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THRESHOLDS OF CORTICAL ACTIVATION OF BABOON α- AND γ-MOTONEURONES DURING HALOTHANE ANAESTHESIA

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SUMMARY

1. In the halothane anaesthetized baboon the threshold for cortically elicited primary and secondary spindle afferent discharge was compared to the threshold at the 'best point' for a cortically elicited tibialis anticus tension change.

2. The cortical threshold for eliciting a tension change was greater than the cortical threshold for eliciting spindle afferent discharge for some afferents and less for other afferents.

3. The pattern of cortically elicited spindle afferent discharge during muscle stretch suggested static γ-motoneurone activation. No convincing evidence of dynamic γ-motoneurone activation was seen.

4. Inhibition of spindle afferent discharge was occasionally found and the 'best point' was the same as the cortical 'best point' for eliciting monosynaptic α-motoneurone activity. This result suggested that the cortical projections which inhibit the pre-existent activity of static γ-motoneurones are in a close anatomical relation to those cortical projections which elicit activity of α- and static γ-motoneurones.

INTRODUCTION

Koeze, Phillips & Sheridan (1968) reported the effects of cortical stimulation upon the discharge of five extensor digitorum communis spindle afferents and muscle tension at different levels of anaesthesia in the barbiturate anaesthetized baboon. During deep anaesthesia the cortical threshold for activation of four of the spindle afferents was lower than the cortical threshold for eliciting a muscle contraction. During light anaesthesia the cortical threshold for eliciting a muscle contraction was lower than the cortical threshold for eliciting a spindle afferent discharge. Similar

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studies of a baboon hind-limb muscle, tibialis anticus (TA) were different in that the threshold for eliciting muscle contraction was always lower than that for eliciting spindle afferent discharge (Koeze, 1968). At all levels of barbiturate anaesthesia spindle afferent discharge acceleration before a detectable tension change (γ-leading) in response to cortical stimulation was never seen.

Studies of the central control of cat γ-motoneurones have shown that the effects on spindle afferent discharge elicited by stimulation of the red nucleus (Appelberg, 1966) and of the sensorimotor cortex (Vedel & Mouillac-Baudevin, 1970) are critically dependent upon the level of anaesthesia. In these studies a short-acting volatile anaesthetic, halothane (Fluothane, ICI), was used and it was possible to selectively activate dynamic and static γ-motoneurones at different levels of anaesthesia.

This study was undertaken to assess the threshold for cortical activation of α- and γ-TA motoneurones of the baboon anaesthetized with halothane. Specifically, two questions were asked. What is the relationship between the cortical thresholds for activation of α- and γ-motoneurones? Are both dynamic and static γ-motoneurones activated during cortical stimulation and if so what is the relationship between their cortical thresholds?

The thresholds were judged by simultaneous comparison of muscle tension and spindle afferent discharge (Granit & Kaada, 1952). Although this method is less direct than recording the axonal discharge of the motoneurones, by examining the effects of cortical activation upon the spindle afferent response during a muscle stretch the type of γ-motoneurone activated can be assessed. Dynamic γ-motoneurone activation influences only the primary spindle afferent response; static γ-motoneurone activation influences both primary and secondary afferent response (Appelberg, Bessou & Laporte, 1966). Comparisons of the discharge of the two types of afferent during cortical stimulation gave additional evidence about the type of motoneurone activated.

METHODS

The studies were performed on the spindle afferents of the right TA muscle of eight immature baboons (Papio sp., average 5·8 kg wt.). The responses of twenty-one spindle afferents were examined. Eleven of these were classified as primary afferents (conduction velocity, c.v. $>$ 68 m/sec), seven were classified as secondary afferents (c.v. $<$ 50 m/sec) and three had intermediate conduction velocities and therefore could not be definitely classified (Koeze, 1973). Threshold studies were made of seventeen afferents. The threshold of the remaining four afferents were not studied either because they showed inhibitory effects (two) or because they were damaged during the threshold testing procedure (two).

Anaesthesia was induced with a mixture of 70% N_2O, 30% O_2 and 3% halothane. The halothane concentration was controlled with a Foregger Fluomatic vaporizer. No premedication was given. Anaesthesia was always maintained with

the same 70 % N_2O and 30 % O_2 mixture supplemented with halothane at a concentration suitable for the depth of anaesthesia desired. In other respects the preoperative, operative, fixation and muscle nerve isolation procedures used were as previously described (Koeze, 1968).

The isolation and identification of the spindle afferents, the measurement of conduction velocity, the recording of the action potentials and the measurement of the 'instantaneous' frequency were the same as reported elsewhere (Koeze, 1968, 1973). The myograph used to record the tension of elicited contractions and the stretcher used to extend the muscle was the same as described previously (Koeze, 1973).

The cortical 'best point' was defined as the point which gave the largest contraction of the test muscle at the lowest current strength for a brief burst of impulses at a frequency at 250 Hz, 0·2 msec pulse duration. The usual procedure was to find the 'best point' during moderately deep anaesthesia before the spindle afferents were isolated. If the anaesthetic level was too deep the concentration of the halothane was reduced until a stimulus of 4–5 mA elicited a small contraction of TA.

The cortical stimulating electrode was a 1 mm diameter silver ball coated with silver chloride and held by a device (Hern, Landgren, Phillips & Porter, 1962) kindly supplied by the late Dr E. H. J. Schuster. This device allowed the points stimulated to be 'mapped' in relation to other structures such as the central sulcus. The stimulus was always constant current and surface anodal. The cathodal electrode was a large silver plate, wrapped in saline soaked cotton and sutured under the skin over the temporalis muscle on the right side. The constant current was obtained from a battery-driven device. Feed-back current amplifiers maintained a constant current so long as the impedance did not exceed 10 kΩ. The duration and frequency of the stimulus was controlled by Tektronix pulse generators.

Spindle afferents were isolated when the animal was deeply anaesthetized with 1–2 % halothane. In this state there were no spontaneous movements, respiration was slow and shallow, the corneal reflex was absent and the spindle afferent discharge was regular. This level of anaesthesia was usually present for 20–30 min before threshold testing began. During deep anaesthesia the threshold was determined for either a small muscle contraction or spindle afferent discharge acceleration for a 3·2 sec stimulation of either 50, 100 or 250 Hz (pulse duration 0·2 msec). After the threshold determination during this deep level of anaesthesia, the halothane concentration was either reduced or discontinued. As the level of anaesthesia decreased, threshold determinations were made at 1 min intervals. These determinations continued until spontaneous movements of tail or leg appeared. The depth of anaesthesia was then increased by increasing the halothane concentration and the procedure was repeated using either the same or some other frequency of stimulation. In some experiments the procedure was prolonged by carefully adjusting the halothane concentration to maintain a certain level of anaesthesia so that very accurate determinations of the threshold could be made. A 1 min interval was always allowed between the 3·2 sec periods of cortical stimulation.

