The past 25 years have seen a resurgence of interest in the basal ganglia. In the 1980s, a series of studies yielded the now classic descriptions of basal ganglia connectivity and information flow that now dominates textbook descriptions. This was followed by the appearance of reward-based learning theories of corticostriatal function in which dopamine played a critical role in modulating corticostriatal connectivity. These advances have contributed enormously to our understanding of the basic principles of some of the circuits within these structures, and have dominated novel approaches to treatment of basal ganglia diseases. Yet even so, many of the common symptoms of Parkinson’s disease or dystonia are incompletely understood, suggesting that these “dark basements of the brain” still harbor hidden secrets.

Keywords: Basal ganglia; movement; dopamine; Parkinson’s disease; deep brain stimulation.

1. Introduction

Although the basal ganglia has a role in cognitive and emotional processing, much of our knowledge, particularly about the pathophysiology of these nuclei, has arisen from studies involving the motor system. From one point of view, these approaches have been highly positive and have led to a resurgence of interest in novel treatments for common diseases of the basal ganglia such as Parkinson’s disease and dystonia. However, from the point of view of a clinician trying to understand the symptoms of the commonest disease of the basal ganglia, Parkinson’s disease, the detailed pathophysiology often seems woefully inadequate: it may have some implications for bradykinesia but is incapable of explaining the other common symptoms of tremor, rigidity and postural instability.

2. Anatomy

The basal ganglia comprise a group of subcortical nuclei comprising the striatum, globus pallidus, subthalamic nucleus (STN), and substantia nigra. The main connections between them and the pattern of information flow between them are summarized in Fig. 1. Note that the striatum in rodents is a single structure whereas
it consists of two separate structures, the caudate and putamen in primates. Conversely, the globus pallidus and substantia nigra are each viewed as consisting of two separate structures. The internal and external segments of the globus pallidus (GPi and GPe) share some but not all of their input and output connections. The substantia nigra pars reticulata (SNpr) has similar connections to the GPi, whereas the pars compacta (SNpc) consists of a quite different population of dopaminergic cells whose axons innervate the striatum. The inputs to the SNpc are not shown on the diagram since the importance of the various known anatomical inputs is not known. However, connections from striatum (particularly caudate nucleus) and habenula may well be important.

2.1. Main connections and information flow

Despite its close involvement in movement control, the basal ganglia have no direct sensory inputs or motor outputs. The main information flow seems to be in a loop from cortex to basal ganglia and back to cortex [1]. Figure 1 shows the striatum can be regarded as the main receiving nucleus for cortical inputs whereas the GPi/SNpr represents the main output channel, via thalamus, back to cortex. Input from the cortex is excitatory and glutamatergic whereas the output is GABAergic and inhibitory. Within the basal ganglia, the striatum is connected to the GPi/SNpr via two separate sets of connections known as the direct and indirect pathways. The striatum inhibits the GPi/SNpr via the direct pathway whereas the overall effect of the indirect pathway is excitatory. Finally, note the additional cortical input to the STN: this is termed the “hyperdirect” pathway since it avoids the striatum [2, 3].

Fig. 1. Summary diagram of the major connections in the basal ganglia. Excitatory connections are shown as black arrowheads with “+”; inhibitory connections are indicated as black circles with “−”. GPe, external segment of globus pallidus; GPi, internal segment of globus pallidus; SNpr, substantia nigra pars reticulata; SNpc, substantia nigra pars compacta; STN, subthalamic nucleus; PPN, pedunculopontine nucleus; SC, superior colliculus. D1 and D2 refer to postsynaptic receptor subtypes of the neurotransmitter dopamine.
2.2. Additional connections

There are two other sets of connections to highlight in this diagram. First, although the majority of the output goes via thalamus back to cortex, there is some output to several brainstem nuclei including the superior colliculus (SC) and pedunculopontine nucleus (PPN). The former is related to the role of the basal ganglia in eye movement whereas the latter may have a role in gait and posture, and has recently been used as a target for deep brain stimulation in patients with Parkinson’s disease who suffer from postural and gait problems [4]. The second connection to note is the input and output from the midline thalamic nuclei that mimics that from the cortex.

