Cooperative Control of Limb Movements by the Motor Cortex, Brainstem and Cerebellum

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Abstract—The model of sensory-motor coordination proposed here involves two primary processes that are bound together by positive feedback loops. One primary process links sensory triggers to potential movements. While this process may occur at other sites, I emphasize the role of combinatorial maps in the motor cortex in this report. Combinatorial maps make it possible for many different stimuli to trigger many different motor programs, and for preferential linkages to be associatively reinforced.

A second primary process stores motor programs and regulates their expression. The programs are believed to be stored in the cerebellar cortex, in the synaptic weights between parallel fibers and Purkinje cells. Positive feedback loops between the motor cortex and the cerebellum bind the combinatorial maps to the motor programs. The capability for self-sustained activity in these loops is the postulated driving force for generating programs, whereas inhibition from cerebellar Purkinje cells is the main mechanism that regulates their expression. Execution of a program is triggered when a sensory input succeeds in initiating regenerative loop activity.

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1. INTRODUCTION

Goal-directed movements of individual limbs are considered to be controlled primarily by the corticospinal and rubrospinal pathways (Kuypers, 1981). The corticospinal pathway originates from the primary motor area in the cerebral cortex whereas the rubrospinal pathway originates from the magnocellular division of the red nucleus in the upper brainstem. Several pathways through which the motor cortex and red nucleus might interact to coordinate the control of limb movements have been discovered (Allen & Tsukahara, 1974). However, the specific mechanisms whereby a cooperative control of the limb might occur are not very well understood. In this report I suggest that positive feedback loops between the motor cortex, brainstem and cerebellum coordinate the initiation and execution of limb motor programs that involve the rubral and cortical descending systems.

The starting point for this synthesis is a recent model that treats the cerebellum as an array of adjustable motor pattern generators (Houk, 1987). This pattern generator concept evolved from a series of neurophysiological and neuroantomical studies of the cerebello-rubrospinal pathway summarized in Houk and Gibson (1987). Insight gained from this analysis led to the hypothesis that cerebellar modules function as adjustable pattern generators, and that an array of these modules transmits a set of motor commands to local feedback systems in the spinal cord; the latter translate the commands into actual limb motion (Houk, 1988). Recently we presented a computer model of an adjustable pattern generator, a neural network inspired by the modular organization of the cerebellum and the anatomy and physiology of the cerebellorubrospinal system (Houk, Singh, Fisher & Barto, 1989). Simulations demonstrated that these rubrocerebellar modules are capable of storing, retrieving and executing simple motor programs.

In the present chapter, this theory concerning the central representation of motor programs is advanced in several ways. First, I discuss how the adjustable pattern generator concept can be extended to include the corticospinal system. Second, I explore some potential triggering mechanisms for initiating the readout of motor programs. Third, I indicate how positive feedback can bind what I will call "combinatorial maps" in the motor cortex to motor programs in the cerebellum. Fourth, I discuss how a cooperative control model might account for several long-standing puzzles concerning the neurophysiology of normal motor function. Finally, I outline how the model could relate to data on cerebellar lesions and recovery of function.

2. REPRESENTATION OF MOTOR PROGRAMS IN RUBROCEREBELLAR MODULES

The mechanisms suggested below for the representation of motor programs in corticocerebellar modules represent extensions of the ideas already developed for rubrocerebellar modules (Houk et al, 1989). Correspondingly, it will be useful to review how motor programs are postulated to be stored, retrieved and executed by the simpler rubrocerebellar modules. Figure 1 illustrates the main pathways for information flow in rubrocerebellar modules.

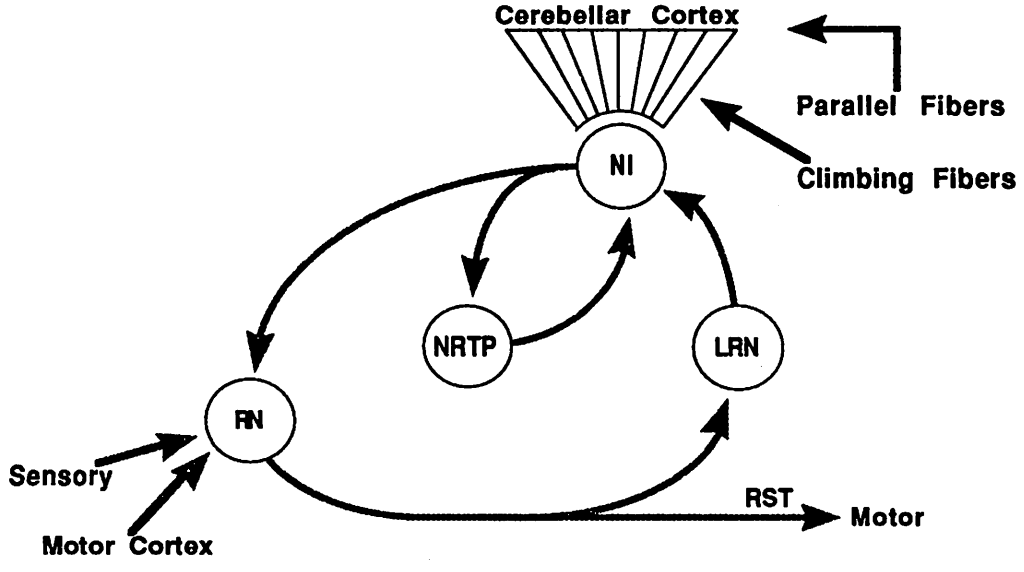

Figure 1: Information flow in rubrocerebellar modules. Note the two positive feedback loops between the nucleus interpositus (NI) in the cerebellum and several brainstem nuclei, and the linkage of these loops to motor output in the rubrospinal tract (RST).

