

3 Overview of Common ERP Components

Overview

This chapter provides an overview of the ERP components that are most commonly encountered in cognitive, affective, and clinical neuroscience research. Some of the individual components could justify an entire chapter, but I will cover just the basics. For more extensive descriptions, see the edited volume on ERP components that Emily Kappenman and I organized (Luck & Kappenman, 2012b).

ERP components are classically divided into three main categories: (1) *exogenous* sensory components that are obligatorily triggered by the presence of a stimulus (but may be modulated to some degree by top-down processes); (2) *endogenous* components that reflect neural processes that are entirely task-dependent; and (3) *motor* components that accompany the preparation and execution of a given motor response. Although these terms provide a useful first approximation for dividing up the various ERP components, the category boundaries are not always clear. For example, is the lateralized readiness potential—which reflects response preparation whether or not a response is actually emitted—an endogenous component or a motor component? Consequently, you should not worry too much about the boundaries between these terms.

It is important for you to learn about all the major ERP components, even the ones that don't seem relevant to your area of research. As an analogy, cardiologists need to know the anatomy and physiology of all the major systems of the human body because the heart influences the whole body and the whole body influences the heart. Similarly, even if your main interest is language, you need to know about ERP components related to all the major systems of the brain. This is important because components from other domains will influence language-elicited ERP waveforms, and also because the components from these other domains may end up being directly useful to you for testing hypotheses about language. As chapter 4 describes in more detail, some of the highest-impact ERP studies have “hijacked” a component from one domain and used it to study a different domain.

One of the central issues that must be addressed for a given component is the nature of the psychological or neural processes that it reflects. Indeed, the conceptual definition of the term *ERP component* that I presented in chapter 2 requires that a component be related to a particular

computation. It turns out that this is a difficult issue to address, and an online supplement to this chapter will consider the general question of whether and how it is possible to assess the mental or neural function represented by a given component.

This chapter will describe the various ERP components in an order that roughly corresponds with their time of occurrence. We will begin with the contingent negative variation (CNV), which precedes a target stimulus, and then move on to the major visual and auditory sensory ERP components, with a brief discussion of sensory components from other modalities. We will then turn to the various components that contribute to the overall N2 wave, including the auditory mismatch negativity and the visual N2pc component (along with the distractor positivity [P_D] and contralateral delay activity [CDA] components, which are closely related to N2pc). This will be followed by a discussion of the P3 family of components and then a discussion of components related to language, long-term memory, emotion, error processing, and motor responses. We will then discuss steady-state ERPs, which are oscillatory neural responses that are evoked by rapidly oscillating stimuli. The final section will describe how four major ERP components were originally discovered. Before discussing individual components, however, it is necessary to say a few words about the naming conventions for ERP components (as briefly mentioned in chapter 1).

Naming Conventions

Unfortunately, the naming of ERP components is often inconsistent and sometimes ill-conceived. The most common convention is to begin with a P or N to indicate that the component is either positive-going or negative-going, respectively. This is followed by a number indicating the peak latency of the waveform (e.g., N400 for a negative component peaking at 400 ms) or the ordinal position of the peak within the waveform (e.g., P2 for the second major positive peak). This seems like a purely descriptive, theory-free approach, but it is not usually used this way. For example, the term *P300* was coined because it was positive and peaked at 300 ms when it was first discovered (Sutton, Braren, Zubin, & John, 1965). In most studies, however, the same functional brain activity typically peaks between 350 and 600 ms, but this component is still often labeled P300. Many investigators therefore prefer to use a number that represents the ordinal position of the component in the waveform (e.g., P3 instead of P300). This can still be confusing. For example, the first major peak for a visual stimulus is the P1 wave, which is observed over posterior electrode sites with a peak latency of approximately 100 ms. This component is not typically visible at anterior scalp sites, where the first major positive peak occurs at approximately 200 ms. This anterior positive peak at 200 ms is typically labeled *P2*, because it is the second major positive peak overall, even though it is the first positive peak in the waveform recorded at the anterior electrode sites.

Another problem is that a given label may refer to a completely different component when different sensory modalities are considered. For example, the auditory P1 wave bears no special relationship to the visual P1 wave. However, later components are largely modality-independent,

and the labels for these components typically refer to the same brain activity whether the stimuli are auditory or visual. For example, N400 refers to the same brain activity whether the eliciting stimulus is auditory or visual.

Although the conventions for naming ERP components can be very confusing to novices, experts usually have no trouble understanding exactly what is meant by these names. This is just like the problem of learning words in natural languages: two words that mean different things may sound exactly the same (homophones); two different words may have the same meaning (synonyms); and a given word may be used either literally or metaphorically. This is certainly an impediment to learning both natural languages and ERP terminology, but it is not an insurmountable problem, and in both cases some work is needed to master the vocabulary.

ERP components are sometimes given more functional names, such as the *syntactic positive shift* (which is observed when the subject detects a syntactic error in a sentence) or the *error-related negativity* (which is observed when the subject makes an obviously incorrect behavioral response). These names are often easier to remember, but they can become problematic when subsequent research shows that the same component can be observed under other conditions. For example, some investigators have argued that the error-related negativity is not directly related to the commission of an error and is present (although smaller) even when the correct response is made (Yeung, Cohen, & Botvinick, 2004).

The Contingent Negative Variation and Stimulus-Preceding Negativity

As you may recall from the brief description in chapter 1, the contingent negative variation (CNV) is a broad negative deflection between a warning stimulus and a target stimulus (Walter, Cooper, Aldridge, McCallum, & Winter, 1964). When the period between the warning and target stimuli is lengthened to several seconds, it is possible to see that the CNV actually consists of a negativity after the warning stimulus, a return to baseline, and then a negativity preceding the target stimulus (Loveless & Sanford, 1975; Rohrbaugh, Syndulko, & Lindsley, 1976). The first negative phase may reflect processing of the warning stimulus, and the second negative phase may reflect the readiness potential that occurs as the subject prepares to respond to the target (for a more nuanced account, see the comprehensive review by Brumia, van Boxtel, & Böcker, 2012).

A related ERP component is the stimulus-preceding negativity (SPN; see review by van Boxtel & Böcker, 2004). The SPN is a negativity that grows as the subject anticipates the occurrence of an information-bearing stimulus, such as a feedback tone, irrespective of whether an overt response is required for this stimulus. The SPN can be one of the subcomponents of the CNV.

It is important for you to know about these components because they can create confounds in your experiments. For example, if the interval between stimuli is relatively long in an experiment, an SPN will occur prior to each stimulus, and this can confound the results if the degree of anticipation differs across conditions.

Visual Sensory Responses

C1

Although the first major visual ERP component is typically the P1 wave, the P1 is sometimes preceded by a component called *C1*, which is largest at posterior midline electrode sites. Unlike most other components, it is not labeled with a P or an N because its polarity can vary. The C1 wave appears to be generated in area V1 (primary visual cortex), which in humans is folded into the calcarine fissure (figure 3.1). The part of area V1 that codes the lower visual field is on the upper bank of the fissure, and the part that codes the upper visual field is on the lower bank. In both cases, the positive side of the dipole points to the cortical surface, but the cortical surface

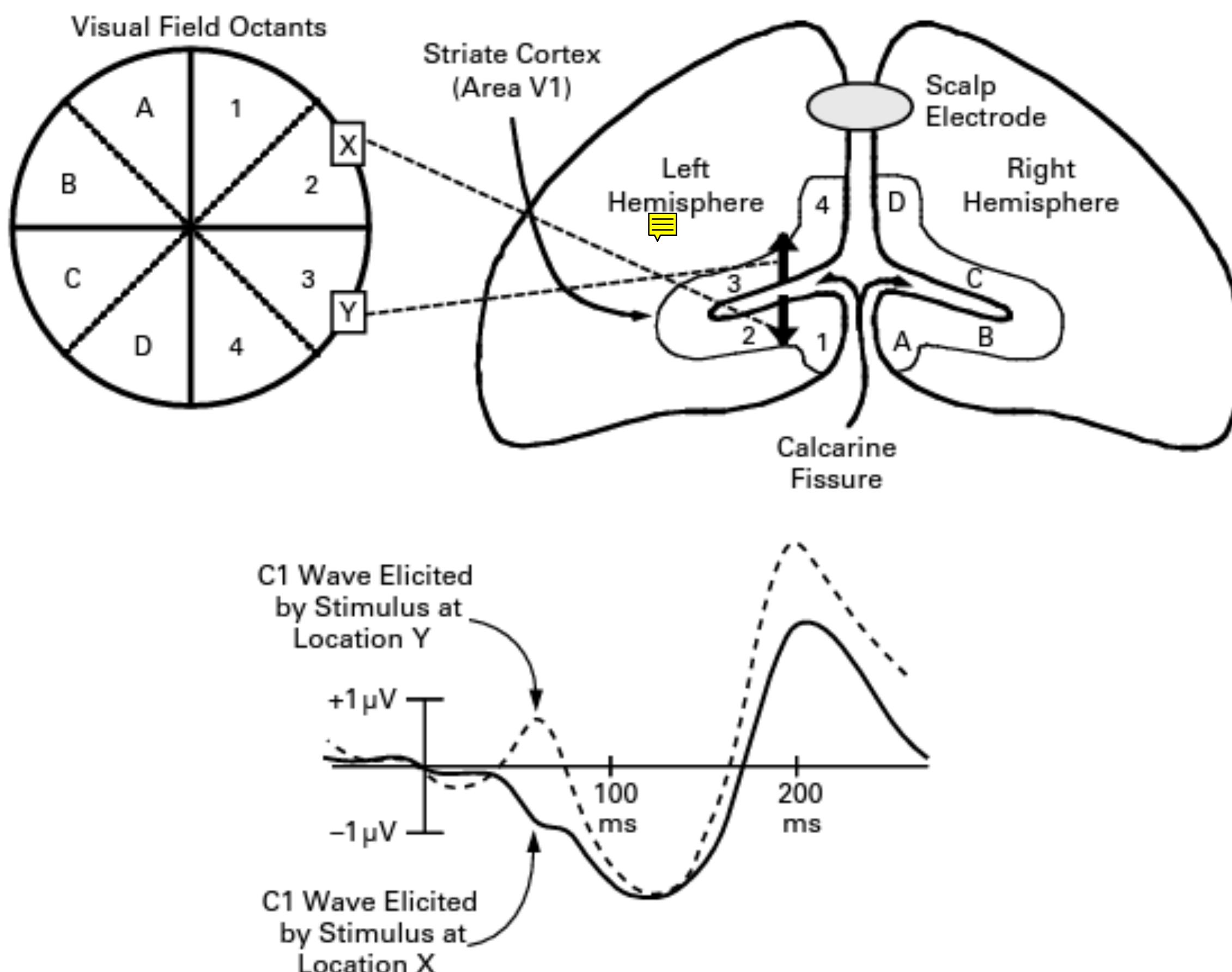


Figure 3.1

Relationship between stimulus position and C1 polarity. If a subject is staring at the center of the circle shown on the left side of the figure, the visual field can be divided into eight octants. Each octant projects to a different region of striate cortex, which wraps around the calcarine fissure as shown on the right side of the figure. If a stimulus is presented at location X in octant 2, just above the horizontal meridian, this will generate a dipole with a positive end that points downward and a negative end that points upward. This will lead to a negative potential recorded at a scalp electrode located on the midline (e.g., at the Oz or Pz electrode locations). If a stimulus is instead presented at location Y in octant 3, just below the horizontal meridian, the dipole will be located on the other side of the calcarine fissure, and the positive end will therefore point upward. This will lead to a positive potential at the scalp electrode.

is flipped for the upper bank of the fissure relative to the lower bank. As a result, the voltage recorded from an electrode above the calcarine fissure is positive for stimuli in the lower visual field and negative for stimuli in the upper visual field (Jeffreys & Axford, 1972; Clark, Fan, & Hillyard, 1995). When the C1 wave is positive, it sums together with the P1 component, creating a single positive-going wave. Consequently, a distinct C1 wave is not usually observed unless upper-field stimuli are used to generate a negative C1 wave (which can easily be distinguished from the positive P1 wave). The C1 wave typically onsets 40–60 ms poststimulus and peaks 80–100 ms poststimulus, and it is highly sensitive to basic visual stimulus parameters, such as contrast and spatial frequency.

The C1 wave provides my favorite example of the localization of an ERP component because the evidence does not just come from mathematical source localization techniques. Instead, the evidence comes from the confirmation of several predictions that derive from the hypothesis that C1 is generated in area V1. The most important of these predictions is that the C1 wave should invert in polarity for upper versus lower field stimuli, which is based on the known anatomy and physiology of visual cortex. There is no other area of visual cortex that is folded in a manner that would lead to this inversion. Moreover, the polarity actually flips slightly below the horizontal midline, which fits perfectly with the details of the mapping of the visual field onto area V1 (as discussed by Clark et al., 1995). The hypothesis that C1 is generated in area V1 also predicts that it should onset before any other visual component, and this hypothesis is also confirmed by the C1 onset latency. This hypothesis also predicts that the scalp distribution of the C1 wave should be consistent with a generator near the location of the calcarine fissure, which has also been verified (Clark et al., 1995; Di Russo, Martinez, Sereno, Pitzalis, & Hillyard, 2002). Thus, strong converging evidence supports the hypothesis that C1 is generated in area V1.

P1 and N1

The C1 wave is followed by the P1 wave, which is largest at lateral occipital electrode sites and typically onsets 60–90 ms poststimulus with a peak between 100 and 130 ms. Note, however, that P1 onset time can be difficult to assess because of overlap with the C1 wave. In addition, P1 latency will vary substantially depending on stimulus contrast. A few studies have attempted to localize the P1 wave by means of mathematical modeling procedures, sometimes combined with co-localization with fMRI effects, and these studies suggest that the early portion of the P1 wave arises from dorsal extrastriate cortex (in the middle occipital gyrus), whereas a later portion arises more ventrally from the fusiform gyrus (see Di Russo et al., 2002). Note, however, that many areas across the cortex are activated within the first 100 ms after the onset of a visual stimulus, and many of these areas presumably contribute to the voltages recorded in the C1 and P1 latency range (Foxe & Simpson, 2002). Like the C1 wave, the P1 wave is sensitive to variations in stimulus parameters, as would be expected given its likely origins in extrastriate visual cortex. The P1 wave is also modulated by selective attention (see review by Hillyard, Vogel, & Luck, 1998; Luck, Woodman, & Vogel, 2000) and by the subject's state of arousal (Vogel &

