

**The Basal Ganglia and the Planning of Actions**

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# The Basal Ganglia and the Planning of Actions

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## Abstract

A model is presented for the low-level operation of the basal ganglia in the brain. The model posits that the basal ganglia are responsible for driving smooth transitions of state (e.g., joint positions) for an organism. This is accomplished through the computation of a potential function on which a gradient descent is performed to the desired state. The model proposes that various contiguous parts of the neostriatum correspond to state spaces, each of which pertains to a different aspect of the organism. The model also proposes that the neostriatum computes a potential function within each of these state spaces via electrotonic coupling, and that the local shape of this function at a given point (i.e., state) is used to drive the organism to a new state via inhibition of the globus pallidus. 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. Some implications of the model for Parkinson's and Huntington's diseases are also discussed.

## 1 Introduction

In this note, 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 its function may in part be characterized as the computation and storage of harmonic functions 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 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? This is a general question which describes a variety of problems faced by different organisms and even designers of robot systems. For example, searching for food is one of the most basic tasks facing any organism. How does the primate find its way to a food source? Any task involving motion falls into the category discussed here. How are humans able to so effectively reach for and pick up objects, even in the presence of obstacles?

Research in cognitive psychology (see, for example, Neisser [1]) has suggested that, at least for certain types of tasks, the brain is capable of spontaneously generating trajectories for limb- and egomotion. Such path generation is not learned [2], but is dependent on the environment. What part of the brain, then, is computing this information, and how is it performing that computation? From ablation and stimulation studies [3], and studies of basal ganglia disease [4], it is well known that the basal ganglia play an important role in determining such trajectories.

## 1.2 Problems in Robotics

A related question in the field of Robotics concerns how to build a robot system which is capable of computing a collision-free path 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. This is the robotic analogue for the type of path generation problem described above for organisms. 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*. Configuration space is simply a space of parameters which can be used to describe the pose of the robot. For a small mobile robot, these parameters are simply 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 robot can be represented as a single point in its configuration space. Changing configuration is represented by the movement of a point in configuration space. The robot path-planning problem, then, can be phrased in terms of this question: How is the robot to proceed from its current point in configuration space to some goal point in configuration space? Figure 1 displays the basic idea. The robot, represented as a point, must be made to go from its current state to some goal state without stopping or hitting any obstacles.

Several approaches have been taken for solving the path planning problem in Robotics [5, 6, 7, 8, 9, 10, 11]. Many techniques are heuristic and incomplete. There is, however, great interest in devising complete path-planning algorithms, i.e., those which are guaranteed to work if a path exists,