There are projections from the nucleus interpositus (NI) in the cerebellum to several brainstem nuclei, two of which are shown in Figure 1. The magnocellular red nucleus (RN) and the nucleus reticularis tegmenti pontis (NRTP) receive this cerebellar input and then project back to NI either directly or via the lateral reticular nucleus (LRN). Electrophysiological studies suggest that these recurrent pathways operate as positive feedback loops (Tsukahara, Bando, Murakami & Oda, 1983; Houk, 1987; Houk & Keifer, 1989). Regenerative loop activity is normally prevented by inhibitory input to NI from Purkinje cells in the cerebellar cortex. When this inhibition is blocked, loop neurons show increased spontaneous activity and an enhanced responsiveness to extrinsic inputs. The loop receives extrinsic inputs from the motor cortex and from ascending pathways that provide direct sensory input from the periphery, as shown in Figure 1.

According to the adjustable pattern generator hypothesis, the fundamental driving force for the generation of a motor program is the tendency for regenerative activity in these positive feedback loops. Under resting conditions this drive is restrained, since loop activity is inhibited by tonic activity in Purkinje cells. In preparation for a movement, the cerebellar cortex is postulated to reduce the level of inhibition by switching off a set of Purkinje cells. This corresponds to retrieving a motor program from memory. Then, one of the extrinsic inputs triggers an onset of loop activity that is subsequently sustained by positive feedback. Triggering corresponds to the initiation phase of a motor program. Next, the motor program goes through an execution phase. In this phase, loop activity is regulated by the degree of inhibition as determined by how many Purkinje cells were switched off during the preselection period. The execution phase is terminated and the motor program is ended by a strong inhibitory input from the cerebellar cortex. In this manner, motor programs are thought to be shaped into useful patterns of loop activity.

In our simulation model, motor programs are stored in the cerebellar cortex by adjusting the weights of parallel fiber synapses onto Purkinje cells. The training rule is modeled after experimental data regarding cellular mechanisms for synaptic plasticity in Purkinje cells and the response properties of climbing fibers. After a period of training, programs that command accurate movements are retrieved from memory and executed in the manner described in the previous paragraph. The trained network automatically adjusts the parameters of a motor program to accommodate different targets and different initial positions of the limb.

One potentially important feature of this model is that it separates the problem of deciding when to take action (the trigger function) from the problem of specifying what action to take (program retrieval and execution). The cerebellar cortex is held responsible for controlling the preselection and execution of a motor program whereas extrinsic inputs to the loop are held responsible for controlling when a preselected program is actually initiated. The positive feedback loops serve to bind these two processes into a coordinated whole. This binding function, postulated for rubrocerebellar loops, has even greater potential when one considers corticocerebellar loops.

3. EXTENSION TO CORTICOCEREBELLAR MODULES

Numerous pathways for information flow between the motor cortex, red nucleus and the cerebellum have been discovered by anatomical and electrophysiological methods (Brodal, 1981; Allen & Tsukahara, 1974). Several of the more prominent ones are included in Figure 2. Note that these corticocerebellar pathways intermingle with the rubrocerebellar pathways illustrated in Figure 1, and, as a consequence, the rubrocerebellar pathways are subsumed within this more complex network. For example, outflow from the motor cortex

(MC) is directed to the red nucleus, NRTP and LRN (all of which are part of the rubrocerebellar network) in addition to forming the corticospinal tract (CST). The output from the cerebellum is now designated as coming from all three of the deep cerebellar nuclei (CN) rather than just from the IP component of CN.

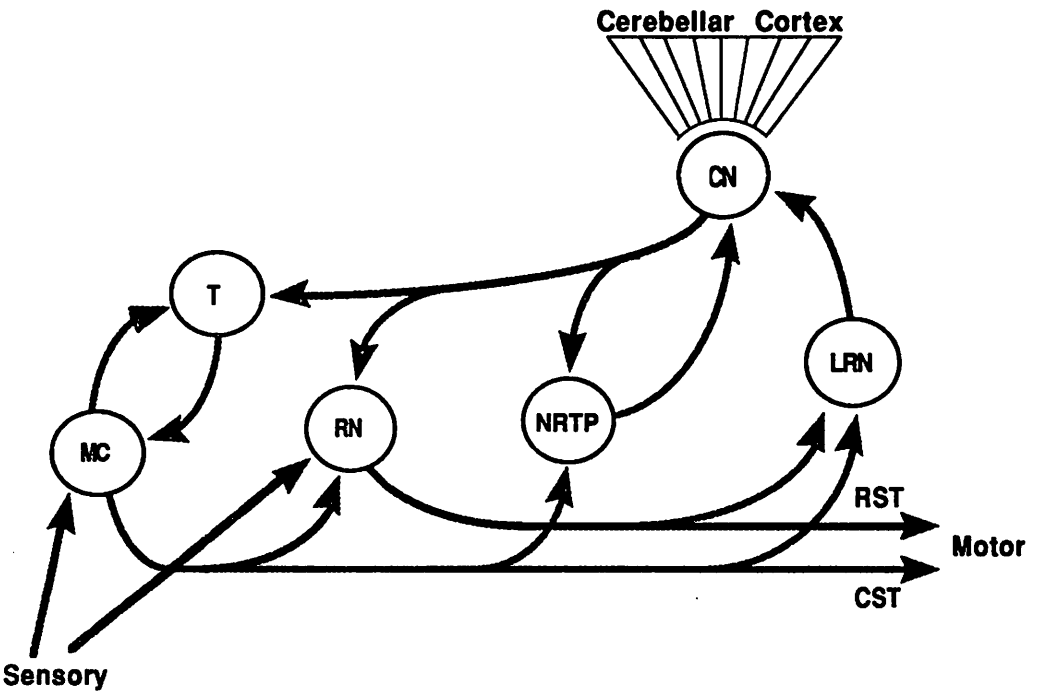

Figure 2: Information flow in corticocerebellar modules. Note that there are five recurrent loops that are subjected to inhibitory regulation by the cerebellar cortex. The controlled spread of activity in these loops is postulated to coordinate motor outflow via the RST and CST.

