A Model for the Functioning of the Striatum

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Abstract

A model is presented for the operation of the striatum. The model posits that the basal ganglia are responsible for driving smooth transitions of state for an organism. We propose that this is accomplished through the computation of a potential function within the striatum on which a gradient descent is performed toward the goal state. The model suggests that various somatotopic regions of the striatum correspond to state spaces, each of which pertains to a different aspect of the organism. This paper discusses this model only in the context of motor control, i.e., egomotion and limb movement. The model appears to account for a variety of experimental results, and for some unusual properties of the striatum.

1 Introduction

The basal ganglia have been one of the more enigmatic structures in the brain. While they have been extensively studied (DeLong and Georgopoulos 1981; Wilson, et al. 1990; Kandel, et al. 1991), and have been very clearly implicated in motor function (Yahr 1976; Alexander, et al. 1990), their exact function remains unclear.

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In this paper, a theory is put forth on the mechanisms of the planning of goal-oriented and obstacle-avoiding behavior in biological systems. It is argued here that the basal ganglia may be an important locus for aspects of these activities. Furthermore, the mechanisms for basal ganglia function may be characterized in part as the computation and storage of harmonic functions. These functions are computed in parameter spaces associated with the positions and movements of various body parts, and possibly additional representations of action and intention.

1.1 Goals and obstacles: a class of behaviors and control problems

The problem considered in this paper is that of planning the purposeful state changes of a mechanical system or organism. In other words, how does the system make its way from its current state to some goal state, avoiding undesirable, or even dangerous states? This is a general question which describes a variety of problems faced by different organisms and the designers of robot systems, e.g., in reaching and egomotion.

Research in cognitive psychology (see, for example, Neisser 1976) has suggested that, at least for certain tasks, the brain is capable of spontaneously generating trajectories for limb- and egomotion. Such path generation is not learned (Menzel 1973), but is dependent on the environment. What part of the brain, then, is computing this information, and how is it performing this computation?

1.2 Problems in Robotics

A related problem in robotics concerns how to build systems which are capable of deriving collision-free paths from one point to another. We would like to further motivate our model by considering what is known in robotics as the *path-planning* problem. There are many different types of robots, ranging from mobile robots which roam in a two-dimensional environment to complex robot arms with several degrees of freedom. For each of these robot types, one can describe robot position and motion in terms of the robot's *configuration space* (Lozano-Pérez 1981, Udupa 1977). Configuration space is the space of parameters which can be used to describe the pose of the robot. For a small mobile robot, these parameters are the x and y positions of the robot on the floor (with respect to some fixed coordinate frame). For a jointed robot arm, the parameters are the angles of each joint. Note that any conventional robot (e.g., fixed-base jointed robots) can be represented as a single point in its configuration space. Changing configuration is represented by the movement of a point in configuration

space.

Put Figure 1 near here.

Several approaches have been taken for solving the path planning problem in robotics (Schwartz and Sharir 1983; Khatib 1985; Arkin 1989; Canny and Lin 1990). Recent work in robot path planning (Connolly, et al. 1990; Connolly and Grupen 1992) revolves around the use of harmonic functions for complete robot path planning. Harmonic functions are solutions to Laplace's Equation:

$$\sum_{i=0}^{n} \frac{\partial^2 f}{\partial x_i^2} = 0 \tag{1}$$

which describes several physical phenomena including fluid flow and the distribution of electrical charge in a medium. In Equation 1, f is a harmonic function, n refers to the number of variables for the function, and each x_i is a function variable. This equation, then, says that all second derivatives of the harmonic function f sum to zero. Analog computers have been constructed to compute solutions to this equation, and consist of resistive grids (McCann and Wilts 1949). Since Kirchhoff's Laws[1] represent a discrete form of Laplace's Equation, such grids are able to compute discrete solutions to Laplace's Equation very rapidly as voltages. Recently, Tarassenko and Blake (Tarassenko and Blake 1991) employed a resistive grid to solve the path-planning problem for a mobile robot. The path-planning problem is primarily concerned with qualitative solutions. Hence, resistances within the grid need not be precise, nor does the grid need to be connected in a regular fashion. The former property is exploited by Stan and Burleson (Stan and Burleson 1992) in their path-planning grid.

Figure 1 shows an example of a two-joint robot, its configuration space, and a path generated by a harmonic function. Shaded areas represent obstacles, while solid black areas are goal regions. The left-hand side of the figure shows the robot in its environment. Several robot positions along the path have been superimposed so that the reader can see how the path progresses. On the right-hand side is the configuration space for the robot. The horizontal axis represents the first joint angle, while the vertical axis is the second joint angle. In this space, the robot is represented as a point. The shaded areas on the right-hand side are the result of mapping obstacles from Cartesian space into the robot's configuration space. Now the problem of moving the robot between two configurations (left side) has been reduced to determining a suitable path for a point (right side).

