# Why Transcortical Reflexes?

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*"Between the brain and the muscle there is a circle of nerves; one nerve conveys the influence of the brain to the muscle, another gives the sense of the condition of the muscle to the brain."*

Charles Bell (1826)

SUMMARY: *Experiments in humans and in monkeys have indicated that load perturbations, occurring during voluntary movements and postural activity, may be automatically compensated for. Overall muscle stiffness opposing load changes is determined by the viscoelastic properties of the muscle, by segmental reflex actions and finally by long-loop reflexes. Under certain circumstances, for instance when the subject or the experimental monkey is "prepared" to counteract perturbations which are unpredictable in time, the long-loop "reflexes" appear to be responsible for most of the corrective muscle tension. Experiments in anaesthetized monkeys revealed that signals from stretch afferents reach neurons of the motor cortex, possibly via a relay in the cortical area 3a. The latencies of these responses to well controlled muscle stretches were in the same range as motor cortical cell discharges recorded in alert monkeys subjected to load perturbations. Furthermore, these responses of cells in the motor cortex also had the appropriate timing to indicate a causal relationship with the long-latency electromyographic responses to load changes referred to above. These experimental results therefore strongly support the hypothesis, first proposed by Phillips (1969), of a transcortical servoloop adjusting motor cortical output according to the load conditions in which movements are performed.*

*The major advantage of transcortical regulations as opposed to segmental regulations, seems to be a powerful gain control acting at the cortical level; it was repeatedly shown that the long-loop reflexes are strongly modifiable and under voluntary control. It is suggested that an adaptive gain control at the cortical level is a prerequisite to preserve the complex capabilities of the motor cortex as the chief "executive" for skilled, preprogrammed movements. A loss of this adaptive gain control may be, at least partly, the cause of motor disorders such as rigidity in Parkinsonian patients, as reported by Tatton and Lee (1975). It is suggested that further investigations of the control of transcortical reflexes may aid in the understanding of the pathophysiology of motor disabilities.*

RÉSUMÉ: *Les expériences chez les humains et les singes ont indiqué que les perturbations survenant durant les mouvements volontaires et l'activité posturale peuvent être compensées automatiquement. Dans certaines circonstances, par exemple quand le sujet ou le singe expérimental est "préparé" à neutraliser les perturbations qui sont imprévisibles dans le temps, les "réflexes" à long trajet semblent être responsables pour la plus grande partie de la tension correctrice du muscle. Nos résultats expérimentaux appuient fortement l'hypothèse, proposée premièrement par Phillips (1969), d'un servo-mécanisme transcortical ajustant l'efférence corticale motrice (output) selon les conditions de charge dans lesquelles les mouvements sont accomplis.*

*L'avantage majeur des régulations transcorticales opposées aux régulations segmentales, semblent être un contrôle de gain puissant agissant au niveau cortical; il fut souvent montré que les réflexes à long circuit sont fortement modifiables et sous contrôle volontaire. Il est suggéré qu'un contrôle de gain adaptable au niveau cortical est un prérequis pour préserver les capacités complexes du cortex moteur en tant que directeur des mouvements pré-programmés. Une perte de ce contrôle de gain adaptable peut être, au moins partiellement, la cause de désordres moteurs, telle la rigidité chez les patients parkinsoniens, comme le rapportait Tatton et Lee (1975). Il est suggéré qu'une investigation plus poussée du contrôle des réflexes transcorticaux pourrait aider dans la compréhension de la pathophysiologie des désordres moteurs.*

## INTRODUCTION

Experiments in human subjects (Marsden, 1973; Marsden, Merton and Morton, 1973) led to the concept that long loop reflexes may play an important role in compensating for sudden load changes interfering with voluntary movements or postural activity. Similar experiments in monkeys, which allowed single unit recordings in the motor cortex, furthermore indicated that the motor cortex represents a relay for supraspinal "stretch reflexes" (Evarts, 1973). However, the motor cortex in primates is generally regarded as the chief "executive" of the brain, addressing the motor apparatus of the brainstem and of the spinal cord. The functional importance of this highly complex neural structure would appear to be much reduced if one viewed it merely as a reflex center. Thus, we will make an attempt to reconcile the seemingly paradoxical hypotheses of motor cortical function.