Entirely new features in Figure 2 are the reciprocal pathways between the thalamus (T) and the motor cortex and the input to the thalamus from the cerebellar nuclei. Most of the new connections are excitatory, and they form additional positive feedback loops that would be expected to result in an intensification and a spatial spread of loop activity. However, the motor cortical projections to thalamus and red nucleus have inhibitory components as well, and the latter may serve to restrain loop activity and limit its the spatial spread.

It is apparent from Figure 2 that the cerebellar nuclei play a special role in this system. Collectively, they serve as a focal point for the regulation of many positive feedback loops that operate in parallel. Five of the six loops illustrated in Figure 2 circulate through the cerebellar nuclei, the exception being the loop between thalamus and motor cortex. This design would appear to facilitate the postulated role of the cerebellar cortex in controlling the selection and execution of motor programs that regulate both spatial and temporal

aspects of motor coordination. The presence of multiple pathways that converge upon this focal point allows the cerebellar control process to operate simultaneously on a whole array of command signals, essentially the entire array of commands that is being distributed to many different muscles via corticospinal, rubrospinal and other descending pathways to the spinal cord.

In addition to providing more feedback loops, the corticocerebellar network also presents many additional points at which trigger signals might act to initiate loop activity. There are new trigger points in the thalamus, the cerebellar nuclei and the motor cortex. The potential of the motor cortical sites for triggering loop activity are particularly interesting to analyze, since so much is known about the input-output relations of motor cortical cells.

4. TRIGGERING FEATURES BASED ON COMBINATORIAL MAPS

There is a rich literature that compares the sensory and motor properties of single neurons in the motor cortex (Asanuma, 1981; Fetz, 1981; Goldring & Ratcheson, 1972; Lemon, Hanby & Porter, 1976). The motor properties are revealed by a cell's activity in association with different movements, by contractile responses to electrical stimulation, or by mapping projections to motor neurons. Individual corticospinal neurons branch to target different combinations of muscles and thus can best be thought of as controlling different elemental movements. The number of cells is very large such that any given movement is multiply represented by a whole colony of neurons with similar output connections. Colonies tend to be grouped into cortical columns, and the columns are organized in topographical maps that spread across the cortical surface.

The sensory properties of motor cortical cells are revealed by responses to touch, joint movement, sounds or visual stimuli. Somatosensory responses to touch or joint movement are most common and often show the strongest responses. Amongst cells with somatosensory responses, many have sensory receptive fields that are near the muscles targeted by the cortical cell. However, other cells have their best sensory responses to stimulation remote from the targeted muscles, and some cells have large receptive fields that include most of a limb or even portions of the trunk. Although certain patterns may be preferentially represented, the basic design of the system seems to be one of insuring that a great variety of sensory-motor combinations is represented in the motor cortex. The significance of this variety has not been adequately explained in the past.

The variety of sensory-motor relations found in the motor cortex results in a sensory-motor map that is well suited, at least in some respects, for triggering movements. To explain this idea I will use an oversimplified model of sensory-motor processing in this section, and append more realistic features in the next section. Consider a colony of cortical neurons that is homogeneous in motor function. This situation is represented in Figure 3 by the set

of 6 motor cortical cells m_j that each control the same movement, designated movement j. I further assume that heterogeneity in sensory function is superimposed upon this homogeneity in motor function . Specifically, I will assume that each cell in the m_j colony receives a different sensory input, the s_i in Figure 3.

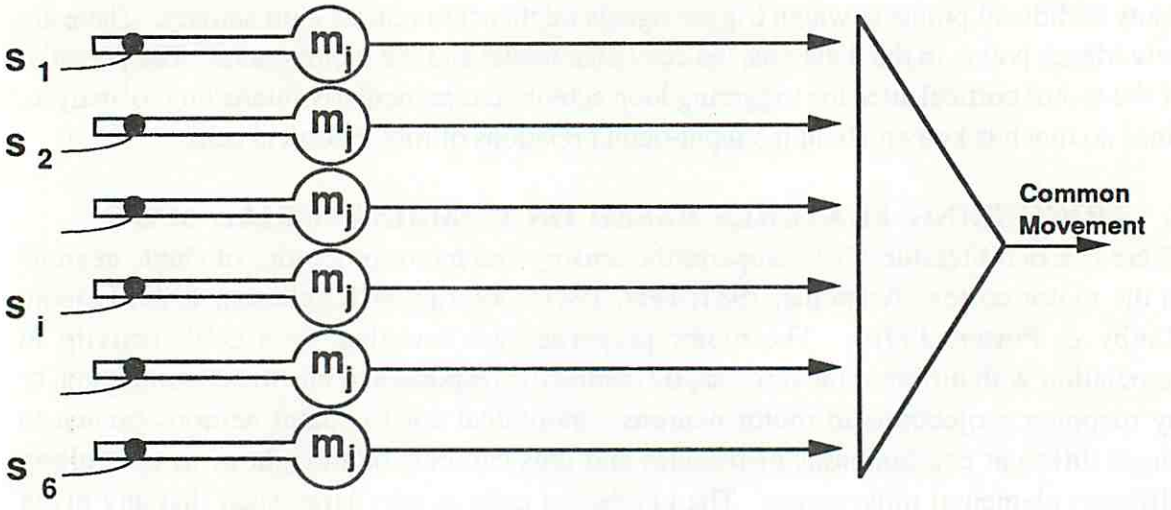

Figure 3: Organization of a colony of motor cortical cells that control a common movement j. Each cell in the m_j colony is postulated to receive a different sensory input, the s_i .

