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2000-09

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September, 2000

Technical Report CAS/CNS-2000-020

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Cortical Models for Movement Control

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Introduction: Constraints on modeling biological neural networks.

The goal of this chapter is to describe recent elaborations of a neural network model of cerebral cortical contributions to the planning and control of voluntary movements. A model is a system of structural and functional relations intended to be interpreted and critically evaluated as a partial representation of some other system. Although many kinds of models of cortical control of voluntary movement are possible, this chapter's focus is on neuronal and neural network modeling, which seeks to represent both structural and functional relationships among the cellular constituents of the brain as part of an attempt to explain intelligent behavior. By using the mathematics of differential equations as the primary representational language, we can use computer simulations to compute the dynamical real-time implications of a very large set of locally specified processes – and thus escape the severe predictive limitations of unaided imagination.

Because of the complexity of neurons, of the networks that they compose, and of the sensory-motor systems with which these networks interface, any model of movement control must be abstract, i.e., highly selective in the set of natural properties highlighted by the model. To ensure that such abstractness does not come at the cost of neural realism, it is important to avoid assuming any structural or functional relationships that contradict known limitations of the modeled neural system. For systems-level neural network modeling, which aims to explain adaptive behavioral phenotypes – such as our ability to reach to an object's location under visual guidance – the most important data constraints on neural modeling fall into four classes: psychophysics, circuit neuroanatomy, circuit neurophysiology, and task-dependent cellular firing patterns.

Here, *psychophysics* refers to the study of the perception, cognition, and behavior of the whole animal from a parametric functional perspective. Psychophysicists seek to quantify relationships between physical variables and psychological variables in order to characterize the abilities and

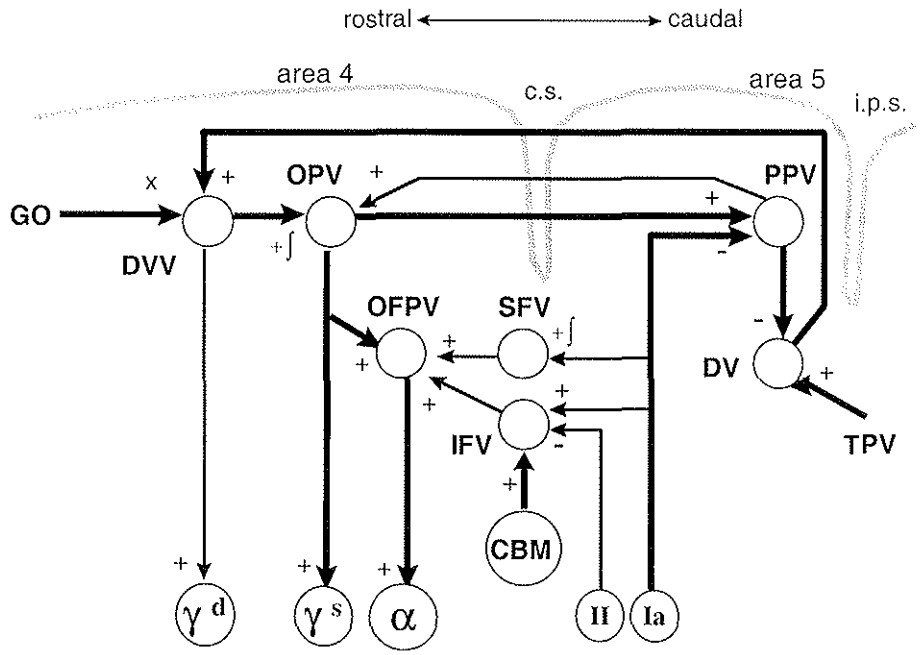
limitations of the animal under well-controlled conditions. *Circuit neuroanatomy* is the study of anatomically identifiable cell types and their roles in the synaptic organization of the central nervous system (CNS). If by "synapse" we mean "locus of neuronal inter-action", then the synaptic organization sets limits on potential interactions among neurons and other cells (e.g., neuroglial cells) intrinsic to the nervous system. *Circuit neurophysiology* is the study of the dynamic interactions that may and do occur within and between cellular constituents of the CNS. Here, although the focus of systems modelers has typically been on excitatory and inhibitory synaptic actions, and on the inherently nonlinear relationships between inputs and outputs for single neurons, recent systems models have begun to incorporate further dynamical properties, such as the slow processes made possible by intracellular metabolic cascades and second messengers (e.g., Fiala et al. 1996). Finally, the study of task-dependent *cellular firing patterns* observable in alert behaving animals has created an inventory of physiologically identifiable cell types, i.e., cells that can be classified on the basis of distinctive and reliable response profiles that are revealed when firing rates plotted versus time are time-aligned relative to key task events, such as target stimulus onset, trigger signal (go stimulus) onset, or movement onset.

Recent rapid expansions of the data base in these four areas – psychophysics, circuit neuroanatomy, circuit neurophysiology, and task-dependent cellular firing patterns – have created a very rich set of constraints that provide guidance in the model development process. The next section briefly reviews a set of the preeminent data constraints in preparation for presentation of a functional, network-level, explanation.

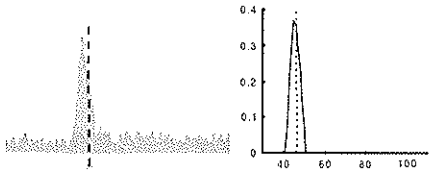
Cellular firing patterns in monkey cortical areas 4 and 5.

Cell populations in areas 4 and 5 of the cerebral cortex have been shown to be strongly active during voluntary reaching movements. Lesion studies have shown that all such movements requiring independent control over individual contributing joints, or individual fingers, strictly require the primary motor cortex (cf. Passingham, 1993), area 4, which is tightly linked with parietal area 5. The data discussed here are drawn primarily from studies of monkeys making reaches to specified spatial targets following onset of a go stimulus scheduled by the experimenter (e.g., Georgopoulos et al. 1982; Kalaska et al. 1989; Kalaska & Drew, 1993).

