Published in final edited form as:
Psychophysiology. 2008 January ; 45(1): 152–170.

Influence of cognitive control and mismatch on the N2 component of the ERP: A review

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Abstract

Recent years have seen an explosion of research on the N2 component of the event-related potential, a negative wave peaking between 200 and 350 ms after stimulus onset. This research has focused on the influence of "cognitive control," a concept that covers strategic monitoring and control of motor responses. However, rich research traditions focus on attention and novelty or mismatch as determinants of N2 amplitude. We focus on paradigms that elicit N2 components with an anterior scalp distribution, namely, cognitive control, novelty, and sequential matching, and argue that the anterior N2 should be divided into separate control- and mismatch-related subcomponents. We also argue that the oddball N2 belongs in the family of attention-related N2 components that, in the visual modality, have a posterior scalp distribution. We focus on the visual modality for which components with frontocentral and more posterior scalp distributions can be readily distinguished.

Descriptors

N2; N200; Cognitive control; Error-related negativity; Feedback-related negativity; Novelty

The P300 may be the most studied component of the event-related potential (ERP). Its amplitude is sensitive to stimulus probability and task relevance of the eliciting stimulus, and its latency reflects stimulus evaluation time (Johnson & Donchin, 1980, Chapter 12; Verleger, 1997). The P300 is commonly divided into two subcomponents with different scalp distributions and different functional correlates: a frontally maximal P3a that reflects the orienting of attention to unexpected or significant events in the environment and a parietally maximal P3b that indexes the updating of working memory (Courchesne, Hillyard, & Galambos, 1975; Debener, Makeig, Delorme, & Engel, 2005; Dien, Spencer, & Donchin, 2004; Donchin, 1981; Friedman, Cycowicz, & Gaeta, 2001; Goldstein, Spencer, & Donchin, 2002; Näätänen & Gaillard, 1983; Polich & Comerchero, 2003; Spencer, Dien, & Donchin, 1999, 2001; Squires, N. K., Squires, & Hillyard, 1975).

Commonly observed in combination with the P3a and P3b is the smaller, earlier N2. The label "N2" refers to the second negative peak in the averaged ERP waveform and is labeled as such because it follows a prominent frontocentral negative peak at around 100 ms in the auditory modality or a prominent temporo-occipital negative peak at around 180 ms in the visual modality. So linked were the N2 and P3 in early research that they were often referred to as the "N2-P3 complex," and some studies measured them in combination for purposes of

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This review formed part of a dissertation submitted in partial requirement for a doctorate in Psychology from the University of Arizona.

correlation with stimulus probability (Squires, K. C., Petuchowsky, Wickens, & Donchin, 1977; Squires, K. C., Wickens, Squires, & Donchin, 1976).

Recent years have seen a renewed interest in N2 components of the ERP as functionally distinct from P3 components and useful for understanding the nature and sequence of cognitive processes. For instance, a large literature has emerged focusing on the role of anterior N2s in cognitive control. *Cognitive control* is partly defined as the monitoring or regulation of strategy ("How fast am I responding?" "How fast *should* I be responding?") and the processing of feedback that is informative for strategy regulation ("Another mistake"; "That reward was worse than I expected"; etc.). Additionally, the concept of cognitive control covers immediate control of action, such as canceling a prepared response. However, studies of cognitive control exist against a background of both older and recent work showing N2 modulations that are more readily attributed to the detection of novel stimuli and to the orienting of visual attention.

The goal of the current article is to review both classic and recent studies that show what experimental factors modulate N2 amplitude and to propose a classification scheme for what are clearly multiple negative components peaking 250–300 ms after stimulus onset. We will suggest that the N2 elicited by visual stimuli should be divided into at least three subcomponents: a frontocentral (anterior) component related to the detection of novelty or mismatch from a perceptual template when the eliciting stimuli are attended, a second frontocentral component related to cognitive control (encompassing response inhibition, response conflict, and error monitoring), and one or two posterior N2s related to some aspects of visual attention. Some of these components—particularly those with posterior scalp distributions—are likely to be specific to the visual modality, whereas the modality dependence of anterior N2s is less clear, particularly in cases where they might overlap with the auditory mismatch negativity (MMN) and make component identification difficult. We thus focus on the visual modality here, with auditory studies noted only as background (for reviews of the MMN, see Näätänen, 2001; Näätänen & Alho, 1997; Ritter, Deacon, Gomes, Javitt, & Vaughan, 1995).

Organization of the Review

We begin with a synopsis of N2 classification schemes proposed in the 1970s through early 1990s. This is followed by a brief review of two-stimulus "oddball" paradigms manipulating stimulus probability and response probability; these provide the first suggestion of distinct anterior and posterior N2s and indicate that the anterior N2 is primarily elicited by trials with no response (*no-go* trials). We then review studies using a variety of paradigms suggesting that an anterior N2 is also sensitive to perceptual novelty or template mismatch and can be dissociated from both the frontal P3 (P3a) and more posterior N2s. We then review evidence that the anterior N2 is sensitive to cognitive control and the arguments for associating N2 effects with response inhibition, response conflict, and error monitoring. We conclude with evidence from both ERP and hemodynamic imaging studies regarding associations and dissociations between perceptual novelty and cognitive control, factors that are both reflected by anterior N2s.

N2 Classification Schemes: 1970s through Early 1990s

In the late 1970s and early 1980s, it was proposed that, like the P3, the N2 could be divided into subcomponents called the N2a and the N2b (for reviews, see Näätänen & Gaillard, 1983; Näätänen & Picton, 1986). Somewhat confusingly, the N2b, not the N2a, was consistently observed in combination with the P3a, and, based on further oddball studies in the visual modality, it was proposed that a third N2 subcomponent, the N2c, was the companion of the P3b (Ritter, Simson, Vaughan, & Friedman, 1979; Ritter, Simson, Vaughan, & Macht, 1982).

This classification scheme was formalized in an influential review by Pritchard, Shappell, and Brandt (1991) that laid out the properties of the N2a, N2b, and N2c in the auditory and visual modalities. Key differences between the components were that the N2b and N2c—but not the N2a—required attention to the eliciting stimulus and were always accompanied by P3 components.[1] The historical label "N2a" has now been largely replaced by *mismatch negativity* for the auditory modality and the putative visual correlate of this potential by "visual MMN," so that we use this more modern terminology below. In the 1991 scheme, the N2b was larger to nontargets than to targets but elicited by both, had a central scalp distribution in both the auditory and visual modality, and was accompanied by a P3a. The N2c was distinctive in that, unlike the MMN and N2b, its latency covaried with reaction time, it was larger to targets than to nontargets, and its scalp distribution was modality specific, being posterior in the visual modality and frontocentral in the auditory modality. The N2c was thought to reflect a subprocess of stimulus classification whereas the MMN and N2b/P3a complex were thought to index different stages of mismatch detection. Näätänen and Gaillard (1983) argued that these mismatch detectors played a key role in triggering the orienting reflex.

If this characterization of the three N2 subcomponents is correct, the frontocentral N2 to rare auditory targets is often a mixture of the MMN, N2b, and N2c, all of which have central or frontocentral scalp distributions in the auditory modality. In revisiting some of the older literature, therefore, we will refer mainly to studies using visual stimuli, as we consider them to be somewhat less ambiguous in differentiating the N2b from the N2c. Because we will conclude that the N2b and N2c may also need subdivision, we will initially adopt neutral terms of *anterior N2*, referring to a negative-going wave with a frontal or central scalp maximum and corresponding to Pritchard et al.'s (1991) N2b, and *posterior N2*, referring to a negative-going wave with a parietal or more posterior scalp maximum and corresponding to Pritchard et al.'s N2c.

The Two-Stimulus Oddball Paradigm: Influences of Stimulus Probability and Response Requirements

The two-stimulus oddball paradigm is one of the simplest and most frequently used in ERP research. In the most common version, a rare target stimulus receives an overt (button press) or covert (silent count) response whereas a frequent standard receives no response. In this most standard version of the oddball paradigm, rare visual targets elicit a larger N2 over parietal, temporal, and occipital scalp, followed by a larger P3b (Ritter et al., 1982; Ritter, Simson, & Vaughan, 1983; Simson, Vaughn, & Ritter, 1977). Like the P3, the latency of this posterior N2 covaries with reaction time (Ritter et al., 1982, 1983). Posterior negativities in the N2 time range have been extensively studied in the context of attention paradigms, including visual search (N2pb and N2pc; Conci, Gramann, Müller, & Elliott, 2006; Hickey, McDonald, & Theeuwes, 2006; Hopf et al., 2000; Hopf, Luck, et al., 2006; Luck & Hillyard, 1994a; Schubo, Wykowska, & Muller, 2007; Woodman & Luck, 1999, 2003) and attention to relevant stimulus features (selection negativity or SN; Anllo-Vento, Luck, & Hillyard, 1998; Eimer, 1997; Harter & Guido, 1980; Harter & Previc, 1978; Jonkman, Kenemans, Kemner, Verbaten, & van Engeland, 2004; Kasai & Morotomi, 2001; Kenemans, Lijffijt, Camfferman, & Verbaten, 2002; Martin-Loeches, Hinojosa, & Rubia, 1999; Martinez, Di Russo, Anllo-Vento, & Hillyard, 2001; Okita, Wijers, Mulder, & Mulder, 1985; Smid, Bocker, van Touw, Mulder, & Brunia, 1996; Smid, Jakob, & Heinze, 1997, 1999). Both the N2pc and SN are enhanced by the presence of relevant elements of a stimulus array or relevant stimulus features, and both components are largest contralateral to those stimulus elements when they are presented lateral

[1] In the years since Pritchard et al.'s 1991 review, it has become clear that the N2a/MMN is sometimes followed by a P3a and that modulations of other N2s can be observed without changes in P3 amplitude. The MMN is not covered here, but the remainder of the review will make the relative independence of the visual N2 and P3 clear.

to fixation (Anllo-Vento & Hillyard, 1996; Hillyard & Münte, 1984; Karayanidis & Michie, 1996; Lange, Wijers, Mulder, & Mulder, 1998; Luck & Hillyard, 1994a, 1994b). Second, both the SN and N2pc have likely sources in occipito-temporal and occipital cortex, including area V4 (Anllo-Vento et al., 1998; Hopf et al., 2000; Hopf, Luck, et al., 2006; Luck, Chelazzi, Hillyard, & Desimone, 1997; Luck, Girelli, McDermott, & Ford, 1997; Martinez et al., 2001). Although a thorough review of this literature is beyond the scope of this article, we note that these well-documented posterior negativities in the N2 time range likely share functional correlates with the posterior N2 elicited in the two-stimulus oddball paradigm.

