panied by reduced ERN amplitudes (142, 504–506). In contrast, in another patient group with large pMFC lesions, no impairments in error- and conflict-induced performance adjustments were found (171), except for slowed error corrections in a flanker task (360). This suggests that pMFC lesions interfere with the solution of response conflict, because fast error corrections result from concomitant activation and sequential execution of the incorrect and correct responses (496, 533). In line with this, TMS of the pre-supplementary motor area (pre-SMA) affects processing of competing response tendencies (512) and intracranial stimulation of the pre-SMA in monkeys replaced automatic incorrect responses by slower correct responses (270). Moreover, patients with pMFC lesions showed increased false alarm rates in a two-back task associated with higher subjective confidence in action selection (518). This seems to be in line with findings that macaques with sulcal anterior cingulate lesions show increased impulsivity and reduced impact of reinforcement history in choice behavior (289). In sum, the pMFC appears involved in and necessary for rapid online adjustments, evaluation of confidence with choice, and between-trial adjustments. Lesions of the pregenual and subcallosal ACC have been shown to result in strongly reduced ERN amplitudes (499) and difficulties in conflict-induced trial-by-trial adjustments (142). These conflict-driven sequential adaptations seem particularly impaired when stimuli had an emotional component such as face expression (333).

Different subregions of the lateral prefrontal cortex (LPFC) have been implicated in maintaining and updating task representation, goals, and contextual information (51, 139, 551), and in exerting top-down control in mutual interaction with the pMFC (291, 331). Its necessity for performance monitoring is strongly suggested by the fact that ERN generation is impaired in patients with LPFC lesions comprising the middle and inferior frontal gyri, anterior insula, and underlying white matter: the amplitudes of ERN and CRN did not differ from each other (199, 535, 538). Whereas post-error slowing appeared unaffected, in some patients error corrections were diminished (199), particularly when white matter tracts connecting LPFC, pMFC, and the striatum were affected (535). The importance of the fiber connections at the base of the middle and inferior frontal gyri is also reflected in a reduced ERN in patients with isolated lesions in that area (245).

Whereas patients with lesions of the posterior orbitofrontal cortex (OFC) extending into the subgenual ACC and the ventromedial prefrontal cortex (vmPFC) showed severe attenuation of the ERN and error corrections (520), more anterior OFC and frontopolar lesions did not interfere with performance and ERN generation in a flanker task (538). Nevertheless, vmPFC and OFC play a major role in motivated and adaptive behavior, particularly with respect to establishing stimulus-outcome associations, credit assignment, task and reinforcement history, and value-guided choice. These aspects will be discussed in section V*D* on monitoring-based learning processes.

B. Subcortical Circuitry

Cortico-striato-thalamic circuits are highly important for action selection and learning. They can thus be expected to be an essential part of the performance monitoring network. The basal ganglia are closely connected to the cortical areas involved in performance monitoring and adaptive behavior, such as pMFC, LPFC, vmPFC, and OFC. Particularly, spiraling connections from the striatum to the dopaminergic midbrain and back, as well as nonreciprocal cortico-thalamic projections appear important in connecting the different basal ganglia loops (215). Thus the subcortical circuitry discussed below is involved in performance monitoring by receiving and sending information about outcome predictions, aversive and appetitive outcomes, prediction errors, and adaptive signals from and to the relevant cortical areas.

The necessity of the basal ganglia in performance monitoring is demonstrated by the finding that focal and isolated damage in the striatum disrupts the generation of the ERN (535). Performance monitoring is also impaired in degenerative diseases of the basal ganglia. Parkinson's disease (PD) is characterized by a gradual loss of dopaminergic neurons that progressively affects dorsal and later ventral striatal function. PD patients quite consistently display reduced ERN amplitudes (162, 498, 579; but see Ref. 257), but no significant effects of medication on the ERN have been observed. Less is known about the FRN in PD, although behavioral studies show an imbalance of preference versus avoidance learning depending on whether the patients are medicated or not (187, 188). Generally, the FRN might be affected later in the course of disease progression than the ERN because the ventral striatum, relevant for reward processing, seems to be affected by dopamine depletion only at more severe stages of PD (104, 105).

Huntington's disease (HD) is an autosomal dominant neurogenetic disorder characterized by striatal degeneration. In HD patients, a consistent reduction of the ERN was found that was related to the genetic mutation and the reduction of gray matter volume in pMFC (37, 38).

Therapeutic high-frequency deep brain stimulation (DBS) opens up the opportunity to investigate the effect of stimulating subcortical brain regions. In a patient treated for intractable alcohol dependence, bilateral DBS in the nucleus accumbens over 12 mo reduced clinical symptoms significantly reflected in subpathological clinical scores at the end of the observation period. In addition, DBS nearly doubled the ERN amplitude in a flanker task (315). This effect was reversible when stimulation was temporarily turned off and

replicable over several measurements. Notably, behavioral performance remained unaffected. It suggests fast functional interactions of the ventral striatum and the medial frontal ERN generators.