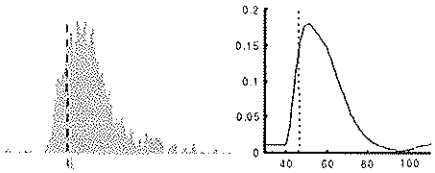
Figure 1. (Next page). Model circuit for 6 physiologically identified cell types in areas 4 and 5 of the cerebral cortex. Upper part: Proposed cortical circuit with links to alpha and gamma motoneurons and muscle receptors. CBM = input from cerebellum (via thalamus, as in Figure 3). Lower part: Columns 2 and 4 show *simulated* activation profiles corresponding to the *observed* cellular activation profiles shown in columns 1 and 3.



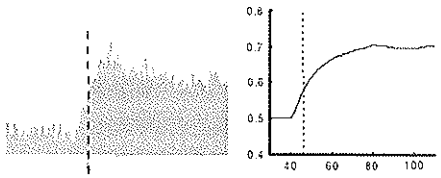
(a) area 4 phasic RT (**IFV**)



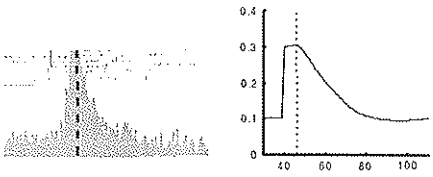
(b) area 4 phasic MT (**DVV**)



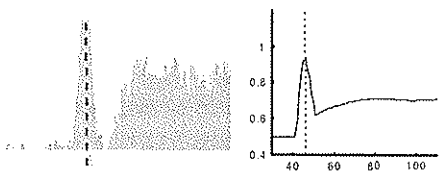
(c) area 4 tonic (**OPV**)



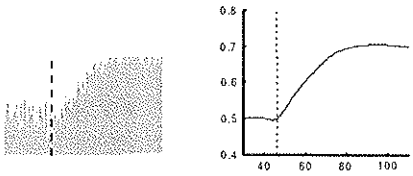
(d) area 5 phasic (**DV**)



(e) area 4 phasic-tonic (**OFPV**)



(f) area 5 tonic (**PPV**)



In Figure 1's lower half, the gray response profiles (firing rate vs. time) on the left side of panels (a)-(f) show physiological "signatures" of six distinctive physiologically identified cell types from areas 4 and 5 of monkey cerebral cortex (Kalaska et al., 1989; 1990). For each type, Table 1 typically lists two or more experimental reports, from distinct labs, that have documented the existence of these cell types. For each profile, the vertical dashed line indicates the time of movement onset. The functional names given to these cell types capture the repeatable, task-dependent properties of the named cell types' response profiles just before, during, and after a voluntary reaching movement made with the contralateral arm.

Model element	**Cell type by physiology**	**References**
Desired velocity vector (DVV)	area 4 phasic movement-time	(Fromm, et al., 1984; Georgopoulos, et al., 1982; Kalaska, et al., 1989)
Outflow position vector (OPV)	area 4 tonic	(Fromm, et al. 1984; Kalaska, et al., 1989; Kettner, et al., 1988)
Outflow force + position vector (OFPV)	area 4 phasic-tonic	(Cheney & Fetz, 1980; Cheney & Fetz, 1984; Fromm, et al., 1984; Kalaska, et al., 1989)
Inertial force vector (IFV)	area 4 phasic reaction-time	(Kalaska, Cohen, Hyde, & Prud'homme, 1989)
Static force vector (SFV)	area 4 tonic or subcortical?	see above
Difference vector (DV)	posterior area 5 phasic	(Burbaud, et al., 1991; Chapman, et al., 1984; Crammond & Kalaska, 1989; Kalaska, et al., 1990; Lacquaniti, et al., 1995)
Perceived position vector (PPV)	anterior area 5 tonic	(Burbaud et al., 1991; Kalaska & Hyde, 1985; Kalaska, et al., 1990; Lacquaniti, et al., 1995)
Target position vector (TPV)	area 5 or area 7b	(Lacquaniti, et al., 1995; Robinson & Burton, 1980; Dum & Strick, 1990)
GO signal	globus pallidus	(Horak & Anderson, 1984b; Horak & Anderson, 1984a; Kato & Kimura, 1992)

Table 1. Proposed correspondence between model elements and cell types. An expanded set of citations can be found in Bullock et al. (1998) and Cisek et al. (1998).

The Figure 1 profiles have *phasic*, i.e., transient, components, as well as *tonic*, i.e., quasi steady-state, components. Moreover, the phasic component may be largely restricted to the reaction time (RT) interval that

extends from the go stimulus till the time of movement onset (see panel a), or it may begin in the RT interval and extend well into the movement time (MT) interval (see panels b & d). Of the 6 cell types shown, 3 are phasic, two are tonic, and one (see panel e) is phasic-tonic. Other cell types are also observable in these areas (cf. Kalaska et al. 1997), but these types represent a majority of the strongly movement-related types that have been reliably observed in association with shoulder, elbow and wrist movements contributing to primate reaches. These profiles present a key part of the puzzle to be solved. Unlike the responses often observed in sensory cortex, they are not stimulus driven. What we want to understand is how these profiles are so reliably structured in time, and how this reliable temporal structuring contributes to the movements known to depend on an intact motor cortex. Are these temporal structures mutable patterns that emerge in real time from network computations sensitive to the evolving context? Or are they being retrieved as preformed patterns from memory? Or might there be a *mixture* of computation-based and memory-based components in these profiles? The model to be presented is a mixture model, but the primary emphasis is on the enduring need in voluntary movement for mutability. Hence we emphasize how these patterns can arise from network computations that enable the sensory-motor system to generate movements that remain sensitive to the evolving context of action.

Anatomical links between areas 4 and 5, spinal motoneurons, and sensory systems.

