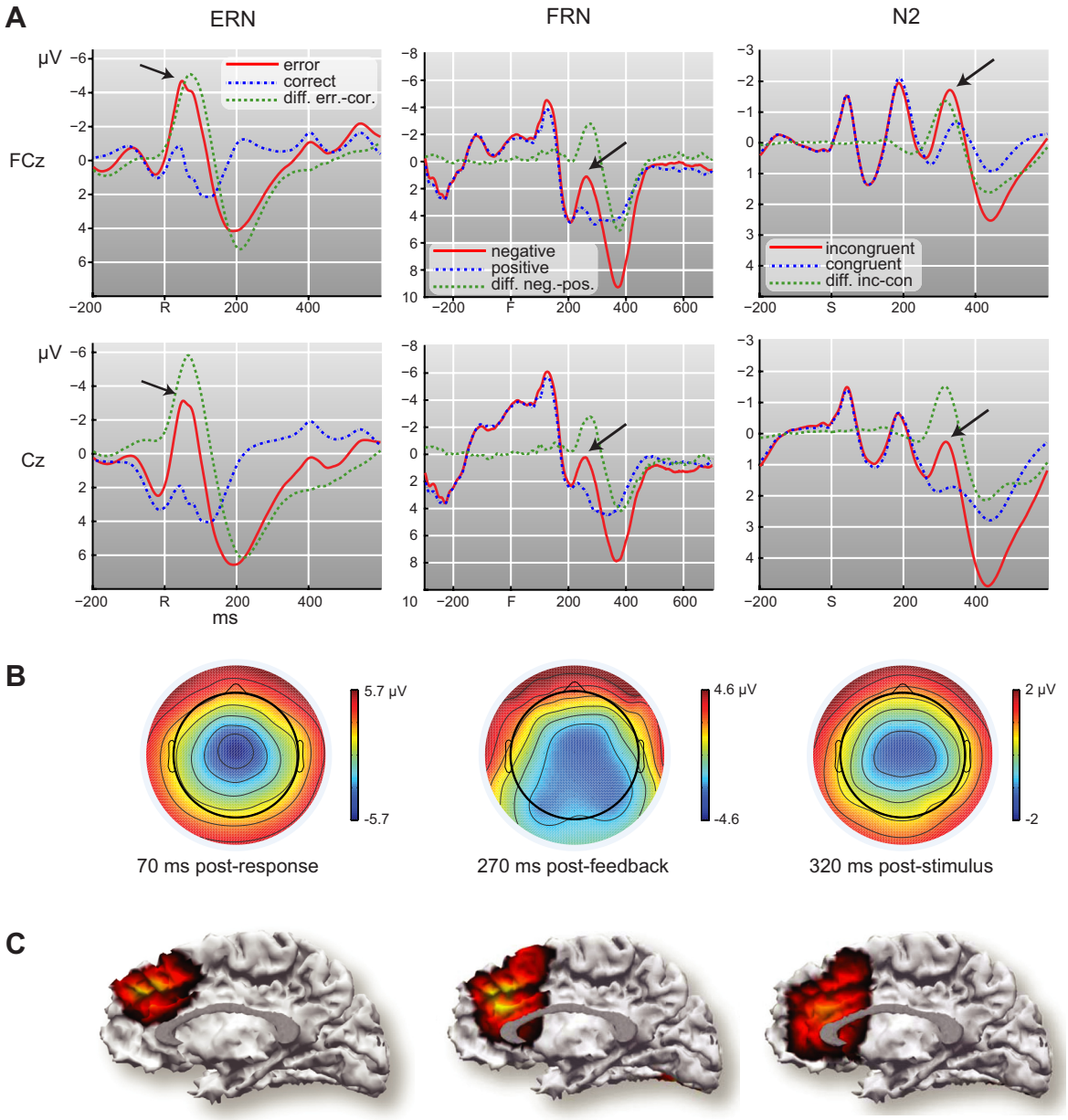

FIGURE 4. Event-related potentials associated with performance monitoring. *A*: prototypical ERPs in response to errors (ERN, response-locked), negative feedback (FRN, feedback-locked), or preresponse conflict (N2, stimulus-locked) at midline electrodes FCz and Cz. *B*: corresponding topographies of difference waves from *A*. *C*: source localizations ERN, FRN, and N2. R, response; F, feedback; S, stimulus. [From Gruendler et al. (213).]

