Error correction and the basal ganglia: similar computations for action, cognition and emotion?

Andrew D. Lawrence

The information-processing capacities of anatomically defined basal-ganglia–thalamocortical circuits have received much attention recently, leading to the development of several computational models of their function[1]. We have previously argued[2] that to understand these complex neural circuits, a strategy involving the study of patients with striatal pathology and animals with specific interventions, combined with data from functional neuroimaging and the development of information processing models, will be required. Three recent studies[3–5] have gone some way to using such strategies to help increase our understanding of the functions of these regions.

Smith *et al.*[3] have examined the motor impairment in people with Huntington's disease (HD). HD, an autosomal dominant neurogenetic disorder, is associated with neuronal loss within corticostriatal circuits, and as such provides a valuable model for understanding the role of these circuits in normal behaviour, and their disruption in disease. The most striking neuropathological changes observed in HD are found within the striatum, with GABA-containing medium-spiny striatal projection neurones bearing the brunt of the pathology[2]. However, atrophy of the neocortex becomes increasingly evident with the progression of HD, and so special emphasis should perhaps be placed on studying very early mutation-positive, but clinically asymptomatic, HD patients.

Impaired error correction in HD motor control

In their recent paper, Smith *et al.*[3] compared the motor control of patients with clinically manifest HD, asymptomatic gene carriers (AGC) and healthy controls. Participants were asked to move a two-jointed object with their arm, from a central position to one of eight targets on a surrounding circle. Healthy controls make small corrections to their initial arm trajectories as they approach the target, which means that jerkiness occurs towards the end of a movement. HD patients generally made much jerkier movements that failed to stop smoothly and accurately on the target; however, although the AGC subjects had little difficulty in calculating the initial trajectory of the movement, they had trouble implementing the appropriate corrections that are needed towards the endpoint. To examine this further, Smith *et al.* deliberately perturbed the initial trajectory of the arm movements via an occasional brief (70 ms) force pulse shortly after movement initiation, given randomly on a minority of trials. Again, the corrective movements of AGC subjects were disturbed to a far greater extent than those of healthy controls. This finding was different from that seen in a group of patients with cerebellar dysfunction, in whom the initial trajectory of movements was more irregular than normal, but the reaction to the external perturbation was normal. To explain these findings, Smith *et al.* suggested that an error-dependent feedback control process might be disturbed early in the course of HD, encompassing both self-generated and externally induced errors.

The phenomenon of feedback control has been much criticized because, it is argued, sensory feedback through the periphery is slow, and thus feedback control mechanisms are prone to instability[6]. Closed loop control need not be rejected, however, if it is appreciated that central or internal feedback can supplement peripheral feedback mechanisms. Fast internal feedback loops help stabilize feedback control systems[6]. In predictive control, a so-called 'forward model' is used to provide internal feedback of the predicted outcome of an action, which can be used before the availability of sensory feedback, helping to prevent instability[6,7]. The control signals obtained within the inner loop are sent to the periphery, and the body 'moves along in tandem'[6]. Only unpredictable components of feedback are used in correcting errors within the feedback loop through the periphery[6]. In this regard, it is of some note that in primates, striatal neurones appear to carry predictive information related to movement and reward and hence could participate in comparing motor output to an internal model or prediction[7,8]. The main output of the basal ganglia modulates the action of the thalamus, which relays sensory information to the cortex and basal ganglia[9]. This information stream, Smith *et al.* suggest, is likely to participate in error feedback control.

The notion of an error-correction dysfunction following basal ganglia damage is not a new one (although the cerebellum has most often been attributed such a role[10], a suggestion seemingly ruled out by Smith *et al.*'s results). For example, Rosvold[11] proposed that the caudate nucleus forms part of a neural mechanism for achieving error correction in the motor system, and Angel *et al.*[12] attributed some of the motor deficits observed in Parkinson's disease to slowed error correction mechanisms. In a single unit recording study of basal ganglia activity, in which animals learned a motor sequencing task, cells in the caudate fired only following an incorrect press, supporting a role for the caudate in mechanisms of error correction[13].

