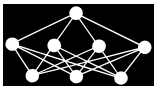

NEUROCONTROL III: DIFFERENCING MODELS OF BASAL GANGLIA–THALAMOCORTICAL LOOPS

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Abstract:

Two models of basal ganglia–thalamocortical loops are proposed, both of which feature population coding and utilize local approximations. The first model is a temporal differencing scheme similar to the temporal differencing striosome models [1, 2]. In this model the basal ganglia perform temporal differencing by means of their two arms, viz. the direct and the indirect pathways. The second model involves control architeture based on the so called position–and–direction–to–action (PDA) map [3, 4]. The architecture utilizes this PDA map for both static and dynamic state (SDS) feedback to achieve precise control under perturbed conditions [5]. Here it is argued that the differencing part of the SDS scheme may be viewed as a model of the basal ganglia, while the full SDS scheme may be considered as a model of basal ganglia – thalamocortical loops. The SDS scheme is different from the temporal differencing model as it may also generate correcting commands if control is taken over and carried out by independent motor programme executing units. Relationships to neurobiological and psychophysical findings are discussed.

Key words: *Neural network, neurocontrol, dynamic feedback, basal ganglia*

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1. Introduction

Over recent years a considerable amount of research has been carried out to characterize the structure and the functional organization of the basal ganglia (BG). In spite of many useful findings, it is still not known how these nuclei contribute to behavioural and motor control. Various functional and computational models have

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been proposed that emphasize different aspects of the basal ganglia–thalamocortical interaction.

For example, electrotonic coupling (EC) model of the BG [6, 7] argues that the BG are responsible for driving smooth transitions of state. These works suggest that the corticostriatal arborizations implement a mapping from Cartesian coordinates (i.e., from a description formulated in terms of external space) to configurational space (i.e., to a description formulated in joint coordinates). The model proposes that the corticostriatal projections realize one of the steps of the computational sequences of control subserving goal-directed movements. According to the EC model the activated corticostriatal projections correspond to states of joint kinematics that are needed to achieve the planned trajectory. The model is based on dye coupling experiments [8, 9, 10, 11, 12] suggesting the possibility that electrotonic coupling via gap junctions takes place between striatal medium spiny neurons (MSNs). Then, according to the model, the striatum generates a control surface, or potential function, for guiding state changes by means of those contiguous regions which are cellular representations of state spaces. These state spaces would correspond to various aspects of organism functions. Each region continually computes (deforms) a harmonic function based on cortical input consisting of obstacle and goal information. In the putamen, the somatotopic regions are suggested as being 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.

With regard to the winner-lose-all (WLA) model [13], this is centred around the specific architecture of BG. It is based on the observation that the anatomy of the BG, and the connectivity of excitatory and inhibitory neurons may be interpreted as a WLA circuit. The main elements of the WLA circuit are as follows: a multimodal sensory and cognitive map from the cortex projects in a convergent manner to the striatum; the prefrontal cortex contains replications of the thalamic output integrated over different time scales; processed sensory representation of the environment is received by the striatum. The striatum sends two projections: a highly convergent one to the external segment of the globus pallidus (GPe), and a moderately convergent one to the internal segment of the globus pallidus (GPi). The first pool of striatal neurons that reaches the firing threshold inhibits its target in the GPi. This inhibition leads to the disinhibition of the corresponding thalamic targets and the gating of ascending information to the corresponding cortical motor neurons. At the same time, inhibition of the GPe neuron results in the disinhibition of the corresponding neuron of the subthalamic nucleus (STN), which then diffusively excites the other GPi neurons. This diffuse excitation is then assumed to prevent all but the first action from being selected. The cortico-STN pathway acts as negative feedback that turns off selected actions within the loop. The primary assumption for the WLA mechanism is the existence of streams of information that remain segregated from striatum to thalamus and that each of the segregated pools of neurons is devoted to a particular action.

The adaptive critic temporal differencing models of striosomes [1, 14, 2, 13] are neural models that address the temporal credit assignment problem [15, 16, 17] through the detection of events that predict subsequent reinforcement. Both models are based on the anatomy and physiology of the striosome compartments of the striatum and on the signalling properties of the dopamine neurons of the BG. In

these models the striosomes generate the prediction of reinforcement versus time during the course of an action.

The present paper offers new type model. This new type of model is related to the earlier models in the following way:

- it deals with the matrisomes of the BG and may augment the models of the striosomes;
- it agrees with the EC model in that the outputs of the BG are related to different configurational states, although the model does not assume that the outputs specify configurations;
- it agrees with the WLA model in that the outputs of the BG correspond to actions, but it does not assume any direct WLA mechanism, nor that there would be a decision made at BG level to accomplish a given particular discrete action.

The SDS model features the following properties:

- it solves the path planning problem without explicit trajectory formation;
- it suggests that the cortex may formulate the actions to be executed in terms of speed field tracking;
- it eases the degrees–of–freedom problem in motor planning [18] by avoiding (i) phase–space discretization and, possibly, (ii) planning in configurational space;
- it offers a solution to the learning/adaptation dilemma;
- it formulates a hierarchy of training signals.

In short, the SDS model assumes that the two main pathways of the BG, i.e., the direct and the indirect pathways both having functionally discrete channels of information processing, serve as the dynamic state feedback route. The functionally discrete channels correspond to different sets of sign–proper control correcting outputs to different body parts organized in a somatotopic manner. Sign–proper control means whether a given correcting control signal requires the enhanced use of the "extensor" or the "flexor". The two interpretations outlined in the paper both suggest differencing schemes. In the first case the BG function is interpreted as temporal differencing. This interpretation of the functioning of the matrisomes is in line with the striosomal models [14, 13] and suggests a general strategy of BG computations.

In the other interpretation of the BG function the GPi computes the difference between the desired and the experienced control values and feeds it back to the control areas of the cortex via the thalamus. The direct pathway corresponds to the computed desired control values and tends to decrease BG output by directly suppressing activity at the level of GPi. The result is the disinhibition of the thalamocortical pathway. In contrast, the indirect pathway corresponds to experienced moves and thus to the control values that would have been computed to accomplish these experienced moves. The indirect pathway tends to increase BG output

by increasing neuronal activity at the level of the output nuclei — in one case by disinhibiting the STN having excitatory projections to GPi and to the substantia nigra pars reticulata (SNr), and in the other by directly disihibiting GPi and SNr. This interpretation allows correcting signals to be produced if control is executed by other unit(s) and not the BG. Then the BG function is that of controlling *and* of creating a hierarchy of training signals.

The paper is organized as follows: first the anatomy, the functional organization and some particular neurotransmitters of the BG and the BG–thalamocortical loop will be detailed. Then the two pathways and the experimental findings about the working of the BG will be reviewed. After these sections a description is given of the position–and–direction–to–action (PDA) map that may be learnt by direct associative identification and models the inverse dynamics of a plant. The PDA map is utilized to formulate the temporal differencing model of BG. Then the SDS model and the SDS model of BG are presented. The discussion section details the features of these differencing models and relates these features to other neurobiological and psychophysical findings, in particular to (i) the bimodal somatosensory receptive fields in the putamen [19] and the ventral premotor cortex [20] that provide a gelatinous medium surrounding the body in the form of a distributed representation suitable for coding joint configurations and to (ii) the selective aspects in the early acquisition of skill [21]. The discussion section also treats the models in relation to the BG diseases. At the end of this section a comparison is made between the EC, the WLA and the SDS models of BG. Conclusions are drawn at the end of the paper.

2. Outline of the organization of basal ganglia

The basal ganglia have as their main components, the caudate and lentiform nuclei, both of which are large subcortical structures [22]. The lentiform nucleus comprises of two parts, the putamen and the globus pallidus (GP), these differ largely in histological structure and anatomic relationships. The putamen – the lateral part of the lentiform nucleus – is similar to the caudate nucleus, and the two structures together form the striatum.

Also included in the BG are the substantia nigra (SN) and the subthalamic nucleus (STN). The SN is to be found on the dorsal side of the basis pedunculi and extends throughout the mesencephalon from the rostral border of the pons into the subthalamic area. In its cell rich part, the substantia nigra pars compacta (SNc), dopamine is synthesized. The dopamine-synthesizing cells are also to be found in the ventral tegmental area (VTA) too. The cell-sparse part of the SN, the substantia nigra pars reticulata (SNr), is located ventrally and adjacent to the dopaminergic cell groups of the SNc. The SNr and the GP are the output structures of the BG. The STN influences motor activities primarily through its prominent projections to these two output structures. The STN receives direct input from motor regions in the cerebral cortex.

The major input to the BG is from the cerebral cortex, and the whole cortical mantle is involved in this highly organized projection system to the striatum. The somatosensory and motor cortices project somatotopically to the putamen [23, 24, 25]. The putamen also receives direct projections from parietal area 7b [26, 27].

The cortical association regions in the frontal, temporal, and parietal lobes project to the caudate nucleus.

GABAergic MSN projections are the most abundant neurons in the striatum [28]. Only the axons of the MSNs reach the two output structures of the BG, the GP and the SN, through the direct and the indirect pathways. These projection neurons are divided into two subpopulations: one projecting to the external segment of the globus pallidus (GPe), the other projecting either to the internal segment of the globus pallidus (GPi) or to the SNr [29].

The GPi and the SNr, that project to the thalamus and the brain stem, form the major output structures of BG. The pathways to the thalamus are topographically organized: the GPi projects to the ventral anterior – ventral lateral (VA-VL) complex and the ventral pallidum primarily to the mediodorsal thalamic nucleus (MD). The SNr projects to the VA-VL complex and MD nuclei. The VA-VL complex and MD, in turn, project to motor-premotor and prefrontal cortical areas. Other projection sites include the superior colliculus and the reticular formation in the mesopontine tegmentum. The GPe sends inhibitory, feedforward projections to the reticular thalamic nucleus (RTN). The RTN sends GABAergic projections to the BG recipient nuclei of the ventrolateral thalamus.

The nigrostriatal dopaminergic pathway forms one of the side loops of the BG connection system. The dopaminergic cells of the SNc and the VTA project in a more or less topographic fashion to the caudate and the putamen.

The most important side loops involve the STN; these are inputted from the GPe and influence the GPi and the SNr. These side loops form the indirect pathway [30] in contrast to the direct pathway which is formed by direct striatopallidal MSN projections.

All five major structures are functionally subdivided into skeletomotor, oculomotor, associative, and limbic territories based on their physiological properties and their interconnections with cortical and thalamic territories having the same functions [31].