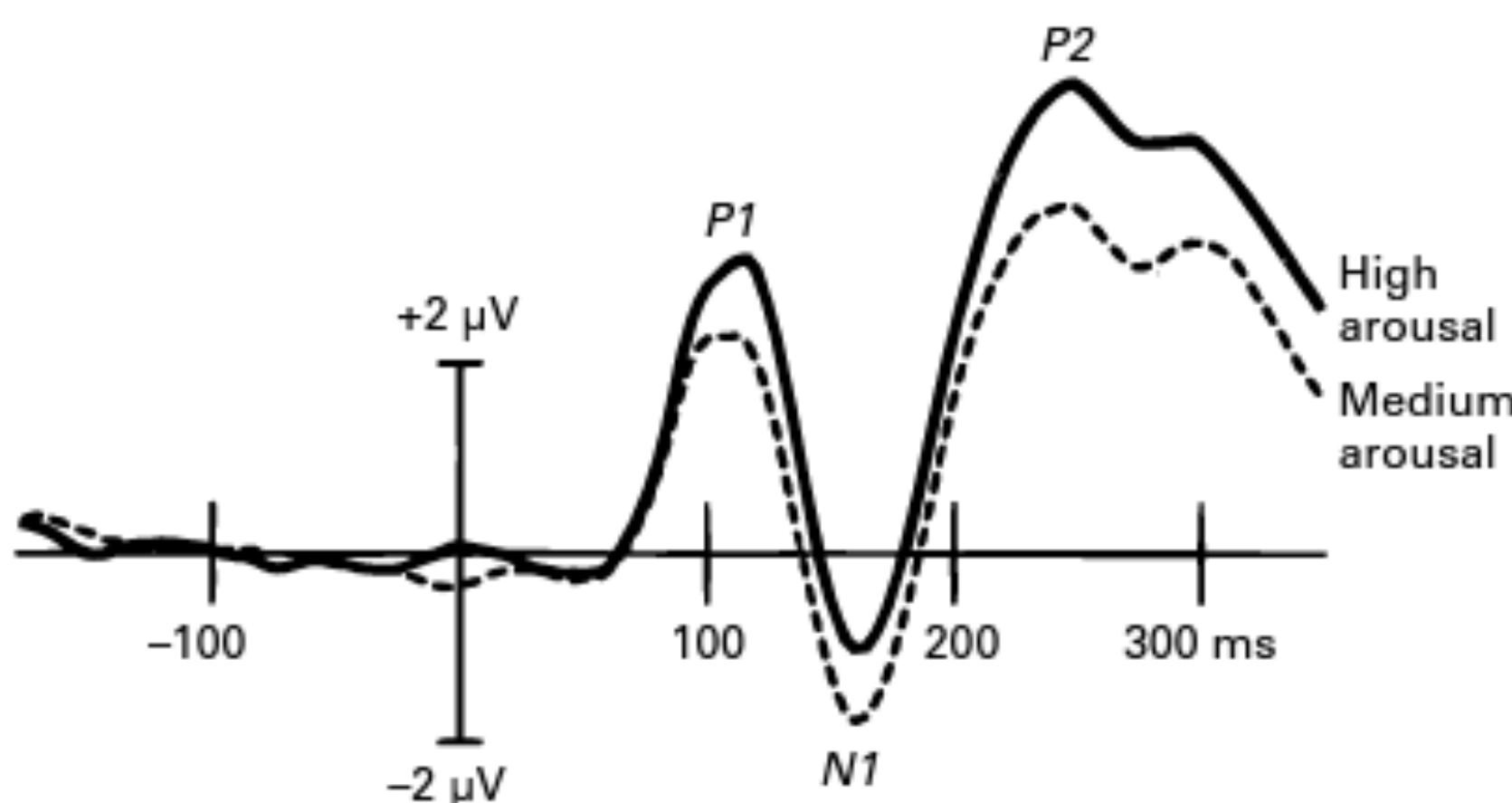


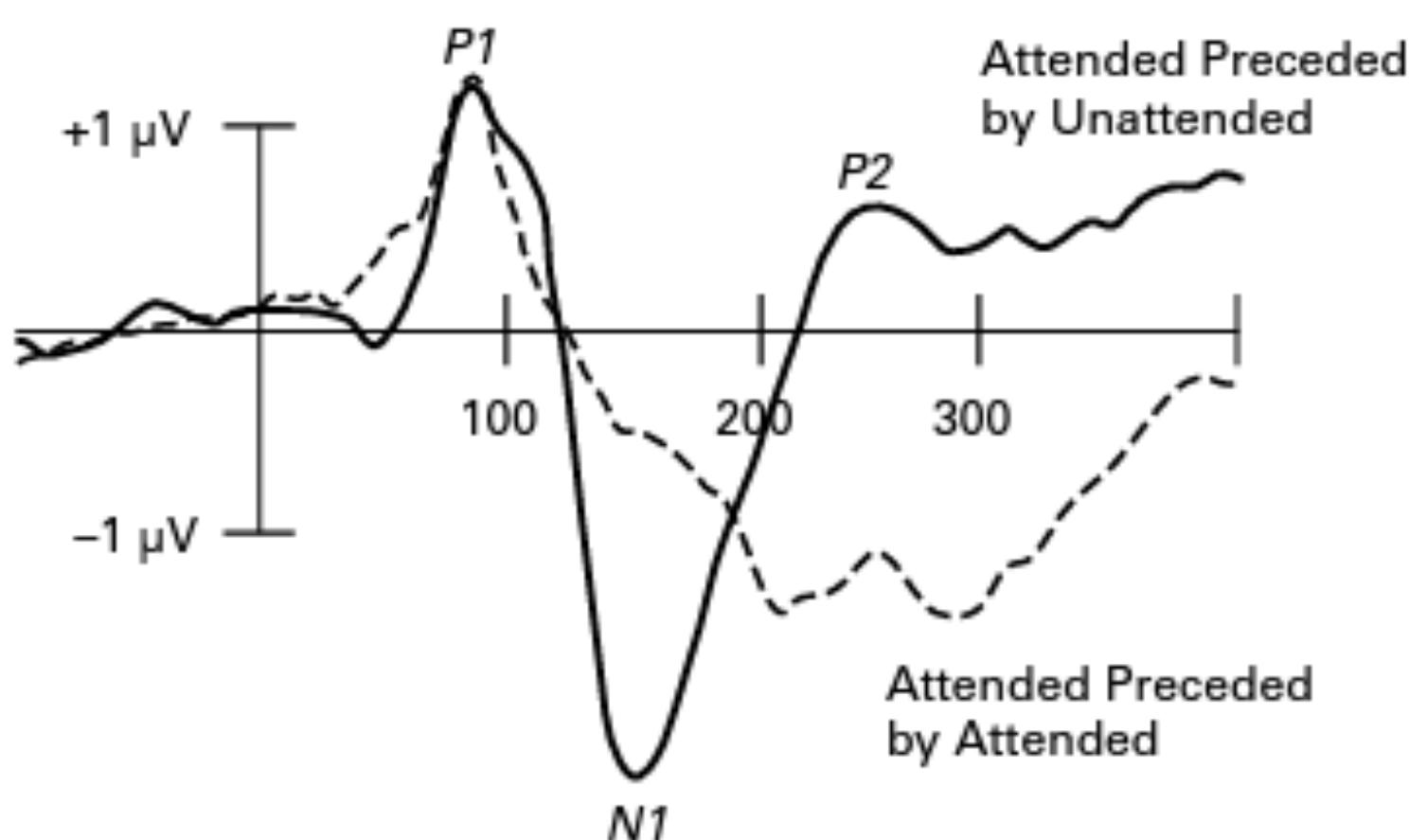
Figure 3.2

ERPs elicited by foveal visual stimuli under conditions of medium versus high arousal (from the study of Vogel & Luck, 2000). Note that arousal influences the entire waveform, beginning with the P1 wave.

Luck, 2000). Other top-down variables do not appear to reliably influence the P1 wave. In particular, P1 amplitude is not sensitive to whether a stimulus matches a task-defined target category (Hillyard & Münte, 1984).

The effect of arousal is illustrated in figure 3.2. In this experiment (Vogel & Luck, 2000), subjects pressed a button whenever they detected a stimulus (a foveal array of five letters). In the *medium arousal* condition, subjects were simply given our usual instructions for RT tasks; namely, “to respond as quickly as possible.” In the *high arousal* condition, subjects were encouraged to respond even faster. They were given feedback at the end of each block of trials that consisted of their mean RT for that block and a message stating that they must respond even faster in the next block. This manipulation was effective: mean RT was 292 ms in the medium arousal condition and 223 ms in the high arousal condition. As shown in figure 3.2, the amplitude of the P1 wave was increased in the high arousal condition relative to the low arousal condition. However, the difference between conditions persisted well after 300 ms, influencing the amplitude over the entire waveform. It is not clear which underlying components were affected later in the waveform, but it is clear that arousal can influence the measured value at virtually any point in the ERP waveform. It is therefore important to control for arousal in ERP experiments. For example, if two conditions are tested in separate trial blocks, and one condition is more difficult than the other, this may lead to arousal differences that confound the ERP measurements (see chapter 4 for additional discussion of this issue).

The P1 wave is followed by the N1 wave. There are several visual N1 *subcomponents*. That is, there are several distinct components that sum together to form the N1 peak. These components are not necessarily functionally related, and they are called *subcomponents* only because they contribute to the same visually salient deflection in the waveform. The earliest N1 subcomponent peaks 100–150 ms poststimulus at anterior electrode sites, and there appear to be at least two posterior N1 components that peak 150–200 ms poststimulus, one arising from parietal

**Figure 3.3**

ERPs elicited by lateralized visual stimuli at an attended location that were preceded either by a stimulus at the same attended location or at an unattended location in the opposite visual field (from the study of Luck et al., 1990). Note that amplitude of the N1 wave is greatly reduced when the previous stimulus was at the same location.

cortex and another arising from lateral occipital cortex. Many studies have shown that all three N1 subcomponents are influenced by spatial attention (see reviews by Mangun, 1995; Hillyard et al., 1998). In addition, the lateral occipital N1 subcomponent appears to be larger when subjects are performing discrimination tasks than when they are performing detection tasks, which has led to the proposal that this subcomponent reflects some sort of discriminative processing (Ritter, Simson, Vaughan, & Friedman, 1979; Vogel & Luck, 2000; Hopf, Vogel, Woodman, Heinze, & Luck, 2002).

The visual N1 component is highly *refractory*. That is, if a stimulus at a particular location is preceded by another stimulus at the same location at a short delay, the response to the second stimulus is greatly reduced. This is illustrated in figure 3.3 (from the study of Luck, Heinze, Mangun, & Hillyard, 1990). The figure shows the ERP recorded at temporal-occipital electrodes in response to an attended stimulus that was preceded by either an attended stimulus at the same location or an unattended stimulus at a different location. Although the P1 wave was approximately equivalent for these two trial types, the N1 wave was dramatically reduced when the attended stimulus was preceded by an attended stimulus in the same location. Many ERP components show effects such as this under some conditions, and this creates confounds in many experiments (to read about how this confounded some of my own experiments, see box 4.5 in chapter 4).

N170 and the Vertex Positive Potential

Jeffreys (1989) compared the responses to faces and non-face stimuli, and he found a difference from 150 to 200 ms at central midline sites that he named the *vertex positive potential* (VPP; note that the Cz electrode site at the very top of the head is sometimes called the *vertex* site). Jeffreys noted that this effect inverted in polarity at more lateral sites, but he did not have any

recordings from electrode sites over inferotemporal cortex. More recent studies using a broader range of electrode sites have found that faces elicit a more negative potential than non-face stimuli at lateral occipital electrode sites, especially over the right hemisphere, with a peak at approximately 170 ms (Bentin, Allison, Puce, Perez, & McCarthy, 1996; Rossion et al., 1999). This effect is typically called the *N170* wave (see figure 1.2 in chapter 1). It is likely that the N170 and the VPP are just the opposite sides of the same dipole (see review by Rossion & Jacques, 2012); the relative sizes of the N170 and VPP depend on what reference location is used (see chapter 5 for an in-depth discussion of reference electrodes). Note that the N170 is one of the subcomponents of the N1 wave. As was discussed in chapter 2, the voltage measured at 170 ms reflects the sum of the N170 component and other face-insensitive components, and you should be careful not to assume that the voltage elicited by a face in this time range solely reflects the face-sensitive N170 component.

A key question is whether the N170 effect (the difference between face and non-face stimuli) truly reflects face-specific processing or whether it reflects low-level properties that just happen to be more prominent in faces. Several lines of evidence now provide strong evidence that this effect is truly related to face perception (reviewed by Rossion & Jacques, 2012). For example, research by the late, great Shlomo Bentin showed that simple stimuli, which are not ordinarily perceived as faces, will elicit a larger N170 when subjects are primed to perceive these stimuli as faces (Bentin & Golland, 2002; Bentin, Sagiv, Mecklinger, Friederici, & von Cramon, 2002).

Much fMRI research has asked whether the same neural mechanisms that are ordinarily used to process faces are also used to process other complex stimuli for which the perceiver is an expert (e.g., cars for people who are car experts). The N170 has been used to address the same issue. Although ERPs lack the anatomical specificity of fMRI, their greater temporal resolution makes it possible to ask whether the timing of the processing is the same for faces and non-face objects. This is important because fMRI effects that are the same for faces and non-face objects might reflect processes that occur long after perception is complete. By showing that both faces and non-face objects elicit the same negative-going component at 170 ms, we can have more confidence that these effects reflect perception rather than some later postperceptual process. Supporting this possibility, initial studies demonstrated that bird and dog experts exhibit enhanced N170 responses for birds and dogs, respectively (Tanaka & Curran, 2001). But how can we know that this effect reflects the same neural circuits that produce the N170 for faces? A very clever set of experiments by Bruno Rossion and his colleagues answered this question by showing that the simultaneous presentation of the object of expertise and a face will reduce the N170 elicited by the face, which is typically taken as a sign of competition for the same neural circuits (Rossion, Kung, & Tarr, 2004; Rossion, Collins, Goffaux, & Curran, 2007).

Several studies have attempted to localize the N170 and its magnetic counterpart, the M170 (reviewed by Rossion & Jacques, 2012). Different studies have come up with substantially different estimates of the generator location, leading Rossion and Jacques (2012) to conjecture that

several different anatomical areas (e.g., the fusiform face area and the occipital face area) contribute to the scalp N170/M170.

P2

A distinct P2 wave follows the N1 wave at anterior and central scalp sites. This component is larger for stimuli containing target features, and this effect is enhanced when the targets are relatively infrequent (see Luck & Hillyard, 1994a). For example, figure 3.4B shows data recorded from the Cz electrode site in a visual oddball paradigm, in which the oddballs elicited a larger P2, N2, and P3 relative to the standards. Although both P2 and P3 are larger for oddball stimuli in some paradigms, the P2 effect occurs only when the target is defined by fairly simple stimulus features, whereas the P3 effect can occur for arbitrarily complex target categories. At posterior

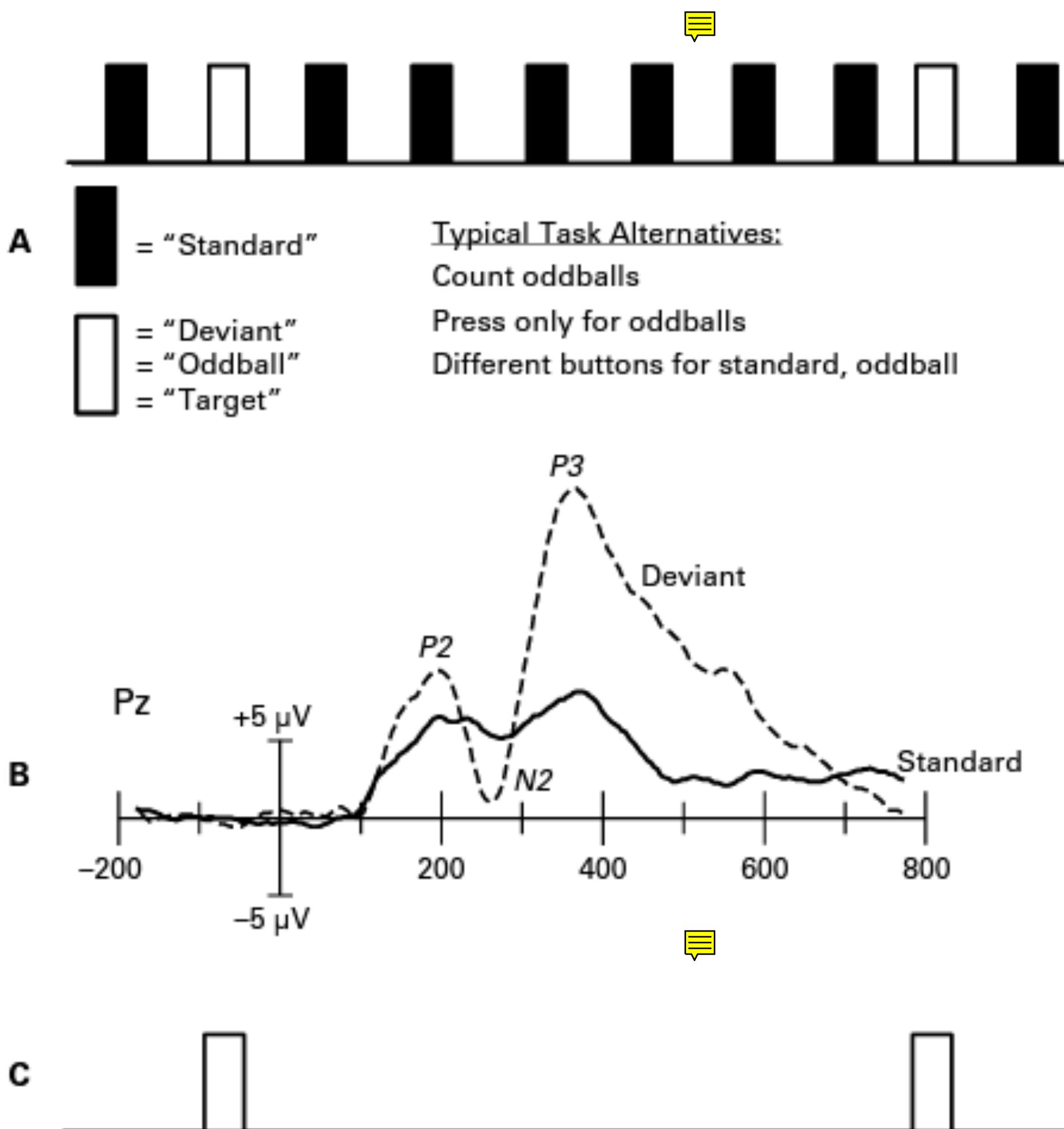


Figure 3.4

(A) Stimulus sequence in a typical oddball experiment, in which 80% of the stimuli are standards and 20% are targets (also known as oddballs or deviants). (B) Typical ERPs elicited at the Pz electrode site by standards and deviants in a visual oddball paradigm. (C) Target-only paradigm, in which the targets are presented at the same time as in the oddball paradigm, but without the standards. The P3 in this paradigm is nearly identical to the P3 in the traditional paradigm.

sites, the P2 wave is often difficult to distinguish from the overlapping N1, N2, and P3 waves. Consequently, not as much is known about the posterior P2 wave.

Auditory Sensory Responses

Figure 3.5 shows the typical ERP components evoked by the presentation of an auditory stimulus (see reviews by Picton, 2011; Pratt, 2012). If the stimulus has a sudden onset (such as a click), a distinctive set of peaks can be seen over the first 10 ms that reflect the flow of information from the cochlea through the brainstem and into the thalamus. These *auditory brainstem responses* (ABRs) are typically labeled with Roman numerals (waves I–VI). They are highly automatic and can be used to assess the integrity of the auditory pathways, especially in infants.

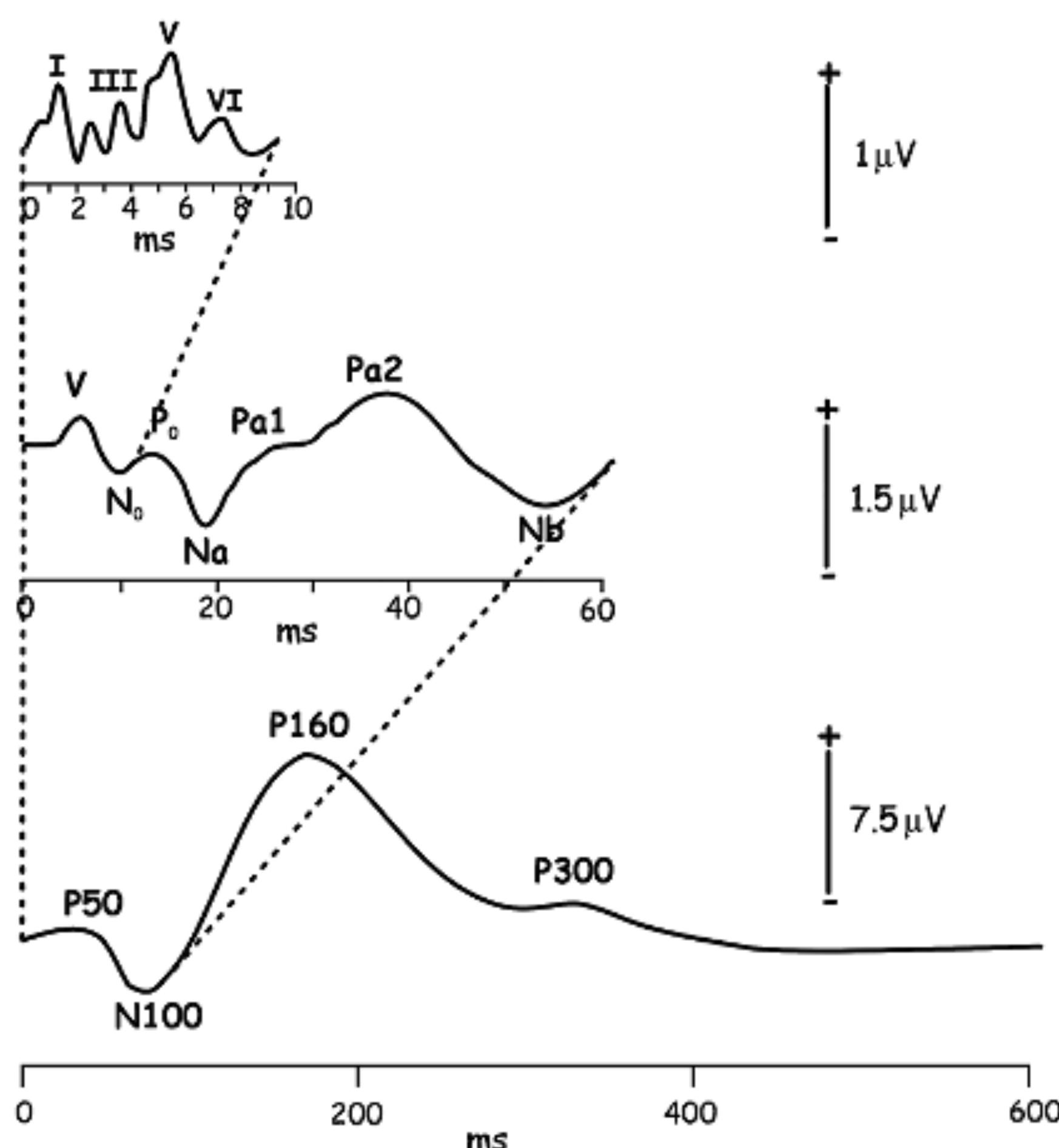


Figure 3.5

Typical sequence of auditory sensory components. The waveform elicited by a click stimulus is shown over different time ranges with different filter settings to highlight the auditory brainstem responses (top), the midlatency responses (middle), and the long-latency responses (bottom). Adapted with permission from Pratt (2012). Copyright 2012 Oxford University Press.