Although the oddball N2 difference wave is much more prominent over posterior than anterior electrodes (where it is often completely absent), a small anterior N2 difference with later peak latency has been observed inconsistently (Breton, Ritter, Simson, & Vaughan, 1988; Czigler, Csibra, & Ambro, 1996; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). Partially accounting for this result is the fact that, in the standard oddball paradigm, stimulus probability and response probability (whether overt or covert) are perfectly confounded, in that rare stimuli call for go responses and frequent stimuli call for no response (no-go). Several studies have separated the influences of visual stimulus probability from go versus no-go response requirements in two-stimulus paradigms. Pfefferbaum, Ford, Weller, and Kopell (1985) first demonstrated that when targets (go) and nontargets (no-go) were presented with equal probabilities, no-go trials elicited a larger N2 at a midline frontal scalp site (Fz) in both button-press and counting tasks, followed by a larger frontal P3 (see also Kok, 1986). Czigler et al. (1996) extended this result by crossing stimulus probability (20% vs. 80%) with go/no-go response requirements. This study was primarily directed at age differences, such that the N2 results from young adults are described but not statistically analyzed. Figures show that a larger N2 for the low-probability stimuli was observed for both go and no-go responses at posterior scalp sites T5, T6, and Oz. However, the probability effect on the N2 recorded at Fz and Cz was much larger in the no-go case than the go case.

Bruin and Wijers (2002) similarly crossed stimulus probabilities of 25%, 50%, and 75% with go/no-go response requirements, but with statistical analysis of N2 at midline sites Fz, Cz, Pz, and Oz. As the probability of no-go (nontarget) stimuli decreased, the N2 elicited by these events increased in amplitude, with the largest probability effect at Fz. However, the N2 elicited on go (target) trials was reportedly diminished by decreasing probability at both Fz and Cz. The authors suggest that the latter result was less secure than the former, because of the possibility of overlapping potentials from motor preparation. Nieuwenhuis et al. (2003) also crossed three levels of stimulus probability (20%/50%/80%) with go/no-go requirements. A clear effect of probability was apparent for an anterior N2 (measured at FCz) on no-go trials, but only a weak nonsignificant probability effect was observed for go trials in stimulus-locked averages. In averages time-locked to the button-press response rather than stimulus onset, low-probability go trials elicited a larger negative wave peaking at ~75 ms prior to the response than high-probability go trials, but this effect remained smaller than the probability effect for no-go trials.

Interim Summary

In paradigms with two stimuli—only one of which calls for a response—a posterior N2 is larger for rare targets than for common nontargets and precedes a parietally maximal P3 (P3b) that is similarly sensitive to target status and probability. At frontal and/or central scalp sites, an N2 is instead larger for nontarget than target trials, those calling for no response. Ample subsequent studies have confirmed the reality of the *no-go N2* (Eimer, 1993; Jodo & Kayama, 1992; Kok, 1986; Kopp, Mattler, Goertz, & Rist, 1996), such that the latency of this potential has been a useful tool for tracking the timing of access to different properties of a stimulus (Schmitt, Münte, & Kutas, 2000; Schmitt, Rodriguez-Fornells, Kutas, & Münte, 2001; Schmitt,

Schlitz, Zaake, Kutas, & Münte, 2001). The no-go N2 is evident primarily when no-go trials are less probable than go trials, but can also be observed when go/no-go probabilities are equal. Similar to the association between the posterior N2 and the parietal P3 (N2c and P3b in Pritchard et al.'s 1991 terminology), the anterior no-go N2 is typically followed by a frontal P3 (N2b and P3a).

Based on the no-go results and other evidence, some investigators have argued that the anterior N2 reflects the monitoring of response conflict and is generated in the anterior cingulate cortex (Nieuwenhuis et al., 2003; Yeung, Botvinick, & Cohen, 2004). This hypothesis stresses that no-go trials require overriding a prepotent response, particularly when no-go trials are less probable than go trials. The smaller no-go N2s obtained when go and no-go trials are equiprobable is attributed to a continuing bias to respond, due to the speeded response requirements that are typical in two-stimulus target detection paradigms. Finally, Nieuwenhuis et al. have argued that the small stimulus probability effect they observed for go trials (in response-locked averages) indexes a degree of response conflict when “go” is the low-probability response.

N2 results during tasks that tax response inhibition and other aspects of cognitive control are reviewed extensively later in this article. Just below, however, we turn to studies suggesting that an anterior N2 is sensitive to the detection of perceptual novelty or attended deviation from a visual template. The novelty/deviation/mismatch studies also provide further support for the dissociation between anterior and posterior N2s and additionally dissociate the anterior N2 from the frontal P3a.

The Frontocentral N2 Is Sensitive to Visual Novelty and Attended Mismatch from a Visual Template

The Oddball Paradigm Revisited

The sensitivity of the P3b to probability is usually described in terms of *categorical probability*, or the class to which a stimulus is assigned by a set of experimental instructions (Johnson & Donchin, 1980). For instance, Kutas, McCarthy, and Donchin (1977) recorded a large parietal P3 to the rare stimulus “John” as compared to the frequent stimulus “Mary” when participants were asked to discriminate male from female names, but an equally large P3 to a variety of male names mixed with a variety of female names that occurred with higher categorical probability. This result indicates that P3b amplitude is not determined by stimulus probability per se, or else the variable-name list would have led to equivalent P3s for each (equally low-probability) name.

Kutas et al. (1977) did not report N2 amplitudes, but a later study by Breton et al. (1988) examined the impact of target stimulus variability and obtained a clear dissociation between N2 and P3b. One block of trials was a standard 80/20 oddball paradigm, with responses to a rare target letter and no response to a different single letter. The response requirements and categorical probabilities were the same in another block of trials, but the targets now consisted of any of 25 possible letters. The targets in this second block were thus 20% probable collectively, but less than 1% probable individually. The parietal P3 and posterior (temporal and occipital) N2 were larger for rare targets than frequent nontargets in both blocks, but unaffected by the variability in physical identity of the target. In contrast, the N2 at Fz and Cz was larger for the 25-target condition than the unitary target condition. The influence of target variability on anterior N2 amplitude was unaccompanied by any amplitude change in positive components. Comparison of a third and fourth block of trials confirmed the effect of pure stimulus probability on the frontocentral N2. In the third condition, called “paired deviants (2)”, an “X” appeared on 68% of the trials, and an “O” appeared on 16% of the trials, which

could be followed by another "O" (8%) or an "I" (8%). Participants responded to the second "O" with one finger and to the "I" with another finger, but we are currently concerned only with the initial stimulus that called for no response, but instead signaled an upcoming stimulus that needed to be identified. In the "paired deviants (25)" condition, 25 different letters appeared with a collective probability of 16%, and were thus individually very rare. As before, the variable set of stimuli elicited a larger anterior N2 than the rare but unitary stimulus, and this effect could not be attributed to response probability.

The Frontal Novelty Effect: N2 and P3a

Courchesne et al. (1975) were the first to report a novelty effect for visual stimuli. Stimuli were 80% nontargets (the numeral "2"), 10% targets that were silently counted (the numeral "4"), and 10% nontargets that were unique on each trial. For some participants, the rare nontargets were complex and random colored patterns (*complex novels*); for others, the novel stimuli were evenly divided between complex novels and *simple novels* consisting of interpretable black and white stimuli (words, faces, geometric shapes, etc). As compared to either the frequent nontargets or the rare targets, the complex novel stimuli elicited a larger frontal N2 and P3. Although equally rare, the simple novels were much less effective in eliciting this frontal novelty response. Other results from the same large study showed that (1) the N2/P3a response to complex novels habituated rapidly if stimuli were repeated and (2) that the frontal novelty response was not augmented by making these stimuli targets, although the parietal P3b was increased.

Multiple studies have replicated the impact of visual novelty on the frontocentral N2, with variable degrees of dissociation from the P3a and from the posterior N2. Polich and Comercho have examined visual ERPs to rare nontargets in the context of frequent standards and rare targets (Comerchero & Polich, 1998, 1999; Demiralp, Ademoglu, Comerchero, & Polich, 2001; Polich & Comerchero, 2003). They found that, as in auditory studies, both simple repeated nontargets and unique novel nontargets elicited frontal P3a components and that these P3a's were larger when the target/standard discrimination was more difficult. N2 components were not analyzed in these studies, but inspection of figures suggests that amplitude of an anterior N2 was more sensitive to the degree of perceptual deviation from other stimuli and less sensitive to task difficulty than the P3a. In one paradigm, difficulty of target–standard discrimination (easy: letter "A" vs. letter "B"; difficult: large circle vs. slightly smaller circle) was crossed with four types of rare nontarget stimuli: the letters "C" thru "Z," a square with an area similar to the target and standard stimuli, a much larger square, and complex colorful squares that were unique from trial to trial (Demiralp et al., 2001; Polich & Comerchero, 2003). The frontal P3 to all types of rare nontargets was larger in difficult than easy tasks, and larger for both large squares and novel colorful squares than for letters and small squares. In both task conditions, the P3 to large squares and colored squares was identical at Fz. Inspection of the waveforms, however, suggests that the frontal N2 was evident primarily for novel stimuli and not influenced by task difficulty.

Other studies have explicitly analyzed N2 components in addition to positive components. Thomas and Nelson (1996) demonstrated an age effect on the anterior N2 response to visual novelty. Their stimuli were color photos of a frequent target face (60%), a target face (20%), and trial-unique nontargets that were a mixture of objects, scenes, and abstract patterns. ERPs from young adults and eight-year-old children showed a larger parietal P3b for both targets and novels relative to the frequent standard, but in contrast to many studies, no sign of a frontal N2/P3a complex in response to novels in the adults. The children showed a substantial anterior N2 for the novel photos, unaccompanied by a subsequent frontal P3. The lack of a novelty response in young adults in this study is surprising. It is possible that this absence arises from a slightly higher proportion of novel stimuli than in other studies; the impact of the probability

of novel stimuli has not yet been investigated. However, the child data in this study provide a convincing dissociation between the anterior N2 and frontal P3a.

Czigler and Balázs (2005) likewise found a decline in the N2 novelty response with increasing age. Stimuli were four letters arrayed at the corners of an invisible square; on different runs, participants were instructed to respond to matching letters on one of the two diagonals of the square. Stimulus probabilities were 65% for arrays with no matching letters, 15% with a match on the attended diagonal, 15% with a match on the unattended diagonal, and 5% trial-unique black and white line drawings of familiar objects. In young adults, but not in older adults, the novel stimuli elicited a very large N2, which was of maximal amplitude at the central midline (Cz), slightly smaller at the frontal midline (Fz), and barely visible at both the prefrontal and occipital midline sites (Fpz and Oz). In this study, the N2 portion of the frontal novelty complex was very much larger than the P3a, which was only visible at Fpz. There was little sign of a posterior N2 difference among any of the conditions, in contrast to some of the studies described above in which target stimuli elicit reliable posterior N2s as compared to nontarget stimuli.

What Makes a Stimulus Novel?