It is proposed that motor commands, issued from the cortex, may or may not, depending on the context, be subjected to feedback modification by afferent signals generated in muscle spindles (and probably also other receptors). In this scheme proprioceptive feedback would have merely a modifying function to reinforce or to suppress, a central command. There is indeed experimental evidence (which will be discussed in detail) indicating that access of peripheral feedback signals

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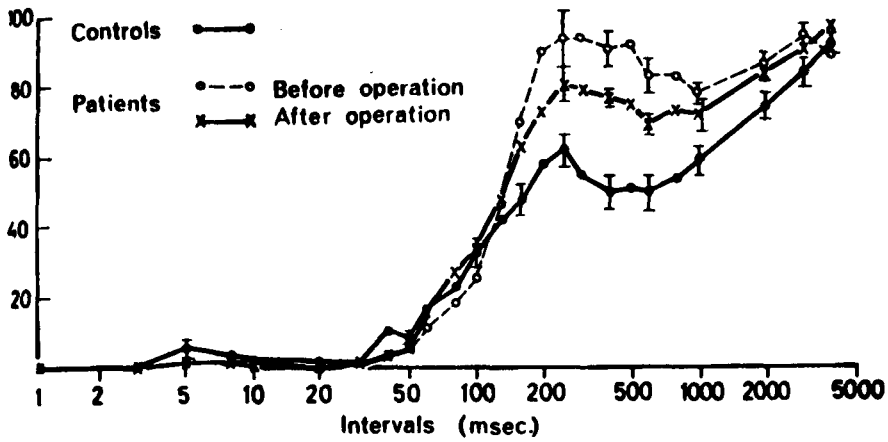


*Figure 1*—H-reflex recovery curve (Zander Olsen and Diamantopoulos, 1967). Note "late facilitation", starting at about 100 msec., which may reflect facilitation of motoneurons via a transcortical loop. The "late facilitation" was found to be much more pronounced in Parkinsonian patients than in normal subjects. Stereotaxic surgery in Parkinsonian patients reduced the "late facilitation". Ordinate: percentage of test response.

to output cells of the motor cortex is facultative and determined by the context in which a particular motor act is performed. Phillips (1969) was the first to propose that feedback signals from muscle spindles may be used at the cortical level for automatically adjusting the output of cortical neurons. This hypothesis of transcortical load compensation requires i, a neural pathway from muscle spindle afferents to the motor cortex representing the afferent limb of the 'transcortical reflex'; ii, an adaptive control of the input-output gain, at the cortical level, for signals generated in muscle spindles. This latter requirement appears to be necessary in order to free the motor cortex from a rigid reflex connotation. The purpose of this paper is thus to summarize experimental evidence for transcortical servoactions and to discuss the possible advantages and disadvantages of transcortical regulation as compared with segmental regulation by feedback signals. Furthermore, long-loop stretch reflexes appear to be interesting from the clinical point of view: there is growing evidence that an increased gain at the cortical level may explain some aspects of the pathophysiology of increased muscle tone.

*Long latency responses evoked by stretch afferents*

In 1956, Hammond described the EMG responses to sudden pulls of the forearm flexor muscles in the human subject; this author particularly emphasized the presence of a second, temporally dispersed component which followed the early synchronized EMG potential. The short latency and biphasic form of the first potential identified it easily as the monosynaptic stretch reflex. The latency of the second component, although around 50 msec., was still much less than a voluntary reaction. The nature of this second component was interpreted much later as a transcortical "reflex" by Evarts (1973) and Marsden, Merton and Morton (1973). Characteristically, the magnitude of the stretch-evoked long-latency response was found to depend on previous instruction given to the human subject or to the monkey: when the instruction was "resist", the response was large and contributed a major part of the reflex tension; but if the instruction was "let go", it was much weaker. Similar observations were made in man by Melvill Jones and Watts (1971) who recorded the stretch response in the ankle extensors to sudden dorsiflexion of the foot. Impressed by the prominence of the long-latency stretch response they termed it "Functional Stretch Reflex".

The involvement of supraspinal structures upon activation of stretch afferents was also discussed in studies of the H-reflex in man and animals (reviewed by Wiesendanger, 1972). Most investigators plotting the recovery curve of the H-response noted a "late facilitation" starting at about 120 msec. In patients with increased muscle tone, an abnormally large late facilitation was often the most consistent deviation from normal curves (Zander Olsen and Diamantopoulos, 1967; Fig. 1). A transcortical reflex loop seems to be the most probable cause, although there are a number of other possibilities to explain the late facilitation (Wiesendanger, 1972).