Excessive halothane concentrations can produce profound hypotension and cardiac arrhythmias. The respiratory CO_2 and aortic blood pressure were continuously monitored in these experiments. The systolic blood pressure was kept above 70 mm Hg by decreasing the halothane concentration. The usual range of systolic blood pressure was 80–120 mm Hg. As far as could be determined the cortical circulation was adequate. Examination of the cerebral vessels with a dissecting microscope did not show marked vasoconstriction and the cortical thresholds were comparable with those at higher blood pressures. Arrhythmias were detected as changes in cardiac rate and were easily corrected by reducing the concentration of halothane.

Occasionally towards the end of a long experiment the blood pressure fell below 70 mm Hg. Attempts were made to raise the blood pressure with various vasoconstrictive drugs and plasma expanding compounds. Any restoration of the blood pressure was usually only temporary and the experiments had to be ended.

RESULTS

Simultaneous recordings of muscle tension and spindle afferent discharge were made during cortical stimulation. If these records showed, for example, a minimal contraction (1–10 g wt.) without any evidence of spindle afferent coactivation, the strength (mA) of the cortical stimulus was taken as the threshold for α-motoneurone activation and that threshold was judged to be lower than the threshold for γ-motoneurone activation. If the spindle afferent discharge accelerated without an associated muscle tension change then the strength (mA) of the cortical stimulus was judged to be at the threshold for γ-motoneurone activation.

At the threshold for the α-motoneurone activation there was usually a small tension increase during the last second of stimulation when the frequency of stimulation was 50 or 100 Hz. At threshold the 250 Hz stimulation often elicited a small initial increase in tension followed by relaxation and another tension increase during the last second. The strength of the stimulus required for the threshold effects appeared to be independent of the stimulus frequency but this was not carefully checked in every experiment.

TABLE 1. Effect of the level of anaesthesia on the cortical threshold for activation of α- and γ-motoneurones

Spindle afferent	α-threshold $< \gamma$ at all levels	α- and γ-threshold approx. equal at all levels	α-threshold $< \gamma$ during deep levels but γ-threshold $\lessapprox \alpha$ during light levels	γ-threshold $< \alpha$ during deep levels but α-threshold $\lessapprox \alpha$ during light levels	γ-threshold $< \alpha$ at all levels
Primary (c.v. > 68 m/sec)	2	1	2	1	2
Non-classified (c.v. 51–67 m/sec)	—	1	—	2	—
Secondary (c.v. < 50 m/sec)	2	1	2	—	1

The threshold for α- and γ-motoneurone activation was compared at different levels of anaesthesia. The motoneurone activation was assessed by examination of the discharge pattern of eight primary, six secondary and three non-classified spindle afferents. A summary of the results of this comparison is shown in Table 1. Nearly every possible permutation of interaction between the threshold of α- and γ-motoneurone activation during light and deep anaesthesia was found.

Fig. 1 illustrates a secondary afferent response which was interpreted as indicating a lower threshold for α-motoneurone than for γ-motoneurone activation during deep anaesthesia. During light anaesthesia the thresholds for α- and γ-motoneurone activation were about the same. An increase in tension elicited by a 4 mA, 100 Hz 'best point' cortical stimulation during deep anaesthesia is illustrated in Fig. 1*A*. There was no associated spindle activity. The animal had been breathing N_2O–O_2 with 1·5 % halothane for 45 min. The small tension change suggested that the stimulus strength was close to the threshold for α-motoneurone activation. Six minutes after this record was made the halothane concentration was reduced to 0·5 %. A spontaneous tail movement occurred 5 min later. Seven minutes after the halothane concentration was reduced the records shown in Fig. 1*B* were made. The stimulus strength was 3 mA. The spindle afferent discharge accelerated and a small tension change was evident. The stimulus was judged to be near the threshold for both α- and γ-motoneurone activation. The halothane concentration was maintained at 0·5 %. Three minutes after the records of Fig. 1*B* were made, the spindle afferent began to discharge spontaneously. Four minutes after the spontaneous discharge began the records of Fig. 1*C* were made at a stimulus strength of 2·85 mA. This stimulus strength was much greater than the threshold for both α- and γ-motoneurone activation.

Fig. 2 illustrates the reverse for a primary afferent from the same animal. When the records of Fig. 2*A* were made the baboon had been anaesthetized with 1·5 % halothane for 52 min. The spindle afferent discharge was regular and there were no spontaneous movements. A 4 mA, 100 Hz 'best point' cortical stimulus elicited afferent acceleration with no evidence of extrafusal muscle contraction. Fig. 2*B* illustrates records made when the animal had been breathing 0·5 % halothane and the N_2O–O_2 mixture for 14 min. Spontaneous movements were present. A 2·6 mA, 100 Hz cortical stimulus elicited a large contraction. The spindle afferent acceleration was less than that found with a larger stimulating current at a deeper level of anaesthesia. On the basis of this record it was judged that during light anaesthesia the threshold for α-motoneurone activation was lower than the threshold for γ-motoneurone activation.

In several experiments the threshold determinations were repeated two

or three times using the same afferent. The relationship between the α- and γ-threshold and the depth of anaesthesia were consistent although the absolute strength of the cortical stimulus at threshold was often different.

An acceleration of spindle afferent discharge either in the absence of or before tension change, 'γ-leading', was seen in the records of ten spindle afferents (five primary, two secondary and three non-classified) at some time during the threshold determination.