2.3. Parallel organisation

Although the cortex innervates the striatum topographically, with prefrontal cortex projecting onto the head of the caudate and sensorimotor cortex onto the putamen, the overlapping dendritic trees of receiving neurons in the nuclei of the basal ganglia would appear to be designed to integrate information from many cortical areas [5]. Despite this, physiological studies suggest that input from particular cortical areas remains separate on its way through the basal ganglia circuits. Most authors describe four or five main circuits [1]: this article focuses on the two of them, the oculomotor loop from frontal eye fields to caudate without output back mainly to the superior colliculus, and the sensorimotor loop from sensory and motor cortical areas to putamen and thence back to cortex or pedunculopontine nucleus. In normal conditions, the information is segregated even within these pathways. For example, motor inputs from premotor, supplementary motor and primary motor areas project to different subregions of the GPi. Sensory inputs from arm and leg also remain separate. However, in the absence of dopamine, segregation breaks down and somatotopy becomes much less clear.

2.4. Striatal anatomy and dopaminergic input

Ninety percent or more of the neurons in the striatum are medium spiny neurons; the remainder are mostly cholinergic and GABAergic interneurons [6]. The spiny neurons receive cortical (and thalamic) inputs onto the tips of the dendritic spines and have axons that project to the globus pallidus. One subpopulation of neurons send axons directly to GPi/SNpr whereas the other forms the origin of the indirect pathway and projects to GPe. In addition, there is a diffuse dopaminergic input from the SNpc that terminates at the base of the dendritic spines, in a strong position to modulate the action of the cortical and thalamic inputs at the tips. Neurons of the direct pathway contain mainly D1-type dopamine receptors whereas those of the indirect pathway are mainly of the D2 variety.

GABA interneurons also receive input from cortex and densely innervate the cell bodies of spiny neurons. Their function is unknown but they could mediate a feedforward cortical inhibition of spiny neurons that might terminate initial
excitation. Cholinergic interneurons are large and are active in the absence of inputs ("tonically active neurons", TANs). They have widespread terminals and cholinergic receptors are found on spiny neurons and at dopaminergic and glutamatergic synapses.

3. Physiology of Sensorimotor Basal Ganglia

The first ideas about the role of the basal ganglia in motor control were driven by the observation that at rest, the output neurons of the GPi/SNpr fire at sustained high rates of 60 Hz or more [7]. Given that they are GABAergic, this was thought to provide tonic inhibition of the thalamus and thus withdraw facilitation from motor cortex. If inhibition was removed, facilitation would be restored and movements could occur. Observations of activity in the oculomotor loop during visually triggered saccadic eye movements were consistent with this. During fixation, activity in the part of the SNpr that projects to superior colliculus was high, but was suppressed shortly after a visual stimulus was presented in the periphery. This was followed by a burst of activity in the superior colliculus and movement of the eyes. Shortly afterwards, SNpr activity rose to baseline levels. It appeared as if appearance of the visual stimulus had released the brake of the basal ganglia and allowed the eyes to move.

However, a further experiment showed that this link was not obligatory [8]: depression of SNpr activity did not necessarily cause movement of the eyes. In this experiment, monkeys were trained to make a memory guided saccade. They fixated a central spot; a target was presented briefly to the right or left, but the monkey had to retain fixation until the central spot disappeared, at which time it had to make a saccade to the remembered position of the target. Discharge rates in SNpr declined on presentation of the target and stayed depressed until the fixation spot was extinguished and the eyes had moved to the remembered target. In this case, it was as if the depression of SNpr activity served as a form of memory of the target location. However, eye movement itself was delayed and presumably initiated by some other mechanism.