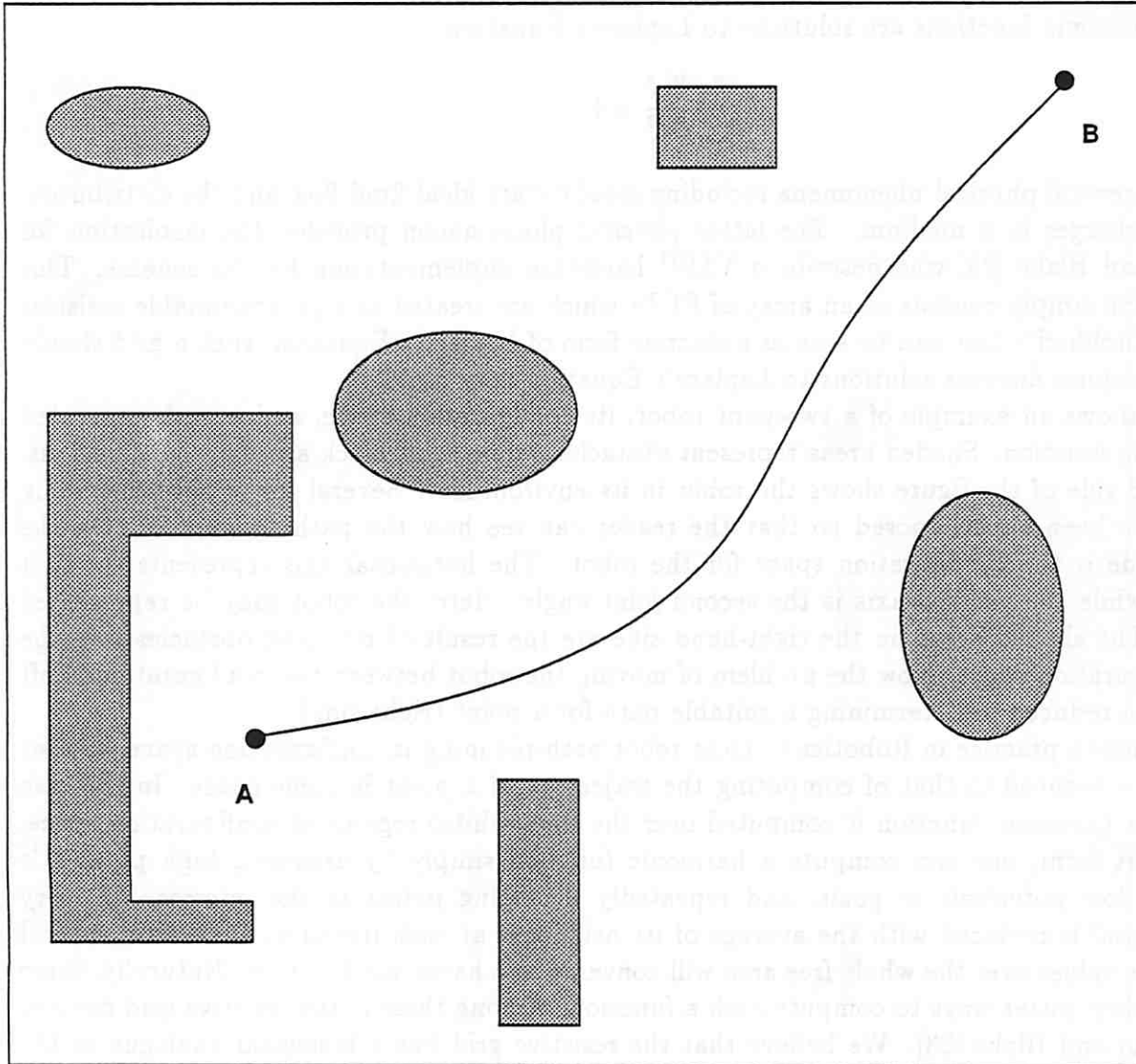


Figure 1: A schematic showing the path-planning problem. How is the robot to proceed from its original state (point A) to the desired goal state (point B)?

and which are able to detect cases where a path does not exist [12, 13, 14, 15, 16, 17, 18, 19, 20, 21]. Recent work in robot path planning by Connolly, et al. [22], Tarassenko and Blake [23], and Connolly and Grupen [24] revolves around the use of harmonic functions for complete robot path planning. Harmonic functions are solutions to Laplace's Equation,

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

and describe several physical phenomena including steady-state ideal fluid flow and the distribution of electrical charges in a medium. The latter physical phenomenon provided the inspiration for Tarassenko and Blake [23] who describe a VLSI<sup>1</sup> hardware implementation for the scheme. This implementation simply consists of an array of FETs which are treated as a programmable resistive grid. Since Kirchhoff's Law can be seen as a discrete form of Laplace's Equation, such a grid should be able to compute discrete solutions to Laplace's Equation very rapidly.

Figure 2 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. Here, the robot may be represented as a point. The shaded areas on the right-hand side are the result of mapping obstacles 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).

It is a common practice in Robotics to treat robot path-planning in configuration space, so that the problem is reduced to that of computing the trajectory of a point in some space. In the case shown here, a harmonic function is computed over the free (white) regions of configuration space. In its simplest form, one can compute a harmonic function simply by assigning high potentials to obstacles, low potentials to goals, and repeatedly averaging points in the interior. If every point's potential is replaced with the average of its neighbors at each iteration, then after several iterations, the values over the whole free area will converge to a harmonic function. Naturally, there are several other, faster ways to compute such a function. Among these is the resistive grid devised by Tarassenko and Blake [23]. We believe that the resistive grid has a biological analogue in the basal ganglia.

To see how the resistive grid might be used, consider that two possibilities exist for designating obstacles: 1) obstacles may be held at a fixed high potential, so that the function always slopes away from such points, or 2) obstacles may be rendered impenetrable, so that the function gradient

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<sup>1</sup>VLSI: Very Large Scale Integration. FET: Field Effect Transistor.

