can be drawn through the consequences of errors and other performance problems. In the 1960s, Rabbitt (429, 430) described typical behavioral changes after errors in speeded choice reaction time tasks: immediate error corrections by a second key press and response slowing after errors. Sometimes performance was also found to improve on trials after errors (316, 317), and error signaling (pressing a specific button whenever an error was encountered) was introduced to study conscious error detection (427, 431, 432).

Also in the 1960s, the first physiological evidence for a performance monitoring system in the human brain was reported by Bechtereva and colleagues (27–30), who by means of depth electrodes found increased neuronal activity in caudate and putamen specifically after errors but not after correct responses in psychological tests, which they interpreted as activity of "error detectors" of the brain.

1. Invasive electrophysiology

Single-unit recordings in the anterior cingulate cortex (ACC) of monkeys revealed that a subset of neurons specifically increased firing rate after errors and after omission of reinforcement on correct trials (380) (for anatomical details and nomenclatures used in this review, see **FIGURES 2** and **3**). A study on motor learning identified transcortical field potential changes in macaques' ACC area 24 at the ventral bank of the cingulate sulcus that were associated with erroneous and unrewarded movements (201). More recent experiments using a saccade countermanding task in which monkeys had to withhold prepotent prosaccades to a visual target upon occurrence of a rare stop signal extended these findings (271, 459, 500). This task allows the study of error-related activity on incorrectly executed saccades (errors of commission) and activity related to response conflict, which arises by competition between the impending prosaccade and the intention to withhold it. This response conflict (see sect. III*B* for a detailed discussion of this concept) was reflected by simultaneous activation of movement and fixation neurons in the frontal eye fields and superior colliculus. It was shown that neurons in the dorsal bank of the anterior cingulate sulcus (in the rostral cingulate motor area, CMAr) responded to errors (271). Half of these neurons were also responsive to omission of re-