It should be noted that the novel stimuli in Czigler and Balázs (2005) appear to be like Courchesne et al.'s (1975) “simple novels”—interpretable rather than meaningless random shapes. In the earlier study, the simple novels were relatively ineffective in producing a frontal novelty response. Because Courchesne et al. presented simple novels intermixed with complex novels in the same blocks, the contrasting results may indicate that what counts as “novel” may depend on the overall composition of a stimulus list, rather than only the eliciting stimuli themselves.

Daffner and colleagues returned to the question raised by Courchesne et al.'s (1975) initial report of the visual novelty response: Why did the meaningless “complex novel” stimuli elicit a larger frontal N2/P3a response than the interpretable “simple novel” stimuli? Both were equally rare in the context of the experiment, but one could argue that the “complex novels” were more deviant from long-term memory representations. Daffner et al. (2000) compared three variants of the three-stimulus odd-ball paradigm, in which stimuli consisted of a single frequent standard (70%), a single rare target (15%), and rare nontargets (15%) that were unique on each trial. Stimuli were simple geometric shapes or “unusual” shapes—black and white line drawings of pseudo-objects that could not exist in three-dimensional space and complex scribblelike shapes. In the *all-simple* block, standards, targets, and nontargets were all simple geometric shapes and no N2 differences among conditions were observed. In the *mixed* block, standards and targets were single complex shapes and nontargets were multiple simple shapes. The mixed block is of particular interest because it tests the hypothesis that the N2 is sensitive not to complexity or unfamiliarity per se, but to a salient difference in category between the standard and rare nontarget stimuli. If only unfamiliarity drives the N2, one would expect the simple nontargets in the mixed block to elicit the same N2 amplitude as the stimuli in the all-simple condition. If a contrast of category between deviants and standards partly drives the N2, one would expect the deviants in the mixed block to elicit a larger N2 than the all-simple stimuli. In fact, influences of both the short-term experimental context and longer term unfamiliarity were observed. The frontocentral N2 for simple deviants in the mixed block was larger than the N2s to target, standard, and deviants in the all-simple block, which did not differ from each other. This pattern of results is consistent with a description of the N2 novelty effect as arising from deviation from a predominant stimulus category. At the same time, there was also a clear and large effect of long-term unfamiliarity. Unusual stimuli elicited larger anterior N2s than simple stimuli, whether they served as standards, targets, or rare nontargets. Across all of the blocks, the parietal P3b was largest for targets, of intermediate amplitude for rare

nontargets, and smallest for standards. Frontal positivities were generally small in amplitude, but followed the same gradient in all three blocks, such that the results securely dissociate the P3a from an anterior N2 (consistent with our visual analysis of the results of Demiralp et al., 2001, and Polich & Comerchero, 2003).

Overall, novelty oddball paradigms show that, to effectively drive the novelty N2, visual stimuli must be either highly unfamiliar and thus deviate from long-term context or deviate considerably from short-term context. Simple shapes that are merely infrequent appear to be insufficient to yield a substantial anterior N2, although they may elicit a frontal P3a and/or a *posterior* N2 (Alho, Woods, Algazi, & Näätänen, 1992; Maekawa et al., 2005).

Dissociating the Anterior Novelty N2 from Posterior Target N2

A study by Suwazono, Machado, and Knight (2000) provides one of the clearest dissociations between an anterior N2 elicited by novelty and a posterior N2 sensitive to target status. Stimuli were 70% frequent standards and 20% rare targets (upright and inverted triangles), together with trial-unique color photos that served as rare novel nontargets on 10% of the trials. All stimuli were presented unpredictably to the left or right visual field. In a between-participants manipulation, novel stimuli predicted that the subsequent stimulus would be a target with 100% validity, 40% validity, or 20% (chance) validity. Target stimuli elicited a parietal P3b, preceded by a pronounced N2 at temporal and occipital scalp sites. As in earlier reports (Heinze, Luck, Mangun, & Hillyard, 1990; Woods, Alho, & Algazi, 1992), the target N2 was larger over scalp sites contralateral to the field of stimulus presentation. When targets were completely predictable from the preceding stimulus, the target P3 was substantially reduced in amplitude, and the posterior N2 was essentially eliminated. When novel stimuli had no predictive value, they elicited an N2 largest over frontal and central scalp, with no bilateral asymmetry. When novel stimuli served as completely valid cues for upcoming targets, they additionally elicited a targetlike posterior N2. Increasing the predictive value of the novels also yielded an increase in amplitude of the N2 recorded at frontocentral sites; because this increase was more evident at central than frontal sites, it is difficult to determine if it reflects a modulation of the anterior N2 per se or spillover from the more posterior N2. Novel stimuli also elicited a small frontal P3a in this study, which was reported as smaller when the perceptually novel items served as signals for upcoming targets. This latter finding may also be subject to difficulty in isolating the P3a from an overlapping anterior N2, but it remains clear that the anterior N2, posterior N2, and positive components responded differently to the manipulations of target status, novelty, and target predictability in this study.

Suwazono et al. (2000) concluded that the posterior N2 reflected the degree of attention required for processing stimuli in visual cortex whereas the novelty N2 reflected a “general alerting system” (p. 38) that was not modality specific. Although this interpretation emphasizes the orienting rather than mismatch detection aspect of the novelty N2, orienting and mismatch detection could be two sides of the same coin. It is possible, for instance, that mismatch detection is amplified when mismatches cue a significant event.

Dissociating the Anterior Novelty N2 from the Posterior N2 in Visual Search Paradigms

Another dissociation between anterior and posterior N2 components comes from a series of experiments by Luck and Hillyard (1994a) in which participants fixated on a central point while presented with an array of simple bars spanning all four visual quadrants. Participants responded to laterally located “pop-out” elements within these arrays that differed from the background elements in color, orientation, or size. Depending on the particular experiment, the target was sometimes any pop-out and sometimes a pop-out of a particular feature. When compared to homogenous arrays, arrays with target pop-outs elicited a pattern very similar to the one elicited by relevant stimuli in the multidimensional target detection tasks described in

the previous section: an early frontal positive difference, followed by a posterior negative enhancement from about 200 to 275 ms and a central negativity from about 250 to 325 ms. At posterior electrodes, the negative enhancement was larger contralateral than ipsilateral to the target pop-out from 200 to 300 ms. This lateralized negative difference was called the N2pc. Across multiple experiments, it was further observed that the anterior N2 was elicited by both target and nontarget pop-outs, whereas the N2pc was only elicited by targets. Finally, neither the anterior N2 nor the N2pc was affected by the probability of target pop-outs, but lower probability targets did enhance the posterior N2 *bilaterally*. This pattern suggested the presence of three separate N2 components: the N2pc, the anterior N2, and a bilateral posterior N2 called the *N2pb* that was sensitive to target probability, similar to the P3b (Luck & Hillyard, 1994a). Subsequent experiments have implicated the N2pc in the suppression of visual noise surrounding a target (e.g., Hopf, Boehler et al., 2006; Luck & Hillyard, 1994b), but no experiments to our knowledge have jointly investigated the anterior N2 and bilateral posterior N2 (*N2pb*) identified by Luck and Hillyard (1994a).

The lack of sensitivity of the anterior N2 to target probability tends to suggest a mismatch rather than control interpretation, the mismatch in this case being between the homogenous background elements and the pop-out elements used by Luck and Hillyard (1994a). However, because more recent studies of visual search have focused on the N2pc, the particular sensitivities, replicability, and localization of the anterior N2 and N2pb in visual search paradigms remain to be fully understood.

Summary of Novelty Manipulations

Taken together, the results above are consistent with an N2 sensitive to the mismatch of a stimulus with a mental template but suggest the extension of the concept of "template." Specifically, mental templates can be formed by long-term or short-term experience, and multiple templates may be active simultaneously. They also suggest a dissociation between the anterior N2 and P3a, with the anterior N2 more sensitive to departures from predominant stimulus and stimulus category and the P3a more sensitive to distraction from mental set.

Novelty N2 and response inhibition?—Although the amplitude of an anterior N2 is enhanced on no-go as compared to go trials (introduced above under "Two-stimulus oddball paradigms" and reviewed in more detail under "The no-go N2 and cognitive control," below), it is difficult to see a relationship between the anterior novelty N2 and inhibition of a prepotent response or response conflict. In the three-condition oddball paradigms reviewed above, both frequent nontargets and novels are associated with no response, yet novels elicit larger anterior N2s. Moreover, frequent nontargets are more perceptually similar to targets than are the novels and should thus engender more conflict as to whether or not to respond, rather than less. There remains one sense in which a novelty response could be tied to a more general sort of motor inhibition, in that cessation of ongoing behavior has traditionally been considered a part of the orienting response to change in the environment, together with a host of autonomic responses (Sokolov, 1963; for a review, see Öhman, Hamm, & Hugdahl, 2000). However, even this idea is somewhat difficult to apply to the experiment of Suwazono et al. (2000), in which novel stimuli were predictive of upcoming targets. Even when targets occurred 200 ms after a novel —during the anterior N2—reaction times were faster than when targets followed a frequent standard stimulus. This finding seems inconsistent with the notion that the novelty N2 indexes a brief period of motor inhibition.

N2s in Sequential Matching Tasks

Above, we have suggested that the anterior N2 "novelty response" may reflect deviation from a perceptual template, based on both long-term experience with visual objects and shorter-term exposure to recurring standards and targets in the novelty oddball paradigm. An early hint at

this idea comes from the finding that, in contrast to the standard oddball paradigm, the frontocentral N2 is enhanced when oddball targets are gaps ("missing stimuli") in a train of regular standard stimuli (Czigler et al., 2006; Simson, Vaughan, & Ritter, 1976). Sequential matching tasks—in which participants are asked to judge whether a second stimulus is the same or different from an initial stimulus—may tap a similar sort of "template-matching" process. This possibility is examined below.

The Sternberg paradigm for studying short-term memory is one such paradigm: Participants receive a small memory set of one to four items, followed by a probe item that requires a positive or negative judgment depending on whether it appeared in the memory set. Many studies have examined ERPs in the Sternberg paradigm, with a focus on the latency of the decision-related P3b and its relation to memory set size, but with little attention to other components (e.g., Ford, Roth, Mohs, Hopkins, & Kopell, 1979; Marsh, 1975; Pelosi, Hayward, & Blumhardt, 1995; for a review, see Verleger, 1997). In some studies, only positive probes (those in the memory set) are assigned to a response, and negative probes are no-go trials, so that the effect of memory-set/probe mismatch cannot be examined separately from the effect of response requirements. However, at least two studies with responses to both types of probes indicate that negative probes elicit a larger negative potential, with peak latencies of 270 and 340 ms in the different reports (Kotchoubey, Jordan, Grözinger, Westphal, & Kornhuber, 1996; Kramer, Strayer, & Buckley, 1991). In both reports, the probe-type effect was largest at frontal sites, of intermediate size at central sites, and small to absent at parietal and more posterior sites. Kotchoubey et al. additionally reported that the mismatch effect was largest when the memory set consisted of only a single item, namely, trials in which the comparison and probe stimulus occurred in immediate succession. The match/mismatch effect was still observed with memory sets of two and four items, but by chance, some of these trials would also have included matching memory set items and probes that occurred in immediate succession, so that it is not possible to derive a function of how the match/mismatch effect might decline across intervening stimuli.