A key basis for understanding how these firing patterns could arise in principle from network computations is information about how cells in areas 4 and 5 are linked to other brain areas and to both sensory and motor neurons. The upper part of Figure 1 schematizes the left cerebral hemisphere and shows some of the known pathways that link areas 4 (rostral to the c.s., central sulcus) and 5 (rostral to the i.p.s., intraparietal sulcus) to each other and to sensory and motor neurons, and Table 2 cites the empirical reports that document the depicted pathways. Regarding the schematic circuit, two caveats are in order. First, the circuit incorporates only a subset of the known pathways into and out of these areas. Second, the circuit in some respects goes beyond what is currently known, by suggesting not only fiber projections between areas, but also that certain pathways between areas make functional contacts with only one or another cell type within the target area. In fact, the circuit incorporates a set of modeling hypotheses of the following general form: the known projection between (broad) areas A and B can be used as part of a computational model to help explain the physiological signatures of multiple cell types in areas A and B on the assumption that the projection encompasses one or more functionally distinct pathways, e.g., one linking cell type A1 to type B1 and one linking cell type A2 to type B2. Such

a proposal is often necessary to complete a model because it is rarely possible to find data capable of definitively aligning a neuroanatomical map with a neurophysiological map of cell types. The general features of such a proposal are also justified on empirical grounds. First, barring intrinsic differences, two cells with equal connectivity would not be expected to have such divergent behavior as to fall into different classes. Thus a difference of connectivity must exist to explain divergent behavior when the cells are not intrinsically different. Second, it is the norm in neuroanatomy to discover that any large scale projection is composed of numerous distinct pathways.

Even if data were available to allow a definitive alignment of the cell types in the lower part of Figure 1 with the nodes in the connectivity scheme of the upper part of Figure 1, we would still be far short of an understanding of the system. For understanding, we need a theory of how the circuit supports behavioral functions that are vital to the animal's adaptive success. Such a theory is implicit in the proposed *functional/computational* names (e.g., DV, difference vector) of the cell types given in Table 1 and used to label nodes of the circuit in the upper part of Figure 1. We now turn to a summary of the theory (Bullock, Cisek & Grossberg, 1998) behind this set of functional names.

Model connection	**Corresponding pathway**	**References**
spindle - SI	spindle to SI	(Oscarsson & Rosen, 1963; Phillips, et al., 1971; Prud'homme & Kalaska, 1994)
SI - PPV	SI to area 5	(Jones, et al., 1978)
OPV - PPV	area 4 to area 5	Evarts, 1974; Jones, et al., 1978; Pandya & Kuypers, 1969)
PPV - OPV	anterior area 5 to area 4	(Johnson, et al., 1993; Jones, et al., 1978; Strick & Kim, 1978; Zarzecki, et al., 1978)
DV - DVV	posterior area 5 to area 6	(Jones, et al., 1978)
	area 5 to area 4	(Johnson, et al., 1993; Strick & Kim, 1978; Zarzecki, et al., 1978)
OPV - gamma MNs	area 4 to gamma MNs	(Brooks, 1986; Pandya & Kuypers, 1969)
OPPV - alpha MNs	area 4 to alpha MNs	(Brooks, 1986; Pandya & Kuypers, 1969)

Table 2. Evidence for some of the connectivity assumed in the model.

How insertion of a *time delay* can create a niche for deliberation.

To understand the potential computational role of the above reviewed anatomy and physiology of the cerebral cortex, it is useful to begin with the distinction between voluntary and involuntary movement. The paradigm of an involuntary movement is unconditional reflexive movement, which cannot be suppressed by anything short of an external physical constraint or a neural network lesion. Whatever else the animal may be doing, an external stimulus unconditionally elicits the reflexive movement after a fixed latency that depends solely on the intrinsic dynamics of a short, often monosynaptic, sensory-motor linkage. In contrast, the paradigm of voluntary movement is conditional action undertaken after a variable period of preparation that imposes a "cost" in the form of a longer response latency. But there are enormous benefits associated with this extensible time delay, because it allows the completion of slower processes that may either enhance the efficacy of, or suppress and replace, the prepotent candidate act. At least three benefits are notable. An extensible time delay creates the space within which true deliberation emerges on the evolutionary scene. This deliberation involves a covert search for alternatives and evaluation of their relative merits vis-à-vis learned criteria applied to the expected consequences of enacting each alternative. Second, a time delay allows preparation of sensory, neural, and mechanical preconditions for successful execution of whatever act is selected by the deliberative process. Finally, a time delay allows initiation of the chosen act to be optimally timed vis-à-vis the evolving context of action.

A volition-deliberation nexus and voluntary trajectory generation.

Among the neuroscience issues that arise out of the popular distinction between voluntary and involuntary movement, my efforts have focused on the nexus of issues that emerge with the idea that alternative voluntary movements can be primed during a period of deliberation. By closing a gate across the sensory-motor pathway, a brain can create an interval during which slower-to-complete analyses can contribute to an ultimate decision to act. Once that decision is made, the gate can be opened in only that sensory-motor pathway associated with the chosen action. We can further imagine such a gate opening at varying rates, or to varying degrees, in order to control the rate of movement execution. We have used these ideas to build a neural network model of how we voluntarily vary speed without disrupting the form – distance and direction – of a planned point-to-point movement. The name of this circuit is the VITE model, which has now undergone several stages of progressive elaboration to explain a diverse array of data sets (Bullock & Grossberg, 1988a, b; 1991; Bullock, Bongers, Lankhorst, and Beek, 1999; Bullock, Grossberg & Guenther, 1993; Bullock, Cisek & Grossberg, 1998; Cisek, Grossberg, and Bullock, 1998; Jacobs and

Bullock, 1998). Here "VITE" is after the French for "quickly" (because the model seeks to explain voluntary speeding of movement), but is also an acronym for **V**ector **I**ntegration **T**o **E**ndpoint, as explained below.

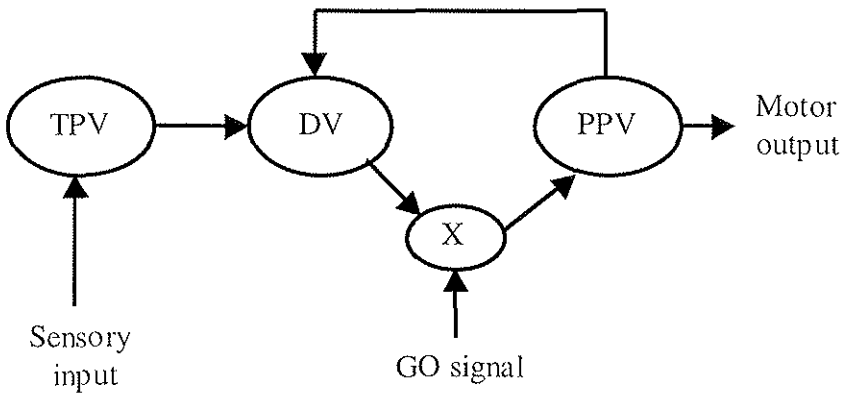


Figure 2. The VITE circuit. A volitional GO signal determines whether a computed movement specification, the DV (difference vector) is allowed to control action of the effectors. TPV = target position vector; PPV = present position vector; X = multiplicative interaction.