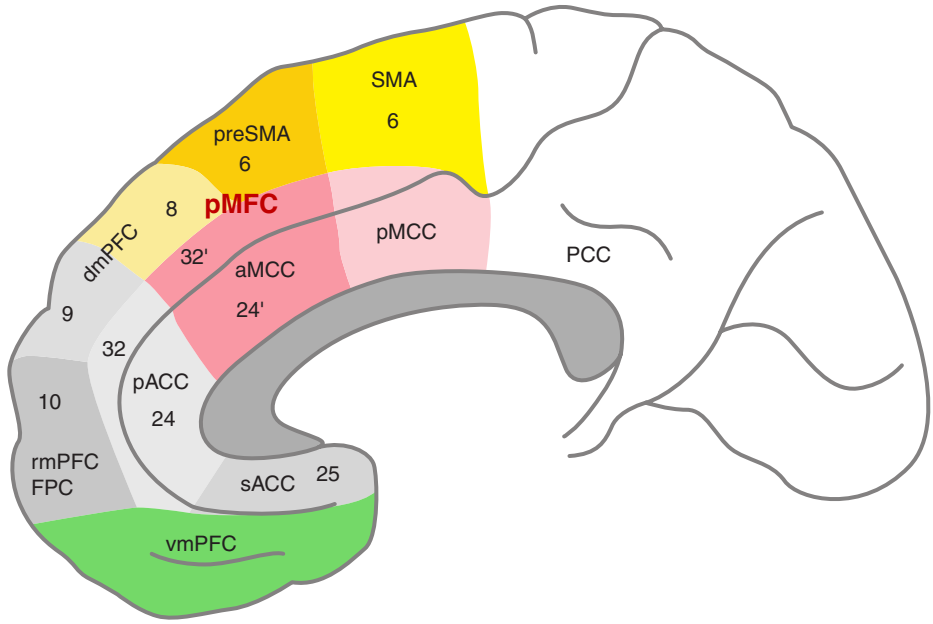

FIGURE 2. Subregions of the medial frontal wall. Based on cytoarchitectonical and functional criteria, the medial frontal cortex can be divided into cingulate and isocortical areas. The cingulate cortex has been divided into anterior, middle, and posterior portions, which are further subdivided into the subcallosal and pregenual anterior cingulate cortices (sACC and pACC, respectively), the anterior and posterior midcingulate cortices (aMCC and pMCC, respectively), and the posterior cingulate cortex (PCC) (556). Another terminology of the regions around the dorsal section of the cingulate sulcus has been suggested in an early meta-analysis attempting to link human cortical areas to their monkey's homologs (412, 413): the anterior and posterior parts of the rostral cingulate zone (RCZ), largely overlapping with the aMCC, are assumed to be the homolog of the monkey's rostral and ventral cingulate motor areas (CMAr, CMAv), respectively; and the caudal cingulate zone (CCZ) might correspond to the monkey's dorsal cingulate motor area (CMAd). The isocortical regions can be divided into ventromedial, rostromedial, and dorsomedial prefrontal cortex (vmPFC, rmPFC, dmPFC, respectively) and the presupplementary and supplementary motor areas (preSMA and SMA, respectively) (160). Hemodynamic signals in neuroimaging experiments often coactivate several neighboring regions such that the descriptive term posterior medial frontal cortex (pMFC) has been used to commonly refer to pre-SMA and aMCC, as well as adjacent SMA, posterior dmPFC, and pMCC (439). The numbers refer to cytoarchitectonic areas. FPC, frontopolar cortex.

