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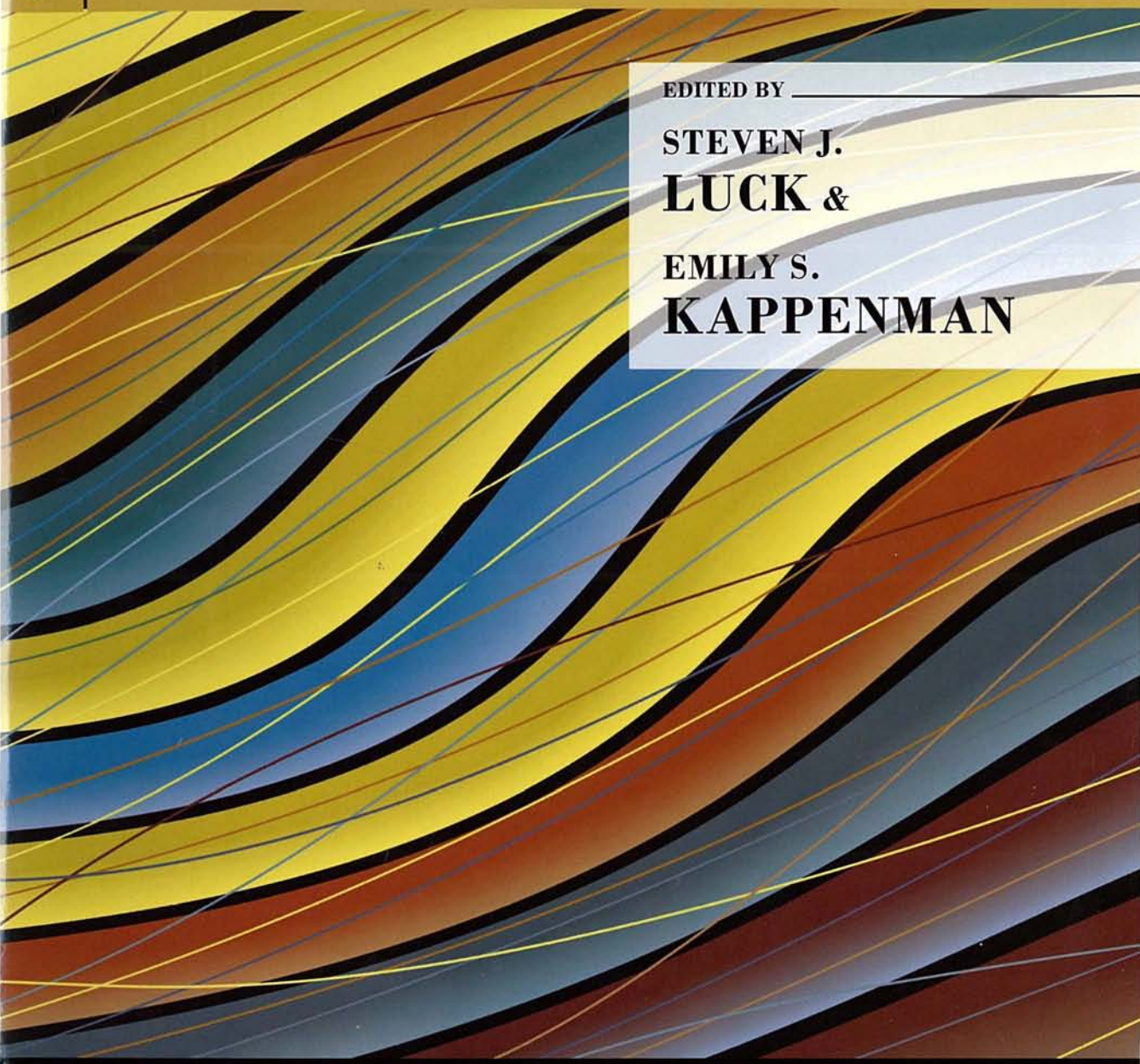

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The Oxford Handbook *of* EVENT-RELATED POTENTIAL COMPONENTS

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CHAPTER

10 The Error-Related Negativity (ERN/Ne)

William J. Gehring, Yanni Liu, Joseph M. Orr, *and* Joshua Carp

Abstract

We review two decades of research on the error-related negativity (ERN or Ne), a component of the event-related brain potential that accompanies errors in speeded performance. Theories of the ERN must contend with a wealth of experimental data, both in healthy subjects and in individuals with neurological and psychiatric conditions. Data regarding a number of other components, including the error positivity, feedback-related negativity, correct response negativity, and theta oscillations are thought by many to also constrain ERN theorizing. We attempt to characterize the past highlights and current trajectory of theorizing, computational modeling, and empirical research. We consider how the way in which ERN research is conducted affects its success, and we discuss some promising trends for the future. Although two decades have resulted in impressive theories and data, the ERN community awaits breakthrough developments by new investigators.

Keywords: error-related negativity, ERN, error negativity, Ne, feedback-related negativity, FRN, error detection, response conflict, reinforcement learning, anterior cingulate cortex.

Introduction

It has been 20 years since the first reports of an event-related brain potential component associated with error commission in choice reaction time performance (Falkenstein et al., 1989, 1990, 1991; Gehring et al., 1990, 1993). The anniversary provides a good opportunity to review the state of research on this component, which is known as the *error negativity* (Ne) or *error-related negativity* (ERN). Here we refer to it as the *ERN*. Over the two-decade span, a large number of studies have followed, and ERN research has proven influential to scientists in diverse fields both inside and outside the ERP research community.

In this chapter, we present a somewhat selective overview of ERN research. Excellent reviews of major experimental findings already exist (Falkenstein, 2004a; Falkenstein et al., 2000; Holroyd et al., 2004b; Nieuwenhuis et al., 2004a; Olvet & Hajcak, 2008; Overbeek et al., 2005; Ullsperger, 2006) and collections of papers concerning the ERN and related topics appear regularly in special issues of journals (e.g., Elton et al., 2000; Falkenstein, 2004b, 2005; Kok et al., 2006; Mars et al., 2008). Instead of exhaustively reviewing the ERN literature, our goal here is to step back and characterize the big picture—what we can safely conclude about the functional significance of the ERN, how the way in which ERN research is conducted has affected this knowledge, and how considering these things can suggest some new directions for future research. Our review focuses primarily on the classic response-locked ERN, referring to other components only insofar as they shed light on the ERN.

In evaluating the progress of over 20 years of research on the ERN, it is instructive to consider the comparable 20-year span in the investigation of the P300 component (see Chapter 7, this volume). Sutton and Ruchkin (1984) looked back on the P300 research that had taken place since the initial report of the P300 by Sutton and colleagues (1965). They did not see 20 years of increasing clarity

regarding the significance and neural origins of the P300. Rather, the primary message of their chapter was that 20 years of research had made the situation remarkably complicated. In particular, they noted problems with "issues related to the increase in the number of components that have been identified and to the problem of deciding which components are being dealt with in a particular experiment" (p. 1). Our survey of ERN research suggests that Sutton and Ruchkin's remarks on the P300 of 1984 could easily apply to the ERN of today. Still, although the ERN is more complicated than we thought 20 years ago, the level of theoretical and methodological sophistication has grown at a remarkable pace, and the degree to which ERN researchers have influenced—and been influenced by—the larger community of cognitive neuroscientists is unusual in the history of ERP research.

The ERN

The ERN was first observed in speeded choice reaction time tasks (Falkenstein et al., 1989, 1991; Gehring et al., 1990, 1993; see Renault et al., 1980, for what is probably the earliest appearance of this component in published data), where it appeared in response-locked waveforms as a difference between error trials and correct trials. Figure 10.1 shows the ERN waveforms reported in Falkenstein et al. (1991) and Gehring et al. (1993); note that negative is plotted downward in Panel A of the figure and upward in Panel B. The onset of the ERN occurs at or shortly before the moment of the erroneous button press and peaks around 100 ms later. The precise latency depends on the time-locking event: the ERN will appear later in a waveform time-locked to the onset of electromyogram (EMG) activity than in a waveform from the same set of trials time-locked to the button-press switch closure. The lateness of the ERN seen in Figure 10.1B compared to that in Figure 10.1A may be in part because the data in Figure 10.1B were time-locked to EMG onset and those in Figure 10.1A were time-locked to the button-press switch closure. It is also likely that some of the variability in ERN latency in the literature is related

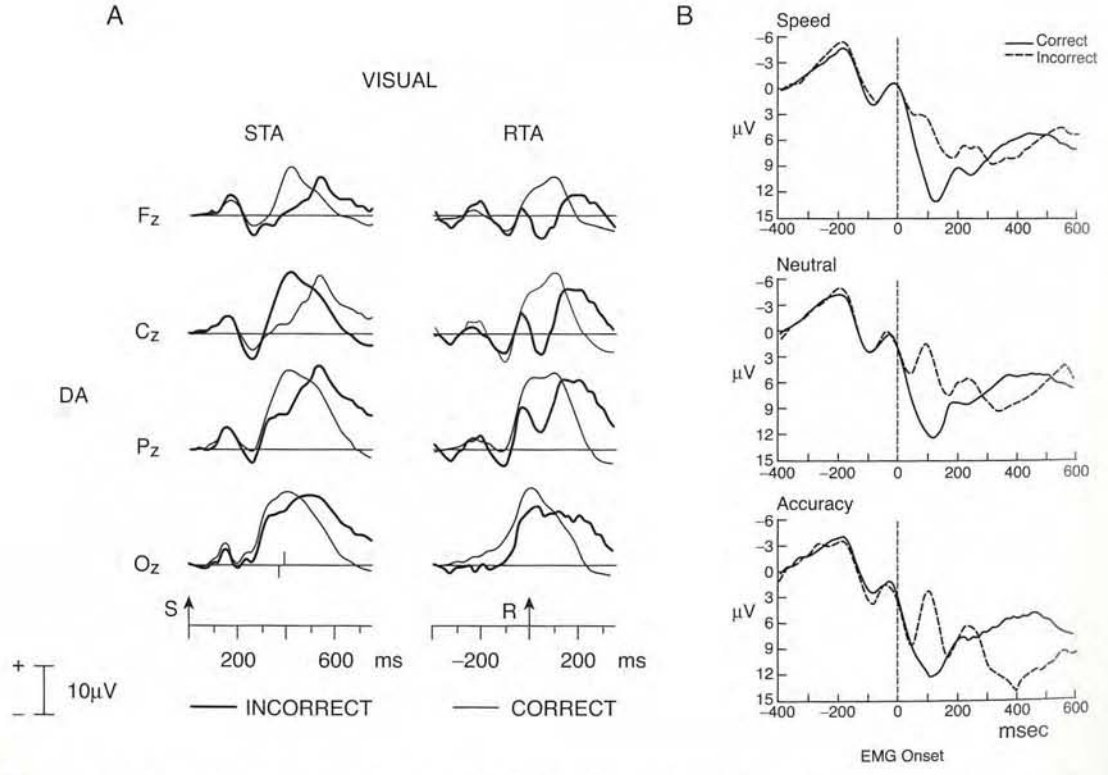

Fig. 10.1. Initial reports of the error-related negativity (ERN/Ne). (A) Stimulus-locked (STA) and response-locked (RTA) grand average ERP waveforms for correct and error trials evoked during a visual discrimination task with divided attention blocks (DA). Response-locked data used the button-press switch closure as the time-locking event. Modified from Falkenstein et al. (1991), Figure 2, reprinted with permission from Elsevier. (B) Response-locked grand average ERP waveforms for correct and error trials evoked during a flanker paradigm. The time-locking event was the onset of electromyogram (EMG) activity associated with a squeeze response. From Gehring et al. (1993), Figure 1, reprinted with permission of John Wiley & Sons, Inc.

to variability across different response devices in the time it takes for a button press to travel from a resting position to switch closure. Studies have found the scalp distribution of the ERN to be maximal at midline frontocentral scalp locations, most typically the 10–20 location FCz. The ERN occurs on error trials in a wide variety of speeded-response tasks (Falkenstein et al., 1995; Gehring et al., 1995) involving visual (Falkenstein et al., 1991; Gehring et al., 1993), auditory (Falkenstein et al., 1991), and tactile (Forster & Pavone, 2008) stimuli, and unimanual (Falkenstein et al., 1991; Gehring et al., 1993), bimanual (Murata & Katayama, 2005), foot (Forster & Pavone, 2008; Gehring & Fencsik, 2001; Holroyd et al., 1998), oculomotor (Endrass et al., 2005; Nieuwenhuis et al., 2001), and vocal (Masaki et al., 2001) responses. It may even be elicited by auditory, visual, and somatosensory error feedback stimuli (Holroyd & Coles, 2002; Miltner et al., 1997) and by losses in gambling tasks (Gehring & Willoughby, 2002).

The ERN has attracted a great deal of interest, both within the ERP research community and in cognitive neuroscience more generally. Much of this interest arose because of evidence that the ERN is generated in the anterior cingulate cortex (ACC) and because of the burgeoning interest in the role of the ACC in those *cognitive control* functions that enable the brain to adapt behavior to changing task demands and environmental circumstances (Botvinick et al., 2001; Ridderinkhof et al., 2004). Cognitive control functions include processes that detect when control is needed—as when performance breaks down—and processes that implement control through changes in attentional focus and other strategic adjustments. Because an error is a salient marker that performance has broken down, the ERN is generally thought to reflect a process involved in evaluating the need for, or in implementing, control. As we describe in this chapter, the quest to determine the precise nature of that process has spawned a rich and fascinating debate involving evidence that includes neuroimaging, neurological and psychiatric patient studies, animal neurophysiology, neuropharmacology, and computational modeling.

The ACC

Gehring et al. (1993) suggested that the ACC and the adjacent supplementary motor area (SMA) were likely candidates for the neural generator of the ERN. A short time later, Dehaene and colleagues (1994), using Brain Electromagnetic Source Analysis (BESA; Scherg, 1990), showed that an equivalent dipole within the ACC accounted well for the midline-frontal scalp distribution of the ERN (Figure 10.2). Several subsequent BESA modeling efforts have supported an ACC locus (e.g., Holroyd et al., 1998; Mathewson et al., 2005; van Veen & Carter, 2002).

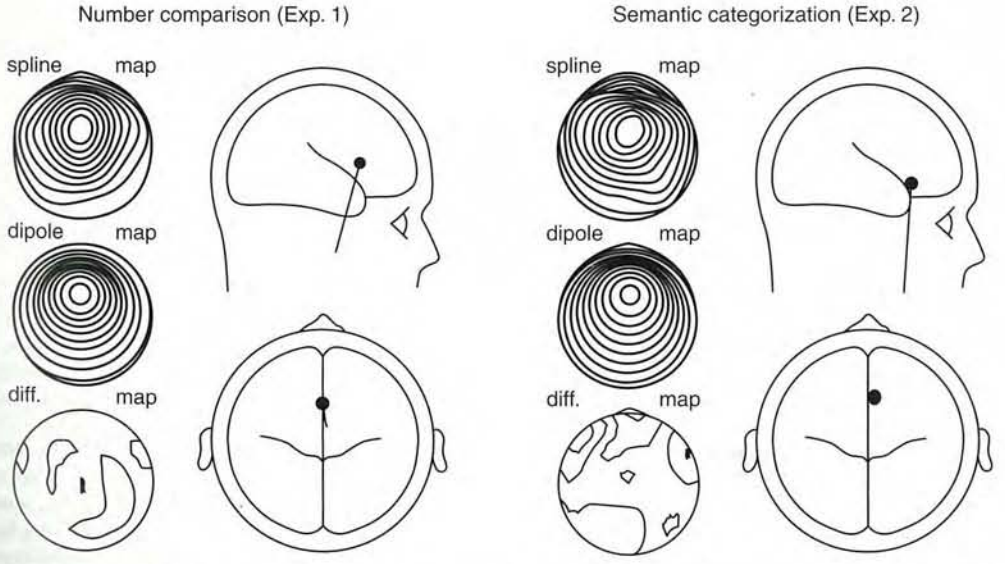

Fig. 10.2. Dehaene and colleagues (1994), using Brian Electromagnetic Source Analysis (BESA) to analyze data recorded with a dense electrode array, showed that an equivalent dipole placed roughly in the ACC accounted well for the midline-frontal scalp distribution of the ERN. From Dehaene et al. (1994), Figure 2, reprinted with permission of John Wiley & Sons, Inc.

Consistent with this ERP evidence, several functional magnetic resonance imaging (fMRI) studies showed error-related blood oxygen-level dependent (BOLD) signal increases in the ACC (Figure 10.3; Carter et al., 1998; Kiehl et al., 2000; Menon et al., 2001; Ullsperger & von Cramon, 2001; for reviews, see Hester et al., 2004; Ridderinkhof et al., 2004; Taylor et al., 2007). For example, Carter and colleagues showed that a region of the ACC responsive to conflict was also involved in processing errors in a modified version of the continuous performance task (see Figure 10.3A). In addition, one magnetoencephalographic study in humans has identified an ACC source (Miltner et al., 2003; but see Stemmer et al., 2004b). The ACC encompasses several subdivisions along its rostral-caudal extent, and the precise locus of the ERN generator within this region is usually argued to be in a dorsal region of the ACC (e.g., Debener et al., 2005; Garavan et al., 2003; Holroyd & Coles, 2002; Holroyd et al., 2004b; Ridderinkhof et al., 2004; Yeung et al., 2004b).

Neurophysiological studies in nonhuman primates support the existence of ACC activity related to errors. Early studies found ACC single-unit activity related to the absence of an earned reward (Niki & Watanabe, 1979), and a subsequent study using local field potentials (LFPs) found ACC activity during intermediate stages of learning in a task where monkeys had to learn the appropriate response to a visual cue (Gemba et al., 1986). A series of recent studies has used an oculomotor stop-signal task that is similar to the speeded tasks typically used to elicit the ERN. In the stop-signal task, an error is a trial where subjects are directed by a stop signal not to respond, yet they fail to withhold the response. Both single-unit (Ito et al., 2003) and LFP (Emeric et al., 2008) recordings in monkeys

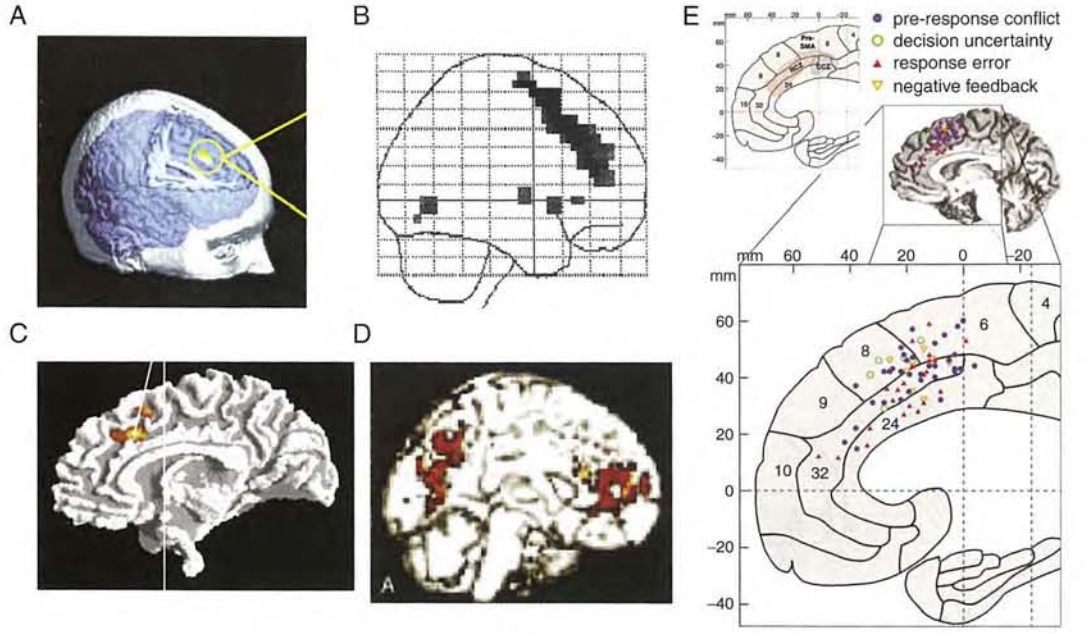

Fig. 10.3. Error-related fMRI activations. Studies showing the medial frontal loci of fMRI BOLD response activations associated with errors. (A) Carter and colleagues showed that a region of the ACC that responded to processing conflict also was involved in processing errors. The fMRI activations were elicited by a modified continuous performance task. From Carter et al. (1998), Figure 2, reprinted with permission from AAAS. (B) In a go/no-go task, errors of commission were associated with BOLD activation in the medial frontal cortex consisting of voxels in the caudal ACC, rostral ACC, and medial frontal gyrus. From Kiehl et al. (2000), Figure 1A, reprinted with permission of John Wiley & Sons, Inc. (C) Errors in a flanker task were associated with fMRI activations in the cingulate motor area (CMA). From Ullsperger and von Cramon (2001), Figure 2, reprinted with permission from Elsevier. (D) In a go/no-go task, errors of commission were associated with fMRI activations in the ACC and medial prefrontal cortex, as well as the insula and precuneus/posterior cingulate. From Menon et al. (2001), Figure 1A, reprinted with permission of John Wiley & Sons, Inc. (E) Ridderinkhof and colleagues (2004) presented a meta-analysis of fMRI studies finding activations in the medial frontal cortex related to performance monitoring. Error-related activations were found in various regions of the medial frontal cortex, including the rostral cingulate zone (RCZ), the caudal cingulate zone (CCZ), and the pre-SMA. From Ridderinkhof et al. (2004), Figure 1, reprinted with permission from AAAS.

have shown ACC activity related to errors (see Figure 10.4). Interestingly, analogs of the ERN may even be evident in rats when they make errors, in medial-frontal cortex locations homologous to the human ACC (Smith et al., 2009).

If the ERN is generated in the ACC, lesions there should reduce ERN amplitude. There are only a few studies of humans that have tested this prediction, in part because well-circumscribed ACC lesions are not common. Swick and Turken (2002) described an individual with a focal left-hemisphere lesion encompassing the rostral to middorsal ACC who failed to show an ERP difference between error and correct trials, which could be consistent with a reduction in the ERN. Nevertheless, the individual showed a large negativity on both error and correct trials, which could indicate that the structures necessary for generating the ERN were intact but receiving faulty input. Stemmer and colleagues (2004a) reported that three of five individuals with ACC damage showed no ERN, but that the other two showed an ERN in at least one of the two flanker

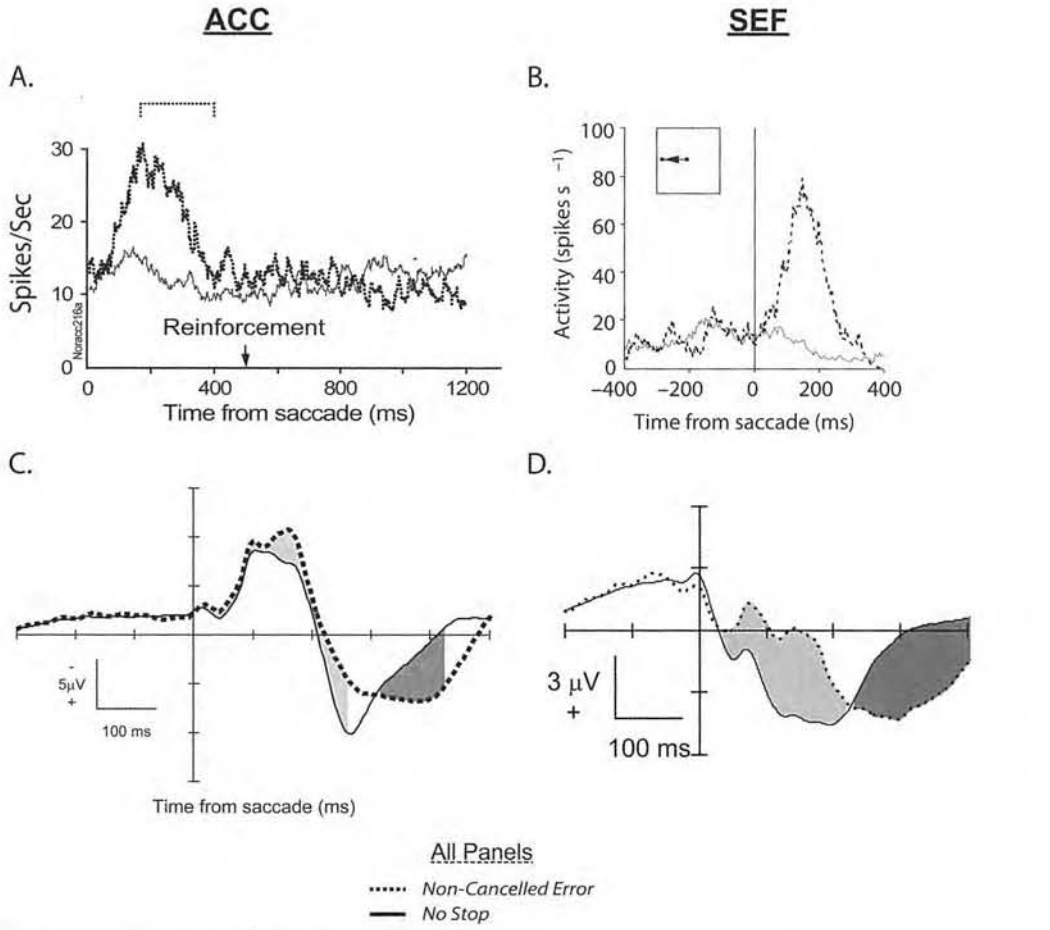

Fig. 10.4. Single-unit (top) and local field potentials (bottom) recordings in macaque monkeys comparing activity for correct no-stop signal trials and erroneous noncanceled trials. These figures demonstrate error activity that manifested earlier in the supplementary eye field (SEF) than in the anterior cingulate cortex (ACC). (A) The average spike rate in an ACC neuron peaked at about 200 ms after the initiation of an erroneous noncanceled saccade. The bracket reflects the range of saccade initiation times for errors. From Ito et al. (2003), reprinted with permission from AAAS. (B) The average spike rate in an SEF neuron peaked at about 180 ms after the initiation of an erroneous noncanceled saccade. From Stuphorn et al. (2000), reprinted with permission from Macmillan Publishers Ltd. Error activity peaks earlier in the SEF (panel B) than in the ACC (panel A). (C) Local field potentials averaged across multiple sites from the ACC revealed a greater negativity for errors that peaked at about 180 ms after saccade onset and a greater positivity for errors that peaked at about 400 ms after saccade onset. From Emeric et al. (2008), reprinted with permission from the American Physiological Society. (D) Local field potentials averaged across sites in the SEF revealed a greater negativity for errors that peaked at about 80 ms after saccade onset and a greater positivity for errors that peaked at about 300 ms after saccade onset. From Emeric et al. (2010), reprinted with permission from the authors. Both the early error negativity and the later error positivity peaked earlier in the SEF (panel D) than in the ACC (panel C).

task variants used in that study. Precise lesion loci were not reported, but Stemmer et al. suggested that the individuals with no ERN had damage in the rostral ACC and those with spared ERNs tended to have damage in the most ventral (subgenual) ACC (see also Stemmer et al., 2000). However, performance measures differed between patients and controls in these studies. Thus, although the studies report results consistent with an ACC source of the ERN, the inconsistency of lesion locations and effects on the ERN, as well as behavioral differences between groups, point to the need for further work.