tients, the so-called correct-related negativity (CRN) (181, 199, 538). In young participants it can be revealed by Laplacian transforms of the EEG data (553, 554) which filter out low spatial frequencies masking the focal CRN activity. Recent studies using blind source separation by temporal independent component analysis (ICA; Refs. 335, 336) strongly suggest that ERN and CRN stem from the same or largely overlapping generator structures (202, 441, 573). A temporospatial principal component analysis (PCA) revealed two temporospatial factors contributing to both ERN and CRN (156). In agreement with the ICA findings, a central factor was found that was sensitive to response correctness and task difficulty. A more frontal negative factor was present for all responses and was not modulated by accuracy and difficulty. It has been suggested that the CRN reflects partial error processing on correct trials with high uncertainty about the action outcome, for example, due to stimulus ambiguity (101, 401, 460). Notably, partial error execution observable by isometric muscular activity of the incorrect response hand is not a necessary prerequisite for the CRN (553). Rather, the amplitude of the response-related negativities CRN and ERN seems to reflect the objectively accumulated evidence in favor or against having made an error (574). It has been suggested

that both, ERN and CRN, signal the need for adjustments (22). This is consistent with the finding that the CRN amplitude is smaller on trials preceding errors and the resulting notion that it is involved in keeping up resources (effort) allocated to the task at hand (5, 219, 437). The above-mentioned finding that the CRN is larger and easier to detect in older subjects and some patient groups has not been explained unambiguously, yet. Perhaps several factors such as higher motivation (and thus higher effort invested in the task), higher uncertainty, as well as delay and increased jitter of other onging EEG activity with the potential to mask CRN activity contribute to this effect.

As shown in **FIGURE 4**, the ERN is followed by positive deflections specific to error trials, collectively referred to as error positivity (Pe) (165, 166). The frontocentral positive deflection following the ERN is sometimes called "early Pe"; a positive slow wave at parietal electrodes sustained from 300 to 500 ms post response is the classical Pe, sometimes called "late Pe" (546). Dipole localization studies suggest the early Pe to be generated by the same generators as the ERN, whereas parietal cortex and rostral ACC seem to contribute to the late Pe (232, 546). However, these results have to be interpreted with caution due to methodological limitations such as low electrode number and localization based on grand mean average activity. The functional significance of the Pe is still rather unclear. It appears to reflect the accumulated subjective evidence for an error associated with conscious perception of the error (398, 497, 526, 574). Furthermore, it seems to share features with the P300 observed on infrequent target stimuli in oddball tasks (12, 126, 438).