The simplest version of the VITE model, schematized in Figure 2, consists of the minimal processing stages necessary for the priming and voluntary generation of variable speed PTP (point to point) movements, e.g., of a hand reaching to a target or goal position. The target position vector (TPV) stage shown in Figure 2 represents desired positions, such as the positions of visible targets, in body centered coordinates. These body centered coordinates might be motor coordinates, such as joint angle or muscle length coordinates, or spatial coordinates, such as polar or cartesian coordinates. The present position vector (PPV) stage represents the actual position of the hand in the same coordinate system. The discrepancy between TPV and PPV is continuously computed at the difference vector (DV) stage, using excitatory signals from the TPV and inhibitory signals from the PPV. Difference vectors represent both magnitude and direction information, specifying the displacement needed to contact the target. At the DVxGO stage, the DV output signal is multiplied, or gated, by a GO signal. While the GO signal is zero valued, any DV command is prevented from execution. Thus instating a TPV and computing a DV while GO is zero constitutes the operation of motor priming, which can be detected by measuring cell discharges in frontal cortex (e.g., Georgopoulos et al., 1993; Kalaska & Crammond, 1995). Such priming is necessary for deliberative activity and for minimizing the reaction time of movements whose success depends on quick

execution in response to external signals. When the GO signal becomes positive, the PPV stage starts integrating signals at a rate proportional to DV times GO. Because the GO signal is a scalar multiplier, voluntary changes in the amplitude of the GO signal can modulate movement rate without affecting the direction coded by the difference vector (DV).

Because of these relations, the DVxGO signal can be interpreted as a movement velocity command, and the PPV can be interpreted as a present position command. In fact, the global shape of the temporal evolution of the model's DVxGO variable has been shown to match experimental velocity profiles for PTP movements of the hand quite well, and significantly better than alternative models (Bullock & Grossberg, 1988a, b, 1991; Nagasaki, 1989; Zhang & Chaffin, 1999). In a more complete version of the VITE model, a distinction is made between a present position command and a perceived position vector (Bullock, Cisek & Grossberg, 1998; see below), but for present purposes, these two functional roles may be attributed to a single PPV stage. In that case, PPV output defines that position of the endeffector that lower-level, force-generating, circuits attempt to instate. Among the models pertinent to lower level control is a sensory-spino-muscular circuit known as the Factorization of LEngth and TEnsion (FLETE) model, which explains how positions can be maintained at variable joint stiffness levels (Bullock, 1995; Bullock & Contreras-Vidal, 1993; Bullock & Grossberg, 1991; van Heijst, Vos & Bullock, 1999). Thus the overall theory suggests that variable speed control and variable stiffness control are achieved at distinct levels within the motor hierarchy.

While performing a normal PTP movement, the TPV is constant during the entire movement. Initially, some discrepancy between the PPV and TPV is registered at the DV. When the GO signal is activated, the product DVxGO becomes positive. As a result, the PPV begins to change in the direction of the TPV. This causes the arm to move, and as it approaches the target the discrepancy between TPV and PPV, computed as the DV, declines toward zero. However, this does not immediately reduce the velocity, because whereas the DV is declining, the GO signal is growing. The product of these two signals first grows and then declines, giving rise to a bell-shaped velocity profile. Ultimately, the movement causes PPV to match TPV, at which time DV reaches zero, as does DVxGO. Therefore the PPV ceases to change, and the arm stops moving (assuming that the PPV command is well followed with help from lower order circuits, e.g., spinal circuits incorporated in the FLETE model). Note that the PPV command can stop changing even if the GO signal is large, provided that the DV is zero.

Because all outflow signals from the DV stage are multiplied by the same GO signal, whose value grows smoothly in the course of the movement, the components of a synergy of contracting muscles tend to complete their motions synchronously, whatever the relative initial sizes of the components of the DV command. This temporal equifinality property even holds when the

different DV components that are multiplied by a given GO signal have different onset times (Bullock & Grossberg, 1988a,b), provided that the range of onset times is not too large a fraction (e.g., > 50%) of the movement time. This property promotes stable control of rapid sequences of movements.

Given this heuristic introduction to the model, it is possible to state a set of six hypotheses regarding correspondences between the computational stages of a more elaborate version of the model (Figure 1, upper part) and the six cell types described above. Mathematical specification of this extended circuit model recently allowed us to perform the simulations summarized in columns 2 and 4 of the lower part of Figure 1. These simulations allowed systematic comparison of the model's predicted activation profiles for 6 idealized neuron types with the activation profiles of the 6 known types. The hypotheses (Bullock, et al. 1998; Cisek et al. 1998) are:

1. An arm movement Difference Vector (DV) is computed in parietal area 5 from a comparison of a Target Position Vector (TPV) with a representation of current position called the Perceived Position Vector (PPV). The DV command may be activated, or primed, prior to its overt performance.
2. The PPV is also computed in area 5, where it is derived by subtracting a feedback of position error from an efference copy of an Outflow Position Vector (OPV) command arising in area 4. The position error signal used for this computation is generated by spindle receptors in skeletal muscles and, after ascending the spinal cord, is routed to area 5 via area 2.
3. The primed DV projects to a Desired Velocity Vector (DVV) stage in area 4. A voluntarily scaleable GO signal gates the DV input to the DVV in area 4. By virtue of the scaled gating signal, the phasic cell activity of the DVV serves as a volition-sensitive velocity command, which activates lower centers, including gamma-dynamic motoneurons of the spinal cord.
4. The DVV command is integrated by a tonic cell population in area 4, whose activity serves as an Outflow Position Vector (OPV) to lower centers, including alpha and gamma-static motoneurons. This area 4 tonic cell pool serves as source of the efference copy signal used in area 5 to compute the Perceived Position Vector (PPV). As the movement evolves, the difference vector (DV) activity in area 5 is driven toward baseline. This leads to termination of excitatory input to area 4 phasic cells, and thus to termination of the movement itself.
5. A reciprocal connection from the area 5 Perceived Position Vector (PPV) cells to the motor-cortical tonic cells (OPV) enables the area 4 position command to track any movement imposed by external forces. This reciprocal connection also helps to keep muscle spindles loaded and to avoid instabilities that would otherwise be associated with lags due to finite signal conduction rates and loads.