The striatum has a modular structure with two types of modules, the striosomes [32, 33, 34] and the matrisomes [35, 36, 37]. Both the striosomes and the matrisomes have interconnectivity within the modules, but not between them. The tonically active neurons (TANs) of BG tend to lie at striosome–matrisome boundaries [38]. The striosomes are neurochemically specialized patchy input–output zones that tend to collect inputs related to the limbic system and to project to the dopamine–containing SNc. In contrast, the matrisomes receive sensorimotor and associative inputs and project to the output nuclei of the BG.

Any given matrisome receives overlapping inputs from the same body-part representation in different subareas of the sensorimotor cortex, so that several sorts of information relevant to that body part converge. Experiments in which anatomical tracers were placed into monkey brains to label simultaneously anterograde sensorimotor inputs to the striatum and retrograde striatal outputs to the pallidum showed that labelled input fibre clusters can overlap clusters of backward labelled projection neurons quite precisely [39, 40, 41]. The emerging picture is that the information is dispersed to distributed modules in the striatum, but it can be brought together again at the next stage of processing, i.e., at the pallidum. This pattern has been observed both for striatal projections to the GPe and for projections to

the GPi [42].

The organization of the BG can thus be viewed as a family of reentrant loops that are organized in parallel, each taking its origin from a particular set of functionally related cortical fields passing through the functionally corresponding portions of the BG, possibly undergoing extensive remapping and returning to parts of those same cortical fields by way of specific BG recipient zones in the dorsal thalamus. Because of their parallel organization, corresponding stations along each of the functional loops are thought to carry out similar operations [43].

The convergence of direct and indirect projections to the GPi is estimated as 1000:1 [44, 45, 46, 47, 48]. The convergence is manifested by the wide dendritic arborization of pallidal and nigral neurons oriented at right angles to the incoming striatal axons with small number of contacts between the striatal axon and a single pallidal cell. Transneuronal retrograde tracing indicates that the BG – thalamocortical loops are organized to form multiple segregated output channels [49].

3. Direct and indirect pathways of the basal ganglia

As is known, the corticostriatal projections are glutamatergic and excitatory whereas the striatopallidal and the pallidofugal connections are GABAergic and inhibitory. Within the motor circuitry of the BG there are two major projections. The direct pathway projects from a certain subpopulation of striatal neurons in the putamen to the GPi. With regard to the indirect pathway, one arm has its origin in a different population of striatal neurons, then proceeds through the GPe and the STN before reaching the GPi. The second arm of the indirect pathway is made up of GPe projections to the output nuclei themselves. All intrinsic projections are GABAergic and thus inhibitory, except the projections from the STN to the GPi, which are excitatory. The two main arms of the two pathways are illustrated in Fig. 1.

These pathways are complemented by the GPe–RTN–ventrolateral thalamus pathway. Also, (i) most of the putamen-projecting sensorimotor areas send excitatory connections directly to the STN, and (ii) the STN sends glutamatergic connections back to the GPe and the putamen.

There appears to be a functional consistency among the various GPe and GPi projections [43]. Activation of MSNs associated with either arm of the indirect pathway will tend to increase BG output by increasing neuronal activity at the level of the output nuclei. In one case the BG output is increased by disinhibiting the STN with its excitatory projections to the GPi and the SNr, and in the other by directly disinhibiting the GPi and the SNr. In contrast, activation of MSNs associated with the direct pathway tends to decrease BG output by directly suppressing activity at the level of GPi and SNr. The net result is that cortically initiated activation of the (indirect) direct pathway will tend to (suppress) enhance reentrant thalamocortical excitation by (increased) decreased inhibitory outflow from BG to thalamus.

The functional consistency of various GPe and GPi connections is further emphasized by the dopamine pathways. Dopaminergic input to the putamen consists of nigrostriatal projections that originate in the SNc. The cortical motor and

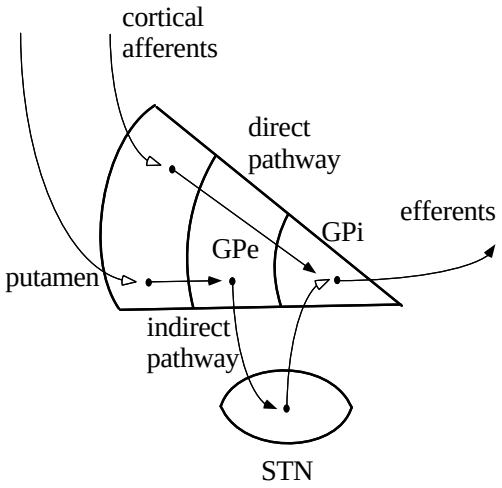

Fig. 1. Schematic representation of parts of the direct and the indirect pathways. *Both pathways are excited by corticostriatal projections. The part of the direct pathway shown in the figure inhibits the pallidofugal connections of the internal segment of the globus pallidus (GPi) and thus disinhibits the thalamocortical connections. The part of the indirect pathway shown in the figure inhibits the projections of the external segment of the globus pallidus (GPe) and, in turn, disinhibits the excitatory projections of the subthalamic nucleus (STN) to the GPi and thus reinforces the pallidofugal connections and, in turn, suppresses the thalamocortical projections. Open and filled arrows represent excitatory and inhibitory connections, respectively.*

premotor areas receive separate dopaminergic projections from the VTA. At the network level dopamine has an inhibitory action on the striatal GABAergic cells projecting to the GPe, but an excitatory action on the neurons projecting directly to the GPi and the SNr. The role of dopamine could be viewed as increasing (decreasing) the effects of the direct (indirect) pathway.

Dopamine has also been shown to have a role in synaptic plasticity within the striatum being implicated in both long-term potentiation (LTP) and long-term depression (LTD) [50]. It seems that dopamine has a permissive role in striatal synaptic plasticity: The presence of dopamine at striatal synapses appears to be necessary for LTP/LTD to occur with additional factors required such as the activation of corticostriatal inputs and/or the associated depolarization of the postsynaptic MSN [51].

4. Experimental findings on the working of the basal ganglia

In this section first the pioneering works of Alexander, Crutcher, DeLong and Strick [52, 53, 54, 55] about the dissociation of preparative, directional, muscle-like, pre- and post-instruction, etc., neuronal activities will be reviewed. Then the intriguing results of Schultz and colleagues [56, 57, 58] will be outlined. These results will

play an important role when arguing about the WLA interpretation [13]. We shall also review here the findings on synaptic plasticity, including the properties of the dopamine neurons of the SNc [59, 60] and the properties of TANs [42].

It has been shown [53, 54, 55] that neural responses in the supplementary motor area (SMA), primary motor cortex (MC) and putamen associated with the planning and execution of visually guided limb movements are very similar and largely overlapping, with some delay in the neural responses within the putamen. Neurons could be sorted into different groups and to some extent these groups were not disjunct but overlapping. Movement related cells were classified as directional if they showed an increase in discharge rate predominantly or exclusively during movements in one direction and did not have significant static or dynamic load effects. A cell was classified as muscle-like if its directional movement related activity was associated with static and/or dynamic load effects and if the activity pattern was similar to that of flexors or extensors of the forearm. Cells with directional activity appeared to be coded for trajectory/kinematics-level variables while cells with muscle-like activity appeared to be coded for dynamics/muscle-level variables. Also, cells were found that showed preparatory activity that stopped at the onset of movement. In most cases preparatory activity was found to be independent of the loading conditions. Moreover, there were both preparatory and movement related cells which showed limb-dependent preparatory activity, irrespective of the visual position of the target and appeared as coding trajectory/kinematics variables. At the same time other cells selectively discharged to the direction of the visual position of the target and were termed as "target-dependent" cells. These studies revealed that all of these areas, i.e., the SMA, the MC and the putamen, contain neural representations of several different levels (i.e., trajectory level and dynamics level) of motor processing. The differences between neuronal populations of different cell types in the SMA, MC and putamen were not extensive and did not allow one to determine functional differences for the studied, relatively simple, stimulus-triggered movements.

In the experiments of Romo, Scarlatti and Schultz [56, 57, 58] another aspect of movement control was studied in the anterior striatum and the SMA. These studies investigated the differences between the stimulus-triggered and self-initiated movements. Again, the studies pointed to the combined role of SMA and the striatum in the internal generation of individual behavioural acts and in the preparation of behavioural reactions. The large temporal overlap of activity – found in these studies – that preceded externally and internally initiated movements suggested that this activity may not originate from a single group of neurons or a single structure. Rather, as was suggested in these studies, it may develop through interactions in neuronal groups linking several cortical and subcortical structures. Preparatory activity is found at several levels of cortico-basal ganglia loops, such as the globus pallidus [61, 62], SNr [63, 64], and according to these studies also at the frontal cortex, the caudate nucleus and the putamen.

The dopamine neurons located in the SNc and the VTA play an essential role in both the primary reinforcement of behaviour and in guiding preparatory behaviour based on the likelihood of delayed reinforcement [65, 66, 60]. Microelectrode recordings from dopamine neurons reveal that at the first stage of learning of a new behavioural task, dopamine neurons discharge in response to the primary

reinforcement. At a later stage of learning, dopamine neurons start to discharge in response to stimuli that more or less regularly precede the primary reinforcement. At the same time the responses to the primary reinforcement progressively disappear.

In the work of Graybiel, Aosaki, Flaherty, and Kimura [42] the role of TANs in BG plasticity was studied. It was found that TANs were widely distributed through the striatum. There was a pronounced tendency for TANs to lie at striosome-matrix borders. TANs were distributed over broad regions of the striatum and showed temporally coordinated activity after behavioural conditioning. It was suggested that these striatal neurons could facilitate coordinated changes in the activity of other striatal neurons, including the MSN projection neurons during learning. Also, Schultz and colleagues [67] found a high degree of homogeneity in the responses of the dopamine neurons. These facts are very much in line with the findings that MPTP treatment, that destroys dopamine-containing fibres resulted in the partial loss of the temporally coordinated responses of TANs and that the response could be reinstated by systemic injection of apomorphine. This led to the suggestions that the expression of the acquired responses of the TANs requires tonic dopaminergic input and that the dopamine signal needed to express the response may be spatiotemporally permissive [42].

Another study has dealt with the activity correlations of GP neurons. It was shown that in contrast with cortical and thalamic neuronal activity, almost all pairs of GP neurons in the normal monkey were not driven by a common input [38].

We shall argue in the discussion that these features are consequences of the differencing models of BG.