*The evidence for cortical participation in the long-latency stretch response*

The evidence that the late EMG response evoked by sudden muscle stretch is mediated by the motor cortex has so far been indirect. Evarts (1973) first showed that sudden passive displacements of a handle which a monkey was trained to hold in a narrow zone resulted in a response of cells of the motor cortex governing arm muscles. The motor cortical response preceded the late EMG response by an interval appropriate to suggest a causal relationship. Similar observations were made by Conrad, Matsunami, Meyer-Lohmann, Wiesendanger and Brooks (1974) and Conrad, Meyer-Lohmann, Matsunami and Brooks (1975) when the perturbation was "injected" in the initial phase of a trained arm movement. Computer-generated histograms furthermore uncovered a tight temporal relation between oscillations of cortical cell discharges and EMG bursts elicited by the perturbation, thus providing a further indication of a causal relationship between the two. Ablation of the postcentral gyrus abolished the early cortical response to stretch (Tatton, Forner, Gerstein, Chambers and Liu, 1975), probably by destroying the first cortical relay in area 3a (see below). The crucial experiment of ablating or blocking the motor cortex has however not yet been done.

*Modification of long-latency muscle responses to stretch*

Whether a "perturbation" of a

movement causes a prominent long-latency "stretch reflex" or not was found to be highly dependent on the strategy of movement execution. This aspect, first noted by Hammond (1956) and by Hammond, Merton and Sutton (1956), was systematically investigated in alert monkeys by Evarts and Tanji (1974). Perturbation of a postural contraction resulted in an early burst from pyramidal tract cells which preceded the secondary EMG burst. Prior instruction modified the motor cortical response in parallel with the long-latency EMG response, again suggesting a causal relationship. It appeared that the "instruction" to the monkey (similar to the "resist" — "let go" strategies used by Hammond, 1956) modulated the tonic background activity of pyramidal tract cells. Thus, the reactivity of output cells of the motor cortex critically depended on modulating influences. This gating effect appears to be essentially under voluntary control and obviously depends on past experience (in conditioning experiments: feedback by reinforcement) and on the actual context in which the movement is executed. The hypothetical neural circuitry will be discussed below (see The control of transmission . . .).

The recent discovery by Tatton and Lee (1975) that long-latency EMG responses evoked by load pulses are very prominent in Parkinsonian patients and that these responses are hardly modifiable is of the greatest importance. The results, if confirmed, would indicate that long-loop reflexes would have to be considered in explaining the pathophysiology of rigidity. Milner-Brown, Stein and Lee (1975) have also made the interesting observation that weight lifters may have more prominent long-loop reflexes than average human subjects. This may signify that the efficiency of weight lifters to cope with heavy loads depends on the progressive development, by training, of long-loop reflexes.