In the case shown in Figure 1, a harmonic function is computed over the free (white)

[1] The governing laws for resistances in a circuit: Kirchhoff's current law states that current flowing into a junction must equal the current flowing out of a junction.

regions of configuration space. One can compute a harmonic function on a digital computer simply by assigning high potentials to obstacles, low potentials to goals, and repeatedly averaging points in the interior. If every free point's potential is replaced with the average of its neighbors at each iteration (leaving goals and obstacles fixed at their initial potentials), then after several iterations, the values over the whole free area will converge to a harmonic function. Similarly, in a resistive network, the voltage at any free node after settling will be the average of the voltages at neighboring nodes. In a resistive network, obstacles may be represented either by fixed high potentials (resulting in repulsion) or by increased resistances (resulting in the absence of flow across obstacles). Mathematical literature refers to the former case as the Dirichlet boundary condition, while the latter is known as the Neumann boundary condition. In either case, the obstacle is avoided. For our model, it suffices to think of the majority of striatum cells as behaving in a manner similar to the resistive elements described here. Figures 2 and 3 show a sample problem for the Dirichlet and Neumann obstacle boundary conditions, respectively.

Put Figure 2 near here.

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Using a grid-based representation, the mapping from Cartesian to configuration space (e.g. joint angles) can easily be accomplished. Such mappings take into account the possible collisions of the effector with other obstacles and with itself. Once this mapping is obtained, it is permanent for a given effector, and can be used to set up obstacles and goals in state space, given information about their three-dimensional form in Cartesian space. Figure 4 illustrates the nature of the configuration space mapping. Grid points in Cartesian space are marked as either occupied or empty. Each point in state space corresponds to an arm state, which consists of several Cartesian space points. If any one of these Cartesian space points is occupied, the corresponding point in state space is also marked as occupied, otherwise it is marked empty. A harmonic function may then be computed over the free (unoccupied) points of the configuration space grid.

2 The Basal Ganglia

The basal ganglia constitute a large subcortical system that is densely connected to the cerebral cortex, thalamus and other brain centers. There is considerable evidence, including degenerative diseases which are largely restricted to the basal ganglia, that they are essential to motor control (Yahr 1976; Kandel, et al. 1991). Though their exact role is still not clear, there is growing neurological, anatomical and physiological support for the view that the basal ganglia may be involved in the planning aspects of behavior (Alexander, et al. 1992).

Put Figure 4 near here.

The basal ganglia seem to be organized into separate functional circuits; the two major circuits are involved in motor and cognitive functions, respectively. Though these circuits seem to operate in a separate, parallel fashion (Alexander and Crutcher 1990), they share much the same physiology and disease pathology, including their degeneration in Parkinson's disease (PD), which principally affects the basal ganglia.

An important pattern emerges in the cognitive deficits studied in PD patients (Taylor, et al. 1986; Brown and Marsden 1990). In a recent survey and analysis of these studies, Brown and Marsden conclude that the deficits are best characterized as a general dysfunction in planning; i.e., the ability to generate novel responses to novel situations and organize the correct sequence of actions. Across a wide variety of cognitive tasks, including visual-spatial and memory recall, it is the break-down of the planning aspect of the response that seems to best explain the behavior of the PD subjects. Taylor, et al. also report difficulties in forming cognitive strategies in subjects with Huntington's chorea, a degenerative disease restricted to the basal ganglia.

There is growing evidence that the motor circuit of the basal ganglia may also be involved in planning, at the motor level. Anatomically, it is connected to areas of the cortex associated with motor planning. An important input area to the basal ganglia motor circuit is the premotor cortex, which appears to be crucial for such functions as obstacle avoidance during reaching in monkeys (Moll and Kuypers 1977). A major output target of the circuit is the supplementary motor area, which appears important in programming motor sequences and coordinating bilateral movements (Kandel, et al. 1991). In general, the anatomical connections of the motor circuit of the basal ganglia indicate an emphasis on preparing motor actions rather than simply executing them (Kandel, et al. 1991).

The physiological evidence is also consistent with this interpretation. Experimental studies have yet to pinpoint the details of basal ganglia function during sensory-motor behavior; however, given recent cellular recording studies (Mink and Thach 1991a; Kimura 1990), it is unlikely that the basal ganglia are simply involved in the initiation of actions or their guidance through sensory feedback while movement is taking place. Mink and Thach recorded basal ganglia firing patterns during five different types of motor tasks, which included an open-loop visually guided task, as well as two classified as memory-guided and two which were described as closed-loop visual tracking. Given the cell activity, these researchers conclude that basal ganglia activity is strongest during the visually guided open-loop task. In this task, the monkey had to move to the correct location as indicated by a randomly positioned

visual cue. As described in (Mink and Thach 1991a), this task involves a "pre-programming" of the motor system based on sensory cues – rather than the use of these cues as feedback control during movement.

In general, the nature of basal ganglia input and output, and the cellular response timing during sensory-motor tasks seems appropriate for a planning role. This is reflected by the firing activity of basal ganglia cells studied by Kimura (Kimura 1990), and also by the work of Crutcher and DeLong (Crutcher and DeLong 1984a, 1984b). Two important classes of cells reported by Kimura were referred to as IIa and IIb. The IIa cell activity is correlated with external sensory input relevant to the trained task. It occurs during the sensory phase of the task: soon after the sensory stimuli and not during the actual movement of the monkey. The IIb cell activity is much more directly related to the movement of limbs. It appears too late to be signaling the initiation of the motor phase of the task, but could be involved in the ongoing control. Given the behavior of these two cell types, it is not unreasonable to assume that the basal ganglia could be generating limb control instructions based, in part, on sensory processing occurring much earlier than the motion – too early to be used strictly as sensory feedback during motion.