5. Two differencing models of the basal ganglia – thalamocortical loops

First the PDA map is briefly reviewed (for detailed descriptions, see [3, 4]) and, after which possible interpretations of the BG – thalamocortical loops will be constructed.

The starting point of these models is that the set of possible trajectories is too large to be directly specified and, instead, a dynamic and distributed representation is created by the object under control (i.e., the limb), the target to be reached, and the obstacles to be avoided. This point is supported by biological findings since there is no evidence of signals in the brain that would specify trajectories. At the same time signals that are capable of specifying the positions of targets in the extrapersonal space can be found in the parietal association cortex [68].

Also, the PDA map provides a distributed representation of motor commands, the so called populations coding [69].

5.1 The PDA map

The architecture of the feedforward inverse dynamics controller is shown in Fig. 2. It is a fully self-organizing architecture that forms a Position-and-Direction-to-Action (PDA) map introduced earlier [3]. It has four layers: a sensory layer (SL), a geometry discretizing layer (GDL), an interneural layer (INL), and a control layer

(CL). The layers are connected as follows. Spatially tuned feedforward connections bring the sensory information to the GDL. This has recurrent self-organized intralayer geometrical connections (GCs) that connect neighbouring nodes of the GDL and govern a diffusive equation. The sustained input activities of the SL give rise to a steady state diffusion field (SSDF). It is assumed that sensory inputs corresponding to start, target, and obstacle entities are segmented and recognized by some higher order system and the start, target and obstacle nodes of the GDL are identified. The start and target inputs serve as sources and sinks of the diffusion, respectively on the GDL. The obstacle inputs form the forbidden zone: diffusion through GCs of nodes receiving obstacle inputs is inhibited. Under these conditions the diffusion has its maximum at the start node(s), its minimum at target node(s), and it avoids the obstacle regions. The model is an extension of previous works [70, 71, 72, 73]. The diffusing activities are sensed by the interneurons. These are simple linear I/O units that serve as sensors and also as starting points for associative feedforward connections towards neurons of CL. The network is fully self-organizing and utilizes Hebbian-learning [4]. The said higher order recognition module that initializes the formation of the steady state GDL activities is not modelled. The INL to CL connections (that are called control connections (CCs)) form the direct, associative identification of the inverse dynamics in the form of a PDA map. Here it is assumed that the direction of the desired motion is formulated in the external space using body centered coordinates and that the PDA is modulated by the configuration of the joints. The crucial points of the assumption are that (1) this modulation is a relatively smooth function of the configuration that allows coarse coding for the modulation function and that (2) the controller is robust against imprecisions. Biological findings support the assumption of smooth configuration modulated dynamic remapping that subserves motor control [20]. It should be noted that the outputs of the PDA map do not correspond to the final control values but form one of the control terms. The free space learnt CCs will move the plant along the gradient of the SSDF if the CCs belonging to the interneurons of start node GCs control the motions. It is emphasized that the SSDF on the GDL forms a distributed diffusion field [3, 4]. In other words, without the explicit representation of the trajectory the plant may start to follow the trajectory defined in the external space.

The obstacle avoidance without further training is a natural consequence of the structure, if diffusion to and from nodes that are identified as obstacles is inhibited (cf. [3]). Learning properties are discussed in [4].

Since the controller should move the plant along the SSDF, the control problem of a first order plant may be formalized as follows. The equation of motion is given as [74]

$$\dot{\mathbf{q}} = \mathbf{b}(\mathbf{q}) + \mathbf{A}(\mathbf{q})\,\mathbf{u} \tag{1}$$

where $\mathbf{q} \in \mathbf{R}^n$ is the state vector of the plant, $\dot{\mathbf{q}}$ is the time derivative of $\mathbf{q}$, determined by the SSDF, $\mathbf{u} \in \mathbf{R}^m$ is the control signal, $\mathbf{b}(\mathbf{q}) \in \mathbf{R}^n$, and $\mathbf{A}(\mathbf{q}) \in \mathbf{R}^{n\times m}$ that admits a generalized inverse, $\mathbf{A}^{-1}(\mathbf{q})$. We assume that the domain (denoted by D) of the state variable $\mathbf{q}$ is compact and is simply connected; that $n \leq m$, and for each $\mathbf{q} \in D$ the rank of matrix $\mathbf{A}(\mathbf{q})$ is equal to n; that is, the matrix is non-singular. As a consequence the plant is strongly controllable. In this case the inequality $n < m$ means that there are more independent actuators than

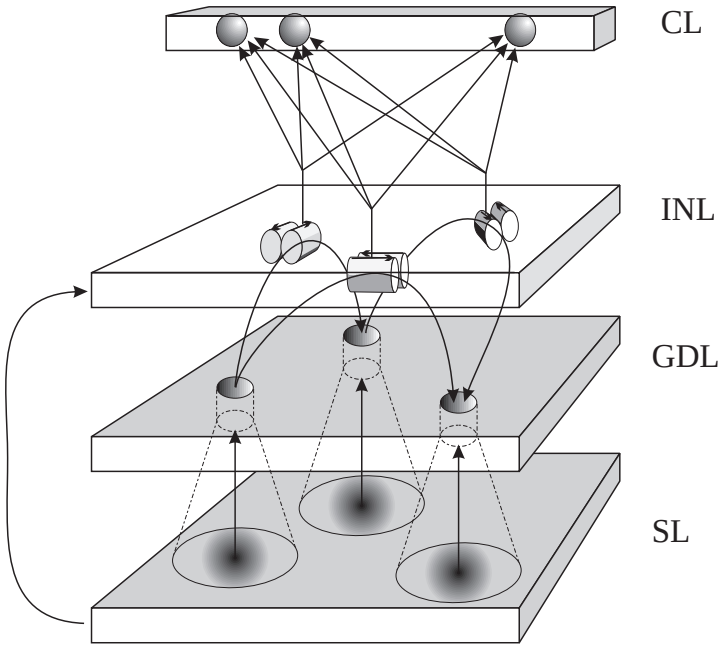

Fig. 2. Scheme of the self-organizing PDA architecture. *CL: control layer, INL: interneuronal layer, GDL: geometry discretizing layer, and SL: sensory layer.*

state vector components, i.e., the control problem is redundant. (The problem of higher order plants will be discussed later.)

Let $\mathbf{v} = \mathbf{v}(\mathbf{q})$ be a fixed n dimensional vector field over D. The *speed field tracking task* is to find the static state feedback control $\mathbf{u} = \mathbf{u}(\mathbf{q})$ that solves the equation

$$\mathbf{v}(\mathbf{q}) = \mathbf{b}(\mathbf{q}) + \mathbf{A}(\mathbf{q})\mathbf{u}(\mathbf{q}). \tag{2}$$

Conventional tasks, such as the *point to point control* and the *trajectory tracking* tasks cannot be exactly rewritten in the form of speed field tracking, and speed field tracking is more robust against noise than these conventional tasks.

Speed field tracking is robust in the following sense. Assume, that the controller is such that it can follow the speed field $\mathbf{v} = \mathbf{v}(\mathbf{q})$ and that it results in collision free motion. Then for any nonvanishing and bounded scalar field $\lambda = \lambda(\mathbf{q})$ the speed field $\hat{\mathbf{v}} = \lambda(\mathbf{q})\mathbf{v}(\mathbf{q})$ results in a collision free motion too [4].

Given the plant's dynamics stated in the form of Equation (1) the main value of the inverse dynamics of the plant being modelled by the PDA map may be written as follows:

$$\mathbf{p}(\mathbf{q}, \dot{\mathbf{q}}) = \mathbf{A}^{-1}(\mathbf{q})\Big(\dot{\mathbf{q}} - \mathbf{b}(\mathbf{q})\Big), \tag{3}$$

The control signal $\mathbf{u}(\mathbf{q}) = \mathbf{p}(\mathbf{q}, \mathbf{v}(\mathbf{q}))$ can be used to solve the speed field tracking control task.

The working of the PDA map is shown in Fig. 3. The steady state diffusion field values at the GDL are denoted by σ^*. The s_i and t_i activities denote start and target activities, respectively. The geometry connections of the GDL are denoted by w_{ij}. The neuron of the INL belonging to connection w_{ij} is Y_{ij} and its output is modulated by start activity s_i. This modulation property of the model has the immediate consequences that (i) the trajectory is not explicitly represented, but (ii) instead, its distributed and indirect representation is created by means of a diffusion field, even so (iii) the plant will follow the trajectory specified by the gradient of the speed field given by the SSDF [4]. The contribution of neuron Y_{ij} to the control vector depends on its output and on its learnt connection strengths to the control units. The control units simply sum the individual contributions. At the level of the PDA map the speed field tracking task can be speeded up or slowed down simply by rescaling the speed field. As a consequence the output of the PDA map will be proportional to the velocity of the motion if the controller hierarchy is capable of following the speed field.

The PDA map provides population coding [75]. Typical coding properties are shown in Fig. 4 where a multigrid-like set of neuron layers was developed to represent a two-dimensional pixel discretized external space. The start position of the plant is in the middle of the figure, while in the different trials the target positions were set to the corners and to the centres of the border-lines. The control vectors belonging to individual interneurons are depicted at the appropriate target position (although they are situated at around the centre of the figure) and point in their own directions. The vectorial sum of each cluster of control vectors is also shown (not to scale).

5.2 The PDA map models the neocortex

The PDA map could be thought of as a crude model of the neocortex. Recognition is implicitly modelled by the PDA map when it is assumed that the position of the target, that of the obstacle(s) and that of the plant are given. The PDA map models the sensorimotor part of the neocortex as an associative direct inverse system identification unit. The PDA model of the sensorimotor part of the neocortex features the following properties:

- although it serves path planning it does not have a direct representation of the trajectory;
- the PDA map represents directional actions in a local manner;
- the PDA map sends outputs of many control units with different preferred directions at a time;
- the PDA outputs are modulated by the configuration of the joints.