Although much of the evidence discussed thus far is consistent with a dorsal ACC generator, a number of considerations suggest that areas outside of the dorsal ACC—such as the rostral ACC and the pre–supplementary motor area (pre-SMA)—should also be considered as candidate neural generators for the ERN. Some fMRI studies of errors (Kiehl et al., 2000; Menon et al., 2001) showed error-related ACC activity in the rostral anterior cingulate. Indeed, across the literature, there is substantial variability in fMRI findings: a meta-analysis of fMRI studies by Ridderinkhof and coworkers (2004) found peaks for error-related activity throughout the medial frontal cortex, including areas adjacent to the dorsal ACC, such as the rostral ACC and pre-SMA (see Figure 10.3). Another reason for questioning the dorsal ACC assumption is that it is still an open question whether the fMRI BOLD response and the ERN reflect the same neural activity. The most direct test of this assumption was reported by Debener et al. (2005), who used simultaneous recording of EEG and fMRI to show that a single-trial measure of the ERN predicted the ACC BOLD response on the same trial. Nevertheless, their single-trial measure of the ERN was sensitive to the positive deflection following the ERN in addition to the ERN itself (see the discussion of the early error positivity below). Also, Mathalon and colleagues (2003) measured the ERN and BOLD response in separate sessions and found the ERN to be correlated with a more rostral ACC region in addition to the dorsal ACC (whose activation extended into neighboring Brodmann's area 8). Even reports of dorsal ACC activity should not be taken at face value; Nachev and coworkers (2008) argue that many of the dorsal ACC fMRI activations reported in the literature are more accurately characterized as pre-SMA activations.

The use of BESA equivalent-dipole models that provides much of the support for a dorsal ACC source calls for additional caution. First, some evidence using BESA has pointed to other possible generators, particularly the SMA and a more rostral/ventral part of the ACC (Dehaene et al., 1994; Luu et al., 2003). Moreover, excessive reliance on BESA is itself a cause for concern, because of BESA's limited accuracy in localizing deep sources such as the ACC and because of its inability to distinguish a single deep source from a more widespread and superficial distribution of cortical activity. Even for the most experienced and careful BESA modelers, the number of sources reached by the technique can never deviate from the number of dipoles stipulated in advance by the modeler (see Luck, 2005). In addition, even with the freedom to select any number of dipoles, ERN investigators have reported models that are rather unimpressive in their ability to explain the observed data: in some cases, the unexplained variance in fitting the observed data to the data predicted by the model approaches or even exceeds 10%, making it plausible that alternative models would be more appropriate.

Other methods support the possibility that sources other than (or in addition to) the dorsal ACC contribute to the ERN, including the rostral ACC and the pre-SMA. Studies of nonhuman primates, for example, showed error-related single-unit activity (Stuphorn et al., 2000) and LFPs (Emeric et al., 2010) in the supplementary eye field (SEF) in addition to those in the ACC. The SEF may serve the same function for oculomotor movements that the pre-SMA or SMA does for manual movements (Schall & Boucher, 2007; Stuphorn et al., 2000). Similarly, in a study of humans, Herrmann and colleagues (2004) reported an analysis employing low-resolution electromagnetic tomography (LORETA) that showed a pre-SMA source for the ERN (and, interestingly, a dorsal ACC source for the Pe, which could have influenced the results of Debener et al., 2005, described above). Vidal and coworkers (2000) suggested that the enhancement of the ERN by a surface Laplacian analysis is more consistent with a superficial source such as the SMA than with a deeper source. In the study by Miltner et al. (2003), at least four of the six subjects showing a magnetic equivalent of the ERN showed sources more consistent with a rostral than with a dorsal ACC source. Brazdil and colleagues, using intracranial recordings in humans, identified multiple sources of ERN-like activity, including the rostral ACC and pre-SMA (Brazdil et al., 2002, 2005). One study using transcranial magnetic stimulation (TMS) found that medial frontal stimulation of the pre-SMA led to an attenuation of the ERN (Rollnik et al., 2004).

The orbitofrontal cortex (OFC) has emerged as another possible contributor to the ERN, although the evidence is both limited and mixed. Intracranial ERN-like activity has been observed in the OFC (Brazdil et al., 2002, 2005). Turken and Swick (2008) found that lesions in the OFC reduced the amplitude of the ERN. The lesions in that study extended to the rostral portion of the ACC that was associated with a reduced ERN in the study of Stemmer et al. (2004a) described earlier, so it is possible that the more rostral ACC damage contributed to the effect. Ullsperger and coworkers (2002) failed to find reduced ERNs in patients who had damage to the frontopolar cortex that included the OFC. Still, the plausibility of an OFC source is further supported by neurophysiological recordings in monkeys that show neurons with some similarity to those observed in the ACC (e.g., Thorpe et al., 1983).

Whatever the source of the ERN, connectivity between that structure and the lateral prefrontal cortex (PFC) appears to be critical for generating the ERN. Gehring and Knight (2000) reported that a group of individuals with focal lateral PFC lesions showed ERN activity equivalent to that of controls on error trials, but what appeared to be an ERN on correct trials (see discussion of the correct-response negativity below), resulting in no difference between error and correct trials. Other studies found reduced ERNs caused by lateral prefrontal damage (Ullsperger & von Cramon, 2006b; Ullsperger et al., 2002). Consistent with these findings, lesions from sickle-cell disease—thought to interrupt the communication between the lateral and medial frontal cortices—also reduced the ERN (Hogan et al., 2006; Figure 10.5). Other evidence of communication between the generator of the ERN and the lateral PFC comes from measures of phase coherence between medial and lateral frontal sites (Cavanagh et al., 2009) and a correlational functional connectivity analysis based on LORETA (Holmes & Pizzagalli, 2008).

It may be the case that the source of the ERN uses information supplied by the PFC to distinguish errors and correct responses (Gehring & Knight, 2000) or that the ERN is inhibited on correct trials. Alternatively, damage to the PFC may increase the response conflict on correct trials (Cohen et al., 2000). It is also possible that the ERN detects conflict or errors and signals the PFC to engage cognitive control processes (Kerns et al., 2004), but if the direction of communication runs from the medial to the lateral PFC, some kind of bidirectional communication might be necessary to explain how lateral PFC lesions would diminish the size of the ERN (see, e.g., Banich, 2009). Note, however, that not all evidence points to a role for the PFC in generating the ERN; the TMS study mentioned above found no effect of lateral PFC stimulation on the ERN (Rollnik et al., 2004).

More recent evidence suggests that the generator of the ERN may communicate with a number of regions in addition to the lateral PFC. Cohen (2011) correlated measures of theta-band activity likely to be sensitive to the ERN (see below) and white matter tract strength based on diffusion tensor imaging. The amount of error-related activity was related to the strength of white matter connections

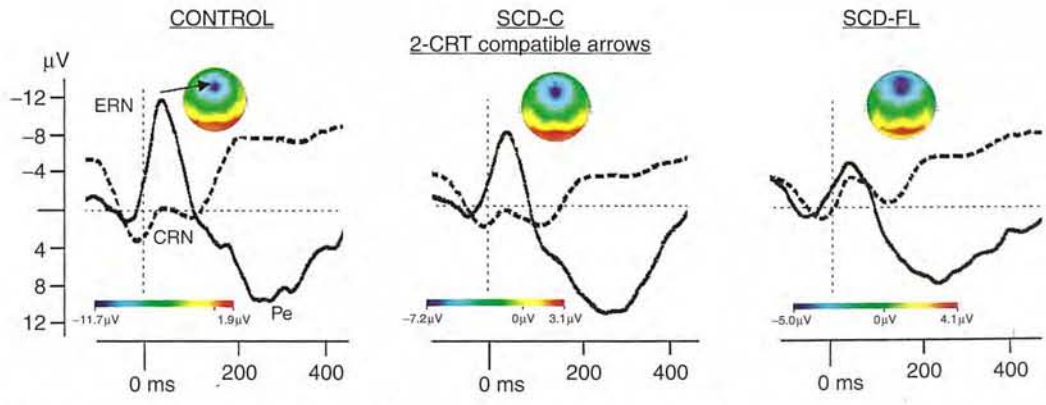

Fig. 10.5. Error-related negativity waveforms from healthy controls, control subjects with sickle-cell disease and an absence of brain lesions (SCD-C), and patients with frontal lesions due to sickle-cell disease (SCD-FL). The lesions in frontal white matter decreased the amplitude of the ERN, supporting a prefrontal contribution to the generation of the ERN. From Hogan et al. (2006), Figure 3, reprinted with permission from Oxford University Press.

linking the medial frontal cortex to the motor cortex, inferior frontal cortex and ventral striarum. Moreover, subjects showing a greater degree of theta-band phase synchrony between FCz and other scalp electrodes showed stronger connections linking the medial frontal cortex to the corpus callosum and to white matter tracts leading to the superior frontal gyrus.

Many investigators assume that the ERN arises from the dorsal ACC, yet the inconsistency of the evidence supporting this hypothesis, coupled with other evidence consistent with alternative sources, raises the intriguing possibility that one or more regions outside the dorsal ACC contribute to the ERN. The ERN could arise because of multiple sources, such as the SMA/pre-SMA, along with a deeper source such as the rostral ACC or the dorsal ACC (Falkenstein, 2004a). It is even possible that the ERN arises from a single region other than the dorsal ACC, such as the rostral ACC, the pre-SMA, or the SMA (Vidal et al., 2000). In any case, it seems prudent for ERN research to consider some of these alternatives rather than to proceed with the assumption that the dorsal ACC is the only region that generates the ERN.

Related Components

Before reviewing the major theories and models of the functional significance of the ERN, we turn to other ERP components that are relevant to these theories. Some are also elicited by errors; others may share a common neural generator.

ERROR POSITIVITY

In the response-locked error-trial waveform, a positivity known as the *error positivity* (Pe) usually follows the ERN (Falkenstein et al., 1990; for a comprehensive review, see Overbeek et al., 2005). The Pe often has a centroparietal topography with a maximum amplitude between 200 and 400 ms after an erroneous response (e.g., see Figures 10.1, 10.5, 10.6, 10.15b, 10.18, and 10.19). The Pe, unlike the ERN, is consistently larger for errors that the subject reports than for errors that go unreported (Endrass et al., 2005, 2007; Nieuwenhuis et al., 2001; O'Connell et al., 2007; Vidal et al., 2000) and is related to the increase in skin conductance response (SCR) following error responses relative to that following correct responses (Hajcak et al., 2003b). Falkenstein (2004a) and Overbeek et al. (2005) review evidence for three hypotheses of the functional significance of the Pe: that it reflects an affective response to the error, that it is involved in awareness of the error, and that it is involved in adapting response strategies following an error.

Nevertheless, definitive conclusions await a better understanding of the component structure of the post-error positivities, an understanding that is just beginning to emerge. There is wide variability in the reported Pe scalp distributions (Arbel & Donchin, 2009; Overbeek et al., 2005), and there is some disagreement about what constitutes a Pe. For example, several investigators suggested that the Pe may be a delayed parietal P300 (P3b) associated with detecting or evaluating an error (Davies et al., 2001; Leuthold & Sommer, 1999; Overbeek et al., 2005), an assertion supported by the similar response of the P300 and the centroparietal Pe to variations in the intertrial interval (Ridderinkhof et al., 2009). It is likely, however, that there are two components: an early fronto-central Pe and a later, more posterior component (Arbel & Donchin, 2009; Ruchsow et al., 2005b; van Veen & Carter, 2002). The anterior Pe may have a neural generator overlapping or near that of the ERN in the medial frontal cortex (Herrmann et al., 2004; van Veen & Carter, 2002). The early Pe and the ERN are also similar in that both are unrelated to error awareness in an antisaccade task (Endrass et al., 2007). As we discuss below, these similarities have led to the suggestion that the ERN and early the Pe are both parts of a single oscillatory potential. As for the later, more posterior positivity, it could be generated in the rostral ACC and reflect error awareness (Endrass et al., 2007) or a subjective affective response (van Veen & Carter, 2002). It could also be a P300 (P3b) associated with the error (Arbel & Donchin, 2009).

N200/N450

Larger stimulus-locked N200s appear on incongruent trials than on congruent trials in conflict tasks like the Eriksen flanker task (Gehring et al., 1992; Kopp et al., 1996) and in other conflict conditions (Nieuwenhuis et al., 2003). In conflict tasks using verbal stimuli, such as the Stroop task, an N450 component on incongruent trials may be a delayed instance of the N200 (Liotti et al., 2000; West, 2003). As we discuss later, the conflict monitoring theory claims that the congruence effect on the N200/N450 and the ERN reflect the same component. It is plausible that the N200 and the ERN might be related, because the scalp distribution and time course of the ERN bear some similarity to those of the classic N200 component (Simson et al., 1976; Squires et al., 1976; for reviews, see Folstein & Van Petten, 2008; Pritchard et al., 1991). Studies relating

the ERN to reinforcement learning show some additional parallels: the N200-like component elicited by error feedback (see below) and the appearance of an N200 in response to stimuli that violate expectancies acquired during sequence learning (Eimer et al., 1996; Ferdinand et al., 2008; Russeler et al., 2003).

CORRECT-RESPONSE NEGATIVITY

Although the ERN is usually much larger on error trials than on correct trials, a negativity often appears on correct trials at the same latency in the response-locked waveform as the ERN (Figure 10.6; Ford, 1999; Gehring & Knight, 2000; Luu et al., 2000b; Scheffers & Coles, 2000; Vidal et al., 2000). The correct-response negativity (CRN) is usually smaller than the ERN, but the two components show a similar scalp distribution (Vidal et al., 2000). In some published waveforms the CRN is quite striking, especially in individual subject data (e.g., Swick & Turken, 2002). Observations of a CRN have raised the question of just how specific the ERN is to errors (Vidal et al., 2000). We will discuss the CRN in more detail later in the chapter.

FEEDBACK-RELATED NEGATIVITY

Miltner and colleagues (1997) reported that error-feedback stimuli (such as tones informing the subject that an error has occurred) elicit activity that resembles the ERN (Figure 10.7; Badgaiyan & Posner, 1998; Gehring & Willoughby, 2002; Holroyd & Coles, 2002; for a review, see Nieuwenhuis et al., 2004a). Whereas the classic ERN is time-locked to the error response, the feedback-related negativity (FRN) occurs approximately 250–300 ms following a feedback stimulus. In the Miltner et al. task, for example, subjects made a time estimation judgment using a button-press response, and the task was difficult enough that subjects did not know the accuracy of their judgment until the feedback stimulus occurred 600 ms later. In gambling tasks, subjects make a choice and later receive random gain or loss feedback (e.g., Gehring & Willoughby, 2002). In some ways, the FRN seems similar to the ERN: it is a negative-going component with roughly the same time course and a frontocentral scalp distribution. The FRN can be modeled as one or two equivalent dipoles in roughly the same location observed in studies of the classic ERN (e.g., Gehring & Willoughby, 2002; Miltner et al., 1997). If the FRN represents the same component as the ERN, the FRN would be evidence that the ERN is a general-purpose system for error detection (Miltner et al., 1997). We discuss this component in more detail in the section on the reinforcement-learning theory of the ERN.

THETA OSCILLATIONS

In numerous reports of the ERN, one gets the visual impression of multiple negative peaks, with a small

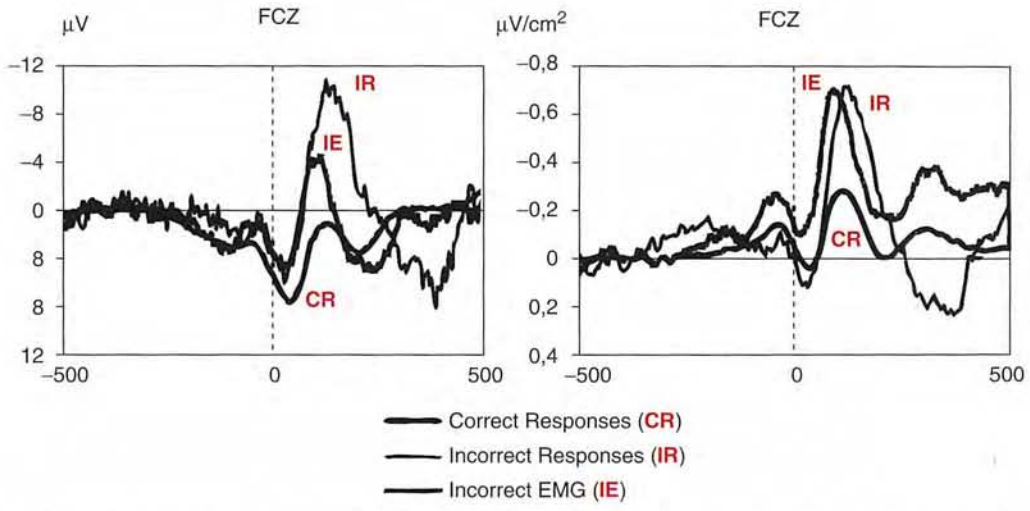

Fig. 10.6. The CRN. Grand average EMG-locked monopolar activity (left) and Laplacian transformed activity (right) elicited during a go/no-go task. The authors compared activity for correct responses, incorrect responses, and partial errors (where incorrect EMG activity was observed but no incorrect response was made). The authors observed ERN-like activity on all three trial types; the ERN was greater for partial errors than for correct responses and greatest for incorrect responses. Modified from Vidal et al. (2000), Figure 2, reprinted with permission from Elsevier.

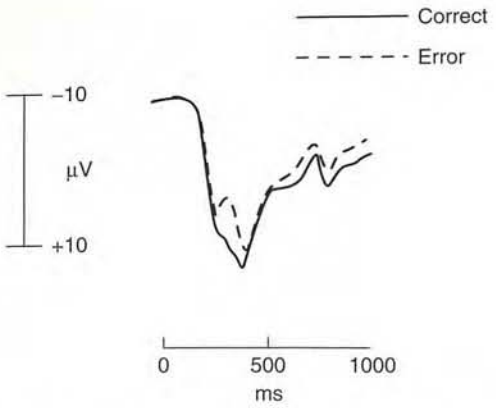

Fig. 10.7. Grand average ERP waveforms evoked by visually presented correct and error feedback stimuli in a time estimation task. This report suggested that the negative-going component elicited by negative feedback is the same as the classic response-locked ERN. Adapted from Miltner et al. (1997), Figure 3, reprinted with permission from MIT Press.

negativity often following the ERN (e.g., Figures 10.1b, 10.6). This peak does not always follow the ERN in average waveforms, and indeed, it may be a correction-related negativity associated with the corrections following an error response, as Fiehler and colleagues (2005) claim. However, Luu and Tucker (2001) proposed an alternative possibility: that the ERN is just one peak in a theta-frequency (4–7 Hz) oscillation. Studies that have applied time-frequency analysis to the ERN have confirmed that the ERN does indeed appear within that frequency range, but thus far, such techniques cannot reveal definitively whether the ERN actually consists of single or multiple peaks (Bernat et al., 2005; Cavanagh et al., 2009; Gehring & Willoughby, 2004; Hall et al., 2007; Trujillo & Allen, 2007; see Figure 10.8). Other studies have identified a delta (1.5–3.5 Hz) contribution to the ERN in addition to the theta activity (Yordanova et al., 2004). These investigators argue that the delta contribution reflects error-specific processing, whereas the theta activity is more generally related to response monitoring because of its presence in both the CRN and the ERN (Yordanova et al., 2004). This argument suggests that the separate time-frequency analysis of both errors and correct trials will be necessary for a complete picture of the time-frequency content of the ERN, because not all time-frequency studies have included correct-trial data.

The notion that the ERN is part of a theta oscillation suggests an alternative interpretation of the early, anterior Pe discussed earlier (e.g., van Veen & Carter, 2002): the ERN and the early, anterior Pe could both be part of a single oscillatory potential. The biphasic nature of intracranial field potentials recorded in monkeys would be consistent with the sources of the ERN and early Pe being close to each other (Emeric et al., 2008). Arbel and Donchin (2009) suggested that the early Pe was not part of the same oscillation that causes the ERN, however, because they found that the ERN and the fronto-central Pe responded differently to speed-accuracy instructions. Still, it is plausible that experimental variables could influence an oscillatory generator differently at different points in the oscillation if

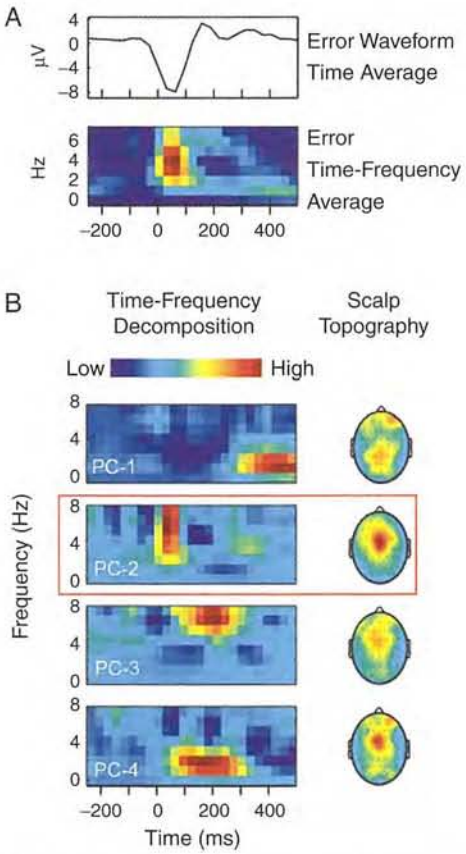

Fig. 10.8. Grand average error waveform (top) elicited by a flanker task and the average time-frequency data showing a peak in the theta range. Time-frequency surfaces show components as colored blobs in the time-frequency plane. Multiple components can occur and overlap in the time-frequency plane much as they do in the time-domain waveforms; thus, such surfaces require techniques to separate one component from another. Here, the authors used principal components analysis to extract the ERN (component 2) from the other overlapping activity. Modified from Hall et al. (2007), Figure 3, reprinted with permission of John Wiley & Sons, Inc.

time-varying information flows continuously to the generator.

A related issue is whether the ERN represents theta activity that is more phase-aligned on error trials than on correct trials. Phase alignment on error trials alone could produce an ERN in the average error waveform merely because the single-trial peak happens to occur more consistently at the same latency on error trials, even if there is no actual change in amplitude. Luu et al. (2004) supported this idea through an analysis of single-trial data filtered for theta-frequency activity (see also Trujillo & Allen, 2007). Yeung and colleagues (2004a, 2007a), however, suggested that such analyses must be viewed with caution. For example, digital filters can cause ringing artifacts that appear as oscillations, and an increase in a single discrete ERN-like event on error trials could cause data to appear as though phase alignment was greater on those trials than on correct trials. (Illusory oscillations could also result from Morlet wavelets, such as those used by Trujillo and Allen [2007].) Nevertheless, the Yeung et al. studies do not rule out either the theta-oscillation or phase-alignment hypotheses, and ringing artifacts may not be a satisfactory explanation for the oscillatory appearance of the ERN (Cavanagh et al., 2009; Trujillo & Allen, 2007). Some evidence indicates that phase alignment and amplitude enhancement both play a role (Cavanagh et al., 2009). Finally, further complicating the picture, a study of the FRN showed evidence for phase-amplitude coupling across frequency bands, raising the intriguing possibility that the computations underlying the ERN and FRN may be more accurately characterized by examining the interactions among activities in multiple frequency bands (Cohen et al., 2009).

Flies in the ERN Ointment

The previous section has already introduced some of the thorny issues in the effort to understand the ERN, and this is as good a place as any to make those issues explicit. First, the range of electrophysiological phenomena to be covered by a theory of the ERN (or by a chapter about the ERN) is changing and is a matter of active debate: Does the ERN include feedback-related components? Does the ERN include the ERN-like peak that happens on correct trials? Does the ERN comprise not only the negative-going peak following the error, but also the sharp fronto-central positivity that sometimes accompanies the ERN? Is the ERN really a multiple-peak oscillation?

Second, as will become clear below, studying a phenomenon related to errors presents unique difficulties: unlike an experiment that can manipulate a critical independent variable—whether a stimulus is attended or unattended, for example—a study of errors cannot specify in advance whether a particular response will be correct. Thus, the analysis of errors must always be to some degree correlational and post hoc. This presents practical problems (designing an experiment in which subjects will produce enough errors for analysis) as well as more conceptual ones (errors have multiple possible causes, and the experimenter cannot know with certainty why a particular error occurred).

Third, the important distinction for the purposes of theory is not what is correct or an error in the eyes of the experimenter, but rather what is deemed correct or an error by the brain of the subject. These are not identical, and some confusion in the literature arises from the assumption that they are. The CRN is an example; it is possible that the CRN occurs because the brain labels a response as an error that is not an error according to the experimenter (Coles et al., 2001). Another example is found in the conflict literature (see below), where it is sometimes assumed that as long as no overt error (like a button press) occurred, there was no error for the brain to detect (see, e.g., Carter et al., 1998), overlooking the fact that the brain might detect errors at a level of response activation lower than that required to produce an overt error (see Gratton et al., 1988; Murthy et al., 2007).

And finally, what is an error at one level of analysis might be correct at another; thus, the brain's response to an error might depend on consequences at some level of analysis other than the one specified by the instructions to the subject. The subject's negative affective reaction to an error might not be determined by the small monetary penalty designated by the experimenter, but instead by the fear of the experimenter's disapproval or the desire to perform better than other subjects or in accord with the experimenter's exhortations. Hence, if changing the penalty from 10 cents to 25 cents fails to affect a component, the reason might be that the component is sensitive to a different, perhaps social, incentive—not that the component is insensitive to incentives.

Functional Significance of the ERN—Major Theories

A spirited debate has emerged regarding the computation represented by the ERN. In this section, we introduce several influential theories of ERN and describe some of the data supporting them.