Finally, experiments studying motor behavior in subjects with PD also indicate an open-loop aspect to the motor control deficit associated with the disease. In Flowers (Flowers 1978a, 1978b), both normal controls and PD subjects were asked to actively track a moving visual target. The tracking trials varied in the quality of the information available to make predictions concerning the target motion: there were regular and irregular paths, and trials where the target disappeared for intervals. As expected, the tracking errors in the normal control group were much lower when the movement was regular than when it was irregular. This degree of improvement was not found in the PD group, in spite of the fact that individuals in this group recognized the regularity and could accurately describe it verbally. Flowers attributes this to an inability to perform open loop or predictive control. In this type of control, the subject is expected to generate a path based on advance knowledge, rather than simply follow the target in a closed-loop fashion. This interpretation is reinforced by experiments in which the target disappeared for periods of time. Subjects in the normal control group were quite competent at tracking in spite of the gaps in visibility. They seemed to be able to effectively utilize the information about the position and shape of the trajectory before the target disappeared to generate the appropriate motion in anticipation of its reappearance. The subjects in the PD group had little or no ability to do this.

2.1 Anatomy

The model is primarily concerned with the putamen and to a lesser extent, the caudate nucleus (collectively termed the neostriatum). This represents the part of the striatum involved in cortical and basal ganglia motor circuits. The intent of this paper is to provide a theory of the functioning of these nuclei in a motor context. It is beyond the scope of this paper to explore implications of the model for other functions of the striatum (e.g. possible involvement in speech, mood, and control of autonomic systems).

Corticostriatal projections arise throughout the cortex, arising mainly from cortex layer V. The putamen receives projections specifically from the primary motor cortex, the supplementary motor area, the premotor cortex, and the somatosensory cortex (Kandel, et al. 1991). These projections are glutamatergic, and are consistent with experiments showing the involvement of the putamen in motor function (Yahr 1976; DeLong and Georgopoulos 1981). Moreover, these projections are somatotopic, and divide the putamen into contiguous regions corresponding (in primates) to the arms, legs, face, and hands.

The striatal primary neurons project to the globus pallidus, and thence to the thalamus. The striatopallidal projection is convergent, (perhaps by a ratio of as much as 200:1; see Alexander, et al. 1992; Shepherd 1990) and inhibitory. Globus pallidus cells are themselves inhibitory and tonically active. Thus, they continually inhibit their target thalamic cells (DeLong and Georgopoulos 1981). There is also evidence that these thalamic cells "close the loop", in the sense that striatal neurons receive afferents from the same cortical areas which are their ultimate targets (via the globus pallidus and thalamus; see, for example, Holsapple, et al. 1991).

In summary, the putamen receives dense, somatotopic input from much of the sensory-motor cortex, and indirectly provides disinhibitory output to the thalamus. Specifically, it projects to those parts of the thalamus that project in turn to the sensory-motor cortex.

2.2 Cytology

The cells in the striatum have been divided into three groups (Shepherd 1990): giant aspiny, medium spiny, and medium aspiny. The giant aspiny neurons are generally regarded as being tonically active and cholinergic. These cells receive cortical afferents, and are interneurons, i.e., their effect is local. Kimura and colleagues (Kimura, et al. 1984) have shown that these cholinergic cells cease firing for a short interval immediately before the onset of voluntary

motion. The medium spiny cells are phasically active and GABAergic,[2] having efferents to the globus pallidus and the substantia nigra. These cells fire in relation to movement. Many studies have been performed on these cells, and several researchers have shown clearly that these neurons fire in direct relation to passive and active movement (Crutcher and DeLong 1984a, 1984b; Kimura 1990; Kimura, et al. 1990). Finally, the medium aspiny neurons are also GABAergic, but are interneurons. They fire in short bursts which have not been correlated with movement (Shepherd 1990).

Table 1 summarizes the characteristics of each of these cells and gives their population percentages (from Shepherd 1990). Though the percentages vary with species, the medium spiny cells generally make up an overwhelming majority. Given this and the synaptic connections of this cell type, Goldman-Rakic and Selemon (Goldman-Rakic and Selemon 1990) conclude:

> In order to discuss [basal ganglia] circuitry, the striatum may be approximated by a simple, 3D conglomeration of medium-sized spiny neurons, each receiving cortical input and sending it outward to the pallidum and [substantia nigra]; interneurons comprise such a small proportion of the neostriatal population that their numbers may be considered negligible.

Thus even though the interneurons are undoubtedly important for its normal functioning, accurate connectionist models of the striatum cannot involve cascades of interneurons or "hidden units".

Put Table 1 near here.