The ERN occurs in speeded reaction time tasks, in which stimuli usually convey the necessary information to unequivocally determine the correct response (given enough processing time). Therefore, errors can be detected without external feedback. In many situations, however, we select our actions under significant uncertainty. In such underdetermined situations, errors can only be detected based on external information about the action outcome provided by the sensory systems. Negative feedback provides evidence to the performance monitoring system that the action was not successful. In a time estimation task, in which participants had to press a button within a small, adaptive time window around 1 s after an imperative stimulus, only feedback can provide sufficient information whether the response was given at the correct time or not. Such time estimation tasks reliably elicit a negative ERP deflection with a frontocentral scalp distribution similar to the ERN (213, 252, 356), the feedback-related negativity (FRN; also called feedback-ERN or feedback negativity). This negative deflection is usually greater on negative than on positive feedback, occurs independently of feedback modality, and peaks approximately between 200 and 300 ms after the feedback. It was also found in gambling tasks and reinforcement learning tasks (200, 249). The FRN amplitude is higher on more unexpected outcomes and was suggested to scale with the RPE, i.e., the difference between predicted and obtained action outcome (84, 96, 249, 252, 255, 375). In contrast, other studies suggest that the FRN amplitude is only influenced by the valence of the outcome in a quasi-binary fashion and that the magnitude of the violation of outcome expectancy is reflected in the amplitude of the subsequent feedback-related P300 deflection (216, 251, 411, 598). Moreover, the FRN is modulated by task context such as the range of possible outcomes (254) and whether attention is directed to the utilitarian (gain/loss) or performance information (error/correct) contained in complex feedback (377). Interestingly, the FRN is also elicited when an unfavorable action outcome requiring behavioral adjustments cannot be attributed to the actor but results from external factors, e.g., technical malfunction (202). Interpreting FRN results requires attention to several caveats. Studies differ substantially in FRN quantification (e.g., difference waves vs. single-condition ERPs, theta-band filtering), baseline correction, and how they deal with the overlap with the subsequent P300 deflection. Moreover, the FRN occurs in the time range of the N2 family of ERPs (180) and may therefore be masked by other factors influencing the N2 (248). A recent systematic review of FRN studies synthesizes the above-mentioned findings and concludes that the FRN represents quantitative RPE, is elicited by outcomes as well as outcome-predicting stimuli, changes (along with behavior) with learning of stimulus-outcome contingencies, and is maximally engaged by volitional actions (563). A recent study comparing EEG activity elicited by real outcomes (reward/punishment) and by fictive outcomes (based on feedback indicating what would have happened had one decided differently) revealed a double dissociation (177). Real outcomes elicited a frontocentral FRN reflecting an RPE and a subsequent frontocentral P3a. In contrast, no FRN or P3a was found in the fictive condition; instead, an earlier occipitoparietal outcome correlate was found. About 400 ms after feedback, brain activity for both conditions converged into a common adaptive pathway, reflected in parietal P3b-like activity that was predictive of future choice behavior. The absence of the FRN for fictive feedback is surprising as even the observation of mistakes made by others elicits similar EEG activity (see further below). It may result from the fact that fictive feedback was given, when participants decided not to actively select a stimulus, which is in line with the notion that the FRN is maximal for volitional motor actions (563).

The initial assumption that ERN and FRN are functionally equivalent (249, 356) has found strong support by electrical source localization (213) and blind source separation using ICA (202), suggesting that the ERN and FRN stem from the same or at least highly overlapping generator structures in the pMFC.

In addition to own, self-generated errors, humans can also detect when other persons make mistakes, adjust their own behavior accordingly, and learn to avoid similar mistakes. The observation of mistakes committed by others elicits a frontocentrally distributed negative ERP deflection similar to the FRN to visual feedback (134, 302, 354, 544). Source localization of this observed-error-related negativity (oERN) again hints at the pMFC. It appears, however, that the observed error must entail a consequence to the observer along a reward-punishment axis to elicit the oERN, because the mere counting of errors in everyday behavior did not evoke ERN-like activity (130). A recent study in macaques revealed that neurons in the pre-SMA and cingulate sulcus increased activity on observed errors associated with omission of the reward delivery to both the observing and acting monkeys (601). Whereas a subset of these neurons did also respond to unexpected reward delivery when the monkey was acting, the overlap with pMFC neurons responding to own errors was negligible. Activity of neurons responsive to observed errors in the cingulate sulcus appeared furthermore to be associated with subsequent behavioral adjustments of the observing monkey.