Error Detection/Comparator Theory

For the first few years of ERN research, the dominant view of the ERN was that it reflected a computation involved in error detection. Specifically, this error-detection theory posited that the ERN reflects a process that compares the output of the motor system (as represented by an efference copy of the movement command) to the best estimate of the correct response at the time of ERN occurrence (Falkenstein et al., 1991; Gehring et al., 1993). The short latency of the ERN and its close temporal association with the response suggest that the ERN is triggered when a comparator receives the efference copy corresponding to the response that is being executed at that moment (Coles et al., 2001). The notion that the ACC might be a comparator was first proposed by the motor physiologist Vernon Brooks (Brooks, 1986). The schematic diagram (Figure 10.9) published by Coles et al. (2001) illustrates this idea: in speeded-response tasks, an error usually occurs because the subject responds before stimulus evaluation is complete. As the response is executed, stimulus processing continues. A comparison process computes the difference between the representation of the correct response (derived from continuing stimulus processing) and a representation of the current, ongoing response (the efference copy). A discrepancy between these two representations gives rise to a mismatch or error signal.

There are several possible ways the ERN could originate from such a system. The ERN could reflect the output of the comparison process (Coles et al., 2001; Falkenstein et al., 1990) or the comparison process itself (Falkenstein et al., 2000; Vidal et al., 2000). Still another possibility is that the ERN reflects a process that uses information in the error signal to prevent or correct the error or to make some kind of strategic adjustment (Gehring et al., 1993; Holroyd & Coles, 2002). Another variant of the error-detection view emphasizes stimulus representations: the premature response is associated with an anticipated stimulus, and it is the mismatch between the anticipated and actual stimuli that causes the ERN (Bernstein et al., 1995; Schmidt & Gordon, 1977).

Conflict-Monitoring Theory

The conflict-monitoring theory of the ACC was originally proposed as an alternative to the error-detection theory (Carter et al., 1998). Proponents of the conflict-monitoring theory argued that the error-detection model was computationally implausible (Botvinick et al., 2001; Carter et al., 1998; Yeung et al., 2004b). In their view, a comparator would have to have information that specified which of the representations being compared was the correct one. For the comparator to have that information, it would have to be able to access information outside of the series of processes responsible for task performance. In particular, the system generating the ERN would have to know the intended (correct) action (Carter et al., 1998). And if the brain

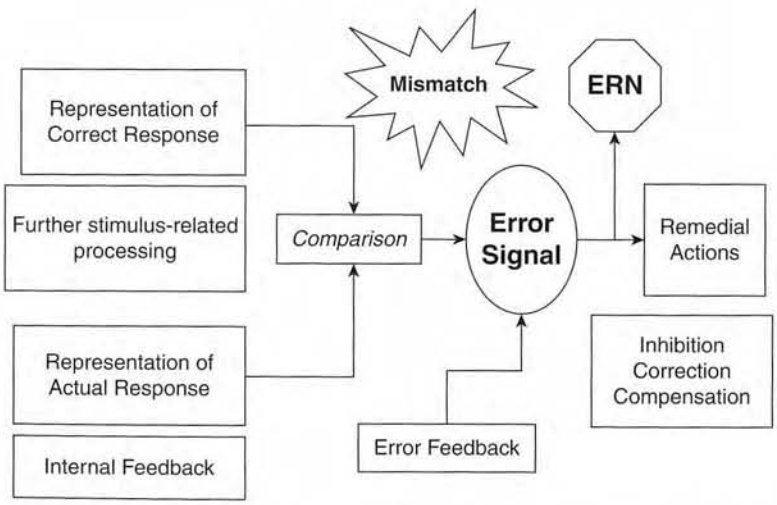

Fig. 10.9. Schematic diagram illustrating the error-detection theory of the ERN, which posits a comparison process that computes the difference between a representation of the correct response (derived from stimulus processing) and a representation of the current, ongoing response (the efference copy). A discrepancy between these two representations gives rise to a mismatch or error signal, which underlies the ERN. From Coles et al. (2001), Figure 1, reprinted with permission from Elsevier.

knows which response is correct, the argument goes, then why isn't the brain executing the correct response?

According to the conflict model, *response conflict*—defined as concurrent activation of multiple competing responses—can account for the ERN without postulating an all-knowing homunculus. In typical choice tasks, the activation of more than one response might signal that something is awry. In this way, response conflict can track performance accuracy without the system "knowing" which response is correct. Conflict signals the need for increased control: following high-conflict trials, the task set is strengthened, leading to improved performance on postconflict trials. Computational implementations of this idea (see Figure 10.10) model response selection using parallel distributed processing networks based on the one originally proposed by Cohen and colleagues (1990). These models define conflict as the Hopfield energy of the response units—in a simple two-choice task, twice the product of the activation of two response units, weighted by the strength of the inhibitory connections among responses.

The conflict model has been used to model performance in a wide variety of tasks (Botvinick et al., 2001), and Yeung et al. (2004b) showed that the simulated conflict signal mimicked the time course of the ERN and its sensitivity to a variety of experimental manipulations, including flanker

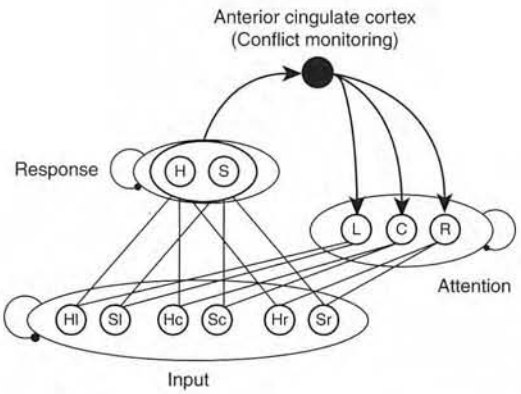

Fig. 10.10. Conflict-monitoring model for the Eriksen flanker task. Stimulus processing units corresponding to the target and flanking letters *H* and *S* activate the corresponding responses. Conflict is generated in the response layer by the coactivation of mutually inhibitory response channels (e.g. "*H*" and "*S*"). The conflict-monitoring process detects the presence of response conflict and signals adjustments in attention that will reduce conflict from the flanking letters on subsequent trials. From Botvinick et al. (2001), Figure 7, reprinted with permission from the American Psychological Association.

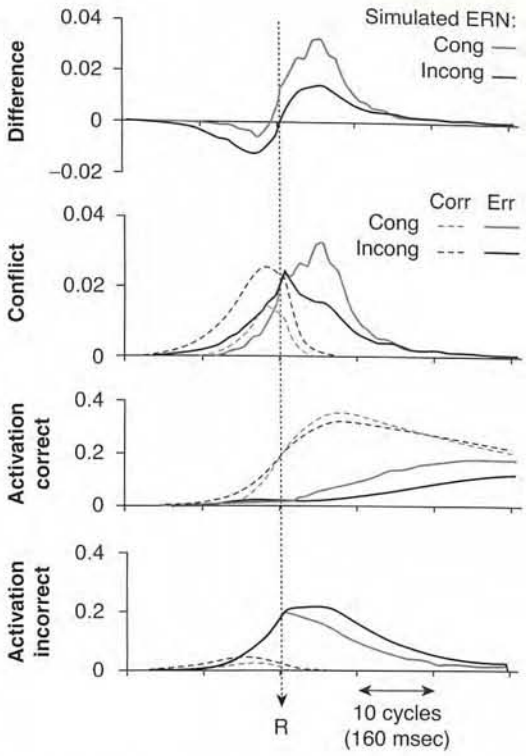

Fig. 10.11. Conflict-monitoring model simulation of response-locked ERN activity for congruent and incongruent trials in a flanker task, shown for correct and error responses. Conflict is the product of activation in the correct channel and the incorrect channel. On congruent error trials, the correct channel is highly activated following the erroneous response, leading to a large simulated ERN. In contrast, conflict is greatest prior to the response on correct incongruent trials, because incorrect response activation from the flanking letters subsides before the correct response. From Yeung et al. (2004b), Figure 4, reprinted with permission from the American Psychological Association.

congruency (see Figure 10.11). For example, the enhanced ERN observed in cases where accuracy is emphasized over speed (see the discussion below and Figure 10.1) can be produced in the conflict model by focusing attention more strongly on the central letter and adopting a more conservative response criterion (Yeung et al., 2004b).

Reinforcement Learning Theory of the ERN

Holroyd and Coles proposed the reinforcement-learning theory of the ERN (abbreviated RL-ERN; Figure 10.12), which is also instantiated as a computational model (Holroyd & Coles, 2002; Holroyd et al., 2005). According to this theory, a monitoring mechanism in the basal ganglia produces an error signal when events occur that are worse than expected, where the expectation has developed

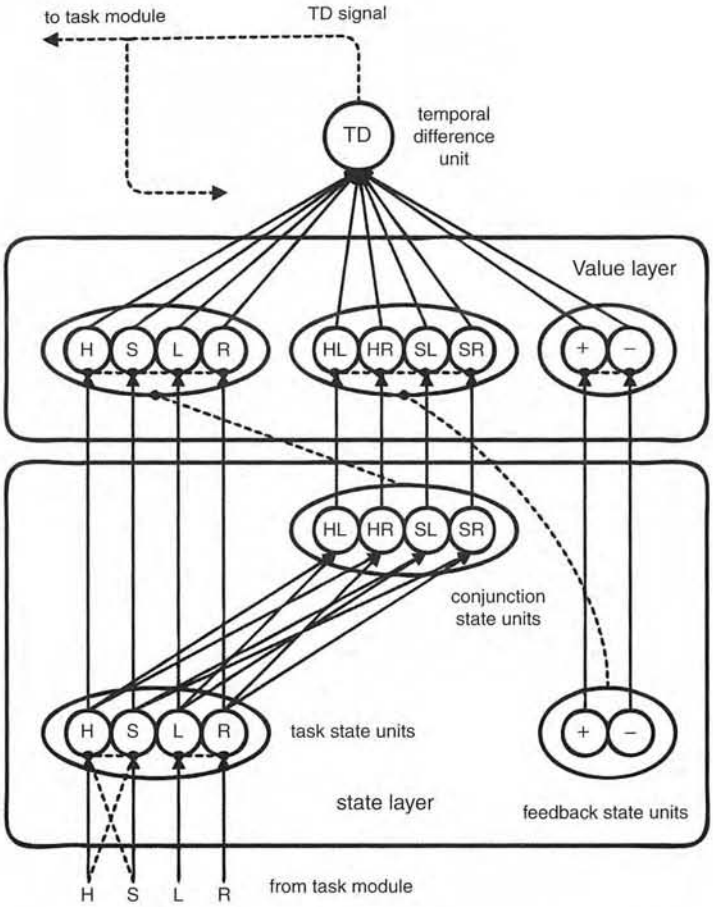

Fig. 10.12. Diagram of the RL-ERN model. A task module (not shown) produces overt behavior in response to external stimulus input, simulating choice reaction time performance. The monitor module (shown) evaluates the output of the task module according to the current context, with additional input from external feedback, and assigns values (good or bad) to the current behavior. In the case of a bad outcome, the monitor module sends an error signal to the task module in order to improve performance. From Holroyd et al. (2005), Figure 8, reprinted with permission from the American Psychological Association.

according to the history of prior reinforcements associated with a response. The midbrain dopamine system conveys this error signal to the ACC, where the signal is used to improve task performance by changing the manner in which control over the motor system is allocated to various competing systems in the brain. The theory has roots both in artificial intelligence models of reinforcement learning (Sutton & Barto, 1988) and in the literature relating dopamine to reinforcement learning (Schultz, 2002). An important prediction of the model is that the monitoring mechanism responds to the earliest information that something has gone wrong. Hence, before a task is learned and early in learning, errors occur because the system does not yet represent the contingencies between stimuli, responses, and reward; thus, the system determines whether performance is good or bad via external feedback. After learning establishes associations between reward values and stimulus–response conjunctions, errors can be detected immediately when a response occurs, without the need to wait for external feedback (Holroyd & Coles, 2002).

A recent extension to the RL-ERN framework is the prediction of response outcome (PRO) theory of Alexander and Brown (2010). The PRO theory says that the medial prefrontal cortex predicts the outcomes of an action based on past experience, and it compares the predicted response outcomes to the outcomes that actually occur. A major difference from the RL-ERN model is that the prediction of response outcomes in the PRO theory does not distinguish between good and bad outcomes. In fact, undesirable outcomes (errors) can be the most likely outcomes, in which case the ERN will occur on correct trials, because it is elicited by the unexpected

outcome (see also Oliveira et al., 2007). An additional difference from the RL-ERN model is that the outcome prediction implemented in the PRO theory is not dependent on a dopaminergic error signal.

Affect/Motivation

As most experimenters know, subjects often become aware of their errors, and the awareness often manifests itself in language unfit for polite company. Some investigators have hypothesized that the ERN represents just such an affective response to errors. Luu and colleagues (2003), for example, suggested that it reflects distress associated with the violation of expectancy caused by the error (see also Tucker et al., 1999). They argued that the ERN reflects part of a limbic circuit for action regulation, a function that, in their view, encompasses both online control of action and learning from action outcomes. Activity in the circuit is coordinated by theta-frequency (4–7 Hz) oscillations, and the ERN reflects one portion of this theta activity (Luu et al., 2004). Luu and Pederson (2004) suggested that this view is not necessarily inconsistent with the conflict-monitoring or error-detection accounts, as the detection of errors, conflict, or a loss of reward could cause an affective response (see also Yeung, 2004). In contrast to the RL-ERN and conflict theories, the affective/motivational theory has not been formalized as a quantitative model.

Core Empirical Phenomena

Although new theories of the ERN (e.g., Jocham & Ullsperger, 2009) and of the ACC (e.g., Anderson et al., 2008; Brown & Braver, 2005) continue to emerge, the four perspectives outlined above have dominated the literature. This section is concerned with the empirical evidence relevant to these theories. First, we review some of the major empirical findings that are not closely aligned to any particular theory of the ERN.

Speed/Accuracy Emphasis (or Error Probability)

The speed–accuracy trade-off describes the fact that subjects can respond quickly, making many errors, or slowly, avoiding errors (Pachella, 1974). Studies that have instructed subjects to perform under varying levels of speed-versus-accuracy emphasis have tended to show that speed emphasis decreases the amplitude of the ERN relative to accuracy emphasis (Falkenstein et al., 1990, 1995; Gehring et al., 1993; see also Ganushchak & Schiller, 2006; Hajcak et al., 2003b; Ullsperger & Szymanowski, 2004). The Gehring et al. study compared error responses of exactly the same latency, establishing that the slowing of responses in the accuracy condition was not responsible for the effect. However, by definition, a speed/accuracy manipulation is confounded with error probability, so it is possible that such findings could be explained by the principle that unexpected outcomes elicit more activity than expected ones (Alexander & Brown, 2010; Oliveira et al., 2007).

Individuals can differ in their speed and accuracy, but studies relating the ERN to individual differences in performance are less consistent in their findings. Some studies have reported that high-accuracy subjects showed larger ERNs (e.g., Hajcak et al., 2003b; Pieters et al., 2007). Others failed to find such a relationship (Falkenstein et al., 2000; Mathewson et al., 2005). A negative finding was also reported in one attempt to relate the ERN to within-subject, block-by-block fluctuations in accuracy (Ullsperger & Szymanowski, 2004). Falkenstein and colleagues (2000) have suggested that it is time pressure per se, rather than error rate, that accounts for the findings of studies that manipulate speed/accuracy emphasis, because subjects in their study who differed in error rate showed equivalent ERNs (Falkenstein et al., 2000; see also Ullsperger & Szymanowski, 2004). Note, however, that analyses relating the ERN to differences in performance across subjects or across task blocks may not be conclusive tests of the hypothesis that speed/accuracy emphasis modulates ERN amplitude. Speed and accuracy are determined by many factors other than strategic trade-offs, including fatigue and endogenous lapses of attention (e.g., Weissman et al., 2006).

Each of the major theoretical approaches can accommodate the effects of speed/accuracy instructions on the ERN. Yeung et al. (2004b), using the conflict-monitoring framework, showed that varying response thresholds and attentional focus affected simulated response conflict in a manner consistent with empirical observations of speed/accuracy effects on the ERN. Error detection accounts of the ERN can also explain these observations. Gehring et al. (1993) argued that instructions emphasizing accuracy over speed enhanced the error-monitoring process underlying the ERN. Similarly, Falkenstein et al. (2000) suggested that accuracy emphasis strengthens the representation of the correct response, yielding a stronger mismatch signal on error trials. The enhanced ERN related to the

unexpectedness of low-probability errors would be consistent with the RL-ERN and PRO accounts. Finally, emotional accounts of the ERN could argue that emphasizing accuracy affects the ERN by making subjects experience errors as more aversive.

Error Detection and Correction

A series of studies conducted by Rabbitt and Laming beginning in the 1960s showed that subjects attempt to prevent error commission and, failing that, try to correct their errors and avoid subsequent mistakes (Laming, 1968; Rabbitt, 1966, 1967, 1968, 1981; Rabbitt et al., 1978). The theories outlined above predict a relationship between the ERN and compensatory behavior. Error-detection accounts of the ERN suggested that corrective behavior would be more likely or greater in magnitude when the ERN accompanying an error is larger (Falkenstein et al., 1995; Gehring et al., 1993). The RL-ERN model says that the ERN represents a process that reallocates control among various motor controllers, suggesting that the size of the ERN is related to changes in response strategy (Holroyd & Coles, 2002). The conflict-monitoring model makes similar predictions, because the conflict-monitoring system can drive changes in response strategy and attentional focus (Botvinick et al., 2001).

ERROR CORRECTION

When an error occurs, subjects often make the correct response soon afterward (e.g., Rabbitt, 1966). Findings concerning the relationship between the ERN and such immediate error corrections are inconsistent. Some studies have found that corrected errors are accompanied by larger ERNs than uncorrected errors (Falkenstein et al., 1995, 1996; Gehring et al., 1993; Rodríguez-Fornells et al., 2002), but others have failed to find such a difference (Fiehler et al., 2005). As for ERN latency, several studies have reported later ERNs when errors are not corrected than when they are corrected, which could mean that the ERN must occur quickly if it is to assist in error correction (Falkenstein et al., 1996; Fiehler et al., 2005; Hoffmann & Falkenstein, 2010). However, Rodríguez-Fornells et al. (2002) failed to find a latency difference. One might also predict that the error trials with the largest ERNs would also be corrected most quickly; this prediction has been confirmed (Gentsch et al., 2009; Rodríguez-Fornells et al., 2002) as well as disconfirmed (Fiehler et al., 2005). Group differences are also inconsistent with a link between the ERN and immediate error corrections. Whereas older adults typically show reduced ERNs relative to younger controls (e.g., Falkenstein et al., 2001b; Gehring & Knight, 2000; Mathewson et al., 2005; Nieuwenhuis et al., 2002), they can show this difference yet still correct their errors equally often (Falkenstein et al., 2001b; Gehring & Knight, 2000).

Most studies record button-press responses, limiting the analysis to the latency of the switch closure and its accuracy. It is more useful when other movement parameters, such as force or velocity, are also recorded. When response force data are available, it is usually observed that errors are less forceful than correct responses, suggesting that subjects may be inhibiting the error as it is being executed (Carbonnell & Falkenstein, 2006; Gehring et al., 1993; Rabbitt et al., 1978). The existence of partial errors, where there is electromyographic (EMG) activity that does not result in a button press, also supports this idea (Burle et al., 2008; Coles et al., 1985). The ERN could reflect activity involved in inhibiting the error in two ways: First, the error signal could give rise to the inhibition (in which case a larger error signal presumably would call for more inhibition); second, the activity itself could have an inhibitory effect. Here again the evidence is mixed. Gehring et al. (1993), using a single-trial measure of ERN amplitude, showed that larger ERNs tended to be associated with less forceful responses. In a go/no-go task, however, Scheffers et al. (1996) found no such relationship. Carbonnell and Falkenstein (2006) did not find that ERN amplitude differed between partial and full errors, but they found that ERN latency was shorter for partial errors than for full errors (see also Vidal et al., 2000; Figure 10.6). Endrass and colleagues (2008) also found that partial errors were accompanied by ERNs that were earlier, but in their study those ERNs were smaller than those associated with full responses. These error-inhibition analyses suffer from some ambiguities: responses might be less forceful or incomplete because of mutual inhibition between the error and the correct response (Ohtsuki, 1981) rather than an inhibitory process undertaken to stop the error. Thus far, the literature has failed to distinguish between these alternatives. Nevertheless, the latency analyses are especially intriguing, because they could indicate that an ERN—if it represents an attempt to stop the error response—has to be early to be effective. If the process intervenes early enough, the response can be stopped, but if it is too late, the response will proceed to completion.

The ERN occurs after lateralized readiness potential (LRP; see Chapter 9, this volume) activity

associated with activating the error response, and in some reports following the onset of LRP activity associated with the error correction (Burle et al., 2008; Rodríguez-Fornells et al., 2002). Some have suggested that this latency makes the ERN too late to reflect activity that inhibits or corrects the error (Rodríguez-Fornells et al., 2002). Burle et al. (2008) conducted perhaps the most detailed study of the timing of the ERN relative to measures of EMG, LRP, and behavior (see Figure 10.13). In their study, the onset of the ERN did not vary in latency when the error corrections were fast compared with when they were slow. However, the duration of the ERN was longer, and the peak was higher (and later) when corrections were late. The ERN itself appeared in the interval between the error and the error correction. As Burle et al. suggest, the results seem as if the ERN was interrupted by the error correction: the ERN began at a set time following the error but was then interrupted by the beginning of the error correction. The authors hypothesized that the ERN represents an alarm signal that lasts until remediation of the error begins.

Nevertheless, it is not hard to come up with alternative hypotheses that are also consistent with the time course of the ERN reported by Burle et al. (2008). For example, the ERN could represent a process necessary for error correction. Late error corrections could be late because they required more of this processing (that is, a greater-amplitude and longer-duration process). In order to execute a fast error correction, it may be necessary to suppress a still-active erroneous motor program to permit the correct response to occur. One computational model includes such a process: in the executive-process interactive control (EPIC) cognitive architecture (Meyer & Kieras, 1997), a motor memory buffer retains recently programmed movement features. Those features remain until a different movement is required. At that point, the movement features must be deleted from the buffer so that the new movement can be programmed. Random variability in the activation of the movement features to be deleted would be enough to produce variability in the time needed to delete them, but there might be other reasons as well. Could the ERN reflect some process that is needed to enable the error correction by getting the just-executed motor program out of the way? It would be difficult to disprove such a hypothesis using ERN latency measures without showing trials where the error correction was completed—and not simply initiated—prior to occurrence of the ERN.

A plausible alternative hypothesis would be compatible with an amended version of the conflict-monitoring theory in which the conflict signal is used as a control signal that resolves conflict (e.g., Seymour & Schumacher, 2009). Some error corrections may have

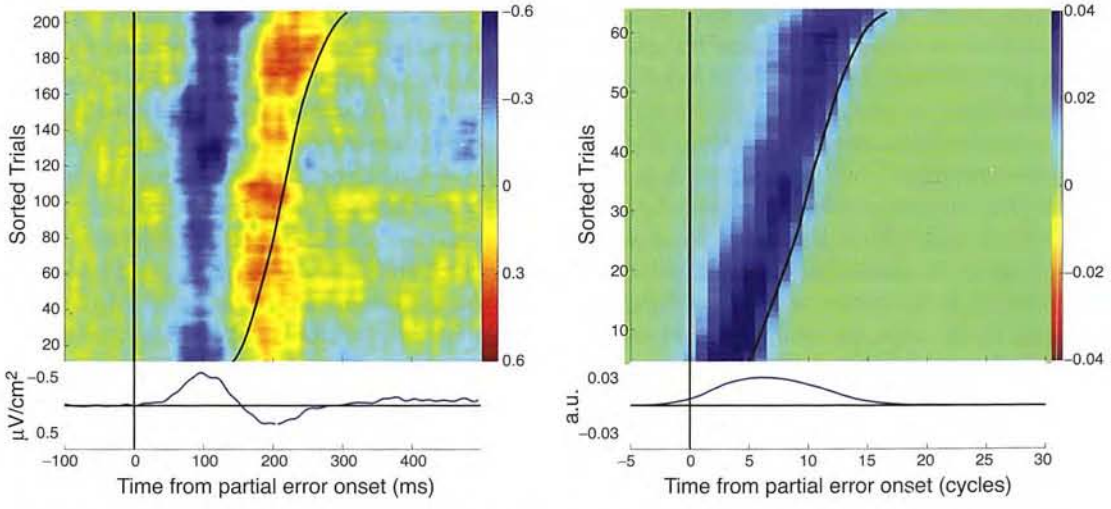

Fig. 10.13. Analysis of single-trial experimental ERN data (left) and simulated data (right) derived from the conflict-monitoring computational model. The trials all consisted of partial error responses followed by correct responses. The surfaces represent those trials time-locked to the onset of the partial error, sorted according to the length of the interval between the partial error and the subsequent correct response (indicated by the black line). In the experimental data, the ERN appears as a blue patch occurring on all trials around 100 ms following the partial error; the amplitude and duration grow larger as the interval between the partial error and the correct response lengthens. In the simulated data, the conflict model predicts that the time of maximum conflict will correspond most closely with the time of the correct response. Thus, the experimental data disconfirm the prediction of the conflict model. From Burle et al. (2008), Figure 3, reprinted with permission from MIT Press.

been late because there was more conflict on those trials, perhaps because of random variability in error-response activation. The ERN could represent a process that resolves that conflict (rather than simply detects it); thus, the size and duration of the ERN would be proportional to the conflict—still present following the error—that must be resolved for an error correction to be initiated.

Consistent with these two possibilities (which might be termed the *error-clearing* and *conflict-resolution hypotheses*, respectively), in the data of Burle et al. (2008) the late corrections were also associated with a greater amount of error EMG activity, such that the amount and duration of the ERN corresponded to the amount and duration of EMG activity associated with the eliciting error. Interestingly, the peak of the ERN in their study corresponded roughly to the offset of the error EMG activity. It is clear that ERN research would do well to probe the generality of the findings of Burle et al., applying their approach to a broader range of tasks and subject strategies.

There is an additional complication that must be considered in relating the ERN to measures of error correction: if the ERN represents a process involved in preventing or suppressing an error, then error trials represent those trials where the ERN occurred but failed. For example, the notion that the ERN, occurring after the error, is too late to reflect suppression of the error response itself overlooks the possibility that errors might be precisely those cases in which ERN activity occurred too late to be effective. Successfully inhibited error trials will (by definition) not occur. Thus, the typical ERN average could suffer from a selection bias: in this case, there could be another set of trials on which error activity occurs but the ERN occurs quickly enough to inhibit the error. Those trials will ultimately appear as correct trials; therefore, the resulting estimate of ERN latency will not include those trials (see Isoda & Hikosaka, 2007, for similar ideas applied to single-unit recordings from the pre-SMA). This ERN would not appear as a classic CRN (following the onset of the response); rather, it would occur prior to the correct response, possibly not even appearing in the average waveform because of latency variability (although the N2 activity on conflict trials identified by Yeung et al., 2004b, would be consistent with such a process).

