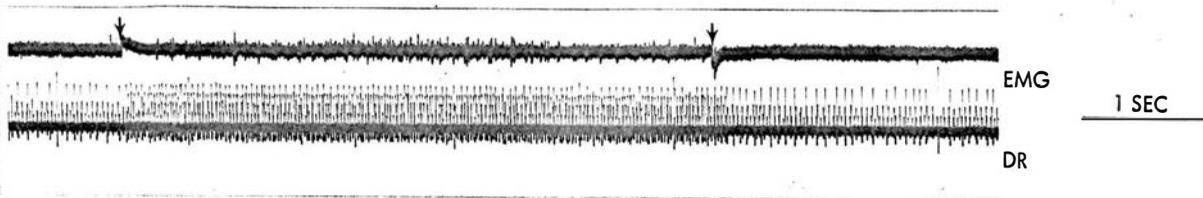
B



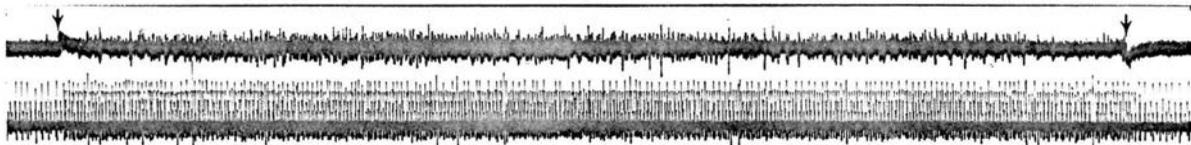
RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENT TO CORTICAL STIMULATION

- Figure 9. Response of anterior tibialis  $I_a$  afferents and EMG to pyramidal stimulation. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.
- A. At 60/sec EMG was preceded by  $I_a$  facilitation.
  - B. At 100/sec latency between stimulus onset and EMG was decreased.  $I_a$  facilitation preceded EMG.
  - C. At 150/sec  $I_a$  facilitation and EMG were co-activated.

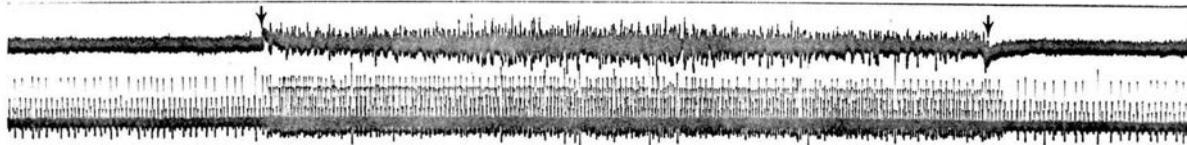
A



B

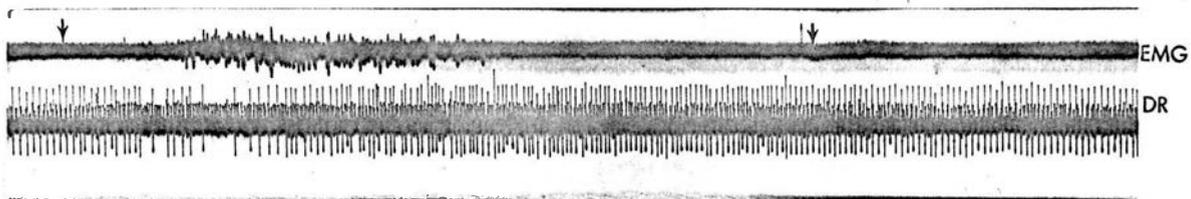


C



RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENT AND EMG TO PYRAMIDAL STIMULATION

Figure 10. Response of anterior tibialis  $I_a$  afferent and EMG to cortical stimulation. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.  $I_a$  firing rate was decreased before onset of EMG activity.



1 SEC

RESPONSE OF ANTERIOR TIBIALIS  $I_A$  AFFERENT AND EMG TO CORTICAL STIMULATION

In all cases deafferentation had no effect on the stimulus threshold levels, latencies or pattern of  $I_a$  facilitation or EMG activity.

## SOLEUS

Generally soleus muscle spindle firing was not as regular as that of the anterior tibialis spindles, but was of phasic nature with periodic increases and decreases. The results of the soleus experiments fell into three classes. Class I represents the most common response of the soleus  $I_a$  afferents. Cortical stimulation resulted in initial firing cessation followed by  $I_a$  firing break-through while pyramidal tract stimulation produced some initial facilitation followed by a reduction in firing rate and post-stimulus firing cessation. Figure 11a,b shows the results at a stimulus frequency of 60/sec.  $I_a$  firing ceased 428 msec after the onset of the stimulus. It can be seen that the spindle began to fire in a phasic manner before the termination of the stimulus, and after the stimulus was terminated the  $I_a$  firing remained phasic in bursts of 2-3 spikes before returning to pre-stimulus levels.

Figure 11c is illustrative of the Class I response to pyramidal tract stimulation. In all cases there is an initial facilitation of  $I_a$  firing followed by phasic inhibition.