*The pathway from muscle afferents to the motor cortex*

In previous experiments on baboons, electrical stimulation of mus-

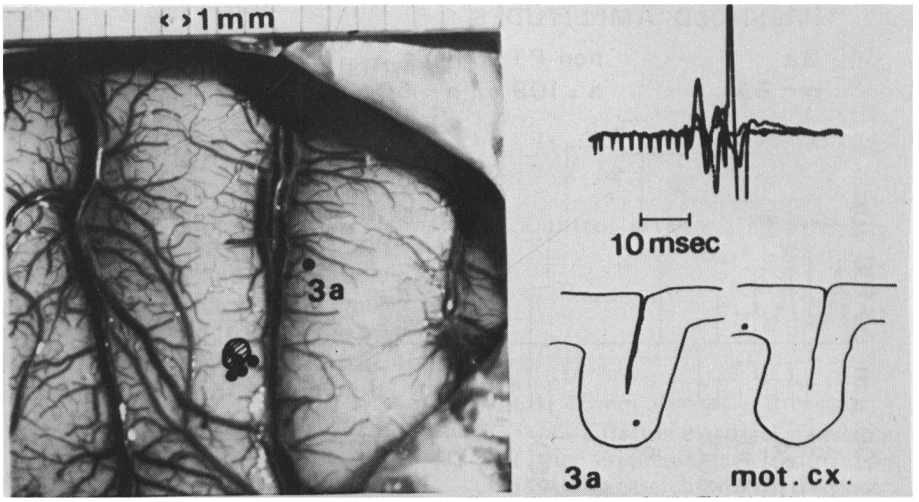


*Figure 2*—Entry points of microelectrode tracks in the hand area of the left peri-Rolandic cortex in a Cebus monkey. In the postcentral gyrus (3a), one electrode track was aimed at area 3a in the depth of the central sulcus. After the recording of stretch-evoked responses, the site was marked by ejection of Fastgreen which was subsequently found in a histological section (right). In this experiment, 5 tracks were made in the precentral gyrus in the area (hatched) from which, upon electrical stimulation, EMG responses in contralateral wrist and finger extensors could be elicited at lowest threshold (about 0.4 mA; above). The deepest point of a microelectrode penetration in the motor cortex was also marked by Fastgreen (right, mot. cx.). From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

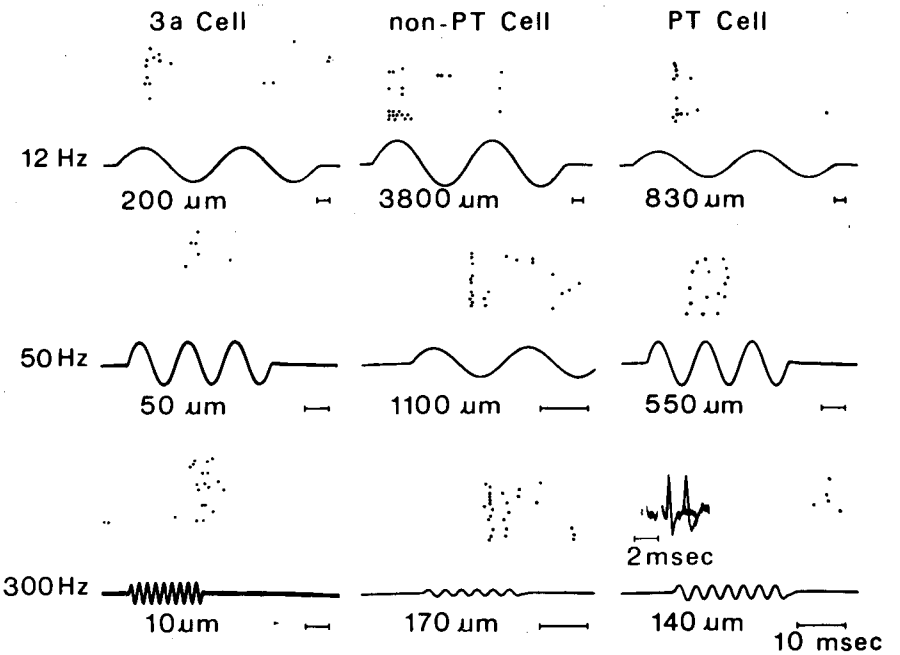


*Figure 3*—Representative response patterns of a neuron in area 3a (3a cell), of a non-corticospinal neuron (non-PT cell) and of a corticospinal neuron (PT cell). Twenty threshold responses to sinusoidal stretching of the finger extensor muscle at frequencies of 12, 50 and 300 Hz are shown as dot rasters. Each dot represents a spike discharge. The minimal amplitudes required to evoke a cortical response are noted for each frequency. Note that, for each cell type, the threshold amplitudes were lowest for the highest frequencies of sinusoidal stretching. Antidromic responses to a double stimulus applied in the contralateral funiculus is shown for the PT cell as an inset (lower right). Horizontal bar represents 10 msec. except for antidromic response (2 msec). From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