When my children were born, they were both given ABR screening tests, and it was gratifying to see that a variant of my main research technique is commonly used in a clinical application.

The ABRs are followed by the *midlatency responses* (MLRs; defined as responses between 10 and 50 ms), which probably arise at least in part from the medial geniculate nucleus and the primary auditory cortex. Attention has its first reliable effects in the midlatency range, but I don't know of any other cognitive variables that influence auditory activity in this time range.

The MLRs are followed by the *long-latency responses*, which typically begin with the P50 (aka P1), N100 (aka N1), and P160 (aka P2). The phrase *long-latency response* is a bit confusing, because these are relatively short latencies compared to high-level cognitive components, such as P3 and N400. However, the transmission of information along the auditory pathway is very fast, and 100 ms is relatively late from the perspective of auditory sensory processing. The long-latency auditory responses can be strongly influenced by high-level factors, such as attention and arousal.

Like the visual N1 wave, the midlatency and long-latency auditory responses become much smaller when the interval between successive stimuli decreases, with refractory periods that may exceed 1000 ms (this is true for sensory components in other modalities as well). Thus, when evaluating an ERP study, it is important to assess whether a difference between groups or conditions might be confounded by differences in the interstimulus interval.

Like the visual N1 wave, the auditory N1 wave has several distinct subcomponents (see review by Näätänen & Picton, 1987). These include (1) a frontocentral component that peaks around 75 ms and appears to be generated in the auditory cortex on the dorsal surface of the temporal lobes, (2) a vertex-maximum potential of unknown origin that peaks around 100 ms, and (3) a more laterally distributed component that peaks around 150 ms and appears to be generated in the superior temporal gyrus. Further fractionation of the auditory N1 wave is possible (see, e.g., Alcaini, Giard, Thevenet, & Pernier, 1994). The N1 wave is sensitive to attention: Although some attention effects in the N1 latency range reflect the addition of an endogenous component, the N1 wave itself (or at least some subcomponents) can be influenced by attention (Woldorff et al., 1993).

Figure 3.5 illustrates a common feature of ERP waveforms, namely, that the peaks tend to be narrow early in the waveform and become progressively broader later in the waveform. The ABR peaks, for example, last only 1–2 ms each, whereas the MLR peaks last 10–20 ms each, and some of the “cognitive” peaks such as P3 may last for several hundred milliseconds. This is not a coincidence. In almost any physical system, timing precision relative to a starting point becomes less and less precise as time progresses. This simply reflects the accumulation of timing errors. For example, if there is a 1-ms variance in timing a 10-ms interval, and we want to time a 70-ms interval, then the 1-ms variance associated with each of the seven successive 10-ms intervals will add together, and there will be a 7-ms variance in the timing of the 70-ms interval. Consequently, the variance across trials in the starting time of a given ERP component will tend to be larger for later components. This trial-to-trial variance will cause the component to become

temporally “smeared” when the individual trials are averaged together (chapter 8 and online chapter 11 will provide a more precise way of describing and understanding the exact nature of this smearing, using the mathematical concept of *convolution*).

Somatosensory, Olfactory, and Gustatory Responses

The vast majority of cognitive ERP experiments use auditory or visual stimuli, so I will provide only a brief mention of components from other modalities. The response to a somatosensory stimulus (see review by Pratt, 2012) begins with one of the rare ERP components that reflects action potentials rather than postsynaptic potentials, arising from the peripheral nerves. This *N10* response is followed by a set of subcortical components (ca. 10–20 ms) and a set of short- and medium-latency cortical components (ca. 20–100 ms). An N1 wave is then observed at approximately 150 ms, followed by a P2 wave at approximately 200 ms.

It is difficult to record olfactory and gustatory ERP responses, largely because it is difficult to deliver precisely timed, sudden-onset stimuli in these modalities (which is necessary when averaged ERP waveforms are computed). However, these potentials can be recorded when appropriate stimulation devices are used (see, e.g., Wada, 1999; Ikui, 2002; Morgan & Murphy, 2010; Singh, Iannilli, & Hummel, 2011).

The N2 Family

Many clearly different components have been identified in the time range of the second major negative peak (for reviews, see Näätänen & Picton, 1986; Folstein & Van Petten, 2008; Luck, 2012b). Early reports of N2 components typically came from oddball experiments, in which infrequently occurring targets (aka oddballs or deviants) are interspersed among frequently occurring standards (see figure 3.4A; see also the discussion of the oddball paradigm near the beginning of chapter 1).

Subdivision into N2a, N2b, and N2c Subcomponents

As described by Näätänen and Picton (1986), a repetitive, nontarget stimulus will elicit an N2 deflection that can be thought of as the *basic N2* (although it doubtless contains several sub-components). If deviant stimuli are occasionally presented within the repetitive train (e.g., in an oddball paradigm), a larger amplitude is observed in the N2 latency range for the deviants. Early research (summarized by Pritchard, Shappell, & Brandt, 1991) suggested that this effect could be divided into three subcomponents—termed *N2a*, *N2b*, and *N2c*—on the basis of manipulations of attention and stimulus modality. The N2a is an automatic effect that occurs for auditory mismatches, even if they are task-irrelevant. This effect is more commonly known as the *mismatch negativity* (MMN). Task-irrelevant visual oddballs can also elicit a negative-going wave from approximately 100 to 200 ms (Czigler, Balazs, & Winkler, 2002), but the early portion of this effect appears to reflect temporal rareness rather than mismatch detection per se (Kenemans,

Jong, & Verbaten, 2003). Thus, the MMN appears to be a mainly auditory phenomenon. The MMN will be discussed in more detail in a later section.

If the deviants are task-relevant, then a somewhat later N2 effect is observed, largest over central sites for auditory stimuli and over posterior sites for visual stimuli (Simson, Vaughan, & Ritter, 1977). These anterior and posterior effects were labeled *N2b* and *N2c*, respectively. More recent research has shown that an anterior N2 effect can also be observed for visual stimuli under some conditions (as described in the next section). The terms *N2b* and *N2c* have therefore been largely replaced by the terms *anterior N2* and *posterior N2*.

Anterior N2

There are several anterior N2 (*N2b*) effects that have been repeatedly reported in the literature (see the excellent review by Folstein & Van Petten, 2008). One common effect is related to response inhibition. Figure 3.6, for example, shows an example from the go/no-go paradigm (Bruin & Wijers, 2002). In this paradigm, subjects make a manual response for one stimulus and withhold this response for the other stimulus. When the no-go stimulus is presented, this produces conflict between the go response and the no-go response, especially when the go stimulus is more common than the no-go stimulus. Accordingly, the no-go N2 is largest when the go stimulus is more common than the no-go stimulus.

The anterior N2 is also sensitive to mismatch, but only when the stimuli are attended. That is, subjects must be performing some kind of task with the stimuli, even if it does not explicitly involve discriminating mismatches. For example, Suwazono, Machado, and Knight (2000) presented subjects with 70% standard stimuli (simple shapes), 20% targets (triangles), and 10% novel nontarget stimuli (color photographs). Even though the task did not require subjects to discriminate between the simple standards and the novel stimuli, the novel stimuli elicited an enhanced anterior N2. Similarly, Luck and Hillyard (1994a) presented subjects with homogeneous arrays

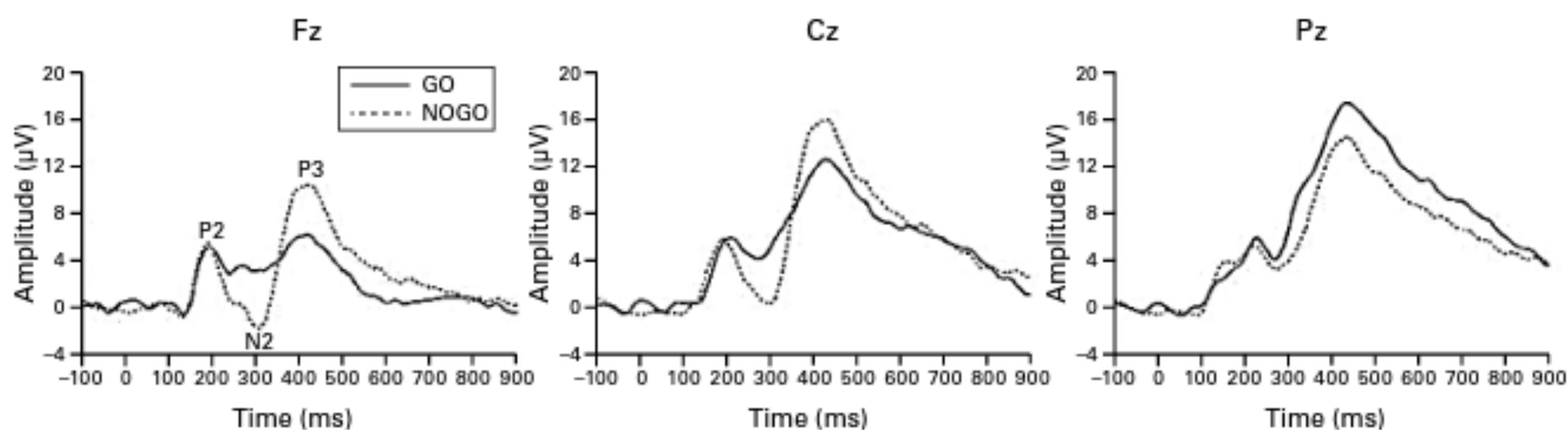


Figure 3.6

Grand average ERPs from the go/no-go study of Bruin and Wijers (2002). In this study, the letters M and W were presented at fixation, and the subject was instructed to make a finger-lift response for one stimulus but not the other (counterbalanced across trial blocks). The probabilities of the go and no-go stimulus were .25/.75, .50/.50, or .75/.25. The data shown here overlay the waveforms for go stimuli when they had a probability of .25 and no-go stimuli when they had a probability of .25. Adapted with permission from Bruin and Wijers (2002).

of colored rectangles or arrays that contained a “pop-out” item, which could be deviant in color, shape, or size. One of the three deviant types was the target, but both the target and nontarget deviants elicited a larger anterior N2 than the homogeneous arrays. Moreover, this effect was relatively independent of the probability of the deviants. However, it disappeared when the subjects were not attending to any of the three deviants. Thus, subjects must be attending to the stimulus arrays for this effect to be observed, but they do not need to be attending to the specific mismatch dimension.

Several anterior N2 effects are related to conflict between competing response alternatives. The no-go N2, for example, may reflect competition between the go response and the no-go “response.” An increased anterior N2 is also seen in the Eriksen flankers task. In this task, subjects make one of two responses to a central stimulus, and simultaneous task-irrelevant flanking stimuli may be assigned to the same response as the target (*compatible* trials) or to the opposite response (*incompatible* trials). The anterior N2 is typically larger for incompatible trials than for compatible trials (Folstein & Van Petten, 2008), presumably because the flanking stimuli activate the incorrect response to some extent, which then conflicts with the activation of the target response (e.g., Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Gehring, Gratton, Coles, & Donchin, 1992; Yeung et al., 2004). In fact, Yeung et al. (2004) proposed that the anterior N2 is actually the same as the error-related negativity (described later); as the activation of the incorrect response increases, the likelihood that the incorrect response is actually emitted also increases, so this brain activity will be larger when subjects actually make an incorrect response. A larger anterior negativity is also seen on incompatible trials relative to compatible trials in the Stroop paradigm (West & Alain, 1999, 2000; Liotti, Woldorff, Perez, & Mayberg, 2000). This effect is considerably later than the anterior N2 effect in the flankers paradigm, but it is possible that it nonetheless reflects the same process.

The anterior N2 component is also observed in the stop-signal paradigm. In this paradigm, the subject tries to make a speeded response to a visual target, but on some trials a tone occurs a few hundred milliseconds after the target, indicating that the response should be withheld. The anterior N2 is larger when subjects are unsuccessful at withholding the response than when they are successful (Pliszka, Liotti, & Woldorff, 2000; van Boxtel, van der Molen, Jennings, & Brumia, 2001), consistent with the idea that conflict should be greatest when go representation is so strong that it cannot be overcome by the stop representation.

An anterior N2 effect is also observed for feedback indicating whether a response was correct or incorrect. This is called the *feedback-related negativity* (FRN), and it is typically larger for negative feedback than for positive feedback (Holroyd & Coles, 2002; Nieuwenhuis, Yeung, Holroyd, Schurger, & Cohen, 2004; Hajcak, Holroyd, Moser, & Simons, 2005). Again, this can be considered a case of conflict between the response that was made by the subject and the correct response that was indicated by the feedback.

It is tempting to assume that all of these anterior N2 effects reflect the same underlying component, generated in medial frontal cortex (possibly the anterior cingulate cortex). However, fMRI data indicate that several distinct frontal brain regions exhibit activity related to the vari-

ables that influence the anterior N2 (see, e.g., Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). It is therefore quite plausible that multiple ERP components contribute to the anterior N2, just as multiple ERP components may contribute to the N170.

Posterior N2

The typical posterior N2 (N2c) is very much like the P3 wave, insofar as it is seen for task-relevant targets and is larger for rare targets than for frequent targets. Renault, Ragot, Lesevre, and Redmond (1982) proposed that this component reflects the process of categorizing a stimulus, because the duration of the component (measured from the target-minus-standard difference wave) depends on the difficulty of the categorization. However, increasing the difficulty of categorization also increases the onset latency of the P3 wave, and this may artificially change the apparent duration of the N2c component. Thus, the functional significance of the posterior N2 component is not clear.

The N2pc (N2-posterior-contralateral) component occurs during approximately the same time interval as the N2c component. The N2pc component is observed at posterior scalp sites contralateral to an attended object and reflects some aspect of the focusing of attention. The N2c and N2pc components can be distinguished from each other because the N2c is highly sensitive to the probability of the target whereas the N2pc is not (Luck & Hillyard, 1994a). The N2pc component will be discussed in greater detail in a later section of this chapter.

The Mismatch Negativity

The mismatch negativity (MMN) is a relatively automatic response to an auditory stimulus that differs from the preceding stimuli. It typically peaks between 160 and 220 ms, with a fronto-central midline scalp maximum.

Figure 3.7 shows the results of a typical MMN experiment (Näätänen & Kreegipuu, 2012). Tones were presented at a rate of approximately 1 per second while the subjects read a book and ignored the tones. Most (80%) of the tones were 1000-Hz standards, and the rest (20%) were slightly higher-pitched tones (1004, 1008, 1016, or 1032 Hz). The brain cannot easily discriminate between 1000, 1004, and 1008 Hz, so there was little or no difference in the ERP waveforms elicited by these three tones. However, the 1016- and 1032-Hz deviants elicited a greater negativity than the 1000-Hz standards from approximately 100 to 220 ms, and this greater negativity is the MMN. This MMN is often isolated from the rest of the ERP waveform with a deviant-minus-standard difference wave (shown in the right column of figure 3.7).

The most widely accepted theory of the MMN proposes that it reflects the comparison of a short-lived memory trace of the standards with the current stimulus (see review by Näätänen & Kreegipuu, 2012). However, the MMN effect is often contaminated by other brain activity. As was described earlier in this chapter, the auditory N1 wave is highly refractory. When the same pitch is presented many times, the neurons responding to this pitch will become less responsive, leading to a smaller N1 wave for this pitch. When a different pitch is presented, a somewhat

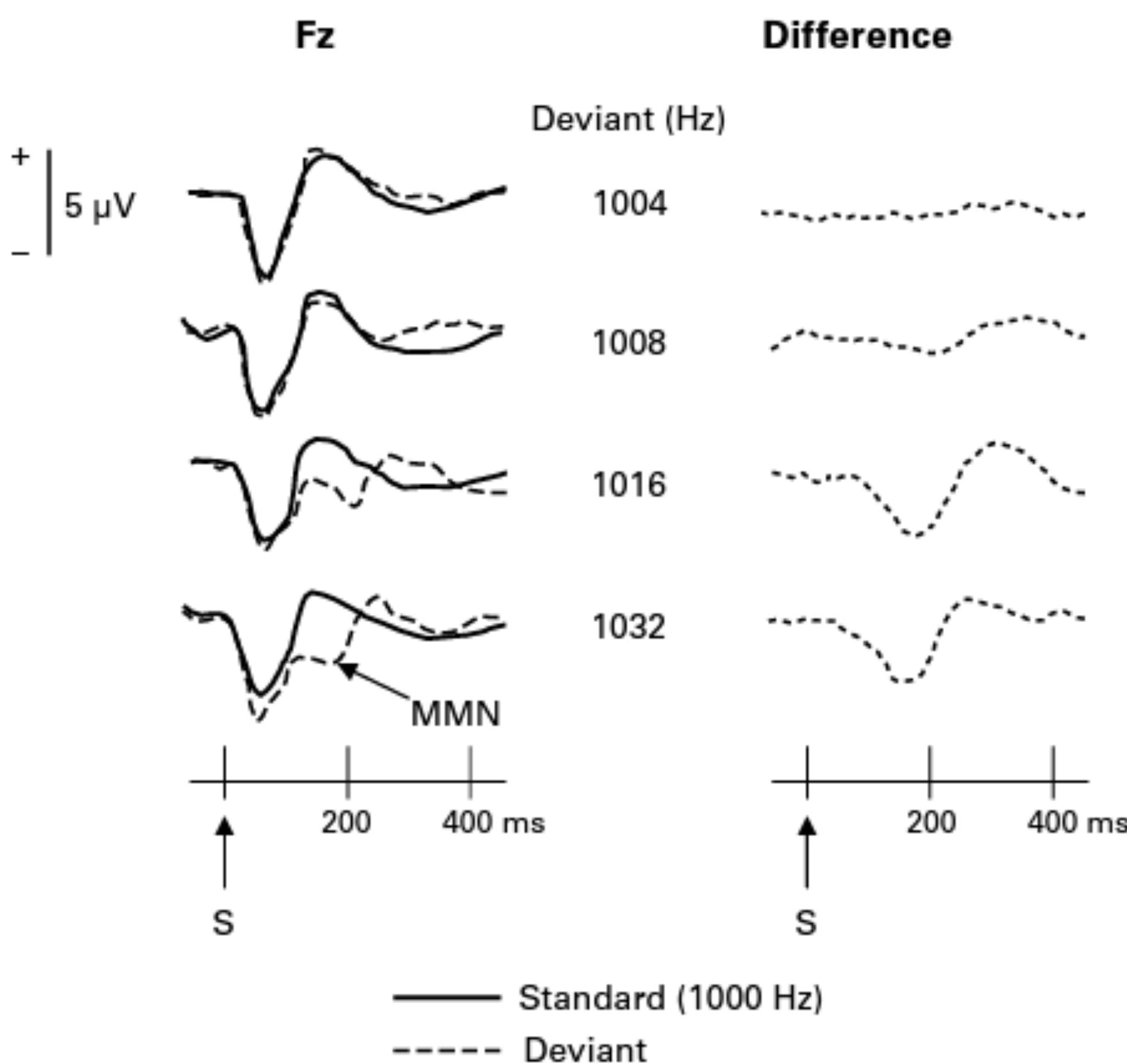


Figure 3.7

Example mismatch negativity results. While the subjects read a book, brief tones were presented at a rate of approximately 1 tone per second, with 80% standard tones (1000 Hz) and 20% deviant tones (1004, 1008, 1016, or 1032 Hz). The mismatch negativity is isolated by difference waves (right column) in which the ERP elicited by the standard stimulus is subtracted from the ERP elicited by the deviant stimulus. Adapted with permission from Näätänen and Kreegipuu (2012). Copyright 2012 Oxford University Press.

different population of neurons will be stimulated, and these neurons will produce a larger response than the neurons that code the standard pitch. This N1 difference between standards and deviants may therefore contribute to the MMN effect.