5.3 The basal ganglia as a differencing entity

The main observation that helps to set up models for BG comes from the construct of the BG itself. One arm of the direct as well as one arm of the indirect pathway are shown in Fig. 1. The corticostriatal projections are glutaminergic and excitatory whereas the next two projections of the direct pathway, the striatopallidal

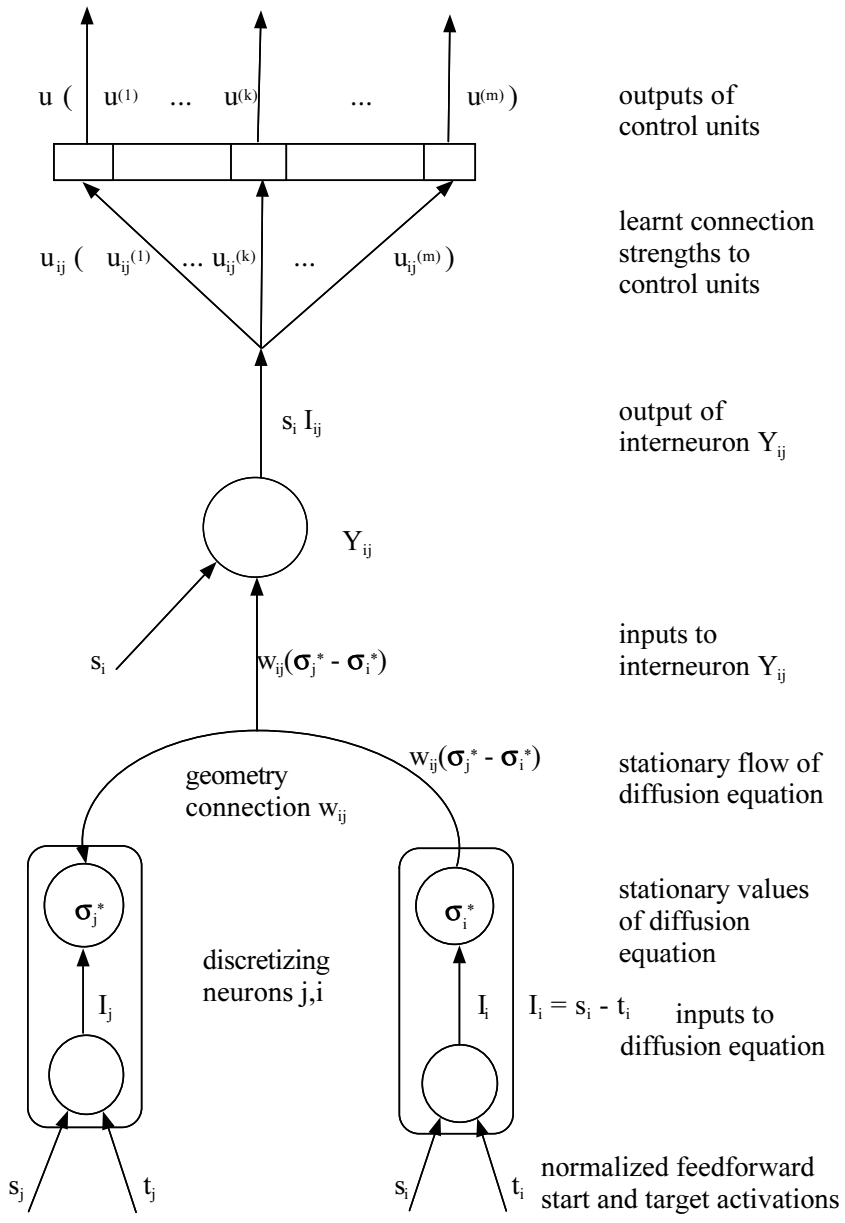

Fig. 3. Working of the PDA network

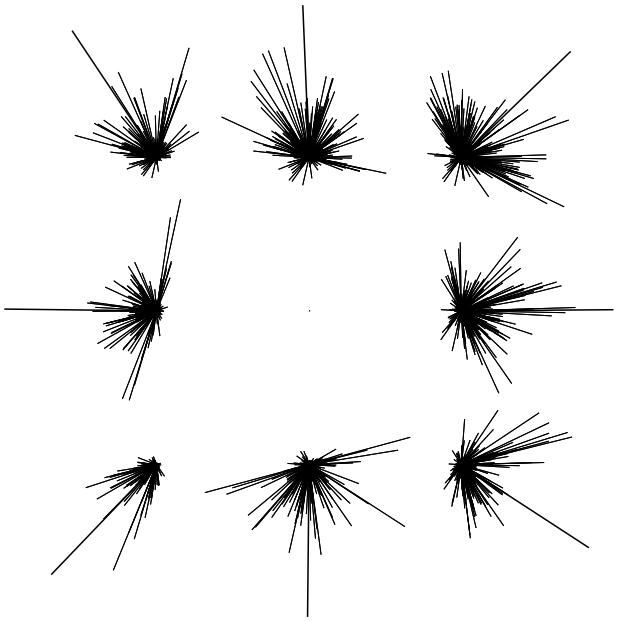

Fig. 4. Population coding properties of the PDA map

and the pallidofugal connections, are GABAergic and inhibitory. This means that activation of the corticostriatal direct pathway system disinhibits the thalamic and the brain stem regions innervated by the GPi projections. The indirect pathway originates in a distinct population of MSNs and proceeds through the GPe and the STN before reaching the GPi. Of the three sets of connections starting at the putamen and ending at the GPi, two are inhibitory, whereas the projection from the STN to the GPi is excitatory. This means that activation of the corticostriatal indirect pathway system inhibits the thalamic and the brain stem regions innervated by the GPi projections. This difference, i.e., the reinforcing action vs. the suppressing action of the direct vs. the indirect pathway, is emphasized in the literature (see, e.g., [43]).

5.4 Temporal differencing model of the basal ganglia: inertia as a perturbation

The PDA framework assumes that directional actions are identified under the assumption that the momentum is zero. Such directional actions are, however, not suitable if the momentum of the plant is non-zero. To give an example, consider the problem of moving round a circle at constant speed. If the directional action is constructed by taking the difference between the desired next position (on the circle) and the present position (on the circle) then the result is the acceleration along the path, i.e., the constant speed requirement will be spoilt. Moreover, this

action will result in leaving the desired path since the desired path requires a force orthogonal to the path. In this case, a correcting term is needed. If we assume that the direction of a move is approximately kept without further action due to the inertia of the limb then the control is needed to change the momentum. That is, the control values may be estimated as the temporal change, i.e., the approximate temporal derivative, of the PDA's desired output. In other words, assume that the PDA suggests the control action

$$\mathbf{u}_{PDA} = \mathbf{A}^{-1}(\mathbf{q})\Big(\dot{\mathbf{q}}_d - \mathbf{b}(\mathbf{q})\Big), \tag{4}$$

that may be rewritten in the form:

$$\mathbf{u}_{PDA} = \alpha(\mathbf{q})\dot{\mathbf{q}}_\mathbf{d} - \beta(\mathbf{q}), \tag{5}$$

where $\dot{\mathbf{q}}_d$ denotes the desired momentum. An approximation of the force may be gained by computing the difference between the force suggested by the PDA at time t, and the force suggested by the PDA somewhat earlier, at time $t - \tau$:

$$\mathbf{u}_{approx} = \mathbf{u}_{PDA}(t) - \mathbf{u}_{PDA}(t - \tau) \tag{6}$$

where τ is some delay. It is easy to see that the controller will accelerate the limb approximately in the desired direction provided that the directional derivatives of $\alpha(\mathbf{q})$ and $\beta(\mathbf{q})$ as well as the velocity are small. Still, this way the BG extends the domain of the PDA map to nonzero velocities.

In this temporal differencing model of the BG it is assumed that the main role of the direct and indirect pathways is to introduce (i) sign difference and (ii) delay to approximate the correct control from the output of the PDA map. The direct control effect of the PDA map (versus the control effect of the feedback channel after temporal differencing) should be small.

It is likely, however, that the correct differencing will not be achieved. The main reasons are that (i) the conditions on $\alpha(\mathbf{q})$, $\beta(\mathbf{q})$ and the velocity may not be satisfied, (ii) the temporal differencing instead of differentiation may be too crude, and (iii) the equal contributions of the two pathways cannot be guaranteed. These shortcomings will result in deviations from the speed field to be followed. These shortcoming are, indeed, properties of the BG and can be experienced, for example, in the case of cerebellectomy. The robustness and the stability of this scheme are nevertheless questionable. At the same time the eventual problem of controlling the limbs and the body and their interactions is to find the optimal control action combination out of the many possibilities for a many degrees of freedom limb system with control units that overcome in number the degree of freedom of the motion. In this respect this model of BG may provide suitable inputs (possibly in a non-optimal combination) for all the control units.

The temporal differencing model of BG has, however, biological implications since the direct and the indirect pathways are fed by different populations of striatal neurons. In order to maintain the model as a valid model of BG one has to assume that pallidal neurons receive direct and indirect inputs from the same cortical neurons (or neuronal groups or, at least, the same neuronal functional groups, where the function is defined by the PDA concept itself and may correspond to a

large set of cortical neurons due to the population coding properties of this part of the cortex [69]). The divergence–reconvergence pattern [42] may allow this feature. Beyond its limited controlling capabilities the other weak point of the temporal differencing model of BG is that the extensive divergence–reconvergence pattern is not justified by the model.

5.5 Dynamic State Feedback Control

Here we review recent developments [5] that allow one to set up another differencing scheme of BG. The approximation errors of the inverse dynamics can be viewed as permanent perturbation to the plant's dynamics. Thus we assume that instead of Equation (1) the plant follows

$$\dot{\mathbf{q}} = \hat{\mathbf{b}}(\mathbf{q}) + \hat{\mathbf{A}}(\mathbf{q})\mathbf{u}, \tag{7}$$

where $\hat{\mathbf{A}}(\mathbf{q})$ is another nonsingular matrix field. Let us first assume that we seek a static state feedback compensatory control signal, $\mathbf{w} = \mathbf{w}(\mathbf{q})$, such that the control signal $\mathbf{u}(\mathbf{q})+\mathbf{w}(\mathbf{q})$ solves the original speed field tracking problem for the perturbed plant.