Error correction and the songbird basal ganglia

However, before we can attribute a role for the basal ganglia in error correction mechanisms an alternative strategy is required for making causal inferences about the functions of the basal ganglia[2]. Specific interventions in experimental animals are necessary to investigate the functions of the basal ganglia at a systems level of analysis. In an elegant study, Brainard and Doupe[4] have produced just such evidence. They found that the de-terioration in the songs of adult zebra finches following deafening was prevented when deafening was paired with a lesion of a specialized basal ganglia circuit within the song system, the anterior forebrain pathway (AFP), apparently because the lesion removed an error signal that is produced by (or routed through) this pathway. Brainard and Doupe[4]

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argued that cortical–basal-ganglia circuits might participate in the evaluation of sensory feedback during calibration of motor performance. In related work Solis and Doupe[5] argued that the AFP participates in the comparison of auditory feedback of a bird's own song to a tutor song template, and is sensitive to how well this match can be accomplished.

Impaired error correction in HD cognition

Several authors have suggested that there might be a similarity of computational operations performed by the parallel corticostriatal circuits[2,14]. In a study of cognitive function in AGC of HD (Ref. 15), we saw cognitive deficits suggestive of impaired error correction. AGC subjects were impaired on a discrimination learning task, which involved learning to make different responses to particular stimulus conditions. Specifically, participants had to learn to reliably identify, among competing stimuli, the one that exhibited a single attribute on one of various perceptual dimensions (e.g. a particular shape). HD patients were selectively impaired in discrimination shift learning, being unable to shift responding to a previously irrelevant dimension (e.g. from line to shape) following a change in reward contingencies. Follow-up experiments suggested that these deficits could be attributed to increased perseverative errors, consistent with impaired error correction[16]. In addition, in the linguistic domain, Ullman *et al.*[17] found overactive or excess '-ed' suffixation-rule use in HD patients, who showed a pronounced over-regularization rate (e.g. producing digged as the past tense of dig), and showed numerous instances of the superfluous and perseverative addition of the '-ed' suffix, as if a 'suffixing rule' was overactive or disinhibited. Both impaired discrimination learning and over-regularization can plausibly be attributed to impairments in error correction[18]. Thus, there is a striking similarity in the effects of basal ganglia lesions on action, cognition and language, with such lesions leading, in all cases, to a disruption in the capacity to modify behaviour adaptively.

Dysregulated error correction in OCD?

The basal ganglia have also been associated with several affective disorders, including depression, schizophrenia and, in particular, obsessive compulsive disorder (OCD). The notion of error correction might have particular relevance for our understanding of OCD. Pitman[19] proposed a cybernetic model of OCD (see also Ref. 20), postulating that the essential problem is mismatch between perceptual and reference (internal) signals, subjectively manifest as a sense of incompleteness (absence of consummation). This mismatch is detected by a comparator, which results in the generation of an error signal that activates a behavioural output mechanism in a futile attempt to reduce this mismatch. Gehring *et al.*[21] found evidence for exaggerated compensatory behaviour in OCD, and they also concluded that the basal ganglia, which might be overactive in OCD, implements action correction. Such mechanisms also appear relevant to the complex stereotypies associated with increased dopamine-mediated activity in the striatum[22,23]. Cools[24] applied control systems theory to the results of experimental manipulations of the basal ganglia in animals, which implicates the striatum in arbitrarily programming the ordering and sequencing of behavioural states of varying complexity. On the basis of pharmacological manipulations, Cools and van den Bercken[25] concluded that dopamine-mediated activity in the striatum increases the magnitude of error signals, leading to stereotyped behaviour. 'When the error signal remains larger than zero as a result of the intervention, […] the organism continuously displays attempts to execute the program without being successful' (p. 128).

That the striatum might play similar roles in action, cognition and affect makes good evolutionary sense. Evolution prefers to adapt old structures to new functions rather than creating novel structures from scratch. Mechanisms for detecting and correcting cognitive errors that adapt and extend mechanisms that had already evolved to support motor learning are more plausible than cognitive mechanisms with unclear or unknown evolutionary antecedents[18].