6. Phasic-tonic force-and-position-related (OFPV) cells in area 4 enable graded force recruitment to compensate for static and inertial loads, using inputs to area 4 from cerebellum and a center that integrates muscle spindle feedback. These area 4 phasic-tonic cortico-motoneuronal cells enable force of a desired amount to be exerted against an obstacle without interfering with accurate proprioception (PPV), and while preserving a target posture (TPV) should the obstacle give way.

In summary, the VITE model provides an integrative perspective on cortical control of voluntary movement by providing a unified account of the signatures of willful action: command priming, delay for deliberation, execution gating, variable-speed action, smooth bell-shaped velocity profiles, voluntary exertion of force against surfaces, and voluntary relaxation under load. At the same time, the assumed gating operation allows the model to be articulated with future models of decision making and task-dependent reconfiguration of the brain achieved in cooperation with subcortical circuits. Some reasons for attributing circuit gating functions to subcortical circuits are treated in the next section.

Cortical-subcortical cooperation for deliberation and task-dependent configuration.

In the past, much of neural network theory has analysed associative learning of sensory-motor transformations – mappings achieved by adaptively weighted pathways – that define one or another task-specific machine. Such analyses, by themselves, do not deal with cognitive and volitional aspects of intelligence. Eventually, animals capable of growing sensory-motor networks would have had many machines in one brain, and so would have needed to evolve mechanisms for switching intelligently among them. Therefore, neural network theory must also analyze adaptive switching and gating circuits, which enable on-the-fly reconfiguration in order to match sensory-motor flows to the present task. Such analyses add a more cognitive and volitional dimension to neural network theory, and directly raise issues of hierarchical organization in intelligence.

The idea that by learning we have grown many alternative pathways, a subset of which we can activate in order to make ourselves into the machine required for the current task, carries with it some interesting implications. One is that most of the resultant, potentially useful, pathways are normally closed, and that activating a particular pathway therefore amounts to opening a subset of normally-closed switches or "gates". This invites us to search the brain for neural systems that govern sensory-motor pathways, and whose normally inhibitory outputs show selective pausing (removal of inhibition) in association with current task demands.

In the popular imagination, the cerebral cortex is seen as the dominant site for learning, and this is probably true for the declarative, factual, learning that depends on the high level perceptual and conceptual analyses performed by cerebral circuits. However, on the basis of a wealth of interdisciplinary data, it is possible to propose, as a general hypothesis, that the brain also has two major adaptive extra-cerebral systems, each of which works with the cerebral cortex to reconfigure the brain for new tasks by rapidly switching among the different sensory-motor pathways that have been grown. One of these systems, the **basal ganglia complex** (a subcortical part of the telencephalon, or forebrain), is sensitive to information needed for intelligent voluntary decision making (e.g., Brown, Bullock & Grossberg, 1999; Schultz, 1998). This system both chooses among and regulates actions by opening normally closed gates that control activation of the frontal cortex of the brain (Bullock & Grossberg, 1991; Chereul et al. 1994; Hkosaka & Wurtz, 1989; Redgrave, Prescot & Gurney, 1999). Among such actions are sensory orientation (information gathering actions), working memory storage (information holding and purging actions) and manipulation (situation changing actions). The other system, the **cerebellar complex** (composed of the cerebellum proper and related pontine and olivary nuclei of the hindbrain), is sensitive to information needed for the successful realization of desired actions. It acts by opening normally closed gates that control sensory-motor transforms and motor command timing (e.g., Fiala et al. 1996; Kettner et al. 1997; Rhodes & Bullock, 2000; 2001).

The upper part of Figure 3 schematizes the two great subcortical systems just implicated in switch-like control of the current brain configuration and therefore of the current behavioral "set". In particular, the upper part of the figure shows that the flows from perceptual to motor regions of the cerebral cortex are influenced (1) by tonic inhibition from the **basal ganglia** to the motor thalamus, and (2) by tonic inhibition from the **cerebellar cortex** to the (interposed and lateral) deep cerebellar nuclei (DCN), which in turn excite the thalamus. This same scheme suggests that the manner of action of these two sub-cortical switching networks differs, because whereas the basal ganglia can directly inhibit cortico-thalamic loops, the cerebellum cannot. This anatomical and physiological asymmetry corresponds to a well established difference in the syndromes associated with damage to these systems. Damage to the basal ganglia complex can result in catastrophic cessation of all voluntarily initiated behavior. This occurs in advanced stages of Parkinson's disease, and can be seen as due to an inability to voluntarily open normally-closed gates that ultimately control activation of the muscles. By contrast, damage restricted to the cerebellum, which cannot directly inhibit cortico-thalamic loops, never causes a cessation of voluntary action. Instead, cerebellar damage typically leads to errors in the context-dependent details needed to correctly implement intended actions. Such

errors include errors of timing, direction, and amplitude of the motion components that constitute action.