A similar principle makes it difficult to come up with an airtight prediction about the relationship between the ERN amplitude and the likelihood of an error correction. If the process reflected by the ERN is involved in clearing or deleting portions of the error motor program to enable correction of the error, the appearance of an error correction will depend on whether the process represented by the ERN is successful. If a lot of activity is required on some trials because clearing out the error motor program is difficult on those trials, it is possible that the process could fail to delete the error program more frequently on those trials, creating an inverse relationship between the size of the ERN and the likelihood of error correction. Running counter to this tendency, however, could be a (possibly coexisting) dependence of the ERN amplitude on the likelihood of detecting the error, in which larger ERNs would appear when error corrections are more likely. Thus, the relationship between the amplitude of the ERN and parameters of error correction behavior could depend on the precise quantitative relationship between the detectability of an error (which itself could depend on the particular stimuli and movements involved) and the relative difficulty of correcting one type of error when another type is occurring. These and other task- and context-dependent factors have been mostly ignored by computational models and verbal theories.

STRATEGIC ADJUSTMENTS

Detection of an error can also lead to strategic adjustments—changes in strategy, attentional focus, response bias, or other parameters that will decrease the likelihood of future errors. The distinction between within-trial error corrections, reviewed above, and such strategic adjustments is similar to the distinction drawn by Donchin and coworkers (1978) between tactical and strategic information processing: the subject not only has the tactical goal to be correct on a single trial, but also strategic goals: to achieve an overall level of accuracy and response time, to please the experimenter, to obtain course credit or payment, and so on. Post-error slowing is generally assumed to be a strategic adjustment in which a subject slows down after making an error that happened when the subject responded too quickly (Rabbitt, 1981). If so, the size of the error signal could be related to the amount of slowing undertaken afterward. Gehring et al. (1993) used a single-trial measure of ERN amplitude and showed greater ERN amplitudes associated with increased post-error slowing. Other studies reported similar findings (e.g., Debener et al., 2005; Ladouceur et al., 2007; Rodríguez-Fornells et al., 2002). Some, however, failed to find a significant relationship (Dudschig & Jentzsch, 2009; Gehring & Fencsik, 2001; Hajcak et al., 2003b; see Figure 10.14).

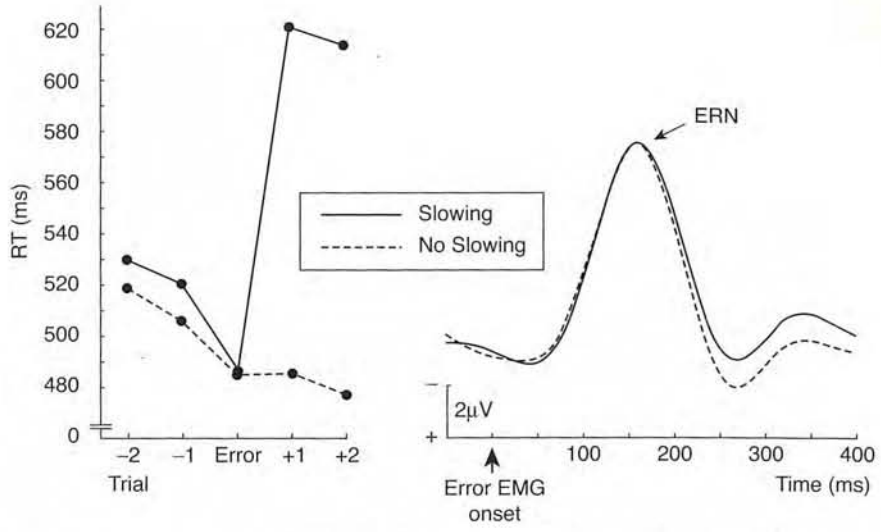

Fig. 10.14. Event-related potential waveforms from error trials that demonstrated post-error slowing (solid lines) or an absence of post-error slowing (dashed lines). The authors found no relationship between ERN amplitude and the degree of post-error slowing. Error trials in the two averages were paired according to reaction time to eliminate any confound between error reaction time and post-error slowing (as shown by the corresponding reaction time data on the left). From Gehring and Fencsik (2001), Figure 8, reprinted with permission from the *Journal of Neuroscience*.

A reason for some of the inconsistency may be that many processes can intervene between the ERN elicited by an error and the trial that follows that error, and that slowing depends in part on those processes that follow the ERN. Pe is one example of such a post-ERN process (Overbeek et al., 2005), but there are other post-ERN processes: Marco-Pallarés et al. (2008) found an oscillatory potential in the beta frequency range that correlated with the theta power underlying the ERN and (like the theta power itself) predicted post-error slowing.

Several dissociations between ERN amplitude and post-error slowing have appeared in the literature. Ullsperger and Szymanowsky (2004) found that emphasizing accuracy increased both the amplitude of the ERN and the degree of post-error slowing. Nieuwenhuis and coworkers (2001) compared errors of which subjects were aware with those that they did not detect. Perceived errors were associated with post-error slowing but unperceived errors were not, despite equivalent ERN amplitudes. Drug effects provide similar dissociations: for example, neither yohimbine (Riba et al. 2005a), alprazolam (Riba et al., 2005b), nor oxazepam (Johannes et al., 2001a) affected post-error slowing, although the former drug increased the size of the ERN and the latter two reduced it.

Various between-group associations and dissociations involving the ERN and post-error slowing have been reported. Alain and colleagues (2002) found both a reduced ERN and a reduction in post-error slowing in individuals with schizophrenia, whereas reduced ERNs with no changes in post-error slowing have been observed in schizophrenia (Mathalon et al., 2002) and Huntington's disease (Beste et al., 2008). Studies have shown that individuals scoring high on a worry questionnaire (Hajcak et al., 2003a), individuals with obsessive-compulsive symptoms (Hajcak & Simons, 2002), and individuals with major depression (Chiu & Deldin, 2007) showed increased ERNs relative to controls but the same level of post-error slowing. Gehring and Knight (2000) found that individuals with PFC lesions and age-matched (older) adults showed reduced ERNs, but equivalent post-error slowing, relative to young subjects. Swick and Turken's (2002) patient with ACC damage and a reduced ERN showed normal post-error slowing (complementing other reports of normal post-trial adjustments in individuals with ACC damage; Fellows & Farah, 2005; Modirrousta & Fellows, 2008). Band and Kok (2000), however, found that older subjects had a reduced ERN and an increase in post-error slowing (see also Falkenstein et al., 2000).

Several problems limit the utility of studies of post-error slowing in testing the putative link between the ERN and strategic adjustments. First, analysis of post-error effects is susceptible to confounds between previous-trial accuracy and previous-trial response latency. If error and correct trials show systematic differences in response time, then so-called post-error effects could be attributable

to previous-trial response time instead of previous-trial accuracy. Few ERN studies have matched post-error and post-correct trials on foregoing reaction time; for exceptions, see Gehring and Fencsik (2001), Hajcak and Simons (2002), and Hajcak et al. (2003b).

A more insidious problem in studying post-error slowing is identifying the function of slowing. Most of the research has simply assumed that post-error slowing reflects a strategic increase in control (e.g., Botvinick et al., 2001). Suprisingly, this assumption has remained relatively untested until recently. Some evidence supports it: Hajcak and Simons (2008) found that errors that followed errors were associated with reduced post-error slowing, implicating insufficient strategic control as a cause of the double errors. Nevertheless, post-error slowing might instead occur because the same problem that caused the error in the first place (such as a lapse of attention) persists until the subsequent trial. Alternatively, subjects may divert attention to process the error, interfering with primary task processing. Supporting this explanation is evidence showing that post-error slowing is enhanced at short response–stimulus intervals (Dudschig & Jentzsch, 2009; Jentzsch & Dudschig, 2009). The infrequency of the error relative to other events may cause automatic attentional capture that is unrelated to the status of the event as an error (Notebaert et al., 2009), or the shift of attention may occur because of a capacity-limited error-monitoring process that can contribute to strategic control (Dudschig & Jentzsch, 2009; Jentzsch & Dudschig, 2009). Such alternative accounts are supported by reports of decreased accuracy on trials following errors relative to those following correct trials (Hajcak et al., 2003b) and of groups (such as the individuals with depression in Compton et al., 2008) that show enhanced post-error slowing but decreased accuracy following errors relative to controls. Future research would do well to take these alternative explanations for post-error slowing more seriously. In particular, it should no longer be assumed that post-error slowing is a straightforward index of cognitive control.

ERROR DETECTION

The issue of whether the ERN is related to conscious detection of the error is distinct from the issue of whether it is related to error correction: a correct response can follow an error simply because of continued processing of the stimulus and thus does not necessarily indicate that the error was detected (Gehring et al., 1995; Ullsperger & von Cramon, 2006a). Consequently, definitive evidence of error detection is possible only if the subject makes a response signaling that an error has occurred (Rabbitt, 1968). Findings relating the ERN to such error detection responses are inconsistent. Two studies using antisaccade tasks have reported that ERN activity is as large for errors that the subject fails to detect as it is for those that the subject can report (Endrass et al., 2007; Nieuwenhuis et al., 2001). In contrast, Wessel and colleagues (in press) recently reported two antisaccade experiments in which the ERN was larger in response to perceived errors than to unperceived errors. To explain the discrepancy, the authors pointed out that the ERN was numerically larger for perceived errors in the Endrass et al. study (implicating an issue with statistical power), and that task requirements in the Niewenhuis et al. study may have introduced a response bias toward not signaling an error.

Some studies of manual responses have shown larger ERNs when subjects signal that an error has occurred than when they are uncertain or fail to report the error (Scheffers & Coles, 2000; Ullsperger & von Cramon, 2006a), supporting a link between the ERN and awareness of the error, but other studies suggest the link might be tenuous. Steinhauser and Yeung (2010) reported that the ERN was larger for detected errors than for undetected errors, but (somewhat paradoxically) that the ERN was not affected by an incentive manipulation that changed the subjects' criterion for signalling that an error had occurred. Because of the latter finding, the authors suggested that the ERN was not directly involved in error detection, but instead that the difference between detected and undetected errors might have reflected different reaction times (and response conflict) associated with the two types of errors. Maier and coworkers (2008) reported a flanker study in which subjects detected errors on incongruent trials more often when the response did not match the flanker letter (nonflanker error) than when the response and the flanker letter matched (flanker error). Paradoxically, the latter condition showed the largest ERN. This result is difficult to interpret, however, because the two conditions were not matched: the a priori probability of an error being a nonflanker error was twice that of its being a flanker error, presenting a potential confound in the perceived likelihood of the two error types.

Evaluation

How the ERN relates to measures of compensatory behavior and conscious error detection is far

from clear. At least some of this ambiguity may result from the difficulty of controlling for all possible confounds, as in the example of post-error slowing discussed above. The lack of robust findings relating the ERN to post-error behavior would seem to be evidence against the RL-ERN and conflict-monitoring theories, because both theories argue that some relationship should hold between the amount of ERN activity and post-error strategic adjustments (Botvinick et al., 2001; Holroyd & Coles, 2002). However, in both cases, there is no reason why one could not devise models that employ the same computations in the service of immediate error correction or inhibition rather than strategic control. Thus, some central ideas of these models—that the ERN represents a signal sensitive to the reward properties of the response outcome, or that it reflects conflict between responses—could potentially be instantiated in different computational models that make different predictions. This situation points to the need for greater effort in developing and evaluating competing computational models and alternative architectures. The ambiguity in these studies also points to a need for a better understanding of the underlying processes that result in post-error slowing and error detection responses.

Tests of the Theories

In this section, we focus on studies that seem to be more relevant to some subset of the theories than to the others. In some cases, it is apparent that the various theoretical perspectives on the ERN have led to different research agendas: some phenomena that are important to one theory are relatively unimportant to or discounted by others.

Error Detection and Conflict Monitoring

ERROR-CORRECT MISMATCH

Each of the major theories of the ERN links the ERN to a computation representing the difference between information corresponding to the correct response and information corresponding to the error. According to error-detection accounts, the ERN reflects a comparison between information corresponding to the actual (erroneous) and the intended (correct) response. The RL-ERN model links the ERN to learned values of stimulus–response conjunctions (Holroyd et al., 2005); the ERN reflects the comparison of the values of correct and incorrect stimulus–response conjunctions. Although the conflict-monitoring account is sometimes positioned as incompatible with one based on a mismatch-based error detector, the conflict computation is nevertheless sensitive to the difference between the response representation associated with an error and that derived from continued processing of the stimulus (see Holroyd et al., 2005, and Yeung et al., 2004b, for detailed comparisons of the mismatch theory and conflict-monitoring theory).

Because each of these models relates the ERN to error-correct mismatch in the sense we have described here, each one predicts (or could predict) that the amplitude of the ERN should be sensitive to the similarity of the representations involved in the comparison or conflict computation. Nevertheless, surprisingly, the bulk of the empirical studies and computational models in ERN research have limited themselves to two-choice tasks that permit little exploration of the role of representational mismatch. Here, we discuss the few exceptions to this tendency (for additional discussion of this issue, see Falkenstein et al., 2000).

A small number of studies have examined the effects of response similarity on the ERN. According to hierarchical models of response selection, the choice of an effector begins with relatively coarse decisions (e.g., right hand vs. left hand) and progresses to increasingly specific decisions (e.g., right index finger vs. right middle finger; Bernstein et al., 1995). The later in selection two responses diverge, the more similar they are. Using this logic, Bernstein et al. (1995) and Falkenstein and colleagues (1996) found that ERN amplitude increased as error and correct representations grew more dissimilar, consistent with a mismatch account. In contrast, Gehring and Fencsik (2001) reported greater ERN amplitudes for errors with representations similar to the correct response. Neither type of result, however, is definitive: the studies assume a certain kind of response representation (based on side or effector), and it is possible that similarity might be based on other movement parameters (such as velocity, force, etc.). Alternative computational models that incorporate different conceptions of response similarity would be helpful in testing the response mismatch hypothesis.

Another important unresolved issue is the role of stimulus similarity. Mismatch could involve the stimulus associated with (or predicted by) the error response and the stimulus that actually occurs (Schmidt & Gordon, 1977). Although Bernstein et al. (1995) found that stimulus similarity did not affect ERN amplitude significantly, the direction of the effect was consistent, with larger ERNs being associated with dissimilar stimuli than with similar stimuli. That study did not present waveforms, and

it involved numerous other conditions, suggesting that additional study is warranted. Elton et al. (2004) also reported that stimulus mismatch did not affect the ERN in an experiment using auditory stimuli separated by different degrees of pitch (Figure 10.15A). However, their data actually showed a significant interaction with electrode site, such that at Cz the ERN amplitude was greater for errors with large stimulus deviations relative to small-deviation errors. Perhaps the most surprising study examining stimulus mismatch is that of Yeung et al. (2007b), who showed that simply increasing the brightness of the stimulus increased the amplitude of the ERN (Figure 10.15B). Orr and coworkers (2008; see Figure 10.15C) showed that ERN amplitude was greater when stimuli could be distinguished on the basis of simple visual features than when the stimuli shared common features. Similarly, Vocat et al. (2008) found that false-alarm errors in a go/no-go task caused by a salient stimulus dimension elicited a larger ERN than those caused by a less salient stimulus dimension. Although the idea of mismatch is generally associated with the error-detection theory, results of this kind are consistent with all extant theories of the ERN (as Yeung et al., 2007b, note): whether the ERN reflects error detection, conflict detection, or the motivational or emotional significance of error commission, stronger representations of the correct response should produce larger ERNs. Note that studies of mismatch must be careful that the representational mismatch manipulation does not produce stimuli that are so difficult to discriminate as to make subjects uncertain about which stimulus actually occurred (Pailing & Segalowitz, 2004a).

The congruency manipulation in conflict tasks provides another way to manipulate the level of mismatch: when the irrelevant and relevant dimensions of a stimulus both correspond to the correct response (as in a compatible flanker stimulus such as *HHHHH*), there is more information to serve as the basis of error detection or to cause response conflict than when only the relevant dimension signals the correct response (as in *SSHSS*). Here, the results seem to depend on the task. Scheffers and Coles (2000) found that congruent stimuli in a flanker task were associated with larger ERNs than were incongruent stimuli (but see Bartholow et al., 2005). More recently, Forster and Pavone (2008) found that congruent stimuli were associated with larger ERNs than incongruent stimuli in a task with tactile target stimuli and spatially incompatible visual distractors. Christ and colleagues (2000) found no difference in the ERNs elicited by congruent and incongruent stimuli in both a Simon and a spatial-Stroop task. There are a number of differences in the paradigms, methods, and subject performance that could account for these inconsistencies. In particular, it is important to note that although these studies all involve conflict tasks, they differ in the manner in which the overlap between stimulus and response dimensions engenders conflict (see Kornblum et al., 1990). That is, the studies differ in which parts of which representations are in conflict. It appears that to resolve some of these contradictory findings, the ERN literature would benefit from further study of how the effects of congruency on the ERN may depend on the specific aspects of the stimulus or response representations that are incongruent.

In sum, evidence for roles of both response and stimulus mismatch in ERN generation is limited and mixed. Studies supporting a role of response similarity in mismatch are contradictory and have not considered thoroughly the stimulus and response representations involved in the computations that result in an overt response. Evidence against a role of stimulus similarity is not convincing. The studies reviewed above employed a wide range of tasks and manipulations of response and stimulus similarity, complicating direct comparisons among studies and a synthesis of results. Based on the evidence, some role for stimulus representations in computing mismatch is plausible and worthy of further experimentation, although experiments must be careful to control for the effects of error probability and to dissociate the role of stimulus similarity in the mismatch computation from its role in activating the competing responses.

LEVEL OF RESPONSE CONFLICT

Conflict theory asserts that greater amounts of response conflict should be associated with larger ERNs, and several studies have tested this claim. Some investigators have attempted to measure response conflict directly, based on the assumption that error corrections will compete with the errors that they follow. Carbonnell and Falkenstein (2006), for example, measured the force of the error and correct responses, reasoning that, all things being equal, a more forceful error response will compete more with the subsequent correct response than will a less forceful error. Their results showed no difference in the size of the ERN on the two types of trials. In a similar fashion, Masaki and coworkers (2007) used EMG measures to show that conflict was greater in a difficult task condition than in an

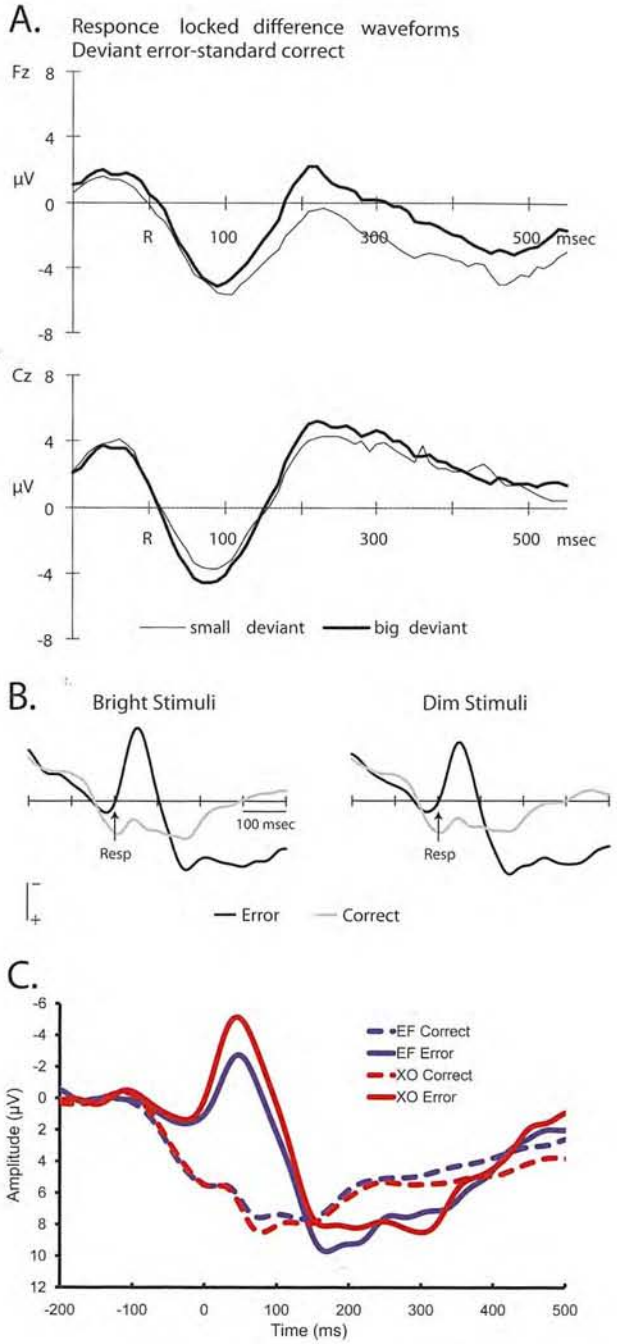

Fig. 10.15. Mismatch effects on ERN amplitude. (A) Response-locked error-correct difference waveforms elicited in a go/no-go task where the go stimulus was a standard auditory tone and the no-go stimuli were small or large auditory deviants. Errors were classified as no-go commission errors. There was no main effect of deviance on ERN amplitude, but a deviance by electrode (Fz vs. Cz) interaction revealed a significantly greater ERN at Cz for large deviants compared to small deviants. The opposite effect was reported at Fz, although the baseline and time course suggest that the Fz effect may have resulted from the Pe. From Elton et al. (2004), Figure 4, reprinted with permission of John Wiley & Sons, Inc. (B) Grand average response-locked ERP waveforms for correct and error trials in a flanker task with bright and dim (i.e., high- and low-luminance) stimuli. The authors found that ERN amplitude was reduced for dim stimuli. Modified from Yeung et al. (2007b), Figure 2, reprinted with permission from the authors. (C) Grand average response-locked ERP waveforms for correct and error trials in a variant of the flanker task examining the role of stimulus similarity on the ERN. Stimuli were composed of perceptually similar letters (EF trials; e.g., *EEEEE* and *FFEFF*) and perceptually dissimilar letters (XO trials; e.g., *XXXXX* and *OOXOO*). The ERN was larger on error trials in the perceptually dissimilar condition than in the perceptually similar condition. From Orr et al. (2008). Reprinted with permission from the authors.

easy one, yet the ERN amplitude was the same in the two conditions. Here, however—as we saw in the discussion of ERN latency above—the complicated logic of inhibitory processes makes the conclusions less compelling. If the error response on one trial is less forceful than the error response on another trial, it could be the case that the less forceful error was less forceful precisely because there was a great deal of response competition, and that the more forceful error occurred because there was little competition between the error and the correct response.

Studies such as this are on firmer ground in testing the conflict-monitoring model if they are based on simulations that establish the amount of response conflict in the conditions where ERNs are compared. For example, Burle et al. (2008) used conflict-model simulations to establish that—according to the computational model—there is less conflict when an error precedes an error correction by a long interval than when the two responses occur close together in time. Contrary to the predictions of the model, however, the ERN was larger when the interval between the error and the correction was long than when it was short (see Figure 10.13). Nevertheless, even this study might not be as damaging to the notion of conflict monitoring as it appears. To understand why this is the case, one must keep in mind that there is a distinction between the concept of conflict monitoring, which could be implemented computationally in a variety of ways, and the particular computational models of Botvinick et al. (2001) and Yeung et al. (2004b). The Burle et al. (2008) study showed that the predictions of that particular computational model did not hold. However, in alternative implementations of conflict monitoring, response conflict might not be evident in overt measures such as muscle activation or overt movement. Rather, the response conflict might be limited to covert activity, at the level of the primary motor cortex, or possibly even earlier in processing. Peripheral inhibitory processes can intervene between the cortex and overt behavior (De Jong et al., 1990; Ohtsuki, 1981), such that a high degree of conflict at the level of motor cortex might not result in overlapping movements. Such processes are not represented in the models of Botvinick et al. (2001) and Yeung et al. (2004b). Thus, it would appear worthwhile to explore alternative ways that conflict monitoring could be implemented computationally, with more detailed representations of the motor system, before drawing firm conclusions about how the size of the ERN should or should not vary with observable indications that conflict is present in the motor system.

N200/N450

According to the conflict model, ERN activity will occur on correct trials in conditions of high response conflict (Yeung et al., 2004b), appearing as an N200 preceding the correct response. Some studies, however, have reported dissociations of the N200 and ERN and argued against the similarity of these components. The ACC lesion patient of Swick and Turken (2002) showed a reduced ERN but a normal N200. Similarly, administering alcohol reduced the size of the ERN but did not affect the N200 (Ridderinkhof et al., 2002). Here, computational modeling has proven useful in showing that such dissociations need not imply that the components are distinct (Yeung & Cohen, 2006). In the case of alcohol, for example, a combined deficit in perceptual and attentional processing will reduce the size of the ERN but will not affect the amplitude of the N200 even if both represent the same conflict signal (Yeung & Cohen, 2006; Yeung et al., 2007b).

THE CRN

Observing the CRN sparked a challenge to the idea that the ERN represents part of an error-detection system: if an ERN can occur on a correct trial, then how could it represent the detection of an error (Vidal et al., 2000)? Recall that the CRN is an ERN-like potential whose peak follows the correct response by the same latency as the ERN peak follows the error response. As Vidal et al. point out, incorrect muscle activity cannot be the sole cause of the CRN on correct trials, because if there is some incorrect EMG activity, that activity will typically occur prior to the correct response, not after it. Possible explanations for the CRN include that it is an artifact of a stimulus-related N200 appearing in the response-locked waveforms, or that it is the result of correct responses that the subject's brain somehow mislabels as errors (Coles et al., 2001; Vidal et al., 2000). Supporting the latter interpretation are cases where an ERN-like potential accompanies slow, correct responses—responses that involve the correct effector but exceed a specific experimenter-imposed response deadline (Heldmann et al., 2008; Luu et al., 2000b). If subjects were to evaluate responses according to an internal deadline or some other subjective criterion, an ERN could occur (appearing as a CRN), despite the objective correctness of the response. This slow-trial CRN would grow larger as responses get later, making it consistent with

a mismatch- or comparator-based error detector, where the standard that defines an error is determined in part by the subject's internal criteria. It is less clear how the conflict-monitoring theory would predict a slow-trial CRN. Not all instances of the CRN are easily explained by implicit response deadlines, however: Vidal and colleagues (2003) reported one study without an explicit deadline in which the CRNs did not differ on fast and slow correct trials. They argued that the CRN is therefore not likely to reflect error detection. They suggest that the ERN and CRN are the same component, which may represent a comparison process that precedes error detection per se, or else an emotional response to the error, rather than error detection itself.