With Class II soleus spindles, cortical stimulation produced an initial burst of  $I_a$  firing followed by a cessation of spindle firing with intermittent  $I_a$  firing. Pyramidal tract stimulation produced less reduction in spindle firing but resulted in a post-stimulus cessation of firing. Figure 12 shows a soleus record at 80/sec. After the onset of the stimulus there was a period of  $I_a$  facilitation and no EMG. This was followed by a cessation of  $I_a$  firing followed by a period of decreased  $I_a$  firing for the remainder of the stimulus period. However, during the remainder of the stimulus period

there were bursts of  $I_{\alpha}$  potentials with no EMG. At the termination of the stimulation period there was a period of phasic EMG activity in which each EMG burst was followed by a burst of  $I_{\alpha}$  firing. Each  $I_{\alpha}$  burst was followed by a reduction of or a cessation of  $I_{\alpha}$  firing. This period was followed by a resumption of  $I_{\alpha}$  activity prior to the next EMG burst. After the last EMG burst there still remained a period of phasic  $I_{\alpha}$  firing which gradually ceased. At a stimulus frequency of 100/sec (Fig. 13) the period of initial  $I_{\alpha}$  facilitation occurred after the onset of the stimulus while the period of  $I_{\alpha}$  reduction occurred 288 msec later. For the remainder of the stimulus period there was a reduction in  $I_{\alpha}$  firing interrupted by  $I_{\alpha}$  burst, however, in the post-stimulus period there was no EMG although the phasic  $I_{\alpha}$  firing was still present.

All pyramidal tract stimulations gave the same results and Figure 14 at a stimulus frequency of 100/sec is a representative example. After the onset of the stimulus there was an initial  $I_{\alpha}$  facilitation followed by firing cessation 1276 msec later. The remainder of the stimulation period shows  $I_{\alpha}$  firing cessation interrupted by  $I_{\alpha}$  burst. The post-stimulus period shows some firing cessation followed by a return to pre-stimulus levels. There was no EMG activity. In general, pyramidal tract stimulation had less effect than cortical stimulation.

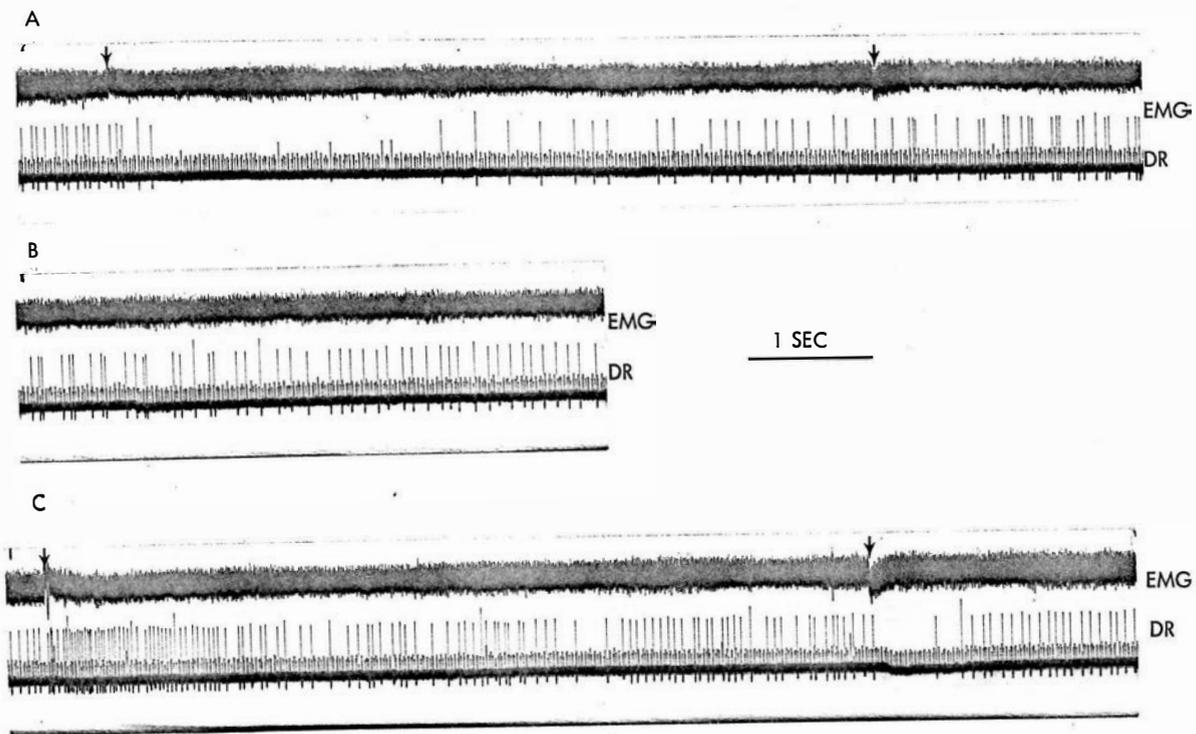
Class III soleus spindles are represented in Figure 15. With these spindles cortical stimulation (Fig. 15a) did not produce any change except for an off response which resulted in a period of firing cessation with phasic bursting until the return to pre-stimulus levels. Note that the apparent change in spindle activity during the stimulus period is the continuous changing spontaneous activity described earlier for these soleus spindles. Pyramidal tract stimulation (Fig. 15b) produced no change in

these spindles except for a slight decrease in firing rate as an off response.