In a series of studies, Wang and colleagues have observed that N2 amplitude is enhanced when the second stimulus in a pair does not match the first. In a 2003 study, stimuli were geometric figures with four possible shapes, four possible colors, and a small gap in the outline that occur in one of four positions (Wang et al., 2003). The second stimulus of a sequential pair matched the first on all three features (33%), matched on none (33%), or was a partial match (33%, with all six varieties of partial match equally represented). Data from only the two conditions of complete three-feature matches or complete mismatches were presented. In one block of trials, participants judged colors alone as matching or mismatching, signaled by two different button presses (choice RT, 50/50 response probability). A negative potential peaking at 270 ms was larger for mismatches than matches. In other blocks of trials, participants judged stimuli as matching or mismatching on both color and shape and all three features. In these tasks, partial matches on the task-relevant dimensions were no-go trials (and not analyzed). Across blocks, the number of task-relevant dimensions were thus either one, two, or three. The N2 mismatch effect did not vary across number of relevant dimensions: Complete mismatches elicited equally large N2s and complete matches equally small ones. The N2 effect in this study appeared to be of nearly equal amplitude at frontal, central, and parietal sites but much smaller occipitally. Although clearly distinct from the posterior N2 target effect in other studies, this topography appears to extend somewhat more posterior than that of the novelty effects reviewed above (see also Cui, Wang, Wang, Tian, & Kong, 2000; Kong et al., 2000).

A follow-up experiment by Wang, Cui, Wang, Tian, and Zhang (2004) provides more information about the impact of mismatching features in task-irrelevant dimensions. Stimuli possessed two features of shape and color, and participants performed three tasks: match on color alone (choice response, 50/50 response probability), match on shape alone (50/50

response probability), or match/mismatch on both (25% positive response, 25% negative response, 50% no-go for partial matches). For the single-dimension tasks, N2 amplitude followed a clear gradient: largest amplitudes when both features failed to match the comparison stimulus, intermediate amplitudes when the task-relevant feature matched but the irrelevant feature was a mismatch, and smallest when both features matched. As in the previous study, the mismatch effect had a fairly broad frontal-central-parietal scalp distribution. When the complete (two-feature) matches were contrasted with complete mismatches across the three tasks, the N2 difference was, surprisingly, slightly smaller in the two-dimension task than the one-dimension tasks, but was followed by a later negative difference peaking at ~400 ms. In contrast to the one-dimension task, the two-dimension task required analysis of a conjunction of features because the three cases of "both same," "both different," and "one same, one different" were assigned to three different responses (two buttons and no-go). The late negative mismatch response may have reflected a second processing stage of comparing a conjunction of current attributes to a prior stimulus.

Kimura, Katayama, and Murohashi (2006) further explored the effect of change in irrelevant stimulus dimensions. Participants responded to rare changes in orientation between pairs of gratings. For the majority (80%) of paired stimuli with no orientation change, half differed on the irrelevant dimension of spatial frequency. These irrelevant-change trials elicited a larger frontocentral N270 component than no-change trials, and the amplitude of the negative component tracked the magnitude of the task-irrelevant change, as did the false-alarm rate to the irrelevant changes. An interesting contrast to both Wang et al. (2004) and Kimura et al. is provided by a third study that also investigated the effect of irrelevant change between paired stimuli (Fu, Fan, & Chen, 2003). Rather than rare *changes*, participants detected rare *target* gratings (those with a particular spatial frequency), which could occur in either first or second position of the pair. Of the frequent pairs in which neither position contained a target, the second stimulus either matched or mismatched the first stimulus on the irrelevant dimension of orientation. Task-irrelevant mismatches/changes elicited no sign of an enhanced anterior N2 in this study, suggesting that active attention to change is necessary for the elicitation of this component.

Yet another study by Wang and colleagues (Zhang, Wang, Li, & Wang, 2003) suggests modulation of N2 by both perceptual matching and response conflict. In a modified Sternberg task, three simple shapes were presented as a memory set, followed by three probe items, each of which required a choice response of "match" or "mismatch." A probe item could (1) match a memory set item in both shape and sequential position (i.e., a circle presented as the second probe would match a circle presented second in the memory set), (2) be a shape not included in the memory set (complete mismatch), or (3) have occurred in the memory set but not in the same sequential position (partial match, calling for a negative response). Conditions (2) and (3) both elicited more negative ERPs than the matching condition in the 200–300-ms latency range, but this match/mismatch effect is hard to interpret due to the presence of a large P3 for matching items, which were associated with a lower probability response (33% vs. 67%). Of greater interest is the observation of a larger N2 and longer RTs in the partial match than complete mismatch condition, although both called for the same response. The partial match condition is one that could be expected to elicit more response conflict. This result appears to be discrepant with Wang et al. (2004), in which partial matches yielded an N2 that was intermediate between complete matches and complete mismatches, as one would expect from a template-matching process. However, a critical difference between the experiments is whether or not a partial match occurred in a dimension that was irrelevant (Wang et al., 2004) or relevant (Zhang et al., 2003) to the assigned task. The differing results may suggest that N2 amplitude can be driven more by template matching or more by response conflict, depending on how stimulus features are mapped onto decision or response requirements.

A study by Gehring, Gratton, Coles, and Donchin (1992) that combined a flanker task with a precue was consistent with the conclusion that N2 amplitudes reflect both template mismatch and response conflict. In the flanker task (Eriksen & Eriksen, 1974), participants respond with a left or right button press to the central letter of a five-letter display; flanking letters can be the same as the central letter or associated with the opposite response (e.g., if a central *H* signals a left-hand response and *S* a right-hand response, then *HHHHH* is a *compatible* array and *SSHSS* an *incompatible* array for a left-hand response). Multiple ERP studies have used this paradigm and are reviewed in more detail under "The anterior N2 and cognitive control." Briefly, the standard result is that incompatible arrays elicit slower responses, a larger frontocentral N2, a delayed P3b, and signs of incorrect motor preparation as compared to compatible arrays. Gehring et al. included an *H* or *S* letter cue prior to the array; in different blocks, the cue was the same as the central letter of the subsequent array on 80% of the trials, the opposite of the central letter on 80% of the trials, or had no predictive value. The authors employed a "vector filter" to analyze ERPs elicited by the arrays, which gave greater emphasis to either frontal or central scalp sites. When frontal sites were more heavily weighted, the N2 was sensitive to the compatibility manipulation—larger for incompatible arrays that invoked a response conflict—and relatively unaffected by the nature of the preceding cue. When central sites were more heavily weighted, N2 amplitude largely reflected the cue/array relationship. In the "80% same" block, N2 showed an amplitude gradient that followed the degree of perceptual mismatch between cue and target array. That is, after a cue of *H*, N2 amplitudes followed a gradient of *SSSSS* > *SSHSS* > *HHSHH* > *HHHHH*. This pattern of results provides a strong hint that, although anterior N2s reflect both perceptual matching and response conflict, it may be possible to decompose what appears to be a unitary physiological response into frontally maximal and centrally maximal components with differing functional attributes. Unfortunately, the apparent central maximum of the perceptual match effect in Gehring et al. is not in close agreement with the Sternberg paradigms indicating a frontal maximum for the component that differentiates positive and negative probe trials (Kotchoubey et al., 1996; Kramer et al., 1991).

Further details of the Gehring et al. (1992) study are relevant for an understanding of what we propose as the "template mismatch" aspect of the anterior N2. Comparison of the "80% different" and "80% same" blocks may tease apart the predictive value of the cues from their pure perceptual overlap with the target arrays. If passively perceived mismatch were the sole determinant of frontal N2 amplitude, one would expect the 80% different block to be identical to the 80% same block: *SSSSS* preceded by *H* would elicit the largest N2, although participants were aware that *H* tended to predict *S*. In contrast, if N2 amplitude were driven by internally generated expectations, one would expect the results of the 80% different block to be a perfect reversal of the 80% same block. The observed results showed a mild reversal, too weak to be statistically significant, but clearly in the opposite direction of the 80% same block. This outcome indicates that although internally generated expectations may not always be strong enough to override a simple perceptual matching process, the anterior N2 cannot be considered an index of a completely passive visual comparator either.

Finally, we note some apparent exceptions to our suggestion that the anterior N2 indexes deviation from an actively maintained visual template, coming from ERP studies of change blindness (Eimer & Mazza, 2005; Koivisto & Revonsuo, 2003). In one study, Eimer and Mazza (2005) had participants view sequentially presented pairs of arrays separated by 500 ms. Each array consisted of four black-and-white photographs of faces, arranged in a cross left, right, above, and below fixation. In the first experiment, participants indicated whether or not one of the faces on the left or right had changed. Relative to undetected change trials, detected change trials elicited a posterior N2 enhancement contralateral to the detected change, but there was no sign of an anterior N2, seeming to contradict the results of Wang and colleagues discussed above. We suspect that the key difference was the difficulty of this task in which the faces were

highly confusable and presented off fixation. The fact that participants detected only 71% of changes suggests that the task was challenging even though attention was limited to the left and right faces, in contrast to, for instance Wang et al. (2003) and Wang et al. (2004), in which accuracy exceeded 95%. This study therefore suggests that there may be a threshold for degree of mismatch below which the anterior N2 is not triggered.

A second study, conducted by Koivisto and Revonsuo (2003), is more difficult to integrate into our framework. Participants viewed sequentially presented pairs of arrays containing eight oriented lines arranged in a roughly circular pattern around fixation (eight-item condition) in order to detect whether one of the elements changed orientation. As in the Eimer and Mazza (2005) study, participants detected only 71% of the changes in this condition, and detected changes again elicited only a posterior N2 relative to no-change and undetected-change trials. More bothersome was a one-item control condition in which arrays contained only a single oriented line, also presented off fixation, and participants judged simply whether orientation changed between the first and second stimulus of the pair. Predictably, accuracy was quite high in this condition (98%), but there was still no sign of an anterior N2 to detected changes. This appears to be a fairly direct failure to replicate several of the sequential matching tasks discussed above.