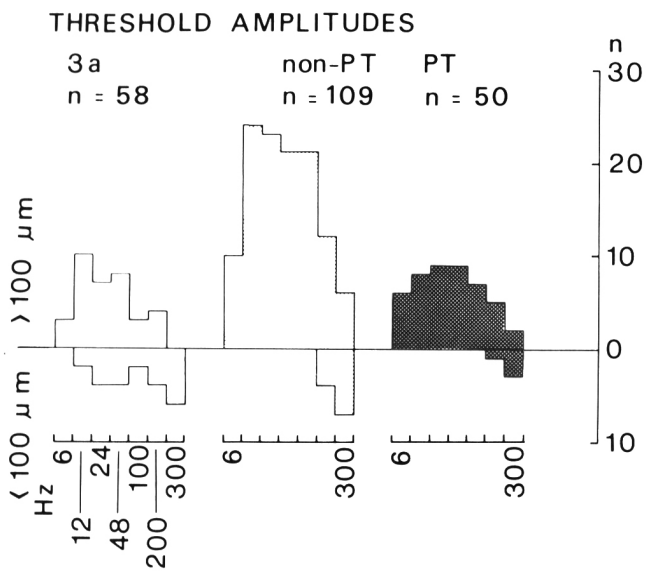


*Figure 4*—Division of cortical neurons which responded to sinusoidal stretching of finger-extensors at minimal amplitudes > 100 $\mu$ m (above horizontal line) and < 100 $\mu$ m (below horizontal line). The numbers are represented as columns for each frequency tested. Most "low-threshold" neurons were found among 3a neurons, much lower proportions among cells of the motor cortex. Note that, for corticospinal and non-corticospinal neurons, low thresholds (< 100 $\mu$ m) were disclosed only if "dynamic" stimuli of 200-300 Hz were used. From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

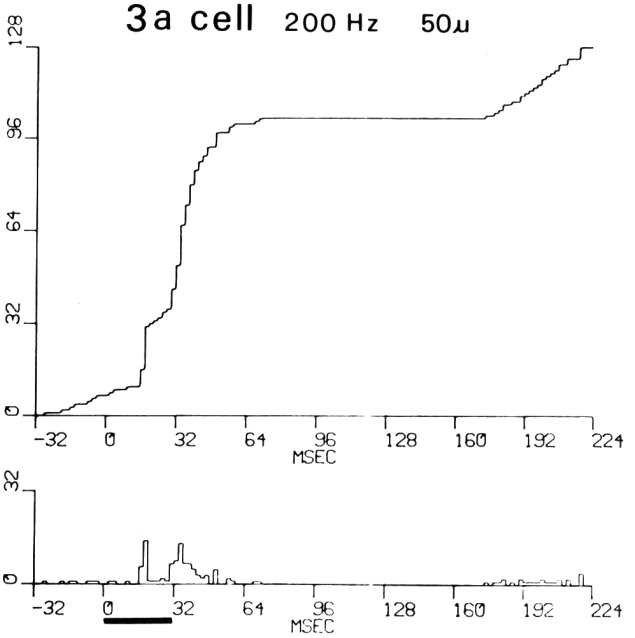


*Figure 5*—Typical response pattern of a 3a neuron which responded to sinusoidal stretching of the EDC muscle at a frequency of 200 Hz and at a minimal amplitude of 50 $\mu$m. The duration of the vibration train is indicated by a solid bar. Computer generated cumulative distribution function (above) and post-stimulus time histogram (below). Two peaks show up in the histogram. The phasic response is followed by an inhibition lasting about 100 msec; ordinate: number of spikes. From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

cle nerves of the forearm was used to establish the existence of a powerful projection from low-threshold muscle afferents to area 3a at the bottom of the central sulcus (Phillips, Powell and Wiesendanger, 1971). Cytoarchitectonically, this area is transitional between koniocortex (area 3b) and agranular cortex (area 4) and no evidence was found electrophysiologically that this projection area for low-threshold muscle afferents has output neurons to the spinal cord. Neurons of the motor cortex were also activated by electrical stimulation of muscle nerves, but less powerfully than 3a neurons (Wiesendanger, 1973). Repetitive stimulation at intensities above threshold for group II afferents had to be used to excite neurons, both corticospinal ("PT-neurons") and non-corticospinal ("non-PT neurons"). It was therefore assumed that the "error signals" to the output cells of the motor cortex were provided by secondary rather than primary muscle spindle endings as originally proposed by Phillips (1969).

In recent experiments on Cebus monkeys, the question was reinvestigated by using controlled stretches of forearm muscles (Lucier, Rüegg and Wiesendanger, 1975). Since high frequency longitudinal vibration of tendons represents an optimal stimulus for exciting primary muscle spindle endings (Matthews, 1972), it was hoped that weak effects from primary muscle spindle endings could be disclosed more easily with trains of vibration than with electrical stimulation at intensities below group II threshold (and therefore submaximal for the group I volley). The objective was to compare the response pattern of neurons in area 3a with those in the motor cortex, corticospinal and non-corticospinal.