The stimulus-locked N2 deflection comprises a family of ERP responses which, in part, are related to performance monitoring and cognitive control (180). Simultaneous activation of competing response tendencies is associated with increased frontocentral N2 amplitudes (306, 546). Particularly in the framework of the response conflict monitoring theory discussed further below, a functional equivalence of the stimulus-locked N2 on correct high-conflict actions and the response-locked ERN has been suggested (125, 593, 595). Frontocentral N2 modulations are also found during inhibition of preactivated motor responses in GoNogo and stop signal tasks (180). It has been argued that the N2 increase in GoNogo tasks can be explained by a conflict between executing and inhibiting a response (146, 158, 378). A comparison of scalp distribution and results of source localization suggested a large overlap between N2, FRN, and ERN (213). In addition to a pMFC source found for all three ERPs, an additional N2 source was found in the right inferior frontal cortex, which is in line with other findings suggesting an involvement of this area in motor inhibition (14, 15). Intracranial recordings also showed conflict-related theta-band activity coupled with frontocentral surface N2-like EEG activity (97).

Salient and surprising events such as novel stimuli are also associated with modulations of the stimulus-locked N2-P300 complex (110, 180, 420, 492). Recently, it has been shown that ERN sources estimated with ICA can explain a substantial part of novelty N2 activity, suggesting overlapping sources of brain activity related to errors and valence-free surprising action outcomes (573). In a number of studies the FRN appears to be more driven by unexpectedness of an outcome than by its valence (145, 173, 248), also suggesting that this ERP wave is to some extent susceptible to valence-free expectancy violation (i.e., surprise). This is in line with recordings from the aMCC homolog in monkeys showing neuronal activity coding valence-independent prediction errors (227, 346) and novelty of stimuli (347).

In sum, invasive recordings in rodents and primates have consistently demonstrated pMFC responses coding various pieces of information needed for action valuation and evaluation such as reward expectation, reward proximity, reward delivery, errors, surprise, effort, and observed errors. Thus pMFC neuronal activity appears to integrate multiple decision parameters using a common valuation currency (288). These information contents appear to also modulate scalp-recorded ERPs related to performance monitoring such as the ERN, FRN, oERN, N2, Pe, and P3. It appears, however, that a one-to-one matching of type of information and ERP is not possible, although these ERPs are modulated differentially.

B. Functional Neuroimaging

As a result of its high spatial resolution, functional neuroimaging has been particularly influential in performance monitoring research. In line with the electrophysiological findings, fMRI signal increases related to various evaluative signals have been found in the pMFC with great consistency (for reviews and meta-analyses, see Refs. 296, 374, 439, 534). For example, hemodynamic signals related to errors in speeded forced choice task (76, 196, 256, 290, 292, 536); to conflict between competing response tendencies (44, 76, 195, 291, 352, 536, 545); to decision uncertainty, outcome predictability, and volatility of the environment (33, 406, 407, 558, 559); to feedback and punishment (256, 297, 339, 361, 532, 565); to reward, reward anticipation, and reward prediction errors (282, 300, 301, 397); to the observation of errors committed by others (131, 478); to externally induced failure to achieve action goals (528); and to valence-free surprising action outcomes (573) have been reported. Furthermore, pMFC activity has been shown to correlate positively with effort and negatively with the subjective action value discounted by effort (121, 422). This fits with the finding that in two-choice decisions an inverse value difference signal was found in aMCC. The hemodynamic signal increases when the difference between the values of the choice options decreases, i.e., when value comparison becomes more difficult and time-consuming (24, 224).

Furthermore, appreciation and expression of negative emotions and acute pain, observation of others experiencing pain, and social exclusion are associated with signal increases in this region, too (152, 160, 318, 475, 555). These latter situations have in common with the performance monitoring signals that they are evaluative and may trigger adaptive actions (530). This notion has been addressed and

elaborated in a recent review demonstrating overlapping activation in aMCC to pain, negative effect, and cognitive control resulting in the adaptive control hypothesis (475). Briefly, this hypothesis suggests that the aMCC "represents a hub, where information about pain, and other, more abstract kinds of punishment and negative feedback could be linked to motor centers responsible for expressing emotion on the face (a communicative means of adaptation; note from the authors) and coordinating aversively motivated instrumental behaviours" (p. 156; Ref. 475). We will return to this hypothesis and reconcile it with other theories in section IV.