2.3 Neurotransmitters

The cortex provides input to the putamen and caudate nuclei via glutamate, an excitatory amino acid. Herrling, et al. and Heinonen et al. have shown that glutamate elevates membrane potentials in medium spiny cells (Herrling, et al. 1983; Heinonen, et al. 1990). Shimizu et al. and Krebs et al. provide evidence that glutamate is at least partly responsible for facilitating dopamine release (using microdialysis; Shimizu, et al. 1990; Krebs, et al. 1991; see also Roberts and Sharif 1978). The results of Leviel, et al. show that at low concentrations (10^{-8}M), glutamate can induce dopamine release, whereas higher concentrations (10^{-4}M) inhibit dopamine release (Leviel, et al. 1990; in vivo, under anesthesia). Pin and Bockaert also show that glutamate also induces Ca^{++}-independent GABA release (Pin and Bockaert

[2]Neurotransmitter abbreviations: GABA is γ-aminobutyric acid, ACh is acetylcholine, AChE is acetylcholinesterase, GLU is glutamate, and DA is dopamine.

1989; in culture). This release is apparently dendritic, rather than via the conventional action potential/exocytosis mechanism. Thus, glutamate appears to be performing at least three main functions:

- medium spiny cell depolarization
- DA release, resulting in medium spiny cell desensitization
- dendritic GABA release from medium spiny cells

Since the main glutamatergic pathway in the striatum is corticostriatal, these functions all appear to be under cortical control.

Dopamine is provided to the striatum by the substantia nigra. There is evidence for both D_1 and D_2 dopamine receptors in the striatum (Calabresi, et al. 1987). Calabresi et al. have also shown that D_2 receptors are primarily responsible for presynaptic inhibition of glutamate release. D_1 receptors are responsible for desensitizing medium spiny cells (in vitro). Bernath and Zigmond (Bernath and Zigmond 1990) show that dopamine may also influence GABA release. Williams and Millar (Williams and Millar 1990) show that dopamine at low concentrations (roughly 100nM) apparently induces cell firing, while higher DA concentrations (1-10μM) inhibited the cells. Herrling and Hull (Herrling and Hull 1980) report a DA-induced slow depolarization without an increase in firing rate. At high concentrations, DA does not seem to affect membrane potentials. It seems possible, then, that the main purpose of dopamine is to act as a thresholding mechanism.

Acetylcholine is produced by the giant aspiny cells. Glowinski (Glowinski 1990) reports that acetylcholine presynaptically regulates dopamine release in the striatum. The giant aspiny cells fire tonically, hence these cells must be responsible for providing a continual release of DA from nigrostriatal terminals.

The medium spiny cells project to the substantia nigra and the globus pallidus. These efferents are GABAergic. As mentioned previously, GABA is released both through exocytosis and by reversal of the dendritic GABA uptake system. Many studies have suggested an apparently contradictory role for GABA. Some studies report hyperpolarization (Lighthall, et al. 1981; Herrling, et al. 1984) while others report depolarization (Misgeld, et al. 1982). Nonetheless, the consensus appears to be that GABA is an inhibitory, hyperpolarizing neurotransmitter. $GABA_A$ receptors are associated with Cl^- channels, and the equilibrium potential for Cl^- is -75mV (Shepherd, 1990). GABA is probably acting in the striatum as a sort of voltage clamp somewhere near this potential.

2.4 Neuronal Response Characteristics

Researchers have uncovered some unusual characteristics of the firing patterns of neurons within the striatum. Relatively few cells within the striatum fire spontaneously. The giant aspiny cells appear to fire tonically, but at somewhat random intervals. The bulk of the cells are medium spiny cells, which rarely fire. When these cells do fire, at least in the putamen, it is related to voluntary movement. Kimura has shown that some of these cells will also fire in response to passive limb movement. Wilson (in Shepherd 1990) notes that the medium aspiny cells fire in bursts.

Crutcher and DeLong (Crutcher and DeLong 1984a, 1984b) and Kimura and his colleagues (Kimura, et al. 1984; Kimura 1990) have performed studies of the primate putamen which strongly indicate that medium spiny cells are sensitive to joint motion, often in a directionally-dependent manner. These cells usually correspond to joints rather than muscles. Crutcher and DeLong have found that neurons related to joints are grouped in clusters (100-500 μ) which are widely dispersed through the corresponding somatotopic region of the putamen. They further determined that many of these neurons were sensitive to the direction of joint movement, whether active or passive.

2.5 Electrotonic Coupling

Recent dye-coupling experiments (Cepeda, et al. 1989; Walsh, et al. 1989; Cepeda, et al. 1991; Walsh, et al. 1991; O'Donnell and Grace 1991; Onn, et al. 1991; Grace, et al. 1992) suggest the possibility that electrotonic coupling via gap junctions exists between striatal medium spiny cells. A survey by Pasik, et al. (Pasik, et al. 1990), also cites possible micrographic evidence for gap junctions between GABAergic cells in striatum. Gap junctions allow a cytoplasmic (and hence electrical) connection between cells. It is known that dopamine modulates electrotonic coupling in a variety of other brain structures (Grace and Bunney 1983). If electrotonic communication is indeed occurring among the medium spiny cells of the striatum, then these cells are effectively behaving as (nonlinear) resistors. Cell groups which do respond to dye-coupling are affected by dopamine agonists (Grace, et al. 1992) and thus intercellular coupling appears to be regulated by dopamine.