Conclusions from Visual Novelty and Sequential Matching Paradigms

Nonmatching stimuli in sequential matching tasks and in cue-target sequences elicit larger frontocentral N2s as compared to matching stimuli, and this anterior N2 appears similar to that elicited by complex unusual stimuli in visual novelty paradigms. Mere deviation from preceding stimuli appears to be insufficient to elicit an anterior visual N2 (or it would routinely appear for rare targets differing from more frequent standards). The novelty paradigms indicate that if deviant stimuli serve as nontargets and do not require participants to override a prepotent response, they must be very unusual (typically unique) as compared to both their experimental context and long-term experience to elicit a substantial anterior N2. Large frontocentral N2s are elicited in two other sorts of circumstances. One is when the second stimulus of a two-stimulus sequence is discrepant from the initial stimulus and the assigned task encourages or demands perceptual matching of the two, as in sequential matching tasks, Sternberg tasks, and the modified version of the Eriksen flanker task using an informative precue. The second is when the eliciting stimuli are either targets or cues for an upcoming target but comprise a perceptually variable set of items that must be individually identified and then classified as belonging to a larger set before their target status or signal value can be appreciated (Breton et al., 1988).

The shared property of these diverse results appears to be actively attended mismatch between a stimulus and a mental template. The most straightforward case is provided by the sequential matching paradigms, in which (a) mismatch detection is central to the task and (b) the N2 effect is eliminated when mismatch becomes task irrelevant. Also reasonably intuitive is the novelty effect, which suggests that mismatch between a stimulus and long-term memory can “grab” attention as long as the magnitude of mismatch is considerable. This leaves Breton et al.'s (1988) observation that diverse (but simple) targets elicit larger anterior N2s than equally rare but homogeneous targets. We suspect that the key factor is in the strategy encouraged by the two types of oddball. When the relevant oddball is a single simple stimulus, the most natural strategy is to hold the target in mind and search for matches. When the relevant oddball category has too many exemplars to hold in working memory but the *standard* is a single stimulus, the most natural strategy is to hold the standard in mind and search for mismatches, which will drive an anterior N2.

Our emphasis on “mismatch” as a precondition for the elicitation of an anterior N2 in the visual modality may suggest some similarity to the auditory mismatch negativity, but the anterior

visual N2 differs from the auditory MMN in several functional properties. First, there has been no suggestion that anterior N2 responses to visual stimuli appear in the absence of focal attention to the eliciting stimuli, whereas the MMN can be elicited by acoustic deviants as participants read a book and ignore auditory events. Second, as described above, the sensitivity of the visual N2 to perceptual mismatch is strongly modulated by the task relevance of perceptual changes across sequential stimuli. The dissociation between the anterior visual N2 and preattentive change detection is evidenced by studies of unattended visual deviance aimed at eliciting the visual analogue of the mismatch negativity (*vMMN*). These studies typically require participants (a) to perform a demanding visual task at fixation while irrelevant visual oddball events are presented in the near periphery or (b) to detect auditory oddballs while irrelevant visual oddballs are presented at fixation. Unattended visual deviants elicit early and posterior negative effects that often end at or before 200 ms, prior to the N2 epoch. Some have argued that this effect might be closely related to the visual N180 component and reflects a "release from refractoriness" when a visual stimulus with new physical properties stimulates a new pool of visual neurons as compared to repetitive identical stimuli (Kenemans, Jong, & Verbaten, 2003; but see Czigler et al., 2006; for a review, see Pazo-Alvarez, Cadaveira, & Amenedo, 2003). In any case, it is clear that, when preconditions for the MMN are replicated in the visual modality, an anterior N2 is never the result.

We have argued in this section that the anterior N2 is driven by attended mismatch from a visual template and that this effect is not an epiphenomenon of cognitive control processes. Equally clear, however, is the sensitivity of an anterior N2 to cognitive control. We review the evidence for this claim below, arguing that the control N2 constitutes a separate component.

An Anterior N2 Is Sensitive to Cognitive Control

We argued above that an anterior N2 component was modulated by attended mismatch between a stimulus and a mental template and that the majority of observed template-mismatch effects could not be described in terms of response inhibition, detection of response conflict, or strategic performance monitoring—a collection of processes broadly termed "cognitive control." In this section, we describe conditions explicitly designed to vary in their requirements for cognitive control, which also result in modulation of an anterior N2, and discuss their relationship (or, mostly, lack of relationship) to perceptual template matching.

Experimental paradigms commonly used in the study of response inhibition and/or response conflict include go/no-go tasks, the Eriksen flanker task, and the stop-signal paradigm, each reviewed below. These are contrasted to a paradigm that appears to include similar cognitive elements ("negative patterning") but that leads to little if any modulation of an anterior N2. Paradigms designed to evaluate response-monitoring and/or the adjustment of strategies are those in which participants are expected to make errors and to notice them or those which include feedback about performance. After review of error and feedback studies, we discuss the relationship among the variety of N2-like responses elicited in paradigms that tax cognitive control and N2 responses that appear to be triggered by mismatch to a perceptual template.

The No-Go N2 and Cognitive Control

Several findings from go/no-go studies support the hypothesis that the no-go N2 is driven by inhibition of a planned response. First, even though larger N2s are elicited by no-go than go trials in silent counting tasks, it tends to be somewhat larger in tasks for which an overt response must be withheld (Bruin & Wijers, 2002; Pfefferbaum et al., 1985). Second, the no-go N2 is increased by pressure to respond quickly (Jodo & Kayama, 1992). This sensitivity to speed instructions may bear on the difference between tasks with overt motor responses and covert responses like silent counting as it is more difficult to mandate speed for a covert response. Third, the no-go N2 is larger in participants with low than high false alarm rates, suggesting

an association between amplitude and successful response inhibition (Falkenstein, Hoormann, & Hohnsbein, 1999).

Finally, there is some evidence that the no-go N2 is larger when no-go stimuli share features with target stimuli and thus trigger preparation of an incorrect response that must be suppressed. Azizian, Freitas, Parvaz, and Squires (2006) compared ERPs elicited by a rare target stimulus (10%), a frequent nontarget standard with a very different shape (60%), a rare nontarget with a similar shape (10%), and two other nontargets with intermediate similarity (10% each). The similar nontarget elicited a much larger frontocentral N2 than any of the other nontarget conditions. Other studies that include attentional manipulations have also observed centrally and sometimes frontocentrally distributed N2 enhancements to nontargets that share features with rare targets, sometimes locating dipole sources in medial frontal cortex, consistent with source localization of the no-go N2 in more standard paradigms (Kenemans et al., 2002; Lange et al., 1998; Nieuwenhuis et al., 2003). These observations of larger N2s for nontargets that resemble targets are the opposite of what might be expected for a component sensitive to perceptual mismatch alone and thus provide strong evidence that the N2 also indexes cognitive control.

The evidence that difficult no-go trials (those in which the no-go stimuli resemble the go stimuli) elicit larger N2s than easy no-go trials is, however, not perfectly uniform. In a two-stimulus go/no-go task (25% probability nontargets), Nieuwenhuis, Yeung, and Cohen (2004) presented either the visually distinct but acoustically similar letters *F* and *S* or the visually similar but acoustically distinct letters *F* and *T* in separate visual and auditory blocks. The frontocentral N2 to auditory no-go stimuli was larger during the difficult (*F* vs. *S*) than easy discrimination (*F* vs. *T*), which did not elicit a significant no-go N2. There was, however, no difference between easy and difficult visual discrimination, both of which elicited large no-go N2s. The authors' interpretation was that discriminating even the easy visual stimuli posed sufficient difficulty such that participants prepared a response on the no-go trials. An experiment from a different laboratory (Senkowski & Herrmann, 2002) used three colored circles, one light green, one dark green, and one red, each with a probability of 33%. The dark green circle and one of the other circles were always no-go stimuli, and the third circle was the rare target. The N2 to the dark green circle was measured when the light circle was the target (difficult discrimination) and when the red circle was the target (easy discrimination). Although the N2 to the dark green circle was enhanced during difficult discrimination, the scalp distribution was clearly posterior rather than frontocentral, suggesting an attentional rather than control-related enhancement.

Perhaps more consistently effective than manipulating the discriminability or presence of target features on no-go trials is the manipulation of the *location* of target features. This is the strategy employed in the Eriksen flanker task and its variants, which we now review.

The Eriksen Flanker Task

In addition to variants of the two-stimulus oddball task, several tasks have been designed specifically to investigate the influence of cognitive control. One such task is the Eriksen task (Eriksen & Eriksen, 1974), in which participants perform a choice reaction time task on the central letter of a letter string. Typically, the central letter is flanked on either side by either identical letters (e.g., *HHHHH*, *congruent noise* condition), or letters indicating the opposite response (e.g., *SSHSS*, *incongruent noise* condition). Relative to congruent flankers, incongruent flankers elicit delayed reaction times, ERP correlates of initially incorrect motor preparation (*lateralized readiness potentials* or *LRPs*) that resolve before the actual response, an enhanced frontocentral N2, and a delayed parietal P3 (Bartholow et al., 2005; Coles, Gratton, Bashore, Eriksen, & Donchin, 1985; Gehring et al., 1992; Gratton, Coles, Sirevaag, Eriksen,

& Donchin, 1988; Heil, Osman, Wiegelmann, Rolke, & Hennighausen, 2000; Kopp, Rist, & Mattler, 1996; Yeung et al., 2004).

Several findings demonstrate that the N2 effects in the flanker task are not elicited by template mismatch but instead by the need to control incorrect response preparation. First, the probability of the response categories and of the congruent and incongruent flankers is usually 50%, eliminating the possibility that the frontal N2 is driven by mismatch with a template developed for frequent stimuli. Two studies have investigated the interaction between expectation and flanker compatibility. A study by Gehring et al. (1992), already described above, crossed congruent and incongruent flankers with precues that validly or invalidly predicted the correct response. Whereas the N2 at central electrodes was sensitive to the extent of mismatch between the cue and the overall array, the N2 at frontal electrodes was sensitive to noise congruity and relatively insensitive to cue validity. More difficult to interpret is a study by Bartholow et al. (2005) that manipulated not probability of response category but the probability of incongruent flankers. The scalp distribution of the N2 to incongruent flankers was more frontal when incongruency was common relative to uncommon, perhaps suggesting that cognitive control was elicited more readily when it was more frequently necessary. In any case the N2 at Cz was significant in both expectation conditions, showing that it was not contingent on the predictability of congruent trials. One might argue that the N2 in the flanker paradigm is driven by mismatch between the relevant central element and the incongruent flankers, but this explanation is eliminated by studies manipulating the response values of mismatching flanker elements. These show that the N2 is larger when mismatching flankers are associated with the opposite response to the central element rather than with the same response or with a no-go response (Heil et al., 2000; van Veen & Carter, 2002).