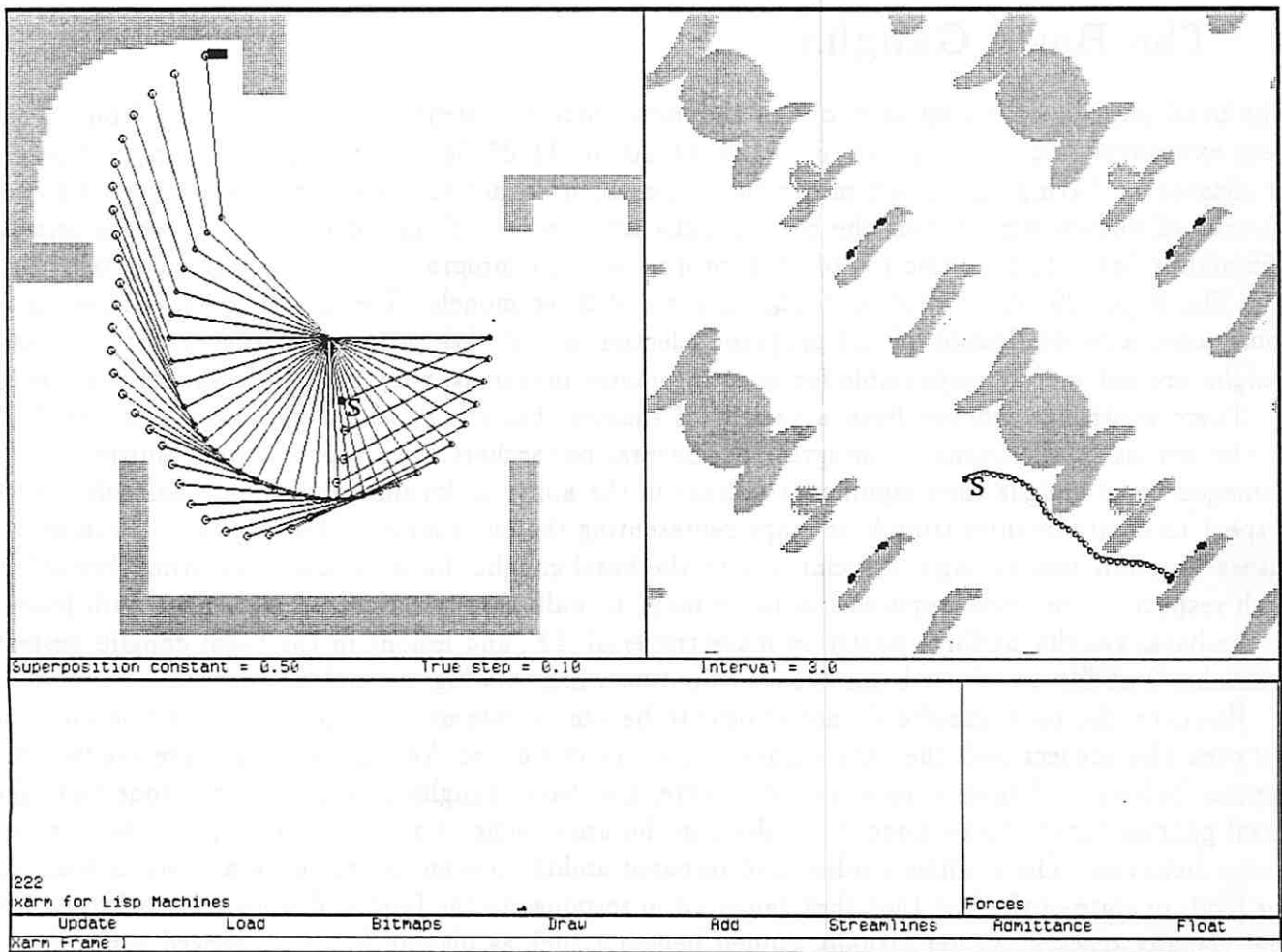


Figure 2: An example of a simulated two-jointed robot (left) and its configuration space (right). The path shown has been generated using a harmonic function computed over the configuration space.

runs parallel to these points. All goal points are held at a fixed low potential. For the resistive grid, case 1 implies raising the potentials of obstacle points to some predetermined high value, while case 2 raises the resistances between such obstacle points so that no current flows through them. Case 1 results in an obstacle which the point must actively avoid (i.e. be repelled from). Case 2 results in a flow around the obstacle, but no repulsive force exists. In either case, the obstacle is avoided. For our model, it suffices to think of the majority of neostriatum cells as behaving in a manner similar to the resistive elements described here.

## 2 The Basal Ganglia

The basal ganglia have long been one of the more enigmatic structures in the brain. While it has been extensively studied [4, 25, 26, 3, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36], and has been very clearly implicated as having important motor functions [4], its exact function remains unclear. Current theories of its role suggest that the basal ganglia act as a sort of “motor jukebox”, wherein cortical stimulation leads to the selection of an appropriate motor program [37]. Recently, however, Mink and Thach [38, 39, 40] have shed doubt on some of these models. The model presented here is in some sense a generalization of the program selection model. We propose, however, that the basal ganglia are not merely responsible for selecting motor programs; they actually *create* the program.

There is strong evidence from a variety of sources that the basal ganglia are deeply involved in the details of sensorimotor integration. Several researchers have reported that subjects with damaged basal ganglia have significant deficits in the ability to localize and orient themselves with respect to environmental stimuli or maps representing the environment. Patients with Parkinson’s disease, an affliction strongly associated with the basal ganglia, have difficulty orienting themselves with respect to the scene vertical and using maps to walk through a course [41]. Rats with lesions in the basal ganglia perform poorly in maze traversal [42], and lesions in the basal ganglia destroy a monkey’s ability to adjust hand-eye coordination when viewing through prisms [43].

However, the basal ganglia do not appear to be simply representing the geometric relationships between the subject and the environment. Denny-Brown and Yanagisawa [44] have studied the general behavior of several monkeys with extensive basal ganglia lesions and conclude that the basal ganglia appear to be directly involved in the use of sensory information to generate complex motor behavior. The animals studied had impaired ability to even initiate such actions as reaching for food, in spite of the fact that they salivated in response to the food and were able to fixate on it and visually track it. Other visually guided behavior such as placing limbs on viewed surfaces and avoiding walls while walking are destroyed or severely affected by lesions of the basal ganglia.