These assumptions capture one facet of the variety in sensory-motor relations found in the motor cortex, namely a variety of sensory inputs to a colony of cells that control the same movement. If different colonies of motor cortical cells controlling different movements were innervated by a similarly diverse set of sensory inputs, as seems likely, the motor cortex would contain a representation of essentially all possible sensory-motor combinations. I will refer to this model of motor cortex as a combinatorial map.

Modeling the motor cortex as a combinatorial map explains how different sensory inputs might trigger the same movement and how the same sensory input might trigger different movements. However, there are several problems with this model in its simplest form. First, we have to assume that each cell has a powerful effect on the muscles it targets, such that its individual activity is sufficient to produce the requisite movement. Second, we need to postulate a rather complex gating mechanism for controlling which sensory-motor relations are expressed at any instant in time. If the latter control were absent and the former assumption were true, virtually any sensory input would provoke a global contraction of all of the muscles of the body. Third, a simple combinatorial map provides

no provision for responding to a given stimulus with several different temporal patterns of motor discharge. This contrasts with the ability of subjects to vary the velocity and duration of movement over a broad range. The latter shortcoming results because the simple combinatorial model includes no provision for elaborating a motor program.

5. BINDING COMBINATORIAL MAPS TO MOTOR PROGRAMS

The reciprocal loops discussed earlier (Figure 2) offer a means for binding combinatorial maps in motor cortex to motor programs stored in the cerebellum. This mechanism simultaneously addresses the other problems with the simplified model. Figure 4 adds two positive feedback loops through the cerebellum to the combinatorial map of Figure 3. I assume that collaterals from each of the cortical cells in colony m_j project to each of the two cerebellar nuclear cells shown in Figure 4 (n_k and n_{k+1}). The nuclear cells in turn project back to the cortical cells to complete several feedback loops. Because of the divergence pattern assumed to exist in these loops, the response of any given cortical cell to its particular sensory input can result in a recruitment of activity in the remainder of the cortical colony. This eliminates the need for postulating powerful actions of individual cortical cells, since movements will now be produced by the collective actions of many cells in a cortical colony. Positive feedback thus solves the first problem noted for the simple combinatorial model of sensory-motor processing.

The probability that a particular cortical colony will be recruited by positive feedback through a cerebellar loop will depend on the state of inhibitory input from the cerebellar cortex onto the loop's nuclear cell. This cerebellar agency for gating permissible sensory-motor combinations seems a natural addition to the temporal shaping features of cerebellar cortical regulation discussed earlier. Input from the cerebellar cortex thus solves both the second and third problems noted for the simple combinatorial model. In this manner, all three of the problems raised in the previous section are addressed by using positive feedback loops to bind combinatorial maps in the motor cortex to motor programs stored in the cerebellum. I will refer to this overall model as a cooperative control model of sensory-motor integration.

The cooperative control model offers a new framework for evaluating neurophysiological and behavioral data collected from subjects performing various sensory-motor tasks. I will provide some examples to demonstrate how this framework can shed new light on previous data that have been difficult to interpret.

A problem that has remained in the literature for many years is that of understanding what goes on during a reaction time. Subjects typically require in excess of one tenth of a second to respond to a sensory cue. While some of this latency is mechanical, involving the conversion of motor action potentials into muscle force and movement, the remaining

central component of reaction time usually occupies a period of 60 to 100 msec or more. This is surprisingly long since conduction from sensory receptors, through motor cortex and back to muscle takes only 20-50 msec as estimated electrophysiologically. Why aren't motor responses produced at electrophysiological latencies, and what neural events occupy the reaction time?