#### MEDIAL AND LATERAL GASTROCNEMIUS

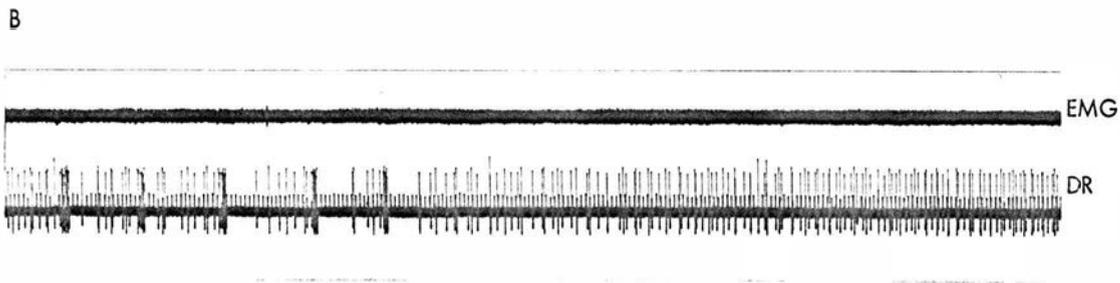
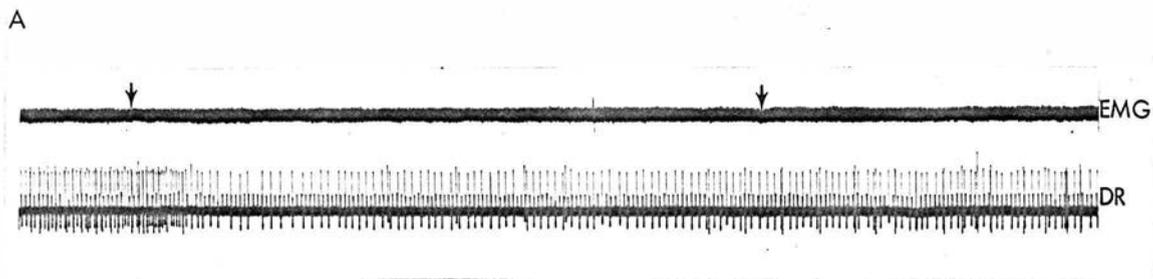
Cortical and pyramidal tract stimulation had no effect on  $I_{\alpha}$  firing from these spindles and EMG activity occurred only at suprathreshold intensities which produced post-stimulus epileptoid contractions. The same was also true for the soleus.

- Figure 11. Response of Class I soleus  $I_a$  afferent and EMG to cortical and pyramidal stimulation. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.
- A and B. Continuous records of response to cortical stimulation which resulted in inhibition of  $I_a$  firing.
- C. Response to pyramidal stimulation which resulted in initial  $I_a$  facilitation followed by inhibition.



RESPONSE OF SOLEUS CLASS I<sub>A</sub> AFFERENTS AND EMG TO CORTICAL AND PYRAMIDAL STIMULATION

Figure 12. A and B are continuous records of Class II soleus  $I_a$  afferents and EMG response to cortical stimulation at 80/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Stimulus produced initial facilitation followed by inhibition. Stimulus termination resulted in phasic EMG and  $I_a$  bursts.



1 SEC

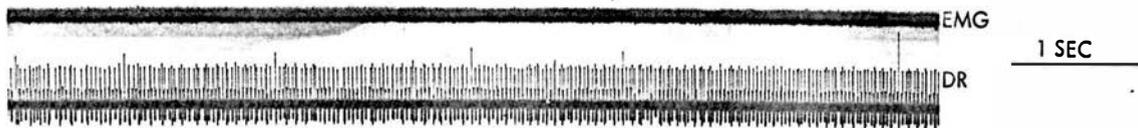
RESPONSE OF SOLEUS CLASS II  $I_A$  AFFERENT AND EMG TO CORTICAL STIMULATION

Figure 13. A and B are continuous records of Class II soleus  $I_{\alpha}$  afferents and EMG to cortical stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Stimulus resulted in initial  $I_{\alpha}$  facilitation followed by inhibition. The post-stimulus period was characterized by phasic  $I_{\alpha}$  burst and no EMG.