A series of later experiments in a number of different laboratories attempted to extend such observations to the control of upper limb movements. However, the results were unclear. Arm movement-related cells were found in striatum and pallidum, but their relationship to movement parameters was complex and confusing [9]. In retrospect, this may have been because the tasks studied were similar to those designed to explore primary and secondary motor areas of cortex, where there is a much closer relationship of cell discharge to movement. In the basal ganglia in contrast, there was no clear relationship of cell discharge to movement speed, extent, direction or whether the movement was slow and smooth or quick and ballistic. In fact, the discharges could appear linked to movement extent in one task but not in another. In most cases, cells changed their discharge rate after movement onset, suggesting basal ganglia was not important in the initiation of movement,
which seemed surprising in view of the difficulty that patients with Parkinson’s
disease have in starting to move. Finally, lesioning the output nuclei with kainic acid
produced little change in movement, except to slow it a little and increase the
amount of co-contraction around wrist or elbow joints [10].

The results suggested that the basal ganglia output was not essential for move-
ment, at least in the adult animals in which it was tested. Instead, it seemed to be
necessary to facilitate movement, perhaps reducing the computational overhead of
cortical areas to which it projected. Mink’s model of basal ganglia output incor-
porates this idea within a center-surround framework [11]. He proposed that the
basal ganglia output was focused spatially, and perhaps temporally such that it
would facilitate (by withdrawing ongoing inhibition) some movements and suppress
others. It did not trigger movement onset but refined cortical output patterns. This
explained why neurons sometimes seemed to discharge completely differently in
apparently very similar tasks. It also accounted for the increase in co-contraction
that had been observed when GPi output was interrupted either by kainate injection
or by electrical stimulation [10].

In the initial formulation of Mink’s model, the STN played an important role in
providing a background tonic facilitation of the GPi/SNpr, and hence a generalized
inhibitory output to motor structures. The direct pathway from striatum was viewed
as providing a focused input that suppressed GPi/SNpr neurons in highly specific
spatial patterns. This inhibitory input led to focal reductions in the inhibitory output
of the basal ganglia which were ultimately responsible for focusing activity in motor
cortex. The global role of the STN input resulted from a belief that STN projections
were distributed widely to the dendrites of GPi/SNpr neurons whereas the direct
input from striatum was much less divergent and could thus issue a focused motor
command. However, this dichotomy may not be correct, and it seems likely that
patterned activity in both direct and indirect projections would be needed to produce
a focused output as envisaged by Mink.

In summary, the basal ganglia are seen as providing a general inhibitory output to
movement. The circuitry within these structures is thought to release inhibition in a
highly selective way, both in space and time so that in any given context, appro-
priate movements are facilitated whereas others are suppressed. Importantly, the
outputs are not believed to trigger movements but only to bias the motor system
towards certain patterns of movement that are likely to be encountered as the
animal moves in its environment. The next question becomes how the patterns of
output are selected from the many inputs that the basal ganglia receive. This is the
realm of dopamine and reinforcement learning.

3.1. The role of dopamine

In the model of Mink, the patterned output from GPi/SNpr is the result of a pat-
terned input from striatum. But how this pattern is set up? A series of experiments
by Schultz and others on the role of dopamine provides a possible explanation [12].
Dopaminergic projection neurons in the SNpc normally fire at a slow rate of around 5 Hz, but occasionally produce short bursts of higher frequency activity. Schultz found that the bursts occurred when animals received an unexpected reward.

The dopamine neurons have highly divergent terminations in the striatum with each axon forming connections with many hundred striatal neurons. Thus, a burst of dopaminergic firing will lead to very widespread dopamine release in the striatum. As noted above, this input, at the base of the dendritic spines of the medium spiny neurons is in a position where it could modulate the input which arrives from the cortex at the tips of the spines. It has been postulated that a correctly timed dopamine input could change the efficiency of cortical inputs, such that unexpected rewards would reinforce immediately preceding inputs that might have led to the reward. Effectively, this is the basic premise of all striatal models of learning. It allows the striatum to learn patterns of outputs that in any given context bias movement selection towards actions that lead to reward. The striatum is seen as an essential learning machine that automatically decides (on the basis of prior experience) which movement is likely to lead to the best outcomes.