Three pieces of evidence indicate that, although this type of refractory confound can contribute to the MMN, a portion of the MMN cannot be explained in this manner. Specifically, an MMN can be elicited by tones that deviate from the standard by having a lower intensity (e.g., Woldorff, Hackley, & Hillyard, 1991; Woldorff, Hillyard, Gallen, Hampson, & Bloom, 1998), by tones occurring at an unexpectedly early time (Ford & Hillyard, 1981), and by the omission of a stimulus (Rüsseler, Altenmüller, Nager, Kohlmetz, & Munte, 2001). None of these results could be explained by a release from refractoriness when the deviant is presented. Studies that wish to isolate the “true” MMN from N1 refractory effects should use one of these procedures.

The MMN is often called *preattentive* and *automatic* because it is observed even if subjects are not using the stimulus stream for a task (e.g., if they are reading a book while the stimuli are being presented). However, the MMN can be eliminated for stimuli presented in one ear if the subjects focus attention very strongly on competing stimuli in the other ear (Woldorff et al., 1991). The problem with the terms *preattentive* and *automatic* is that attention is a complex set of cognitive processes that operate at different stages of processing under different conditions (see Luck & Vecera, 2002; Luck & Kappenman, 2012a), making it difficult to draw a clear line between *preattentive* and *attentive* activity or between *automatic* and *controlled* processing. It is best to steer clear of these terms when describing the MMN and stick to the facts. That is, you might describe the MMN as, “a response that is sufficiently automatic that it can be observed when the subject is reading a book.”

Because of its relatively high degree of automaticity, the MMN has been very useful as a means of assessing processing in individuals who cannot easily make behavioral responses, such as preverbal infants (Csepe, 1995; Trainor et al., 2003) and people who are comatose (Fischer, Lüaute, Adeleine, & Morlet, 2004). For example, the MMN can be used to assess infants’ sensitivity to various linguistic contrasts (Dehaene-Lambertz & Baillet, 1998; Cheour, Leppanen, & Kraus, 2000).

As reviewed by Näätänen and Kreegipuu (2012), at least two cortical generator sources contribute to the MMN. One is located in the auditory cortex of the supratemporal plane, just anterior to the N1 generator site. This generator leads to a negativity over the front of the head and a positivity at inferior posterior electrodes (when a nose reference site is used), and it is thought to reflect the mismatch detection process itself (see chapter 5 for more on the effects of reference electrode placement). The second is thought to arise from the prefrontal cortex and to reflect processes involved in shifting attention to the deviant sound. There is some intriguing evidence that the MMN specifically reflects the flow of current through NMDA receptor-mediated ion channels (Javitt, Steinschneider, Schroeder, & Arezzo, 1996; Umbricht et al., 2000; Kreitschmann-Andermahr et al., 2001; Ehrlichman, Maxwell, Majumdar, & Siegel, 2008; Heekeren et al., 2008; Tikhonravov et al., 2008), but some contradictory evidence has also been reported (Oranje et al., 2000).

N2pc, Distractor Positivity, and Contralateral Delay Activity

In this section, we will consider three high-level visual components that are isolated from the rest of the ERP waveform by measuring the difference in voltage between electrodes contralateral and ipsilateral to an object that is being attended (N2pc), an object that is being suppressed (distractor positivity; P_D), or an object that is being maintained in working memory (contralateral delay activity; CDA). The ability to effectively isolate these components from other components has made them extremely valuable for answering specific questions about high-level visual processes.

N2pc

As described earlier in this chapter, the posterior N2 contains a subcomponent that is called *N2-posterior-contralateral* (N2pc) because it is larger at contralateral sites than at ipsilateral sites relative to the location of an attended visual object (see review by Luck, 2012b).

Consider, for example, the experiment shown in figure 3.8 (from the study of Luck et al., 2006). Each stimulus array contained one red square, one green square, and a large number of black distractor squares. The locations of the individual squares varied at random from trial to trial, with the constraint that the two colored items were always on opposite sides of the display. The subject was instructed to attend either to red or to green at the beginning of each trial block

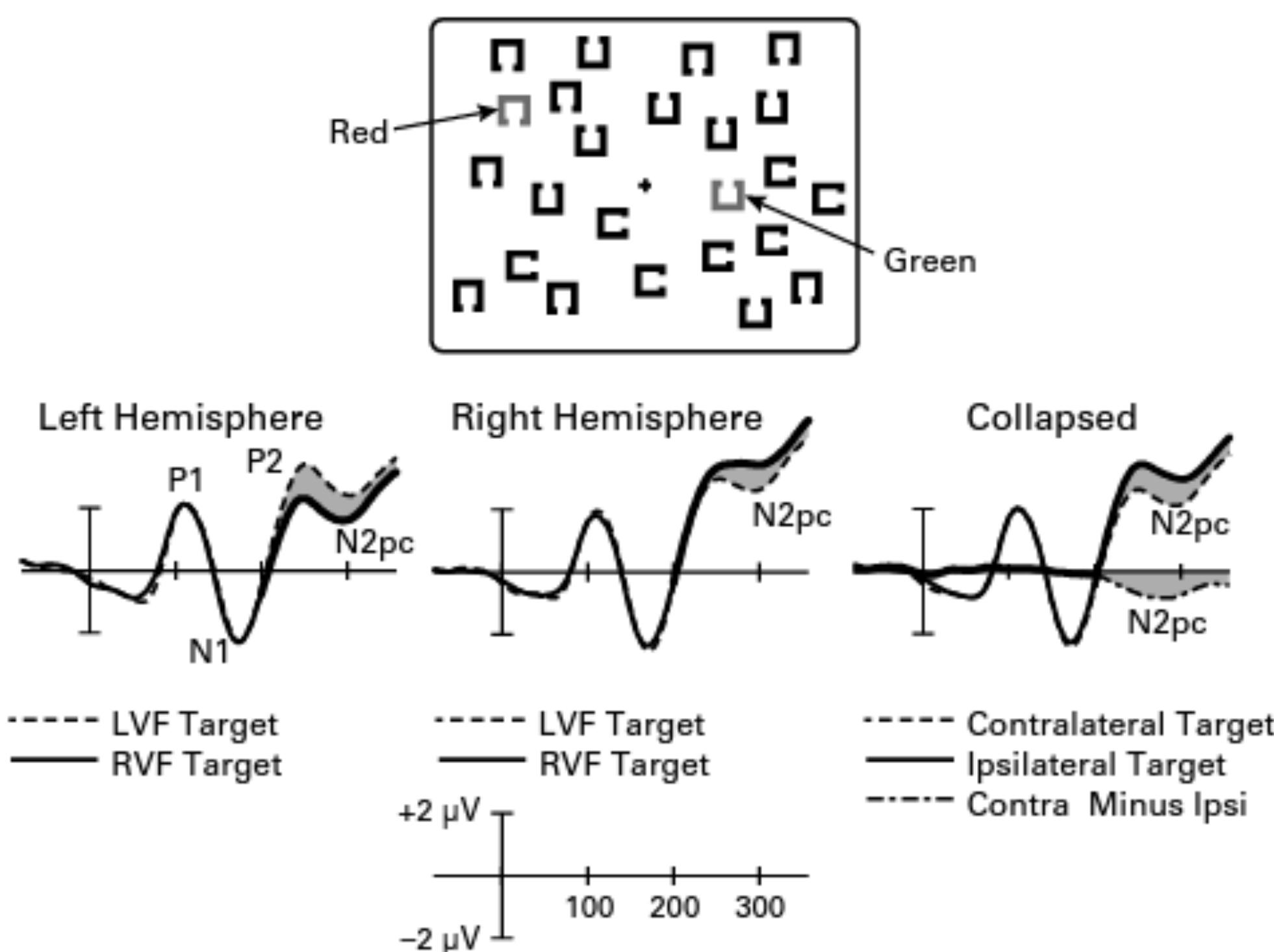


Figure 3.8

Typical N2pc paradigm and grand average ERP waveforms from posterior occipito-temporal electrode sites (from the study of Luck et al., 2006). To avoid any possibility of physical stimulus confounds, each stimulus array contained a distinctly colored item on each side, and one of these two colors was designated as the target color in the instruction screen for each trial block. Thus, the same stimulus array could be used to induce subjects to focus on either the left or right visual field, depending on which color was defined as the target. The locations of the items varied at random from trial to trial, except that the two pop-out colors were always on opposite sides. The subject was required to press one of two buttons to indicate whether the gap in the target item was at the top or the bottom of the square. The voltage in the N2 latency range over the left hemisphere was more negative when the target was in the right visual field than when it was in the left visual field, and the voltage over the right hemisphere was more negative when the target was in the left visual field than when it was in the right visual field. The data were collapsed into an ipsilateral waveform (left hemisphere/left target averaged with right hemisphere/right target) and a contralateral waveform (left hemisphere/right target averaged with right hemisphere/left target). The N2pc is defined as the difference between these contralateral and ipsilateral waveforms (shown as the shaded region), which was made explicit by constructing a contralateral-minus-ipsilateral difference wave. Adapted with permission from Luck (2012b). Copyright 2012 Oxford University Press.

and to press one of two buttons on each trial to indicate whether the attended-color object contained a gap on its top or a gap on its bottom. Because either red or green could be the target color (depending on the instructions given to the subject at the beginning of the trial), the same physical stimulus arrays could be used for trials that required focusing attention on the left (e.g., attend-red for the array shown in figure 3.8) or on the right (e.g., attend-green for the array shown in figure 3.8). This was important for avoiding a variety of stimulus confounds.

The N2pc component consists of a greater negativity when the attended item is contralateral to the recording electrode than when the attended item is ipsilateral (see the end of the chapter for the story of how N2pc was discovered). It typically occurs during the time range of the N2 wave (200–300 ms) and is observed at posterior scalp sites over visual cortex, with a maximum voltage near the PO7 and PO8 electrodes. In figure 3.8, the N2pc can be seen over the left hemisphere as a more negative voltage for targets in the right visual field (RVF) than for targets in the left visual field (LVF), and it can also be seen over the right hemisphere as a more negative voltage for LVF targets than for RVF targets. To avoid overall differences between LVF and RVF targets and overall differences between the left and right hemispheres, it is useful to create a collapsed contralateral waveform (the average of RVF for the left hemisphere and LVF for the right hemisphere) and a collapsed ipsilateral waveform (the average of LVF for the left hemisphere and RVF for the right hemisphere). The difference between these contralateral and ipsilateral waveforms is used to isolate the N2pc component from other overlapping ERP components. The rightmost portion of figure 3.8 shows the contralateral-minus-ipsilateral difference wave, illustrating how this difference wave eliminates the other components (e.g., P1 and N1). If you study the auditory modality, you might be interested to know that Marissa Gamble and I discovered an auditory analog of the N2pc component, which we called the *N2ac* component because it was observed over anterior contralateral electrode sites (Gamble & Luck, 2011). This is shown in figure 10.6 in chapter 10.

The N2pc component is useful for determining whether attention has been covertly directed to a given object and for assessing the time course of attentional orienting. For example, although an N2pc is typically observed for both simple feature targets and more complex conjunction targets, it is larger for conjunction targets than for feature targets (Luck, Girelli, McDermott, & Ford, 1997), and it can be eliminated for feature targets but not for conjunction targets when subjects are performing an attention-demanding secondary task (Luck & Ford, 1998). Similarly, N2pc can be used to determine whether attention is automatically captured by salient but irrelevant objects (Eimer & Kiss, 2008; Lien, Ruthruff, Goodin, & Remington, 2008; Sawaki & Luck, 2010). N2pc has also been used to show that masked, subliminal objects can nonetheless attract attention (Woodman & Luck, 2003a). The timing of the N2pc component has been used to show that objects associated with reward trigger faster shifts of attention (Kiss, Driver, & Eimer, 2009), that attention shifts serially from object to object in some visual search tasks (Woodman & Luck, 1999, 2003b), and that people with schizophrenia can shift attention just as quickly as healthy control subjects under some conditions (Luck et al., 2006).

Combined MEG/ERP/fMRI studies have shown that the topography of the N2pc component is consistent with a generator source in area V4 and the lateral occipital complex (LOC) (Hopf et al., 2000, 2006). Notably, the V4 source may be present only when the scale of the competition between the target and the distractors is fine enough to occur within the receptive fields of individual V4 neurons; when the scale of distraction is coarse, the N2pc may arise solely from the LOC (Hopf et al., 2006). This stretches the conceptual definition of the term *ERP component* provided in chapter 2. That is, the V4 and LOC sources may reflect the same computational operation (suppression of a competing distractor), but applied to different scales of information in distinct but nearby regions of cortex. Do these sources therefore reflect different components or should they be treated as the same component? If it turns out that we can reliably distinguish between these different sources and be certain that they reflect the same computational operation, I would be so happy that I wouldn't care about picky little definitional issues!

I originally hypothesized that the N2pc reflects the suppression of the distractors surrounding the attended object (Luck & Hillyard, 1994b), and subsequent research has confirmed that it is sensitive to the proximity of the distractors (Luck et al., 1997; Hopf et al., 2006). However, a clever study by Clayton Hickey, Vince Di Lollo, and John McDonald provided strong evidence that N2pc does not directly reflect distractor suppression (Hickey, Di Lollo, & McDonald, 2009; see box 3.1 for a story about this study). They presented arrays containing only one target and one distractor, and they borrowed a trick that Geoff Woodman and I previously developed for

Box 3.1
Honor

You might notice that the experimental design shown in figure 3.9 contains a low-level sensory imbalance, with a lateralized stimulus on one side of the screen but not the other, which could have contributed to the contralateral-minus-ipsilateral differences found by Hickey et al. (2009). To minimize this problem, Hickey et al. used colored stimuli that were the same luminance as the background, which reduces the sensory ERP amplitudes. I was one of the reviewers when this paper was submitted for publication to the *Journal of Cognitive Neuroscience*, and in my review I noted that it was still possible—although unlikely—that lateralized sensory responses were contaminating the N2pc and P_D effects. The study was done in the lab of John McDonald, who had trained as a postdoc in Steve Hillyard's lab several years after I left the lab, and I knew him fairly well. As a result, I had a little bit of fun in my review (which I signed), noting that, "I view this as a matter of honor more than a matter of publishability." The authors rose to the challenge and ran a control experiment ruling out contributions from sensory lateralizations, and this control experiment ended up providing additional evidence about the link between the P_D component and distractor suppression. Honor is important!

Several years later, Clayton Hickey was asked to review a paper that Risa Sawaki and I had submitted to *Visual Cognition*. He pointed out a potential alternative explanation of our results, and he brought up the issue of "honor" that I had raised in my review of his paper. He even quoted from my review of his paper. Risa and I were able to argue convincingly that this alternative explanation was unlikely, but it was quite amusing to see my own review used against me.

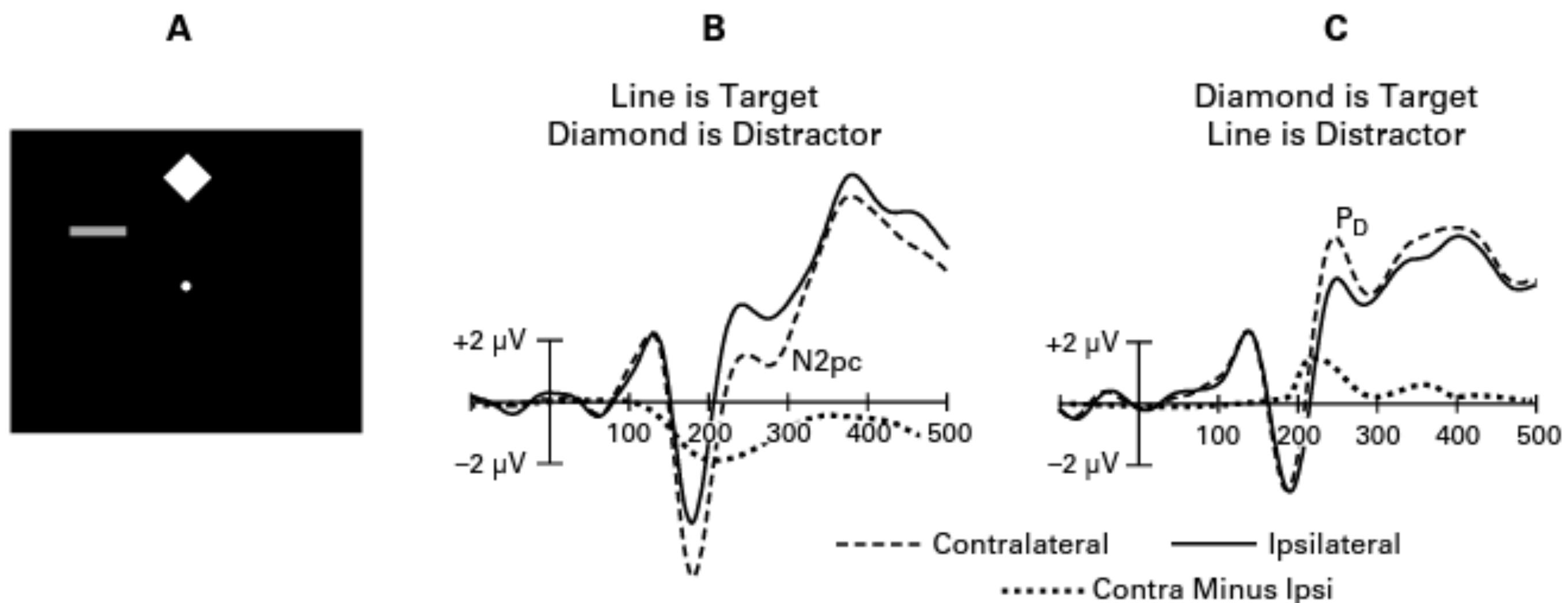


Figure 3.9

Example stimuli and grand average ERP waveforms from the study of Hickey et al. (2009). (A) Each array contained a bright green square or diamond along with a short or long red line that was isoluminant with the background. The red stimulus elicited almost no lateralized sensory activity, making it possible to attribute any later lateralized activity to top-down processing. (B) When the red line was the target, a contralateral negativity (N2pc) was elicited by the red item. (C) When the green diamond was the target, the ERP was more positive contralateral to the red distractor item than ipsilateral to this item. This contralateral positivity is called the distractor positivity (P_D). Waveforms courtesy of John McDonald.

isolating the N2pc to one of two items (Woodman & Luck, 2003b). Specifically, one of the two items was presented on the vertical midline, and the other was on the left or right side (figure 3.9A). There is no contralateral or ipsilateral side for the item on the vertical midline, so any brain activity elicited by this item should cancel out in a difference wave constructed by subtracting the waveform ipsilateral to the other item from the waveform contralateral to the other item.