The relationship of the cortical area from which this effect can be elicited to the cortical 'best point' for α-motoneurone activation was of some interest. Attempts to map the cortical area were made in a few experiments. Fig. 2*C*, *D* and *E* illustrate one such attempt. The records showed that the

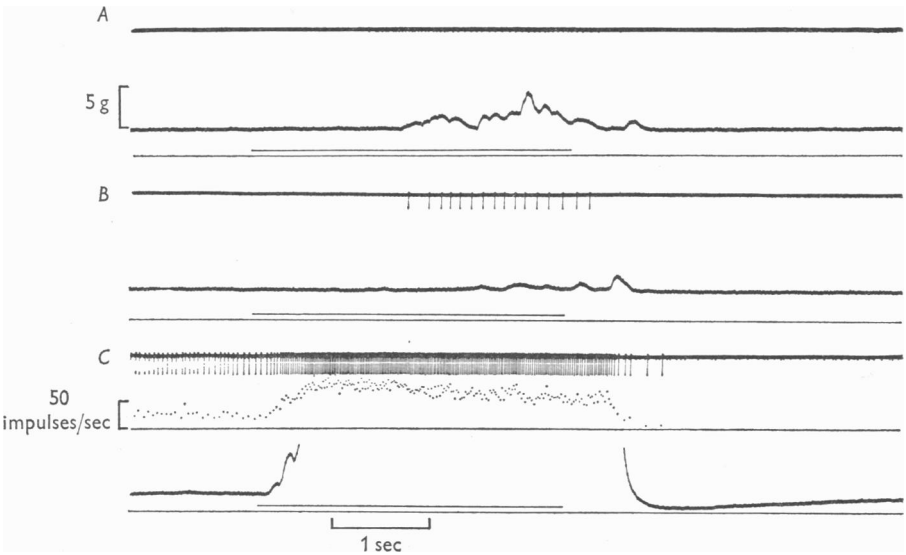

Fig. 1. Effect of cortical stimulation on a secondary spindle afferent (c.v. 43 m/sec) during different levels of anaesthesia. *A*, cortical stimulation 4 mA, 100 Hz. Deep anaesthesia, 1·5 % halothane and 70 % N_2O, 30 % O_2 for 45 min. No resting discharge or elicited spindle discharge present. Top record, spindle afferent discharge; middle record, tension change and bottom record, stimulus duration. *B*, cortical stimulation 3 mA, 100 Hz. Moderate anaesthesia, animal breathing 0·5 % halothane and 70 % N_2O, 30 % O_2 for 7 min. Record taken 13 min after *A*. Occasional spontaneous tail activity present. Order of records as in *A*. *C*, cortical stimulation 2·85 mA, 100 Hz. Light anaesthesia. Animal breathing same anaesthetic as *B*, for 14 min. Record made 7 min after *B*. Top record, afferent discharge; frequency meter record directly underneath; below this the tension record and bottom record, stimulus duration. In most Figures the frequency meter and cortical stimulus monitor have been retouched for reproduction. In all Figures the dorsal roots were largely intact unless it is specifically stated that they were cut.

area was closely confined to the previously mapped 'best point' for tibialis anticus α-motoneurone activation.

Occasionally a clonic convulsion was inadvertently elicited during the threshold determinations. This usually occurred during very light anaesthesia when spontaneous movements were present and the stimulus strength was inappropriate for the level of anaesthesia. The spindle afferent discharge pattern during the convulsion resembled that reported previously (Koeze *et al.* 1968). There was a considerable increase in the spindle afferent discharge during the clonic contractions, but the amount of γ-motoneurone coactivation was poorly correlated with the amount of tension recorded. There were occasional periods of spindle acceleration without detectable tension changes and occasional muscle contractions without evident spindle afferent acceleration. The only difference between the primary and secondary spindle afferent discharge during the clonic contractions was a greater frequency of discharge for the primary afferents.

During light anaesthesia spontaneous spindle afferent discharge acceleration with and without associated muscle tension increase was recorded. When spontaneous spindle afferent discharge accleration occurred threshold measurements were not determined so as to avoid confusion between spontaneous and elicited activity. Fig. 3 illustrates records of two primary spindle afferents from the same animal. A spontaneous TA contraction and the associated spindle afferent acceleration is shown in Fig. 3*A*. When the spontaneous spindle afferent discharge acceleration subsided and the background spindle afferent discharge decreased the cortex was stimulated at 2·4 mA, 50 Hz (Fig. 3*B*). This stimulation elicited a large contraction. The associated spindle afferent discharge acceleration was about the same as that found during the spontaneous contraction and its relationship to the cortical stimulation may be questioned. Fig. 3*C* illustrates intense spindle afferent discharge from another primary afferent when the anaesthesia was light. A slight acceleration was associated with each contraction. Much of the spindle afferent discharge before and during the contraction was quite irregular. A feature of this irregularity was the occurrence of periodic bursts of 2–4 impulses. These bursts suggested that part of the spindle afferent activity was due to static γ-motoneurone activity (Appelberg *et al.* 1966; Bessou & Pagès, 1969).

Cortical stimulation elicited considerable discharge of some secondary spindle afferents (Fig. 1). More often, however, the cortically elicited secondary afferent activity was meagre when compared to that elicited in primary spindle afferents. An example of this is shown in Fig. 4. The records were taken from a primary and secondary spindle afferent isolated at the same time in the same animal. Fig. 4*A* shows the recordings of a primary spindle afferent response during a 2·7 mA, 100 Hz motor cortex

stimulation. The animal had been breathing N_2O–O_2 without halothane for 4 min. Some spontaneous movements were present. The stimulus was considerably above threshold for both α- and γ-motoneurone activation. One minute after this record the discharge of a secondary afferent was recorded during cortical stimulation of the same current strength and stimulus frequency. This record is shown in Fig. 4*B*. There was a very modest increase in spindle afferent discharge. Fig. 4*C* is a recording of the