Area 3a was first localized by using low intensity (about twitch-threshold) electrical stimulation of the deep radial nerve. Figure 2 illustrates a location of a typical electrode penetration which, in the depth, traversed area 3a receiving a projection from the deep radial nerve. The area of the motor cortex which, upon electrical stimulation with a short train, elicited an EMG response at minimal intensity was the best site for finding stretch-evoked responses. Several penetrations made in this area are marked on the photograph of the brain surface. An attempt was made to test each cell systematically with sinusoidal stretching of finger and wrist extensor muscles at frequencies of 6, 12, 24, 50, 100, 200 and 300 Hz and with step stretches. For each type of stimulus, the responses were photographed first at threshold amplitudes then at 100 times threshold amplitudes. Figure 3 illustrates representative response patterns for three selected frequencies and for the three cell types: 3a neurons, non-PT neurons, and PT-neurons. These examples show, and this was typical

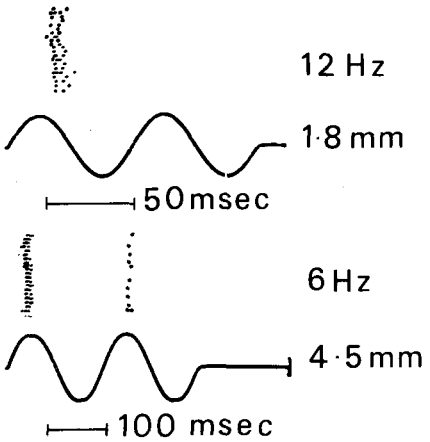


*Figure 6*—Phasic response of a corticospinal neuron which, at low frequencies of sinusoidal muscle stretch, responded only at high amplitudes of stretching. Note partial driving at 6 Hz and complete suppression of evoked activity to the second cycle at 12 Hz. From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

for the sample, that minimal amplitudes, required to activate cortical cells decreased as the applied frequency of sinusoidal stretching was increased. With a highly dynamic stimulus such as a 300 Hz vibration, 3a neurons sometimes discharged at amplitudes which were within the range of the thresholds of primary muscle spindle receptors. More surprisingly, however, the threshold of neurons of the motor cortex was also drastically decreased when high frequency vibration was used. As shown in Figure 4, a small number of cells in the motor cortex, especially of non-PT neurons, were activated with amplitudes below 100 μ m. This (and other criteria described in the original paper) indicated to us that primary muscle spindle endings may, if maximally excited with trains of vibration, contribute to the stretch evoked responses.

Dynamic responses (short bursts) were the dominant pattern for all cell types. Figure 5 is an example of a dynamic response of a 3a neuron; the post-stimulus time histogram reveals a clustering of the discharges with the formation of two clear peaks followed by a prolonged inhibition. Partial driving was observed at frequencies of up to 100 Hz in some 3a neurons and non-PT neurons of the motor cortex. Partial driving at the lowest frequencies (6 Hz) was common for all cell types (Fig. 6). It was thus established in these experiments that a fairly restricted input from stretch afferents was indeed capable of activating cells of the motor cortex. Secondary muscle spindle endings, and to some extent also primary muscle spindle endings, were considered to be the most likely receptors involved in producing the motor cortex response. A similar conclusion was reached by Murphy, Wong and Kwan (1975) in similar experiments in cats.

*The control of transmission at the cortical level*

Electrophysiological studies in anaesthetized monkeys showed that transmission from stretch receptors to area 3a was rapid and secure (Phillips et al., 1971; Lucier et al., 1975). Whether area 3a is the first link in a transmission line to the motor cortex has not been established electrophysiologically, although latency measurements would be compatible with a transmission of signals from area 3a to non-PT neurons and finally to PT-neurons. In degeneration studies, it was found that small lesions in area 3a of Cebus monkeys resulted in degeneration in area 4 (Wiesendanger and Rüegg, in preparation). Whatever the pathway for signals from muscle spindles to the motor cortex may be, the electrophysiological studies of Lucier et al. (1975) made it clear that the security of transmission at the level of output cells in the motor cortex is low. All in all the number of output