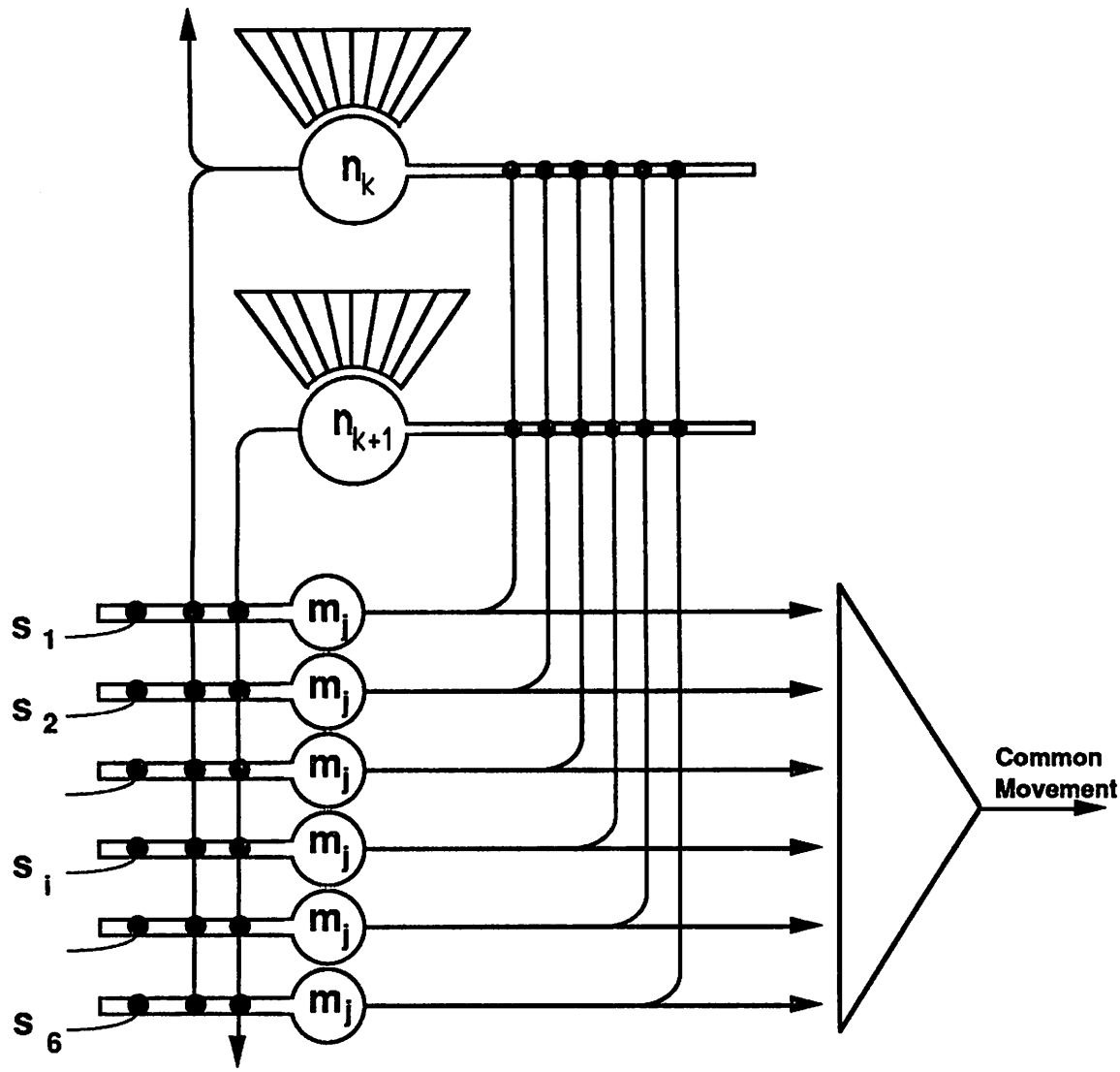

Figure 4: Cooperative control of a movement by a colony of motor cortical cells that is linked to alternative motor programs by positive feedback loops through the cerebellum.

In special cases one can observe motor responses to sensory cues at electrophysiological latencies (Luschei, Saslow & Glickstein, 1967). However, this requires averaging electromyographic potentials from many trials. The short-latency electromyographic responses to the sensory cue are too small to produce significant mechanical outputs. Motor responses instead are associated with larger components of electromyographic response that are delayed by a reaction time.

These previously unexplained results are readily accounted for by the cooperative control model in Figure 4. In fact, they lend strong support to this model. Let the sensory cue be one of the s_i inputs to the m_j colony of motor cortical cells. One of the m_j will thus receive a sensory input and transmit a small excitation to the muscle, the short-latency response that is revealed only by averaging. Activation of the remainder of the m_j cells must await a buildup of positive feedback in the recurrent loop through the cerebellum. It is this later component that produces the actual motor response.

Evarts and his colleagues introduced a behavioral task for dissociating between sensory cue and motor response in animal subjects. Microelectrodes were then used to sample the responses of motor cortical neurons while the animals performed this task (Evarts & Tanji, 1976). Using this paradigm, it was possible to demonstrate short-latency responses to limb perturbations; these components were designated "reflex" since they depended on the sensory input independent of the motor response. These authors also demonstrated longer-latency responses that were designated "intended" components since they correlated with the motor output independent of the sensory stimulus. I prefer to call these two response components "sensory" and "motor"; they are analogous to the two components of electromyographic response discussed in the previous paragraph. The findings of Lamarre and his colleagues (Lamarre, Busby & Spidalieri, 1983) indicate that similar "sensory" and "motor" components of response can be observed in motor cortical cells when visual and auditory signals are used as sensory cues. In both sets of experiments, the number of cells that showed sensory responses was fewer than the number that showed motor responses.

These formerly puzzling results are also readily accounted for by the cooperative control model in Figure 4, using arguments similar to those given above. However, in these cases the data involve cortical recordings and thus relate much more directly to the model. The growth of a sensory component of response into a motor component, seen in some cells, is explained as a case of successful transmission through one of the positive feedback loops that pass through the cerebellum. The failure of a sensory component to grow into a motor component, seen in other cells, is explained by an inhibition (by Purkinje cells) of transmission through the loop that returns to the recorded cell. An absence of a sensory component along with the presence of a motor component, seen for many cells, is explained by a divergence in the positive feedback loop that recruits the recorded cell. The

model similarly explains why cells that show only a motor component of response to one sensory cue show both sensory and motor components of response to another cue. In all cases, the motor components are delayed in a manner that is consistent with a progressive buildup of positive feedback in corticocerebellar recurrent loops.

Another literature that warrants reanalysis in the light of the cooperative control model advanced here concerns the study of stimulus-response compatibility (Fitts & Seeger, 1953; Bauer & Miller, 1982). According to this concept, the speed of reaction to a given stimulus is not just dependent upon the characteristics of the stimulus or the characteristics of the response, but in addition depends on the relationship between the two. More "compatible" relations are said to give rise to shorter reaction times. While this principle has been repeatedly invoked, it has never been given an adequate explanation. The cooperative control model provides a useful framework for considering neural mechanisms that may underlie stimulus-response compatibility; one example will be given in this section and another in the following section.

Movements cued by proprioceptive stimuli have shorter latencies than do movements cued by visual stimuli, and this difference has been demonstrated for matched movements (Newell & Houk, 1983). Shorter proprioceptive latencies are readily explained by the cooperative control model on the basis that more motor cortical cells receive proprioceptive input than receive visual input and proprioceptive responses are typically more vigorous and of shorter latency than visual responses. The shorter latency of proprioceptive responses in motor cortex accounts for part of this effect. In addition, it is likely that a buildup of positive feedback in corticocerebellar loops would be more rapid with the stronger and more extensively represented proprioceptive stimuli.