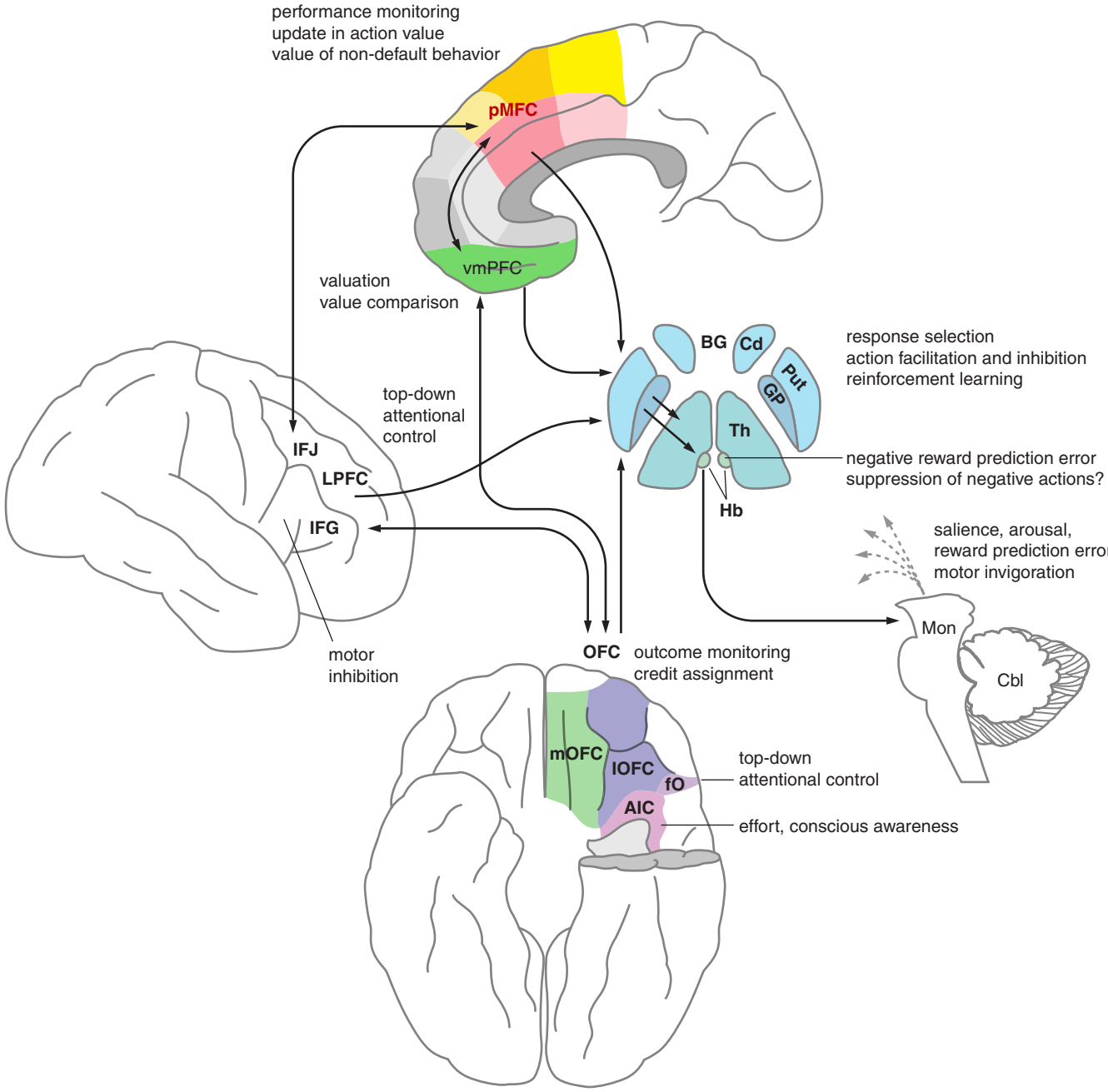

FIGURE 3. Important brain structures involved in performance monitoring and adaptive behavior, their function, and interaction. pMFC, posterior medial frontal cortex (see **FIGURE 2** for details); vmPFC, ventromedial prefrontal cortex; LPFC, lateral prefrontal cortex; IFJ, inferior frontal junction; IFG, inferior frontal gyrus; OFC, orbitofrontal cortex; m/lOFC, medial/lateral OFC; AIC, anterior insular cortex; fO, frontal operculum; BG, basal ganglia; Cd, caudate nucleus; Put, putamen; GP, globus pallidus; Th, thalamus; Hb, habenula; Mon, mesencephalic and pontine monoaminergic nuclei; Cbl, cerebellum.

wards. A different subset of ACC neurons specifically responded to earned and unexpected reward. Whereas none of the recorded ACC units showed activity related to response conflict, the supplementary eye fields contained distinct neuronal populations that were responsive to errors, reinforcement, or response conflict, respectively (500). In contrast, studies in humans undergoing cingulotomy for medically intractable psychiatric disorders reported neurons sensitive to response conflict in the anterior midcingulate cortex (aMCC), the putative homolog of the monkey's CMAr (127, 482).

A combined single-unit recording and inactivation study showed that the monkey's CMAr does not only respond to undesired action outcomes but is also involved in subsequent adaptive changes in action selection (485). In a modified response reversal task, the reward magnitude was gradually decreased on correct actions until the monkey

switched to the alternative action, which then became the rewarded response. Interestingly, 37% of CMAr neurons specifically increased firing *1*) after the reward was reduced and only *2*) before the monkey selected an alternative response on the subsequent trial. When the CMAr was reversibly inactivated by the injection of the $GABA_A$ agonist muscimol, the monkeys showed perseverance even when the reward was considerably reduced and spontaneous response switches in trial series with constant maximal reward. Nearly identical results were found in intraoperative single-unit from recordings the aMCC in humans undergoing surgical cingulotomy (580). Thus aMCC activity seems to translate changes in action outcomes into the need for adaptive behavior. This view is supported by findings that CMAr neurons encode action sequences and multiple feedbacks during exploration (423, 424). This activity ended as soon as enough information was accumulated to adapt behavior and exploit the gained knowledge. Action outcomes are informative with respect to the necessity of adaptation when they deviate from expectation, which can be expressed formally as reward prediction error (RPE). Several studies have demonstrated that CMAr neurons code RPEs (7, 288, 346, 455). Other variables needed for action valuation and assessing the need for ongoing and future adjustments also appear to be coded by anterior cingulate neurons, for example, reward expectancy and reward proximity (342, 484) as well as effort (111, 243, 244), often in an action-specific fashion. In addition to CMA, adjacent supplementary motor area (SMA) and pre-SMA also contain neurons that code evaluative signals such as expected outcome, actual outcome, RPE, and surprise (458).