Studies in non-human primates suggest that the aMCC receives inputs from the basal ganglia circuitry mostly via the ventral anterior (VA) and ventrolateral anterior (VLa) nuclei of the thalamus (226, 543), a region which also integrates cerebellar circuitries assumed to be involved in motor control (262). In addition to the VA/VLa nuclei the mediodorsal (MD) thalamus sends fibers to the pMFC (483, 557). Compared with healthy controls, patients with thalamic lesions show impaired conscious error detection, post-error slowing, post-error accuracy improvement, and smaller error-related ERPs (410, 471). A detailed analysis revealed a double dissociation within the thalamus: lesions affecting VA and VLa abolish the ERN while leaving the Pe nearly intact. In contrast lesions encompassing MD only slightly decrease the ERN amplitude and completely wipe out the Pe (299, 471). This suggests a differential involvement of thalamic nuclei in different stages of performance monitoring.

The subthalamic nucleus (STN) is an important modulatory part of the cortico-striato-thalamocortical circuits. It has been suggested to modulate cognitive control and decision making by providing a general NoGo signal unspecifically inhibiting motor response tendencies under situations of high response conflict and uncertainty (14, 183). Indeed, high-frequency DBS in the STN for PD results in more impulsive decisions (reflected in shorter reaction times in high-conflict conditions) in decision making and more errors of commission in simple motor Go/NoGo tasks, respectively (17, 80, 187). It has been suggested that evaluative signals stem from pMFC via a hyperdirect pathway and that STN then implements motor inhibition to slow down reactions on conflict-laden decisions (183), thereby allowing to accumulate additional information useful for more specific adaptation. This might also apply to errors; in section VC we present evidence for involvement of the STN in post-error slowing. However, DBS in STN seems to yield paradoxical effects dependent on the stage of decision making and response selection: fast impulsive errors appear disinhibited, but slow selective inhibition of incorrect responses is improved at later stages (590). The effect of DBS on impulsivity appears to also depend on the subtype of PD (591). In any event, the STN appears to use performance-monitoring signals to on-line modulate motor behavior. In line with this notion, depth recordings from STN revealed conflict- (80) and action-outcome-related signals that were related to motor adjustments on subsequent trials (62) and modulated by motivation (313).

The habenular complex (Hb) is another key subcortical structure for performance monitoring and adaptive behavior. It is part of a major top-down pathway from forebrain to midbrain modulating the activity of dopaminergic and serotonergic brain stem nuclei (230, 231, 241, 324). Neuroimaging in humans and invasive recording in animals showed that the Hb codes negative prediction errors (59, 259, 344, 454, 532). In animals, electrical stimulation of the lateral Hb strongly inhibits firing of dopaminergic neurons in the ventral tegmental area (VTA) and substantia nigra pars compacta (SNc) (88, 277, 344) and of serotonergic raphé neurons (567). Conversely, Hb lesions result in increased striatal and prefrontal DA turnover (327, 381). Thus the Hb seems to influence approach-avoidance decisions, impulsivity, and acute pain- and error-induced remedial actions; in particular, it has been suggested to regulate motor suppression induced by these diverse situations (241). Support for this hypothesis comes from the finding that electrical stimulation of the lateral habenula after rewarding saccades can delay subsequent saccadic eye movements (343). Similarly, in gerbils, stimulation of the lateral habenula and VTA, respectively, had opposite effects on the acquisition of avoidance behavior (487).

IV. PSYCHOLOGICAL AND NEUROBIOLOGICALLY INFORMED MODELS

A. Mismatch Theory

Early evidence on nearly immediate error corrections, post-error slowing, and post-error improvements in accuracy suggested a genuine error detection mechanism (10, 11, 428–430). The discovery of the ERN triggered the proposal of the mismatch theory of error detection. It assumes a comparator detecting a discrepancy (i.e., mismatch) between the representations of the executed and the correct actions, which then is reflected in the ERN and error-related aMCC activity (101, 165, 166, 198, 536). On the basis of behavioral observations, the early onset of the ERN which sometimes even precedes the erroneous motor response, and a single-case study showing a normal ERN in a patient with sensory deafferentation (6), it has been argued that the representation of the executed response cannot be derived from proprioceptive feedback but rather from a central efference copy of the motor program. The representation of the correct action directly results from ongoing, even after response elicitation continued perceptual processing and stimulus-response mapping according to the task rules. In line with this view, the ERN is often seen on premature erroneous responses executed before completion of stimulus-response mapping. Factors that affect the representations of either executed or correct action reduce the amplitude difference between ERN and CRN, reflecting disturbance of the comparison process (101, 460). On the basis of continuous flow of information accounts of stimulus-response mapping (208), it has been assumed that the action comparator issues its signal when the motor efference copy

arrives and uses whatever information about the correct response is available at this stage (101). The mismatch theory has been extended to explain the FRN occurring when external feedback information is available (356). Here, the mismatch between representations of the intended outcome, derived from reward prediction, reinforcement learning, and forward modeling, and the actually achieved outcome gives rise to the error signal driving the pMFC activity. Limitations of the mismatch theory are that it is, at least in its original form, not mathematically formalized and that it cannot explain pMFC activity and N2 modulations on correct actions involving response competition and/or higher effort.