This can be solved by letting $\mathbf{w}(\mathbf{q})$ satisfy the equation $\mathbf{u}(\mathbf{q}) = \mathbf{p}\left(\mathbf{q}, \mathbf{v}\left(\mathbf{q}, \mathbf{w}(\mathbf{q})\right)\right)$, where $\mathbf{v}(\mathbf{q}, \mathbf{w}) = \dot{\mathbf{q}} = \hat{\mathbf{b}}(\mathbf{q}) + \hat{\mathbf{A}}(\mathbf{q})\Big(\mathbf{u}(\mathbf{q}) + \mathbf{w}\Big)$. Although $\mathbf{w}(\mathbf{q})$ can be explicitly expressed from this it contains terms like $\hat{\mathbf{A}}^{-1}(\mathbf{q})$ and $\hat{\mathbf{b}}(\mathbf{q})$ and thus to estimate $\mathbf{w}(\mathbf{q})$ on-line is approximately the same as retaining the adaptivity of the feedforward controller. This problem can be alleviated by introducing dynamic state feedback for estimating the compensatory control signal. The simplest corresponding error-feedback law is to let $\mathbf{w}$ change until $\mathbf{w}(\mathbf{q})$ satisfies the optimality condition:

$$\begin{aligned} \dot{\mathbf{w}} &= \Lambda\Big(\mathbf{u}(\mathbf{q}) - \mathbf{p}(\mathbf{q}, \mathbf{v}(\mathbf{q}, \mathbf{w}))\Big) \\ \dot{\mathbf{q}} &= \hat{\mathbf{b}}(\mathbf{q}) + \hat{\mathbf{A}}(\mathbf{q})\Big(\mathbf{u}(\mathbf{q}) + \mathbf{w}\Big), \end{aligned} \tag{8}$$

where Λ is a fixed positive number, the gain coefficient of dynamic feedback. If the speed of the plant is measurable then Equation System (8) can be realized by a compound control algorithm. The compound control algorithm works by duplicating the PDA map in order to compute the two terms of the first equation of ((8)). The first term may be thought of as the control vector suggested by the PDA map to move the plant into the desired direction. The second term is the control vector computed by the PDA map when the experienced direction is given to it. The block diagram of the compound controller is given in Fig. 5. The compound controller was termed as the **S**tatic and **D**ynamic **S**tate Feedback Controller (SDS Controller) [5].

If $\mathbf{S}$ is a symmetric real matrix then let $\lambda_{\min}(\mathbf{S})$ be denoted by the minimum eigenvalue of $\mathbf{S}$. Let $||\cdot||$ denote the Euclidean norm and let $\mathbf{z} = \hat{\mathbf{A}}(\mathbf{q})\mathbf{w} - \Big(\mathbf{v}(\mathbf{q}) - \hat{\mathbf{v}}(\mathbf{q})\Big)$. Simple calculation yields that $\dot{\mathbf{q}} = \mathbf{v}(\mathbf{q}) + \mathbf{z}$ and thus $\mathbf{z}$ is the error of tracking $\mathbf{v}(\mathbf{q})$. In order to guarantee the robustness of tracking it is sufficient to guarantee that the error of tracking is small. Since the conditions of the theorem will be important in the discussion below, the theorem [5] will be repeated here:

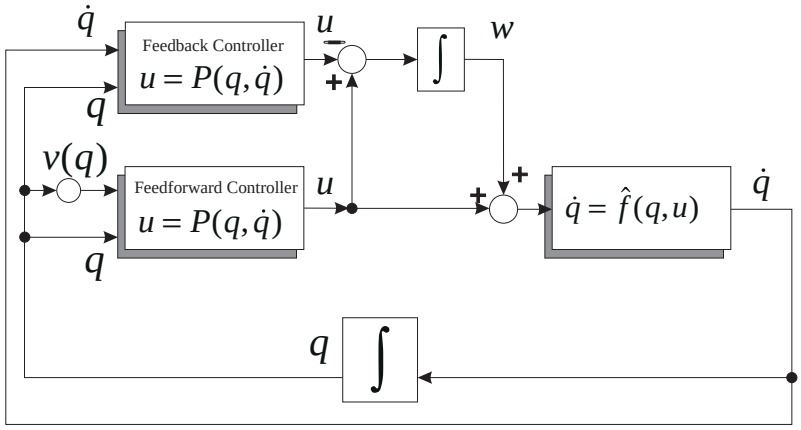

Fig. 5. Compensatory control by doubling the inverse dynamics controller. *The same inverse dynamics controller plays two roles: it is used to compute the control signal (i.e., it is used as a Feedforward Controller, FFC), as well as the compensatory signal (Feedback Controller, FBC).*

THEOREM 51 *Assume that the perturbation of* $\mathbf{A}(\mathbf{q})$ *can be decomposed as* $\hat{\mathbf{A}}(\mathbf{q}) = \mathbf{D}(\mathbf{q})\mathbf{A}(\mathbf{q})$. *Suppose that* $\mathbf{A}(\mathbf{q})$, $\mathbf{b}(\mathbf{q})$, $\mathbf{v}(\mathbf{q})$ *and* $\mathbf{D}(\mathbf{q})$, $\hat{\mathbf{b}}(\mathbf{q})$ *have continuous first derivatives and the constants* $a = \inf\{\, ||\mathbf{A}(\mathbf{q})|| \mid \mathbf{z} \in D \,\}$, $d = \inf\{\, ||\mathbf{D}(\mathbf{q})|| \mid \mathbf{z} \in D \,\}$, *and* $\lambda = \inf\{\, \lambda_{\min}(\mathbf{D}(\mathbf{q}) + \mathbf{D}^T(\mathbf{q})) \mid \mathbf{q} \in D \,\}$ *are positive. Then for all* $\epsilon > 0$ *there exists a gain* Λ *and an absorption time* $T > 0$ *such that for all* $\mathbf{z}(0)$ *that satisfy* $||\mathbf{z}(0)|| < K\Lambda$ *it holds that* $||\mathbf{z}(t)|| < \epsilon$ *provided that* $t > T$ *and the solution can be continued up to time* t. *Here* K *is a fixed positive constant and* $\mathbf{z}(0)$ *denotes the initial value of* $\mathbf{z}$. *Further,* $\Lambda \sim O(1/\epsilon)$.

We say that a perturbation of Equation (1) is *non-invertive* or *uniformly positive definite* if λ is positive. The proof is based on a modification of Liapunov's Second Method with the semi-Liapunov function $V(\mathbf{x}) = \mathbf{z}^T\mathbf{z}$. If $\mathbf{z}$ satisfies the conclusions of Theorem then it is said to admit the property of uniform ultimate boundedness (UUB).

It may be worth emphasizing that no perturbations should be reflective, in this way one can keep the error term bounded. Reflective perturbations may originate, for example, from the task (the load, the context, etc.), the approximate nature of the configuration modulated PDA map, and so on. Reflective perturbations, however, could be recognized, e.g., by the sudden growth of error when correcting. Under the conditions that (i) a recognition module is added, (ii) subsequent reflective perturbations are predictable and recognizable, and (iii) the number of reflective perturbations is limited, then the PDA map could be extended. Different options include that the recognition module performs dynamic remapping [76] on the PDA, or that the PDA has differing versions and the recognition module can select from those. Here, the robustness of the SDS model allows the second option

via the selection of the sign–proper feedback channels.

It will be argued that the putamen MSNs represent different configurations and tasks separated if the sign of feedback they need is different. These are then collected into sign-proper output channels that are less specific in regard to the configuration and the task, by the GPi.

Generalization of the first order equations is straightforward. To this end Eq. (8) is rewritten in the form:

$$\dot{\mathbf{w}} = \Lambda\Big(\mathbf{u}(\mathbf{q}) - \mathbf{u}(\mathbf{q})_e\Big) \tag{9}$$

where $\mathbf{u}(\mathbf{q})$ and $\mathbf{u}(\mathbf{q})_e$ denote the control vector suggested the PDA map and the experienced control vector, respectively. The experienced control vector, just as in the first order case, is the control vector computed by the copy of the PDA map inputted by the experienced direction. The desired direction can be given as the difference between the desired speed and the actual speed and it approximates the desired acceleration. The experienced direction can be given as the experienced acceleration.

5.6 The SDS model of the basal ganglia – thalamocortical loops

If one considers the SDS model as a model of the BG–thalamocortical loops then the first feature suggested by the model is related to the divergence-reconvergence pattern found between the part of the cortex that represents a particular body part, the striatum and the GP [39]. The need for sign–proper feedback within the SDS model requires such a pattern. Then the divergence from the cortex to the striatum may be considered as a context (task) driven sign–proper division that serves the SDS feedback loop and works in a competitive manner in order to reconverge and collect the control information into sign–properly reorganized output channel(s). In other words, depending on the context (task), the configuration, and the momentum of the different body parts the feedback may require a particular sign combination for sign–proper feedback. The SDS model assumes that the sign–proper domains of the configurational phase space are large and it might be possible to break up these domains into subdomains of different body parts that would allow for the somatotopic organization of the putamen [52]. In order to create such a sign–proper pattern the configuration of the limb (or the whole body) should be available to the neurons of the putamen.

The SDS scheme (Fig. 5) works as follows. The PDA map has two identical copies. The first copy computes the control vector that would accelerate the plant into the desired direction. The second copy computes a control vector based on the experienced acceleration. These two copies may correspond to different parts in the cortex, e.g., to the supplementary motor area and to the motor cortex, respectively [77]. The BG computes the difference of the two maps; it corresponds to the upper small circle of +/- sign in the figure. The BG's output is time integrated either in the BG itself or in the cortex and it is added to the output of the controller. The SDS scheme differs from the temporal differencing one since in the SDS scheme an external motor programme executing unit will modify the BG's output whereas in the temporal differencing scheme it will not. Since – in the case of the BG model – we are looking for a solution to govern overcontrolled systems and in this respect

we expect the BG to provide sign–proper feedback signals, the following notes are relevant:

- the BG signal may not fully disappear if the controller in charge is not the BG but an optimized controller (e.g., the cerebellum) since the BG provides sign–proper non-optimized outputs in all of the controlling dimensions;

- the BG signal in all of the controlling dimensions will be sign–proper if the optimizing controller happens to commit an error due to unexpected perturbations or to non-satisfactory tuning. In other words, the BG output may be used for launching correcting motor programmes or for tuning the systems that run these motor programmes.

Preliminary computer studies [78] show that in a large region of parameters the SDS controller is capable of controlling realistic simulations of 3D robotic arms with crude models of the inverse dynamics. The system is robust against imprecisions in the differencing, which is crucial from the viewpoint of the present model of BG.

This model of BG may imply that external optimizing systems could use the output of the BG for training. This optimization, however, may depend on many constraints, e.g., on observations whether the experienced error should be used for tuning or not, etc. That is, one may not expect the direct use of the BG's output for tuning and that may account for the lack of apparent direct connections between the thalamic recipient regions of BG's output and the cerebellum or the inferior olive [79]. Also, the SDS model requires the separate "measurement" of the control signal that corresponds to the experienced direction. This measurement, however, is an important ingredient of the direct identification procedure of the PDA map. It is one of the advantages of the SDS scheme that the doubling of the PDA map alleviates the credit assignment problem since associative direct identification is always between the experienced move and the control combination applied [5]. It is an open question whether desired as opposed to experienced control signals could be distinguished in the case of the inputs to the BG or within the BG itself.