When the target was lateralized and the distractor was on the midline, Hickey et al. (2009) found that the voltage was more negative over the hemisphere contralateral to the target than over the ipsilateral hemisphere (figure 3.9B). That is, they found the typical N2pc pattern, even though there were no lateralized distractors. This is pretty strong evidence that the N2pc is not directly related to the processing of the distractor but instead reflects processing of the target. Moreover, when the distractor was lateralized and the target was on the midline, the voltage was actually more positive and not more negative over the contralateral hemisphere relative to the ipsilateral hemisphere (figure 3.9C). Hickey et al. termed this positivity the *distractor positivity* (P_D). If the N2pc component directly reflected suppression of the distractor, then a negativity rather than a positivity should have been observed contralateral to the distractor.

What, then, does the N2pc component reflect? It is clearly related to the focusing of attention onto an object (for a discussion of the evidence for this claim, see Luck, 2012b), and its amplitude is influenced by the presence of nearby distractors, although it does not reflect processing of these distractors. The best current explanation is that it reflects the allocation of some kind

of limited-capacity process to one or more relevant objects (Ester, Drew, Klee, Vogel, & Awh, 2012), but in a way that is influenced by the presence of nearby distractors. Note that it is entirely possible that the N2pc does not reflect a single specific process, but instead reflects multiple processes that are applied to an attended stimulus and that are stronger in the contralateral hemisphere than in the ipsilateral hemisphere. That is, the N2pc may reflect multiple *consequences* of focusing attention onto a lateralized object.

By now, you should be noticing a theme: A given ERP effect may reflect multiple related processes (multiple face-related processes for the N170 component, multiple control-related processes for the anterior N2 component, and multiple attention-related perceptual processes for the N2pc component). Given the enormous complexity of the human brain, a difference wave based on a fairly subtle experimental manipulation (e.g., ipsilateral versus contralateral target location) may include activity from several processes that are implemented in nearby regions of cortex during the same general time period.

Distractor Positivity

As shown in figure 3.9C, the distractor positivity (P_D) component consists of a more positive voltage over the hemisphere contralateral to a distractor than over the ipsilateral hemisphere. Three findings from that study suggest that it reflects some kind of suppressive or inhibitory process that is applied to the distractor. First, it is lateralized with respect to the distractor, not with respect to the target, which indicates that the P_D reflects a process that operates on the distractor. It seems very likely that a process that operates on a distractor will be inhibitory in nature. Second, the polarity of the P_D component is opposite to that of the N2pc component, but the scalp distributions are quite similar. If we assume that the N2pc represents some kind of excitation (because it is applied to the target), then a reversal from an excitatory postsynaptic potential to an inhibitory postsynaptic potential in the same population of neurons would give us the opposite polarity. This is only weak evidence that the P_D represents an inhibitory process because it rests on difficult-to-test assumptions, but it is at least consistent with an inhibitory process. Third, Hickey et al. showed that the P_D was eliminated if subjects were required only to detect the presence of the target item rather than to discriminate its identity, which presumably reduced the need to actively suppress the distractor.

Additional evidence that the P_D reflects a suppressive process comes from subsequent studies conducted by Risa Sawaki in my lab. These studies showed that distractor items presented within a bilateral array will elicit a P_D rather than an N2pc if the distractor is highly salient (e.g., a color pop-out) or partially matches a working memory representation or target template (Sawaki & Luck, 2010, 2011; Sawaki, Geng, & Luck, 2012). A similar effect was reported by Eimer and Kiss (2008), but this was before Hickey et al. (2009) isolated the P_D component and proposed that it reflects distractor suppression, so Eimer and Kiss didn't realize they were seeing an inhibition-related component. Risa has also shown that P_D amplitude may be correlated with behavioral measures of attentional capture, with a larger P_D associated with less capture (Sawaki et al., 2012). Thus, there is growing evidence that the P_D reflects a process that is involved in

Box 3.2

Ignorance Is Not Always Bliss

Ever since the first N2pc experiments that I conducted in graduate school, I noticed that the N2pc was often followed by a contralateral positivity. For example, you can see this positivity following the N2pc component in figure 2 of Luck and Hillyard (1994b). This positivity was present in some experiments more than in others, but I could not decipher any consistency in the pattern of results. I decided that it was going to be nearly impossible to understand this positivity because it was partially canceled by the N2pc, making it difficult to measure separately from the N2pc. Because I didn't want to deal with this positivity, I started publishing N2pc studies in which the figures ended at approximately 300 ms so that people couldn't see the positivity that followed N2pc (see, e.g., figure 3.8 in this chapter and figure 5 in Luck et al., 1997).

When Risa Sawaki started getting interested in the P_D, she proposed that this positivity that followed the N2pc was the same as the P_D component; that is, the N2pc reflects the focusing of attention onto an object, and the P_D reflects the cancellation of attention after perception of the object was complete. I explained to Risa my skepticism about being able to figure out this subsequent positivity, but she ignored my advice. As I mentioned in box 3.1, it is sometimes good to ignore your mentor when he or she tells you that something can't be done, and Risa demonstrated this principle by running several experiments that explored the P_D that follows the N2pc component, leading to a very nice paper in the *Journal of Neuroscience* (Sawaki et al., 2012). This required developing a new way of measuring and analyzing the N2pc and P_D components to minimize the problem of cancellation (which you can read about in the paper).

the suppression of potentially distracting visual objects. Notably, an N2pc component is often followed by a P_D component, which appears to reflect a cancellation or resetting of attention after perception is complete (Sawaki et al., 2012) (box 3.2).

Contralateral Delay Activity and Working Memory

Ed Vogel and Maro Machizawa conducted a beautiful set of experiments extending the basic N2pc approach into the domain of working memory (Vogel & Machizawa, 2004). Previous studies by Dan Ruchkin and others had described a sustained negative voltage during the maintenance period of working memory tasks, which they called the negative slow wave (NSW; see review by Perez & Vogel, 2012). The NSW increases in amplitude as the memory load increases, and it has a frontal distribution for verbal memory tasks and a temporo-parietal distribution for visual memory tasks. Although the NSW has been a useful component, it faces two problems as a specific measure of working memory. First, to demonstrate that a neural measure actually reflects stored working memory representations per se, it is necessary to show that the measure is closely related to the amount of information that is stored in working memory. A given measure may increase as the load increases simply because the task is becoming more difficult, and not because of changes in the amount of information being stored in memory. Second, it is important to be able to isolate a memory-related component from other components that might be present during the delay interval, such as anticipation-related components that may

be present as subjects wait for the cue to report the stimuli at the end of the trial (see the section on the contingent negative variation and stimulus-preceding negativity later in this chapter). Vogel and Machizawa solved both of these problems by means of a contralateral-minus-ipsilateral difference wave (as described near the end of the chapter in the section on the discovery of the CDA).

The basic paradigm of Vogel and Machizawa (2004) is shown in figure 3.10A. Each trial began with a *cue arrow*, which told the subjects which side should be remembered on that trial. The cue was followed by a *sample array*, which contained one to four colored squares on each side of the fixation point. Subjects were supposed to store the items on the cued side of the array in memory, completely ignoring the items on the uncued side. At the end of the trial, a *test array* was presented, and subjects reported whether one of the items on the cued side had changed

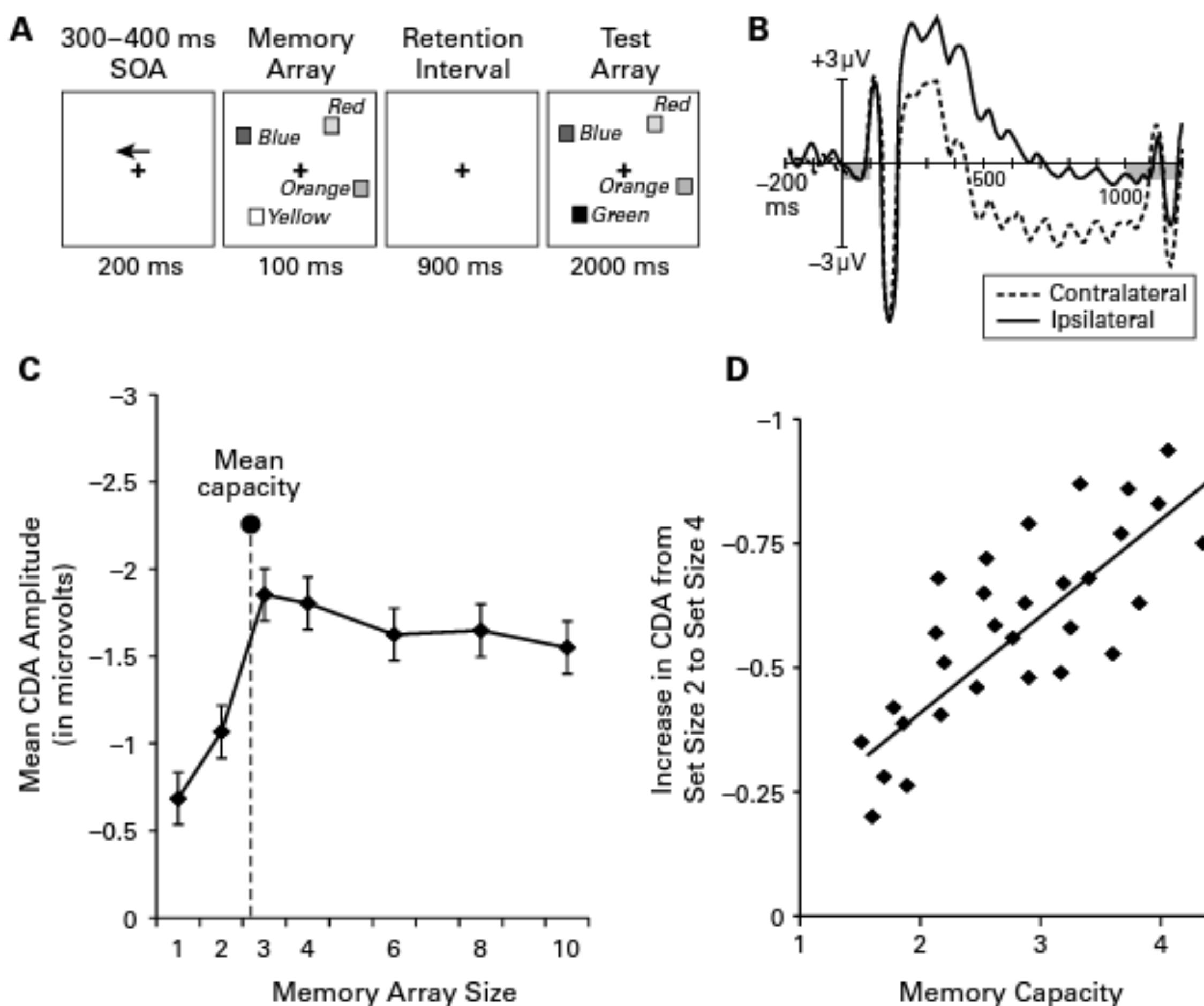


Figure 3.10

ERP results from Vogel and Machizawa (2004) showing the contralateral delay activity (CDA). (A) Experimental design. (B) Grand average ERP waveforms, time-locked to the sample stimulus and extending through the delay interval. (C) Mean CDA amplitude as a function of memory set size. (D) Correlation between subject's visual working memory capacity, measured behaviorally, and the magnitude of the increase in CDA between set sizes 2 and 4. Adapted with permission from Perez and Vogel (2012). Copyright 2012 Oxford University Press.

color between the sample and test arrays. The colors on the uncued side always remained the same between the sample and test arrays, giving subjects a strong incentive to remember the items on the cued side but not the items on the uncued side.

Figure 3.10B shows the ERP waveforms recorded over posterior scalp sites contralateral and ipsilateral to the cued side, time locked to the sample array and extending through the delay interval until the time of the test array. The voltage was more negative over the contralateral hemisphere than over the ipsilateral hemisphere from approximately 200 ms after the onset of the sample array until the onset of the test array. The early portion of this contralateral negativity presumably consists of an N2pc component, but the N2pc is not ordinarily sustained for more than a few hundred milliseconds. Thus, the sustained activity during the later portion of the delay interval represents an additional ERP component, which Vogel and Machizawa (2004) called *contralateral delay activity* (CDA). Although both the N2pc and CDA are contralateral negativities over posterior scalp sites, the CDA has a more parietal distribution than the N2pc, and MEG studies suggest that the CDA is generated in posterior parietal cortex (Robitaille, Grimault, & Jolicoeur, 2009), whereas the N2pc appears to be generated in ventral occipito-temporal cortex (Hopf et al., 2000, 2006).

Figure 3.10C shows that CDA amplitude is closely tied to working memory capacity. Average storage capacity in this experiment, measured behaviorally, was slightly under three items, and the CDA reached an asymptote at a set size of three items. This demonstrates that the CDA is related to memory capacity per se and does not just increase as the task becomes more difficult. Moreover, individual differences in working memory capacity, as measured behaviorally, were tightly correlated with the set size at which CDA amplitude reached asymptote. Specifically, figure 3.10D shows that a given subject's memory capacity was correlated with the change in CDA amplitude between set size 2 and set size 4. This tight correlation between the CDA and behaviorally measured capacity provides excellent evidence that the CDA is closely related to the actual maintenance of information in working memory. This also shows how ERPs can be extremely useful in assessing individual differences (see also Vogel, McCollough, & Machizawa, 2005; Leonard et al., 2012).

The P3 Family

Varieties of P3 Components

There are several distinguishable ERP components in the time range of the P3 wave (for a review of the P3 wave, see Polich, 2012). The first major distinction was made by Squires, Squires, and Hillyard (1975), who distinguished between a frontally maximal P3a component and a parietally maximal P3b component. Both were elicited by unpredictable, infrequent changes in the stimuli, but the P3b component was present only when these changes were task-relevant. When ERP researchers (including myself) refer to *the P3 component* or *the P300 component*, they almost always mean *the P3b component* (in fact, I have already used the term *P3* to refer to the P3b component several times in this book).

Other studies have shown that an unexpected, unusual, or surprising task-irrelevant stimulus within an attended stimulus train will elicit a frontal P3-like response (e.g., Courchesne, Hillyard, & Galambos, 1975; Soltani & Knight, 2000; Polich & Comerchero, 2003), but it is not clear whether this response is related to the P3a component as originally described by Squires et al. (1975). For example, Verleger, Jaskowski, and Waushckuhn (1994) provided evidence that the P3b component is observed for targets that are infrequent but are in some sense expected or awaited, whereas the frontal P3 wave is elicited by stimuli that are truly unexpected or surprising. However, it is not clear that this frontal P3 is as automatic as the P3a observed by Squires et al. (1975).

Theories of Functional Significance

Given the thousands of published P3 experiments, you might think that we would have a very thorough understanding of the P3 wave. But you'd be wrong! We know a great deal about the effects of various manipulations on P3 amplitude and latency, but there is no clear consensus about what neural or cognitive processes are reflected by the P3 wave. The most widely cited theory was developed by Donchin (1981), who proposed that the P3 wave is related to a process he called "context updating." This is often interpreted to mean that the P3 wave reflects the updating of working memory, but this is not what Donchin meant. He likes to point out that he never used the phrase *working memory*, and if you read his paper (which I strongly encourage), he uses the word *context* to mean something very different from working memory (and very different from the way that *context* is used by people like Jonathan Cohen). For Donchin, *context* representations are broad representations of the overall state of the environment, not specific representations of individual items or tasks.

The evidence supporting the context updating theory of the P3 wave is pretty sparse, and very few direct tests have been performed in the time since Donchin's original description of the theory. If you are interested in the P3 wave, you should probably read Donchin's original paper (Donchin, 1981), Verleger's extensive critique of this theory (Verleger, 1988), and the response of Donchin and Coles to this critique (Donchin & Coles, 1988). In my own laboratory's research on attention, we have frequently assumed that the P3 wave reflects working memory updating, and this has led to a variety of very sensible results (e.g., Luck, 1998a; Vogel, Luck, & Shapiro, 1998; Vogel & Luck, 2002). But this assumption certainly carries some risk, so you should be careful in making assumptions about the meaning of the P3 wave.

Another key concept, again raised by Donchin (1981), is that the process reflected by the P3 wave is *strategic* rather than *tactical*. A tactical response is something that is done to deal with the current situation (e.g., when a pilot suddenly banks to the left to avoid a flock of birds). A strategic response is something that is done to prepare for the future (e.g., when a pilot chooses a different route to avoid turbulence that is predicted to occur in an hour). Donchin proposed that the P3 wave reflects a strategic process rather than a tactical process because it frequently occurs too late to have an impact on the behavioral response. Moreover, Donchin argued that

the amplitude of the P3 elicited by a stimulus is predictive of later memory for that stimulus (for a review of memory-related ERP effects, see Wilding & Ranganath, 2012).