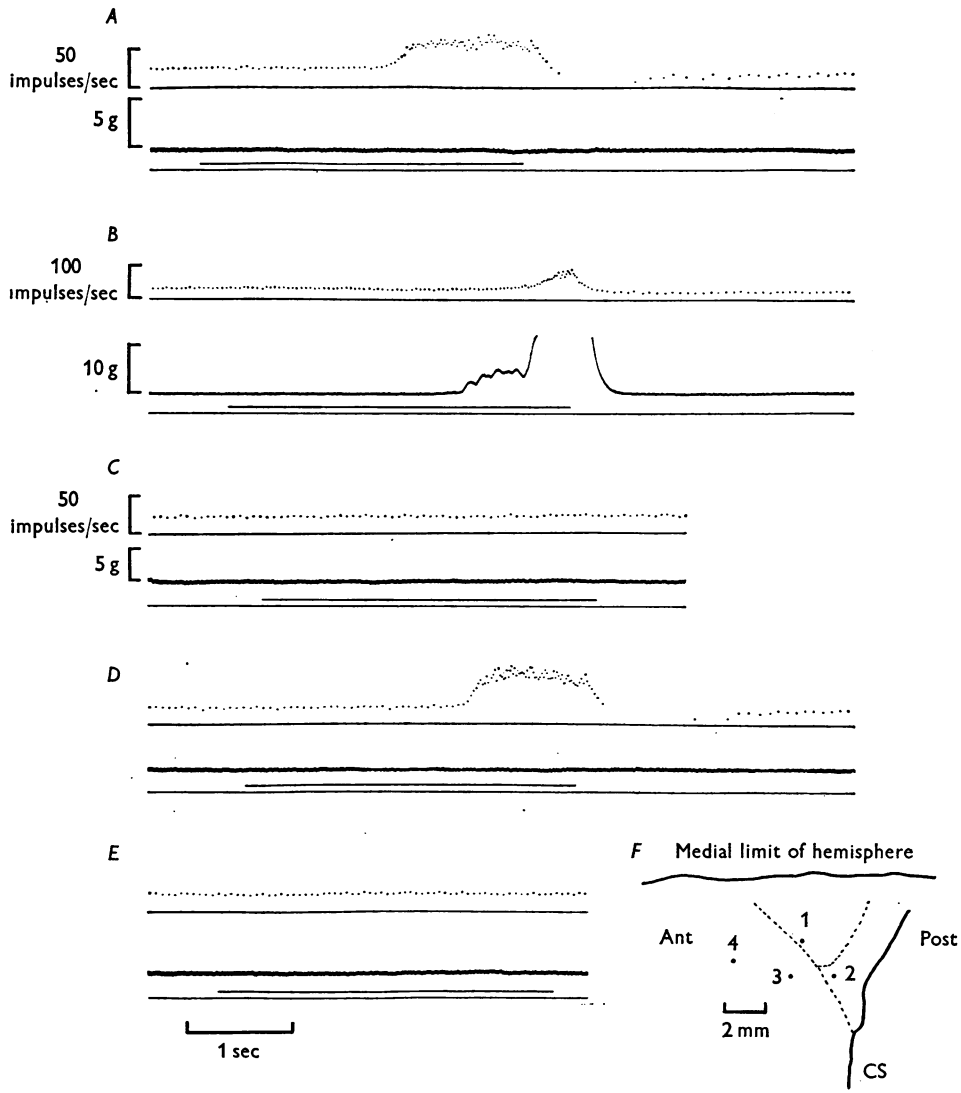

Fig. 2. For legend see facing page.

primary afferent response to the same stimulus 1 min after the records of Fig. 4*B* were made. The tension change and spindle discharge closely resembles that of Fig. 4*A*, which suggests that the anaesthetic level had not changed appreciably during the 3 min interval.

This difference between primary and secondary afferents might be explained by cortical activation of dynamic γ-motoneurones with little or no activation of static γ-motoneurones. That this was not the case is illustrated by the records in Fig. 4*D* made 1 min after those of Fig. 4*C*. The cortical stimulus was unchanged. When the muscle was stretched during the cortical stimulus the afferent responded with a pattern typical of static γ-motoneurone activation. The peak frequency and static was increased but the dynamic index was decreased.

At whatever depth of anaesthesia the spindle afferent discharge elicited by cortical stimulation was almost always judged to be due to static γ-motoneurone activation on the basis of the muscle stretch records made during cortical stimulation. Fig. 5 illustrates records that suggest static γ-activation. Fig. 5*A* shows γ-motoneurone coactivation during a contraction. The animal was breathing only N_2O and O_2. Spontaneous movements were present. The periodic acceleration of spindle afferent discharge which occurred during stretch made before the cortical stimulation suggested that spontaneous static γ-motoneurone drive was present. Cortical stimulation elicited a considerable increase in spindle afferent discharge. The dynamic index is difficult to measure accurately, but it appears to be decreased by the activity elicited by the cortical stimulus.

No certain evidence of dynamic γ-motoneurones activation elicited by a cortical stimulus was found. Two spindle afferents in different animals responded to cortical stimulation with an increase in afferent discharge

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Fig. 2. Effect of cortical stimulation on a primary spindle afferent (c.v. 74 m/sec). Order of records from above down, frequency meter, tension and stimulus monitor. *A*, cortical stimulation 4 mA, 100 Hz at, 'best point', point 1 shown on map in *F*. Deep anaesthesia. Animal breathing 1·5 % halothane and 70 % N_2O and 30 % O_2 for 52 min. *B*, cortical stimulation 2·6 mA, 100 Hz at, 'best point', point 1 on map in *F*. Light anaesthesia. Spontaneous tail movements present. Animal breathing 0·5 % halothane and 70 % N_2O and 30 % O_2 for 14 min. Records in *B* taken 85 min before those in *A*. *C*, stimulation during deep anaesthesia at point 2 on map in *F*. *D*, stimulation during deep anaesthesia at point 3 on map in *F*. *E*, stimulation during deep anaesthesia at point 4 on map in *F*. Cortical stimulation current and frequency of *C*, *D* and *E*, same as in *A*. Record *C* made 1 min before *A*, record *D* made 1 min after *A* and record *E* made 3 min after *A*. During *C*, *D* and *E* the animal was breathing the same gas mixture as in *A*. *F*, cortical map showing 'best point' (1) and three other points used for stimulation in records *A* to *E*. Dashed line represents a small cortical blood vessel. CS, central sulcus.

and dynamic index above the control stretch values. The dynamic index, however, was increased only by about 10 impulses/sec and it is doubtful if this small increase can safely be attributed to dynamic γ-motoneurone activation. In a few experiments an increase in spontaneous dynamic γ-motoneurone drive was elicited by decreasing the level of anaesthesia.

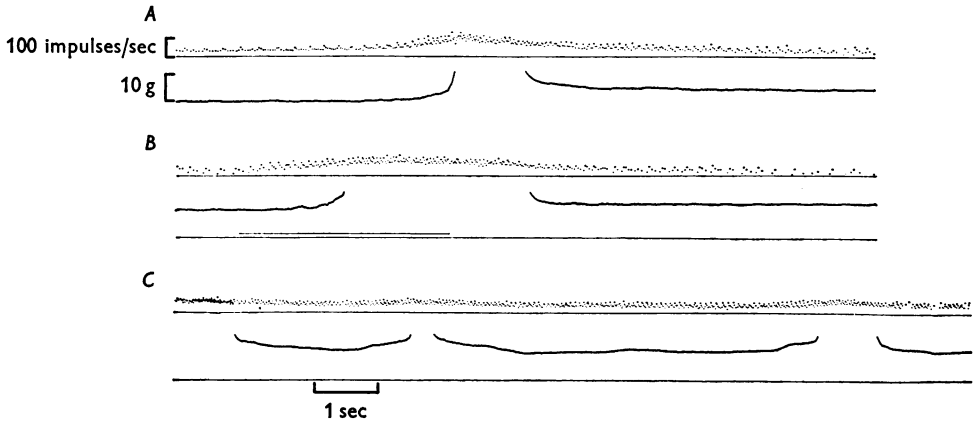

Fig. 3. Spindle afferent discharge during spontaneous TA contractions. *A*, primary spindle afferent (c.v. 74 m/sec). Spindle afferent coactivated with a spontaneous contraction. *B*, continuation of record *A*. Cortical stimulus 2·4 mA, 50 Hz. Light anaesthesia. Animal breathing 0·25 % halothane and 70 % N_2O and 30 % O_2 for 8 min. *C*, primary spindle afferent (c.v. 74 m/sec) from same animal in *A* but a different afferent. Light anaesthesia. Animal breathing 0 % halothane, 70 % N_2O and 30 % O_2 for 4 min. In all records periodic accelerations suggest static α-motoneurone activity. Order of records as in Fig. 2.