Conclusions

An important future direction will be to combine behavioural evidence of a deficit in error correction following basal ganglia damage with computational models of information processing based on the detailed neuroanatomy of these regions[1]. One particularly influential model of basal ganglia function[26] posits that striatal spiny neurones are trained by a dopamine-mediated reinforcement signal to recognize and register salient contexts and/or states that are likely to be useful in guiding behaviour. It would seem possible to apply this framework to Smith *et al.*'s results, if one assumes that match/mismatch detection is a special case of context recognition/registration/negation[2,26]. Another influential theory posits that the striatum is a central selection device[27,28]. Such a theory could also explain Smith *et al.*'s findings, if one assumes that the HD patients' errors are due to errors in selection. Schmidt[29] defined two ways that a person can make an error in achieving an environmental goal. One is called an error in selection, and the second is called an error in execution. With an error in selection, the subject's problem is that she chooses a motor 'program' that is inappropriate for the environmental situation. Correction for errors in selection requires that the subject perceives that an error in selection is being made during the course of the movement, and it requires that a new movement be executed. Schmidt estimated that errors in selection require corrections that have about a 200 ms latency, because attention is required for correcting an error in selection. In Smith *et al.*'s study[3], movements of HD patients began to become irregular 200–300 ms into their course, which would make them consistent with a deficit in correcting an error in selection, thus suggesting that the deficit seen in HD by Smith *et al.* might be part of a more general deficit in action selection.

One important area for future research will be the specification of the error correction mechanism subserved by the basal ganglia. For example, will it be based on a scalar broadcast signal, as assumed in reinforcement learning theory, or a vector, as used in, for example, supervised learning algorithms[30]?

Nevertheless, regardless of the specific computational mechanisms by which the basal ganglia can perform an error correction function, Smith and colleagues are to be congratulated for their work, which will have major implications, not just for theories of basal ganglia function, but more importantly for our understanding, and potential treatment, of HD and other devastating basal ganglia disorders.

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Error correction and the basal ganglia

Response to Lawrence (2000)

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In a recent paper we showed that motor signs of error-feedback control dysfunction were prominent through a great deal of the course of Huntington's disease (HD), even as long as 7–10 years before clinical onset of symptoms[1]. In making simple reaching movements, HD subjects displayed dysfunctional reactions to both self-generated errors and errors produced by external perturbations. As the pathology of HD is believed to be restricted to the basal ganglia early in the disease course, our findings suggest that the basal ganglia take part in or have substantial influence on the pathways through which online error correcting responses are generated.

We appreciate the parallels that Lawrence draws between our findings of error-feedback control dysfunction in HD and other error-correction deficits associated with basal ganglia dysfunction [Lawrence, A.D. (2000) Error correction and the basal ganglia: similar computations for action, cognition and emotion? *Trends Cognit. Sci.* 4, 365–367][2]. In particular, we were struck by the connections he made between the error-feedback control dysfunction in HD and the more cognitive disturbance seen in obsessive–compulsive disorder. We would like, however, to point out that the term 'error correction', which he uses extensively, is quite broad and it is important for readers to appreciate the differences between various types of error correction in digesting these parallels. We would also like to take this opportunity to draw parallels between error-feedback control dysfunction and properties of the vocal tics that occur in Gilles de la Tourette syndrome.

Two distinctly different types of processes can operate to make use of sensory information to correct errors in discrete tasks such as point to point reaching. The first is online error correction, where errors are compensated 'in flight', and the second is trial-to-trial learning, in which errors from one trial influence the motor output on subsequent trials to prevent similar errors from occurring. In our paper we analyzed online error correction, known to control systems engineers as error-feedback control[1]. This process makes almost immediate use of real-time sensory information to compensate for errors in an ongoing movement. The sensorimotor processing, transmission and actuating delays are large compared with the frequency components present in our movements[3]. For example, the arm movements in our task could be almost halfway over before compensatory motion could be generated from the first sensory information acquired after movement onset.

Because of these long sensorimotor loop delays, a simple comparison of the sensory feedback with the desired behavior and production of a proportional response would be intrinsically unstable, and could lead to wild oscillations in arm motion. Online error correction during movement may, therefore, present a formidable challenge to the central nervous system, and whatever ability

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we have to accomplish this process seems remarkable.