A

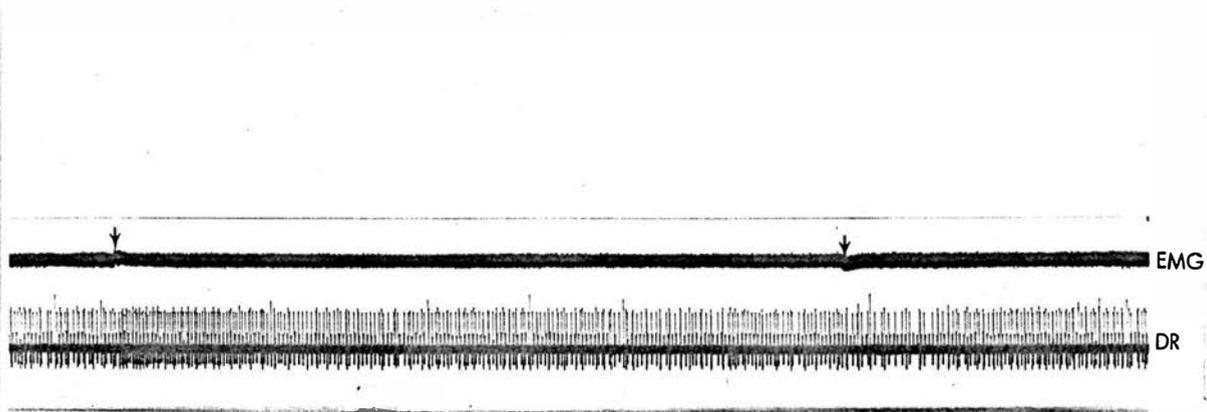


B



RESPONSE OF SOLEUS CLASS II  $I_A$  AFFERENTS AND EMG TO CORTICAL STIMULATION

Figure 14. A and B are continuous records of Class II soleus  $I_a$  afferents and EMG response to pyramidal stimulation at 100/sec. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow. Stimulus produced initial  $I_a$  facilitation followed by inhibition.



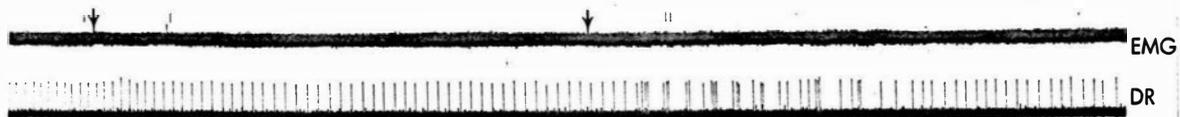
1 SEC

RESPONSE OF SOLEUS CLASS II  $I_A$  AFFERENTS TO PYRAMIDAL STIMULATION

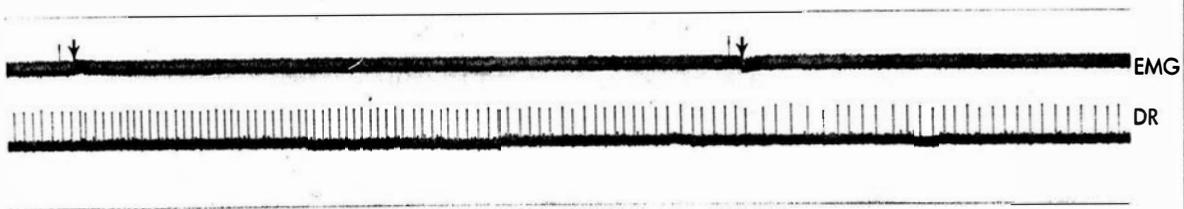
Figure 15. Class III soleus  $I_{\alpha}$  afferents and EMG response to cortical and pyramidal stimulation. The stimulus onset is indicated by the first arrow and the termination of the stimulus is indicated by the second arrow.

- A. Cortical stimulation resulted in post-stimulus inhibition.
- B. Pyramidal stimulus produced slight post-stimulus reduction of  $I_{\alpha}$  firing rate.

A



B



1 SEC

RESPONSE OF CLASS III SOLEUS  $1_A$  AFFERENTS AND EMG TO CORTICAL AND PYRAMIDAL STIMULATION

## DISCUSSION

## Anesthesia

Any neurophysiological study is influenced by the anesthesia. Initially alpha chloralose which has properties of being both an anesthetic and stimulant (Balis and Monroe, 1964) was selected. It is well known that alpha chloralose produces a state of motor hyperexcitability. It was considered that this hyperexcitability is due to actions on the cerebral cortex since pigeons, which have little cortex, do not show the hyperexcitability, and in mammals the hyperexcitability is abolished by decerebration. However, the threshold of the cortex to electrical stimulation was found to be elevated with alpha chloralose and the stimulus intensity had to be increased with each successive stimulus so that after 6-8 test stimuli the current intensity was so high that it resulted in burning of the cortex. This observation has also been reported by Wyss and Obrader (1937) and Asanuma et al. (1968). Although Nembutal is considered as highly depressant to the central nervous system, Keller and Fulton (1931) reported that Dial, sodium amytal, chloral hydrate and sodium barbital leave the cortex excitable even when suprasurgical doses are administered. Nembutal was stated to have little effect on cortical excitability when given in light anesthetic doses (25-30 mg/kg) but under full surgical doses (35-40 mg/kg) the excitability of the cortex was depressed. In the present experiments the data were obtained under conditions of light anesthesia and there was no difficulty in obtaining modification of  $I_G$  activity. Woolsey et al. (1952) found that the results in mapping studies are more constant and reproducible from one stimulation to the next and from one animal to the next with Nembutal than with other anesthetics. Furthermore, since spontaneous activity of the cortex is re-

duced, the cortex is more stable than when electrical excitability is marked. Finally, comparisons between decerebrate and lightly anesthetized cats (Brookhart, 1952) showed that although anesthesia slightly depresses motoneuron response, it seems to emphasize preexisting differences between muscle groups. Therefore, results obtained under Nembutal probably reflect the basic organization of the efferent cortical pathways quite accurately.