Effects of Probability

Although we do not know exactly what the P3 wave means, we do know what factors influence its amplitude and latency (for extensive reviews of the early P3 literature, see Pritchard, 1981; Johnson, 1986; for more recent reviews, see Picton, 1992; Polich & Kok, 1995; Polich, 2004, 2012). The hallmark of the P3 wave is its sensitivity to target probability. As shown in great detail by Duncan-Johnson and Donchin (1977), P3 amplitude gets larger as target probability gets smaller. However, it is not just the overall probability that matters; local probability also matters, because the P3 wave elicited by a target becomes larger when it has been preceded by more and more nontargets.

A crucial detail is that the probability of a given physical stimulus is not the relevant factor. Instead, it is the *probability of the task-defined stimulus category* that matters. For example, a classic experiment by Marta Kutas showed that if subjects are asked to press a button when detecting male names embedded in a sequence containing male and female names, the amplitude of the P3 wave will depend on the relative proportions of male and female names in the sequence even though each individual name appears only once (see Kutas, McCarthy, & Donchin, 1977). Similarly, if the target is the letter E, occurring on 10% of trials, and the nontargets are selected at random from the other letters of the alphabet, the target will elicit a very large P3 wave even though the target letter is approximately four times more probable than any individual nontarget letter (see Vogel et al., 1998).

For decades, people assumed that the amplitude of the P3 depends on the *sequential probability* of the stimulus category (i.e., where the probability is the number of stimuli in a particular category divided by the total number of stimuli). However, more recent studies indicate that P3 amplitude is largely dependent on the *temporal probability* of the stimulus category (i.e., where probability is the number of stimuli in a particular category divided by the time period over which the stimuli are presented) (Polich, 2012). These two factors are typically confounded. For example, figure 3.4A shows a series of stimuli in which the target stimulus is both sequentially rare (because only two of the 10 stimuli are targets) and temporally rare (because only two target stimuli are presented over a fairly long period of time). If you simply leave out the standards, but maintain the same timing of the targets (as in figure 3.4C), the P3 elicited by the targets is about the same as it would be with the standards included, even though the sequential probability of the targets is now 100% (Polich, Eischen, & Collins, 1994; Katayama & Polich, 1996).

P3, Resource Allocation, and Task Difficulty

P3 amplitude is larger when subjects devote more effort to a task, leading to the proposal that P3 amplitude can be used as a measure of resource allocation (see, e.g., Isreal, Chesney, Wickens, & Donchin, 1980). However, P3 amplitude is smaller when the subject is uncertain of whether

a given stimulus was a target or nontarget. Thus, if a task is made more difficult, this might increase P3 amplitude by encouraging subjects to devote more effort to the task, but it might decrease P3 amplitude by making subjects less certain of the category of a given stimulus. Consequently, there is no simple rule for determining whether the P3 will get larger or smaller for more difficult tasks. Johnson (1984, 1986) proposed that the variables of probability (P), uncertainty (U), and resource allocation (R) combine to influence P3 amplitude in the following manner: $P3\text{ amplitude} = U \times (P + R)$.

P3 Latency and Stimulus Categorization

The fact that P3 amplitude depends on the probability of the task-defined category of a stimulus has an important but often-overlooked consequence. Specifically, it is logically necessary that a difference in P3 amplitude between the rare and frequent trials means that the subject has begun to categorize the stimulus according to the rules of the task by the time the difference is present. As an example, consider figure 3.11, which provides a cartoon version of the Kutas et al. (1977) experiment in which subjects were instructed to categorize names as male or female. The category of female names was rare and the category of male names was frequent, but each individual name occurred only once. In this cartoon, the difference between the two categories deviated from 0 μ V at approximately 300 ms. This provides essentially bullet-proof evidence that the brain had begun to determine the category of the names by 300 ms (with the caveat, described in chapter 2, that the onset of a difference will reflect the trials with earliest onset).

Because a difference in P3 amplitude between rare and frequent stimuli cannot occur until the brain has categorized the stimulus as rare or frequent, any manipulation that postpones stimulus categorization (including increasing the time required for low-level sensory processing or higher-level categorization) must necessarily increase the onset time of P3 probability effect. That is, anything that increases the amount of time required for the brain to determine whether a given stimulus falls into the rare category or the frequent category will necessarily increase

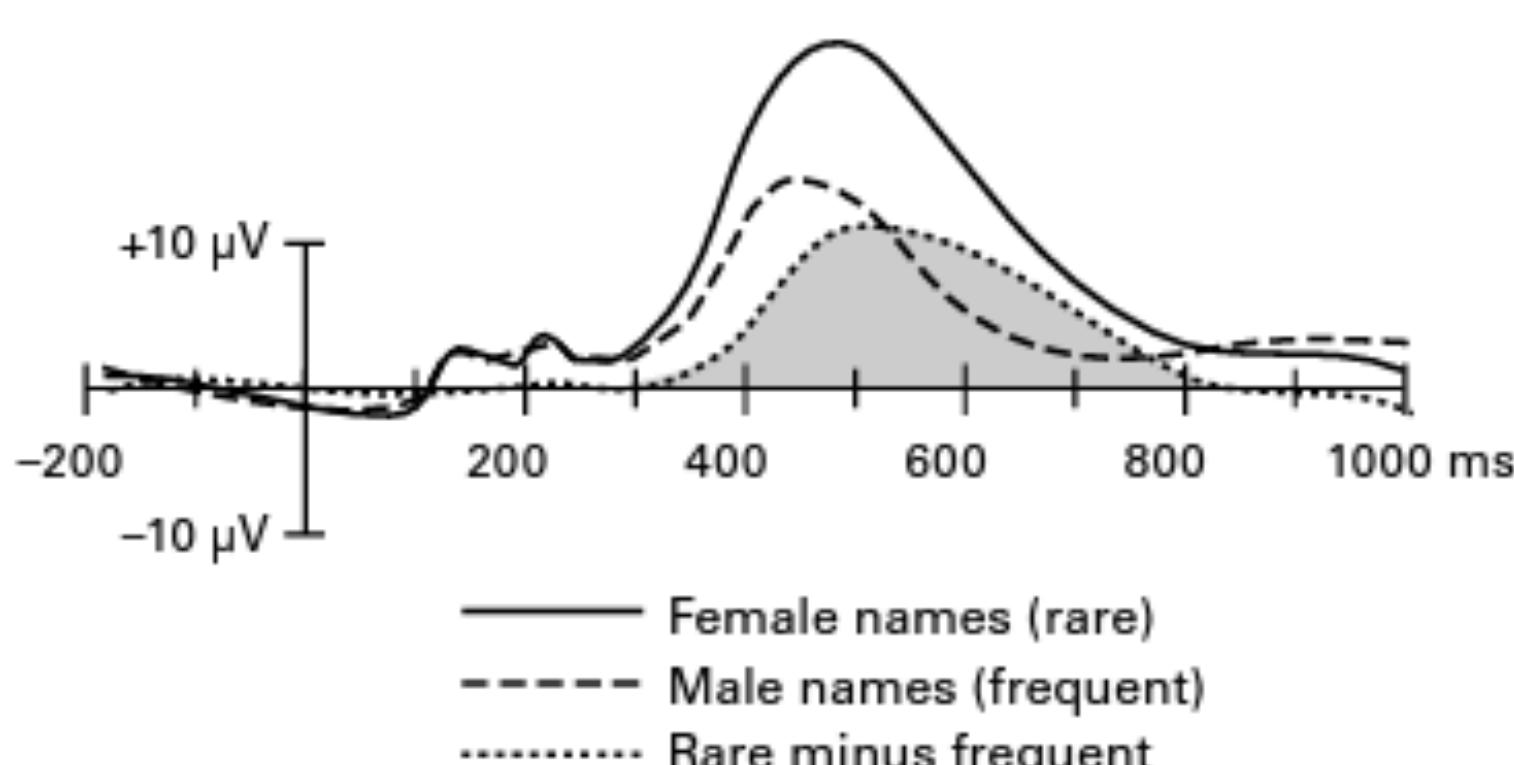


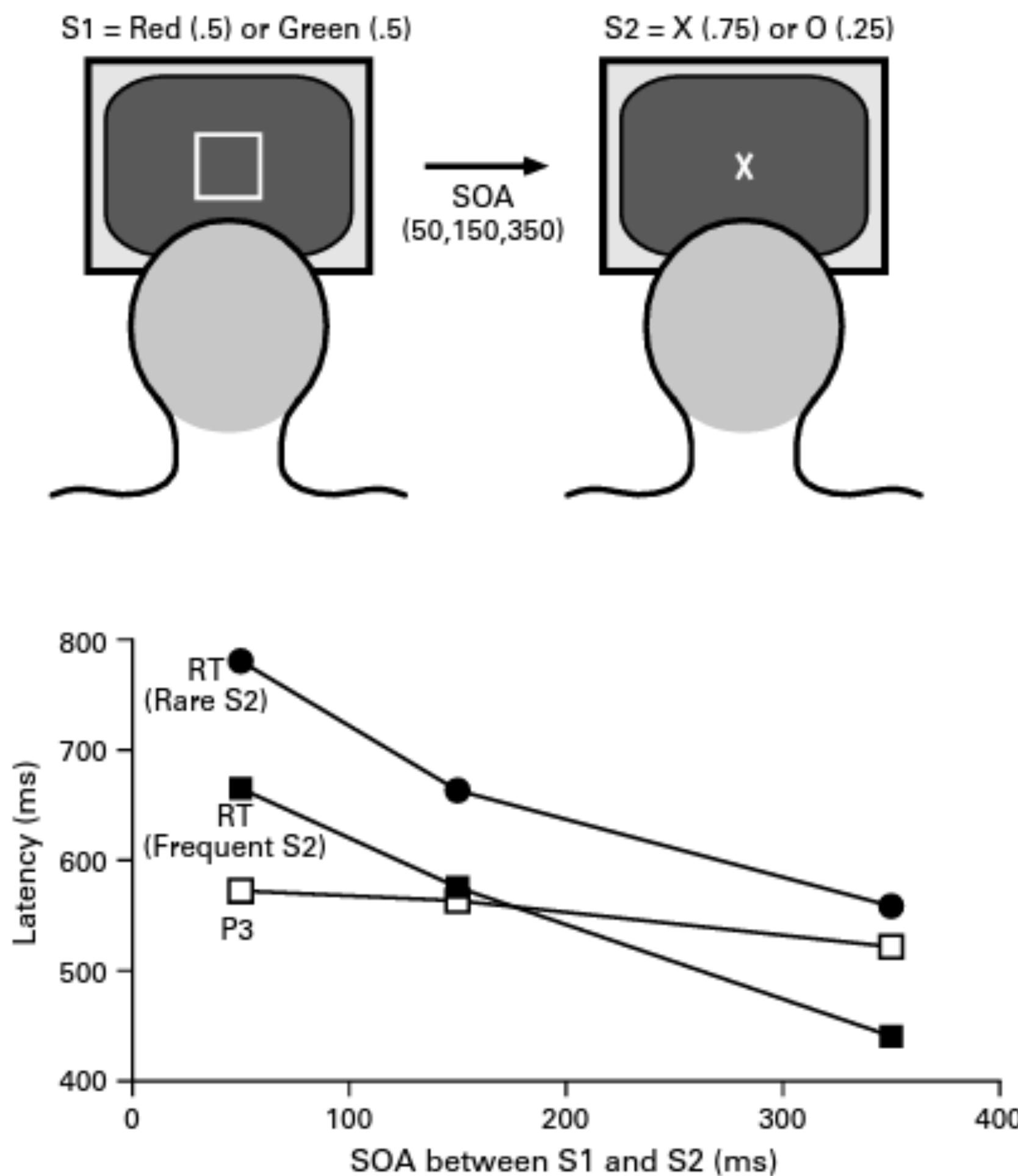
Figure 3.11

Cartoon version of the ERP waveforms from the study of Kutas et al. (1977). These waveforms are not intended to represent the actual waveforms reported by Kutas et al., but instead show the general principle involved in using a rare-minus-frequent difference wave to assess the timing of stimulus categorization.

the onset time of the difference in brain activity between rare and frequent trials. Not only is this logical, it has been confirmed in countless studies. Kutas et al. (1977) framed this general concept in terms of the idea—which was common then but is no longer widely accepted—that the P3 on rare trials reflects surprise, and they noted that, “before a stimulus can surprise it must be identified.” They therefore proposed that P3 latency for rare stimuli reflects *stimulus evaluation time*. The Donchin lab mostly focused on the latency of the P3 peak rather than P3 onset time, and they focused on the ERPs elicited by the rare stimuli rather than focusing on the rare-minus-frequent difference wave. Consequently, their notion that P3 peak latency on rare trials reflects stimulus evaluation time is not quite as precise as the statement that the onset of the difference between rare and frequent trials cannot logically occur until after the brain has begun to determine whether the stimulus falls into the rare category or the frequent category. But these two ways of thinking about P3 latency are very closely related.

When rare-minus-frequent difference waves are used, very strong conclusions can be drawn about whether a given experimental manipulation influenced the amount of time required to categorize a stimulus. An example of this is provided in figure 3.12, which shows the task and results from a study using the psychological refractory period paradigm (Luck, 1998b). In this task, subjects saw two targets on each trial, separated by a variable SOA, and they made speeded responses to both targets. At short SOAs, the brain will still be busy processing the first target when the second target appears, and this delays the response to the second target. Thus, we found that the response to the second target was delayed by hundreds of milliseconds at short SOAs compared to long SOAs. Hal Pashler has theorized that this delay for the second target reflects a postponement of *response selection*, the process of determining which response should be made once the stimulus has been identified (see review by Pashler, 1994). Earlier processes, such as the perception and categorization of the stimulus, should not be delayed at short SOAs. If this theory is correct, then the amount of time required to perceive and categorize the second target should be relatively unaffected by differences in the SOA. The experiment shown in figure 3.12 tested this hypothesis by having one version of the second target be rare and the other be frequent, making it possible to construct a rare-minus-frequent difference wave for the second target. The P3 latency in this difference wave was slowed by only 51 ms at the shortest SOA compared to the longest SOA, which was less than 25% of the total RT slowing seen behaviorally. This result indicated that the RT slowing was mainly due to a slowing of processes that follow stimulus categorization, not stimulus categorization itself, consistent with Pashler’s theory. A related study by Allen Osman and Cathleen Moore showed that the lateralized readiness potential (LRP) was strongly delayed at short SOAs (Osman & Moore, 1993). As will be discussed later in this chapter, the LRP reflects response preparation, so this effect provided further evidence that the slowing of RT was mainly a result of slowed response selection.

This study is related to an important point about the design of ERP experiments (which will be discussed in more detail in chapter 4). Specifically, the P3 component itself does not arise from the process of categorizing the stimulus. Rather, stimulus categorization must occur *before*

**Figure 3.12**

Example of how a rare-minus-frequent difference wave can be used to assess the timing of stimulus categorization in the psychological refractory period paradigm. In this study, subjects saw a red or blue box, followed after a variable stimulus onset asynchrony (SOA) by a second target (an X or an O). One letter was rare and the other was frequent. Subjects were instructed to make a rapid response indicating the color of the box and another rapid response indicating the letter identity. P3 latency and reaction time were measured for the second target (the letter) as a function of the SOA between the first target and the second target. P3 latency was measured as the 50% area latency from rare-minus-frequent difference waves. Adapted with permission from Luck (1998b). Copyright 1998 Association for Psychological Science.

the P3 can be elicited (or, more precisely, before the rare-minus-frequent difference can deviate from zero). Consequently, the latency of the P3 can be used in this manner to assess the timing of processes that must have occurred prior to the time at which the waveforms diverge for the rare versus frequent stimuli. When a component is used in this manner to assess the logically prior cognitive operations, we don't need to know what process the component itself reflects. Thus, many of the most powerful ERP experiments use a given ERP component to study the processes that logically preceded the generation of the component, not the processes that actually generated the component.

P3 and Postcategorization Processes

Although the onset of the rare-minus-frequent difference must logically follow stimulus categorization, this does not tell us whether P3 latency also depends on postcategorization processes. Several studies have provided evidence that P3 latency is sensitive *only* to the time required to perceive and categorize the stimulus and is not sensitive to the amount of time required to select and execute a response once a stimulus has been categorized (see, e.g., Kutas et al., 1977; Magliero, Bashore, Coles, & Donchin, 1984). For example, if subjects press a left-hand button when they see the stimulus LEFT and a right-hand button when they see the stimulus RIGHT, P3 latency is no faster or slower than when they are asked to make a left-hand response for RIGHT and a right-hand response for LEFT (which is known to increase the time required to perform stimulus-response mapping). In contrast, if the stimuli are perceptually degraded, then P3 latency is delayed for these stimuli.

Other researchers have disputed the conclusion that P3 latency is insensitive to manipulations of response-related processes (see review by Verleger, 1997). However, most of the research has not focused on the onset latency of rare-minus-frequent difference waves. For example, consider the study of Leuthold and Sommer (1998), who presented stimuli on the left or right side of the screen (with equal probability) and required subjects to report the side of the stimulus by making a left-hand or right-hand button-press response. In the *compatible* condition, subjects responded with the left hand for the left stimulus and with the right hand for the right stimulus; in the *incompatible* condition, this mapping was reversed. This kind of manipulation is known to primarily influence response selection processes, and it should have little or no impact on the time required to determine which side contains the stimulus. P3 latency was measured by simply finding the peak voltage between 250 and 850 ms in the ERPs elicited by the left-side and right-side stimuli. This peak latency was increased by about 20 ms in the incompatible condition relative to the compatible condition. The problem is that these ERP waveforms presumably contained many overlapping components during this broad time range, so there is no way to know whether this change in peak latency reflected the timing of the underlying P3 component. For example, the effect might have been due to motor potentials during this period, which everyone would expect to vary with stimulus-response compatibility. Moreover, the logic I have described mainly applies to the onset of the P3, not the peak. Thus, the existing evidence provides good support for the proposal that P3 latency—measured as the onset of the rare-minus-frequent difference

wave—reflects the time required to categorize a stimulus and is insensitive to subsequent response-related processes.

P3 and Schizophrenia

Well over a hundred studies have examined the P3 in people with schizophrenia (see meta-analysis by Jeon & Polich, 2003). Most of these studies used simple auditory oddball tasks in which subjects silently counted the oddballs, but many other paradigms have also been used. The peak amplitude of the P3 elicited by the rare (oddball) stimuli is reliably reduced in patients relative to controls, with an average effect size (Cohen's d) of 0.89 for auditory oddball experiments. This is a large effect, and it exhibits excellent stability and reliability, making it a potential biomarker (Luck et al., 2011). Although many interesting and useful conclusions about schizophrenia have been drawn from these experiments, the fact that we do not have a well-accepted theory of the processes reflected by the P3 means that we cannot draw a precise and broadly meaningful conclusion from the simple fact that P3 amplitude is reduced in people with schizophrenia (see box 3.3 for further concerns about the P3 reduction in schizophrenia).