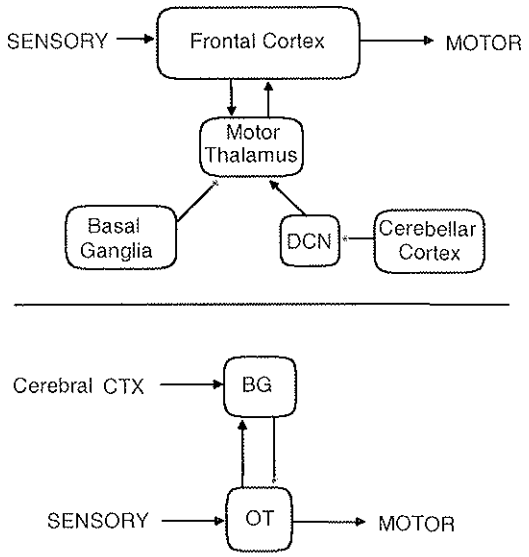


Figure 3. Adaptive switching circuits responsible for reconfiguring the sensory-motor system in order to match current task demands. Arrowheads denote excitatory links, ballheads inhibitory. **Upper part:** The basal ganglia and cerebellar cortex work by selectively pausing the tonic inhibition they normally exert on sites capable of activating sensory-motor pathways through frontal cortex. DCN = deep cerebellar nuclei. **Lower part:** The basal ganglia complex (BG) uses analyses provided by the cerebral cortex to decide which sites to disinhibit in the optic tectum (OT; homologous to the superior colliculus in mammals) which controls orienting (information gathering) actions in amphibians and terrestrial vertebrates.

Further evidence for the volitional gating role of the basal ganglia comes from consideration of the effects of lesions of another of its output pathways, that schematized in the lower part of Figure 1. Here the target of inhibition by the basal ganglia system (Hikosaka & Wurtz, 1989) is a brainstem structure, the optic tectum (OT, also known as the superior colliculus), which though still present in primates is the most important "higher" sensory-motor interface in more ancient vertebrates such as frogs. Lesions that remove basal ganglia inhibition of the OT result in a "volitionless" animal that reflexively responds to stimuli on the basis of their salience, without regard to the expected utility of the response (cf. Butler & Hodos, 1996; Ewert, 1997).

The contrasting connectivity of these subcortical elements, when combined with evidence from lesion studies, suggests that the basal ganglia are critical for "rational" choice among alternative actions and for graded voluntary activation of the chosen alternative, whereas the cerebellum is responsible for filling in the fine details that allow rapid, high performance

realization of action plans. This picture is reinforced by a consideration of the informational inputs to the two systems. In particular, the basal ganglia complex receives all the types of information needed to assist frontal cortex in high quality decisions to act (Passingham, 1987, 1993), including information about object identity, response options, and expected utility. By contrast, the cerebellum appears to receive no inputs from cerebral regions responsible for object recognition, nor any inputs regarding the utilities of behavioral alternatives. Instead, its inputs carry information about the evolving sensory-perceptual context of action, and about errors of realization of the desired motion.

In interdisciplinary terms, the basal ganglia system may be seen as a key part of a distributed, net utility computing, "organ of rational decision making" which corresponds in some respects to the bounded rationality models of microeconomics, whereas the cerebellum may be seen as an "organ of adaptive control" corresponding in some respects to the real-time control circuits of modern cybernetics and robotics. By contrast, the cerebral cortex emerges as an organ for analysis and synthesis of object-oriented representations of the scenario of action, for episodic or declarative memories, and for the flexible composition of conditional plans and desired trajectories.

Cortical layers, neural population codes, and posture-dependent recruitment of muscle synergies.

Any adequate theory of the cortical contribution to action composition must address data of several additional types. For example, the theory presented above proposes that the GO signal required to gate voluntary movement onsets arrives in the frontal cortex via a thalamic pathway that is dominated by the inhibitory output of the basal ganglia (Figures 1 and 3). This idea must be developed in the form of a model that incorporates data regarding the layers of frontal cortex that receive appropriate thalamic inputs. In a recent paper (Brown, Bullock, and Grossberg, 2000), we have developed such a laminar cortical model to explain how the basal ganglia complex gates learned eye movements in tasks where reward contingencies are used to train cued oculomotor actions. In this case, the modeled cortical data were drawn from studies of the frontal eye fields (FEF; part of area 8, rostral to area 4), but the concepts introduced are also applicable to learned control of arm movements.

Two additional phenomena that must be addressed by any theory of cortical control of arm movement are (1) the existence of populations of movement-related neurons of a given type, and (2) the fact that recruitment from such populations is posture-dependent. For example, for each node in Figure 1 (or Figure 2) interpreted as existing in area 4 (the primary motor cortex), there actually exists a corresponding population of cells. In the

middle 1980's, observations (e.g., Georgopoulos, et al. 1982) on such cells were interpreted by some experimentalists to imply that members of these populations were tuned to a distinct preferred direction in a Cartesian-like task space, and that their recruitment (activation) during a movement was solely a function of the match between their preferred direction in task space and the direction of the hand's motion in task space during the movement. However, several modelers (Bullock & Grossberg, 1988a,b; Mussa-Ivaldi, 1988; Sanger, 1994) pointed out that these cells could also be interpreted as having preferred directions in a space more closely related to muscle variables, e.g., joint angle space or muscle length space (or the associated velocity spaces). The model developed in Bullock, Grossberg & Guenther (1993; Guenther & Barreca, 1997) showed that direction-dependent tuning curves like those observed in motor cortex could arise as a result of a learning process within a circuit that incorporated the VITE model principles. This adaptive model was called DIRECT, because it explained how the brain could learn a DIrection to Rotation Effector Control Transform. This learned transformation is capable of recruiting the appropriate vector of joint rotations (or "movement synergy") for any desired direction of end-effector (hand or tool) motion. A key idea of the DIRECT model – inspired by the geometric properties of multi-joint limbs – is that the transform from directions in task space to directions in joint or muscle space is inherently posture dependent.

Thus it is of great interest to ask whether the directional preferences of motor cortical cells are posture-dependent. In fact, they are very strongly posture-dependent, as shown conclusively by Scott & Kalaska (1997) among others. Recently, we have completed two papers (Ajemian, Bullock & Grossberg, 2000a,b) which explore the predictive consequences of three alternative coordinate system assumptions that might plausibly be made regarding coding by motor cortical cells. These papers develop a general framework, based on vector fields, that can be used to study posture-dependent changes in the directional tunings and the gains (depth of modulation of the tuning curve) of movement-related cells. One conclusion is that *the strong evidence of both kinds of posture-dependent changes in most area 4 cells rules out the hypothesis that these motor cortical cells use Cartesian-like task-space coordinates* to encode movement variables. In contrast, the hypothesis that many area 4 cells are tuned to joint or muscle variables, as assumed in VITE and DIRECT, is consistent with the posture-dependent effects seen in the data.