Experimental evidence has yet to pinpoint the details of basal ganglia function during sensorimotor behavior; however, given recent cellular recording studies [38, 35, 45], 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 [38] 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 termed 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 [38], 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 sensorimotor tasks seems appropriate for a planning role, as well as other aspects of motor control. This is reflected by the firing activity of basal ganglia cells studied by Kimura [35, 45], and also by the work of Crutcher and DeLong [27, 28]. 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 signalling 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.

## 2.1 Anatomy

The basal ganglia are comprised of several subparts. We will be primarily concerned with the putamen and to a lesser extent, the caudate nucleus (collectively termed the striatum). The intent of this paper is to provide a theory of the functioning of these nuclei. It is beyond the scope of this paper to suggest implications of the model for other functions of the striatum (e.g. possible involvement in speech, mood, and control of autonomic systems). We wish only to suggest a model for basal ganglia involvement in motor planning.

Corticostriate projections arise throughout the cortex, emanating mainly from lamina V. The putamen receives projections specifically from the primary motor cortex, the supplementary motor area, the premotor cortex, and the somatosensory cortex [46]. These projections are glutamatergic, and are consistent with experiments showing the involvement of the putamen in motor function [3, 4]. Moreover, these projections are somatotopic, and divide the putamen into contiguous regions corresponding (in primates) to the arms, legs, face, and hands. Most researchers divide the putamen and caudate into striosome and matrix compartments. We also note here that PET scans using



<i>type</i>	<i>firing</i>	<i>neurotransmitter</i>	<i>afferent</i>	<i>efferent</i>
Giant	tonic	ACh	cortical	striatal
medium aspiny	burst	GABA	cortical	striatal
medium spiny	phasic	GABA	cortical	GP and SN

Table 1: Types and characteristics of striatal neurons.

labelled glucose indicate that the striatum is among the most metabolically active regions in the brain [46].

## 2.2 Cytology

The cells in the neostriatum have been divided into three groups [37]: 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 [29] has 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,<sup>2</sup> 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 [27, 28, 35, 45]. Finally, the medium aspiny neurons are also GABAergic, but are interneurons. They fire in short bursts which have not been correlated with movement [37]. Table 1 shows the characteristics of each of these cells.

## 2.3 Neurotransmitters

The cortex provides input to the putamen and caudate nuclei via glutamate, an excitatory amino acid. Herrling, et al. [47] and Heinonen et al. [48] have shown that glutamate elevates membrane potentials in medium spiny cells. Shimizu et al. [49] and Krebs et al. [50] provide evidence that glutamate is at least partly responsible for facilitating dopamine release (microdialysis).<sup>3</sup> The results of Leviel, et al. [51] show that at low concentrations ( $10^{-8}$ M), glutamate can induce dopamine release, whereas higher concentrations ( $10^{-4}$ M) inhibit dopamine release (in vivo, under anesthesia). Pin and Bockaert [52] also show that glutamate also induces  $Ca^{++}$ -independent GABA release

<sup>2</sup>Neurotransmitter abbreviations: GABA is  $\gamma$ -aminobutyric acid, ACh is acetylcholine, AChE is acetylcholinesterase, GLU is glutamate, and DA is dopamine.

<sup>3</sup>See also Giorgiueff in *Neuroscience Letters* 6:73-77; 1977, and Roberts in *Brain Res.* 157:391-395; 1978

(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 are all 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 [53]. 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 [54] show that dopamine may influence GABA release. Williams and Millar [55] show that dopamine at low concentrations (roughly 100nM) apparently induces cell firing, while higher DA concentrations (1-10 $\mu$ M) inhibited the cells. Herrling and Hull [56] 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 [57] 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 [58, 59] while others report depolarization [60, 61]. The consensus appears to be that the change in cell polarization due to GABA is relative to the pre-existing membrane potential. If the cell is held at potentials in the range of -70 mV, GABA induces a depolarization. At ranges closer to 0mV, GABA induces a hyperpolarization. Mercuri et al. report that the null potential for GABA is at about -50mV. Herrling [59] supports the notion that GABA appears to be driving the cell toward the -50mV level, even when glutamate is applied to depolarize the cell. GABA<sup>-</sup> could be acting as a sort of voltage clamp to the -50mV potential.

## 2.4 Firing patterns

During the past two decades, researchers have uncovered some unusual characteristics of the firing patterns of neurons within the neostriatum. Relatively few cells within the neostriatum fire spontaneously. The giant aspiny cells appear to fire tonically, but at somewhat random intervals. The bulk of the cells (70-90%, depending on the species) 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. In Shepherd [37], Wilson notes that the medium aspiny cells fire in bursts.