6. PROGRAMMING THE DIRECTION OF MOTION

Each colony of motor cortical neurons targets a different set of muscles, and therefore promotes a different "preferred" direction of limb motion. Movements of the limb in a particular direction might be controlled only by motor cortical or red nucleus cells with preferred directions in the desired direction of motion. Instead, experiments designed to test this idea have revealed that a population of cortical colonies with a variety of preferred directions is recruited (Georgopoulos, Caminiti, Kalaska & Massey, 1983). It is the vector combination of these preferred directions, weighted by the intensity of each colony's activity, that predicts (and presumably controls) the actual direction of motion. This contravariant coding scheme is the one expected for an output stage of the motor system (Pellionisz & Llinás, 1979). Indirect evidence suggests that similar principles may apply to the red nucleus (Gibson, Houk & Kohlerman, 1985) and to the cerebellar nuclei (Thach, Perry & Schieber, 1982), and recently direct support for contravariant population coding in

both cerebellar Purkinje and nuclear cells has been provided (Fortier, Kalaska & Smith, 1989).

While Figure 4 shows only one colony of motor cortical cells, mechanisms for coordinating populations of motor cortical colonies can be contemplated by focusing upon the two cerebellar nuclear cells n_k and n_{k+1}. Although both nuclear cells reinforce activity in the m_j colony, they also branch to innervate additional colonies of cortical neurons. Thus, each nuclear cell controls a population of cortical colonies, and the equivalent vector of the nuclear cell will be the vector combination of the population vectors of the individual cortical colonies, weighted by the synaptic strengths of the inputs from the nuclear cell. Since the different nuclear cells branch to innervate different combinations of colonies, their equivalent vectors will point in different directions. Movements in different directions could be controlled by activating individual nuclear cells or by activating populations of cells. Microelectrode recording studies suggest that sizeable populations of cells are active in coordinated reaching and exploratory movements of the limb, although smaller groups of cells, some of which are intensely activated, are used when isolated tracking movements are made (MacKay, 1988; Van Kan, Houk & Gibson, 1986).

In section 2, I summarized a model for the storage of motor programs in the cerebellar cortex. According to this model, the cerebellum can be viewed as an array of adjustable pattern generators. However, I discussed only the manner in which a single pattern generator is presumed to operate. While an individual pattern generator can be adjusted to regulate the temporal features of a motor program, groups of pattern generators are required to regulate spatial features. This is because individual pattern generators are assumed to have relatively stable connections with the colonies of motor cortical cells discussed above, and with analogous colonies of red nucleus neurons. As a consequence, each pattern generator controls movement in a single direction, and the only way to change the direction of a movement is to activate a different pattern generator, or a different population of pattern generators. The evidence reviewed in the previous paragraph suggests that coordinated movements of the limb as a whole should be represented by activity in a population of pattern generators, with different directions of motion being represented by differently weighted combinations of pattern generators.

The multiple recurrent loops in Figure 2 promote an image of how populations of pattern generators might be recruited to control movements of a limb. Since there is likely to be significant divergence and convergence in the various loops, it would be difficult to place spatial boundaries on individual pattern generators -- instead, pattern generators are probably best thought of as partially overlapping modules. A sensory trigger to either the motor cortex or red nucleus would be expected to have a succession of actions. The first action would be an activation of a small fraction of cells in several colonies of cortical or

rubral neurons, which would result in a short-latency, but insignificantly small, motor response. This small response would also traverse positive feedback loops through the cerebellum and back to the colonies of cortical and rubral cells that initiated it. Depending on the state of Purkinje inhibition from the cerebellar cortex, this might be sufficient to trigger the initiation of a motor program in a small number of pattern generator modules. Once a program is initiated in a few colonies of cortical and rubral neurons, it would tend to spread to other colonies (and hence to other pattern generators) due to divergence in the positive feedback loops. Purkinje inhibition would be an important factor in regulating this spread and thus controlling the spatial features of the motor program.

The image of a progressive growth of activity in positive feedback loops between the cerebellum, motor cortex and brainstem nuclei may account for previously unexplained data concerning the timing of activity in these structures and the timing of movement. A broad range of lead (and some lag) times has been observed for the onsets of single unit activity with respect to the onset of movement, and there is very little difference between the means derived from recordings in motor cortex and cerebellum (Fetz, 1981; Thach, 1975). This result, which was disappointing to early investigators since it did not help to answer the question as to which structure comes first in the initiation of a movement, is well accounted for by the concept of recruitment of neurons in divergent recurrent loops. A related result that is also well explained is the observation that cooling of cerebellar nuclei delays the onset and buildup of motor cortical discharge while simultaneously delaying the onset of movement (Meyer-Lohmann, Hore & Brooks, 1977). Cooling would be expected to slow down the growth of regenerative activity in the various loops.

The discussion of stimulus-response compatibility in the previous section dealt with the compatibility of different modalities of stimulus, whereas the concept has usually been applied to compatibility within the same modality. A good example in the realm of visuomotor performance concerns the compatibility between the direction of a visual stimulus and the direction of the required motor response. Reaction times are shortest when the stimulus and response are in the same direction, and they increase progressively as the angle between the stimulus and response is increased. This observation has promoted the hypothesis that subjects need to perform a progressive mental rotation of a target vector before they can make their movement (Shepard & Cooper, 1982). Recently it has been shown that the population vector recorded by sampling motor cortical cells actually undergoes a progressive rotation during the interval prior to the initiation of a movement, suggesting that single-cell recordings can reveal the neural correlates that underlie cognitive processes (Georgopoulos, Lurito, Petrides, Schwartz & Massey, 1989). The authors hypothesized that the directional transformation required by the task was achieved by a rotation of an imagined movement vector, as represented by discharge in a

population of motor cortical cells. A specific neural mechanism capable of regulating this rotation was not provided.