If the ERN is a reflection of conflict monitoring, one can see that CRNs occurring in conditions of high response conflict could reflect the detection of conflict at the time of a correct response. Consistent with such an idea, Bartholow and colleagues (2005) found that the CRN in a flanker task was affected by the presence of incongruent flanker stimuli on correct trials, and that this effect was influenced by the probability of incongruent flankers: conditions where congruent stimuli were frequent—and the flanker (conflict) effect on RT was largest—showed the largest CRN on incongruent-flanker trials. Response conflict could also explain the CRNs observed when individuals respond deceptively in a recognition task (Johnson et al., 2004). However, the conflict monitoring model predicts ERN-like activity *before* the correct response in conditions of high conflict (Yeung et al., 2004b); thus, the CRN, if it is indeed an ERN on correct trials, would argue against the conflict-monitoring model. Nevertheless, the existence of CRNs could be consistent with some alternative implementation of conflict theory or alternative parameterization of the current models. To our knowledge, there have been no attempts to see whether a model based on conflict monitoring could produce a CRN, although early conceptions of conflict monitoring seem to allow for this possibility (Cohen et al., 2000). As for the other theories, it is also plausible that ideas of affective distress could accommodate the existence of the CRN. It is less clear how the RL-ERN theory will treat the CRN. In the past, this theory explicitly concerned the difference between error and correct trials, and thus appeared to say nothing about the CRN (Holroyd & Coles, 2002). More recent work, however, has argued for the existence of positive-polarity activity occurring on correct trials (Holroyd et al., 2008), suggesting that future RL-ERN modeling work may have to specify why some correct trials yield a CRN and some yield a positivity. One clue may lie in the role of uncertainty, as some evidence suggests that the CRN may occur when subjects are more uncertain of the accuracy of their responses (Pailing & Segalowitz, 2004a).

One criticism of the ERN literature in general, which is particularly important in the case of the CRN, is the lack of a systematic attempt to compare the ERN and CRN to other motor potentials, especially those potentials recorded in voluntary movement tasks. For example, classic studies of movement-related potentials recorded during self-paced finger-flexion responses showed an N+50 component with a latency and a scalp distribution very similar to those of the ERN, although with a slightly larger amplitude contralateral to the response (Shibasaki et al., 1980a, 1980b). It is possible that the CRN is a distinct movement-related potential like the N+50 whose appearance depends on particular task or response parameters. With several other components occurring within the 100 ms just prior to and just after a voluntary response, determining whether the ERN and CRN are fundamentally the same will require a larger comprehensive effort to identify and compare all movement-related potentials in that time range. Such an effort would be a welcome development in the ERN literature.

The existence of the CRN and the possibility that there are other negative-going potentials similar to the ERN at the time of a movement highlight the difficulty in determining how best to define and measure the ERN: defining it as the difference between error and correct trials ignores the possibility that a CRN exists, yet defining it as a negative-going peak in the unsubtracted error- or correct-trial waveform risks misidentifying some other movement-related potential (such as the N+50) as the ERN. It is probably best to adopt a research strategy that incorporates both kinds of analysis (see, e.g., Tops et al., 2006, who refer to the difference between correct and error trials as the ΔERN).

PARSIMONY

One argument that has been put forth as favoring conflict theory is that the conflict model is more parsimonious than the error-detection model because the error-detection model requires a homunculus that can determine which response is correct (Botvinick et al., 2004; Carter et al., 1998; Yeung et al., 2004b). This argument has several weaknesses. First, claims of parsimony are impossible to support without a unified theory of cognition

(Newell, 1990) that describes a complete functional architecture of the brain, specifying how the elements in the model interact with other cognitive, perceptual, and motor systems. Simplicity in one part of such a theory (embodied in the model) might come about by making some part external to the model more complex. For example, because the conflict model does not represent the occurrence of an error, the model must be amended to achieve an explicit (conscious or declarative) representation that an error occurred. Yeung et al. (2004b) showed that adding a threshold to the conflict model can yield a signal that an error has occurred. Nevertheless, such a signal is achieved by sacrificing the simplicity that the conflict model claims as a virtue. In addition, the conflict-based error detector creates other issues to be resolved: is a homunculus necessary to set the threshold? Can a threshold-based error detector apply to more complicated movements that involve multiple effectors? Can the output of a threshold-based system yield a computation of error-correct similarity such as the one thought to underlie the mismatch computation in the error-detection model? Moreover, the work of Holroyd et al. (2005) showed that—contrary to the claims of the conflict theorists—an error-detection model need not invoke a homunculus. Perhaps even more important, claims of parsimony are not particularly relevant: in the philosophy of science, the notion that the simpler theory is more likely to be true is problematic (Hempel, 1966), one reason being that reality isn't necessarily simple. Or, as Gordon Logan once bluntly put it, "Parsimony is overrated" (G. Logan, personal communication).

EVALUATION

The debate between the conflict- and error-detection camps is far from over. All attempts thus far to measure conflict directly have disconfirmed, rather than confirmed, the predictions of the conflict-monitoring model. Nevertheless, difficulties with measuring conflict, and the possibility of amending the conflict model, suggest that these results are not conclusive. Despite the debate between the conflict monitoring theorists and others in the ERN community, some work has suggested that some of the findings explained by the conflict-monitoring theory can also be accommodated by a model with explicit error detection (Holroyd et al., 2005). With the exception of the CRN, which might disconfirm the timing of correct-trial ERNs predicted by the conflict-monitoring model, most findings could be accommodated within either framework. Better modeling work would attempt to compare error detection and conflict monitoring on an equal footing, within the same architecture, to come up with predictions that are unique to one or the other theory and would disconfirm the other (at least within that specific architecture). And for any model, alternative parameterizations should be explored that would make clearer the range of phenomena the model can predict (Roberts & Pashler, 2000).

The Reinforcement Learning Theory of the ERN

THE FRN

For the most part, the RL-ERN theory has been tested using the FRN rather than the classic ERN. The FRN has spawned its own active and growing literature (see Nieuwenhuis et al., 2004a). Some theorists propose that the FRN reflects another manifestation of the temporal-difference reinforcement-learning signal that causes the ERN (Holroyd & Coles, 2002). If so, studies of the FRN can provide a whole range of constraints on ERN theories. For example, the RL-ERN model of Holroyd and Coles (2002) predicts that early in learning, before subjects have learned the mapping between stimuli and responses, the error signal will tend to be elicited by feedback stimuli, but as learning progresses—once subjects know the mapping—the information necessary to produce the error signal will be available at the time of the error response. Thus, with practice, the FRN elicited by error feedback will grow smaller and the ERN elicited by an error response will grow larger. Several studies have confirmed that the ERN elicited by erroneous responses grows larger as subjects learn the stimulus–response mappings (Holroyd & Coles, 2002; Morris et al., 2008; Nieuwenhuis et al., 2002). The FRN results have been less consistent, with the predicted decrease absent in one study (Holroyd & Coles, 2002) and not significant in another (Nieuwenhuis et al., 2002). A third study showed a reversal in which, after learning, correct feedback elicited the FRN and error feedback did not (Morris et al., 2008). One key to understanding the discrepant results may lie in the findings of Eppinger and coworkers (2008), who did find the predicted learning-related decrease in the error-correct FRN difference waveform but also found that the learning-related effects were actually caused by the positivity elicited by correct feedback, not by the FRN.

Several studies have tested the claim that the FRN represents the detection of unexpected, unfavorable

outcomes (Holroyd & Coles, 2002). The evidence is mixed. While several studies have shown that the more unexpected the negative feedback, the greater the amplitude of the FRN (Bellebaum & Daum, 2008; Hajcak et al., 2007; Holroyd et al., 2003; Potts et al., 2006b), some studies have shown that the FRN also responds to unexpected yet favorable outcomes (Donkers & van Boxtel, 2005; Donkers et al., 2005; Oliveira et al., 2007), and Hajcak and colleagues have shown that the FRN is not always sensitive to probability manipulations (Hajcak et al., 2005a, 2007). Rather, the sensitivity of the FRN to probability might be limited to situations in which there is a learnable contingency between responses and outcomes (Holroyd et al., 2009).

Until recently, the consensus in the literature has been that the FRN varies with the outcome value in a binary fashion, reflecting the evaluation of outcomes as good or bad but not capturing the gradations in outcome value that would seemingly be predicted by the RL-ERN theory. When subjects can gain money, lose money, or break even (gain or loss of zero), the FRN does not distinguish losses from neutral outcomes (Holroyd et al., 2004a, 2006). Moreover, when losses that differ in monetary value are possible, FRN amplitude is equivalent for large and small losses (Hajcak et al., 2006; Nieuwenhuis et al., 2004b; Yeung & Sanfey, 2004). Although a binary response seems inconsistent with the sensitivity to value implied by the RL-ERN model (Holroyd & Coles, 2002; Holroyd et al., 2005). Holroyd et al. (2006) suggested that cognitive categorization processes could be added to the RL-ERN model to account for binary ERN responses. More recent evidence, however, complicates this picture. Goyer et al. (2008) observed FRN activity that was modulated by the magnitude of the reward, such that larger losses elicited greater FRN activity. Nevertheless, the time course and scalp distribution of the magnitude effect differed from those of the outcome valence effect, suggesting that the magnitude effect most likely represents a different component. Similarly, Bellebaum et al. (2010) reported FRN amplitudes on loss trials that grew larger with the magnitude of an unobtained reward, although the positivity preceding the FRN may have contributed to the peak-to-peak measure underlying their effect.

Whereas most of the initial work on the FRN examined the role of negative events like losses in eliciting the component, a more recent trend is a focus on how gains can affect the FRN. In the literature on dopamine, an unexpected positive reward causes a phasic increase in dopamine that is involved in reinforcement learning (Schultz, 2002). The RL-ERN theory contends that a positive dopaminergic response should result in a positivity observable at the scalp, just as a negative response results in a negativity. Studies have therefore begun to focus on whether positive outcomes can affect the FRN waveform. Potts and colleagues (2006b) proposed that the P2a elicited by unpredicted reward feedback represents this positive dopaminergic response. Similarly, Holroyd and coworkers (2008) argue that the FRN is the same ERP component as the stimulus-locked N200 and that the error–correct difference in feedback-locked waveforms results from the summation of the N200 with a gain-related positivity—the feedback correct-related positivity—that occurs when there is correct feedback. This interpretation suggests that the null effects of reward expectation on loss waveforms may not be inconsistent with RL-ERN theory: expectation effects may simply be more evident in gain waveforms. Consistent with this account, Eppinger et al. (2008) found that effects due both to learning and to feedback validity were seen in a positivity—not FRN or ERN. Also consistent with this notion, Santesso et al. (2009) showed that pramiprexole, a dopamine agonist that reduces phasic dopamine levels, blocked a gain-related reduction in the FRN that appeared to occur in controls (although there was no control condition to verify that reduction). A special status for gains is also evident in the finding that reward expectation modulated ERPs and EEG frequency spectra related to gains but not losses in a gambling task (Cohen et al., 2007) and that increasing the frequency of large gain stimuli caused smaller gains to elicit an FRN, but changing the frequency of large losses did not have an effect on small-loss FRNs (Nittono et al., 2008).

There is still work to be done in resolving some inconsistencies among these studies. Some studies argue that unexpected gains will elicit the same effect as unexpected losses, namely, a greater negativity than the corresponding expected condition (e.g., Oliveira et al., 2007), whereas others argue that unexpected gains will cause a positivity rather than a negativity (Holroyd et al., 2008; Potts et al., 2006b). The truth might even be a mixture of these possibilities: single-unit recordings in the ACC have shown some neurons sensitive to rewards, some sensitive to omitted rewards, and others sensitive to both (Ito et al., 2003). Moreover, whether there is a purely feedback-related negativity at all is a new point of contention, with one position being that

FRN effects occur because of an overlapping correct-related positivity (Holroyd et al., 2008). Some recent evidence based on a spatiotemporal principal components analysis (PCA) may support the existence of a reward-related positivity: in data from a gambling task, the only component emerging from the PCA that matched the latency of the FRN was a positivity on gain trials, although the raw data showed the typical FRN on loss trials and no discernible peak on gain trials (Foti et al., 2011). Still, it would be reassuring to see some experimental condition that could actually produce a positive-going deflection at the latency of the FRN. One report showed a so-called reward positivity whose latency was earlier than the FRN, making an account of the FRN based on component overlap untenable (Holroyd et al., 2011). In learning and gambling tasks, the gain trial waveform often shows no peak—positive or negative—whose latency matches that of the FRN (see, e.g., Figure 10.7). To explain those cases as reward-related positivity effects, one would have to assume that there is always a negativity at the same latency that overlaps and precisely cancels out the positivity on those gain trials. Similar logic would also have to apply to the response-locked ERN.

Interestingly, as in the ERN studies reviewed earlier, little work has attempted to theorize about, model, or experimentally manipulate the stimulus representations that give rise to the feedback signal. Indeed, many studies confound the outcome valence with the stimulus representations that signal a gain or loss (e.g., +++ vs.−−−; Holroyd et al., 2004a). A complete theory of the FRN would have to specify the representation used in the underlying computation. In a recent study, we demonstrated a case where the FRN was elicited only when loss feedback could be distinguished from gain feedback on the basis of a distinctive visual feature, not when the discrimination was based on a conjunction of features (Liu & Gehring, 2009). In another study, we asked whether the perceptual similarity of the feedback stimuli affects the FRN (Liu, 2008). We manipulated the similarity of the gain and loss feedback stimuli (*E* vs. *F* in one case and *S* vs. *T* in another) and added irrelevant flanking stimuli. Our results showed that the FRN was larger when the feedback stimuli were dissimilar than when they were similar, and that the irrelevant flanking letters elicited a FRN-like negativity, suggesting that the FRN was modulated by the perceptual properties of feedback stimuli (Liu, 2008; see Figure 10.16). Other studies have found the FRN to be influenced by perceptual factors (Jia et al., 2007). Of course, a difficulty such studies face is that manipulating the stimulus representations that give rise to the FRN could affect other N2-like potentials that overlap the FRN, and not the FRN itself, if the FRN is indeed distinct

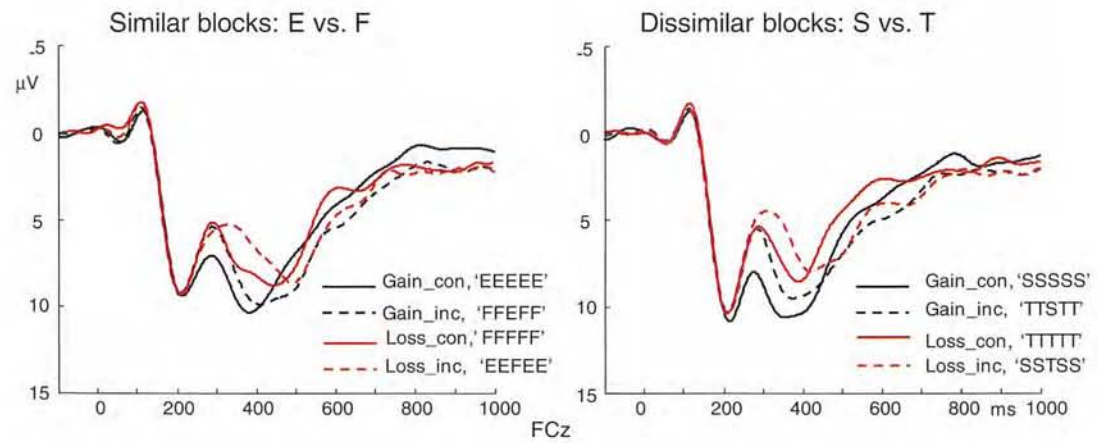

Fig. 10.16. Grand average ERP waveforms elicited by feedback stimuli indicating gains or losses in a gambling task. Feedback stimuli were composed of a central letter that indicated the subject's gain or loss, surrounded by irrelevant flanking letters that were either the same as or different from the central letter. Letters in the feedback stimuli were perceptually similar (*E* vs. *F*, left) or perceptually dissimilar (*S* vs. *T*, right). The feedback related negativity (FRN) was larger when the gain and loss feedback stimuli were perceptually dissimilar (*S* vs. *T*) than when they were similar (*E* vs. *F*). In addition, the FRN was affected by the presence of irrelevant flanking letters, such that a gain stimulus surrounded by irrelevant loss flanker stimuli elicited an FRN (i.e., the *FFEFF* and *TTSTT* waveforms). From Liu (2008). Reprinted with permission from the author.

from the other potentials. A challenge to the FRN literature is to disentangle the multiple N2-like potentials that occur after a feedback stimulus.

EXTENSION OF RL-ERN THEORY TO THE CLASSIC ERN

To extend the RL-ERN model to speeded response tasks, Holroyd et al. (2005) combined the reinforcement learning model with a model of speeded task performance similar to the conflict-monitoring model. The model is fairly complex, but the key insight is that states of the system are assigned values based on past performance outcomes. One layer of units within the system categorizes stimuli and activates responses, not unlike the mechanism in the conflict-monitoring model. Figure 10.12 shows the two additional layers responsible for the ERN. One layer represents the current stimulus and the current response. Critical to the processing in this layer are conjunction units, which are activated by a conjunction of stimulus and response units, such as the *H-left* (HL) unit that is activated when *H* is the stimulus and *left* is the current response. The second layer assigns values to different states within the state layer: if the instructions to the subject are to respond *H* with the left hand, then the *S-left* (SL) state would be assigned a negative value and the *H-left* (HL) state would receive a positive value. Finally, the temporal difference unit compares the current value layer with that predicted based on past reinforcements. If the value is negative, an error is signaled and adjustments are made to the motor control system.

EVALUATION

If the FRN reflects the same process as the ERN, the fact that it occurs well after the response is particularly damaging to a conflict-monitoring account of the ERN, because by the time feedback occurs, any response conflict has dissipated (although it is conceivable that conflict between the expected and actual feedback might be accommodated within a modified conflict model; van Veen et al., 2004). Nevertheless, there is some evidence suggesting that the two components are not identical. First, evidence reviewed below suggests that the ERN, but not the FRN, can be modulated by the value or motivational significance of the eliciting event. Also, although dissociations must be interpreted cautiously (e.g., Yeung & Cohen, 2006), ERN/FRN dissociations have been reported in OCD (Gründler et al., 2009), psychopathy and externalizing psychopathology (Bernat et al., 2011; Borries et al., 2010; Hall et al., 2007), trait anxiety (Gu et al., 2010; Hajcak et al., 2003a), and aging (Eppinger et al., 2008). Moreover, the classic ERN has a symmetrical midline scalp distribution, with its maximal amplitude at the scalp site FCz. The FRN, in contrast, typically has a scalp distribution that is more anterior and, in some cases, lateralized to the right (Gehring & Willoughby, 2004; Müller et al., 2005; Nieuwenhuis et al., 2004b; Potts et al., 2011), possibly because the FRN is generated by two sources, one in the posterior cingulate cortex and one in the rostral anterior cingulate or medial prefrontal areas (Müller et al., 2005; Nieuwenhuis et al., 2005a; van Veen et al., 2004). Consistent with a two-source model, Potts and colleagues (2011) reported a spatiotemporal principal components analysis supporting distinct frontal and central contributions to the FRN, with the latter possibly reflecting an ACC generator common to both the ERN and FRN.

Several other considerations suggest that it would be premature to conclude that the midbrain dopamine system is the sole or primary determinant of the ERN. First, as we review in the section on neurotransmitters below, the evidence linking the ERN to dopamine is mixed. Also, Jocham and Ullsperger (2009) and Frank and colleagues (2005, 2007) have suggested that the link between the ERN and dopamine may actually be opposite to that proposed by Holroyd and Coles (2002): The ERN could be caused by some nondopaminergic signal, with the ERN in turn causing a dopaminergic response in the basal ganglia (see Figure 10.17). Jocham and Ullsperger (2009) note that the midbrain dopaminergic system lacks the speed to generate the ERN as quickly as the Holroyd and Coles model would require, making it more plausible that some other neurotransmitter gives rise to the ERN before dopaminergic responses occur. Contradicting this assertion, however, is a report of intracranial field potentials in an individual with OCD showing error-related activity in the nucleus accumbens 40 ms prior to the ERN recorded at the scalp (Münte et al., 2008). The small sample size and likely presence of overlapping potentials point to the need for further study of this potentially important phenomenon.

ERN as an Affective Response

The proposition that the ERN reflects an affective response to error commission has motivated a search for the personality and emotional correlates of the

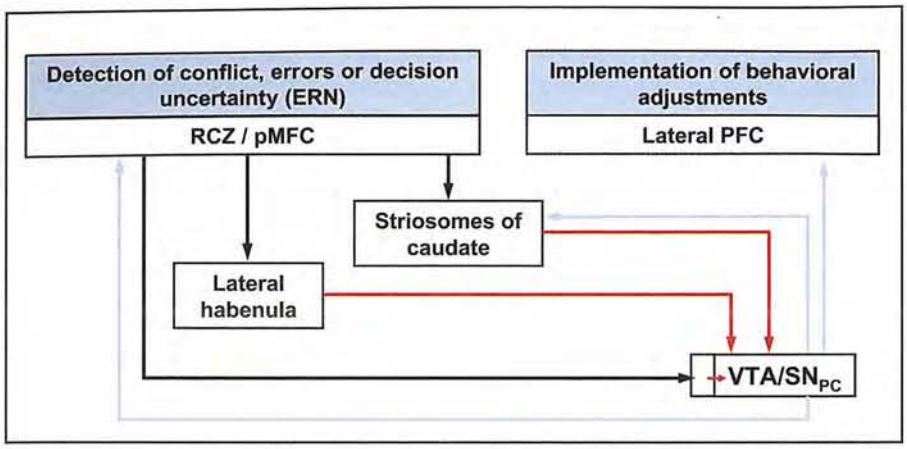

Fig. 10.17. A new model of the neuropharmacology of the ERN, showing three possible pathways through which an error signal from the ACC could inhibit midbrain dopamine neurons. One pathway projects directly from the rostral cingulate zone/posterior medial frontal cortex (RCZ/pMFC) to the ventral tegmental area and substantia nigra pars compacta (VTS/SNpc), which acts through GABAergic interneurons to inhibit dopamine neurons. Also, pathways through the lateral habenula and the striosomes of the caudate nucleus could inhibit midbrain dopamine neurons through GABAergic projections. The blue boxes show the corresponding cognitive processes; black arrows represent excitatory connections; red arrows represent inhibitory connections; blue arrows represent mesocortical and mesostriatal dopaminergic projections. From Jocham and Ullsperger (2009), Figure 2, reprinted with permission from Elsevier.

ERN. A variety of studies have reported links between emotional variables and ERN amplitude—often without providing an accompanying theory at the level of detail offered by the reinforcement-learning and conflict-monitoring models. It is important to note that, broadly speaking, affective and cognitive theories of the ERN are not mutually exclusive: affective states and traits could modulate the cognitive processes that underlie the ERN, or vice versa.

PATIENT STUDIES

The past decade has seen growing interest in the effects of neurological and psychiatric conditions on the ERN (for reviews, see Jocham & Ullsperger, 2009; Olvet & Hajcak, 2008; Ullsperger, 2006). Theories of obsessive-compulsive disorder (OCD) have attributed the exaggerated concerns and repetitive behaviors that characterize OCD to a hyperactive error signal (Pitman, 1987; Schwartz, 1997). Gehring and colleagues (2000) suggested that the ERN might represent such an error signal. They confirmed that individuals with OCD showed an exaggerated ERN relative to controls, and furthermore, that ERN amplitude was correlated with symptom severity (see Figure 10.18A). A number of studies have replicated and elaborated on this finding, both in individuals with OCD (Endrass et al., 2008; Johannes et al., 2001b; Ruchsow et al., 2005a; for an exception, see Nieuwenhuis et al., 2005b) and in individuals with subclinical symptoms (Hajcak & Simons, 2002). This effect appears to be unrelated to medication status when symptom severity is controlled (Hajcak et al., 2008; Stern et al., 2010). One might expect individuals with OCD to classify correct responses as errors, but some studies find elevated CRNs in OCD (e.g., Endrass et al., 2008; Hajcak & Simons, 2002) whereas others do not (e.g., Gehring et al., 2000; Hajcak et al., 2008; Stern et al. 2010). Interestingly, Hajcak and coworkers (2008) found that children with OCD show the exaggerated ERN both before and after treatment with cognitive-behavioral therapy, despite an improvement in symptoms. For this reason, they suggested that the ERN might serve as a useful endophenotype for OCD (Figure 10.18B).

Studies of the ERN in depression are somewhat inconsistent, with some studies reporting no difference between patients and controls (Ruchsow et al., 2004, 2006), others reporting an enhancement in patients (Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008), and another finding a reduction, but only in depressed individuals who also showed psychomotor retardation (Schrijvers et al., 2008). Interpreting these discrepancies is difficult because of differences among the studies in whether patients were in remission or currently depressed, were taking medication, and had a comorbid anxiety disorder, as well as in the severity of their depression. Comorbid

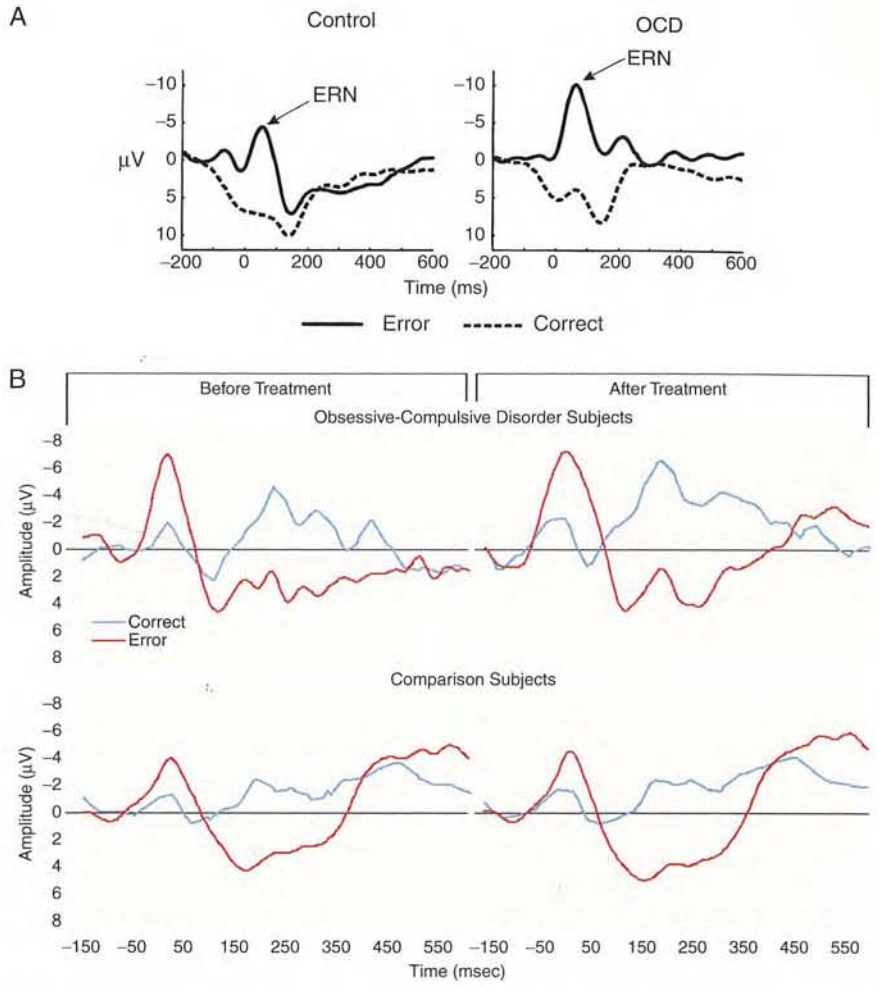

Fig. 10.18. (A) Error-related negativity waveforms from patients with OCD and healthy controls, showing a larger ERN in individuals with OCD. From Gehring et al. (2000), Figure 1, reprinted with permission of John Wiley & Sons, Inc. (B) Error-related negativity waveforms from children with OCD and controls, showing that the exaggerated ERN in OCD does not change with successful cognitive-behavioral treatment. From Hajcak et al. (2008), Figure 1, reprinted with permission from American Psychiatric Publishing, Inc.

anxiety, for example, was not excluded in either of the two studies reporting an enhancement. Schrijvers and colleagues (2009) suggested that ERN enhancement may occur in mild to moderate depression, in which anhedonia, apathy, and psychomotor slowing are less severe and the ERN-enhancing effects of comorbid affective distress are more apparent.