3 The Model

Given the apparent cytological, chemical, and electrophysiological features of the striatum, we wish to suggest the following model. We present the model first in terms of an operating principle, and then suggest possibilities for the physiological realization of this principle.

3.1 Harmonic Functions

The notion that certain aspects of biological systems can be described in terms of control surfaces has been proposed by Arbib (Arbib 1987). Similar schemes have been proposed in robotics for generating trajectories (see for example Arkin 1989). As discussed in Section 1.2, Laplace's equation provides a useful constraint on the potential function (control surface) to be used in generating trajectories. Laplace's equation describes a variety of natural phenomena. Such phenomena (e.g., resistive networks) can be used to compute harmonic functions very quickly. We propose that the resistive network has a biological analogue in the striatum.

In Section 1.2, the mapping from Cartesian space to configuration space was described. The striatum has several interesting properties that suggest that it may well be the repository for such a mapping:

- In primates, the putamen is divided into separate somatotopic regions corresponding to the limbs and face, each reflecting a separate motor circuit (Alexander 1990).
- Each region receives projections from corresponding somatotopic areas in the cortex.
- Substantial arborizations of the afferents from the cortex to the striatum have been observed (Shepherd 1990).
- Cell recordings in the putamen often correspond to specific joint movements.

We suggest that the somatotopic regions of the putamen could be direct representations of state spaces for motor control. The mapping of Cartesian to state space can very easily be accomplished via the corticostriatal arborizations, which could be implementing a many-to-one boolean "or" function of the Cartesian inputs.

If this is the case, then the striatum is in a prime position to be generating a control surface or potential function for guiding state changes. Such a model of neostriatal operation is consistent with recent objections to the "motor programming" concept (Alexander, et al. 1992). The output of the potential function scheme described here is simply a gradient or state-change signal, not a complete path. A planning system based on such a scheme would

be sufficiently plastic to adapt to obstacle and goal configurations that change with time. No reprogramming need occur, since the striatum simply responds to changing input by shifting the potential function into a new shape constrained by the new obstacle/goal configuration. Hence, the only effect on the output of the striatal system is a shift in the path gradient.

In summary, we propose that the striatum is divided into contiguous regions which are cellular representations of state spaces. These state spaces correspond to various aspects of organism function. Each region continually computes (deforms) a harmonic function based on cortical input consisting of obstacle and goal information. The word "obstacle" is used here in a broad sense as "a set of states to be avoided". In the putamen, the somatotopic regions are proposed to be joint spaces, and the output (via the globus pallidus) is proposed to be an encoding of a trajectory vector, defining the next state change for the motor system.

3.2 Biological Realization

Medium spiny neurons may form an electrotonically-coupled resistive network which is programmable via cortical input. Cortical input sets up the potential function by directly depolarizing certain medium spiny cells using glutamate EPSPs, and indirectly clamping other medium spiny cells via GABA. The remainder of medium spiny cells receive no membrane potential regulation except through electrical coupling. The difference in potentials thus produced is propagated electrically among these unregulated striatal neurons, whose membrane potentials are allowed to float.

We propose that electrotonic propagation may be responsible for computing a potential function. The membrane potential of each medium spiny cell corresponds to the function value. Higher potentials represent repelling states, while low potentials indicate goal states (or vice-versa). The spines on medium spiny neurons can act as a low-pass filter (Wilson 1984; Shepherd 1990). This greatly diminishes the effect of an action potential on membrane potentials at the dendrites. Such a filtering effect would tend to protect the integrity of the potential function being computed. Local, high-frequency, temporary changes to a cell's potential would not affect the overall potential function. Consistent with observed behavior, such a system would result in very few neurons actually firing at any given time, since the vast majority of neurons would be computing the harmonic function passively. Electrotonic coupling provided by gap junctions would further enhance the low-pass filtering effect, since gap junctions and cell membranes form an RC circuit.

Once computed, the function can be used to drive state changes within the organism.

Research suggests (Crutcher 1984b; Kimura 1990) that medium spiny cells fire both to induce joint torque (and hence motion), and in response to joint torques (e.g., passive movement). We propose that at any one time during the execution of movement, only a small cluster[3] of medium spiny cells will fire within the striatum. One can make an analogy of this cluster to a ball rolling down the potential surface. The cluster of firing cells changes as movement progresses, but tends to migrate in a continuous fashion toward those cells which represent the organism's goal state (as expressed by the GABA-clamped cells in the network). Once the organism has reached the desired state, the process is shut off.

As clusters of medium spiny cells fire, they inhibit globus pallidus cells, which in turn disinhibit certain thalamic cells. This process induces motion of appropriate joints. Experiments show (Crutcher and DeLong 1984b; Kimura 1984) that such movements can cause certain other medium spiny cells to fire. Most of these cells respond to movement, many to movement only in a particular direction, and some respond to varying load (i.e. joint torque). In addition, it is known that glutamate can presynaptically induce dopamine release from nigrostriatal afferents (Leviel, et al. 1990; Krebs, et al. 1991).