Each striatal medium spiny neuron receives input from several thousand different cortical neurons on its spines. The input from each synapse is weak and many inputs are needed to discharge the neuron. This convergent input from cortex is complemented by the divergent dopaminergic signal from SNpc. Striatal learning models suggest that patterns of input that discharge the neuron and lead to a subsequent unexpected reward will be reinforced by the dopamine burst. Thus, the striatum can be “taught” to recognize patterns of input and on receiving one of these will produce the appropriate output that is most likely to lead to reward. The cortex is therefore using the striatum to select appropriate patterns of output which reinforce behaviors which are likely to lead to reward.

It is important to note that the dopamine burst occurs with unexpected rewards but not with expected reward; indeed, if an expected reward is absent or worse than expected, the dopaminergic activity is reduced. This means that synaptic connections do not get saturated, and that the system is always seeking out the best possible pattern of behavior. The dopamine signal is therefore said to signal reward prediction error. Formal models of this sort of behavior describe what is called “actor-critic” learning. The “actor” is the striatum which is instructed by the “critic” (dopamine) when its performance could be better than it is at present and it is punished (lack of dopamine) when it performance is worse than expected [13].

There is some evidence that the D1 and D2 receptors on the direct and indirect projection neurons may play different roles in this learning scheme [14]. D1 receptors have a lower affinity for dopamine than the D2 receptors, which are thought to be more fully occupied even at basal tonic levels of dopamine firing. Thus, D1 receptors are more likely to be able to respond to bursts of activity and reinforce connectivity in the direct pathway. This would remove inhibition from outputs that lead to reward. Conversely, the D2 system might be more sensitive to withdrawal of dopamine in unrewarded trials. It has therefore been suggested that the D2 system
might operate in the opposite way to the D1 system, reinforcing inputs that are active in the absence of reward. This would mean that activity patterns in the indirect pathway, which maintain strong inhibitory output from the GPi/SNpr are reinforced by lack of reward. In simple terms, punishment (viewed as lack of reward) would be associated with patterns of activity that reduce the probability of performing the punished action.

Although dopamine neurons fire primarily in response to unexpected rewards, they can also in many standard experimental paradigms be shown to shift their firing in time such that rather than firing after presentation of reward they fire after presentation of a signal that predicts the upcoming presence of reward [12]. For example, in a remembered saccade task, a target might appear briefly on the right or left of fixation; after an interval, the fixation light might be extinguished to signal that the animal should move the eyes to the remembered target. If movements to the right are rewarded, then initially, dopaminergic neurons might fire on receipt of the reward. However, after many trials, they may shift discharge to the time of the target presentation, increasing activity when the target signals a forthcoming movement to the right. Such an activity may then strengthen preparatory activity prior to the next movement.

In fact, this type of behavior can be seen in the activity of neurons in the oculomotor circuit from caudate nucleus to SNpr and colliculus [15]. In a left/right remembered saccade task, neurons that fire in response to movements in the rewarded direction increase their firing rate when a target is presented that indicates a forthcoming movement in the rewarded direction. Conversely, they decrease their rate in response to the same stimulus if the reward is switched to the opposite direction. When the saccades are actually made following the disappearance of the fixation light, their onset latencies are more rapid for movements in the rewarded direction. The suggestion is that increased activity after a cue that indicates the next movement will be rewarded prepares the system for the forthcoming movement, such that response times are improved. Interestingly, the changes in firing pattern and onset latency are not observed in the presence of drugs that block D1 receptors.