Finally, a recent computational model of conflict monitoring simulated changes in timing and amplitude of the N2 in the flanker task based on the predicted time course of response conflict (Yeung et al., 2004). The model predicted that the N2 would peak prior to the response on correct trials, after the response on incorrect trials, and be larger to incongruent than congruent trials. Response-locked ERPs confirmed these predictions and further supported the hypothesis that the control N2 is time-locked to the response rather than the stimulus (Nieuwenhuis et al., 2003) by showing that the peak latency of the N2 positively correlated with reaction time. Also predicted by the model was the observation that the *amplitude* of the N2 was positively correlated with reaction time, consistent with the intuitive idea that trials with more conflict elicit longer reaction times. In fact, much but not all of the N2 amplitude difference between congruent and incongruent trials was accounted for by the fact that congruent trials are associated with faster RTs than incongruent trials. Finally, dipole sources for both the N2 to correct and incorrect responses (*error related negativity*; see below) were localized to medial frontal cortex (see also van Veen & Carter, 2002). Hemodynamic imaging studies demonstrate a role for midline frontal cortical areas in mediating response conflict in incompatible flanker trials, similar to that observed for no-go trials (for a review, see Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). This similarity indicates that complete inhibition of a response and selection of an alternate response engage the same brain region (but for suggestions that a region of lateral frontal cortex—the inferior frontal gyrus—may also be involved in response inhibition, see also Aron, Fletcher, Bullmore, Sahakian, & Robbins, 2003; Aron & Poldrack, 2006; Aron, Robbins, & Poldrack, 2004).

In summary, incompatible stimuli in the flanker task elicit a central or frontocentral N2 that is relatively insensitive to probability and is difficult to account for within a mismatch-based theory. However, we will ultimately conclude that this “control N2” is frequently mixed with an N2 that is driven by perceptual mismatch.

The Stop Signal Paradigm

In stop signal tasks, participants perform a visual choice reaction time task to an initial stimulus (S1) that is sometimes followed by a "stop" stimulus that signals the need to withhold the response (e.g., De Jong, Coles, Logan, & Gratton, 1990; Logan, Cowan, & Davis, 1984). Frontocentral N2s are elicited by stop signals and, like the N2 for incongruent trials in the flanker paradigm, are larger when instructions emphasize the importance of fast responses on go trials than when instructions request a balance between speed and accuracy (Band, Ridderinkhof, & van der Molen, 2003).

Stop signals are presented with variable delays after the initial stimulus, resulting in more successful stops at short delays and more incorrect responses when the delays are longer and the prepared response can no longer be inhibited. It is thus possible to combine the distribution of reaction times to trials with no stop-signal and the distribution of latencies of stop signals eliciting unsuccessful stops to calculate the *stop signal reaction time*, the amount of time it takes to inhibit a response. There is some evidence that the N2 elicited by the stop signal is larger in participants with fast stop-signal RT (i.e., efficient inhibitors) than those with slow stop-signal RT (van Boxtel, van der Molen, Jennings, & Brunia, 2001). Another study reported that the stop-signal N2 was reduced over right inferior frontal electrodes in children with attention-deficit-hyperactivity disorder (ADHD) relative to normal controls. This was true for both successful and unsuccessful stop trials. Over these same electrodes, N2 amplitude was correlated with percentage of successful inhibitions in both normal and ADHD children. N2 amplitude was also correlated with degree to which short-delay stop signals improved inhibition success, a measure related to stop-signal RT (Pliszka, Liotti, & Woldorff, 2000; see also Schmajuk, Liotti, Busse, & Woldorff, 2006).

Two further facts are revealed by direct comparison between successful and unsuccessful stop trials. First, the latency of the N2 is longer in unsuccessful than successful trials (Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Ramautar, Kok, & Ridderinkhof, 2004, 2006). Second, N2 amplitude is consistently larger for trials with unsuccessful than successful inhibition (van Boxtel et al., 2001) and this difference has usually been observed to have a centroparietal or posterior scalp distribution (Kok et al., 2004; Pliszka et al., 2000; Ramautar et al., 2004; but see Ramautar et al., 2006). This last result has led some to suggest that the stop-signal N2 does not reflect response inhibition, but instead evaluation of the stop signal (Ramautar et al., 2004, 2006). More specifically, it was hypothesized that, on unsuccessful trials, participants are already aware at the time of the stop signal that it will be ineffective, increasing the "meaningfulness" of the stimulus and amplitude of the N2. In our framework, one might think of the stop signal on these trials as taking on characteristics of a target with high signal value, resulting in increased attention and a posterior N2. An alternative interpretation, suggested by a reviewer of the present article, is that the N2 on unsuccessful trials is comprised of an N2 plus an error-related negativity elicited by the realization of an impending mistake (negativities elicited during erroneous trials are reviewed below under "Anterior negative ERPs elicited by errors and negative feedback"). This interpretation would suggest somewhat different scalp distributions for negativities related to response inhibition versus error detection, one facet of ongoing disputes about the relationship between N2s related to cognitive control and the negative component observed just after an erroneous response (taken up again below).

Overall, the stop-signal N2 may contain multiple subcomponents, one anterior and related to inhibition or control and one posterior related to evaluation of the stop signal. Future studies should attempt to separately manipulate these subcomponents.

The Stroop Task

The Stroop task is very commonly used in the study of cognitive control and the prefrontal cortex. Briefly, participants view color words printed in ink of the same color as that referred to by the word (congruent condition) or a different color (incongruent condition) and say the color of the ink. Reaction times are slower in the incongruent condition than in the congruent condition, and patients with damage to the frontal lobes make an increased number of errors to congruent Stroop stimuli, frequently producing the written word rather than the ink color (Lezak, 1995).

In the most typical adaptation of the Stroop task to ERP recording, participants use four buttons to indicate the color of letters on a computer screen, ignoring as usual the actual color word spelled by the letters. Compared to congruent stimuli, incongruent stimuli often elicit a negative deflection peaking at around 450 ms and overlapping a centrally distributed slow negative potential. In some cases, N450 manifests as a negative peak, clearly distinct from and later than the central N2, maximal at frontocentral electrodes but extending to parietal electrodes, where it appears as a negative deflection of the P3 (Liotti, Woldorff, Perez, & Mayberg, 2000; West & Alain, 2000). In other studies, the incongruity effect appears as an extension of the central N2 in time and is more difficult to distinguish from the central negative slow wave (West & Alain, 1999, 2000). Two other studies failed to observe anything resembling an N450, but these studies were primarily interested in the P3 component and applied conservative filters ideal for studying the P300 (Duncan-Johnson & Kopell, 1981; Ilan & Polich, 1999). We note, however, these were the only two Stroop studies that did not use a reference computed over all scalp sites, as compared to the majority of N2 studies covered here using mastoid, earlobe, or nosetip references.

One study replicated the N450 in three different task conditions: overt vocal color naming, covert vocal color naming, and the usual manual response version (Liotti et al., 2000). The N450 effect was largest and most frontal in the overt naming task, somewhat smaller in the covert naming task, and smallest and most posterior in the manual task, possibly accounting for the somewhat inconsistent scalp distribution across manual studies. This study also localized a dipole source for the N450 in the anterior cingluate, consistent with results from neuroimaging studies (e.g. Cabeza & Nyberg, 2000; Leung, Skudlarski, Gatenby, Peterson, & Gore, 2000; Milham et al., 2001; van Veen & Carter, 2005).

Also of note was a study that examined the impact of the proportion of incongruent trials on the N450 incongruity effect (West & Alain, 2000). When incongruent trials were rare, the effect was larger than when they were frequent, but only for trials with fast reaction times. Note that this is somewhat inconsistent with the findings of Bartholow et al. (2005), who found that incongruent flankers enhanced the N2 at Fz only when trials were mostly *incongruent*.

Although the N450 has a later onset and peak than the N2, it has a similar central scalp distribution and is similarly sensitive to manipulations of cognitive control. On the other hand, the current data suggest that increasing the proportion of incongruent trials increases the N2 to incongruent flankers but decreases the N450 to incongruent Stroop stimuli. Because the effects in both of these studies were subtle, being only significant at Fz in the Bartholow et al. (2005) and significant only for fast trials in West & Alain (2000), this puzzling inconsistency should be treated with caution pending replication.

Negative Patterning: A Limiting Case for the Control N2?

We have reviewed several control-related N2 and N2-like results related to action monitoring and apparently generated by similar sources in medial frontal cortex. We now note a study

where stimuli with apparently conflicting information did not elicit an enhanced N2, suggesting a limit on the generality of this phenomenon.

Fox, Michie, Wynne, and Maybery (2000) employed a negative patterning paradigm (Spence, 1952) to study the effect of configural and elemental no-go stimuli on the no-go N2. In this paradigm, participants respond to two stimuli, labeled "A+" and "B+", but must not respond to a combination of those two stimuli, labeled "AB−" or, in this case, *configural no-go.* Given the studies reviewed so far, one might consider the configural no-go stimulus ideal for eliciting a frontocentral N2. Not only is it a no-go stimulus, but it is composed of elements that individually indicate a go response, introducing conflicting information. Furthermore, the no-go trials were rare (25%) in Fox et al.'s experiment, and we have seen that low-probability no-go trials are particularly potent in eliciting anterior N2s (Bruin & Wijers, 2002; Nieuwenhuis et al., 2003).

The details of the study were as follows. The A+ and B+ were not in this case individual stimuli, but rather stimulus features, A+ being the color blue and B+ being the letter *O*. A+ stimuli were the letters *N*, *J*, and *W*, all colored blue and the B+ stimuli were the letter *O* colored either green, red, or yellow. The AB− (configural no-go) stimulus was a blue *O*. The design further included a CD−, or *elemental no-go* stimulus, that had a unique color and letter, in this case a pink *E*. Each individual stimulus constituted 12.5% of the trials, so that, together, the two types of no-go stimuli (elemental and configural) constituted 25% of the trials. Contrary to prediction, the N2 to configural no-go stimuli was only slightly larger than the N2 to go stimuli and did not reach the authors' criterion of significance. The elemental no-go stimulus, on the other hand, elicited a robust N2 that, interestingly, peaked later than the configural no-go N2. This even though accuracy to the configural no-go stimuli was slightly (but not significantly) worse than the accuracy to the elemental no-go stimuli.

Why was the configural no-go N2 so small? One possibility is that it represents the effect of cognitive control in direct opposition to the effect of perceptual mismatch. Even though all individual stimuli in this experiment were equally probable, the stimulus features were not. In fact, 50% of the stimuli were blue and 50% were the letter *O*, making blue and *O* the most frequent features. Because the configural no-go stimulus was unique but similar to most of the other stimuli, the most intuitive strategy would be to hold the blue *O* in mind and search for a *match*. The features of the elemental no-go stimulus, on the other hand, were unique and very rare (12.5% each), possibly resulting in a mismatch effect summating with the effect of cognitive control.

The results of Fox et al.'s (2000) study emphasize the importance of considering the influence of mismatch on the anterior N2 when designing experiments to investigate cognitive control and further suggest that the presence of conflicting information is not always effective in eliciting this component, particularly during complex categorization rules.