## 2.5 Electrotonic Coupling

Recent dye-coupling experiments [32, 62, 63, 33] indicate the possibility that electrotonic coupling via gap junctions exists between medium spiny cells. Gap junctions allow a cytoplasmic (and hence electrical) connection between cells. It is known that dopamine modulates electrotonic coupling within the visual system and cardiac neurons [64], and the dye-coupling experiments of Cepeda, et al. suggest that this is also true in the striatum. These studies were performed on the basal ganglia of rats and cats. If indeed electrotonic communication is occurring among the medium spiny cells of the neostriatum, then these cells are effectively behaving as (nonlinear) resistors. Gap junctions which do respond to dye-coupling appear to be regulated by dopamine. Kawaguchi, et al. [65] note that biocytin-stained medium spiny cells exhibited coupling in their experiments. They propose the possibility that this is due to electrotonic coupling, since biocytin is a relatively small molecule. This appears to be further evidence of electrotonic coupling. To date, the authors are not aware of any studies showing the presence of mRNA for connexins (the gap junction proteins) in medium spiny cells.

## 2.6 Spatial Organization

Crutcher and DeLong [27, 28] and Kimura and his colleagues [29, 35] 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 [27] have found that neurons related to joints are grouped in clusters (100-500  $\mu$ ) which are widely dispersed through the corresponding somatotopic region of the putamen. They further determined [28] that many of these neurons were sensitive to the direction of joint movement, whether active or passive.

### 3 The Model

Given the apparent cytological, chemical, and electrophysiological features of the neostriatum, we wish to suggest the following model. It should be noted that the model has two aspects: The first is the actual computation of the harmonic function within the striatum, and the second is the read-out mechanism that drives cortical state changes. The latter aspect is much more speculative than the former.

Medium spiny neurons form an electrotonically-coupled resistive network which is programmable via cortical input. It is well established (Liles, '79, [3, 27]) that somatotopic projections exist from the cortex into contiguous, mostly non-overlapping regions within the putamen. This suggests that the putamen can be divided into functional units corresponding to the limbs and face. We propose that each of these units is map of the state space of the corresponding limb (or face). In the case of the limbs, this is merely a joint space. 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 dendritically-released GABA. The remainder of medium spiny cells receive no membrane potential regulation except via gap junctions. The difference in potentials thus produced is propagated electrically among these unregulated striatal neurons, whose membrane potentials are allowed to float.

Electrotonic propagation is responsible for actually computing the potential function. The membrane potential of each medium spiny cell corresponds to the function value. Higher (glutamate-induced) potentials represent repelling states, while low (GABA-induced) potentials indicate goal states. It has also been shown that the spines on medium spiny neurons act as a low-pass filter [66, 37]. This greatly diminishes the effect of an action potential on membrane potentials at the dendrites. This is important, because 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 therefore not affect the overall potential function. 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 via gap-junctions. The power consumption of such a network may very well be reflected in the rather high rate of glucose metabolism within the striatum, as evidenced by PET scans [46]. Although these cells are not firing, they are consuming energy in order to maintain the required potential differences for the computation of the harmonic function.

Once computed, the function can be used to drive state changes within the organism. Research has shown that medium spiny cells fire both to induce joint torque (and hence motion), and in response to joint torques (e.g., passive movement) [28, 35]. We propose that at any one time during the execution of movement, only a small cluster<sup>4</sup> of medium spiny cells will fire within the

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<sup>4</sup>We denote this as the "current-state cluster".

neostriatum. 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. Crutcher and DeLong [28] and Kimura's [29] experiments show 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 [51, 50].

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 3). 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.

Migration of the current-state cluster is accomplished when a joint is moved in a particular direction. This causes the cortex to stimulate the torque-sensitive striatal cells. We believe that such cells are offset from the cluster in a particular way, so as to bias the migration of the cluster. Figure 4 shows an idealized diagram denoting the arrangement of cells. Open circles represent cells which are stimulated by passive movement. Filled circles represent cells whose primary function is to induce movement. It is assumed that each of these cells receive dopaminergic input from the SN. Each dimension of the grid represents a state parameter, e.g., the angle of a particular joint. To see how the migration of the current-state cluster might work, imagine that the cluster is to move to the right, representing an increase in joint angle 1. As the first cluster fires, the joint moves according to the surface provided by the potential function. The gradient of the potential surface (i.e., the actual membrane potentials) prescribes the relative firing rates of cells within the cluster. This induces a particular joint motion. This joint motion, in turn, causes cell 2 to fire, depriving its immediate environment of dopamine. Corticostriatal afferents simultaneously fire in the vicinity of cell 1 to presynaptically stimulate dopamine release from nigrostriatal cells afferent on neighbors of cell 1. This leads to more frequent cell firing in the neighborhood of cell 2, again regulated by the local shape of the potential surface, and again inducing a joint motion. Decreased firing is seen at cell 1. This process is repeated until the cluster (and hence the joints) reaches a goal state. It