3.2. Dopamine and saliency

Evidence supporting the role of dopamine as a reward prediction error signal is strong: the dopamine burst declines as situations giving rise to rewarded actions are repeated; bursts of dopamine can transfer to stimuli that predict the presence of a forthcoming reward; and absence of expected reward leads to reduced dopaminergic firing. However, dopaminergic bursts occur not only to reward but also, in novel environments to any unexpected stimulus. In addition, the latency can be short, around 70–100 ms after presentation of the stimulus [16, 17]. In such circumstances, there is no time for the stimulus to have been evaluated and characterized by reward. In fact, no reward may even have occurred by the time of the burst itself.
In such circumstances, it has been proposed that dopamine is signaling the presence of a salient stimulus in the environment.

It has been argued that these qualities of the dopamine signal could be used by the animal to determine the source of the salient event [16]; that is, was it caused by a prior action of the animal or was caused by an external event. If the signal was always associated with a particular behavior of the animal, that behavior is likely to have some causative action and will be reinforced by the dopamine burst. If there is no regular pattern of behavior associated with the burst, no actions are reinforced. The net result is that if the dopamine burst is signaling a salient event, rather than reinforcing action—reward, it will (less specifically) reinforce action–outcome coupling. It will build up a repertoire of knowledge that in specific circumstances, a given action is likely to lead to a certain outcome, irrespective of whether the outcome is rewarded or not.

This can be interpreted as the first stage in constructing an optimal pattern of behavior. First of all, we need to know what the consequences of an action might be, with a second stage then being to determine which consequences are most useful (rewarding). In the saliency scheme, this second stage may be controlled by later arriving signals that have had time to evaluate whether an outcome is good or bad or indifferent. Whether this occurs in a different part of the basal ganglia or in frontal areas of cortex is unknown.

At the present time, the distinction between dopamine’s role in reward versus saliency is unresolved. Although the majority of work has tackled the reward hypothesis, it is worth recalling that the experiments in which this is tested are sometimes far removed from a naturalistic setting. Monkeys may make the same movements many thousands of times, often within a highly controlled environment. Perhaps the saliency properties of the dopamine signal would be more important in natural settings.

### 3.3. Habitual versus goal-directed action

In the description above, the basal ganglia operates as a learning machine that associates particular inputs with specific outputs in order to maximize behavioral rewards. In the animal literature, such instrumental learning is often subdivided according to the strength of the link between input and behavior into what are termed goal-directed and habitual behaviors. A habitual behavior is difficult to devalue [5]. A rat might have learned to press a lever in order to obtain a reward. If the animal continues to press the lever even when it is no longer rewarded, its response to the lever is termed habitual; alternatively, if the rat gives up pressing the lever after a few unrewarded trials, it is termed a goal directed response. That is, the rat presses the lever in order to obtain a goal (reward).

These two sorts of learning appear to be represented in slightly different parts of the cortico-basal ganglia—cortex loop. In rodents, lesions of the dorsolateral striatum (corresponding roughly to the posterior putamen in primates) impair habitual
responding while leaving goal-directed responding unaffected. In contrast, lesions of the dorsomedial striatum (corresponding to anterior putamen and caudate in primates) impaired goal-directed behavior but retained habitual responding. A similar effect is seen in monkeys, where posterior putaminal lesions (inactivation with the GABA agonist, muscimol) disrupt well-learned sequences whereas anterior lesions that also involve the caudate affect the acquisition of new sequences [5].

It can be imagined that output from posterior putamen is more likely to target primary motor areas, thus influencing motor outputs more directly that output from anterior putamen and caudate which may target secondary motor areas and more frontal regions of cortex. As learning proceeds, basal ganglia output becomes more and more likely to engage direct motor outputs and provoke automatic (or habitual) responding. There is some evidence that this also occurs in humans during extensive learning of a complex finger-tapping sequence: fMRI studies show initial activation of caudate and anterior putamen which then moves more posteriorly at a stage when subjects can perform the sequence even when they are doing a second task at the same time [18].