Data from our own laboratory are generally consistent with this latter point. In one early study, we did observe an enhanced anterior N2-like component to stimuli that offered conflicting information in a complex categorization task. Briefly, participants used a disjunctive three-out-of-five rule to categorize artificial stimuli with five relevant dimensions. *Near boundary* stimuli had three features belonging to one category and two features belonging to another category whereas *Far boundary* stimuli had four features belonging to one category and only one feature belonging to the opposite category. Near boundary stimuli, which had more conflicting information than far boundary stimuli elicited an enhanced anterior N2 (Folstein & Van Petten, 2004). Our subsequent experiments, however, have failed to elicit any N2 enhancement to stimuli sharing features with multiple categories, whereas novel features have been highly effective in eliciting the anterior N2. In one experiment, a conjunction of two

features indicated Category A, a conjunction of two different features indicated Category B, and anything else indicated Category C. Category C included stimuli with one Category A feature and one Category B feature (*conflicting condition*) as well as stimuli with unfamiliar features not associated with either Category A or B (*novel condition*). The conflicting Category C stimuli elicited an N2 with the same amplitude as the familiar Category A and B stimuli whereas the novel Category C stimuli elicited a large enhanced anterior N2 (Folstein, Van Petten, & Rose, 2007). In a second experiment, participants categorized stimuli using a two-out-of-three rule very similar to the three-out-of-five rule employed in the initial experiment. Again, near boundary stimuli did not elicit a larger anterior N2 than far boundary stimuli, but a third category in which the stimuli had novel features did elicit an enhanced anterior N2, again emphasizing the effectiveness of novelty in driving the anterior N2 (Folstein, 2007; Folstein, Van Petten, & Wong, 2007).

Anterior Negative ERPs Elicited by Errors and Negative Feedback: The Error-Related Negativity and the Feedback-Related Negativity

Anterior negative components are also elicited by participants' errors and by negative feedback about task performance. These components have been variously called the *error related negativity* (*ERN*), the *feedback ERN*, the *feedback-related negativity* (*FRN*), and the *medial frontal negativity*. For clarity, we will use the term ERN for the response-locked component and FRN for the component time-locked to a feedback stimulus.

The response-locked ERN is a frontocentral negative wave peaking around 50–60ms after a response that is larger for errors than correct responses (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, & Meyer, 1993). Some studies suggest that the ERN reflects adjustment of both short- and long-term response strategies after a mistake. High ERN amplitude predicts a higher probability of a compensatory second response and a slower response on the subsequent trial, linking this component with short-term strategic monitoring (Gehring et al., 1993). Frank, Woroch, and Curran (2005) showed that participants who made use of negative feedback to associate symbols with probabilistic rewards had larger ERNs than participants who made more use of positive feedback, linking the ERN with longer term strategic processes as well. ERN amplitude increases with the significance of errors, as when accuracy is emphasized over speed (Gehring et al., 1993), participants believe that their performance is being evaluated by an observer, and when correct trials have high monetary value (Hajcak, Moser, Yeung, & Simons, 2005). These results suggest that the ERN is specifically related to the detection of errors rather than to the detection of a rare event.

The FRN is a frontocentral negative wave peaking around 250 ms after stimulus onset that is larger to negative than positive feedback, regardless of sensory modality of the feedback signal, and is also larger to negative than positive outcomes in gambling tasks (Badgaiyan & Posner, 1998; Gehring & Willoughby, 2002, 2004; Hajcak, Holroyd, Moser, & Simons, 2005; Holroyd & Coles, 2002; Miltner, Braun, & Coles, 1997; Yeung, Holroyd, & Cohen, 2005). In gambling tasks, the FRN does not appear to be sensitive to the magnitude of monetary loss, but only to whether an outcome is positive or negative (Gehring & Willoughby, 2004; Hajcak, Moser, Holroyd, & Simons, 2006; Holroyd, Hajcak, & Larsen, 2006; Yeung & Sanfey, 2004). An interesting aspect of monetary feedback stimuli is that a given reward magnitude can indicate an erroneous or correct choice, depending on the alternative outcome (e.g., a decision resulting in a loss of 25¢ could be correct if the alternative was losing 50¢). A recent study showed that the FRN can be selectively sensitive to negative monetary outcomes or to performance errors, depending on which is emphasized by the feedback stimulus (Nieuwenhuis, Yeung, Holroyd, Schurger, & Cohen, 2004).

Response Conflict, Error Detection, Poor Outcomes, and the Medial Frontal Cortex

The evidence generally suggests that both ERN and FRN are generated in medial frontal cortex, although the data are stronger for the ERN than for the FRN. One meta-analysis of functional magnetic imaging (fMRI) studies, including 13 studies of response error and 5 studies of negative feedback, found that all reported activation in medial prefrontal cortex, including Brodmann's areas 24 and 32 of the anterior cingulate (ACC), but also activations in motor areas 6 and 8 (Ridderinkhof et al., 2004; see also Gehring & Willoughby, 2004; Taylor et al., 2006). Comparisons of ERP and hemodynamic data collected in similar paradigms can suggest candidate structures that are critical for an ERP component, but the low temporal resolution of the fMRI response does not allow isolation of activity that corresponds to one ERP component from other brain regions that might be coactive during an experimental condition. However, other sorts of data offer converging support for the idea that the ERN, at least, depends on medial frontal cortex. Patients with Obsessive Compulsive Disorder, who are thought to have overactive anterior cingulates, have larger ERNs than normal controls (Fitzgerald et al., 2005; Gehring, Himle, & Nisenson, 2000; see also Hajcak & Simons, 2002; but see Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005). Conversely, patients with damage to medial prefrontal cortex, including a patient with a focal ACC lesion, have been observed to have attenuated ERNs (Stemmer, Segalowitz, Witzke, & Schönle, 2004; Swick & Turken, 2002; see also Gehring & Knight, 2000; Ullsperger & von Cramon, 2006; Ullsperger, von Cramon, & Müller, 2002). Source localization methods applied to both ERP and MEG data have shown that dipole sources in medial frontal cortex adequately account for both ERN and FRN (Gehring & Willoughby, 2002; Gehring et al., 2000; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Miltner et al., 1997, 2003; Nieuwenhuis et al., 2003; Ruchsow, Grothe, Spitzer, & Kiefer, 2002; van Veen & Carter, 2002; Yeung et al., 2004). Finally, Debener, Ullsperger, et al. (2005) used simultaneous recording of EEG and fMRI to demonstrate that the amplitude of the ERN from trial to trial is correlated with the BOLD signal from an area in the ACC.

In contrast to the strong agreement that the ERN has a source in the anterior cingulate, considerable doubt has recently been cast on an ACC source for the negative-feedback effect in the time estimation task used by Miltner et al. (1997; Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005; van Veen, Holroyd, Cohen, Stenger, & Carter, 2004). Gehring and Willoughby (2004) have argued that the FRN to monetary losses has contributing sources that are more anterior than the ERN elicited by errors in the flanker task (see also Taylor et al., 2006). Therefore the exact relationships among the ERN, FRN, and particular regions of medial frontal cortex remain an active area of study.

Also active is the question of whether the ERN (van Veen et al., 2004) indexes the detection of errors/negative outcomes or the detection of response conflict, as proposed for the anterior N2. One model of conflict monitoring, for instance, was able to account for the timing of the N2 and ERN effects elicited by incongruent flanker stimuli by assuming that both had a medial frontal dipole source (Yeung et al., 2004). Also, the ERN is larger when the error response is motorically similar to the correct response as compared to dissimilar errors, further supporting a conflict interpretation (Gehring & Fencsik, 2001). Another study, however, found that a patient with an anterior cingulate lesion had a reduced ERN but an *enhanced* N450 effect to incongruent stimuli in a Stroop-like task, supporting a dissociation between error and conflict detection (Swick & Turken, 2002; for a dissociation due to administration of alcohol, see also Ridderinkhof et al., 2002). The relationship between response conflict and error detection remains a contentious topic; Yeung and Cohen (2006) suggest that their computational model can account for dissociations between the ERN and control-N2 while maintaining a central role for the anterior cingulate in both ERPs.

A particularly influential alternative to the conflict hypothesis was proposed by Holroyd and Coles (2002), who hypothesized that both the ERN and FRN are generated by a dopaminergic

signal to the anterior cingulate that a given outcome was worse than expected. They predicted that the relative amplitudes of the ERN and the FRN would change depending on the validity of feedback stimuli. Specifically, when negative feedback is valid, there should be a large ERN following an erroneous response because realization of one's own mistake is a disappointment, but the subsequent FRN elicited by the feedback stimulus should be small because that information is consistent with expectation. When feedback is invalid, the FRN should be larger than the ERN, because error feedback would be less expected. This is exactly what Holroyd and Coles (2002) observed in an experiment that compared blocks of completely valid feedback to blocks with inconsistently valid feedback (50% accurate feedback, 50% inaccurate) mappings between stimulus and response. Also as predicted, the ERN to errors increased in amplitude over the course of learning as errors became less expected. Also as predicted, the feedback ERN in the 50% validity condition remained constant throughout the experiment. The authors argued that the ERN and FRN were generated by a single source in anterior cingulate cortex that is sensitive to worse-than-expected outcomes (for further review, see also Nieuwenhuis, Holroyd, Mol, & Coles, 2004).

Holroyd and Coles' (2002) unification theory of the ERN and FRN is, of course, a kind of mismatch detection hypothesis, except that that (a) the mismatch is between an expectation and an event, rather than between external events alone, and (b) the detected mismatch must be negatively valenced. It may be possible to relate this hypothesis to our description of the N2 as indexing attended mismatch with a perceptual template if we assume that participants have a bias toward expecting good outcomes in paradigms in which stimuli carry a valence. Although speculative, this idea is suggested by studies in which participants predict gambling outcomes from trial to trial and are shown to be biased toward expecting positive outcomes (for a review, see Krizan & Windschitl, 2007). Studies exploring the relationship between the mismatch/novelty N2 and either the ERN or FRN are very few in number, but below we describe two sources of extant data: first, ERP experiments that manipulate both outcome valence and perceptual mismatch and then hemodynamic imaging experiments that may shed light on whether or not perceptual novelty effects and “cognitive control” effects share a common neural source.

Impact of Outcome Probability and Perceptual Mismatch on the Feedback-Related Negativity

A handful of recent studies have examined the relationships among outcome valence, outcome probability, and stimulus mismatch as determinants of FRN amplitude. In reviewing these studies, our primary goal is not to evaluate the relative merits of the conflict and outcome detection hypotheses, but to consider the relationship between the FRN and template-mismatch N2.