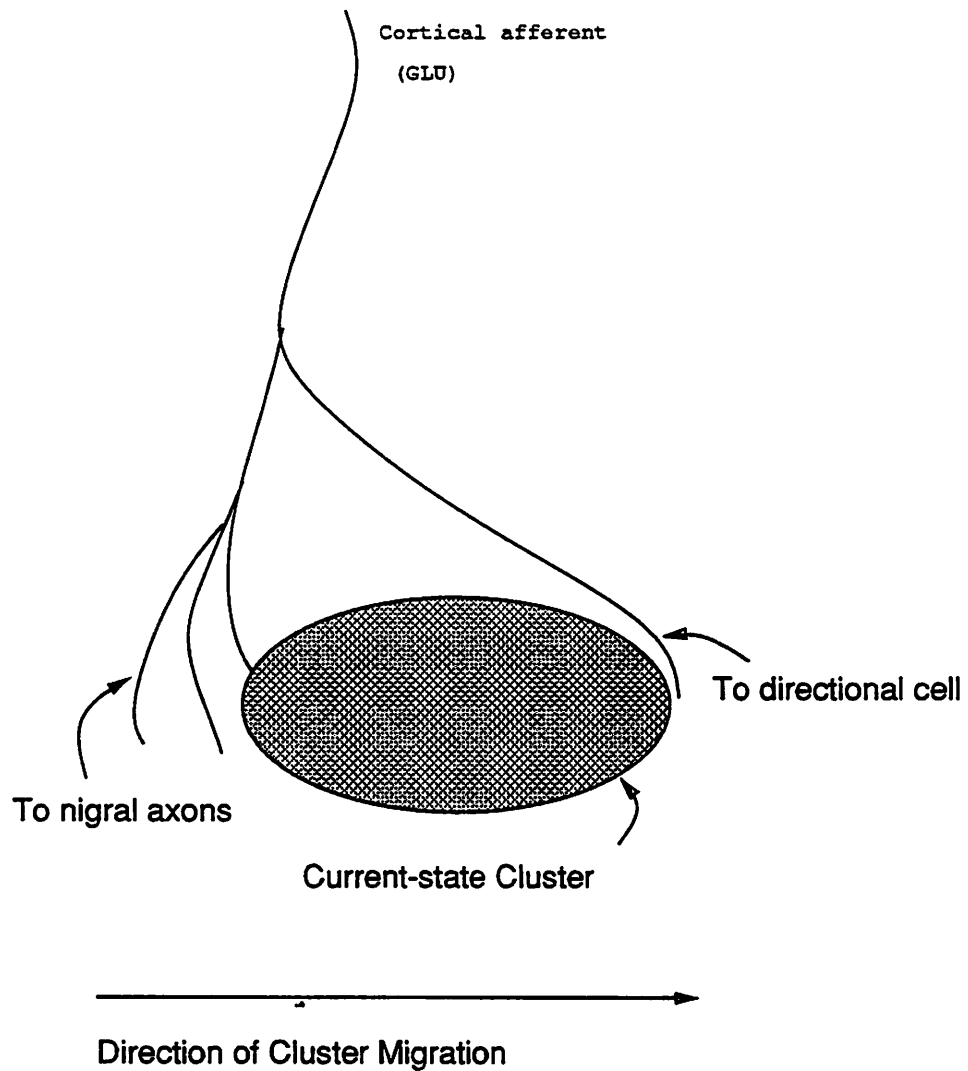


Figure 3: Schematic of the migration of a current-state cluster within the striatum. Glutamate is supplied via cortical afferents. This in turn releases dopamine toward one direction, and excites cells (and probably inhibits dopamine release) in the other direction. The resulting dopamine imbalance shifts the firing cluster in the appropriate direction.

should be noted that some researchers have observed the network-like arrangement of groups of cells in the striatum [62, 33, 67].