Language-Related ERP Components

Several ERP components are sensitive to linguistic variables (see review by Swaab, Ledoux, Camblin, & Boudewyn, 2012). The best-studied language-related component is the N400, which was first reported by Kutas and Hillyard (1980). The story of the discovery of the N400 is described at the end of the chapter, and the main results from this experiment are shown in figure 3.13.

The N400 is a negative-going wave that is usually largest over central and parietal electrode sites, with a slightly larger amplitude over the right hemisphere than over the left hemisphere. It is typically seen in response to violations of semantic expectancies. For example, if sentences are presented one word at a time on a video monitor, a large N400 will be elicited by the last word in the following sentence: “While I was visiting my home town, I had lunch with several old shirts” (see figure 3.13). Little N400 activity would be observed if the sentence had ended with *friends* rather than *shirts*. The words can be presented in naturally delivered speech rather than in discrete visual words, and the same effects are observed. An N400 can also be observed to the second word in a pair of words if the second word is semantically unrelated to the first. For example, a large N400 is elicited by the second word in “tire . . . sugar” and a small N400 elicited by the second word in “sweet . . . sugar.” This may actually reflect the associative strength between the words rather than the semantic relationship per se (Rhodes & Donaldson, 2008). Some N400 activity is presumably elicited by any content word you read or hear, and relatively infrequent words like *monocle* elicit larger N400s than those elicited by relatively frequent words like *table*.

The N400 (or N400-like activity) can also be elicited by nonlinguistic stimuli, as long as they are meaningful. For example, a line drawing will elicit an N400-like component if it is incon-

Box 3.3

Is It Really the P3?

It should now be apparent to you that many different brain processes contribute to the voltage that we measure in the P3 latency range on rare trials in the oddball task, including processes that are specific to the rare stimuli and processes that are present for both the frequent and rare stimuli. As far as I can tell, researchers have not grappled much with the question of whether the reduced amplitude for rare stimuli in schizophrenia reflects a reduction in the underlying P3 component, a reduction in some other positive component, or even an increase in some negative component. One way to address this question would be to ask whether this effect interacts with target probability. For example, I described an experiment in chapter 1 in which we examined the rare-minus-frequent difference waves in schizophrenia patients and control subjects (Luck et al., 2009). Although the peak voltage in the P3 latency range was reduced on rare trials in patients compared to controls, this voltage was also reduced on frequent trials, and there was no difference in P3 between patients and controls in the rare-minus-frequent difference wave (see figure 1.4 in chapter 1). Similar results were observed in a study by Geoff Potts and his colleagues (Potts, O'Donnell, Hirayasu, & McCarley, 2002). This questions the assumption that the P3 effect in schizophrenia really reflects a bona fide reduction in the underlying P3 component (that is, the probability-sensitive P3b component).

Most oddball experiments in schizophrenia have used auditory stimuli, whereas we had used visual stimuli, so I spent some time reading the literature to see if the rare-minus-frequent difference wave is reduced in schizophrenia patients for auditory stimuli. However, I could not find a single paper that plotted rare-minus-frequent difference waves for the auditory oddball paradigm. In fact, most of the papers I read didn't even report P3 amplitude for the frequent stimulus. However, a few did, and it seems that the P3 elicited by the frequent stimulus is not reduced in amplitude as much as the P3 elicited by the rare stimuli, which implies that the rare-minus-frequent difference is smaller in patients than in controls for auditory stimuli. This still doesn't provide strong evidence that the patient deficit solely reflects a reduction in the amplitude of the underlying P3 component rather than some other component (or a combination of components). In addition, given the fact that the P3 appears to be a largely modality-independent component, the finding that the rare-minus-frequent difference wave is reduced for auditory stimuli but not for visual stimuli in schizophrenia patients suggests that the patient impairment reflects an impairment in auditory-specific processes that provide the input to the P3 rather than an impairment in the P3 generator system itself. This is just a speculation, but it seems plausible given the existing data.

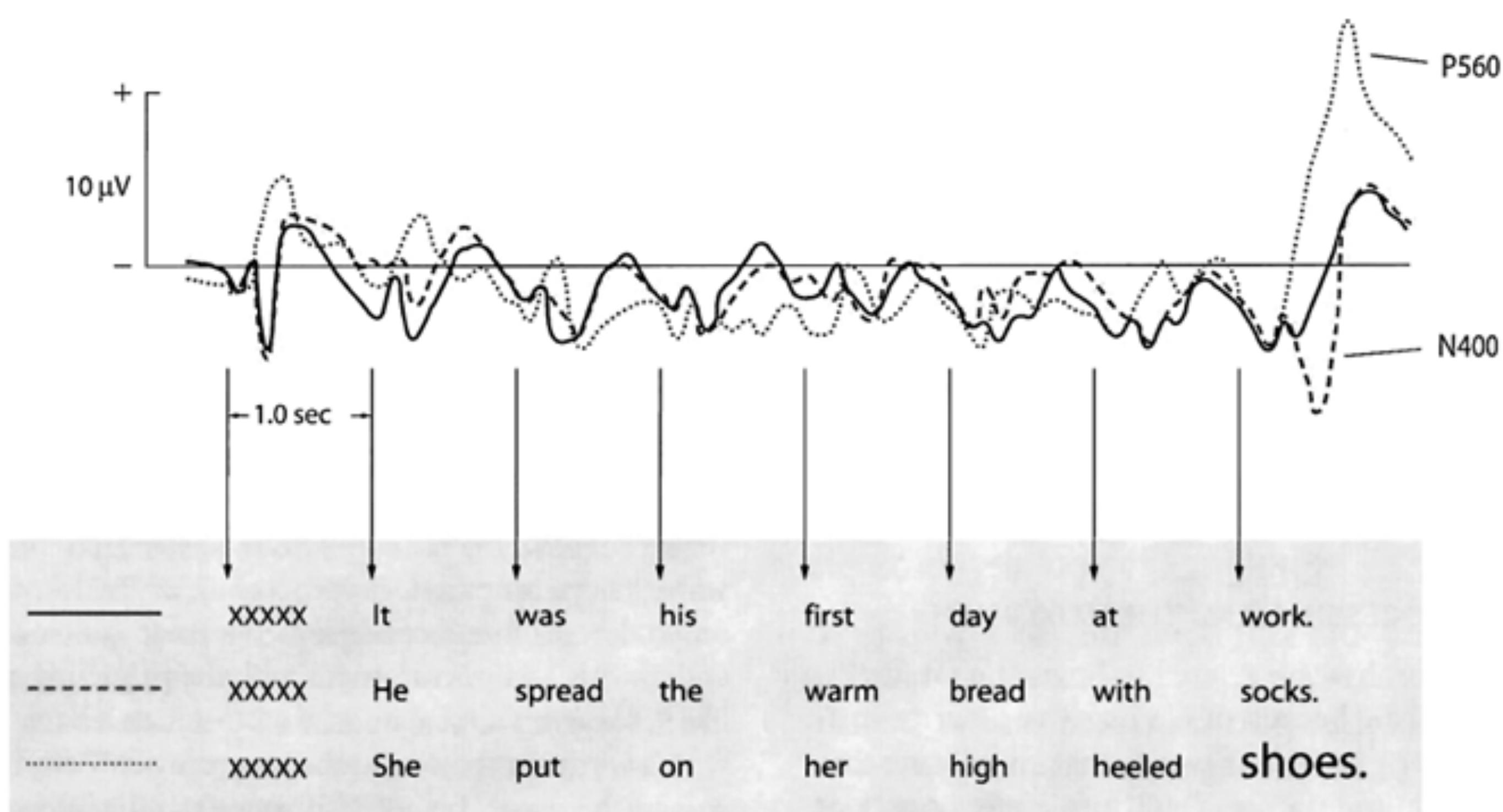


Figure 3.13

Original N400 paradigm used by Kutas and Hillyard (1980). The individual words of a sentence were presented sequentially at fixation, with one word presented per second. The last word of the sentence could be congruent with the meaning of the sentence, incongruent with the meaning of the sentence, or drawn in a larger font. An N400 was observed when the final word was incongruent, whereas as P3 (P560) was observed when the final word was drawn in a larger font. Adapted with permission from Swaab et al. (2012). Copyright 2012 Oxford University Press.

sistent with the semantic context created by a preceding sequence of words or line drawings (Holcomb & McPherson, 1994; Ganis, Kutas, & Sereno, 1996). However, it is possible that subjects in these studies name the stimuli subvocally, so it is possible that the N400 component reflects language-specific brain activity. Moreover, nonlinguistic stimuli typically elicit a more frontally distributed effect than linguistic stimuli (Willems, Özyürek, & Hagoort, 2008).

Although typically larger in right-hemisphere electrodes than left-hemisphere electrodes, the N400 appears to be generated primarily in the left temporal lobe. This apparent discrepancy can be explained by assuming that the generator dipole in the left hemisphere does not point straight upward, but instead points somewhat medially (i.e., upward and toward the right side). Studies of split-brain patients and lesion patients have shown that the N400 depends on left-hemisphere activity (Kutas, Hillyard, & Gazzaniga, 1988; Hagoort, Brown, & Swaab, 1996), and recordings from the cortical surface in neurosurgery patients have found clear evidence of N400-like activity in the left anterior medial temporal lobe (e.g., McCarthy, Nobre, Bentin, & Spencer, 1995). More recent studies also suggest that the left prefrontal cortex may also contribute to the scalp N400 (Halgren, Boujon, Clarke, Wang, & Chauvel, 2002).

Two main theories have been proposed regarding the specific process reflected by the N400. The first, proposed by Marta Kutas and her colleagues, is that the N400 component reflects the neural activity associated with finding and activating the meaning of the word. Because I'm not a psycholinguist, I'll let Marta's own words describe this theory: "Overall, the extant data suggest that N400 amplitude is a general index of the ease or difficulty of retrieving stored conceptual knowledge associated with a word (or other meaningful stimuli), which is dependent on both the stored representation itself, and the retrieval cues provided by the preceding context" (Kutas, van Petter, & Kluender, 2006, p. 669). Thus, the more work required to retrieve the knowledge associated with a word, the larger the N400 will be. The second main theory comes from Peter Hagoort, who proposed that the N400 reflects the process by which the retrieved word meaning is integrated into the preceding discourse (Hagoort, 2007; see also Friederici, Hahne, & Saddy, 2002). The more work required to perform this integration, the larger the N400 will be. Because I'm not a linguist, I do not presume to have an opinion about which theory is more likely to be correct (which may be the only time in this entire book that I do not presume to have an opinion about something).

Syntactic violations also elicit distinctive ERP components. One of these is called *P600* (see Osterhout & Holcomb, 1992, 1995). For example, the word *to* elicits a larger P600 in the sentence "The broker persuaded to sell the stock" than in the sentence "The broker hoped to sell the stock." Syntactic violations can also elicit a left frontal negativity from approximately 300 to 500 ms, which may be the same effect observed when *wh*-questions (e.g., "What is the . . .") are compared to yes–no questions (e.g., "Is the . . ."). Given the important distinction between syntax and semantics, it should not be surprising that words that are primarily syntactic elicit different ERP activity than that elicited by words with rich semantics. In particular, function words (e.g., *to*, *with*, *for*) elicit a component called *N280* at left anterior electrode sites, and this component is absent for content words (e.g., nouns and verbs). In contrast, content words elicit an N400 that is absent for function words.

ERP Components and Long-Term Memory

In addition to the NSW and CDA components that are present in working memory paradigms, several ERP components have been identified that are related to long-term memory (see the review by Wilding & Ranganath, 2012). Separate ERP components have been identified that operate during the encoding and retrieval phases of long-term memory tasks. The ERP elicited by a stimulus during memory encoding will contain many components that are unrelated to encoding. To isolate encoding-related activity, ERP studies commonly sort the single-trial EEG waveforms that were recorded during the encoding phase according to whether or not the stimulus on that trial was later remembered. One averaged ERP waveform is then constructed for trials with stimuli that were later remembered, and another averaged ERP waveform is constructed for trials with stimuli that were later forgotten. Any difference between these ERPs (seen on the remembered-minus-forgotten difference wave) is called a *Dm effect* (difference due

to memory) or a *subsequent memory effect*. In most cases, the Dm effect contains a broad positivity from approximately 400 to 800 ms over centro-parietal electrode sites. However, it may also contain left anterior activity, and the details of the scalp distribution depend on whether the stimuli were words or pictures and on the instructions given to the subjects. Thus, Dm is not a single component, but instead reflects many different processes that can influence whether a stimulus is later remembered.

ERPs can also be examined at the time of memory retrieval by using a recognition task and time-locking the ERPs to probe stimuli that either match a previously studied item (*old* probes) or do not match a previously studied item (*new* probes). Two distinct effects have been observed by comparing the ERPs elicited by old versus new probes. One effect consists of a more negative voltage for new probes than for old probes from 300 to 500 ms, with a maximal voltage at midline frontal electrodes (sometimes called the *midfrontal old-new effect*, and sometimes called *FN400* because it is like a frontally distributed N400). The other consists of a more positive voltage for old probes than for new probes from 400 to 800 ms, with a maximal voltage at left parietal electrodes (called the *left-parietal old-new effect*). The left-parietal old-new effect appears to be associated with what memory researchers call *recollection*, which refers to a clear and distinct experience of the memory that is linked with a particular time and/or place (see review by Yonelinas & Parks, 2007). Some researchers have proposed that the midfrontal old-new effect is associated with *familiarity*, the more diffuse feeling that the probe has been encountered before (e.g., Curran, 2000; Rugg & Curran, 2007). However, Ken Paller and his colleagues have argued that this effect instead reflects a boost in *conceptual fluency*, which is the ease with which meaning is processed and which may be a precursor to familiarity (Paller, Voss, & Boehm, 2007; Voss, Lucas, & Paller, 2012).

Emotion-Related ERP Components

It is difficult to induce intense emotional responses over and over again in a controlled experiment because these responses tend to habituate. Consequently, ERP studies of emotion have tended to focus on how stimuli that are associated with emotions (e.g., photographs of pleasant or unpleasant scenes) are processed differently from relatively neutral stimuli (see the review by Hajcak, Wienberg, MacNamara, & Foti, 2012). It is not clear that the resulting effects reflect emotions that were elicited by the stimuli or, instead, reflect “cold” cognitive responses that are related to the emotional content of the stimuli but are not themselves emotional responses. For example, when I view the stimuli in an emotion ERP experiment and I see a dozen pictures containing snakes over a 10-min period, I don’t feel the kind of fear and revulsion that I experience when I encounter an actual snake while riding my mountain bike on a trail in the woods. My attention is oriented to the picture of the snake, and I realize that it’s something I’d rather not look at, but I don’t feel my heart pounding (in case you can’t tell, I really don’t like snakes). Moreover, it takes a substantial amount of time to have a significant emotional experience, so only the relatively late portion of the ERP waveform is likely to be related to the

phenomenological experience of emotion. But it's still very worthwhile to use ERPs to study emotion-related processes; you just need to make sure that you don't draw unwarranted conclusions about the nature of the processes that are evoked by a long series of photographs in a laboratory experiment.

The emotional content of a photographic image can influence many of the components that have already been described. For example, the P1, N1/N170, N2, and P3 components may all be increased for emotion-relevant stimuli compared to neutral stimuli. Two emotion-related components have been the focus of most research. First, the *early posterior negativity* is a negative potential over visual cortex in the N2 latency range that is enhanced for emotion-inducing stimuli, particularly those with a positive valence (Schupp, Junghofer, Weike, & Hamm, 2003, 2004; Weinberg & Hajcak, 2010). This component is thought to reflect the recruitment of additional perceptual processing for emotion-inducing stimuli. Second, the *late positive potential* (Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000; Keil et al., 2002; Hajcak & Olvet, 2008) is a positive voltage that typically has the same onset time and scalp distribution as the P3 wave (i.e., onset around 300 ms and parietal maximum). It may extend for many hundreds of milliseconds and may become more centrally distributed over time. The initial portion may actually consist of an enlarged P3 component, reflecting an effect of the intrinsic task relevance of emotion-related stimuli.

Error-Related Components

In most ERP studies, trials with incorrect behavioral responses are simply thrown out. However, by comparing the ERP waveform elicited on error trials with the ERP waveform elicited on correct trials, it is possible to learn something about the cause of the error and the brain's response after detection of the error (for an extensive review, see Gehring, Liu, Orr, & Carp, 2012). For example, Gehring, Goss, Coles, Meyer, and Donchin (1993) had subjects perform a speeded response task in which they responded so fast that they occasionally made errors that were obvious right away ("Oops! I meant to press the left button!"). When the ERPs on correct trials were compared to the ERPs on error trials, a negative-going deflection was observed at frontal and central electrode sites beginning just after the time of the response. Gehring et al. called this deflection the *error-related negativity* (ERN), and it was independently discovered by Falkenstein, Hohnsbein, Joormann, and Blanke (1990), who called it N_e (see the end of the chapter for a description of Gehring's discovery of the ERN).