In two studies with overlapping authors, an array with four elements appeared on the screen and participants chose one of the elements, resulting either in a monetary reward or a lack of reward, as signaled by subsequent feedback stimulus. Each participant viewed a list in which negative feedback was frequent and a list in which negative feedback was rare so that feedback valence and probability were fully crossed. In comparisons of positive to negative feedback within each probability level, Holroyd, Nieuwenhuis, Yeung, and Cohen (2003) found that the difference between positive and negative feedback was larger for low probability (25% positive vs. 25% negative) outcomes than high probability (75% positive vs. 75% negative) outcomes. However, in two experiments, Hajcak, Holroyd, et al. (2005) found the standard valence effect (larger FRN for negative than positive feedback), but no interaction between valence and probability. Although these experiments present mixed results, the evidence leans toward insensitivity of the FRN to standard probability manipulations. This is consistent with a bias to expect positive outcomes independent of the actual probability of those outcomes.

Very few experiments have combined the valence manipulations that are popular in the feedback-related-negativity literature with manipulations that we might expect to influence the anterior N2 that is sensitive to perceptual mismatch. Two that appear to do so used a “slot machine task” in which an array of three elements appears on the screen one element at a time at a rate of about one element per second (Donkers, Nieuwenhuis, & van Boxtel, 2005). In both studies, an XXX array signaled a monetary gain or loss indifferent blocks, whereas XXY and XYZ signaled no change in the participant's “account.” This design includes no response selection by the participants, eliminating any adjustments of strategy from consideration of the results (for evidence that negative monetary outcomes elicit FRNs even when participants make no choices, see also Yeung et al., 2005). The comparison of most interest is between the ERP elicited by the third element of the XXX and XXY arrays. Note that in the gain block, XXY indicates a relative loss (given that gain was possible), whereas in the loss block the same stimulus indicates a relative gain (given that loss was possible). In contrast to the results reviewed so far, ERPs were completely unaffected by outcome valence in the initial study (Donkers et al., 2005). However there was a clear and large effect of mismatch in that the final element of XXY arrays elicited a larger negative peak (250 ms poststimulus) than the final element of XXX arrays, regardless of relative loss or gain. The authors referred to the negative peak as a “mediofrontal negativity,” but the more appropriate label is surely N2.

In a second experiment, the authors attempted to eliminate the N2 contribution by crossing the XXX and XXY outcomes with three levels of probability: 37.5%, 25%, and 12.5% (Donkers & van Boxtel, 2005). The largest effect by far was again mismatch, with XXY eliciting a larger anterior negativity than XXX in both the loss and gain conditions. This basic effect of mismatch was modulated by the gain/loss manipulation, with larger amplitudes in the gain block and an effect of outcome probability only in this block. Because XXY specified a relative loss in the gain condition, the authors concluded that outcome valence was the driving factor for the frontal negativity. However, the overall results clearly differ from the standard FRN result of larger amplitudes for losses, as the stimuli signaling *absolute* losses (XXX in the loss block) did not elicit a larger negativity than those signaling absolute gains (XXX in the gain block), and indeed there was a slight trend in the opposite direction (not evaluated statistically, but likely to be insignificant). We suggest an alternate account of these data, namely, that participants were strongly and actively expecting stimulus completions that signaled some absolute financial outcome (e.g., XXX, as compared to XXY, which signaled no change in a participant's account), consistent with the much larger contingent negative variation observed after XX trial onsets than an XY trial onsets in both the gain and loss blocks. In this paradigm, the stimuli signaling an absolute outcome were also ones with three perceptually identical stimulus elements, so that our emphasis on the anterior N2's sensitivity to attended perceptual mismatch would predict much larger frontal negativities for XXY trials. It would be of some interest to try to dissociate the impact of perceptual match/mismatch from the motivational factor of anticipating a financial gain/loss by, for instance, having perceptually dissimilar trials (AAB) signal account changes and perceptually similar trials (AAA) signal no change.

It is clear that Donkers and van Boxtel (2005) also observed a modulation of a frontocentral negative component as a result of global context—larger negativities in the face of potential gain than potential loss—so that pure perceptual mismatch may be insufficient to fully account for the results. We have already noted that frontocentral N2s in the Eriksen flanker task are determined by both perceptual stimulus match/mismatch (larger N2 for *HHHHH* following a cue of *S*) and by participants' predictions (smaller mismatch effect when participants know that *HHHHH* frequently follows a cue of *S*; Gehring et al., 1992). Donkers and van Boxtel's results may indicate that the motivational status of detecting a match or mismatch has a similar modulatory influence.

Anterior Negativities, Cognitive Control, and Mismatch: Conclusions

Anterior negativities in the N2 latency range are elicited by several tasks related to response and strategic monitoring. Several results reviewed above demonstrate a genuine influence of cognitive control independent of mismatch detection. In the case of the no-go N2, the strongest evidence is provided by its sensitivity to speed pressure and the inverse relationship between N2 amplitude and false alarms. Also, the observation of a larger response-locked than stimulus-locked oddball N2 further supports a link between the N2 and response-related processes. In the flanker task, the influence of response monitoring is demonstrated by the greater N2 to response-incompatible flankers than flankers that are only perceptual mismatches. Finally, the sensitivity of the ERN to the significance of errors and its tendency to be larger when errors are more similar to correct responses suggest influences other than mismatch detection.

Although there is good evidence suggesting independence between the frontocentral N2 elicited by a variety of manipulations that tax cognitive control and the frontocentral N2 elicited by perceptual mismatch, we have also argued that the effect of mismatch has occasionally influenced the results of studies designed to evaluate cognitive control (as in Donkers & van Boxtel 2005; Donkers et al., 2005). In the case of negative patterning (Fox et al., 2000), the difference between configural and elemental no-go stimuli could be accounted for in terms of differences between match and mismatch strategies rather than the extent to which they elicited a no-go N2. We have also suggested that the modulation of the feedback-related negativity by outcome valence could be attributed to a “hopeful bias” during gambling tasks, such that poor outcomes represent mismatches between the feedback presented and that expected. Because others have also suggested a parallel between the control N2 and the anterior novelty/mismatch N2, we now turn to a discussion of this issue, including the sensitivity of the anterior cingulate to novelty and mismatch.

Neural Relationship between the Frontocentral Negativities Elicited by Cognitive Control and by Novelty/Mismatch

In the sections above, we have argued that there are at least two functional sources of variance for “N2” ERP components that are maximal over frontal or central scalp sites when elicited by visual stimuli. Experiments employing either unique novel stimuli or match-to-sample tasks have revealed an N2 component related to mismatch detection and deviance. Second, there is a family of control-related N2 components that, with the possible exception of the feedback-related negativity, are independent of mismatch detection.

Recently, some have suggested that the concept of “conflict” should extend beyond conflict between competing responses to include conflict between competing semantic representations or between the stimulus and an expectation (van Veen et al., 2004). A related proposal has been that the “oddball N2,” elicited by rare targets intermixed with frequent nontargets, is the same component as the feedback negativity (Holroyd, 2004). Though we have argued here that the visual oddball N2 has a predominantly posterior scalp distribution and so disagree with this precise formulation, it is clearly of interest to compare the relative sources of the mismatch and control N2 components. There is substantial agreement that at least some portion of the “cognitive control N2,” error-related negativity, and feedback-related negativity are generated in the anterior cingulate gyrus, so that an obvious question is whether the visual novelty/mismatch N2 has a shared neural source. The two paradigms that are most informative in eliciting the mismatch N2 are novelty oddball paradigms and S1-S2 matching paradigms in which match and mismatch trials are equated for probability and response requirements. Although studies with brain-damaged patients have indicated that frontal cortex is critical for the generation of the P3a response to novel stimuli (e.g., Daffner et al., 2003; Knight, 1984), these have been less informative about the novelty N2. We could find no fMRI adaptations or

source localization studies of the matching tasks employed by Wang and colleagues (Cui et al., 2000; Wang et al., 2003, 2004; Zhang et al., 2003), nor could we find successful dipole localizations for the novelty N2 in electromagnetic recordings. We therefore turn to fMRI studies of the novelty oddball paradigm.

Yamaguchi, Hale, D'Esposito, and Knight (2004) used a three-stimulus novelty paradigm with visual stimuli very similar to those used by Suwazono et al. (2000), a paradigm in which novel nontargets elicited robust electrical N2s. One modification was that participants were asked to attend to only one hemifield so that attended and unattended novels could be compared. Relative to standards, attended and unattended novels activated only posterior cingulate areas whereas attended targets activated the anterior cingulate. However, no area in the cingulate differed when novels and targets were compared directly, suggesting that novels did modulate the ACC to some extent. Two other studies have investigated BOLD activation in visual novelty oddball paradigms known to elicit robust novelty N2 components. Kiehl, Laurens, Duty, Forster, and Liddle (2001) found no anterior cingulate activation by novels relative to standards or targets but did find a novelty effect in a nearby region of medial frontal cortex estimated to lie within Brodmann's area 6 (supplementary motor area). Kirino, Belger, Goldman-Rakic, and McCarthy (2000) also observed very little frontal activation of any kind in response to novel photographs even though the same stimuli elicited a robust novelty N2 in electrical recordings. Although the fMRI studies are likely to be incapable, in principle, of distinguishing sources that contribute to an N2 versus a P3a component of a novelty response, the general lack of anterior cingulate activity in visual novelty paradigms provides little support for a source of the novelty N2 in the ACC, in contrast to the stronger data arguing for a “cognitive control N2” origin in this brain region.

The evidence to date is, however, also fairly sparse, and further research is needed to identify the source of the mismatch N2 and its relationship to medial frontal cortex. This research should include (1) source localization and imaging studies of the N270 identified by Wang and colleagues in S1-S2 matching tasks and (2) source localization and imaging studies of the novelty N2 using stimuli well proven to elicit the novelty N2. In particular, we suggest the complex meaningless stimuli used by Daffner et al. (2000) or by Polich and Comerchero (2003). Ideally these studies should include conditions predicted to elicit the control-related N2 so that the two components can be compared directly.

In conclusion, the frontally distributed N2 subcomponents should be further divided into the deviance-related or novelty N2 (for which we advocate the name “N2b”) and the control-related N2, which likely subsumes the ERN, no-go N2, conflict N2, Stroop N450, and FRN and also contributes to the rare target N2 and stop-signal N2. Whether these control-related N2 components reflect different subfunctions of the anterior cingulate or even different mixtures of signals from ACC and other frontal and parietal regions remains to be seen. The fact that, even in visual studies, the frontocentral N2 has multiple functional correlates suggests that it can never be interpreted in isolation. Special care must be taken to separate effects of deviance and expectation from effects related to overriding a prepotent response. Finally, although the two-stimulus oddball task played a pioneering role in the gross delineation of ERP components, it is likely of little further use in the identification of specific brain function (or dysfunction) when used in isolation.

Acknowledgements

We are grateful to Ken Forster, Elizabeth Glisky, and Lee Ryan for serving on the committee. We are also grateful to Erich Schröger and three anonymous reviewers for their careful reading and comments on a previous version. Funding was provided by the National Institute for Mental Health (MH073703). The first author also received partial support from the Cognitive Science Program at the University of Arizona.

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