6. Discussion

This discussion section is only qualitative. Quantitative results are currently being prepared and will be presented elsewhere [78].

6.1 Features of the SDS model

6.1.1 Bypassing computations in joint space

The first feature of the model is that it enables planning in joint coordinates to be bypassed. Instead, the planning is made in external space, say, in body centred or in somatotopic coordinates in the form of a position–and–direction–to–action map. This map is thought to be modulated by the configuration in accordance with the findings of Graziano, Yap and Gross [20]. The computational form of this modulation cannot be specified at this point. Nevertheless, the configurational modulation – instead of the discretization – of the configurational space seems attractive owing

to the relaxed need of computational units: The high dimensionality of the configurational space and the requirements on resolution for precise positioning can set very high limits on the number of discretizing neurons if the PDA map is to be constructed in configurational space. In contrast, the scheme that utilizes (i) high resolution of the external space for precise positioning and (ii) configurational modulation with low resolution to subserve a crude model of the inverse dynamics separates the two problems and may allow the use of a very crude coarse coded representation of the configuration, such as the one found by Graziano, Yap and Gross. This point needs further research.

The PDA map can be formed by the direct associative learning and it corresponds to the rough identification of the inverse dynamics. This identification may, however, be context dependent. The word context here means different tasks, including the configuration and the momentum of the plant that should be recognized due to the approximate nature of the PDA map. The configuration and the momentum are important since a particular intended move of a given segment of the limb may strongly depend on them.

6.1.2 Context modulated sign-proper reorganization in the basal ganglia

The second feature is that the divergence from the cortex to the striatum may correspond to different contexts. The SDS model of BG assumes that the striatum is divided into portions that may be activated (selected) upon the recognition of the context in order to ensure the sign–proper property. The question then arises as to whether the necessary information is available to the putamen.

The putamen receives a topographic projection from the somatosensory and motor cortex, which is in register with the physiological map [23, 25] as well as direct projections from parietal area 7b [26, 27]. The information received from the parietal cortex is not directly fed back to the cortical areas [80] in contrast to the closed loops in the case of the frontal cortex. It has been shown by Graziano and Gross that the visual space is represented by bimodal neurons as a gelatinous medium surrounding the body that deforms in a topology-preserving fashion whenever the head rotates or the limbs move [19, 20]. Such a map gives the location of the stimulus with respect to the body surface in somatotopic coordinates. Based on these findings one may assume that the configuration of the limb (or, maybe, the whole body) is available to the neurons of the putamen.

Then the divergence from the cortex to the striatum may be considered as a context driven sign–proper division that serves the SDS feedback loop and works in a competitive manner: for any task the sign–proper context should be selected. The sign-proper contexts are then processed starting at the putamen and ending at the GPi. However, this processing in the SDS model of BG involves independent processing for all the actuators. This view is supported by the findings of Nini et al. [38] that neurons of the GP do not show correlated activity. The reconvergence to the pallidum may then correspond to the partial loss or, rather, the integration of the context information since the feedback arm should serve controlling and that means recollection after context modulated remapping.

As to whether information about the context of the task is available to the

putamen Alexander and Crutcher have given a positive answer [55]. The authors show that the putamen contains cells with preparatory and with movement related activities which show limb-dependent preparatory activity, irrespective of the visual position of the target and appear as coding trajectory/kinematics variables. At the same time other cells selectively discharge to the direction of the visual position of the target.

Another observation of Alexander and Crutcher [53] reveals that cells are separated into directional cells coding trajectory/kinematics-level variables and muscle-like cells coding dynamics/muscle-level variables. According to the SDS model this information is important for selecting sign–proper feedback channels since it bypasses planning in joint coordinates and thus the questions related to joint coordinates become the questions of sign–proper feedback. Consider, for example the problem of raising a lever: the actual height of the lever, its actual speed, its desired speed, the actual position, and the actual configuration of the limb may all modify the sign of the feedback to be sent to a given control unit. This is in line with the findings of Hoover and Strick that the separation of the output channels of the BG may be due to separate processing of the higher cognitive aspects of the motion and the movement parameters [49].

6.1.3 Solving the learning/adapting dilemma

As it has been emphasized [4, 5], the SDS control scheme offers an attractive answer to the dilemma "when to switch between feedforward and feedback control methods". The dilemma arises when the learning issue is considered, since errors should result in learning. However, if both feedback and feedforward methods are taking place, then the question arises which of the systems should be trained. This problem has a simple answer if the two systems are the same and can be trained by associative learning [5].

Moreover, the feedback system allows the slow training of the feedforward PDA map since the feedback can compensate for perturbations. Then learning is needed only for frequently repeated tasks either by means of tuning the parameters of the PDA map or by recognizing the task and "allocating" a separate recognition channel. Also, the feedback alleviates the requirements against the structural precision of the feedforward controller.

6.1.4 The hierarchy of training systems

The temporal differencing SDS model of BG generalizes the striosome models of Houk [1], Montague, Dayan and Sejnowski [2], Houk, Adams and Barto [14] and Berns and Sejnowski [13]. In these works it is assumed that the striosomal modules, that have a connectivity pattern similar to that of the matrix modules, provide a temporal differencing reinforcement scheme and that the striosome of the striosomal module predicts future reinforcement. The scheme works as follows [14]: the striosomal module is made up of (i) a striosome, (ii) a dopaminergic neuron, (iii) a direct inhibitory projection from the striosome to the dopaminergic neuron, and (iv) the indirect pathway that starts at the striosome, ends on the dopaminergic neuron, passes through the STN, and has an excitatory action. The dopaminergic neurons receive excitatory signals in the case of primary reinforcement. The output

of the dopaminergic neurons reaches the striosomes as well as the matrisomes and serves as the training signal. The indirect pathway, that now does not pass through the GPe, is assumed to be faster than the direct pathway. Thus the result of the computation, i.e., the output of the dopaminergic neuron, is as follows:

$$\hat{r}_t = P_t - P_{t-1} + r_t \tag{10}$$

where P_t is the prediction of future primary reinforcement at step t, and P_{t-1} is the prediction at the previous time step. The differencing takes place at the dopaminergic neurons due to the different actions of the direct and indirect pathways and to their delay structure. The value r_t is the primary reinforcement inputted to the dopaminergic neuron and thus $\hat{r}_t$, i.e., the discharge of the dopaminergic neuron, equals the error of the prediction. This serves as the training signal for the striosomes as well as for the matrisomes.

The present temporal differencing model of BG sets up a similar framework for the matrisomes but with opposite order of delays between the direct and the indirect pathways depending on the differing architecture [58].

The SDS model of BG is, however, different. The SDS scheme may be viewed as a model that leads to a hierarchy of reinforcement learning architectures. The output of the BG is equal to the difference between the control actions corresponding to the goal oriented and the experienced directions. In each control variable there is a change of sign if these roles are exchanged. In other words, after proper tuning of the PDA map, the SDS scheme produces a sign–proper correction and/or training signal for subsequent motor programme executing units.

As was mentioned earlier, the SDS scheme cannot optimize the control actions; the optimization requires an independent system. Suppose now, that there is a system that optimizes control actions and builds up motor programmes. The output of the BG corresponds to all of the directional actions (including mouth, head and limbs) towards the goal. If a motor programme is selected then the optimizing controller should selectively and temporally inhibit the BG modulated output of the cortex to provide the optimal motor programme, This is in line with the inhibitory properties of the Purkinje cells of the cerebellum.

Moreover, for an ongoing motor programme run by the cerebellum, the output of the BG should not correct the moves since these outputs would influence all the possible control entities as they are not yet properly selected. Instead, the feedback received by the cortex, i.e., the correction signal pattern developed by the BG, serves as the source to initiate a correcting motor programme. This series of functioning is supported by the findings of Henish and Flash [81] who showed that perturbed goal oriented moves may be given the interpretation of (i) motor programmes that are initiated and cannot be stopped and (ii) superimposed correcting motor programmes that take into account the consequences of the ongoing programme.

The emerging hierarchy of training signals within the SDS scheme is then as follows:

- there is the system of primary reinforcement that trains the striosomal modules to predict future reinforcements by means of the dopaminergic neurons of the SNc;

- then, in turn, the dopaminergic neurons of the striosomal modules train (reinforce) the matrisomes to select sign–proper division of configurations for feedback action;

- the trained matrisomes of the BG send correcting signals to the cortex to train the command units of the motor programme generator..

6.2 Relationships with psychophysical findings and basal ganglia diseases

6.2.1 Selective aspects in the early acquisition of skill

Identification of sign–proper PDA configurations is the main cornerstone of the model. Should there be feedback of improper sign the move will quickly deviate from the intended direction thereby providing information about the wrong sign of the feedback. The context modulated configurational regions of differing signs should be learnt. Computer simulations also indicate [4, 5, 78] that the sign–properness may be sufficient to control the plant by means of a crude SDS controller. Should this occur, identification of the sign of the feedback will have drastic consequences. As long as the sign of one (or more) actuators is improper the motion quickly deviates from the desired directions and a new direction should be planned. The motion may then become smoother by better identifying the configurational and momentum domains differing in the sign of the feedback channels. The SDS model does not offer any special continuous tuning mechanism for the learning of the sign-proper domains. Instead, the robustness of the SDS control suggests that simple selection may be viable. Then, it is possible that the selective mechanism is a homogeneous reinforcement signal within the striatum that might explain the homogeneous responses of dopamine neurons [67] and the temporally coordinated activities of TANs [42].

These properties of an SDS controller are very much in line with the findings of Thelen and coworkers ([21] and references therein) about the learning of motion of infants. For example, the findings concerning the selective properties of the first attempts at reaching of infants demonstrate considerable variations in kinematics, kinetics and muscle patterning. The common element found in the highly variable moves was that the infants eventually got their hands to the toy. The impression was that initially the infants worked on the shaping of the force dynamics, and that further improvements in kinematics were subsequent to this primary parametrization [21]. If this primary parametrization indeed corresponds to the separation of sign-proper feedback channels according to the suggestion of the SDS model then it may be considered as a selective process [82].

6.2.2 Basal ganglia diseases

The SDS model of BG suggests particular failure modes. Simple failure occurs if the differencing goes to its extremes. In both schemes of this paper the consequences are the same. For example, if the contribution of the direct and indirect pathways is not selected properly, and/or if the selected parts do not forward (project) information properly, i.e., if the projection neurons of the striatum degenerate then the model

predicts movements beyond measure. This consequence is in agreement with the findings on Huntington's disease [83].