B. Response Conflict Monitoring

A broader perspective on cognitive control suggests that not only erroneous actions but also difficulties during ongoing actions can initiate adjustments. This has been formalized in the response conflict monitoring theory (46), which suggests that the performance monitoring system monitors for response conflicts between concomitantly evolving response tendencies rather than for errors per se. Response conflict arises when action implementation simultaneously activates two or more incompatible response tendencies. This may occur when distractors or apparent regularities of the stimuli prime a prepotent but incorrect response. A strong correct response tendency may override incorrect response tendencies. In these cases, the highest overlap of the competing response tendencies occurs before the (correct) response, thus yielding preresponse conflict. If, however, a strong and prematurely executed incorrect response tendency results in an error, response conflict reaches its maximum after the response. Continuously incoming information from stimulus processing is translated into a rising correct(ive) response tendency which conflicts with the executed error. Thus errors in reaction time tasks are typically associated with postresponse conflict.

Computational models have allowed simulations and predictions for numerous task situations which have been compared with empirical evidence (46, 47, 595). Briefly, response conflict signals are assumed to recruit cognitive control processes and to initiate appropriate adjustments. Connectionist parallel distributed processing models of various stimulus-response compatibility tasks (159, 488, 489) form the basis of the response conflict monitoring theory. The models usually comprise three layers of units: an input layer with units representing stimulus features, a response layer consisting of a unit for each response, and an attentional layer with units corresponding to each feature of the stimulus set. The information flow is realized by bidirectional excitatory weights between layers. Competition is elicited by within-layer inhibitory links between the units. The measured response conflict depends on the relative activation levels of the competing response units and is computed by a multiplicative measure, the Hopfield energy (46, 260). A conflict monitoring feedback loop is assumed to reflect the contribution of the pMFC to performance monitoring and initiating adjustments of control. When a stimulus pattern is fed to the input units, activations flow via their connections to the response units. If a response unit crosses an arbitrary response threshold, the corresponding response will be produced by the model, allowing to predict reaction times. When one response unit is active and the others are inhibited, conflict is low or zero. When two or more response units are active, however, the response conflict is large. Adaptation is brought about by feeding back the conflict signal to the attentional layer where it drives attentional refocusing on task-relevant features and/or to the response unit where it modulates response threshold (46, 595). The response conflict model quite accurately predicts behavioral effects of conflict on current and subsequent trials (46, 125, 495, 496).

Numerous neuroimaging studies have demonstrated that the pMFC is engaged when response conflict occurs (20, 44, 53, 76, 77, 291, 348, 353, 439, 536, 545, 570, 604). Also ERP findings, such as the modulation of the stimulus-locked N2, ERN and lateralized readiness potentials by differential stimulus conditions, attentional and pathological states can be explained by conflict monitoring models for the according tasks (125, 495, 593, 595, 597). The N2 is assumed to be modulated by pre-response conflict, whereas the ERN reflects mostly post-response conflict. Strong post-response conflict signals may serve as a reliable basis for internal error detection, rendering a specialized error detection mechanism unnecessary (595). It was furthermore shown that conflict-related pMFC activity varies with the amount of conflict at the response level but not with conflicts at other levels such as stimulus identification (148, 353, 547).

The response conflict monitoring theory has repeatedly been challenged and is still under debate (63, 64, 72, 210, 211, 496, 594). Moreover, the theory cannot account for feedback-related activity in the pMFC that occurs in the absence of motor response activation.

It is noteworthy that invasive studies in monkeys did not find any neurons whose firing patterns coded response conflict (271, 373, but see Ref. 482 for single unit activity in human aMCC associated with response conflict). Perhaps the discrepancy between the ease at which conflict-related signals in pMFC can be measured at the mesoscopic (multicellular) level using EEG and fMRI and the difficulty to find single neurons coding response conflict could be explained by biophysical cortical network attractor models that have been applied successfully to sensory and value-based decision making in parietal and ventromedial prefrontal cortices (265, 278, 585, 586). A substantial number of pMFC neurons show activity specific to a particular action or action sequence from the available action set (423,