While single-unit recordings in humans are performed rather rarely, more evidence on anatomical structures involved in performance monitoring has been collected using intracranial local field potentials (LFPs) reflecting postsynaptic activity. Error-related LFPs were reported in the ACC, aMCC, lateral frontal and mesial temporal cortices, and the amygdala of epilepsy patients (55, 56, 421) and in the dorsal ACC and supplementary eye fields of monkeys (154, 155). This suggests that performance monitoring signals are available throughout a distributed network rather than a single "error detector" region, but the electrophysiological responses to performance problems have been reported most consistently in the aMCC. A study using arrays of microelectrodes (566) in the human aMCC showed larger source currents with inhibited firing (in superficial layers) for errors compared with correct responses and for negative compared with positive feedback. Interestingly, source currents were also increased for novel stimuli and with increasing task difficulty. The authors suggest active inhibition in superficial cingulate layers based on these findings. The neuronal activity from deep layers where the efferents of the ACC arise was not recorded, however.

2. Event-related potentials

In the 1990s, performance monitoring research was significantly boosted by the discovery of error-related event-related brain potentials (ERPs) in the scalp-recorded EEG making the monitoring process observable by noninvasive techniques **(FIGURE 4)**. The error-related negativity (ERN) or error negativity (Ne) was discovered in response-locked ERPs on errors in speeded choice reaction time tasks (165, 198). It is a frontocentrally distributed negative deflection starting around the time of the response and reaching its peak between 50 and 100 ms later. When referenced to averaged mastoids, ERN amplitudes can reach up to 15 μV at the electrodes FCz and Cz. It is preceded by a positive deflection peaking 50 to 0 ms prior to the response and followed by another positive deflection which peaks 100–250 ms after the response. This sequence of frontocentral positive and negative deflections has raised the notion of an oscillation time-locked to the erroneous response in the theta band (5–7 Hz). This theta power increase is larger on errors than on correct trials and has been observed in averaged and single-trial data (79, 136, 328, 330, 379, 599, 600). The question whether the ERN might result from a power increase and/or phase-locking of theta oscillations is still under debate (592). The frontocentral positive deflection directly following the ERN (also called early error positivity, see below) is somewhat broader than the ERN which is in line with findings of an error-specific increase in delta power (1.5–3.5 Hz; Ref. 599). The ERN is independent of stimulus and effector modality, i.e., independent of whether visual, auditory, or tactile stimuli are presented (166, 213) and independent of whether manual, foot, eye-movement, or vocal responses are given (193, 194, 250, 340, 376, 539, 574). Electrical source analyses consistently suggest the ERN and its magnetoencephalography (MEG) equivalent to be generated in the posterior mesial frontal cortex (pMFC), specifically in the aMCC (137, 213, 250, 287, 355, 536). A recent MEG study (1) suggested additional involvement of the posterior cingulate cortex (PCC). In a study with simultaneous EEG and functional magnetic resonance imaging (fMRI), we found a trial-by-trial correlation of the ERN amplitude with the hemodynamic response in the aMCC (136). Moreover, studies in monkeys suggest a close link between error-related LFPs in the anterior cingulate cortex and the scalp-recorded ERN (154, 204). Taken together, source localization findings and invasive recordings in humans and monkeys provide overwhelming evidence that the ERN is generated in the pMFC with a focus in the aMCC. Further research is needed to elucidate the significance of the recently reported additional activity in PCC (1, 83). Various functional interpretations of the ERN will be discussed in section III.

Whereas in young healthy participants correct responses only sometimes elicit a clear response-locked negativity in the raw ERPs, a negativity similar to the ERN, usually of smaller amplitude, is often seen in older subjects and pa-