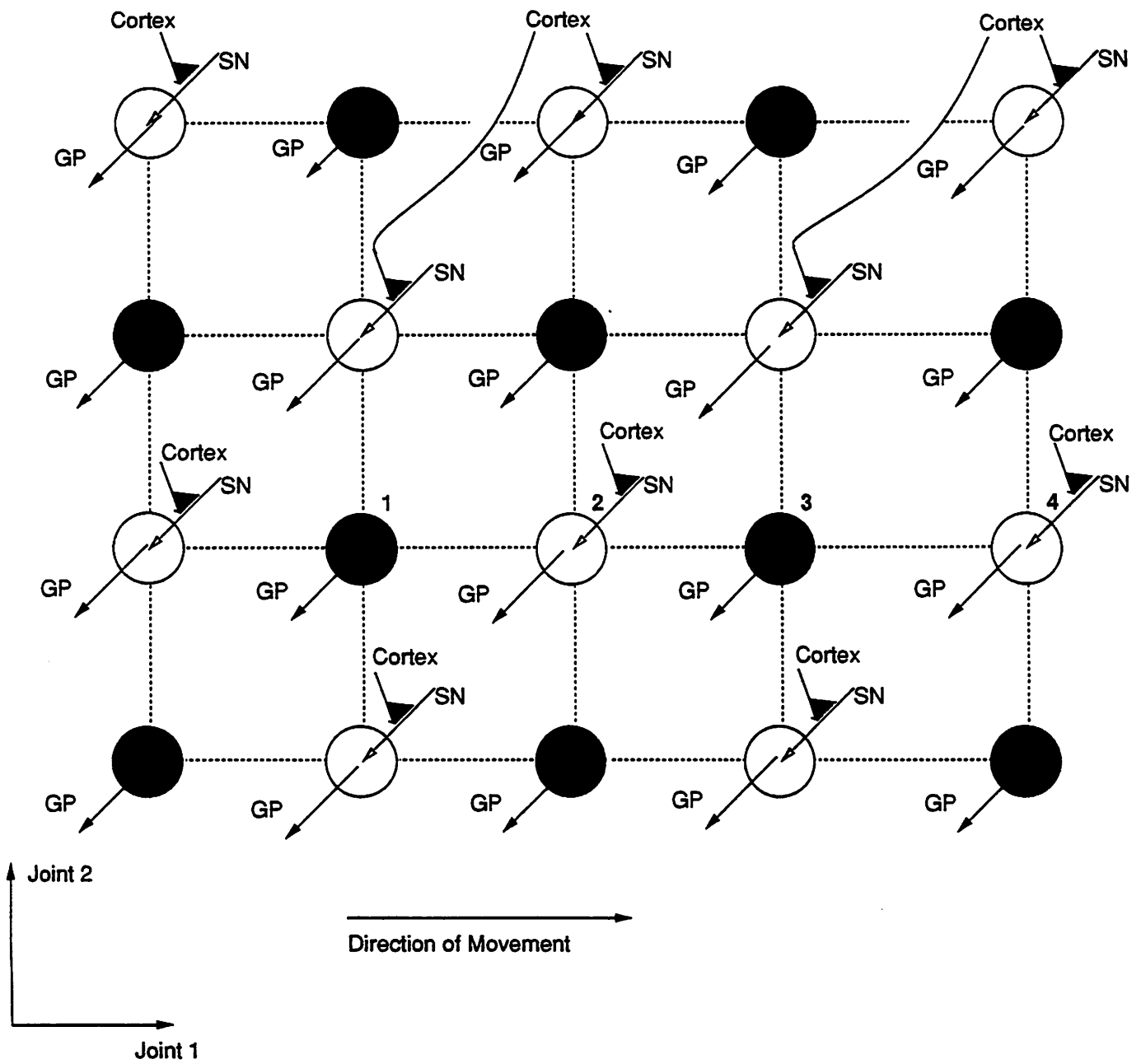
The giant aspiny cells appear to act as dampers or brakes. Their tonic activity tends to prevent spontaneous firing of medium spiny neurons. These cells presynaptically regulate dopamine release, as in the case of some corticostriatal afferents [57]. This tends to prevent spontaneous firing of medium spiny cells, and therefore inhibits inappropriate striatal activity. When these cells stop firing (just before movement), they cease the flow of excess dopamine, in effect releasing the brakes to allow the potential function to affect the motor system. Kimura has noted that some cells (possibly medium spiny) actually fire only once at the outset,<sup>5</sup> and are then silent. It seems likely that the cortex starts the process by inhibiting the giant cells, then firing certain medium cells to start up a current-state cluster, which then triggers the appropriate state changes. It is also conceivable that movement is initiated directly through a cortico-cortical pathway, which in turn causes directional striatum cells to fire, thus starting the current-state cluster. The latter scenario is made more plausible by studies which suggest that the basal ganglia are not involved in the initiation of movement. In any case, the tonic cholinergic cells have an inhibiting role in the striatum, keeping the medium spiny cells quiescent until the striatum is required to generate a state-change path.

## 4 Moot Issues

There are several issues to be addressed with respect to this model. The most obvious is the apparent heterogeneity in the neostriatum itself. Several researchers [31, 68, 69] have noted that the putamen and caudate can be divided into two types of compartments: striosomes, and extra-striosomal matrix. The striosomes are AChE-poor, and appear to have reciprocal striatonigral projections. The predominant cortical afferents and pallidal efferents are based in the matrix. For this reason, we suggest that the primary computation of potential functions is taking place within the matrix portion of the neostriatum. In robot path-planning, there is usually a need to define and avoid self-collision states. In a robot state-space, these are manifested as straight lines denoting the condition that two joint angles are equal. The contiguous tube-like arrangement of striosomes [68] in conjunction with their AChE-poor, and dopamine-rich properties make them likely candidates for self-collision zones. Such zones would of necessity be permanent features. High dopamine levels imply that gap-junction resistance in striosomes would also be high. In other words, such cells are chronically "turned off", meaning that state changes (current-state clusters) would never invade striosomes, but would flow around them. This is consistent with the fact that striosomes are much less plentiful in lower vertebrates; these animals still have a need to maneuver within their

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<sup>5</sup>Could these actually be medium aspiny cells?