We suggest that the aforementioned characteristics may in fact indicate a feedback loop which sustains and moves the current-state cluster. We propose that the same cortical afferents which cause striatal directional cells to fire also presynaptically facilitate dopamine release at the opposite end of a current-state cluster, with respect to the directional cell (see figure 5). This serves to quench the *old* current-state cluster. It also seems plausible that cortical input indirectly inhibits dopamine release in the area of the new current-state cluster. This in turn presents a plausible mechanism for the "ball-rolling" effect. We wish to emphasize that the following discussion is quite speculative, and is intended mainly as an example of how the harmonic function computed by the striatum could be utilized. It is also possible, for example, that thalamostriatal axons (Shepherd 1990) contribute to this feedback loop.

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Migration of the current-state cluster is accomplished when a joint is moved in a particular direction. This causes the cortex to stimulate those striatal cells which are torque-sensitive. It is possible that such cells are offset from the cluster in a particular way, so as to bias the migration of the cluster. This leads to a sort of chain reaction, wherein a firing cluster leads to joint motion, causing certain other striatal cells to fire, and this process in turn quenches the old cluster, perhaps through cortically-evoked dopamine secretion. This process is repeated until the cluster (and hence the joints) reaches a goal state. It should be noted that some

[3] We denote this as the "current-state cluster".

researchers have observed the network-like arrangement of groups of cells in the striatum (Walsh, et al. 1989; Walsh, et al. 1991; Paskevich, et al. 1991).

The giant aspiny cells appear to have a damping effect. Their tonic activity tends to prevent spontaneous firing of medium spiny neurons. Glowinski (Glowinski 1990) suggests that these cells can presynaptically regulate dopamine release. Giant aspiny cells appear to cease firing near the onset of movement (Shepherd 1990), further suggesting that they act as dampers to prevent inappropriate striatal activity.

4 Consequences of the Model

Given the specific model proposed in the previous section, some general predictions can be made of the behavior of the basal ganglia in normal and diseased conditions.

In normal, healthy animals, the following experiments may be useful to determine the soundness of the model:

(1) Depolarize a cell in the putamen and record the level of polarization in nearby cells. If the modulated neuron is a medium spiny cell, there should tend to be a depolarization in the nearby medium spiny cells, correlated with this artificial depolarization. For the theory to be correct, the recorded neurons need not be immediate neighbors, just medium spiny cells in the same functional section of the putamen.

(2) Depolarize or hyperpolarize a medium spiny cell in the putamen and observe the effect on limb movements. A sustained and substantial change in the potential of a cell should distort the potential surface in the extended neighborhood of the cell. The model predicts that this would move a limb associated with the affected section consistently away from or towards some region in joint space and hence a corresponding arm configuration in Cartesian space. It is important to note that our model as it stands does not predict *specific* mappings between brain regions and space regions, just that some such mapping exists.

In the diseases associated with the basal ganglia, there are some known relationships between the pathology and the symptoms that can be predicted. Huntington's chorea is characterized by the progressive death of populations of medium spiny cells (Barbeau, et al. 1972). Given the model, this means that the potential surfaces – and thus motor control surfaces – are being represented by fewer and fewer cells. The coarser, cruder representation of the movement space should lead to coarser, jerkier movements in the subjects, which may explain the wild errors in intentional movement actually found in Huntington's disease. The cell deaths would also produce holes in the network of cells computing the surface. This

would have the effect of producing permanent, non-existent "obstacles" that the subject would appear to be "avoiding". This suggests that the irregularities in the movement of the patients may be repeatable; i.e., the same type of movement should happen if the arm approaches the same point in joint space from the same direction and speed. We do not know if this has been observed, although Bruyn (Bruyn 1972) notes a "conspicuous stereotypy" in the movements of Huntington's disease victims.

In Parkinson's disease, the level of dopamine in the basal ganglia is progressively reduced. Unfortunately, this transmitter has many effects and interactions in the basal ganglia, as well as in the model, which makes it difficult to form predictions. One prediction is that if the level is reduced somewhat, but not completely, the background firing rate of the medium spiny neuron should go up (as seen, for example, in Williams and Millar 1990). This may explain the tremors associated with the disease. When the level of dopamine drops even lower, the firing rate of the medium spiny neurons should decrease again, and finally stop firing altogether. This may explain the severe *akinesia* present in patients with advanced Parkinsonism.

Another effect of dopamine predicted by the model is to alter the resistance in gap junctions and hence the resistance in the network throughout a region of the putamen. With pathologically reduced levels of dopamine, the resistance may go down and the current (ion flow) between primary neurons would increase. The potentials are being maintained by a fixed number of ion pumps, and at some current flow, these membrane mechanisms may not be sufficient to maintain the potentials. This would have the effect of producing a flat potential or control surface, which may lead to the *akinesia* observed in patients. In addition, the loss of dopamine may make it difficult to represent obstacles in the putamen, and thus the resulting potential sufaces would not prevent the subject from hitting them. We do not know whether this has been observed in subjects with PD.