#### Motor Map

The hindlimb motor area was reported to be in the posterior sigmoid gyrus at the junction of the cruciate sulcus and the sagittal fissure by several investigators (King, 1911; Langworthy, 1928; McKibben, 1929; McKibben and Wheelis, 1932; Garol, 1942; Mortimer and Akert, 1961). In the present experiments the hindlimb motor area was found to be on the cruciate sulcus 3-5 mm from the midline, in agreement with Ward and Clark (1936), Clark and Ward (1937), Livingston and Phillips (1957), and Ashly et al. (1972). The finding that the alpha and gamma motor maps were co-extensive is in accord with Mortimer and Akert (1961). The response from a given point in an animal was constant throughout the course of the experiment, which lasted until cortical circulation became poor (Adrian, 1936; Lilly et al. 1952). Variability in response of a given point in acute experiments was reported by Brown and Sherrington (1912), Leyton and Sherrington (1917), Boyton and Hines (1933) and Dusser de Barenne (1936), and Tower (1935) found extensor points in the cat to be variable. In chronic experiments in dogs (Lashly, 1923; Clark, 1941) cats (Clark and Ward, 1937), monkeys (Chang et al. 1946) and man (Penfield and Boldrey, 1937) the response of a

point on a given day was the same, but the response of the point may change from one day to the next. In chronic experiments, rough handling and/or the presence of a loud noise may result in variability of a response (Ward, 1938). The most likely cause for the instability of response observed by earlier investigators was not allowing enough time between stimulations to permit the cortex to recover from the previous stimulation(s) (Dusser de Barenne and McCulloch, 1932; Ward and Clark, 1936; Clark and Ward, 1937; Murphy and Gellhorn, 1945; Clark, 1948; Woolsey et al. 1952; Cure and Rasmussen, 1954).

The hindlimb motor points showed overlaps in two regards: 1) the points for inhibition and facilitation were the same; 2) the muscle representation in the motor cortex was the same. A lack of facilitation - inhibition separation has been reported in the monkey by Preston and Whitlock (1960) and Asanuma and Sakata (1967). The wide degree of muscle overlap is well known in the cat (Ward and Clark, 1936; Bernhard and Bohm, 1954; Chambers and Liu, 1957; Livingston and Phillips, 1957; Uemura and Preston, 1965), primate (Franz, 1915; Dusser de Barenne et al. 1941; Murphy and Gellhorn, 1945; Bosma and Gellhorn, 1947a,b; Liddell and Phillips, 1951; Woolsey et al. 1952; Phillips and Porter, 1964; Landgren et al. 1962a,b; Asanuma and Sakata, 1967), and man (Rasmussen and Penfield, 1947) although more localized muscle representation may be evident with high frequency stimulation (Liddell and Phillips, 1950) and deep anesthesia (Clark and Ward, 1937) and in the monkey there may be restricted bands in which some points produce movement of a single muscle (Foerster, 1931; Chang et al. 1946) or parts of a single muscle (Foerster, 1931). Recently, using

focal stimulation with a microelectrode with a tip diameter of 10-15 $\mu$  and a current spread of only 88 $\mu$  (Stoney et al. 1968) and currents of 2 $\mu$ a Asanuma and Sakata (1967) found muscles to have cortical fields of 0.5 to several millimeters in the cat. Even in these experiments there was an overlap in muscle representation. The lack of discrete localization is not unexpected because of the high degree of apical dendritic branching of the pyramidal tract cells (Kaiserman-Abramof and Peters, 1972) which can lead to the excitation of pyramidal tract cells 4 mm away from the point of cortical stimulation (Phillips, 1956). Also, the lack of monosynaptic synapses on the alpha motoneurons would tend to lead to a less discrete activation of muscles (Bernhard and Bohm, 1954). This is true for primates as well as for cats, since only a portion of the pyramidal tract terminals are monosynaptic (Preston and Whitlock, 1961). It should be observed that in primates the area of widest cortical extent, the hand, also has the highest degree of localization (Beevor and Horsley, 1887; Franz, 1915; Penfield and Boldry, 1937; Walshe, 1943; Liddell and Phillips, 1951; Woolsey et al. 1952; Asanuma and Sakata, 1967). This high degree of localization correlates with the fact that the hand muscles have the highest degree of monosynaptic connections from the cortex (Phillips and Porter, 1964; Preston et al. 1967; Clough et al. 1968).