Our paper did not address the use of error information in trial-to-trial motor learning, but the work of Brainard, Solis, and Doupe mentioned by Lawrence strongly implicates the brain area analogous to the basal ganglia as crucial for this type of learning in the zebra finch songbird[4,5]. In recent years, trial-to-trial procedural learning has received considerable attention. When consistent force[6–9] or visuospatial[9,10] perturbations are made during movements, healthy subjects learn to compensate for these perturbations such that, with practice, they generally make movements that resemble their original unperturbed movements. If the perturbations are suddenly withdrawn, after-effect movements are produced with errors that mirror the original perturbation-induced errors. Learning during these experiments is believed to occur because the brain uses the errors experienced during a movement to adjust motor output on subsequent movements such that the error between the actual and desired trajectories of motion is reduced[11].

Although online error compensation appears to be substantially disturbed in HD, motor learning studies on HD patients have revealed that error-signal dependent trial-to-trial learning is not uniformly disturbed. Individuals symptomatic for HD show intact learning on a mirror-tracing task[12], but impaired learning on rotary pursuit tasks[12,13]. Because both of these tasks require error-dependent, error-correcting learning, the above findings suggest that a dichotomy exists within error-dependent learning. This dichotomy may exist between learning feedforward and feedback control. Rotary pursuit involves long continuous movements, which are largely under closed-loop feedback control. Learning to perform this task requires the ability to modify parameters of an on-line feedback control system. By contrast, mirror tracing of polygons involves multiple, short, discrete movements, which rely more heavily on open-loop feedforward control processes. To perform this task well requires trial-to-trial learning that modifies the commands generated for each movement segment. These segments are largely under feedforward control. Therefore, in HD, motor learning tasks that are largely under feedforward control (e.g. mirror tracing) might be well learned, while tasks that largely stress online feedback control (e.g. rotary pursuit) can display impaired learning.

Preliminary results from our laboratory support this hypothesis. We studied learning to make point-to-point reaching movements in a force field, a task that strongly depends on feedforward predictive processes. In this task a viscous curl force-field perturbs movement in a consistent manner perpendicular to the motion direction over several blocks of trials[14]. This force-field induces a stereotypic pattern of errors, which is reduced with practice in control subjects. We found that neither presymptomatic nor symptomatic HD subjects displayed impaired learning in this task. Both the rates of learning and the magnitude of the learning related effects on performance of this error correcting task were quite similar between groups. These results, taken together with the previously mentioned motor learning studies in HD patients, demonstrate that information from motor error signals can in some cases be used to promote normal learning in HD while in other cases error-signal dependent motor learning appears to be disturbed. This suggests that it is the type of the motor-learning task (i.e. whether the task largely requires modification of feedforward predictive motor commands or instead mainly depends on the adaptation of on-line, feedback-dependent motor responses) and not the general involvement of error signals or error correction that is likely to be the key factor predicting the task performance of HD subjects. To understand the role that the basal ganglia play in motor control and learning, more research is needed to delineate clearly which types of learning are impaired and which are intact in HD and other diseases of basal ganglia function.

In his commentary, Lawrence drew important parallels between dysfunctional online error compensation in our motor task, and the exaggerated compensatory behavior characteristic of obsessive–compulsive disorder (OCD). In OCD, errors (real or imagined, such as one's hands being dirty), draw an exaggerated response (such as compulsive washing). This is quite similar to the inappropriate, often exaggerated, responses that we found to motor errors in HD, whether these errors were extrinsically or self-generated. As Lawrence pointed out, basal ganglia dysfunction is believed to underlie OCD, and so patients with OCD might be expected to manifest some cognitive parallel of the error-feedback control dysfunction in HD. This would suggest that the basal ganglia play an important role in online error compensation for both lower-level motor, as well as higher-level cognitive, processes.

Obsessive–compulsive behaviors occur both in Tourette patients and their family members[15], and there is likely to be a shared genetic basis between Tourette syndrome and some cases of obsessive–compulsive disorder[16,17]. Tourette syndrome is characterized by the occurrence of vocal and other involuntary motor tics. The vocal tics include short comprehensible expressions that often include swear words. Swear words and other common complex vocal tic expressions[18] such as 'shut up', 'stop that', and 'okay honey' can be classified as interjectional, responsive or emotional speech[19–21], as opposed to propositional speech, which makes up the majority of verbal discourse. This division of speech bears a striking resemblance to the feedforward/feedback partitioning of motor control processes that we used to study the motor disorder in HD.