Studies using questionnaire measures are fairly consistent in showing elevated ERN amplitudes associated with negative affect and anxiety. Negative affect, as measured by self-report, has tended to be associated with increased ERN amplitudes (Hajcak et al., 2004; Luu et al., 2000a; but see Compton et al., 2008), although the effect may be limited to the period early in practice, before the individual with negative affect disengages from the task (Luu et al., 2000a). Questionnaire measures of trait anxiety (reflected in a high level of worry) and state anxiety are also associated with an increase in the size of the ERN (Hajcak et al., 2003a; Vocat et al., 2008, respectively). A few studies have examined the relationship between the ERN and the behavioral inhibition and activation systems (BIS/BAS) postulated by Gray (e.g., Gray, 1987). The BIS is associated with sensitivity to punishment, and greater BIS activation is associated with anxiety disorders. Boksem and colleagues (2006b) and Amodio and colleagues (2008) reported an enhancement of the ERN associated with high levels of behavioral inhibition. Cavanagh and Allen (2008), however, failed to find a global ERN difference between high- and low-BIS adults, although high-BIS individuals

showed a relationship between flanker-task ERN amplitude and cortisol reactivity in a subsequent condition involving social evaluative threat. In a longitudinal study, McDermott and coworkers (2009) found that children who showed elevated behavioral inhibition in early childhood (4–7 years old) exhibited enhanced ERN activity as adolescents (15 years old). There was a tendency among a subset of children with high behavioral-inhibition scores to show a relationship between ERN amplitude and the risk of an anxiety diagnosis.

Belief systems that may affect anxiety may also affect the ERN. Inzlicht et al. (2009) reported that individuals with greater religious conviction showed reduced ERNs. They suggested that religion provides a buffer that reduces anxiety, and they argued that political conservatism may provide a similar function (citing the finding of Amodio et al., 2007, that conservative individuals show a reduced ERN).

AUTONOMIC NERVOUS SYSTEM ACTIVITY

If the ERN reflects an affective response to error commission, then it would be expected to covary with error-related changes in autonomic nervous system (ANS) activity. Evidence for this proposition is mixed. Hajcak and colleagues found that errors were followed by greater skin conductance responses (SCRs) and heart rate deceleration relative to correct responses, but that the ERN amplitude did not correlate with either of these measures (Hajcak et al., 2004; see also van Boxtel et al., 2005). Individuals high in trait-negative affect show larger error-related SCRs, as well as larger-amplitude ERNs (Hajcak et al., 2004). One positive finding was that of Hajcak and Foti (2008), who found that startle blinks following errors were greater than those following correct responses and that the ERN predicted the degree of blink potentiation. Also, Dywan and colleagues (2008) reported that respiratory sinus arrhythmia accounted for a relationship between a measure of the ERN and a measure of sadness.

AFFECTIVE AND MOTIVATIONAL VARIABLES

Other studies relating the ERN to affect have manipulated the motivational significance of the error response using monetary rewards and other incentives. Hajcak et al. (2005) manipulated the value of a correct response and found that errors that failed to earn a high-value (100-point) outcome elicited larger ERNs than those associated with a lower value (5 points). In a second experiment, Hajcak et al. (2005) showed that the size of the ERN was increased in the presence of a male experimenter who was seated next to the subject, evaluating the subject's performance. A similar finding was reported in children (Kim et al., 2005). Pailing and Segalowitz (2004b), however, reported that monetary incentive effects on the ERN were limited to subjects scoring high on a neuroticism scale. Ganushchak and Schiller (2008) compared blocks in which trials were associated with a monetary incentive to blocks without the incentive and reported that the incentive blocks yielded larger and later ERNs. Boksem and colleagues (2006a) reported a study of fatigue and the ERN in which a motivational incentive late in task performance caused an increase in response accuracy in some subjects that was associated with an increase in ERN amplitude. It is important to point out that, despite the differing incentives, the motivational effects in the Hajcak et al. and Ganushchak and Schiller studies were not associated with (and therefore were not attributable to) between-condition performance differences.

If the ERN reflects primarily an affective process, it would make sense that inducing short-duration affective states using affective pictures would modulate the size of the ERN. Larson and coworkers (2006) found that pleasant pictures superimposed on flanker stimuli increased the size of the ERN relative to neutral or unpleasant pictures. In contrast, Wiswede and colleagues (2009) found that unpleasant pictures presented 700 ms prior to flanker stimuli enhanced the ERN relative to the neutral or pleasant pictures. As Wiswede et al. note, the different demands on attention in the two tasks could account for the contradictory findings. Indeed, it is difficult to come up with a solid prediction about how such pictures will affect the ERN without a theory that specifies the processing evoked by the pictures in a particular task and how that process affects the computation reflected by the ERN. A gruesome autopsy photograph might make an error seem not so bad by comparison, or it could induce a bad mood that makes the error seem worse than it otherwise would. Either possibility could be compatible with a theory in which the ERN reflects an affective response to the error.

EVALUATION

The data reviewed here suggest that the ERN is sometimes related to variables that reflect or influence affective or motivational processing and is sometimes associated with changes in ANS activity. Nevertheless, such findings could be consistent with almost any theory of the ERN, because most computations that evaluate conflict or error processing

could give rise to an affective response, and because affective and motivational manipulations could influence attention (Yeung, 2004). Thus, although the notion that the ERN is an affective response has had substantial heuristic value and has yielded a number of important findings, it suffers from the same problem as the other ERN theories, namely, that there are multiple alternative theories that can predict the same results and that theorists have thus far failed to carry out truly competitive tests of well-specified alternatives. Computational models that specify how the affective processes differ from the other cognitive and attentional processes would help in this endeavor.

Neurotransmitters

The reinforcement-learning and affective-response perspectives on the ERN have motivated studies of the neurotransmitters involved in the generation of the ERN. Specifically, the RL-ERN theory argues that the ERN is produced by a disinhibition of pyramidal neurons in the ACC following a phasic decrease in the activity of dopaminergic neurons in the basal ganglia (Holroyd & Coles, 2002). This claim has motivated several studies of drug effects on the ERN (for an excellent review of this topic, see Jocham & Ullsperger, 2009). Similarly, researchers have examined the ERN in the context of psychiatric disorders and genetic differences tied to changes in dopamine function. Nevertheless, a fair amount of evidence points to influences from other neurotransmitters.

Dopamine

Several drug studies report results consistent with a link between the ERN and dopamine. Haloperidol, a dopamine antagonist, reduces the amplitude of the ERN (de Bruijn et al., 2006b; Zirnheld et al., 2004), as does the atypical antipsychotic olanzapine (which also blocks serotonin and histamine; de Bruijn et al., 2006b). Amphetamine, which blocks dopamine uptake and promotes its release, enhances the ERN (de Bruijn et al., 2004). Similarly, caffeine, an indirect dopamine agonist, also causes increased ERN amplitude (Tieges et al., 2004). Nevertheless, there are questions about the specificity of both caffeine and amphetamine to the dopaminergic system (see Jocham & Ullsperger, 2009).

Because Parkinson's disease and Huntington's disease patients have low dopamine levels in the basal ganglia, RL-ERN theory predicts an attenuated ERN in those disorders. Several studies have confirmed this prediction (Beste et al., 2008; Falkenstein et al., 2001a; Ito & Kitagawa, 2006; Stemmer et al., 2007). Another found no difference in ERN amplitude between patients and controls (Holroyd et al., 2002). Although the balance of that evidence seems consistent with the RL-ERN theory, the results are complicated by performance differences between patients and healthy controls: in most studies showing a difference, patients were either slower (Beste et al., 2008), less accurate (Stemmer et al., 2007), or both (Falkenstein et al., 2001a; Ito & Kitagawa, 2006). In two studies the Parkinson's disease was relatively mild, so patient and control performance was roughly equal. One study failed to find a difference between groups (Holroyd et al., 2002) and another study, with a larger sample size, showed a reduction in the ERN but not in the CRN, effects that were equivalent in patients on and off medication (Willemssen et al., 2008). As Jocham and Ullsperger (2009) noted, the latter finding could suggest that the ERN is not sensitive to the acute administration of a dopamine agonist, although it is also possible that the 12-hr withdrawal period was simply not long enough to show effects.

Schizophrenia, also thought to involve dopamine dysfunction, is associated with ERN amplitudes that are reduced (Alain et al., 2002; Bates et al., 2002, 2004; Ford, 1999; Kim et al., 2006; Kopp & Rist, 1999; Mathalon et al., 2002; Morris et al., 2006) but can recover somewhat when patients are treated with antipsychotics (Bates et al., 2004). Some of these studies also reported increases of the CRN (Alain et al., 2002; Mathalon et al., 2002) and no effect on the Pe (Alain et al., 2002; Bates et al., 2004; Mathalon et al., 2002; Morris et al., 2006). It does not appear that performance differences alone can account for the ERN reductions (Bates et al., 2004), although the extent to which chronic medication may influence the results is unclear (see, e.g., Morris et al., 2008).

Genetic polymorphisms that affect neurotransmitter systems provide an additional way to assess dopamine contributions to the ERN. For example, the val/met polymorphism of the catechol-*O*-methyltransferase (COMT) gene is associated with levels of dopamine in the frontal cortex. Frank and colleagues (2007) found that the ERN did not differ between met/met individuals (with higher prefrontal dopamine levels) and val/met or val/val individuals. Interestingly, the polymorphism did affect the late Pe. Krämer and coworkers (2007) examined the COMT gene as well as the dopamine D4 receptor gene (DRD4) and found that individuals homozygous for the T allele of the DRD4 gene (associated

with receptor responsiveness to dopamine) showed a larger ERN than individuals homozygous for the C allele. The COMT gene results were less clear, as there was a marginal effect such that the ERN was larger in val/val individuals than in met/met individuals, but only in a stop-signal task. Comparison of the Frank et al. and Krämer et al. studies shows that predictions in such studies are not always straightforward: according to Frank et al., the lower COMT in met/met individuals will cause higher levels of tonic dopamine and should thus result in a larger ERN than in val/val individuals. Krämer et al., however, predicted a smaller ERN in the met/met individuals, noting that lower COMT in those individuals should indeed be associated with greater levels of tonic dopamine but *lower* levels of *phasic* dopamine. (The Holroyd and Coles, 2002, model concerns phasic changes in dopamine.)

Other Neurotransmitters

Several studies have examined drug effects on other neurotransmitter systems. Alcohol reduces the size of the ERN (Easdon et al., 2005; Ridderinkhof et al., 2002), although the effect might be attributable to a degradation in stimulus processing (Yeung & Cohen, 2006; Yeung et al., 2007b) rather than to a direct role for enhancement of GABA receptors on generating the ERN. The benzodiazepines alprazolam (Riba et al., 2005b) and oxazepam (Johannes et al., 2001b) also reduce the ERN, perhaps consistent with a more direct role for GABA. Serotonin involvement is less clear: de Bruijn and colleagues (2006) found that the selective serotonin reuptake inhibitor (SSRI) paroxetine did not affect the ERN. Nevertheless, SSRI effects in the treatment of depression and OCD take several weeks, so that definitive conclusions regarding SSRI effects would seem to require longer-term administration of the drug. Gene studies seem to indicate some serotonin involvement: a study of individuals possessing one or two short alleles of the serotonin transporter gene 5-HTTLPR (thought to be associated with enhanced serotonin levels) showed those individuals to have a larger ERN than individuals with two long alleles (Fallgatter et al., 2004; Figure 10.19A). The finding was replicated in a group of children, although the ERN measure in that study could have been influenced by overlapping ERP components (Althaus et al., 2009). Beste et al. (2010a) reported that another polymorphism related to serotonin, the functional 5-HT1A C(-1019)G polymorphism, shows a relationship to ERN amplitude consistent with that reported by Fallgatter and colleagues, namely, a greater ERN being associated with increased serotonin: the CC genotype group showed a larger ERN than the CG and GG groups. Interestingly, the CRN did not differ between groups (see Figure 10.19B). Nevertheless, caution is warranted: a recent study failed to find a relationship between 5-HTTLPR and the ERN (Olvet et al., 2010).

Evaluation

The Holroyd and Coles (2002) theory has focused the spotlight on dopamine as an important contributor to the ERN, and some of the evidence is consistent with their model. Yet the evidence above suggests that there is room for neurotransmitter models that consider alternative roles for dopamine, such as the Jocham and Ullsperger (2009) model, as well as models incorporating other neurotransmitters. Jocham and Ullsperger (2009), for example, noted that acetylcholine has to date not been studied. Indeed, Sarter and coworkers (2006) pointed out that the functions of acetylcholine in effortful attention suggest that there is good reason to think that this neurotransmitter might be important for generating the ERN.

Development and Individual Differences

Initial studies of children suggested that clear ERNs that were distinguishable from CRNs were not evident until middle to late adolescence (Davies et al., 2004; Ladouceur et al., 2004, 2007). Nevertheless, Friedman et al. (2009) showed clear ERNs in a group of 10-year-old children. In addition, some children in that age range (10–11 years old) with anxiety disorders (Ladouceur et al., 2006) or greater socialization (Santesso et al., 2005) show a clearer ERN than age-matched controls. More recent evidence suggests that the ERN may be evident in children as young as 8 (McDermott & Fox, 2009) or even 5 to 7 years old (Torpey et al., 2009). A report of an ERN in 4-year olds (Brooker et al., 2011) is difficult to evaluate because it did not present an average waveform that isolated that age group. An ERN-like stimulus-locked potential has even been observed in 7-month-old infants observing impossible events (Berger et al., 2006). Although studies seem to indicate a trend for increasing ERN amplitude during adolescence (Davies et al., 2004; Ladouceur et al., 2004, 2007; Santesso & Segalowitz, 2008), there may be an interaction with task difficulty such that the developmental trend during adolescence is only seen for complex tasks (Hogan et al., 2005; but see Santesso & Segalowitz, 2008). The latter finding suggests that task differences might

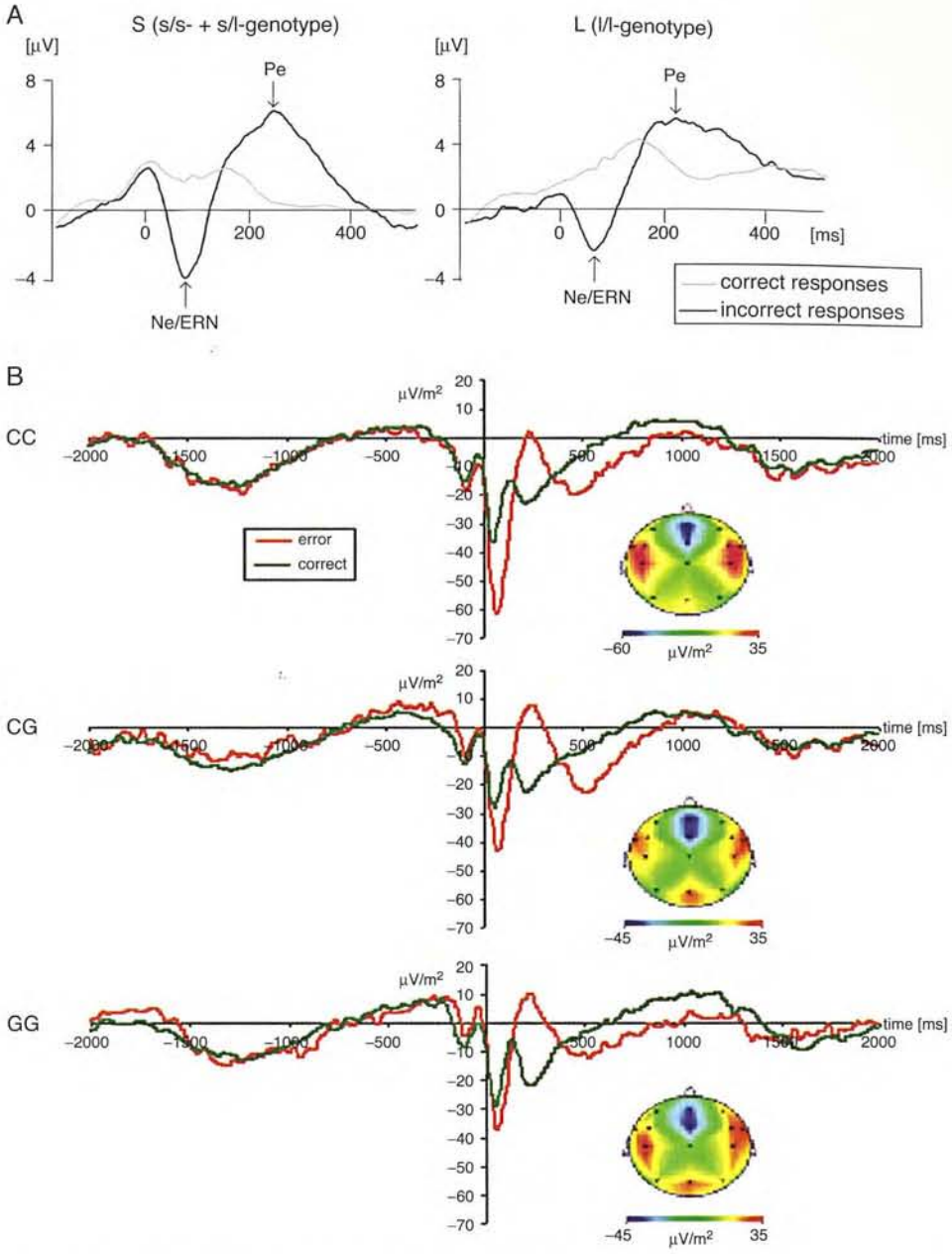

Fig. 10.19. Effects of serotonergic genotypes on the ERN. (A) Error-related negativity waveforms from subjects with short (left) or long (right) allelic variations of a serotonin transporter gene (5-HTTLPR). Individuals with one or two copies of the short allele showed a larger ERN than individuals homozygous for the long allele, suggesting that serotonin influences the generation of the ERN. From Fallgatter et al. (2004), Figure 1, reprinted with permission from Macmillan Publishers Ltd. (B) Response-locked ERPs at electrode Fz for correct (green) and error (red) trials. Separate plots show waveforms for different 5-HT1A genotype groups: top, CC; middle, CG; bottom, GG. This polymorphism influences serotonergic neurotransmission. From Beste et al. (2010), Figure 1, reprinted with permission of John Wiley & Sons, Inc.

explain some of the discrepant findings concerning younger children.

At the other end of the lifespan, numerous studies have reported a reduction in the amplitude of the ERN in older adults relative to younger controls (e.g., Band & Kok, 2000; Falkenstein et al., 2001b; Gehring & Knight, 2000; Nieuwenhuis et al., 2002), as well as an increase in its latency in some tasks (Falkenstein et al., 2001b). Two studies, however, in which older adults responded as accurately as

younger adults failed to find a significant age-related ERN reduction (Eppinger et al., 2008; Friedman et al., 2009). Additional ambiguities remain. Pietschmann and colleagues (2008), for example, reported a study in which ERN amplitudes did not differ in older and younger adults, but in older adults there was no error effect—the CRN was just as large as the ERN. A CRN enhancement was also observed by Eppinger et al. (2008). One key to disambiguating these findings may lie in the effects of cardiorespiratory fitness and physical activity on the ERN in aging adults and the fact that different subject samples might differ in this important variable. Interestingly, however, one study examining this issue found that both younger and older physically active adults showed smaller ERNs than their less active counterparts, which is paradoxical if physical activity is thought to mitigate the effects of aging (Themanson et al., 2006). Nevertheless, studies of aging face the same challenge as studies of Parkinson's disease: the slowing of older adults presents a confound that cannot be corrected simply by speeding the responses of the slower group because of the confound introduced by equating the two groups' performance. In addition, the results of Yeung et al. (2007b) discussed above suggest that future studies should consider whether altered stimulus processing in older adults could be a source of age-related ERN reductions.

We have already reviewed some individual-difference studies related to negative affect and anxiety. Another category of individual differences with obvious relevance to the ERN is that involving measures of impulse control: if the ERN is larger in conditions where accuracy is emphasized over speed, then one might predict that the ERN would be smaller in individuals who are impulsive or have difficulty controlling their behavior. Findings have been inconsistent. Pailing and coworkers (2002) derived a simple measure of impulsivity based on each subject's error trial RT. As predicted, subjects showing the most impulsive behavior showed the smallest ERNs (see also Ruchsow et al., 2005b). It is not necessarily the case, however, that fast responding reflects an impulsive personality; thus, more definitive links to trait-level impulsivity come from studies using questionnaires and other diagnostic means of assessing the impulsive personality. Stahl and Gibbons (2007) used a questionnaire measure of impulsivity to categorize participants in a stop-signal task. They reported less ERN activity in the more impulsive subjects, although their analyses focused on an ERN-like stimulus-locked component. Potts et al. (2006) reported a similar reduction in high-impulsive individuals, although the effect was restricted to a condition where errors were penalized (as opposed to a condition where correct responses were rewarded, where no such difference was observed). At least two studies, however, have failed to find a relationship between the ERN and questionnaire measures of impulsivity (Santesso & Segalowitz, 2009; Vocat et al., 2008).

Studies of clinical impulse-control disorders are also inconsistent. There are reports of children with attention deficity hyperactivity disorder (ADHD) showing reduced (Groen et al., 2008; Liotti et al., 2005), equivalent (Jonkman et al., 2007; Wiersema et al., 2005), or enhanced (Burgio-Murphy et al., 2007) ERNs relative to controls. Wiersema and colleagues (2009) found no significant difference between adults with ADHD and controls, although their waveforms show a larger ERN in the controls. Across studies there are task differences, between-group performance differences, and ERN baseline/measurement issues that could contribute to the inconsistent findings (see Shiels & Hawk, 2010, for a review).

Among the personality disorders characterized by difficulty with impulse control is psychopathy. In the first study of this disorder, Dikman and Allen (2000) found a reduction in the ERN in conditions where errors were penalized for individuals scoring low on a socialization questionnaire thought to reflect proneness to psychopathy. Three studies of violent offenders with psychopathy offer mixed results: two reported a significant ERN reduction in violent offenders with psychopathy (Borries et al., 2010; Munro et al., 2007), although in one of these studies the effect was seen in a task involving face stimuli but not in a flanker task (Munro et al., 2007). Another study using a flanker task failed to find a significant reduction, although the reported ERN amplitude was 2 μV smaller in the offenders (Brazil et al., 2009). Psychopathy in these studies was assessed using the unidimensional Psychopathy Checklist-Revised (Hare, 1991), whereas a large body of evidence suggests that the disorder actually comprises at least two factors, one related to externalizing and the other involving trait fearlessness (Patrick & Bernat, 2009). Externalizing is a personality construct thought by some to be common to a number of different impulse-control disorders, including substance abuse, antisocial behavior, and, in children, conduct disorder. Hall et al. (2007) found that individuals with high scores on an externalizing scale showed a reduced ERN relative to

low-externalizing individuals. As in the studies of ADHD, the numerous differences in subject characteristics, tasks, and ERN measurement methods make it difficult to draw definitive conclusions. If anything, the pattern seems consistent with at least some reduction associated with the externalizing component of psychopathy.

Other individual difference studies are generally consistent with a link between a reduced ERN and impulsive or risky behavior. De Bruijn and coworkers (2006) found a reduced ERN in borderline personality disorder, which is also characterized by impulse-control problems. Pailing and Segalowitz (2004b) found that individuals low in conscientiousness showed a reduced ERN response to monetary incentives. Santesso and Segalowitz (2009) found that individuals scoring high on a measure of risk propensity (comprising risk taking and sensation seeking) showed smaller ERNs. Frank and colleagues (2005, 2007) derived a measure of reward-based learning biases from a reinforcement-learning task, finding that subjects who learned to seek positive outcomes showed a smaller ERN than subjects who learned to avoid negative outcomes.

Evaluation

One criticism of these studies (and the clinical studies reviewed earlier) is that most of them focus on a single personality or clinical construct, without regard for other correlated personality variables or psychopathology. Studies rarely attempt empirically to rule out alternative hypotheses. Yet, it is clear that drawing inferences based on a single personality or psychopathology questionnaire is perilous. In clinical research, questionnaires can be checked against other clinical diagnostic assessments, and here the facts are sobering: the Obsessive-Compulsive Inventory-Revised used to measure obsessive-compulsive tendencies has a sensitivity of 65.6% and a specificity of 63.4% when a cutoff score of 21 is used to decide whether an individual has OCD (Foa et al., 2002). Still, according to Bayes' theorem, if 2% of the population has OCD, an individual scoring over 21 on the OCI-R has less than a 4% chance of actually having OCD.

It is therefore much more informative when studies, such as those of Santesso and Segalowitz (2009), Vocat et al. (2008), and Inzlicht et al. (2009), examine multiple personality constructs in the same individuals. Especially promising are studies that include measures both of personality traits and of psychiatric symptoms (e.g., Chang et al., 2010). In the future, it would seem more useful to have more large-scale studies that simultaneously assess numerous personality and psychopathology constructs rather than continuing to carry out small-scale, single-construct studies. But even research incorporating numerous control assessments leaves open the possibility that some construct that was not assessed is the one really driving the result. Cardiorespiratory fitness, for example, can be associated with changes in ERN amplitude (Themanson & Hillman, 2006; Themanson et al., 2006); in many studies, subject groups could have differed in their fitness levels. Task engagement is an even more troublesome variable. Tops and coworkers (2006) offer a simple explanation for a somewhat paradoxical finding—that higher agreeableness, a positive trait, and higher behavioral shame proneness, a negative one, are both associated with larger ERNs. According to Tops et al., subjects scoring high on either measure tend to be more engaged in the task than their low-scoring counterparts (recall also the finding of Luu et al., 2000a). Thus, individual differences might in many cases be explained by differences in task engagement: two groups of subjects whose brains have exactly the same ability to generate the ERN can show ERN amplitude differences simply because one group of subjects is less involved in the task. Similar undesirable confounds can arise if subjects differ in their representation of or sensitivity to the social evaluation inherent in an experiment (Cavanagh & Allen, 2008; Hajcak et al., 2005b).