The image of spreading activity in corticocerebellar loops promoted earlier suggests a mechanism that might regulate the observed rotation of the cortical population vector. Assume that visual projections to the combinatorial map in motor cortex preferentially innervate cortical colonies that promote movement in the direction of the visual target, due perhaps to the fact that this is the most frequently utilized manifestation of visuomotor coordination. Then, the first cortical colonies to respond during a reaction time in the non-compatible task would be colonies linked to the pattern generators that point toward the compatible target. The observed rotation of the population vector might correspond to a spread of activity to other pattern generators that progressively point in directions closer to the desired direction of motion. This spread of excitation would be accompanied by a progressive inhibition of the pattern generators originally recruited. According to the hypothesis advanced here, the cerebellar cortex would be primarily responsible for regulating the shifting pattern of activity, i.e., the neural correlate of mental rotation. Here again, the cooperative control model provides a novel, and testable, explanation for the neural mechanisms that may underlie performance in a sensory-motor task.

7. CEREBELLAR LESIONS AND RECOVERY OF FUNCTION

The multiple alternative pathways illustrated in Figure 2 suggest various possibilities for recovery of function subsequent to lesions of part of this system. There are alternative trigger points and alternative recurrent loops that might be capable of initiating and sustaining motor programs. In this section I review the effects of cerebellar lesions and subsequent recovery processes from the perspective of the cooperative control model.

The main symptom of cerebellar lesions is an inability to control movements called dysmetria (Holmes, 1939; Growdon, Chambers & Liu, 1967). Lesions of the intermediate and lateral cerebellar nuclei affect mainly goal-directed limb movements whereas medial lesions affect mainly eye, head, trunk and postural limb movements. The inability to control movements fits with the postulated role of the cerebellum as a site for the storage, recall and execution of motor programs. However, the affected movements are only dysmetric (wrong size and wrong direction) rather than being completely abolished, and there is a progressive recovery of limb accuracy that begs explanation. Both of these features are interesting to analyze within the framework of the cooperative control model.

The intermediate and lateral cerebellar nuclei are particularly interesting sites, since lesions here interrupt most of the positive feedback loops between the cerebellum and premotor neurons in red nucleus and motor cortex. Such lesions would be expected to abolish most of the driving force proposed to underlie the generation of motor programs. However,

Figure 2 shows that there is at least one loop that is unaffected by this lesion, the loop between the thalamus and motor cortex. The brief period of complete loss of movements about the affected joints could represent a period during which positive feedback in the thalamocortical loop is strengthened by an adaptive process. If so, then recovery should be reversed by a subsequent lesion of thalamus or motor cortex. In fact, such lesions permanently abolish the independent use of the limb, except under conditions of special training (Growdon, Chambers & Liu, 1967).

While thalamocortical loops may become capable of sustaining motor discharge, they would lack the regulation normally provided by input to the loop from the cerebellar cortex. Since this inhibitory input is thought to be particularly important in regulating the velocity and endpoint of a movement, these fine features of movement might be more permanently affected than simply the ability to move. The former deductions, in as much as they have been tested, appear to fit well with the observed deficits (Goldberger, 1974). Although postures can be held, and movements lacking a precise target can be made, there is a profound inability to stop a movement at a targeted point in space, even after a considerable recovery period. There is less information regarding the ability to move at different velocities, and this might be informative to test in the future.

Lesions of the cerebellar cortex overlying the cerebellar nuclei would be expected to remove the normal inhibitory restraint on positive feedback, thus enhancing evoked excitability and spontaneous activity of motor outflow in both corticospinal and rubrospinal pathways. In line with this prediction, hypersensitivity to touch, exaggerated withdrawal and placing reactions, general hyperreactivity and hypermetric movements are some of the reported signs (Chambers & Sprague, 1955).

Cerebellar lesions interfere with several types of sensory-motor learning (cf. Ito, 1984). Recently, much interest has centered around cerebellar mechanisms that might mediate flexion of a limb or blink of an eye in response to a tone or light conditioned stimulus, since cerebellar lesions have been shown to severely impair or abolish these classically conditioned responses (Glickstein, Yeo & Stein, 1987; Thompson, 1986). Most authors have emphasized the effects of the cerebellar lesions on the associative linkage formed between conditioned and unconditioned stimuli, hypothesizing that this linkage is formed between an unconditioned stimulus transmitted by a climbing fiber input to the cerebellum and a conditioned stimulus transmitted by a mossy fiber / parallel fiber input. However, other authors have emphasized motor deficits, a weakening and delay of response, caused by cerebellar lesions (Welsh & Harvey, 1989). I will attempt to fit these observations into the framework of the cooperative control model of sensory-motor processing.

The present model incorporates two types of learning. The main function of learning mechanisms in the cerebellar cortex is the storage of motor programs. In contrast, the learning of appropriate trigger signals for initiating these programs is an associative process that is more likely to occur in the motor cortex (Woody, 1982), the red nucleus (Tsukahara, Oda & Notsu, 1981) or at one of the other sites along the corticocerebellar or rubrocerebellar loops (Figure 2). I interpret the observation that cerebellar lesions may only weaken or delay learned responses without actually abolishing them as a deficit in the learning of motor programs. The limited recovery observed in some cases might be mediated by thalamocortical loops, as discussed earlier, or by other loops that exist in the brainstem. Older studies have shown that instrumentally conditioned movements can survive removal of both cerebellum and motor cortex (Goldberger, 1974), and it would be interesting to investigate the effects of dual cerebellar and motor cortical lesions on classically conditioned responses.