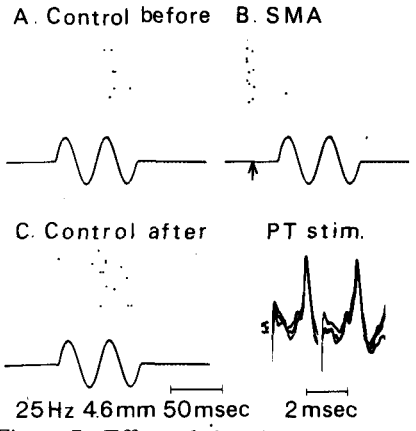


*Figure 7*—Effect of electrical stimulation of the supplementary motor area (SMA) on stretch-evoked discharges of a corticospinal neuron (antidromic invasion to stimulation of contralateral funiculus: PT stim.). A: stretch evoked test response (dot raster as in Fig. 3). B: conditioning stimulus train (15 pulses at 600 Hz, 0.8 mA) suppressed the stretch evoked discharge. The SMA stimulus (arrow) produced some dots because of the stimulus artefacts. C: control as in A. From experiments by Lucier, Rüegg and Wiesendanger; unpublished records.

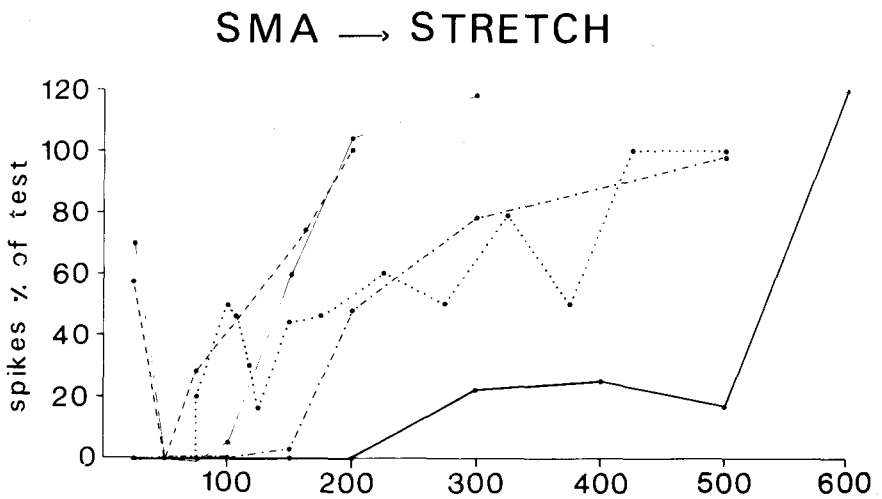


*Figure 8*—Time course of inhibition produced by conditioning stimuli applied to the ipsilateral SMA in five cells of the motor cortex (corticospinal and non-corticospinal neurons). From experiments by Lucier, Ruëgg and Wiesendanger; unpublished records.

cells of the motor cortex activated by stretch stimuli was disappointingly small. These findings have led us to the hypothesis that the output cells are "protected" from feedback signals by a powerful inhibitory control, possibly by the ideally located pericellular plexus from basket neurons (Marin-Padilla, 1969) which are presumed to be inhibitory (Sloper, 1973). According to this hypothesis, structures outside the motor cortex would tonically excite inhibitory basket cells. The "gating of transcortical reflexes" in an appropriate context (Evarts and Tanji, 1974) may be effected by a disinhibition. Because of its structural relationship and because of some clinical observations (reviewed by Wiesendanger, Séguin and Künzle, 1973) we decided to test the possibility that the supplementary motor area (SMA) represents a gating system acting on the motor cortex. Preliminary studies in anaesthetized monkeys were promising. The effect of the SMA was tested by stimulating this area electrically with a 600 Hz train of 15 pulses at intensities ranging from 0.2 mA to 0.8 mA. These conditioning stimuli preceded a stretch stimulus (step or sinusoidal stretch) by 75 msec. All but two of the 16 units in the motor cortex (7 non-PT and 6 PT-neurons; 5 experiments) tested in this way were profoundly inhibited. An example is shown in Figure 7. The effects were long-lasting (100 msec. or more) as illustrated in Figure 8, and may have been mediated directly by fiber connections from the SMA to the motor cortex or indirectly via subcortical loops involving the cerebellum or the basal ganglia. These findings in acute experiments may now provide a guide for chronic experiments. The next step in the examination of transcortical reflexes will be to study the role of gating systems, such as the SMA, in the gain control of long-loop reflexes in awake animals.

*Speculations on the significance of transcortical reflexes*

In view of the existence of a segmental regulatory mechanism for load compensation, well-documented for instance for respiration (Euler, 1966), it might be appropriate to ask what advantage a transcortical servo-loop may have. This question is especially pertinent considering the long time lag involved in the circuit. This long delay makes it unlikely that transcortical load compensation plays a role in the performance of very rapid movements (see also Grillner, 1973); on the other hand long-loop reflexes may be useful in postural stabilization in the presence of background perturbations.