A final important challenge for individual difference research is that it must seek a better understanding of the manner in which the outcome of a study can depend on the particular task used to elicit the ERN and the particular techniques used to measure it (Hogan et al., 2005; Munro et al., 2007). Studies of personality constructs and psychiatric disorders would have limited generalizability if simply changing the subjects' task altered the findings.

New Directions

The preceding sections have sketched some of the major findings and controversies in ERN research. In this section, we outline some of the issues that are emerging as important and will, in our opinion, shape the coming years of ERN research.

Beyond Choice Reaction Time

With the exception of the FRN studies, ERN research has focused almost exclusively on speeded

reaction time (RT) tasks, such as choice RT and go/no-go RT tasks, and most of these have measured only button-press closure, neglecting richer measures such as response force, movement velocity, and EMG. A few recent studies have turned to more realistic aimed-movement and force-production tasks, beginning a likely trend in which the human movement control literature can influence ERN research. Such a trend may help to resolve the extent to which the ERN is involved in immediate error correction versus strategic control.

The complex motor tasks employed thus far include force production (de Bruijn et al., 2003), aimed movement (Anguera et al., 2009; Krigolson & Holroyd, 2007a), pointing (Vocat et al., 2011), and manual tracking tasks (Krigolson & Holroyd, 2006, 2007b). Negative-going potentials have been reported in such tasks when errors occur in the choice of force (de Bruijn et al., 2003) or when external perturbations cause an error in tracking or aimed movements (see Figure 10.20; Anguera et al., 2009; Krigolson & Holroyd, 2006, 2007a, 2007b). Some of these studies have concluded that the ERN is related to high-level errors, where the subject selects an action that violates a task goal, rather than low-level errors, where the subject chooses the correct action but the movement deviates from the planned trajectory (Krigolson & Holroyd, 2006, 2007a, 2007b). Another study has come to the opposite conclusion, however, based on evidence that ERN-like activity is sensitive to the extent of the low-level error (Anguera et al., 2009; see also Vocat et al., 2011). Nevertheless, there is little direct evidence that the slower potentials presented as evidence for these conclusions are indeed the ERN. Alternative possibilities are that the slow potentials represent contingent negative variations (CNVs; see Chapter 8, this volume) preceding anticipated target perturbations and movement-monitoring potentials (MMPs) associated with the ongoing movement (de Bruijn et al., 2003). Additional work is needed to determine more conclusively what components are elicited in these complex motor control tasks. An additional innovation involves measuring ERN-like potentials at the time of the response when an alternative in a gambling task is chosen, where the ERN-like potential appears to be larger for riskier choices (Yu & Zhou, 2009), an effect that is more pronounced in low-impulsive subjects (Martin & Potts, 2009). In these and any other extensions of the traditional paradigm, bridging studies are needed that elicit both the classic ERN in a simple motor task and the negativity in the alternative task, comparing the two components in the same subjects and, ideally, finding intermediate conditions in which the classic ERN transitions to the more atypical response.

The Bridge to the Brain

Another type of bridge that will play a critical role in unraveling the mystery of the ERN is the one that can be established between studies of scalp ERPs in humans and those of single-cell and LFP recordings in nonhuman primates. Thus far, neurophysiological studies of the ACC and other structures have yielded findings that are consistent with each of the major theories of the ERN reviewed here (e.g., Emeric et al., 2008; Ito et al., 2003), but there are also discrepancies (the most glaring being the lack of ACC conflict-related activity; Emeric et al., 2008; Ito et al., 2003). It is still not clear, however, how the various intracranial potentials propagate and summate to produce the scalp-recorded ERN. Links between the monkey studies and those in humans will be strengthened by further studies of saccade countermanding in humans that compare directly to those in monkeys, and by further studies of manual response tasks in monkeys that are more similar to those in humans. Moreover, the scalp-recorded ERN probably reflects the activity of more than one intracranial generator, and even individual LFP recordings can reflect several superposed sources; thus, recordings of current source density across a broad swath of the medial frontal cortex will be especially informative (Emeric et al., 2008).

ERN in the Social World

OBSERVED ERRORS

The idea that the ERN represents performance monitoring leads naturally to the question, does someone show an ERN when he or she sees someone else make an error? Several recent studies have reported evidence that direct experience and vicarious observation of negative events rely on common neural substrates. Both error commission and error observation elicit negative-going ERP components over medial-frontal scalp sites (Bates et al., 2005; Miltner et al., 2004; van Schie et al., 2004). Both components can be explained by a source in ACC (Miltner et al., 2004; van Schie et al., 2004), and both are accompanied by an increase in evoked theta power relative to correct responses (Bates et al., 2005). The so-called observation ERN is elicited by error observation in a choice response task (Miltner et al., 2004), a flanker task (van Schie et al., 2004), and a go/no-go task (Bates et al., 2005), as well as during observation of a human performer (Bates et al., 2005; Miltner et al., 2004)

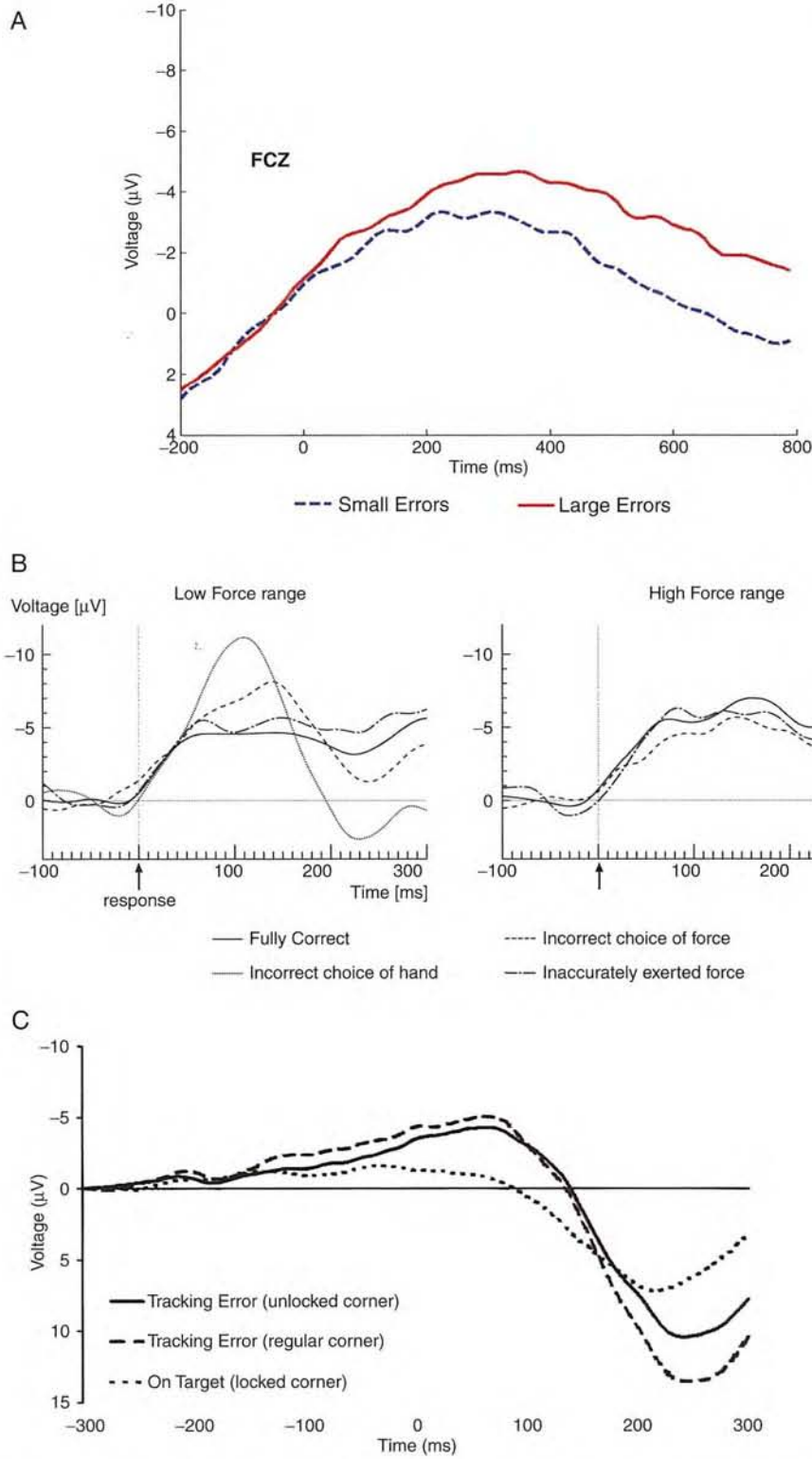

Fig. 10.20. Error-related negativity–like potentials elicited in motor control tasks, showing prolonged error-related activity that bears some similarity to the ERN. (A) Error-related negativity–like activity in a visuomotor adaptation task where participants adapted manual aiming movements to a 30° rotation of the visual feedback display. Movement errors were classified as high (red dashed) and low (blue solid) errors according to the distance of the initial endpoint of the movement from the target. From Anguera et al. (2009).

(*Continued*)

or "virtual feedback" regarding another person's performance (Miltner et al., 2004). An important consideration in these studies is that observed errors tend to be infrequent events, raising the possibility that the observer's ERN is an N200. Supporting this contention, de Bruijn et al. (2007) found that no ERN was elicited in an observer when the observed errors were as likely as correct responses (but see Bates et al., 2005).

Analogous results have been reported for the FRN. Both the direct experience and the observation of losses in a gambling task elicit an FRN-like wave (Fukushima & Hiraki, 2006; Yu & Zhou, 2006). However, the consequences of the observed individual's outcomes for the observer appear to be important: Itagaki and Katayama (2008) found that the FRN in an observer is larger when the observed individual loses than when that person gains—if the two individuals are cooperating. When the two individuals are competing, however, the gain of the observed individual will cause the larger ERN. Effects of cooperation versus competition on the observation FRN may be modulated by the gender and personality of the observer (Fukushima & Hiraki, 2006).

SOCIAL NEUROSCIENCE

The extension of cognitive neuroscience and psychophysiological methods to social, affective, and cultural neuroscience (e.g., Cacioppo et al., 2004) has led to ERN research being extended to the social world (e.g., Amodio et al., 2004, 2006; Inzlicht & Gutsell, 2007). For example, one issue of interest in contemporary social neuroscience concerns implicit attitudes—specifically, implicit race biases—and how individuals might use cognitive control when they are faced with social situations that activate the biases (e.g., Gehring et al., 2003; Richeson et al., 2003). Amodio and colleagues (2004, 2006) found an enhanced ERN in a task where individuals had to determine whether a masked stimulus was a gun or a tool. Preceding the stimulus was a prime stimulus consisting of a black or white face. Basing the study on the conflict model (Botvinick et al., 2001; Yeung et al., 2004b), Amodio and colleagues inferred that a tool stimulus following a black face would be analogous to an incongruent Stroop or flanker trial, involving response conflict between the racially biased, prepotent "gun" response associated with a black face and the correct "tool" response. Errors on such trials were indeed associated with a larger ERN than other trial types. It is not clear, however, that the prediction was derived correctly from the conflict model. The error on a "tool" trial is the "gun" response, which should not conflict with the prepotent "gun" response suggested by the black face.

Social and cultural neurosciences are exciting areas in which the ERN might prove a useful measure. Of course, such studies must include control conditions to show that the effects of a social-level construct on the ERN are not simply explained by the effects of some lower-level variable that is confounded with the construct of interest (such as the possibility that the ERN could be affected by the relative luminance of guns and tools or of black faces and white faces rather than their significance with regard to race biases (Yeung et al., 2007b). As with the individual difference studies discussed above, studies that compare groups of people must take care to eliminate alternative hypotheses by measuring all individual difference variables that might account for observed group differences, rather than presenting groups defined by their responses on a single questionnaire or by their membership in a single social category.

Genetics and the ERN

One of the more exciting areas of recent progress is in studies that relate the ERN to genetic polymorphisms involved in neurotransmitter and neurotrophin function. The studies of Beste et al. (2010a), Fallgatter et al. (2004), Frank et al. (2007), and Kramer et al. (2007) reviewed above show the potential for this technique to shed light on the neural basis of the ERN. It is clear that many more genes are worth investigating. One that seems promising is the glutamate transporter gene SLC1A1, which has been implicated in OCD (Arnold et al., 2006; Dickel et al., 2006; Stewart et al., 2007) and

Fig. 10.20. (Continued)
Reprinted with permission from the American Physiological Society. (B) Grand average response-locked waveforms elicited by a force production task. Errors were classified by errors of hand, errors of force selection, and force exertion. From de Bruijn et al. (2003), Figure 3, reprinted with permission of John Wiley & Sons, Inc. (C) Grand average response-locked waveforms from a motor tracking task on trials in which no tracking error occurred, in which a tracking error occurred for a relatively easy condition ("regular corner"), and in which tracking errors occurred during a relatively difficult condition ("unlocked corner"). Subjects had to maintain a cursor between two moving barriers; zero indicates the time of barrier contact on an error trial and a matched time on correct trials. Modified from Krigolson and Holroyd (2006a), Figure 1, reprinted with permission from Elsevier.

would thus be of interest in ERN studies. Evidence from a twin study for heritability of ERN amplitude provides additional impetus to characterize the genetics underlying the ERN (Anokhin et al., 2008), although heritability of an ERP component could reflect not only neurotransmitter function, but also head shape and brain morphology.

Of course, new methods bring with them new challenges. Sample size is a methodological challenge: early findings of gene–phenotype relationships based on small samples often overestimate true effect sizes compared with later studies based on large samples (Green et al., 2008). Some of the discrepancies noted earlier among genetic association studies and pharmacological studies might result from sample sizes that are too small. Conceptual challenges also abound. Beste et al. (2010b), for example, found a rather striking difference between one group that carried the Met allele of the brain-derived neurotrophic factor (BDNF) Val66Met polymorphism and another that did not. Still, because BDNF controls neurogenesis and cortical morphology (see Beste et al., 2010b), it is not clear to what extent ERP differences could be caused by differences in structural factors such as cortical morphology or synaptic density. Nor is it clear what it would mean functionally if those structural differences did play a role.

Key Issues in Designing and Interpreting ERN Experiments

Despite all the exciting advances we have just reviewed, the ERN waters have been muddied by a number of problems with the quality of the experimentation and data. Here we review these problems and show some examples. Note that we do so not to criticize any individual investigator; therefore, we removed identifying information from the examples we have provided to the extent possible (and we've also included ourselves in the criticism).

Experimental Design and Signal-to-Noise Ratio

Research on the ERN requires a lot of data. In order to achieve an adequate signal-to-noise ratio in the error-trial average, the task design must provide for a relatively large number of trials. Several realities make this difficult. Once a task is learned, errors tend to occur infrequently. An additional complication is that, as reviewed earlier, the bulk of the evidence suggests that a low error rate is necessary to elicit a large ERN, further increasing the amount of data that must be collected. Finally, most within-subject experimental designs require the comparison of at least two experimental conditions, thus doubling the number of trials necessary for the analysis of interest. Unfortunately, investigators have too often allowed the practicalities of experimentation to override the need for clean waveforms, settling for a small number of error trials. Consequently, there are numerous papers where the conclusions are questionable because of the amount of noise in the waveforms.

There is a very simple heuristic that readers can use to evaluate the quality of data contributing to an ERP waveform. A figure comparing two waveforms should theoretically show no difference between the waveforms during the baseline interval (just prior to the stimulus in a stimulus-locked average, or in the corresponding period in a response-locked average, several hundred milliseconds prior to the response). The amount of noise in that interval thus gives a sense of the amount of noise in the portion of the waveform being analyzed. In short, if visual inspection of the ERPs suggests that altering the baseline used for computing a base-to-peak or amplitude measure would alter the outcome of the study, one should be skeptical of the conclusions raised in the study. Figure 10.21 shows several examples where a noisy baseline epoch leads to just this sort of skepticism. Note that in many cases, authors fail to show enough baseline data to draw firm conclusions one way or the other.

Component Overlap

Some of the difficulties with ERN research stem from issues of component overlap. The ERN is a negative-going peak that overlaps at least one major positivity (the P3 or Pe) and most likely other negativities, and investigators must take care to ensure that differences in the ERN waveform are not a result of these overlapping components. Although component overlap is a concern with any ERP component, the situation with the ERN is especially difficult, because the process of response locking does not eliminate stimulus-related potentials occurring at around the same time as the ERN. Despite the ubiquity of this issue in ERP research and the sophisticated analysis techniques that can be employed to deal with it (e.g., Bernat et al., 2005; Woldorff, 1993; Zhang, 1998), numerous examples exist in the literature where overlapping components, rather than the ERN itself, could be responsible for the reported experimental effects (see Figure 10.21). As with noisy data, the baseline interval and other neutral epochs in the waveform

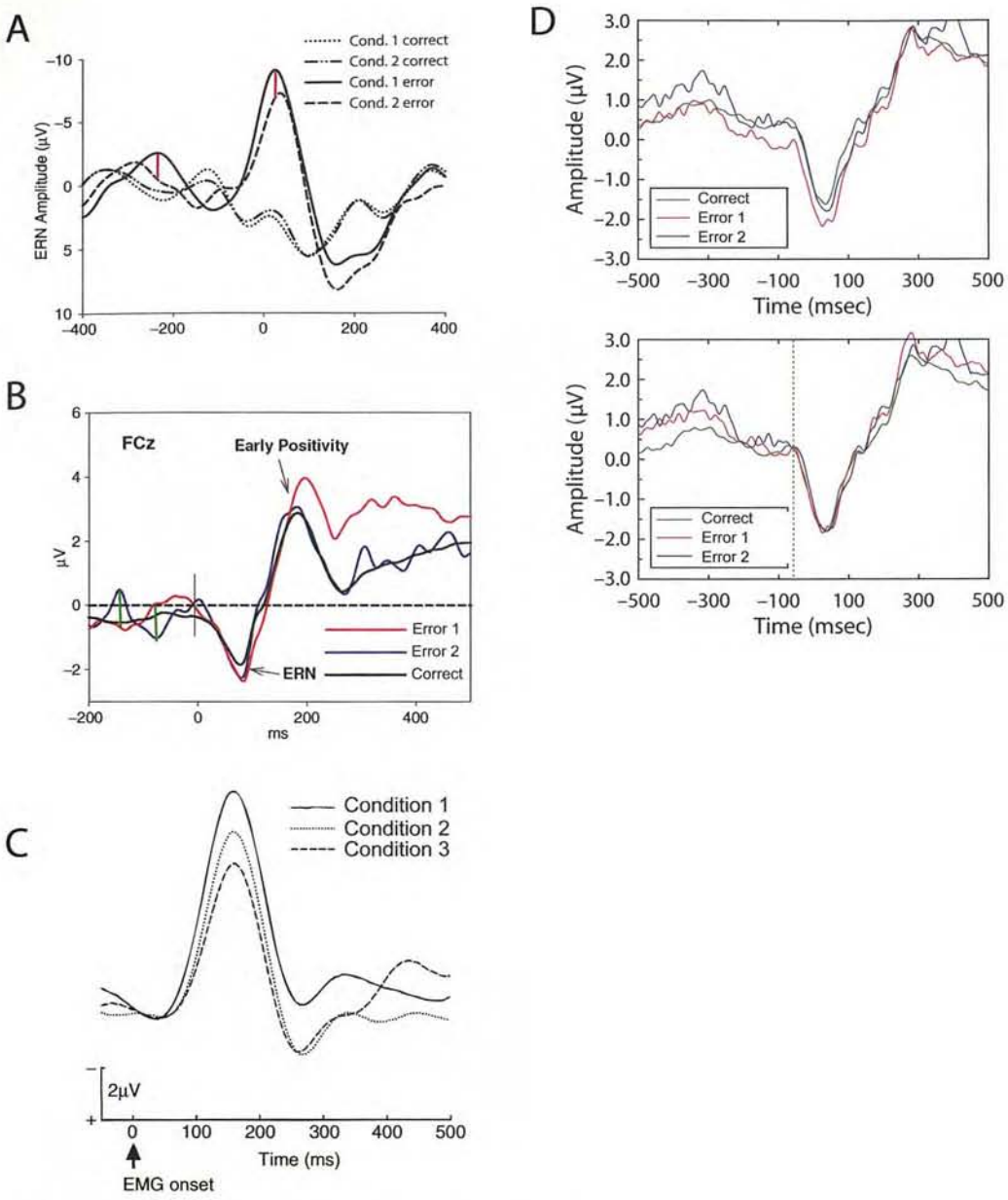

Fig. 10.21. Waveforms illustrating common problems in the ERN literature.

(A) The authors report a difference between the two error waveforms. The amplitude was computed as a negative peak from 50 to 150 ms following the response relative to a –400 to –50 ms preresponse baseline. The vertical red bars show how aligning the waveforms at a different point prior to the response would eliminate the reported effect.

(B) The authors report that there is no difference between the ERNs in the red and blue conditions. Nevertheless, the large, noisy fluctuations prior to the response show that taking the baseline at other plausible points (vertical green bars) would produce other effects, making the red larger than the blue in one case and the blue larger than the red in the other.

(C) The authors report ERN amplitude differences among the three waveforms. The differences in the baseline period and the presence of a difference that continues beyond the offset of the ERN suggest that overlapping components might contribute to the effect. Evaluating these data would be easier if the authors had plotted a longer preresponse interval.

(D) The authors reported that the three conditions differed, with Error-2 > Error-1 > Correct. The waveforms in the upper panel are shown as reported, using a preresponse baseline of –800 to –700 ms, which was not shown in the figure. The ERN was computed as the difference between the first negative peak following the response and the immediately preceding positive peak. Although peak-to-peak measures can help to remove overlapping low-frequency activity, the authors failed to report the time window in which the positive peak was detected. Alignment of the waveforms at the positive preresponse peak shows the amplitude of the ERN relative

(*Continued*)

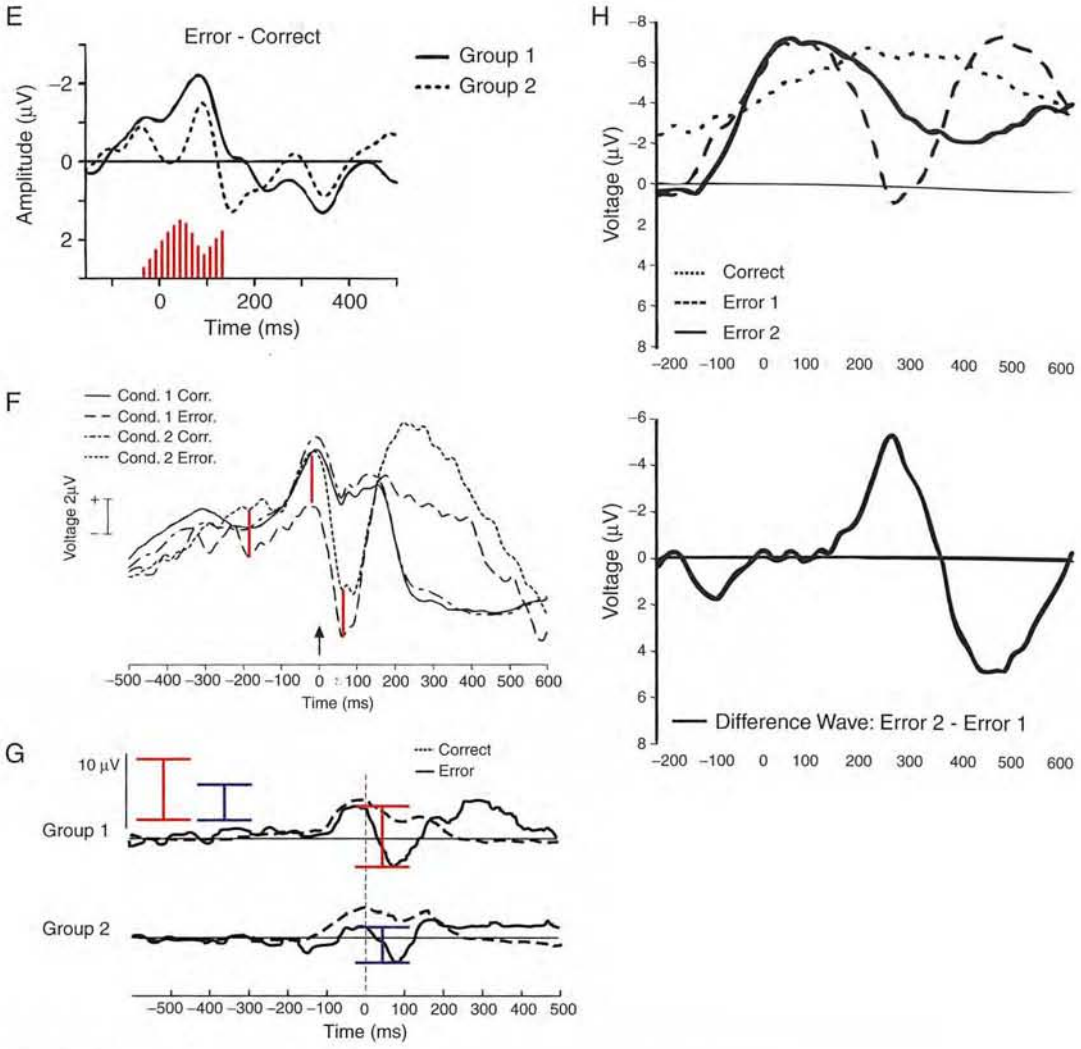

Fig. 10.21. (Continued)
to that peak to be approximately equal in the three conditions, suggesting that the authors' peak-to-peak measure was inappropriately influenced by the much earlier positivity. In addition, the amount of noise is a concern, as there are deflections in the Error-2 waveform prior to the response that exceed the putative postresponse amplitude difference in the ERN.
(E) The authors presented error-trial–correct-trial difference waveforms for two groups of subjects. Waveforms were baseline corrected to the average amplitude between 400 and 50 ms before the response. The entire baseline period is not shown. The height of the red vertical lines shows the difference between the two waveforms at each corresponding point in time. Note that the time course of the difference is certainly consistent with an ERN, but the onset and peak of the difference do not correspond to the onsets or peaks that visual inspection would assign to the ERN. Fortunately, individual averages for correct and error trials were also presented, but those waveforms present similar difficulties, including latency shifts in the underlying CRN and ERN peaks, making it difficult to interpret the effect shown here.
(F) The authors reported that Condition 1 Error shows a larger ERN than Condition 2 Error. The ERN was computed using a mean amplitude measure relative to a baseline interval (which was not shown) from 1000 to 750 ms prior to the response. The vertical red bars (of equal height) show that the difference between ERN peaks following the response is also present prior the response and that alternative baselines or peak-to-peak measures would eliminate the effect.
(G) The authors reported no difference between Group 1 and Group 2 in peak ERN amplitude. However, it appears that if they were to measure the ERN as the peak-to-peak difference (i.e., the difference in amplitude for the first positive peak preceding the response and the first negative peak following the response), there would be a difference between the two groups. The red bar denotes the peak-to-peak difference for Group 1, and the blue bar denotes the peak-to-peak difference for Group 2.
(H) The authors present the negative-going peak in the difference waveform (bottom panel) as evidence that there is an ERN in the Error-2 condition. The difference waveform was created by subtracting the Error-1 (dashed) waveform from the Error-2 (solid) waveform (shown in the top panel). Note that the large positive peak in the Error-1 condition appears to be responsible for the effect

(*Continued*)

(e.g., just prior to the response) can give the reader a sense of the likely contribution of component overlap. Judicious use of difference waveforms can help to increase one's confidence that observed effects show the time course and scalp distribution that an ERN effect should show (although difference waves should never be the sole basis of an analysis).