Evidence that propositional and non-propositional speech are generated differently in the brain comes from studies with aphasic patients[19–21]. These patients can lose the ability for propositional speech entirely, or this speech may be labored and misarticulated. Meanwhile, non-propositional speech can be preserved, well-articulated, and situation-appropriate in these patients. The preserved utterances in aphasia often include swear words, 'yes'/'no' responses, and counting. Further evidence that the verbal tics might indeed be 'feedback' as opposed to 'feedforward' in nature, include findings that the pre-movement EEG potential seen in voluntary speech is absent during verbal tics[22], and the observation that verbal tics occur more frequently at pauses in speech and at points of high indecision than at other times[23,24]. This closely parallels our findings in HD that gross end-movement jerkiness is more likely to occur during trials with larger initial errors in motion. The pathology of Tourette's syndrome is not yet understood, but it is believed to involve the basal ganglia[25,26], and neurotransmitters involved in basal ganglia function such as dopamine[27]. This suggests that Tourette syndrome, obsessive–compulsive disorder, and Huntington's disease might all be manifestations of dysfunctional error-feedback compensatory processes caused by disease affecting basal ganglia function.

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Go with the flow

In a recent article, Wann and Land cast rhetorical aspersions upon the idea that optic flow is used to guide human locomotion[1]. They were especially critical of the notion that the perception of heading (one's instantaneous direction of translation) from optic flow is required for locomotor control in general, and for the task of steering a curved trajectory in particular. Their argument strikes us as full of straw, for to our knowledge no one has actually proposed that heading is required for all locomotor tasks, and researchers have already identified other variables that could be used to steer a curved path to a goal. In this reply we urge the gentle reader not to be swayed by the authors' disputation, and we bring to bear some empirical evidence that rules out their data-free conjectures.

Consider first the significance of optic flow. The authors approvingly cite Harris and Rogers' statement[2] that there is no compelling evidence that optic flow plays a significant role in the control of locomotion on foot. To the contrary, Warren *et al.*[3,4] have shown that when the heading direction specified by optic flow is offset from the walking direction in a virtual environment, it strongly influences the path taken to a target. The data reveal that both optic flow and the visual direction of the target contribute to locomotor control, but the former increasingly dominates as flow and motion parallax are added to the display. This has recently been confirmed in open-field experiments using displacing prisms to offset the flow[5,6]. Another experiment conducted in our laboratory demonstrated that both heading judgments and joystick steering are biased by a moving object in the same manner[7]. These results strongly imply that heading is not merely a '*post-hoc* percept', as Wann and Land suggest, but plays a functional role in guiding locomotion on straight paths.

Next consider the authors' portrait of a heading hegemony. They are surely correct that linear heading is not analytically required for steering a curved path, but then no one has argued that it was. Indeed, a number of alternatives, some based on other flow properties, already exist in the literature[8–11]. Wann and Land favor two purportedly heading-less strategies, one based on the visual direction of the goal (θ) and the other on 'the raw retinal flow'[12], (although they fail to cite the prior publication of the retinal flow theory by Kim and Turvey a year ago[13]). We hasten to point out, however, that θ is defined as the visual angle between the target and heading (Fig. 1), so perceived heading actually *is* required by their visual direction theory. Although Wann and Land argue that heading in a vehicle might be given by the center of the windscreen or a hood ornament, this is clearly a special case. Heading on foot might be given by pedal proprioception, but it is also influenced by optic flow; and heading is not given in numerous other activities (bicycling, skating, skiing, diving, sailing, flying…). Optic flow offers a proven solution, for heading can be accurately judged from flow, on both straight and curved[14] paths. Even the authors later admit that heading from optic flow could be used to specify θ.

We believe that different locomotor tasks are likely to involve different informational variables and task-specific control laws. The point is that this is an empirical matter, and the authors' hypotheses should be regarded as such until they are tested experimentally. As it happens, one of us has recently carried out such tests for steering a curved path[15]. The data