Overall, the data on cerebellar lesions and recovery of function support the concept of cooperative control based on recurrent pathways between the cerebellum and premotor nuclei. According to this concept, well-adapted motor programs are stored in the cerebellar cortex by a motor learning process, whereas the triggers that initiate the execution of these programs are formed as associative linkages in the motor cortex, the red nucleus or at other sites along the positive feedback loops to and from the cerebellum.

8. SUMMARY AND CONCLUSIONS

The model of sensory-motor coordination proposed here involves two primary processes that are bound together by positive feedback loops, as recapitulated in Figure 5. One primary process links sensory triggers to potential movements. While this process may occur at other sites, I have emphasized the role of combinatorial maps in the motor cortex in this chapter. These maps make it possible for many different stimuli to trigger many different motor programs, and for preferential linkages to be associatively reinforced.

A second primary process stores motor programs and regulates their expression. The programs are believed to be stored in the cerebellar cortex, in the synaptic weights between parallel fibers and Purkinje cells. Positive feedback loops between the motor cortex and the cerebellum bind the combinatorial maps to the motor programs. The capability for self-sustained activity in these loops is the postulated driving force for generating programs, whereas inhibition from cerebellar Purkinje cells is the main mechanism that regulates their expression. Execution of a program is triggered when a sensory input succeeds in initiating regenerative loop activity.

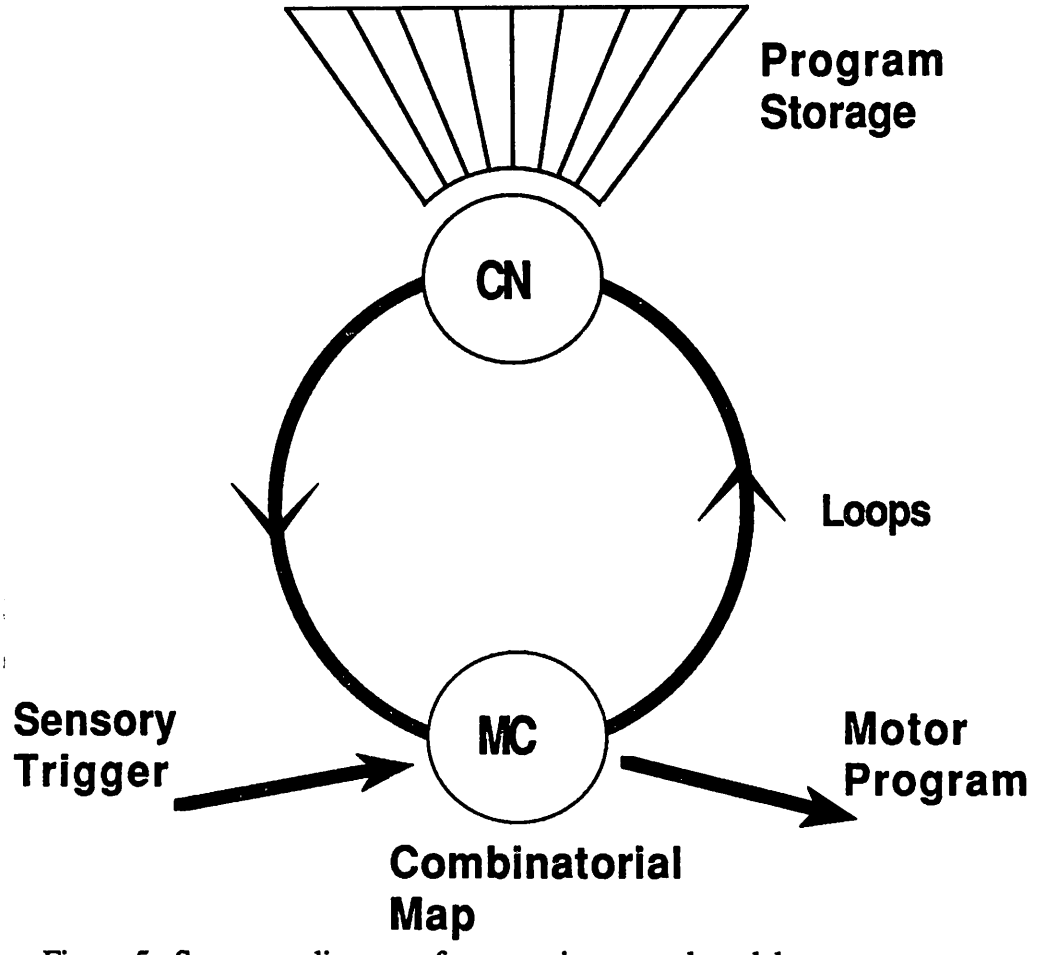

Figure 5: Summary diagram of cooperative control model.

This cooperative control model was motivated by a desire to incorporate diverse anatomical, physiological and behavioral data, rather than by some concept of how the system should be designed. A separate representation of motor programs and their triggering events emerged naturally from the analysis rather than driving it. In fact, the potential utility of this separation is not entirely clear at present. The system appears to represent a compromise between memory-based and computational approaches. A more memory-based approach might store a motor program and its triggering event together, and use additional memory locations to store the same program with each of its alternative triggers. A more computational approach might store a general algorithm that is used iteratively to produce all the different motor programs. Additional theoretical work is needed to determine whether or not the cooperative control scheme is an efficient way of combining memory-based and computational approaches.

The present model does not deal with the nonprecentral areas in the cerebral cortex nor with the basal ganglia, both of which are known to be involved in the control of movement (Alexander, DeLong & Strick, 1986; Humphrey, 1979). In the future it will be profitable to explore extensions of the present theory of sensory-motor representations to include the possible involvement of these other cerebral areas in motor planning, self-initiated movements and the control of action sequences.

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