Fig. 5*B* and *C* illustrates one example. The record in Fig. 5*B* was made when the animal had been breathing 1·5 % halothane and N_2O–O_2 for 19 min. The average dynamic index of four stretches at this level of anaesthesia was 52 impulses/sec. The halothane was reduced to 0·25 % and 9½ min later the stretch records of Fig. 5*C* were made. The average dynamic index of four stretches was 90 impulses/sec. The dynamic index increase of 38 impulses/sec associated with an increase in peak and static frequency suggests a modest increase in dynamic γ-motoneurone drive to the muscle spindle.

A muscle stretch during light anaesthesia occasionally elicited a contraction. Fig. 6 illustrates such a contraction and the associated irregularity and periodic accelerations of the spindle afferent discharge. When the motor cortex was stimulated and the muscle stretched there was an even greater increase in tension and spindle afferent discharge. Two clonic contractions occurred during the release of stretch in the absence of

spindle afferent discharge. The dynamic index of the afferent response was difficult to measure because of the afferent irregularity but it did not appear to be significantly increased. The increased spindle afferent discharge and the absence of any appreciable change in the dynamic index suggested that the recruited spindle afferent acceleration was elicited by static γ-motoneurone activity. The dorsal roots were largely intact and the muscle stretch may have reflexly recruited some of the static γ-motoneurone activity. The absence of a resting discharge before and after muscle stretch supports this idea, but an alternative explanation might be that the muscle spindle was too relaxed to show the effects of γ-motoneurone activation. This activation could have been present but not detected until the muscle was stretched.

It has been shown that the initial length of the muscle may alter the

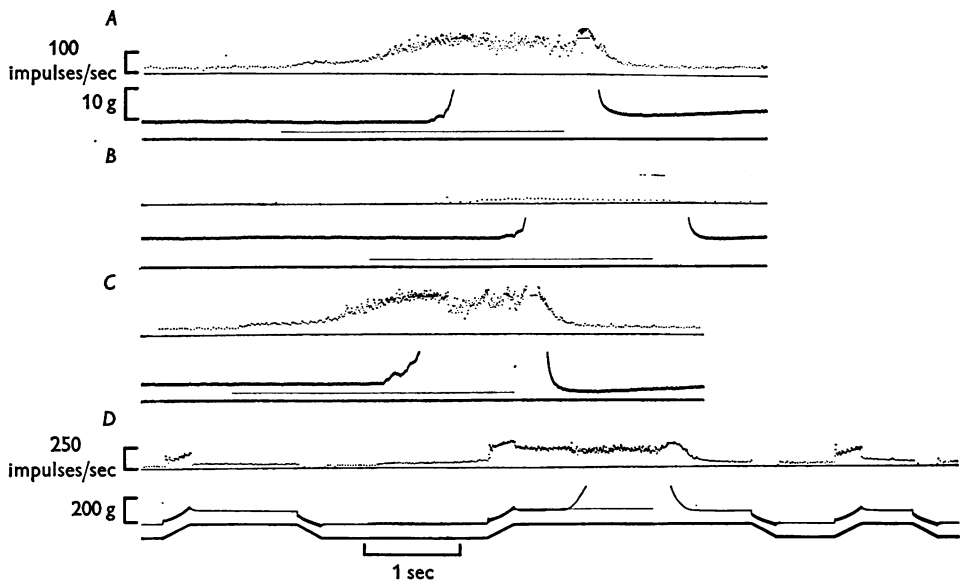

Fig. 4. Effect of cortical stimulation on two afferents during the same level of anaesthesia. *A*, primary spindle afferent (c.v. 74 m/sec). Afferent from different animal than for afferents illustrated in Figs. 1 and 2. Cortical stimulation 2·7 mA, 100 Hz. Light anaesthesia. Animal breathing 0% halothane, 70% N_2O and 30% O_2 for 4 min. *B*, secondary spindle afferent (c.v. 28 m/sec) from same animal. Record made 1 min after *A*. Same cortical stimulus and animal breathing same gas mixture. *C*, same afferent as in *A*. Record taken 1 min after *B*; same cortical stimulus. *D*, same afferent as in record *A*. Records made 1 min after *C*. Muscle stretch 10 mm, 30 mm/sec. Extension record directly under tension record. Cortical stimulus monitor just above and mixed in with tension record. Cortical stimulus as in *A*. Spindle afferent discharge partially unloaded during contraction and accelerated during relaxation in records *A*, *C* and *D*.

apparent relationship between the time of onset of the α- and γ-motoneurone activation and also that an increase in the initial length increases the frequency of spindle afferent discharge elicited by a cortical stimulus (Koeze *et al.* 1968). In these experiments the dorsal roots were intact and it was impossible to say whether or not the increase was elicited by segmental reflexes.