#### Flexor Facilitation Versus Extensor Inhibition

These experiments produced a pattern of flexor facilitation and extensor inhibition with cortical and pyramidal tract stimulation. This pattern with cortical stimulation has been observed in the opossum (Turner, 1924), rabbit (Laughton, 1928), rat (Barron, 1934), dog (Laughton, 1928; Smith, 1933; Woolsey, 1933), cat (Sander-

son, 1874; Warner and Olmsted, 1923; Weed and Langworthy, 1926; King, 1927; Langworthy, 1928; Tower, 1935, 1936; Ward and Clark, 1936; Mettler and Mettler, 1940; Smith et al. 1940; Garol, 1942; Brookhart, 1952; Corazza et al. 1963; Kato et al. 1964; Agnew and Preston, 1965; Jankowska and Tarnecki, 1965; Uemura and Preston, 1965; Asanuma and Sakata, 1967; Preston et al. 1967), monkey (Beevor and Horsley, 1887; Horsley and Schafer, 1888; Brown and Sherrington, 1912; Hines, 1940; Bosma and Gellhorn, 1947; Chang et al. 1946, 1947; Uemura and Preston, 1965), and man (Ranson, 1892; Cushing, 1909; Penfield and Boldry, 1937). Flexion is more common than extension when the latter is present (Brown and Sherrington, 1912; Hines, 1940; Brookhart, 1952; Corazza et al. 1963; Bosma and Gellhorn, 1947; Asanuma and Sakata, 1967). In the opossum (Gray and Turner, 1924) extension occurs only occasionally. Extension is also rare in the monkey and cortical foci for extensors are difficult to find (Chang et al. 1946, 1947). In man, Penfield and Boldry (1937) found a ratio of flexor points to extensor points of four to one. Tower (1935) found the extensor points in the cat to be few and very unstable.

Recording intracellularly from unidentified alpha motoneurons of the cat, Preston and Whitlock (1961) found cortical stimulation to result in EPSPs in 50 of 59 units, IPSPs in 6 units and EPSPs plus IPSPs in 3 units. Similar results were reported in the rat by Bannister and Porter (1967). Lundberg and Voorhoeve (1962) found EPSPs predominately in flexor motoneurons and IPSPs in extensor motoneurons, with EPSPs in all but a few flexor units. Similar studies by Kato et al. (1946) of hindlimb alpha motoneurons showed all flexors to produce EPSPs as a result of cortical stimulation. In the extensors, 6 of 16 units showed EPSPs followed by IPSPs, while the remainder showed

IPSPs (8) or a mixture of EPSPs and IPSPs (2). The absence of extensor facilitation in these experiments is in agreement with King (1911), Weed and Langworthy (1926), Laughton (1928), Woolsey (1933), Smith (1933), Smith et al. (1940), Bosma and Gellhorn (1947) and Kato et al. (1964). A similar picture of flexor facilitation and extensor inhibition has also been seen with pyramidal tract stimulation in the rat (Bannister and Porter, 1967) and in the cat (Landau, 1952; Phillips, 1959; Laursen and Wiesendanger, 1966).

The pattern of flexor facilitation and extensor inhibition has been demonstrated by studies of cortical and pyramidal tract influence on synaptic transmission in the spinal cord. Morrell (1957) stimulated the pyramidal tract in decerebrate cats and found five interneurons were facilitated and three inhibited. Sasaki et al. (1960) found that the monosynaptic reflex to both flexors and extensors was equally facilitated by pyramidal tract stimulation, while Agnew et al. (1963) and Preston and Whitlock (1963) found the  $I_{\alpha}$  monosynaptic path to flexors to be facilitated while the  $I_{\alpha}$  monosynaptic path to the extensors was inhibited. Lundberg and Voorhoeve (1960, 1962) found that stimulation of anterior tibialis  $I_{\alpha}$  afferents produced IPSPs in gastrocnemius and soleus motoneurons which could be enhanced by cortical stimulation. However, Lundberg and Voorhoeve (1960) were unable to show  $I_{\alpha}$  EPSP facilitation due to cortical stimulation. Although the inhibitory actions of the  $I_{\alpha}$  afferents on the antagonist muscle could be enhanced by cortical stimulation in both flexors and extensors, the enhancement was most easily seen in the actions of  $I_{\alpha}$  afferents from flexors acting on extensors (Lundberg and Voorhoeve, 1962).

All polysynaptic reflex pathways to alpha motoneurons can be facilitated by cortical stimulation (Lundberg, 1964). The inhibitory action of  $I_b$  afferents, Group II and III muscle afferents and high threshold joint afferents on extensors, as well as the excitatory actions of these afferents on flexor motoneurons can be enhanced by cortical stimulation (Lundberg and Voorhoeve, 1960, 1962). In flexors the facilitation is predominantly on excitatory pathways while the inhibitory pathways are facilitated in the extensors. These actions are mediated by interneurons. Cortical and pyramidal tract stimulation can cause potential changes in the interneuronal pools producing both facilitation and inhibition (Lloyd, 1941; Lance, 1954; Lindblom and Ottosson, 1957; Hagbarth and Felix, 1959). Interneurons that are excited by Group I muscle afferents and flexor reflex afferents are facilitated (show EPSPs) by cortical stimulation, while those interneurons which are inhibited by these afferents produce IPSPs in response to cortical stimulation (Lundberg et al. 1962). Lundberg (1964) has suggested that the reflex is the unit reaction upon which the corticospinal tract operates.