The other major disease of the BG is Parkinson's disease. It has been argued that the PDA map simply suggests the control action and the SDS model of BG computes a difference based on the PDA map's output. The output of the BG is then added to the output of the PDA map to form the non-optimized control action. However, the strength of the BG's effect on the thalamus is modulated by dopamine. Dopamine has different actions on the direct and indirect pathways. Dopamine increases the contribution of the direct pathway and decreases the contribution of the indirect one. If the dopamine-containing nigrostriatal tract degenerates the model predicts decrease in the positive part of the differencing (i.e., a decrease of the desired component of the motion) and an increase in the contribution of the negative part of the difference. In both schemes the net result is the slowing down of the motion. In both cases the loss of dopamine looks as if the motion were frictional. This is very much in line with one of the symptoms of Parkinson's disease, i.e., with the slowness of movements.

Parkinson's disease has another feature as well; trembling while at rest. This property is not predicted by the model. However, as has been shown by Nini et al. [38] after rendering monkeys Parkinsonian by means of MPTP treatment of the BG, it was found that the neuronal networks of the BG–cortical circuits lose their unique ability to keep pallidal neurons completely independent whereupon phase-locked oscillations appeared in the GPi output. In other words, it is possible that the trembling is a sign of overall neuronal network failure in the absence of dopamine that should be modelled at the neuronal level and it may not be captured at the level of a phenomenological model, i.e., at the present level of the differencing models of BG.

6.3 Relationships with other models of basal ganglia

In this section relationships with the EC model [6] and with the WLA model [13] are detailed. The main difference between the SDS model and the EC model, beyond their distinct use of path planning architectures, is that the SDS model bypasses planning in joint coordinates. Planning in joint coordinates seems to suffer from the problem of dimensional explosion if local representations are to be used about the joint space as is suggested by the EC model. Otherwise, the EC model has close similarities with the PDA map part of the SDS model. The SDS model should be less susceptible to dimensional explosion than the EC model, since the path planning part uses the representation of the external space or, what might be even more advantageous from dimensional considerations, the somatotopic coordinates. The dimensionality of the configurational space enters at the level of sign-properness being much less demanding than the discretization itself.

The main difference between the WLA model and the SDS model is in the assumed role of the BG. The WLA presents a decision making model whereas the SDS model is suggests an error correction model. The WLA model places the competition into the GPi and argues that the STN is the source of inhibition that leads the WLA competition. The role of the WLA competition is to select the action corresponding to the first striatal neurons reaching the threshold. The WLA

model seems to contradict the finding that the motion related neuronal signals in the SMA, the MC and the putamen largely overlap in time [53, 56]. Also, the WLA model would predict correlated acitivity at the GP that seems to disagree with the findings of Nini, Feingold, Slovin and Bergman [38]. The SDS model, in contrast, computes correcting actions. The indirect pathway serves as one set of terms of the correcting actions. The correcting control actions are built up by integration over time (Fig. 5). The SDS model assumes that decision making and movement initiation are not the roles of BG. The integrating property of the SDS scheme seems to better fit the findings of Schultz and colleagues [56, 57, 58], viz. that the activity of the SMA and the striatum may last seconds before self-initiated moves are made and the activity seems to reverberate in cortico–basal ganglia loops.

7. Conclusions

In this paper a model of the basal ganglia and the basal ganglia – thalamocortical loops is suggested that features the following properties:

- the cortex is modelled by a PDA map;
- the PDA map may be learnt by associative direct identification of the inverse dynamics;
- the PDA map represents a population coding scheme for controlling;
- the PDA map serves path planning in the external space although the trajectory is not directly represented by any part of the map;
- the PDA map and a superimposed coarse coding of limb configuration may enable planning in joint coordinates to be bypassed;
- the basal ganglia are the correcting means that derive correcting motor commands by simply computing a difference from the information provided by the PDA map. Two differencing schemes were detailed in the paper: (i) the temporal differencing scheme of the basal ganglia augments the models suggested for the striosomes [1, 2, 14, 13] and (ii) the SDS model of the basal ganglia, on the other hand, offers a hierarchy of training signals for the learning of controlling.
- The differencing models make explicit use of the distinct parallel channels of the basal ganglia by identifying those as feedback channels of sets of control neurons.
- The SDS model explains the extensive divergence-reconvergence pattern that remaps information reaching the striatum as the task dependent selection of sign–proper feedback channels.
- The robustness of the SDS model might allow the use of crude models of inverse dynamics. In particular, identification of the signs of the feedback signals modulated by the configuration might quickly improve the performance during learning and may be considered as a series of selective steps. Then

onwards the BG feedback may serve as the training means for a subsequent motor programme executing unit.

- The predictions of the model on Parkinson's disease and Huntington's disease are in line with experimental findings.

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References

[1] J. C. Houk. Learning in modular networks. In *Proceedings of the Seventh Yale Workshop on Adaptive and Learning Systems*, pages 80–84. Center for Systems Science, New Haven CO, 1992.

[2] P. R. Montague, P. Dayan, and T. J. Sejnowski. Foraging in and uncertain environment using predictive Hebbian learning. In *Neural Information Processing Systems 6*, pages 598–605. Morgan Kaufmann, San Francisco CA, 1994.

[3] T. Fomin, Cs. Szepesvári, and A. Lőrincz. Self-organizing neurocontrol. In *Proceedings of IEEE WCCI ICNN'94, Orlando, Florida*, pages 2777–2780. IEEE Inc., 1994.

[4] Cs. Szepesvári and A. Lőrincz. Neurocontrol I: Self-organizing speed–field tracking. *Neural Network World*, 6:875–896.

[5] Cs. Szepesvári and A. Lőrincz. Neurocontrol II: High precision control achieved using approximate inverse dymanics models. *Neural Network World*, 6:897–920.

[6] C. I. Connolly and J. B. Burns. A model for the functioning of the striatum. *Biological Cybernetics*, 68:535–544, 1993.

[7] C. I. Connolly and J. B. Burns. A new striatal model and its relationship to basal ganglia diseases. *Neuroscience Research*, 16:271–274, 1993.

[8] C. Cepeda, J. P. Walsh, C. D. Hull, S. G. Howard, N. A. Buchwald, and M. S. Levine. Dye-coupling in the neostriatum of the rat: I. Modulation by dopamine-depleting lesions. *Synapse*, 4:229–237, 1989.

[9] J. P. Walsh, C. Cepeda, C. D. Hull, R. S. Fisher, M. S. Levine, and N. A. Buchwald. Dye-coupling in the neostriatum of the rat: II. Decreased coupling between neurons during development. *Synapse*, 4:238–247, 1989.

[10] C. Cepeda, J. P. Walsh, N. A. Buchwald, and M. S. Levine. Neurophysiological maturation of cat caudate neurons: Evidence from in vitro studies. *Synapse*, 7:278–290, 1991.

[11] J. P. Walsh, C. Cepeda, N. A. Buchwald, and M. S. Levine. Neurophysiological maturation of cat substantia nigra neurons: Evidence from in vitro studies. *Synapse*, 7:291–300, 1991.

[12] S. P. Onn, T. W. Berger, and A. A. Grace. Dye-coupling in type I and type II striatal neurons: Alteration by apomorphine and localization to the patch/matrix. In *Proceedings of the 1991 Annual Meeting of the Society for Neuroscience*, 1991.

[13] G. S. Berns and T. J. Sejnowski. How the basal ganglia make decisions. In *The Neurobiology of Decision Making*. Springer-Verlag, Heidelberg, 1995.

[14] J. C. Houk, J. L. Adams, and A. G. Barto. A model of how the basal ganglia generate and use neural signals that predict reinforcement. In *Models of Information Processing in the Basal Ganglia*, pages 249–270. MIT Press, Cambridge, MA, 1995.

[15] A. G. Barto, R. S. Sutton, and C. W. Anderson. Neuronlike elements that can solve difficult learning control problems. *IEEE Transactions on Systems, Man, and Cybernetics*, 13:835–846, 1983.

[16] R. S. Sutton. Learning to predict by the method of temporal differences. *Machine Learning*, 3:9–44, 1988.

[17] A. G. Barto, R. S. Sutton, and C. J. C. H. Watkins. Learning and sequential decision making. In *Learning and Computational Neuroscience: Foundations of Adaptive Networks*, pages 539–602. MIT Press, Cambridge, MA, 1990.

[18] N. Bernstein. *The Coordination and Regulation of Movements.* Pergamon Press, Oxford, 1967.

[19] M. S. A. Graziano and C. G. Gross. A bimodal map of space: Somatosensory receptive fields in the macaque putamen with corresponding visual receptive fields. *Experimental Brain Research*, 97:96–109, 1993.

[20] C. G. Gross, G. S. Yap, and M. S. A. Graziano. Coding of visual space by premotor neurons. *Science*, 266:1054–1057, 1994.

[21] E. Thelen and D. Corbetta. Exploration and selection in the early acquisition of skill. *International Review of Neurobiology*, 37:75–102, 1994.

[22] L. Heimer. *The human brain and the spinal cord.* Springer-Verlag, New York, 1995.

[23] J. M. Kemp and T. P. S. Powell. The cortico-striate projection in the monkey. *Brain*, 93:525–546, 1970.

[24] H. Kunzle. Projections from the primary somatosensory cortex to basal ganglia and thalamus in the monkey. *Experimental Brain Research*, 30:481–492, 1977.

[25] S. L. Liles and B. V. Updyke. Projection of the digit and wrist area of precentral gyrus to the putamen: relation between topography and physiological properties of neurons in the putamen. *Brain Research*, 339:245–255, 1985.

[26] J. T. Weber and T. C. T. Yin. Subcortical projections of the inferior parietal cortex (area 7) in the stump-tailed monkey. *J. Comp. Neurol.*, 224:206–230, 1984.

[27] C. Cavada and P. S. Goldman-Rakic. Topographic segregation of corticostriatal projections from posterior parietal subdivisions in the macaque monkey. *Neuroscience*, 42:683–696, 1991.

[28] C. J. Wilson. Dendritic morphology, inward rectification, and the functional properties of neostriatal neurons. In *Single Neuron Computation*, pages 141–171. Academic Press, Boston, 1992.

[29] A. Parent. Extrinsic connections of the basal ganglia. *Trends in Neuroscience*, 13:254–258, 1990.