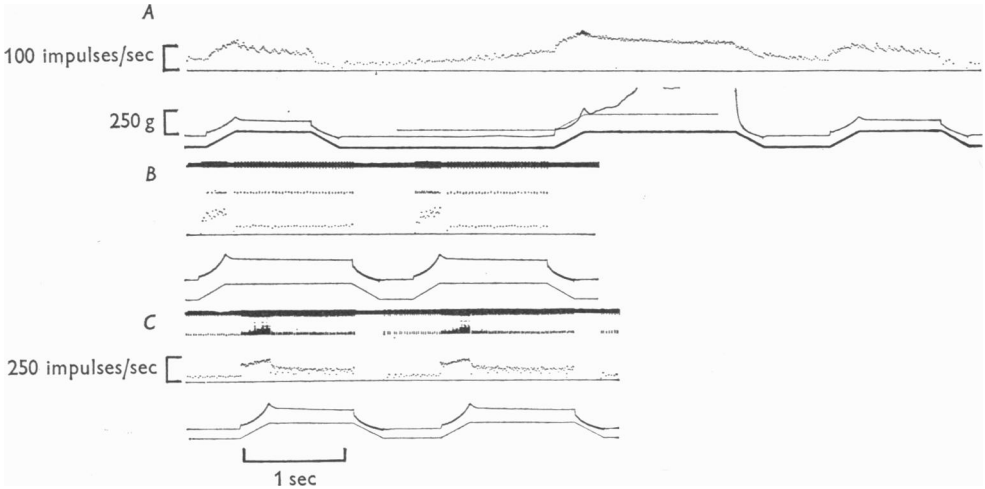

Fig. 5. Spindle afferent activity during muscle stretch. *A*, unclassified spindle afferent (c.v. 57 m/sec). Cortical stimulation 3·4 mA, 100 Hz. Light anaesthesia. Animal breathing 0 % halothane, 70 % N_2O and 30 % O_2. Muscle stretch 30 mm/sec, 10 mm extension. Cortical stimulus monitor record above and mixed with tension record. Spindle afferent discharge suggests static γ-motoneurone activity. *B*, primary afferent (c.v. 81 m/sec). Deep anaesthesia. Animal breathing 1·5 % halothane, 70 % N_2O and 30 % O_2 for 19 min. Muscle stretch and frequency meter calibration as in *A*. *C*, same afferent as *B*. Records made 14½ min after *B*. Light anaesthesia. Animal breathing 0·25 % halothane, 70 % N_2O and 30 % O_2 for 9½ min. Note change in frequency meter gain.

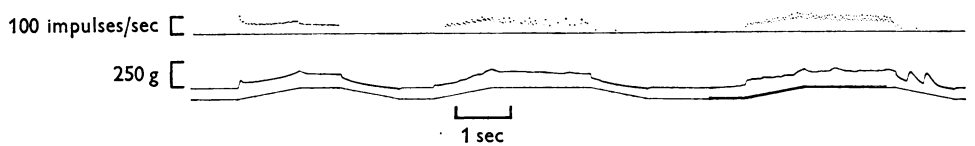

Fig. 6. Muscle contraction and γ-motoneurone activation during stretch. Non-classified spindle afferent (c.v. 62 m/sec). Cortical stimulation 2·5 mA, 100 Hz. Light anaesthesia. Animal breathing 0·25 % halothane, 70 % N_2O and 30 % O_2 for 13 min. Three consecutive muscle stretches, 10 mm/sec, 10 mm extension. Spindle afferent discharge during second stretch suggests spontaneous static γ-motoneurone activity. Note two clonic contractions during release of third stretch.

That segmental reflexes were not always responsible was shown by the records shown in Fig. 7. These illustrate the effect of adding 10 mm to the initial length before and after the dorsal roots were cut. Fig. 7*A*, *B* and *C* were made with a stimulus near the threshold for α-motoneurone activation. The animal was deeply anaesthetized with N_2O–O_2 and 2 % halothane. The cortical stimulus was 4·5 mA, 100 Hz. The dorsal roots were intact except for a small portion used to isolate the afferent. Fig. 7*A* and *C* were made with the muscle at the initial length, Fig. 7*B* with 10 mm added to the initial length. The records were made 1 min apart. The maximum frequency of spindle afferent activity was about 25 impulses/sec greater in *B*. About 45 min later the records shown in Fig. 7*D* and *E* were made. The dorsal roots were cut about 5 min before these records were made. Small extrafusal contractions were present which were probably the result of injury discharges from the cut proximal end of the dorsal roots. They did not appear to affect the spindle afferent discharge. The animal was lightly anaesthetized, breathing only the N_2O–O_2 mixture but there were no spontaneous movements. A 3·5 mA, 100 Hz cortical stimulus was below the threshold for α-motoneurone activation but there was considerable γ-motoneurone activity (Fig. 7*D*). The muscle was lengthened 10 mm and the same cortical stimulus was applied 1 min later. The maximum frequency of spindle discharge was increased by about 30 impulses/sec.

Cortical stimulation inhibited the discharge of two spindle afferents from different animals. The records from one of these are illustrated in Fig. 8. The inhibition appeared to influence static γ-motoneurone activity. In the muscle stretch records of Fig. 8*A* the discharge during tonic stretch was abolished.

A 2·45 mA, 100 Hz stimulation of the 'best point' elicited inhibition of the spontaneous spindle afferent activity (Fig. 8*B*). Stimulation of three other cortical points near the 'best point' are shown in Fig. 8*C*, *D* and *E*. As illustrated, stimulation of two of the points had no effect while that of the third point had a very slight effect. After testing each of the three points, the 'best point' was tested again to make certain that the threshold for the inhibitory effect had not changed.

Inhibition of this afferent by cortical stimulation was not invariably present. Fifty-nine min after the records of Fig. 8*B*–*E* were made a 3·75 mA, 250 Hz 'best point' stimulation elicited considerable spindle afferent discharge with only small tension changes (Fig. 8*F*). The same effect was seen with 100 and 50 Hz stimulation. The level of anaesthesia was similar to the level at the time of the earlier record. In the time between the two sets of records the level of anaesthesia had been decreased until spontaneous movements were present and then increased to its previous level.