Cyto- and receptor architecture as well as connectivity data suggest that the pMFC is subdivided into different regions (31, 284, 404, 405, 556). It is conceivable that these different anatomical subregions, although often coactivated during performance monitoring, serve at least partly dissociable functions. On the basis of electrophysiological data, functional subregions differing with respect to the motor effects of stimulation have been identified in the pMFC of monkeys (413). In humans too, several studies have suggested that the different evaluative signals found in the pMFC are coded in distinct subregions, which has caused an ongoing debate. Although a somatotopy for motor actions has been described for the anterior and posterior rostral cingulate zone (413), the distribution of conflict-related fMRI signals appears to be unaffected by effector modality (19). Some studies, however, showed that response errors predominantly elicited aMCC responses, whereas response conflict on correct trials was more associated with increased activity in the more dorsal mesial area 8 and pre-SMA (53, 195, 534, 536) or, more generally, that pre-response performance problems are coded more dorsally than post-response performance problems (439). In contrast, a more recent analysis showed a somewhat different distribution along an anterior-posterior and inferior-superior axis (374). Other studies found localization differences of response conflict (more anterior) and degrees of freedom in choice of a volitional action (more posterior) within the pMFC (370) (see also Refs. 182, 565). Moreover, it has been suggested that the different subregions of the pMFC might play different roles in the initiated adaptations. Increased arousal and autonomic responses (115, 118, 408) as well as reactive, general cognitive adjustments appear associated with aMCC activity (294, 527). In contrast, proactive, task-specific adjustments may be associated with pre-SMA activity increases (242, 294, 527). Most of the above-mentioned studies were not designed to reveal subtle regional differences and suffered from spatial smoothing and the fact that group analyses ignore individual anatomical variations. Thus, at this point, we have to state that evidence for the suggested subregional dissociations is still too sparse and equivocal to extract a clear functional distribution within pMFC. Therefore, future research aiming at a functional parcellation of the pMFC needs to better take into account individual anatomy, use high-resolution imaging, and combine functional data with structural, e.g., tractographic data. Recent reports suggest that individual patterns of pMFC gyrification influence performance monitoring functions as well as their ERP and fMRI correlates (8, 267).

During performance monitoring, numerous other brain regions are usually coactivated with the pMFC. Most consistently, the anterior insula, parietal cortex, lateral prefrontal cortex, and subcortical regions such as the basal ganglia, thalamus, and midbrain are reported (296). Reward and reward prediction error-related signals are also found in many cortical and subcortical areas, as will be discussed further below.

With the use of separate and simultaneous EEG/fMRI recordings, correlations between the hemodynamic response in the aMCC and scalp-recorded ERN and FRN amplitude, respectively, have been found within and across participants (1, 73, 136, 266, 341). More negative single-trial ERN amplitudes were associated with stronger aMCC responses and increased reaction times on subsequent trials (136).

III. THE PERFORMANCE MONITORING NETWORK: CAUSAL EVIDENCE

Based on the above-discussed electrophysiological and imaging findings and anatomical studies on the connectivity of the pMFC, a basic network of brain structures typically involved in performance monitoring can be derived **(FIGURE 3)**. The causal involvement of the nodes in this performance monitoring network can be addressed in lesion studies in animals and in patients with circumscribed lesions resulting from stroke, tumor excision, traumatic brain injury, etc. Furthermore, stimulation of the network constituents using transcranial magnetic stimulation (TMS) or in patients undergoing deep brain stimulation (DBS) can reveal functional interactions.

A. Cortical Regions

As discussed above, correlative evidence suggests that the pMFC is essential for performance monitoring. Focal lesions of the pMFC, in particular of the aMCC, are rare, and resulting impairments may be transient and can disappear in chronic stages (99). The small number of patients and studies, the uncontrollable size of lesions often extending far beyond pMFC, and the limited amount of behavioral and neurophysiological variables that could be obtained from these patients render the results hard to interpret. Despite these difficulties, a series of studies in patients with unilateral focal lesions of the aMCC demonstrated impairments in post-error and conflict-driven adaptations, accom-