5 Summary

We have described very roughly a model for the low-level functioning of the striatum within the basal ganglia. We have also suggested certain roles for the globus pallidus and the substantia nigra. In short, the model proposes that the basal ganglia are responsible for generating motor or state-change programs which drive the organism in an organized fashion from its current state to some desired state, while avoiding undesirable states. In contrast to models which suggest selection of a motor program, we propose that the basal ganglia

actually create the program by performing a gradient descent on a potential function, thereby generating a trajectory which describes the successive state changes needed to reach the desired state. The potential function is produced by a relaxation process on the membrane potentials within cells in the striatum. In the specific biological realization proposed here, this relaxation process results from two factors: a) cortically "programmed" high and low membrane potentials at different regions within the striatum and b) electrical coupling of the cells via gap junctions.

Given that the structure of the caudate nucleus is similar to the putamen, it seems plausible to apply the same model to the caudate. Here, the states to be maintained appear to be related to egomotion, speech, mood, and even certain autonomic systems. It has already been suggested (Potegal 1972) that the caudate nucleus is involved in localization and egomotion, and the harmonic function approach very easily applies to mobile robots, for which egomotion is the only path-planning problem involved. Certainly, then, the model applies directly to some aspects of the operation of the caudate nucleus. Indeed, one can think of the scheme described here as a constrained interpolation, where the interpolant is a harmonic function. Its purpose then is not necessarily limited to the determination of state changes, and may involve "filling in" missing information. Further study is required before examining this model in the context of the more esoteric functions of the caudate nucleus.

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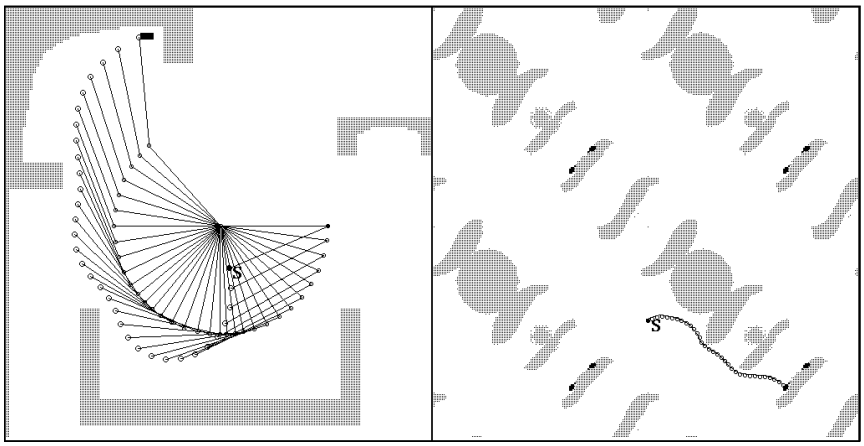

Figure 1: An example of a simulated two-jointed robot (left) and its configuration space (right). The path shown has been generated using a harmonic function computed over the configuration space.

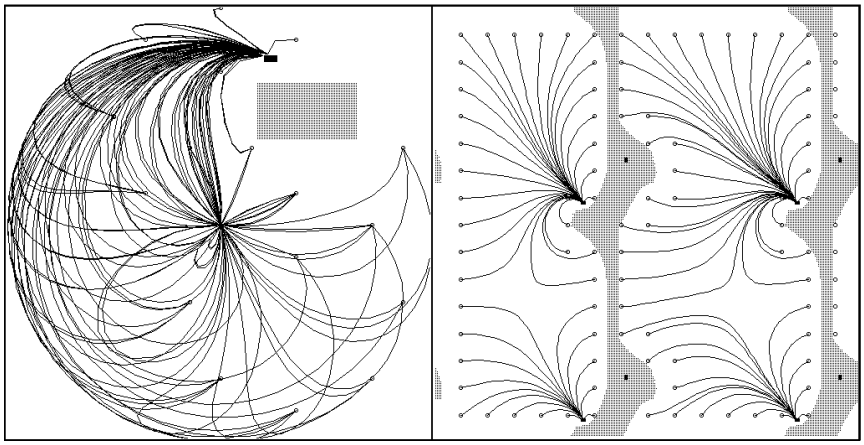

Figure 2: A harmonic function computed using Dirichlet boundary conditions (i.e., obstacles held at a constant high potential). The obstacle potentials are 1.0. The goal potentials are 0.0.

type	*firing*	*neurotransmitter*	*afferent*	*efferent*	*population*
medium spiny	phasic	GABA	cortical	GP and SN	77-95%
medium aspiny	burst	GABA	cortical	striatal	3-25%
giant	tonic	ACh	cortical	striatal	1-2%

Table 1: Types and characteristics of striatal neurons. Note that population percentages are species-dependent.