3.4. Stopping: A role for the hyperdirect pathway?

Although the basal ganglia machinery may usually do a good job of selecting the best movement option given a particular pattern of input, there are occasions when we may need to abort the selected pattern. A green traffic light might have reinforced pressure on the accelerator pedal, but as we approach the road crossing the light may unexpectedly change to red, and we have to stop accelerating and press the brake. There is some evidence the hyperdirect pathway via the STN may play a role in this, and terminate basal ganglia facilitation of movement by generating a rapid global facilitation of the inhibitory output of the GPi/SNpr.

Experiments in monkeys have shown that the unexpected termination of a response produces a burst of activity in the pre-supplementary motor area of cortex that is then followed by activation of STN [19, 20]. If the burst of activity in pre-SMA is delayed, the expected movement fails to be suppressed.

4. The Basal Ganglia and Parkinson’s Disease

The most common disease of the basal ganglia is Parkinson’s disease, in which there is gradual death (over many years) of dopaminergic neurons of the SNpc and other brainstem areas. Initially, the disease affects dopaminergic projections to posterior putamen, and only later in the disease does the denervation become more widespread. Despite the wealth of new information about the basal ganglia summarized in the models above, the data only help explain one of the three main symptoms of Parkinson’s disease. Bradykinesia and associated problems in movement are relatively well addressed by the models, but the causes of tremor and rigidity are still mysterious.
4.1. **History**

Historically, the development of the initial anatomical model (Fig. 1) of information flow in the basal ganglia was an enormous boost for Parkinson’s disease. It suggested (on the basis of a series of animal experiments) that loss of the dopaminergic input to striatum would increase the basal discharge of the output nuclei. Indeed, this is the arithmetical result of adding up all the excitatory and inhibitory connections in the direct and indirect pathways that are affected by dopamine. In this view, the “brake” of the basal ganglia would be increased and movements would become more difficult. Experiments in Parkinsonian monkeys treated with the toxin MPTP, which destroys dopaminergic neurons, showed that there was indeed an increase in GPi/SNpr activity consistent with this model. This line of reasoning led to the reintroduction of neurosurgical lesions of the internal pallidum to treat patients with Parkinson’s disease. Finally, again according to the model, it was shown that the STN nucleus was also overactive in MPTP-treated monkeys, and that lesions here also alleviated symptoms [21], and this is now a commonly used treatment of late-stage Parkinson’s disease.

However, the results of surgery in humans themselves led to a reappraisal of the model. The main observation was that patients with Parkinson’s disease who had severe drug-induced dyskinesias (excess involuntary movements associated with changing levels of dopamine that occur after every dose of LDOPA) also improved after pallidotomy. This was unexpected since dyskinesias, or excess movements, were thought to be the result of reduced basal ganglia output. Thus, they should have been made worse not better by pallidotomy.

The result was that the rationale for surgical intervention was reassessed. The general consensus now is that lesioning the output of the basal ganglia removes a “noisy” and interfering signal from the motor system. Reducing noise allows the rest of the system to function relatively well, although not as fully as normal. Critically, this reinforces the idea that the basal ganglia is not essential for movement, but that they assist preparation for movement. This particular aspect of basal ganglia function has become the focus of much basal ganglia research at the expense of wider questions that explain symptomatology in basal ganglia diseases [22].

4.2. **Present**

The predominant feature in mild to moderately affected patients when they are studied off their normal dopaminergic therapy is slowness of movement, or bradykinesia. In addition, patients complain that moving requires a lot of effort. At first sight, this would be consistent with failure of the basal ganglia to provide automatic selection of appropriate motor plans which normally prepare the system for action [23]. Presumably patients have to replace this automatic procedure by activity from other parts of the motor system [5]. However, there are two problems with this. First, it is not obvious why movements that had already been learned should be affected by reduced levels of dopamine. After all, in the simple models above,
dopamine is only required for learning; once learned, there is no need for dopaminergic involvement. Second, if the automatic contribution of the basal ganglia has to be replaced by input from other regions, why is it that movements are predominantly slow, rather than simply being delayed?