Baseline and Measurement Issues

Excessive noise and overlapping components both complicate the choice of how to quantify the amplitude or latency of the ERN—a choice that in itself can determine the outcome of an experiment. For example, most ERN researchers have had the experience of seeing a published waveform plot and wondering whether the conclusions of the study would have been eliminated or reversed simply by an alternative choice of baseline. Often the baseline interval is not even shown in the plot. Some investigators use a peak-to-peak measure of the ERN, and others filter the waveforms using a high-pass filter designed to eliminate low-frequency activity. Such precautions can help to reduce the contribution of low-frequency activity, but they are not infallible. Digital filters can introduce distortions (Yeung et al., 2004a, 2007a). In particular, while high-pass filters have been used to isolate the ERN from the slower overlapping Pe, they create an effect that is opposite in polarity and earlier in time from the activity in the unfiltered waveform. This artifactual activity can appear during the baseline epoch. Base-to-peak and peak-to-peak measures will thus still be influenced by the overlapping potential, and artificial oscillations will also appear (see Luck, 2005, chap. 5). Even without the use of high-pass filters, peak-to-peak measures are not as foolproof as they might seem for isolating the ERN from overlapping potentials. Peak-to-peak measures are sensitive to whatever is happening prior to the ERN peak, including slow potentials that could cause the peak-picking algorithm to choose the first point of the peak-picking window rather than a true positive peak. Peak measures are also especially susceptible to noise and the effects of component overlap (see Luck, 2005), both of which are concerns in ERN studies. Finally, while time-frequency analysis based on Morlet wavelets or time-frequency distributions might seem promising as a remedy for the problems of digital filters, the techniques are still new and their limitations are not yet well understood.

Difference Waveforms, Scatterplots, and Bar Graphs

When publishing a paper, it is tempting (especially when publishing for a non-ERP audience) to publish easily comprehended bar graphs, scatterplots, and difference waveforms (consisting of the ERP of one condition subtracted from that of another). Such figures simplify presentation of the data, but to a seasoned ERP investigator they are virtually useless unless supporting waveform data are presented with them. A key problem is that such figures do not permit the reader to determine the likelihood that a particular ERN effect could result from overlapping components, baseline problems, or noisy data. The problem is not solved in the numerous papers that show the ERN in an overall error-versus-correct-waveform comparison but then go on to bury the paper's most important finding in a scatterplot or bar graph. Supporting waveform data should always be included: average waveforms should accompany bar graphs or scatterplots, and the two single-condition waveforms used to create a difference waveform should be presented. Indeed, the publication guidelines of the Society for Psychophysiological Research mandate that these waveforms be presented (Picton et al., 2000). Inferences supported by a scatterplot can be strengthened by presenting average waveforms derived from a median split on the variable of interest (see, e.g., Inzlicht et al., 2009). In the case of the ERN, defining the component as the difference between error

Fig. 10.21. (Continued)
seen in the difference waveform. To accept the authors' alternative conclusion—that the negativity in the difference waveform is due to a negativity in the Error-2 condition—one must accept that the Error-2 negativity happens to coincide precisely in latency, frequency, and amplitude with a positivity identical to that seen in the Error-1 condition such that the two components sum together to produce the smooth Error-2 condition waveform. Although such a scenario is not impossible, it seems prudent to at least consider an alternative explanation, namely, that the peak in the difference waveform occurs solely because of the positive peak in the Error-1 condition.

Individual panels reprinted by permission. Copyright by Elsevier, John Wiley & Sons Inc., the Psychonomic Society, and Springer. Note that these waveforms are presented only as examples of common problems and are not meant as a criticism of the work of particular investigators; most other ERN investigators have published waveforms that could have contributed to this figure. Also, the most problematic figures are not included here, because those papers did not present relevant waveforms.

and correct trials carries the assumption that ERN activity does not occur on correct trials, an assumption that is untenable given the many studies showing a CRN.

Some Examples

Figure 10.21 illustrates some examples where the waveforms appear to illustrate one or more of the problems outlined above. Again, we want to stress that we are not attempting to criticize any individual investigator whose work appears in the figure; we have seen examples of these problems in research published by many different laboratories. It is worth noting that the journals in which these problems appear include high-impact general-readership journals in psychology and neuroscience and well-respected psychophysiology specialist journals.

Some Suggestions

With so many things that can go wrong in an ERN study (eliciting multiple ERNs from the experimenter), one might wonder what positive steps can be taken to ensure high-quality ERN data and analyses that will stand up to the scrutiny of the pickiest of reviewers. Of course, all the usual guidelines for ERP recording apply to the ERN (see Handy, 2004; Luck, 2005; Picton et al., 2000). And as with all ERP components, the most important factor determining the quality of the ERN data is simply how clean the dataset is, which is in large part a function of the number of trials contributing to the average. We hesitate to give a specific number of trials that must be in an ERN average, because of the many other factors that contribute to the quality of the data, but we get nervous when an experimental design yields fewer than 40 or 50 error trials. In general, it is not unreasonable to expect a study to require several sessions comprising thousands of trials (e.g., Gehring et al., 1993), especially if the design is complicated (see the discussion below). An effective eye-movement artifact correction procedure (e.g., Gratton et al., 1983) is a helpful way to maximize the trial count by retaining trials for analysis that would otherwise be lost because of blinks and other eye-movement artifacts. In eliciting the desired performance from subjects, we usually try to have subjects respond with an error rate in the range of 5%–10%. Achieving a specific level of accuracy can be a challenge. In our experience, the most effective inducement is frequent feedback (after a block of 40–50 trials) telling the subject to go faster if the error rate was lower than 5% in that block and to be more careful if the error rate was greater than 10%, perhaps coupled with a financial inducement to keep the mean RTs below some threshold value (note that it might be necessary to shift the RT threshold as subjects get faster with practice). The system is not perfect, however, because the instructions to subjects will be different in a between-group study where the groups vary in RT or accuracy.

Having run the study, the investigator is faced with two or more condition waveforms that must be examined for an ERN effect. With so many components occurring in the epoch surrounding a response, it is sometimes tempting to see ERN effects where there are really only amplitude or latency shifts in overlapping components. This temptation is particularly great when there is a desire to publish an ERN result from a study funded by an ERN grant. Nevertheless, reality doesn't always comply with our predictions, even funded ones. To verify that an effect is truly an ERN effect, we advise checking the scalp distribution and the time course of the difference waveform (keeping in mind the caveats regarding the CRN discussed above). The peak of the difference between conditions should occur at about the same time as the peak of the ERN itself. The rise and fall of the difference between conditions should take place at about the same time as those of the ERN, such that the difference shows an ERN-like time course. Although digital filters that eliminate low-frequency activity can be helpful in verifying the time course of the effect, one must remember that such filters can introduce artifacts. The scalp distribution of the peak of the difference waveform should show the same maximum (usually at FCz) as the individual condition ERN waveform. Time-frequency analysis is a promising technique to verify which component caused a particular ERP effect and to remove overlapping components, but time-frequency techniques are not foolproof: the Morlet wavelet, for example, assumes an oscillatory waveform. If the ERN peak is followed by an early Pe peak at the same frequency, the theta power measured with a Morlet wavelet during the ERN time window could reflect not only the ERN, but also the Pe that follows it.

Advice for the Young Investigator

Now we turn to the ERN of the future. What will 20 more years of ERN research give us? Despite the difficulties outlined in the previous section, we hope that junior investigators reading this chapter have seen that the many open questions leave many exciting discoveries and theoretical advances for those

who continue to pursue ERN research. Our look toward the ERN of 20 years from now takes the form of advice to those young investigators.

Model the Competition, Not Just the Conflict

The use of computational modeling in ERN research has been a spectacular advance. We have reviewed cases where a model showed new alternative explanations for empirical phenomena. For example, the conflict model showed that the ERN need not imply the existence of an explicit error detector (Botvinick et al., 2001; Yeung et al., 2004b) and that dissociations between components such as the N2 and ERN need not imply that the components are generated by distinct mechanisms (Yeung & Cohen, 2006). But we have reviewed numerous cases where the fact that a model predicts or does not predict the data leaves open the question of how well other models and theories might fare in predicting the same data. Computational modelers must grapple with the fact that any result predicted by their favored computational model could be predicted by other computational models built within the same architecture, or by other models generated within other architectures, and that support for a model can be achieved only by actually building and testing the alternatives. Modelers should make a good-faith effort to account for and predict experimental findings using different architectures or models, and they should engage in competitive tests of those models, where the alternatives are tested on equal footing. It is unsatisfactory simply to confirm the prediction of a single model without also establishing (1) the ability of other models to predict the same result, (2) the range of other results the model could have predicted given other parameters, and (3) the range of phenomena the model cannot predict (see Roberts & Pashler, 2000). For example, conflict monitoring as modeled in a connectionist framework may not be able to account for the ERN data of Burle et al. (2008), but perhaps conflict monitoring modeled within a symbolic architecture like EPIC (e.g., Seymour & Schumacher, 2009) could do better. And perhaps the key difference isn't whether the architecture is connectionist or symbolic, but some other characteristic of the way the models compute conflict. Only a valiant, systematic effort to compare models and architectures will yield such information.

Navigate the Terrain Hypothesis

We described how the effort to link the ERN with measures of error correction, such as post-error slowing, has resulted in a large number of inconsistent findings. This effort is based in a tradition in ERP research in which investigators look for consequences of the component (Donchin, 1981). That is, to discover the function of a component, it is useful to see whether variation in the size of the component predicts some subsequent behavioral outcome. This logic has been used successfully in studies of memory encoding; ERPs elicited at the time of encoding predict whether the item is later recalled (Fabiani et al., 1986; Paller et al., 1987; see also Chapter 14, this volume).

The situation with the ERN is not so simple. McCarthy and Donchin (1978) proposed the *terrain hypothesis* to describe the considerations one must take into account in predicting the relationship between an ERP component (which in their case was the CNV) and behavior: "As the correlation between the speed of an automobile and the depression of the accelerator depends on the terrain being traversed, so the correlation between measures of the CNV and the organism's performance may depend upon the psychological and physiological terrain over which the organism is traveling" (p. 582). Earlier we discussed some of the task- and context-related factors that make it difficult to predict how ERN amplitude will relate to measures of within-trial error correction. Similar terrain-based considerations apply to the effort to relate the ERN to measures of posttrial strategy change. For example, if a large amount of conflict or a large error signal occurs, the usual hypothesis is that the resulting large ERN signals the need to reduce the conflict on the next trial or to slow down (e.g., Gehring et al., 1993; Jones et al., 2002). In other words, high conflict calls for increased control, or a large mismatch calls for a large correction. Nevertheless, there is no reason to rule out a strategy in which the subject strives for a large conflict signal. After all, on incongruent trials in a flanker task, a large amount of conflict could be an important cue to the subject that he or she is responding at the fastest level possible so as to avoid an error. In such a case, large amounts of conflict in the absence of an erroneous response would indicate that the focus of attention should stay the same. Similarly, what the ERN signal implies for post-error adjustments will depend on the psychological terrain: a large error or mismatch signal might indeed suggest that the subject responded too quickly and should slow down, but at other times it might call for attentional refocusing or no adjustment at all. The findings of Marco-Pallarés et al. (2008) discussed earlier highlight the need to consider the nature of the processes that

intervene between the ERN elicited by an error and the trial following that error—processes that might themselves vary according to the terrain.

In their attempt to navigate the psychological terrain, investigators must be careful to consider all the influences that come "along for the ride" in an experiment. With the ERN sitting at the interface of cognition, attention, emotion, motivation, and cognitive control, there are potential confounds galore in the interpretation of any particular study. As we have seen, two particularly important ones in the case of the ERN are task engagement and social evaluation, either of which can emerge as unwanted confounds in individual-difference studies. Individuals can differ in how engaged they are in the task and in how concerned they are with the wishes of the experimenter, and both factors can either produce misleading effects or mask the effects of interest.

Don't Give Up on Immediate Error Correction

The evidence we reviewed neither strongly supports nor strongly refutes the hypothesis that the ERN reflects a process involved in immediate error correction rather than long-term strategic adjustments. We suggest that there are good reasons to continue pursuing this hypothesis. Certainly the timing of the ERN is not as problematic as some have argued, with single-trial analyses showing that the ERN occurs while the subject is still processing and responding to the stimulus (Burle et al., 2008). Also, we reviewed several studies earlier where corrected or partial errors were associated with earlier ERNs than uncorrected errors (Carbonnell & Falkenstein, 2006; Endrass et al., 2008; Falkenstein et al., 1996; Fiehler et al., 2005; Hoffmann & Falkenstein, 2010; Vidal et al., 2000). Such a pattern would be consistent with the idea that immediate error correction cannot occur if the ERN is too late. There is little direct neurophysiological evidence, but microstimulation of the SEF, which may be involved in generating the oculomotor ERN, can influence the ongoing response (Stuphorn & Schall, 2006). A theoretical consideration is that if the process represented by the ERN evaluates responses and tunes future behavior, the information it has is incomplete, because at the time of the ERN it is not yet clear how the trial has turned out. At the very least, the system would perform better if other subsequent processes were also involved in such adjustments. Why, then, are studies so inconsistent in showing evidence for the immediate-adjustment hypothesis? It might be that the psychological terrain of the choice reaction time task is simply too barren to allow for an adequate test. It is helpful to keep in mind that most movements have a slower time course than a choice reaction time response, and it is probably the case that the process represented by the ERN evolved in the service of movements such as reaching and grasping, not for pressing E-prime button boxes.

As the ERN field turns to more complex motor tasks, a framework that should prove useful in pursuing a potential role for the ERN in immediate error correction comes from theories of internal models in optimal motor control (Wolpert & Flanagan, 2001). According to these theories, the brain predicts the state of the sensorimotor system as a movement occurs, adjusting the movement to recover from deviations from that prediction (but see Krigolson & Holroyd, 2006). A motor system with this ability would require an error signal and response adjustments to occur at a very short latency following the error. If the activity reflected by the ERN is used as corrective response adjustments unfold, then more complex, realistic movements might afford a better opportunity to observe the consequences of the ERN.

Represent the Representation

Despite their explanatory power, computational models such as the conflict model (Botvinick et al., 2001; Yeung et al., 2004b), and the RL-ERN model (Holroyd et al., 2005) have not been specific enough about the representations that they assume. The models also do not specify ways in which they could be extended to tasks for which they were not originally designed. One example is the case of the conflict model depicted in Figure 10.10: the *H* and *S* units do not specify whether activation of one unit over the other requires full categorization of a stimulus as *H* or *S*, or can simply be accomplished by the presence of straight lines and angles (for *H*) and curved lines (for *S*). In addition, the model fails to specify what will conflict and what will not: it does not specify whether responses in a four-choice (two-hand and two-foot) task will conflict more if they are similar (such as two responses by the same limb or two on the same side of the body) or different (e.g., a left-hand response vs. a right-foot response; Gehring & Fencsik, 2001). Nor does the model even specify what constitutes similar or dissimilar responses. The RL-ERN theory is also vague in the representation it assumes: does categorizing of a stimulus as rewarding or nonrewarding require attention? Is preattentive feature analysis sufficient

to elicit the FRN? If a more complex semantic categorization is necessary to determine whether a feedback stimulus conveys a reward, will that stimulus still elicit an FRN? A key insight of the RL-ERN theory—which applies to all of the theories—is that the representations involved in a comparison (or any other computation sensitive to similarity) may actually represent conjunctions of stimulus and response features: notions of stimulus mismatch or response mismatch alone may be too simplistic. The lack of specificity in the ERN modeling to date stems from a more fundamental problem: the models are free-floating in that they do not exist within a broader unified theory of cognition that specifies how the system reflected by the ERN interfaces with computations for evaluating stimuli and producing motor responses. Placing these models within a more comprehensive and general theoretical framework would force them to be more explicit about the representations they assume.

There's More to Life Than the Dorsal ACC and Dopamine

There is enough contradictory evidence to suggest that alternative sources for the ERN in the medial frontal cortex, including the rostral ACC and the pre-SMA, should be considered. The potential role of the SMA and pre-SMA in online control of movements suggests that these structures should be considered seriously as potential generators of the ERN. Moreover, although there has been a great deal of attention on a putative role for dopamine in generating the ERN, other neurotransmitters are also likely to be involved (Jocham & Ullsperger, 2009). New theoretical advances in the effort to explain the ERN will no doubt come about by broadening the structures and neurotransmitters under consideration.

Roll Up Your Sleeves and Collect More Data

Properly designed ERN experiments must involve a greater number of trials in the conditions of interest. All the sophisticated signal analysis in the world can't do very much for an average constructed from only 12 trials. Viable experiments will probably require multiple experimental sessions per subject (e.g., Gehring et al., 1992, 1993). To the extent that an analysis requires eliminating potential confounds, such as controlling for error trial RT when looking at the relationship with post-error slowing, experiments will be even longer. In our view, it would be better to spend 3 years collecting data that answer a single question conclusively than to spend those same 3 years running several experiments whose results are ambiguous. Collecting larger datasets would permit experimenters to deal more successfully with confounds that are no doubt responsible for many of the discrepant findings in the ERN literature. For example, studies that compare ERN waveforms between conditions or groups rarely attempt to equate such things as the distribution of response latencies in the trials that contribute to the average waveform, the proportion of corrected versus uncorrected errors, or the interval between the error and its subsequent correction. It is certainly inconvenient to carry out large-scale studies, but the cost of paying subjects to participate in several ERP sessions is very likely to be less than the cost of (say) running an fMRI study. The feasibility of using an ERN measure based on separate testing sessions is supported by its excellent test-retest reliability (Olvet & Hajcak, 2009a; Segalowitz et al., 2010). Indeed, it seems reasonable for investigators to consider adopting a more psychophysical approach, where a small number of subjects participate for tens of thousands of trials. Replication across studies would then play as important a role as within-study inferential statistics.

Our call to collect more data is contradicted by a recent report examining how many trials must be included in an average ERN waveform before that average becomes stable. Olvet and Hajcak (2009b) defined a stable average as one in which there is a high correlation between the average composed of a randomly chosen subset of error trials and the grand average from which that subset was drawn. They found that just 6 trials were necessary to achieve a high correlation between the subaverage and the overall average (consisting of 27 trials) and that 6 trials were sufficient to achieve a moderate level of internal reliability. Nevertheless, adopting the standard of six trials for ERN experimentation would be unwise. There are several reasons to be cautious. First, the Olvet and Hajcak study was based on a group of healthy subjects, a particular task (flanker), and a high-quality recording system. The extent to which the finding generalizes to other subject populations, tasks, or recording systems is unknown. Second, their finding pertains to within-subject stability of the error-trial waveform. It does not speak to the ability of standard analyses to find between-condition or between-group differences, nor does it address the stability of the error versus correct difference (as the ERN measure consisted only of the error-trial waveform). Finally, the standard used to measure stability—correlation of a grand average

with another average consisting of a subset of those trials—is imperfect, as it does not take into account the noise and overlapping potentials in the grand average that would make a comparison of conditions difficult in an actual experiment. Such complications necessitate more complex analysis methods, which themselves might require more trials. (We note that most of the problematic waveforms in Figure 10.21 included more than six trials.) Interestingly, more consistent with our own informal experience, Larson and colleagues (2010) recently examined a dataset in which adequate test-retest reliability could not be achieved even with an average of 14 error trials. Their grand mean consisting of 42 error trials, however, was temporally stable. Clearly, 42 is not the ultimate answer. With all the factors affecting the quality of ERP data, there is no ultimate answer. But that result does suggest that investigators would do well to design experiments as conservatively as possible to maximize the number of trials.

Ignore Component Overlap at Your Peril

Problems in identifying ERP components and isolating them from the other components that occur at the same time have bedeviled ERP research from the beginning. The problem with many studies not taking these issues seriously is that, in writing this review, we find ourselves in the position of Sutton and Ruchkin (1984): "we cannot be sure in reviewing earlier work in the field what components were, in fact, related to the experimental variables" (p. 1). The way for the field to extract itself from this situation is difficult but necessary: every experiment should use the best tools available for disambiguating the component structure of an ERP waveform. The outcome of using such tools might well be a finding that has nothing to do with the ERN, but it is better to acknowledge such a result than to make the muddy ERN waters even more opaque.

What tools to use? One relatively new method is time-frequency analysis, which offers a way to eliminate the contribution of components that lie outside the frequency band associated with the ERN (Bernat et al., 2005; Gehring & Willoughby, 2004; Hall et al., 2007; Trujillo & Allen, 2007; Yordanova et al., 2004). Figure 10.8 shows a time-frequency plot where ERN activity has been extracted from the lower-frequency (delta) component associated with the P300. In this case, the time-frequency analysis was augmented by a PCA to extract components from the time-frequency surface. Other useful techniques include methods for component identification such as independent components analysis (Debener et al., 2005; Luu et al., 2004) and spatiotemporal PCA (STPCA; Arbel & Donchin, 2009; Krigolson & Holroyd, 2007a). Also useful (but underutilized) are those techniques that capitalize on RT variability to separate stimulus- and response-related components, such as the plotting technique ERP image (Jung et al., 2001; see Figure 10.13), the Adjar method (Woldorff, 1993), and stimulus–response decomposition (Yin et al., 2009; Zhang, 1998).

Educate Your Editors about Converging ERP-erations

The advice outlined here implies that more experiments are needed that address fundamental issues in what Luck (2005) calls "ERPology," that is, experiments directed toward understanding the ERN itself. Important among these are experiments designed to rule out as-yet unnamed components as a source of an ERN effect. If a single experiment finds a putative ERN effect but the waveforms suggest that an overlapping ERP component might account for the effect, follow-up experiments are needed to rule out other components. Such experiments would follow the tradition of *converging operations* in psychology (Garner et al., 1956), where alternative explanations are ruled out by follow-up experiments. Unfortunately, the importance of such experiments might not be obvious to ERP-naive journal editors who, as Figure 10.21 suggests, are often unaware of the need for such work (and most likely don't see the connection between understanding an ERP component and testing a cognitive theory). Thus, writing reviews of journal articles provides a good opportunity to educate editors about the fine points of ERP research and the need for converging ERP-erations.

The ERN Is a Moving Target

It is comforting to think that ERN theories will continue to evolve and that in 20 years the best theory to explain the brain process represented by ERN might be a subpart of a successful grand unified theory of cognition. But it is disconcerting to think that the very definition of the ERN could also be completely different by that time—that in 20 years it might be more accurate to think of the ERN as a multipeaked oscillation, a joint negativity-positivity pair, or even a phenomenon that comprises activity in several frequency bands or several neural sources (Cohen et al., 2009). The problem is that, in assuming a single operational definition of

the ERN, a paper can lose relevance as our understanding of the ERN and the analytical techniques we use to measure it evolve. Even within our own era, it is not uncommon, in our experience, to read a paper and conclude that the author's measure failed to capture the true ERN.

Still, investigators can only dissect their data with the best tools available to them at the time. It would be a shame if all of the ERN studies published now became obsolete simply because ERPers of the future found out that present methods were inadequate. We suggest that the ERNologists of the world today should do more to preserve the fruits of their labor. The continued availability of raw data and the means to revisit them with new analytical techniques would ensure the continued relevance of present-day work. Although neuroimaging data repositories have met with mixed success (Barinaga, 2003), the obstacles seem worth overcoming.

The ERN: Whence? Where? Whither?

Although 20 years of ERN research have yielded an impressive set of findings and a number of interesting theories, it is also clear that things are more complicated than we thought they were 20 years ago. In this respect, we again find ourselves in a position similar to that of Sutton and Ruchkin (1984): "It is not an overstatement to say that, in a certain sense, we know less now than we thought we knew five to ten years ago" (p. 19). Still, we see this high degree of complication as a good thing: it bodes well for 20 more years of interesting and unexpected developments. And indeed, the degree of complication is due in part to the phenomena accumulating faster than satisfactory theoretical explanations for them.

In the same volume as the Sutton and Ruchkin paper, Terence Picton reflected on 20 years of ERP research by asking "Whence? Where? Whither?" (Picton & Cohen, 1984). He noted that ERP research often seemed disowned by—rather than integrated with—the parent disciplines of psychology and physiology and that further progress would require better integration. Drawing an analogy with art, he pointed to the paintings of Paolo Uccello, who had a brilliant understanding of perspective but whose work was limited because he had failed to integrate that knowledge with an accurate depiction of human figures and animals.

By Picton's standard, ERN research has achieved some success, being comfortably situated within the parent discipline of cognitive neuroscience. The challenge ERN researchers face after 20 years is to build a longer-lasting body of work. To draw an analogy to the art world more suitable for our times, Vincent Van Gogh's paintings have proven a challenge to preserve, because the organic red pigment that Van Gogh used fades when exposed to light. Van Gogh was aware that "paintings fade like flowers," and because of this he endeavored to create art that would stand the test of time: "All the colours that Impressionism has brought into fashion are unstable, so there is all the more reason to simply use them too brightly—time will tone them down only too much" (Van Gogh Museum Amsterdam, 2005). There is no doubt that the cognitive neuroscience of 100 years from now will see that contemporary work on the ERN has faded in some ways. But, like Van Gogh, ERN researchers can create a lasting body of work by painting the ERN canvas more thoughtfully, testing well-specified theories with the strongest data and the most rigorous, competitive empirical tests.

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