[30] G. E. Alexander and M. D. Crutcher. Functional architecture of basal ganglia circuits: Neural substrates of parallel processing. *Trends in Neuroscience*, 13:226–271, 1990.

[31] G. E. Alexander, M. D. Crutcher, and M. R. DeLong. Basal ganglia–thalamocortical circuits: Parallel substrates for motor, oculomotor, "prefrontal" and "limbic" functions. *Progress in Brain Research*, 85:119–146, 1990.

[32] A. M. Graybiel and Jr. C. W. Ragsdale. Histochemically distinct compartments in the striatum of human, monkeys, and cat demonstrated by acetylthiocholinesteras staining. *Proc. Natl. Acad. Sci. USA*, 75:5723–5726, 1978.

[33] A. M. Graybiel. Neurotransmitters and neuromodulators in the basal ganglia. *Trends in Neuroscience*, 13:244–254, 1990.

[34] C. R. Gerfen. The neostriatal mosaic: multiple levels of compartmental organization. *Trends in Neuroscience*, 15:133–139, 1992.

[35] R. Malach and A. M. Graybiel. Mosaic architecture of the somatic sensory-recipient sector of the cat's striatum. *Journal of Neuroscience*, 12:3436–3458, 1986.

[36] M. Desban, C. Gauchy, M. L. Kernel, M J. Besson, and J. Glowinski. Three-dimensional organization of the striosomal compartment and patchy distribution of striatonigral projections in the matrix of the cat caudate nucleus. *Neuroscience*, 29:551–566, 1989.

[37] L. D. Selemon and P. S. Goldman-Rakic. Topographic intermingling of striatonigral and striatopallidal neurons in rhesus monkeys. *Journal of Comp. Neurol.*, 297:359–376, 1990.

[38] A. Nini, A. Feingold, H. Slovin, and H. Bergman. Neurons in the globus pallidus do not show correlated activity in the normal monkey, but phase-locked oscillations appear in the MPTP model of parkinsonism. *Journal of Neurophysiology*, 74:1800–1805, 1995.

[39] A. W. Flaherty and A. M. Graybiel. Corticostriatal transformations in the primate somatosensory system. projections from physiologically mapped body-parts representation. *Journal of Neurophysiology*, 66:1249–1263, 1991.

[40] A. W. Flaherty and A. M. Graybiel. Output architecture of the primate putamen. *Journal of Neuroscience*, 13:3222–3237, 1993.

[41] A. W. Flaherty and A. M. Graybiel. Input-output organization of the sensorimotor striatum in the squirrel monkey. *Journal of Neuroscience*, 14:599–610, 1994.

[42] A. M. Graybiel, T. Aosaki, A. W. Flaherty, and M. Kimura. The basal ganglia and adaptive motor control. *Science*, 265:1826–1831, 1994.

[43] G. E. Alexander. Basal ganglia. In *The Handbook of Brain Theory and Neural Networks*, pages 139–144. Bradford Books / MIT Press, Cambridge, MA, 1995.

[44] G. Percheron, G. Yelnik, and C. Francois. In *The Basal Ganglia*, pages 87–105. Plenum Press, New York, 1984.

[45] G. Percheron, G. Yelnik, and C. Francois. A Golgi analysis of the primate globus pallidus: III. Spatial organization of the striato-pallidal complex. *Journal of Neural Computation*, 227:214–227, 1984.

[46] C. J. Wilson. Basal ganglia. In *The Synaptic Organization of the Brain*, pages 279–316. Oxford University Press, New York, 1990.

[47] G. Percheron and M. Filion. Parallel processing in the basal ganglia: Up to a point. *Trends in Neuroscience*, 14:55–56, 1991.

[48] C. Francois, G. Percheron, and G. Yelnik. Three dimensional tracing of individual axons following biocytin injection into the striatum of macaques. In *V-th Meeting International Basal Ganglia Society*, page 19. Plenum Press, New York, 1995.

[49] J. E. Hoover and P. L. Strick. Multiple output channels in the basal ganglia. *Science*, 259:819–821, 1993.

[50] P. Calabresi, P. Maj, and A. Pisani. Long-term synaptic depression in the striatum: Physiological and pharmacological characterization. *Journal of Neuroscience*, 12:4224–4233, 1992.

[51] J. Wickens and R. Kötter. Cellular models of reinforcement. In *Models of Information Processing in the Basal Ganglia*, pages 187–214. MIT Press, Cambridge, MA, 1995.

[52] G. E. Alexander, M. R. DeLong, and P. L. Strick. Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Ann. Rev. Neuroscience*, 9:357–381, 1990.

[53] G. E. Alexander and M. D. Crutcher. Preparation for movement: Neural representations of intended direction in three motor areas of the monkey. *Journal of Neurophysiology*, 64:133–150, 1990.

[54] M. D. Crutcher and G. E. Alexander. Movement-related neuronal activity selectively coding either direction or muscle pattern in three motor areas of the monkey. *Journal of Neurophysiology*, 64:151–163, 1990.

[55] G. E. Alexander and M. D. Crutcher. Neural representations of the target (goal) of visually guided arm movements in three motor areas of the monkey. *Journal of Neurophysiology*, 64:164–178, 1990.

[56] W. Schultz and R. Romo. Role of primate basal ganglia and frontal cortex in the internal generation of movements: I. Preparatory activity in the anterior striatum. *Experimental Brain Research*, 91:363–384, 1992.

[57] R. Romo, E. Scarnati, and W. Schultz. Role of primate basal ganglia and frontal cortex in the internal generation of movements: II. Movements-related activity in the anterior-striatum. *Experimental Brain Research*, 91:385–395, 1992.

[58] R. Romo and W. Schultz. Role of primate basal ganglia and frontal cortex in the internal generation of movements: III. Neuronal activity in the supplementary motor area. *Experimental Brain Research*, 91:396–407, 1992.

[59] W. Schultz and R. Romo. Dopamine neurons of the monkey midbrain: Contingencies of responses to stimuli eliciting immediate behavioral reactions. *Journal of Neurophysiology*, 63:607–624, 1990.

[60] T. Ljungberg, P. Apicella, and W. Schultz. Responses of monkey dopamine neurons during learning of behavioral reactions. *Journal of Neurophysiology*, 67:145–163, 1992.

[61] E. J. Neafsey, C. D. Hull, and N. A. Buchwald. Preparation for movement in cat. II. Unit activity in the basal ganglia and thalamus. *Electroencephalogr. Clin. Neurophysiol.*, 44:714–723, 1978.

[62] A. Nambu, S. Yoshida, and K. Jinnai. Discharge patterns of pallidal neurons with input from various cortical areas during movement in the monkey. *Brain Research*, 519:183–191, 1990.

[63] O. Hikosaka and R. H. Wurtz. Visual and oculomotor functions of monkey substantia nigra pars reticulata. III. memory-contingent visual and saccade responses. *Journal of Neurophysiology*, 49:1268–1284, 1983.

[64] W. Schultz. Activity of pars reticulata neurons of monkey substantia nigra in relation to motor, sensory and complex events. *Journal of Neurophysiology*, 55:660–677, 1986.

[65] J. R. Wickens. Striatal dopamine in motor activation and reward-mediated learning: Steps towards a unifying model. *Journal of Neural Transm.*, 80:9–31, 1990.

[66] P. Apicella, T. Ljunberg, E. Scarnatti, and W. Schultz. Responses to reward in monkey dorsal and ventral striatum. *Experimental Brain Research*, 85:491–500, 1991.

[67] W. Schultz, P. Apicella, R. Romo, and E. Scarnati. Context–dependent activity in primate striatum reflecting past and future behavioral events. In *Models of Information Processing in the Basal Ganglia*, pages 11–27. MIT Press, Cambridge, MA, 1995.

[68] D. Zipser and R. A. Andersen. A backpropagation programmed network that simulates response properties of a subset of posterior parietal neurons. *Nature*, 331:679–684, 1988.

[69] A. P. Georgopoulos, A. B. Schwartz, and R. E. Kettner. Neuronal population coding of movement direction. *Science*, 233:1416–1419, 1986.

[70] G. Lei. A neural model with fluid properties for solving labyrinthian puzzle. *Biological Cybernetics*, 64(1):61–67, 1990.

[71] C.I. Connolly and R.A. Grupen. On the application of harmonic functions to robotics. *Journal of Robotic Systems*, 10(7):931–946, 1993.

[72] R. Glasius, A. Komoda, and S. Gielen. Neural network dynamics for path planning and obstacle avoidance. *Neural Networks*, 1994.

[73] G.F. Marshall and L. Tarassenko. Robot path planning using vlsi resistive grids. In *IEEE Proc., Vision, Image and Signal Processing*, volume 141, pages 267–272, 1994.

[74] A. Isidori. *Nonlinear Control Systems*. Springer-Verlag, Berlin, 1989.

[75] T. Fomin, T. Rozgonyi, Cs. Szepesvári, and A. Lőrincz. Self-organizing mulit-resolution grid for motion planning and control. submitted.

[76] J. Droulez and A. Berthoz. A neural model of sensoritopic maps with predictive short-term memory properties. *Proc. Natl. Acad. Sci. USA*, 88:9653–9657, 1991.

[77] P. E. Roland, B. Larsen, N. A. Larsen, and E. Skinhof. Supplementary motor area and other cortical areas in organization of voluntary movements in man. *Journal of Neurophysiology*, 43:118–136, 1980.

[78] Cs. Szepesvári and A. Lőrincz. Robust control of a realistic manipulator by using approximate inverse dynamics models. manuscript in preparation.

[79] C. Weiss, J. C. Houk, and A. R. Gibson. Inhibition of sensory responses of cat inferior olive neurons produced by stimulation of red nucleus. *Journal of Neurophysiology*, 64:1170–1185, 1990.

[80] L. D. Selemon and P. S. Goldman-Rakic. Longitudinal topography and interdigitation of corticostriatal projections in the rhesus monkey. *Journal of Neuroscience*, 5:776–794, 1985.

[81] E. A. Henish and T. Flash. A computational mechanism to account for averaged modified hand trajectories. In *Advances in Neural Information Processing Systems 4. Part IX*, pages 619–626. Morgan Kaufmann, San Mateo CA, 1992.

[82] G. M. Edelman. *Neural Darwinism: The theory of Neuronal Group Selection.* Basic Books, New York, 1987.

[83] R. L. Albin, A. B. Young, and J. B. Penney. The functional anatomy of basal ganglia disorders. *Trends in Neuroscience*, 12:366–375, 1989.