Medium Spiny Cell Network - Inputs and Outputs

Figure 4: Schematic of striatal medium-spiny clusters, and the migration of the current-state cluster. Dotted lines indicate electrotonic coupling. Arrows denote afferents from the substantia nigra (SN) and efferents to the globus pallidus (GP).



environment, even though they are not equipped with limbs as manipulative as the primates'. Self collision of limbs is less of an issue in lower vertebrates.

This model must also consider the function of the globus pallidus. Clearly, the GP is the major output for the striatum. Mink and Thach [38, 39, 40] have demonstrated that the globus pallidus does not directly encode velocities. We find this to be consistent with our model, since the gradient of a harmonic function as computed by a resistive grid would be highly variable, and will almost certainly require some kind of normalization. For the work described by Connolly [24], the velocity vectors supplied to the robot controller were all normalized for a particular speed. Thus, it is not necessary for this model to be producing speed values. Velocity direction will suffice. Presumably, speed of movement is controlled by some other process. It is also possible that the pallidothalamic pathways are designed for either mass-coding or even digitization. These two forms of encoding are easily accomplished, but would imply that individual GP neurons would not exhibit a clear correlation with motion parameters. Moreover, Aldridge and Gilman [70] show that even within the striatum, the coding scheme used for striatopallidal neurons is unclear.

## 5 Basal Ganglia Disease

This section is also quite speculative, especially given that the authors are not medical professionals. We wish to simply suggest plausible explanations for certain symptoms of basal ganglia disease in light of the model described above.

### 5.1 Parkinson's Disease

Certain aspects of basal ganglia disease can be explained in light of the model presented here. For example, in Parkinson's disease, degeneration of the substantia nigra is seen, with a concomitant loss of dopamine in the striatum. According to our model, this dopamine deprivation would result in a decrease in gap-junction resistance, similar to that observed by Cepeda and co-workers [32, 62, 63, 33]. It is also known, however, that the striatum is not completely starved of dopamine. In fact, remaining nigral cells are found to increase their dopamine production (hence the usefulness of L-Dopa therapy) [71]. The pattern of cell loss suggests that groups of striatal neurons could become tightly coupled due to low gap-junction resistance. This would result in cell islands surrounded by other cell groups which still have active nigral afferents.

The first consequence of decreased dopamine levels is a lowering of the time constant of the cells. Since the time constant  $\tau = RC$  is directly related to resistance, the reduction in resistance due to lower dopamine levels would cause a corresponding reduction in time constant, assuming that capacitance does not increase. This in turn implies that the cell is less effective at providing low-pass

filtering at its junctions with its neighbors. We would thus expect to see greater fluctuations of the membrane potentials of cells in response to potential changes (especially spikes). The integrity of the potential function, then, is less secure.

The second consequence of decreased dopamine levels would be an increase in spontaneous firing rates of striatum cells [55]. Spikes thus generated will tend to jostle the local potential function because of the decreased ability of medium spiny cells to filter these spikes. The combination of these two effects results in inappropriate inhibitory input to the globus pallidus, which in turn results in unintended movement (i.e., the rest tremor). The process is sustained because the movement thus produced causes further firing in the striatum in the regions corresponding to the current limb positions. Note that this explanation for the tremor does not require the cortex to explicitly set up a goal state.

A third consequence of lowered dopamine levels could occur in conjunction with the coupling of cells due to low resistance. This effectively causes a reduction in resolution of the state space. Note that cells so coupled would result in a tendency for them to act as a unit. There would be little change in potential from cell to cell within this unit. Therefore, when movement (i.e., a state change) is required or induced, the state transitions would reflect a more discrete pattern than is normally the case. Since there would be little change in potential within a cluster of dopamine-deprived cells, the gradient of the function in this vicinity is close to zero, and will not be able to affect the globus pallidus in the proper way. We believe that this mechanism could explain the cogwheel rigidity and akinesia seen in Parkinson's patients.

## 5.2 Huntington's Chorea

Huntington's chorea appears to be characterized by medium spiny cell loss within the striatum. One theory for this suggests that excess glutamate leads to cell death [46]. Excess glutamate could be responsible for a variety of problems, among them inappropriate cell depolarization, GABA-polarization (via medium spiny dendrites?), and changes in dopamine levels (via presynaptic regulation?). All three of these effects, if present, would be enough to create an inappropriate harmonic function which results in unintended movement.

## 6 Summary

We have described very roughly a model for the low-level functioning of the neostriatum 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. 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. We hope that the model will beg several useful questions. Among the apparent questions are:

1. What is the spatial firing pattern of the medium spiny cells? In other words, do these cells truly fire in sparse, migrating clusters as a movement progresses?
2. What is the effect of more complex tasks on the firing patterns of medium spiny cells?
3. What is the pattern of cell-to-cell resistances among striatal medium spiny cells?
4. How does the globus pallidus encode its output?
5. Is there evidence for connexin mRNA in medium spiny cells?

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 [42] that the caudate nucleus is involved in localization and egomotion, and the harmonic function approach very easily applies to mobile robots [22], 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 the harmonic function in the context of the more esoteric functions of the caudate nucleus.

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