Thus, the cortical output to motoneurons seems to be one of flexor facilitation and extensor inhibition. However, the inhibition to the extensors is not equal. In the present experiments, the soleus  $I_a$  activity was found to be inhibited while the  $I_a$  activity of the gastrocnemius was unaffected. In the cat (Agnew and Preston, 1965; Preston and Whitlock, 1963; Preston et al. 1967) the soleus is more inhibited by cortical stimulation than the gastrocnemius in both degree and duration. Thus, the degree of inhibition seems to be greater in the slow tonic, antigravity soleus (Denny-Brown, 1929). This is consistent with the observation by Tower and Hines

(1935) that cortical stimulation results in an inhibition of tonic states. Furthermore, in the sloth (Langworthy, 1935) and arboreal opossum (Hare and Porter, 1972) cortical and pyramidal tract stimulation result in extensor facilitation and flexor inhibition. Thus, in these animals, where the functional role of the anatomical flexors and extensors is reversed, the pattern of cortical and pyramidal tract facilitation and inhibition is also reversed. In the arboreal opossum the inhibition is greatest in the flexor digitorum longus, a major tonic antigravity muscle, and the nonpostural gastrocnemius and hamstring muscles are facilitated. Further evidence that the postural muscles are favored by inhibition is the fact that the degree of inhibition to the wrist extensors in the cat is less than to the foot extensors (Preston et al. 1967) and some facilitation is seen in the wrist extensors which relates to a greater use of the forelimb for non-locomotor activities. A similar pattern is seen in the baboon where there is more forelimb flexor inhibition and less extensor inhibition than is seen in the hindlimbs. Finally, unlike the cat, the ankle extensors of the primate show less inhibition and more facilitation (Uemura and Preston, 1965). This also relates to posture since in the primate the angle of the foot is such that the ankle extensors carry less of the antigravity load because the primate heel is on the ground, while in the cat the ankle joint is elevated above the ground and the ankle extensors therefore are more important in posture (Uemura and Preston, 1966; Preston et al. 1967).

The negative results in finding no gastrocnemius inhibition in these experiments may be explained in two ways. First, for the cortex to play a role in the initiation of volitional movement, tonic antigravity postural segmental reflexes must be inhibited (Preston and Whitlock, 1963; Preston et al. 1967). Of the two ankle exten-

sors in the cat, the soleus is the more tonic (Denny-Brown, 1929) and used more in posture (Preston and Whitlock, 1963) and in the standing cat, the soleus is the only ankle extensor showing EMG activity (Smith, 1972). Hence, one would expect to see soleus inhibition and no gastrocnemius inhibition. Second, small doses of pentobarbital (5 mg/kg) reduce or obliterate cortical and pyramidal tract inhibition of monosynaptic reflexes while permitting the facilitation of the monosynaptic reflexes (Preston and Whitlock, 1960). If the inhibitory pathways are more sensitive to anesthesia and the degree of inhibition is less in the gastrocnemius than the soleus, gastrocnemius inhibition may be blocked by the anesthesia under the conditions of these experiments.

The present experiments demonstrate the threshold for gamma activation to be lower than the threshold for alpha activation with cortical stimulation. These results are in agreement with Hern and Phillips (1959) and Hern et al. (1962). The fact that it was not possible to activate alpha motoneurons selectively with pyramidal tract stimulation suggests that there is minimal separation in the projection to these two motoneuron groups.

### Servo-Control Theory

The observation that gamma motoneurons were activated before alpha motoneurons leads to the possibility that voluntary movement could be initiated via the gamma motor system. In 1951, Merton recorded the EMG and myogram from the adductor pollicis in man during steady voluntary contraction and found the silent period was present only if a stimulus to the motor nerve was strong enough to cause

a twitch. Merton (1953) suggested that a facilitation of gamma motoneurons during contraction could prevent the loss of length information during contraction. It was also suggested that if the gamma discharge occurred before alpha activation it would be possible to voluntarily initiate movement via the gamma motor system. The signal for the initiation of a movement would be sent down from supraspinal centers to the gamma motoneuron pool of a muscle eliciting a discharge in the gamma motoneurons. This gamma discharge would result in the contraction of the intrafusal muscle fibers of the muscle spindles which in turn would cause the stretch sensitive receptor area of the spindle to be stretched resulting in the generation of impulses in the  $I_{\alpha}$  fibers innervating the spindle. This  $I_{\alpha}$  discharge would produce a subsequent discharge in the muscle's alpha motoneuron pool thereby initiating contraction of the extrafusal muscle fibers, resulting in movement. Because of the delay due to conduction around the gamma loop, this type of voluntarily induced movement could only be used in slow movements. Supraspinal centers would have two mechanisms for initiating movement: 1) by direct pathways to the alpha motoneurons, and 2) by indirect pathways through the gamma motoneurons via the gamma loop. Lennerstrand and Thoden (1968) showed that dynamic fibers produce an intrafusal contraction which is too slow to keep up with the rapid contraction of the extrafusal fibers. However, Lennerstrand and Thoden did suggest that the static fibers could increase discharge from the  $I_{\alpha}$  fibers quickly enough to transmit a command signal for muscle contraction and to form a part of a servo control system for initiating movement. Recently, Perret and Buser (1972) recorded anterior tibialis  $I_{\alpha}$  afferent fibers in the cat and reported an increased firing rate with each contralateral limb extension with or without EMG activity. When EMG

activity was present, it was always preceded by increased  $I_{\alpha}$  firing which continued during and after the EMG. These findings are almost identical to those of the present study in which the  $I_{\alpha}$  facilitation and EMG were elicited by electrical stimulation of the cortex and pyramidal tract.