In one experiment the ipsilateral sural nerve was stimulated with a

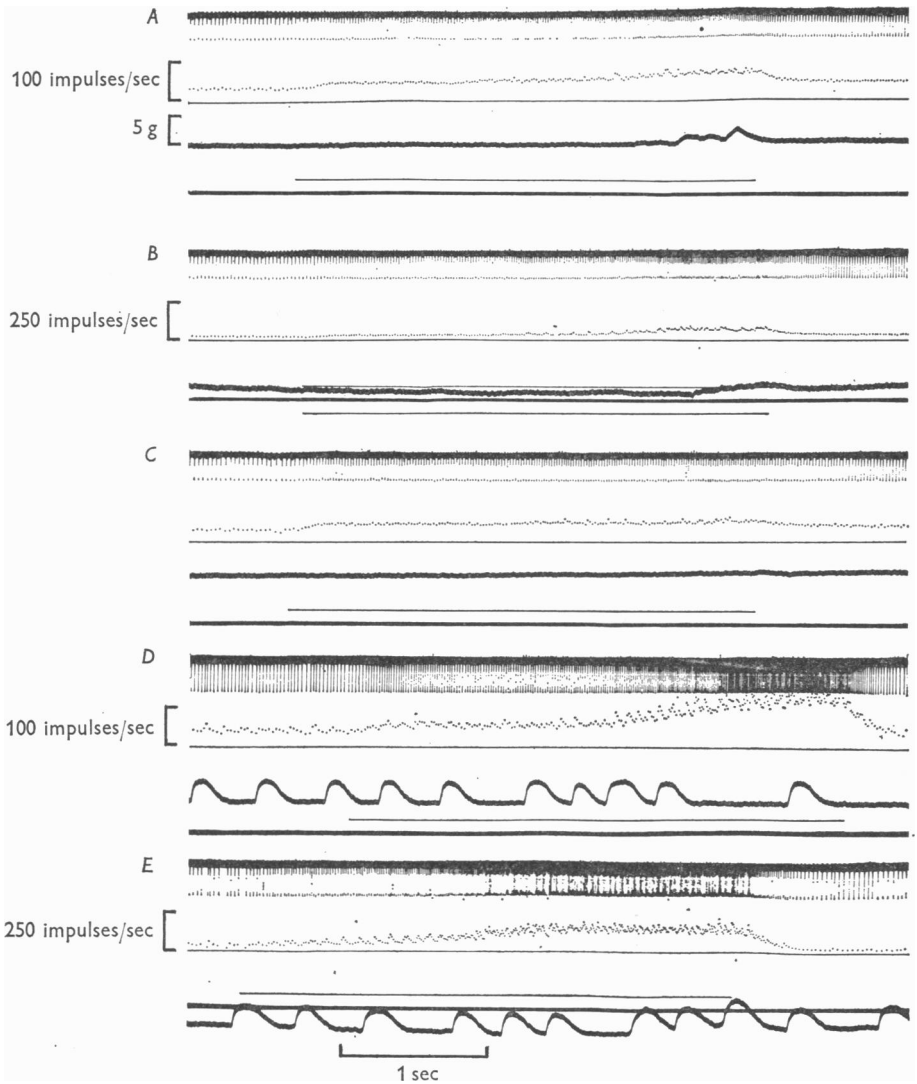

Fig. 7. Effect of initial length and dorsal root section on cortically elicited spindle afferent discharge. Primary afferent (c.v. 74 m/sec). *A*, *B* and *C*, dorsal roots largely intact. *B*, 10 mm added to the initial length. *A* and *C*, muscle at initial length. Records made at 1 min intervals during deep anaesthesia. Animal breathing 2 % halothane, 70 % N_2O and 30 % O_2 for 5 min. Cortical stimulus 4·5 mA, 100 Hz. *D* and *C*, dorsal roots cut. *D*, muscle at initial length. *E*, 10 mm added to initial length. Records made at 1 min intervals during moderate anaesthesia. Animal breathing 0 % halothane, 70 % N_2O and 30 % O_2 for $2\frac{1}{2}$ min. Cortical stimulus 3·5 mA, 100 Hz. Note that frequency meter gain in *C* is same as *A*. Small muscle twitches in *D* and *E* are probably the result of reflex discharges initiated from the cut dorsal roots.

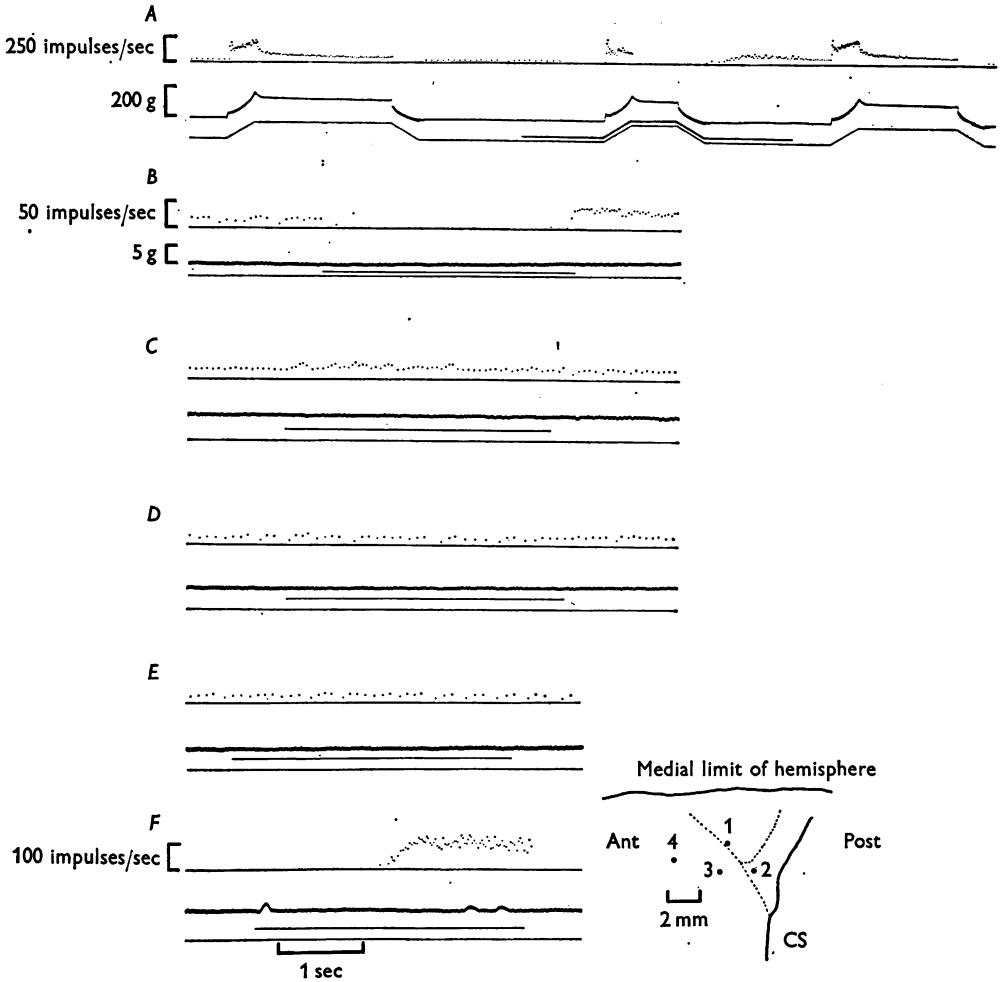

Fig. 8. Cortically elicited spindle afferent inhibition. Primary spindle afferent (c.v. 81 m/sec). *A*, three muscle stretches, 30 mm/sec, 10 mm extension. Middle stretch during cortical stimulation, 2·6 mA, 100 Hz, at the 'best point', point 1 on the map, lower right. Records made during deep anaesthesia. Animal breathing halothane 1·5 %, 70 % N_2O and 30 % O_2 for 18 min. *B*, cortical stimulation, 2·45 mA, 100 Hz at point 1 on map. Records made 10 min after those shown in *A*. Animal breathing same gas mixture as in *A* for records *B*, *C*, *D* and *E*. *C*, cortical stimulation 2·45 mA, 100 Hz at point 2. Records made 1 min before those shown in *B*. *D*, cortical stimulation 2·45 mA, 100 Hz at point 3. Records made 2 min after those shown in *B*. *E*, cortical stimulation 2·5 mA, 100 Hz at point 4. Records made 8 min after those shown in *B*. *F*, cortical stimulation 3·75 mA, 250 Hz stimulation at point 1 on map. Records made at 04.57, 59 min after those shown in *B*. After the records shown in *E* were made the anaesthesia was decreased to 0·25 % halothane at 04.43. At 04.52 the halothane was increased to 1·25 % and at 04.56 to 1·5 %. Records were made during the same experiment as those shown in Fig. 2. See Fig. 2 for details of map.