In contrast to the segmental stretch reflex, which usually consists of a fairly synchronized discharge of motoneurons, the long-loop reflex consists of a much more scattered activation of motor units. This temporally dispersed transcortical activation may effectively counteract an inherent tendency of the monosynaptic stretch reflex loop to oscillate. It is probable that there are more than one supraspinal "delay lines" for signals from stretch afferents (Murphy et al., 1975). Long-loop reflexes with different lag times may have a damping effect and may thus be important to counteract the inherent tendency of oscillations in segmental loops. The most obvious advantage of transcortical as compared to segmental load compensation is the fact that the former is much more amenable to adaptive changes than the latter. The existence of a very powerful adaptive gain control, at the cortical level, has now been demonstrated by Evarts and Tanji (1974). This aspect of transcortical regulation is the most interesting one because an adaptive gain control gives the motor cortex the chance to exert its "higher" role of a chief "executive" of complex motor programs, such as the control of the finger movements during piano playing, simply by shutting off all input from peripheral receptors which, in this context, would only blur the precisely timed dispatch of command signals.

Any control system is of course subject to potential damage. Neurological disorders such as rigidity, and the inability to perform rapid alternating movements, may in fact be partially explained by a pathologically high gain in intracortical transmission. The experimental observations by Tatton and Lee (1975) referred to above point in this direction. Hopefully, some of these speculations will be tested experimentally in patients, and monkeys with experimentally induced abnormalities of muscle tone.

## DISCUSSION

*Abrahams* (Queens) commented on the possible meaning of supraspinal projections of spindle information from the upper cervical cord that is devoid of monosynaptic reflexes. Since head movements, vision, and audition are integrated in the upper cervical cord, it may be functionally meaningful to have more cortical than spinal integration to better utilize feedback in the development of motor outputs.

*Wiesendanger* agreed that this may be so, but that such complexity entailed longer time delays, and therefore spinal loops may still be important as well. In reply to *Marshall's* (Ottawa) question as to whether the inhibition originated only from the supplementary motor area, *Wiesendanger* replied that preliminary work suggested this, because stimulation lateral to area 6 produced no inhibition. He allowed however that circuits through the various cortical areas still have to be worked out. He recalled Denny-Brown's demonstration that ablation of SMA causes forced grasping, and that SMA ordinarily seems to hold the grasping reflex in check. It is not known yet whether cortical basket cells are the inhibitory interneurons as suggested by Sloper.

*Brown* (London) was concerned whether the timing of the supposed transcortical reflex was adequately documented. In Evarts' records 10 msec. elapsed between a signal in the postcentral region and PT discharge in the precentral region. In man the time from arrival of a signal from the hand muscles to precentral discharge would be 40 msec., but the latencies of the V2 response revealed an extra 10 msec. Could "gating" in precentral cortex really take 10-15 msec.?

*Wiesendanger* replied in the affirmative: the time for the pathway from area 3a to precentral cortex was such that it could be the route taken by responses to natural stimulation. He also commented on the use of the term "reflex". If "reflex" is defined as a response whose output magnitude is related to that of the input, then this has been demonstrated in the work as reported by *Brooks* for the relation between load pulses and "early" cortical responses.

*Hore* wanted assurance that input from Paccinian corpuscles elicited by vibratory stimuli had been excluded from the cortical responses in the absence of demonstrated projections of muscle spindle 1a afferents to motor cortex. *Wiesendanger* thought it reasonable that vibration offers a more concentrated stimulus for maximal activation of primary spindles to the exclusion of secondaries, and would therefore be a more optimal input to motor cortex than electrical

nerve stimulation at strengths restricted to primaries, that had failed to reveal primary input. Participation of Paccinian receptors cannot be excluded entirely, but is unlikely because Mountcastle had found them unresponsive to even large amplitude frequencies below 50 Hz: the cortical cells however, did respond even to low amplitudes at 6 Hz. Although, as a further safeguard, radial nerve section had been shown to abolish the responses, one cannot be entirely sure that Paccinian corpuscles in the intraosseous membranes could not have been excited. On balance, however one could conclude that the bulk of the signals about load reaching motor cortex were provided by muscle primary and/or secondary spindle afferents.

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