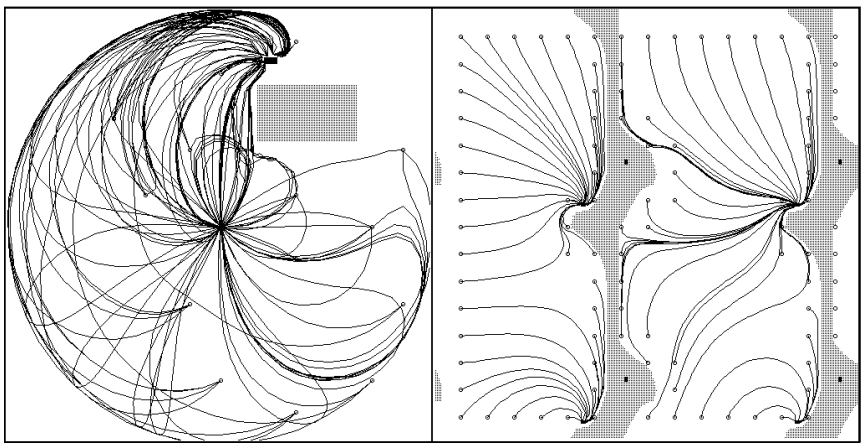

Figure 3: A harmonic function computed using Neumann boundary conditions, where the function gradient is constrained to be parallel to obstacle surfaces.

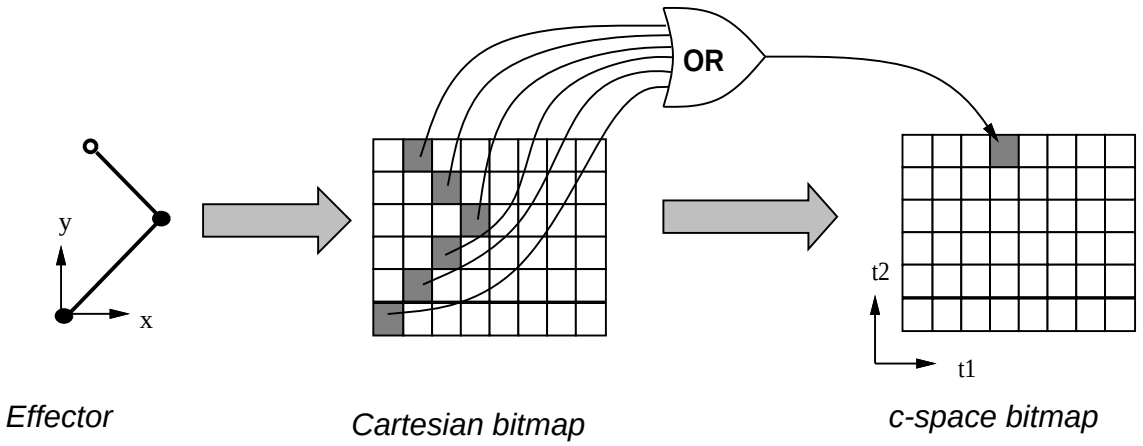

Figure 4: Schematic diagram of the mapping from Cartesian world coordinates into configuration space.

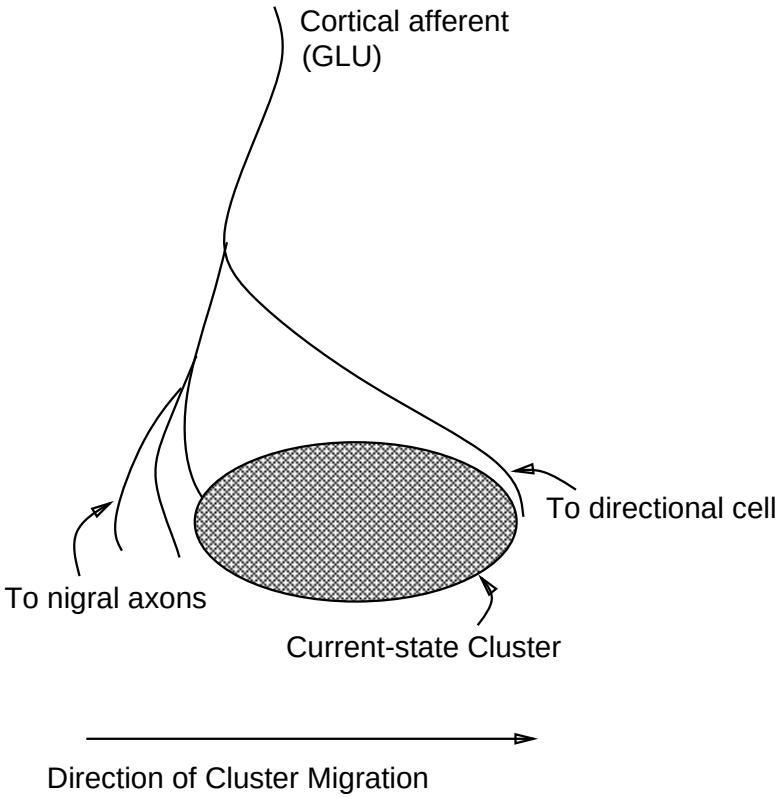

Figure 5: Schematic of the migration of a current-state cluster within the striatum. Directionally-dependent release of dopamine results in cluster movement.

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