100 Hz, 0·5 V stimulus. This stimulation elicited a small contraction of about 5 g wt. and an average spindle afferent discharge of 64 impulses/sec over a 1 sec period. A 250 Hz, 3 mA cortical stimulus elicited a contraction of about 7 g wt. and an average spindle afferent discharge of 43 impulses/sec over a 1 sec period. When the two stimuli were applied simultaneously the muscle tension response exceeded the gain of the recording system and the spindle afferent discharge averaged 82 impulses/sec over a 1 sec period. This suggested that the motor cortex does not inhibit all non-cortically elicited γ-motoneurone activity.

DISCUSSION

Cortical projections to baboon α-motoneurones are both monosynaptic and polysynaptic (Bernhard, Bohm & Petersén, 1953; Preston & Whitlock, 1961; Landgren, Phillips & Porter, 1962*a*, *b*). When the cortical 'best point' is stimulated by a long (3·2 sec) repetitive stimulus, cortical neurones projecting to α-motoneurones by way of monosynaptic, polysynaptic excitatory and polysynaptic inhibitory pathways are activated. In addition, cortical neurones may also be activated which affect α-motoneurones through extra pyramidal pathways.

At or near the 'best point' for cortically elicited motoneurone activation are cortical projections which elicit activity in γ-motoneurones. A portion of this γ-motoneurone activation may be monosynaptic (Grigg & Preston, 1971).

The failure to find spindle afferent discharge acceleration at a lower cortical threshold than that for muscle contraction in the baboon hind limb (Koeze, 1968) might be explained by the effects of barbiturate anaesthesia. For example, it has been reported that a small amount of pentobarbitone blocks cortically elicited α-motoneurone inhibition and late facilitation but not the early, monosynaptic facilitation (Preston & Whitlock, 1960). If the cortical projection to hind limb γ-motoneurones has more synapses than the pathway to forelimb γ-motoneurones then it is possible that the hind limb projection may be more sensitive to barbiturate depression than the forelimb projection.

In the halothane anaesthetized baboon cortical stimulation elicited in some hind limb spindle afferents an increase in discharge at a threshold below that required to elicit muscle contraction. This suggested that during halothane anaesthesia some hind limb γ-motoneurones have a lower threshold for cortical activation than some α-motoneurones. The explanation for this difference from the previously reported study of the barbiturate anaesthetized baboon presumably involves the different mechanisms of action of barbiturate and halothane anaesthetic agents, but until more is known of these mechanisms further speculation is probably not worthwhile.

Activation by cortical stimulation of both dynamic and static γ-motoneurones supplying muscle spindles in both extensor and flexor muscles of the cat has been demonstrated (Vedel, 1966; Fidone & Preston, 1969; Yokota & Voorhoeve, 1969; Vedel & Mouillac-Baudevin, 1970). The cortical threshold for γ-motoneurone activation is lower than that required for α-motoneurone activation (Granit & Kaada, 1952; Yokota & Voorhoeve, 1969; Vedel & Mouillac-Baudevin, 1970).

The results of this study differed from similar cat experiments in two ways. The failure to find a consistent cortical threshold relationship between tibialis anticus α- and γ-motoneurone activation contrasts with the experiments of Vedel & Mouillac-Baudevin (1970) who found that cat tibialis anticus γ-motoneurones had lower thresholds for cortical activation than α-motoneurones at all levels of anaesthesia. The reason for this difference between cat and baboon is not clear but the lack of monosynaptic cortical projections to α-motoneurones in the cat (Lloyd, 1941) may be involved. The second difference was the failure in the baboon to activate substantial dynamic γ-motoneurone activity by cortical stimulation. In the cat such activation has been reported by Vedel (1966) and confirmed by others (Yokota & Voorhoeve, 1969; Vedel & Mouillac-Baudevin, 1970). The explanation for the failure to elicit dynamic γ-motoneurone activation was not clear. It seemed unlikely to be related to the depth of anaesthesia because spontaneous fluctuations of dynamic γ-motoneurone activity were seen during periods of light anaesthesia. Alnaes, Jansen & Rudjord (1965) have argued that dynamic γ-motoneurone activity is largely supported by segmental reflexes. They found non-specific activation of dynamic γ-motoneurone activity in flexor and extensor muscles by stimulation of ipsilateral and contralateral peripheral nerves and believe that dynamic γ-motoneurone activation is part of the basic spinal motor mechanism. It has been shown, however, that dynamic γ-motoneurone activation can be elicited by suprasegmental stimulation after the dorsal roots are cut in the cat anaesthetized with halothane (Appelberg & Jeneskrog, 1968).

Static γ-motoneurone inhibition has been reported previously (Koeze, 1968) and was found in the experiments reported here. In the present experiments the static γ-motoneurone activity from other sources lasted long enough for the cortical site which elicited the inhibition to be mapped. Inhibition could be elicited only from points near the cortical 'best point'. This suggests the presence of cortical projections which provide not only for activation of α- and γ-motoneurones but also for inhibition of static γ-motoneurone activity when it arises from other, non-cortical sources. If the background static γ-motoneurone activity arose from the red nucleus

and if the baboon has the same sort of corticorubral connexions as the cat (Tsukahara, Fuller & Brooks, 1968) then connexions exist by which cortical neurones may suppress red nucleus neurones and thereby decrease static γ-motoneurone activity.

Grigg & Preston (1971) have shown inhibition of γ-motoneurone activity in the 'pyramidal' baboon. Since the rubrospinal and descending brain stem pathways other than the pyramidal tracts are obliterated in this preparation, inhibitory pathways descending in the pyramidal tract must exist. It seemed of interest, therefore, to see what the effect of a 'best point' cortical stimulus that elicited static γ-motoneurone activity would have on motoneurone activity elicited by sural nerve stimulation. The activation from the two sources summed and no inhibition was found. This suggests that cortical inhibition may be selective in the sense that γ-motoneurone activity from some sources is more easily suppressed than from others.

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