Several criticisms to the servo-control theory initiation of voluntary movement have been raised: 1) stimulation of the gamma motor fibers does not result in the production of tension in the extrafusal muscle fibers (Hunt, 1951; Kuffler and Hunt, 1952; Leksell, 1945); 2) the time delay around the gamma loop (11-30 msec) is too long for most voluntary movements (Hunt, 1951; Granit et al. 1959; Andersson et al. 1968); 3) those alpha motoneurons which are the most excited by  $I_{\alpha}$  afferents are the small, slow, tonic, motoneurons involved in posture and not the phasic activity of voluntary movement (Denny-Brown, 1929; Granit et al. 1956, 1957; Eccles et al. 1957a,b; Granit, 1958); 4) the  $I_{\alpha}$  input to the spinal cord is not restricted to the homonomous muscle but is widespread, making discrete or selective alpha activation difficult (Eccles et al. 1957a,b; Eccles and Lundberg, 1958); 5) in the present experiments, deafferentation had no effect on the latencies or pattern of  $I_{\alpha}$  facilitation and EMG activity, in agreement with Lewis and Porter (1971), suggesting that the involvement of the fusimotor system in the production of EMG activity was minimal with electrical stimulation of the motor cortex and pyramidal tract. Most damaging to the servo-control theory as a means of initiating movement has been the failure to observe gamma activity preceding alpha activity under conditions of normal movement. Studies of respiratory muscles by Critchlow and von Euler (1963), Sears (1964) and von Euler (1966) show gamma and alpha motoneurons firing at the same time. Studies made on

decerebrate walking in cats (Severin et al. 1967), jaw movement in cats (Taylor and Davey, 1968), hand movement in the baboon (Koeze, 1968; Koeze et al. 1968) and finger movement in man (Vallbo, 1967, 1969, 1970, 1971; Hagbarth and Vallbo, 1968, 1969) have all shown gamma and alpha motoneurons to fire together with no gamma activity preceding the alpha activity. In all cases studied to date the gamma and alpha motoneurons were co-activated.

Several explanations for the significance of this co-activation have been suggested. The presence of a silent period during voluntary movement would prevent the central nervous system from having information as to the length of the muscle during its contraction. Merton (1953) proposed that gamma induced spindle discharge during voluntary contraction would prevent the loss of this information. Buchwald and Eldred (1961) suggested that gamma activity induced from supraspinal areas may contribute to the general level of spinal cord excitability whereby alpha motoneurons could be partially depolarized in preparation for movement. More widely accepted (Clough et al. 1968; Phillips, 1969; Matthews, 1972) is the view that alpha and gamma motoneurons are co-activated at levels appropriate for an intended rate of shortening. If the rate is slowed by unexpected resistance,  $I_a$  activity due to the gamma bias would be increased and lead to an enhancement of the alpha activity. This mode of movement has been termed servo-assisted (Matthews, 1972), and has been shown to occur in the intercostal respiratory muscles (Corda et al. 1965).

The present experiments suggest the output of the motor cortex and pyramidal tract of the cat is in part designed to inhibit the tonic antigravity extensor muscles and facilitate the flexor muscles. Indeed, the pattern of facilitation of the anterior

tibialis, inhibition of the soleus and unaffected gastrocnemius is what one would expect in a cat initiating a step, since the initial phase of the step cycle is flexion (Goslow et al. 1973) at which time the activity in the postural muscles must be inhibited. These findings are in accord with studies in which lesions of the motor cortex and pyramidal tract were reported to result in extensor hypertonus due to a withdrawal of extensor inhibition and flexor hypotonus as a result of a withdrawal of flexor facilitation.

## SUMMARY

1. The effect of cortical and pyramidal tract electrical stimulation on the gamma motor system was studied in nembutalized cats by recording primary sensory receptor firing over teased dorsal root filaments. The following muscles were studied: anterior tibialis, soleus and gastrocnemius.
2. Alpha and gamma cortical motor areas were located on the cruciate sulcus 3-5 mm from the midline.
3. There was no cortical area separation between hind- and forelimb, between the muscles under study or between facilitory and inhibitory stimulation points.
4. Cortical and pyramidal stimulation resulted in facilitation of anterior tibialis  $I_{\alpha}$  afferents, with gamma motoneuron thresholds lower than alpha motoneuron thresholds with cortical stimulation. With pyramidal tract stimulation, thresholds were the same for both neuron groups.
5. When EMG was present it was preceded by increased  $I_{\alpha}$  firing rates which were maintained during and after the period of EMG activity.
6. Cortical and pyramidal stimulation resulted in inhibition of soleus  $I_{\alpha}$  afferent activity.
7. Cortical and pyramidal stimulation did not affect  $I_{\alpha}$  afferent activity from the gastrocnemius.
8. Deafferentation had no effect on the pattern of  $I_{\alpha}$  facilitation and EMG activity in anterior tibialis muscle in these experiments.
9. It was concluded that one of the functions of the motor cortex and pyramidal tract is to facilitate flexor muscles and to inhibit tonic anti-gravity muscles in preparation for movement.

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