The first question may indicate that there is a role for tonic as well as phasic levels of dopamine. Indeed, a tonic role seems highly likely in view of the constant, but slow tonic firing rates of the dopaminergic neurons of the SNpc. One possibility is that tonic dopamine controls the overall “gain” of the striatal input-output system \[12\]; when it is low, the likelihood that an input will recruit the learned pattern of output is reduced whereas if tonic levels of dopamine are high, outputs may be triggered in the presence of minimal input. Patients off therapy would fail to benefit from the automatic selection of appropriate movements, whereas when dopamine levels were restored on therapy, performance would be relatively normal. The same explanation may also account for the excess of movements that are triggered at high levels of dopamine in peak dose levo-dopa—induced dyskinesia. This may well relate to the discussion on rhythms in the basal ganglia below.

The second question is more difficult to resolve. Again, it may relate to the interest in rhythmic activity covered in the section below. However, there may be a second explanation for slowness of movement that relates to the patients’ perception of the “cost” of moving fast. In an intriguing experiment, Mazzoni et al. \[24\] asked patients with mild Parkinson’s disease to move at various speeds to targets of varying accuracy. They found that patients could move and reach all targets at the required velocity. Furthermore, when they examined those movements that met the speed and accuracy requirement, the patients’ performance was just as quick and accurate as healthy volunteers. The difference was that it took the patients more trials to achieve the criterion than the volunteers. That is, even though patients can make fast and accurate movements, they make slower movements while trying to move at the correct speed than healthy individuals.

If patients (at least in the early stages of the disease) are capable of moving quickly and accurately, why do they tend to move slowly? Mazzoni suggested that patients may be more sensitive to the energy cost of moving fast, so that they perceive it more “difficult”, or more “effortful” to move fast. For example, it could be that on successful trials, there is a smaller dopamine reward burst in patients and this slows reinforcement of the correct motor output. They have to make more movements to reach criterion than normal. Like healthy subjects, movements are more likely to be slower than criterion than faster since they are energetically less demanding.

4.3. **Rhythmic activity in basal ganglia**

Both single-cell as well as local field potential recordings in many nuclei of basal ganglia and cortex have shown that activity in the cortico-basal ganglia—cortex loops tends to be rhythmic \[25, 26\]. Populations of neurons in each area tend to oscillate in synchrony giving rise to field potential activity at a variety of different
frequencies. Recordings from deep brain electrodes in human patients with Parkinson’s disease suggests these oscillations tend to occur within two main frequency bands, broadly defined as $<30\text{ Hz}$ and $>30\text{ Hz}$. When patients are withdrawn from therapy for 12 h or so, then the predominant mode is $<30\text{ Hz}$ (usually between 15–30 Hz, termed the beta band); when on therapy, higher frequencies are more common, particularly at the onset or in preparation for movement when they shift into the gamma range (40–70 Hz).

Slow oscillations involve larger subpopulations of neurons than faster rhythms, and it has been suggested that this makes it more difficult for the basal ganglia to shift into different patterns of activity needed for correct selection of movement. Beta activity effectively reduces information capacity of the basal ganglia network. This impairs processing and will reduce basal ganglia contributions to movement.

5. Conclusions

It is clear, even in a short review such as this, that ideas about the function of the basal ganglia are still being developed. However, the predominant ideas revolve around a role in selection of movements based on learned associations with rewarding or successful events. It contrasts, for example, with current views of the role of the cerebellum, which is often seen as adapting movements on the basis of error signals from peripheral and central feedback. The basal ganglia are no longer what the British neurologist Kinnier Wilson once described as the “dark basements” of the brain; but neither have we climbed to the summit of their contribution to movement control.

References