Trajectory generation in handwriting and viapoint movements.

An iteration of two design principles of the VITE model -- multiplicative gating by a variable scalar, and integration of difference vectors -- led to a neural network for how to vary speed and/or amplitude of

the movements used to write a cursive letter without disrupting the letter's form (Bullock, Grossberg, and Mannes, 1993). Thus a simple extension of the model explains our ability to write the same letter forms at various rates and at a small scale (e.g., in one's agenda) or at a large scale (e.g., on a blackboard). By interpreting this model in terms of a circuit linking basal ganglia, thalamus and frontal cortex, my student J.L. Contreras-Vidal has been able to use it to explain micrographia, a common symptom in Parkinson's disease (Contreras-Vidal, Poluha, Teulings & Stelmach, 1995).

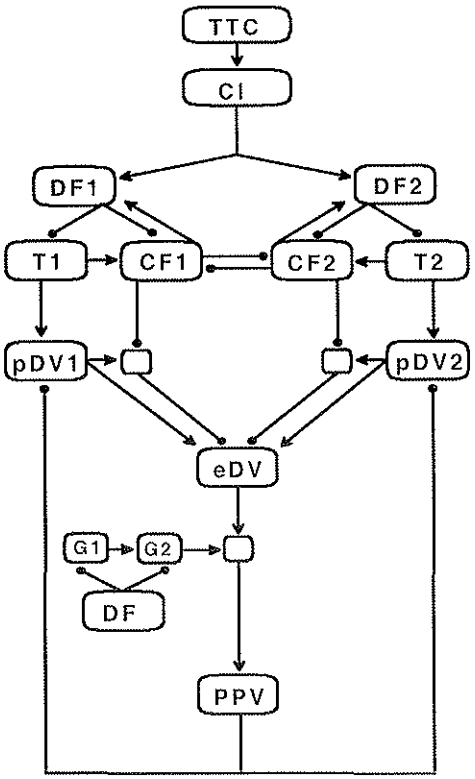


Figure 4. A neural network for serial release of primed movements by successive release of inhibition. Figure adapted from Bullock, Bongers, Lankhorst & Beek (1999). Arrowheads denote excitatory links, ballheads inhibitory.

A further iteration of the VITE model recently extended its explanatory scope to the performance of what experimental psychologists call "viapoint movements" (Bullock, Bongers, Lankhorst, and Beek, 1997). A viapoint movement is simply a continuous movement from point A to point C that is constrained by the experimenter's instruction to pass through an intermediate point B, which is not on the line segment A-C. Such movements exhibit two unique kinematic signatures. The first is an initial deviation of

the trajectory from the straight line linking A to B. This deviation of the initial part of the trajectory is always on the side of the A-B line that is opposite to the side on which C is located. The second kinematic signature is significant curvature in the movement's trajectory in the vicinity of the viapoint, B. These two properties show that viapoint movements do not arise as a mere concatenation of a movement A-B with a movement B-C. Past explanations (Flash & Hogan, 1985) of these kinematic signatures postulated that the entire trajectory is preplanned under a constraint that minimizes the integral of jerk (the time derivative of acceleration) over the whole trajectory. However, this explanation violates the VITE principle – and the apparent implication of neural data like those shown in Figure 3 – that the desired trajectory is being generated on-the-fly. Also, Nagasaki (1989; see also Zhang & Chaffin, 1999) has shown that the minimum jerk principle cannot explain simpler point to point movements. We therefore sought to explain viapoint movements with a VITE model that incorporated a stage that would first choose target B, and gate on the pathway that would generate movement toward it, and then, as contact with target B became imminent, rapidly suppress the choice of target B while choosing target C and gating on the pathway that would generate movement toward C.

The resultant network, shown in Figure 4, was implemented with a large populataion of direction-tuned cells at the eDV stage. Simulations showed the model able to reproduce the two distinctive kinematic signatures of viapoint movements. The rationale behind this architecture is readily stated in terms of a heuristic description of what we believe to be happening during a typical subject's performance of the viapoint task. We imagine that to begin, a subject simultaneously activates two distinct target representations, T_1 (corresponding to viapoint B) and T_2, (corresponding to final point C) in a working memory. For each of these the subject also computes distinct planning difference vectors, pDV_1 and pDV_2, between his current PPV and the represented targets. These pDVs project by separate excitatory and inhibitory pathways to the executive difference vector, or eDV, stage. The inhibitory pathways exist to prevent premature performance of planned DVs. However, to *guarantee* prevention of premature performance, the strength of the inhibitory projections must be stronger than the corresponding excitatory projections – a result that can be achieved by self organization through a local learning rule (e.g., Gaudiano & Grossberg, 1991). Thus, despite the excitatory inputs, there is initially no output from the eDV stage. A competitive choice field with distinct sites CF_1 and CF_2, respectively excited by T_1 and T_2, then chooses/schedules T_1 as the initial target. As a result, CF_1 gates off the inhibitory pathway from the pDV_1 stage to the eDV stage. This unmasks the excitatory inputs from pDV_1 to eDV, and an output is generated from eDV that reflects the evolving mix of the excitatory input from pDV_1 and both the excitatory and inhibitory inputs from

pDV_2. This mix evolves because upon activation of the GO signal, the PPV is updated by eDVxGO, and PPV updating leads to new pDV_1 and pDV_2 values.

During the movement, another, perceptual, network site is computing an estimate of the time to contact (TTC) of the hand with the viapoint, i.e., the perceivable point corresponding to T_1. When the TTC falls below a critical threshold, which indicates that contact is imminent (CI), a pulse is generated that causes rapid suppression of activities T_1 and CF_1. As CF_1 declines, CF_2 grows and quickly wins the competition, i.e., is chosen. Then CF_2 output suppresses the inhibitory projection from pDV_2 to eDV, and thereby unmasks the excitatory input from pDV_2 to eDV. Consequently, eDV output is now dominated by a mix including excitatory input from pDV_2 and any residual excitatory and inhibitory input from pDV_1. However, because T_1 has been suppressed, there is soon no activity at pDV_1. Thereafter, the eDV is wholly determined by excitatory input from pDV_2. As the hand approaches the target corresponding to T_2, TTC once again falls below threshold, and generates a pulse. This time, the pulse suppresses both T_2 and CF_2. In fact, the circuit's structure is such that the pulse generated by perception of imminent contact will always suppress the most recently activated T_j and associated CF_j, j = 1 or 2. Thus the TTC related pulse's action is non-specific, and has the correct effect without need for any target-specific learning.

The above description becomes complete when supplemented by a description of the dynamics of GO signal generation. For simplicity, we assumed that the GO signal is the output of a two cell, excitatory cascade, G_1 followed by G_2. This cascade is excited when the first winner is chosen in the choice field, and is re-excited by any subsequent winners. As a result, excitatory output from G_2 gates on the executive channel from eDV to PPV. When TTC reaches threshold as the hand nears the viapoint, the resultant inhibitory pulse, which suppresses T_1 and CF_1, also inhibits G_1 and G_2. This causes a partial reset of the GO signal pathway. However, its output begins to grow again as soon as the inhibitory pulse has ended. Thus activation and deactivation of the GO pathway is linked to activity in the choice field. At the end of movement, the TTC pulse again inhibits the GO pathway, which then remains inactive because it is no longer excited after all targets have been deleted from the choice field.

Working out the precise correspondence between all the cell types in this model and cell types in the brain is a topic for future work, although we know that suitable sites do exist in the basal ganglia and in thalamic and cortical regions strongly influenced by basal ganglia outputs, in agreement with the general scheme of Figure 4. But one might challenge the main assumption of the model, and ask why the system would be designed to compute the amplitudes of two or more movements simultaneously? Such simultaneous computations would be of adaptive value. For example, a predator capable of performing such computations could thereby make an informed choice to move toward the nearest of several visible prey. The

object of predation could likewise choose to move toward the nearest of several refuges, or to evade the nearer of two predators. However, computation of the dimensions of several forthcoming movements places an animal at risk of premature or even confused performance, because all plans active at the planning vector stage must be capable of exciting the executive vector stage. One way to prevent premature performance is to inhibit the executive vector stage in a way that is sure to cancel each excitatory input to the executive stage until a choice to go ahead with one of the prospective movements has been made. To guarantee cancellation, the inhibitory projections from the pDV to the eDV must be somewhat stronger than the excitatory projections. One consequence is that in the context of a serial plan, the inhibitory input from the second planned movement can cause a perturbation in the trajectory of the first planned movement. This is how we explain the empirically observed deviation in the initial segment of viapoint movements: it is a consequence of an inhibitory "force" required to block premature execution and thus to allow deliberative action by animals.

Satisfying constraints of reaching to intercept or grasp.

All the modeling studies discussed above have pertained to moving to targets in space. Many instances of reaching in humans are reaches to grasp or to intercept objects. In 1994, Peper, Boostma, Mestre & Bakker proposed that reaches to intercept moving objects could be guaranteed to succeed if the GO signal that gated and controlled the rate of zeroing of the difference vector (between hand and object positions) were divided by the object's time to contact with the plane in which interception was desired. The implied circuit continuously adjusts the hand velocity to the "required velocity" for interception. Peper et al. (1994) also showed that subjects behaved in accord with this model. This proposal has been augmented in the RR-VITE model proposed in a forthcoming paper by Dessing, Bullock, Peper & Beek (2000). The "RR-VITE" stands for relative and required velocity integration to endpoint, because in this model, VITE dynamics are adjusted on the basis of both the required velocity constraint identified by Peper et al. and by a relative velocity constraint that tends to ensure that the hand is not moving too fast relative to the object at the time of contact. This extension, which was motivated by psychophysical data, illustrates the relative ease with which VITE can accommodate additional constraints in a natural way.

Another constraint in reaching and grasping is the need to both open and close the hand within the time interval needed to move the hand into contact with the target. A prior model of this competence (Hoff & Arbib, 1993) proposed a series of estimation and differencing operations that yield pre-planned durations for the three component operations of opening, closing and moving the hand to target. However, in a recent paper (Ulloa & Bullock, 2000), we showed that effective coordination of the reaching phase with hand

opening and closing can be achieved dynamically, without any need to precompute durations of the component operations. This construction made use of a fundamental emergent property of a distributed, multi-channel VITE circuit. As noted in Bullock & Grossberg (1988a,b), and above, when multiple negative feedback controllers (such as the separate channels that compose all vector implementations of the VITE circuit) use a common multiplicative GO signal whose amplitude starts at zero and gradually grows larger through the movement interval, the controllers will all tend to reach their endpoints (or their components of a multi-dimensional endpoint) at about the same time. This is true even if some of the controllers don't begin their integration to endpoint until midway through the time used by which ever controller began first. In short, the circuit operates such that late-starting component processes catch up with early starting components. This can be called the synergy synchronization property or, somewhat more generally, the temporal equifinality property, of the VITE circuit. In the model of Ulloa & Bullock (2000), this property helps assure that the hand closure is temporally coincident with the end of the reach even though hand closure – which is variably delayed by the need to first open the hand – doesn't begin until the reach is at least half completed.

Conclusions: Online action composition by cortical circuits.

Neurophysiologists who have studied the activity of the cerebral cortex during movement production have been impressed by the absence of strict serial processing and what may be called late commitment (e.g., Kalaska, Scott, Cisek & Sergio, 1997). It appears not to be the case that entire trajectories are plotted before movements begin, and many aspects of the process of movement planning cannot be temporally separated from movement execution. Abundant evidence instead suggests that the desired kinematics of a movement are computed on-the-fly, in a way that maximizes the ability to respond to unexpected, late-arriving, information regarding such things as target locations, loads on limb segments, or the untoward effects of muscle fatigue. This is what could be expected from an examination of the kind of rich connectivity exemplified in the upper part of Figure 1. The VITE model has incorporated all the connectivity shown there, and further, consistent, extensions of the model have begun to provide the basis for a very wide ranging theory of the neural computations underlying human voluntary action.

Acknowledgements

I am grateful for the many contributions of my collaborators to the modeling studies noted in this chapter. Partial support for the chapter's preparation was provided by the Defense Advanced Research Projects Agency and Office of Naval Research (ONR N0014-95-1-0409).

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