An example set of waveforms is shown in figure 3.14 (from Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). The researchers recorded the electromyogram (EMG) from the thumb in addition to recording button-presses. This makes it possible to define trials that are fully correct (correct thumb button is pressed with no EMG from the incorrect thumb), trials that are fully incorrect (incorrect thumb button is pressed, along with EMG from that thumb), and *partial error* trials (some EMG from the incorrect thumb but without button closure). In ERN experiments, the error-related ERP activity is usually tightly time-locked to the response and more

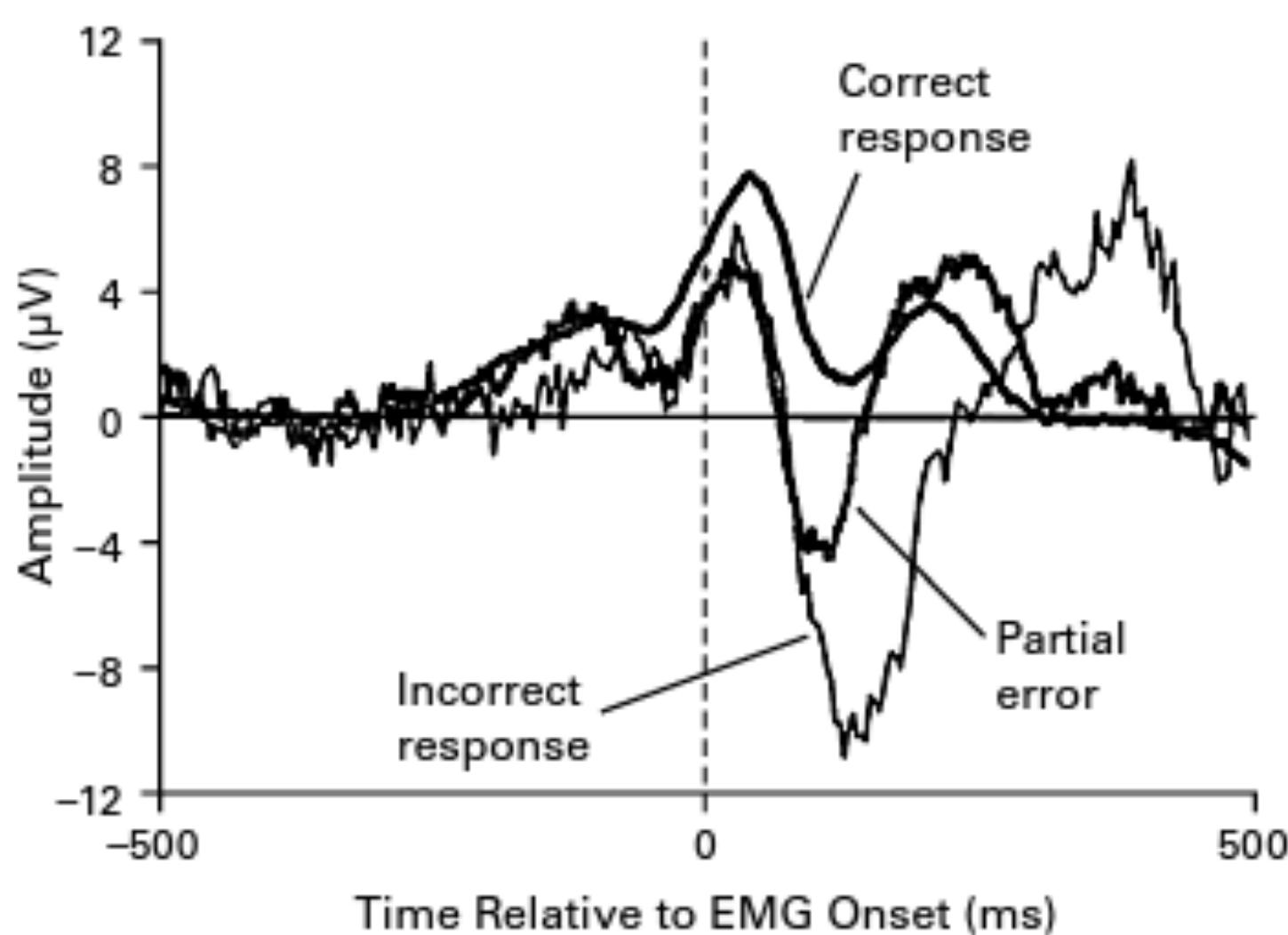


Figure 3.14

Grand average ERPs time-locked to the onset of electromyogram (EMG) activity, which show correct trials, trials with a fully incorrect response, and trials with a partial error (significant EMG without actual button closure). Adapted with permission from Vidal et al. (2000). Copyright 2000 Elsevier.

weakly time-locked to the stimulus, so the ERN is usually visualized in response-locked averages (where the response is defined either by button closure or EMG onset). The waveforms in figure 3.14 are time-locked to EMG onset. The data in this figure illustrate several common findings. First, fully incorrect trials are accompanied by a large negative-going deflection beginning near the time of the response; this is the ERN. Second, the partial error trials are accompanied by a somewhat smaller ERN. Third, a small negative-going potential can also be seen after the response on correct trials, which is termed the *correct response negativity* (CRN). A clear CRN is not always visible because of other overlapping ERPs, but many researchers believe that it is there nonetheless. Fourth, the ERN is followed by a positive deflection, peaking approximately 400 ms after the response in the study shown in figure 3.14. This is called the *error positivity* (P_e). The P_e appears to be associated with awareness of errors, whereas the ERN can occur with little or no awareness of the committed error (Endrass, Reuter, & Kathmann, 2007).

Some complicated technical issues arise in quantifying the ERN. A difference wave approach can be used in which the ERN is quantified as the difference in amplitude between error trials and correct trials. This has the advantage of eliminating any brain activity that is identical on error and correct trials, isolating error-related processes. However, this is not a complete solution. One problem is that the difference may partly reflect a later onset of the P3 wave on error trials than on correct trials rather than a distinct error process. A second problem is that activity prior to the response may differ between error trials and correct trials, distorting the baseline. Both of these problems are related to the fact that the ERN is a correlational effect rather than an experimental effect. That is, the researcher does not control which trials have errors and which trials don't, and some of the differences in the ERPs between correct trials and error trials may

reflect factors that are correlated with errors but not causally related to them. A third problem with difference waves is that a difference across conditions or groups in the strength of the process that generates the ERN may be equally present on correct trials and on error trials. For example, people with obsessive-compulsive disorder appear to have larger ERNs on both correct trials and error trials, perhaps reflecting an aberrant sense of error even when a correct response is made (Gehring, Himle, & Nisenson, 2000). Thus, quantifying ERN amplitude from error-minus-correct difference waves may underestimate the size of increased ERN in these individuals and makes it impossible to test the hypothesis that they also exhibit an enlarged ERN on correct trials. Although difference waves are problematic for quantifying the ERN, it can be even more problematic to measure the ERN from the correct and incorrect trials separately, because that virtually guarantees that the results will be distorted by the many overlapping ERP components (for an extensive discussion, see Gehring et al., 2012).

Most investigators believe that the ERN reflects the activity of a system that monitors responses, is sensitive to conflict between intended and actual responses, or generates emotional reactions depending on responses. Indeed, these processes may be closely interrelated (Yeung, 2004). It is often assumed that the ERN is generated in the dorsal portion of the anterior cingulate cortex (dACC) because fMRI and single-unit studies show that error-related activity is present in this region and because dipole source modeling studies have found that the scalp distribution of the ERN is consistent with a dACC generator location. However, dipole modeling cannot easily distinguish between a single deep dipole and a distributed set of superficial dipoles, and intracranial recordings have found ERN-like responses in multiple cortical areas (Brazdil et al., 2002). Thus, as we have seen with several other ERP components, it is likely that multiple neural sources contribute to the scalp ERN (see discussion in Gehring et al., 2012).

Response-Related ERP Components

If subjects are instructed to make a series of occasional manual responses, with no eliciting stimulus, the responses are preceded by a slow negative shift at frontal and central electrode sites that begins up to 1 s before the actual response. This is called the *bereitschaftspotential* (BP) or *readiness potential* (RP), and it was independently discovered by Kornhuber and Deecke (1965) and Vaughan, Costa, and Ritter (1968). The scalp topography of the readiness potential depends on which effectors will be used to make the response, with differences between the left and right sides of the body and differences depending on which effector is used within a given side (see review by Brumia et al., 2012). The BP/RP can also be observed in tasks that require subjects to make responses to stimuli.

The lateralized portion of the readiness potential is called the *lateralized readiness potential* (LRP), and the LRP has been widely used in cognitive studies (see the review by Smulders & Miller, 2012). As discussed in chapter 2, the LRP is particularly useful because it can be easily isolated from other ERP components. That is, because it is lateralized with respect to the hand making the response, whereas other components are not lateralized in this manner, it is easy to

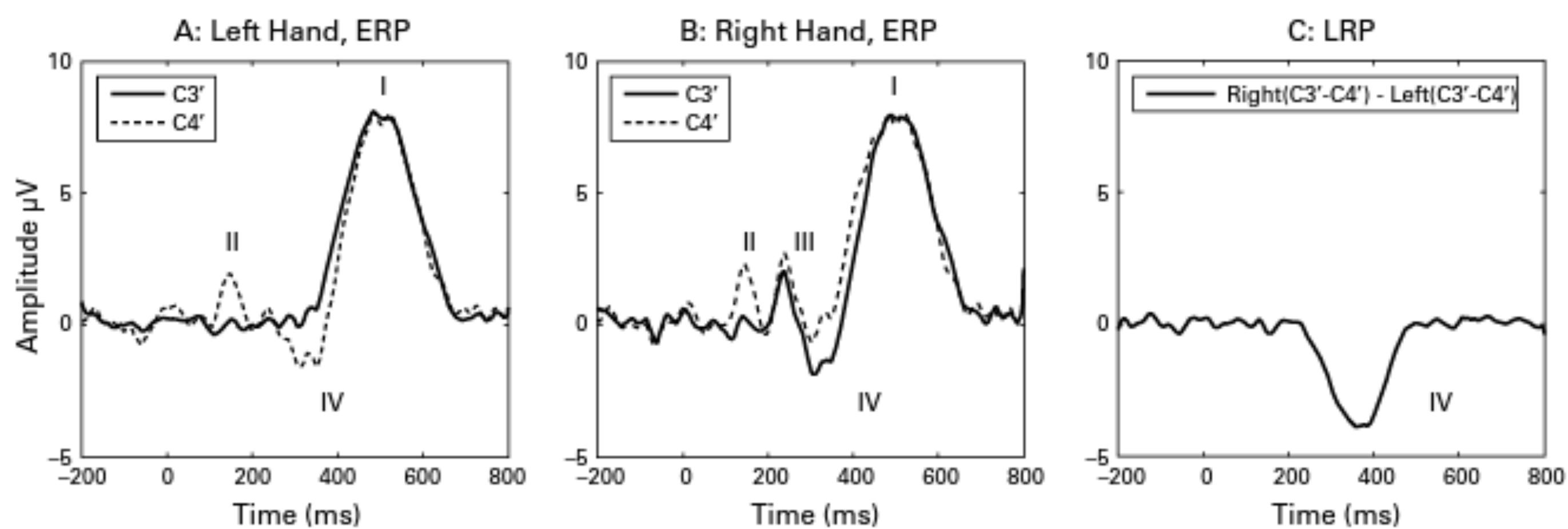


Figure 3.15

Simulated data from a lateralized readiness potential (LRP) experiment in which a stimulus is presented that triggers either a left-hand response or a right-hand response. Adapted with permission from Smulders and Miller (2012). Copyright 2012 Oxford University Press.

tell when a given experimental manipulation has affected the time or amplitude of the LRP. In contrast, it is difficult to be certain that a given experimental manipulation has influenced the P3 component rather than some other overlapping component, and this is one of the main reasons why it has been so difficult to determine what cognitive process is reflected by the P3 component.

Figure 3.15 shows how the LRP is isolated by means of a contralateral-minus-ipsilateral difference wave in an imaginary experiment in which subjects make either a left-hand or right-hand response depending on what stimulus is presented. Panels A and B show the waveforms on left-response and right-response trials, recorded from the C3' and C4' electrode sites (which are just lateral and anterior to the C3 and C4 sites, overlying left and right motor cortex, respectively). There is a large peak (labeled *peak I*) that is the same size irrespective of electrode side or response side (like the P3 wave). There is also a peak (labeled *peak II*) that is larger over the right hemisphere than over the left hemisphere for both left-hand and right-hand responses. Another peak (labeled *peak III*) is larger for right-hand responses than for left-hand responses over both hemispheres. Finally, there is a peak (labeled *peak IV*) that is more negative over the right hemisphere for left-hand responses than for right-hand responses (panel A) and more negative over the left hemisphere for right-hand responses than for left-hand responses (panel B). The LRP is this fourth peak; a greater negativity over the contralateral hemisphere than over the ipsilateral hemisphere (relative to the response hand). The first step in isolating the LRP from the other types of peaks is to compute contralateral and ipsilateral waveforms that combine the two hemispheres. That is, the contralateral waveform is the average of the right hemisphere activity for left-hand responses and the left hemisphere activity for right-hand responses; the ipsilateral waveform is the average of the left hemisphere activity for left-hand responses and the right hemisphere activity for right-hand responses. The second step is to compute the difference between the contralateral and ipsilateral waveforms. As shown in panel C of figure 3.15,

this difference isolates the LRP (peak IV) from the nonspecific (peak I), hemisphere-specific (peak II), and hand-specific (peak III) components. Beautiful! Note, however, that there are two slightly different variations on this approach, one of which doubles the amplitude relative to the other (see Smulders & Miller, 2012).

The LRP is generated, at least in part, in motor cortex (Coles, 1989; Miller, Riehle, & Requin, 1992). The most interesting consequence of this is that the LRP preceding a foot movement is opposite in polarity to the LRP preceding a hand movement, reflecting the fact that the motor cortex representation of the hand is on the lateral surface of the brain, whereas the representation of the foot is on the opposed mesial surface. The LRP appears to reflect some aspect of response preparation: Responses are faster when the LRP is larger at the moment of stimulus onset (Gratton et al., 1988).

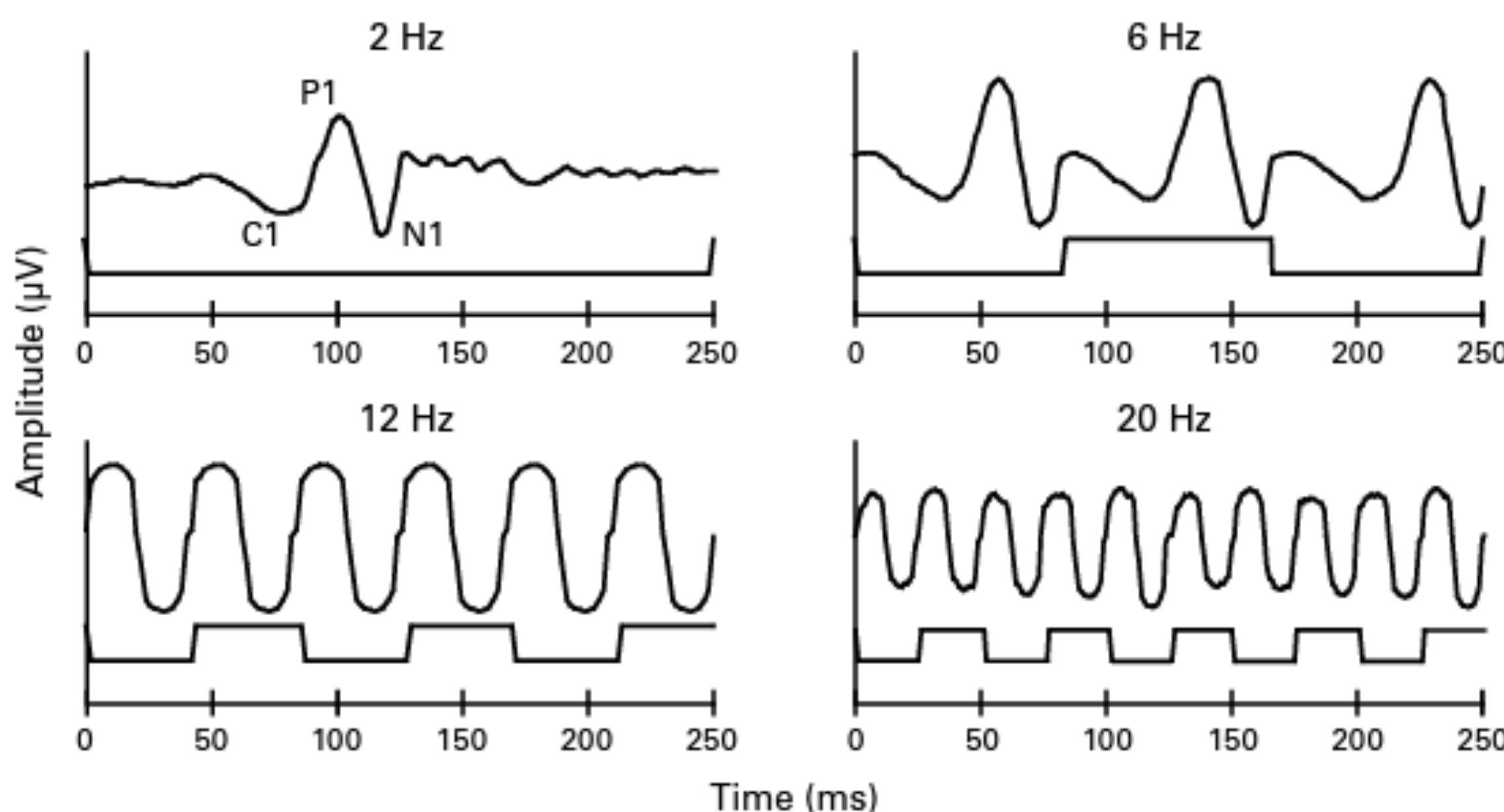
The RP and LRP may be present for hundreds of milliseconds before the response, but other components can be observed that are more tightly synchronized to the response. The early view was that a positive-going deflection is superimposed on the RP beginning 80–90 ms before the response, followed by a negative-going deflection during the response and another positive-going deflection after the response. However, subsequent research identified several additional movement-related components (see, e.g., Shibasaki, 1982; Nagamine et al., 1994).

Steady-State ERPs

In a typical ERP experiment, stimuli are presented at a relatively slow rate so that the brain has largely completed processing one stimulus before the next stimulus is presented. The waveform elicited in this situation is a *transient* response that occurs once and then ends. However, if many identical stimuli are presented sequentially at a fast, regular rate (e.g., 8 stimuli per second), the system will stop producing a *transient* response to each individual stimulus and enter into a *steady state*, in which the system resonates at the stimulus rate. Typically, steady-state responses will look like two summed sine waves, one at the stimulation frequency and one at twice the stimulation frequency.

Examples of transient and steady-state responses are shown in figure 3.16. The upper-left portion of the figure shows the response obtained when the on–off cycle of a visual stimulus repeats at 2 Hz (two on–off cycles per second). This is slow enough that each stimulus onset elicits a transient neural response with multiple distinct peaks. When the stimulation rate is increased to 6 cycles per second (upper-right panel), it is still possible to see some distinct peaks, but the overall waveform now appears to repeat continuously, with no clear beginning or end. As the stimulation rate is increased to 12 and then 20 cycles per second (bottom two panels), the response is predominantly a sine wave at the stimulation frequency (plus a small, hard-to-see oscillation at twice the stimulation frequency).

This steady-state response can be summarized by four numbers; the amplitude (size) and phase (temporal shift) of each of the two sine waves. This is a lot simpler than a transient response containing many different components. In addition, it is possible to collect hundreds of trials in

**Figure 3.16**

Examples of visual responses to repeating stimuli at a rate of 2, 6, 12, or 20 cycles per second.

a very short period of time owing to the fast stimulation rate. As a result, steady-state ERPs are commonly used in the diagnosis of sensory disorders. They have also been used to study the effects of attention on visual processing (Morgan, Hansen, & Hillyard, 1996; Di Russo, Teder-Sälejärvi, & Hillyard, 2003).

Steady-state ERPs have a significant shortcoming, however, which is that they do not provide precise temporal information. For example, if stimuli are presented every 150 ms, the voltage measured at 130 ms after the onset of one stimulus consists of the sum of the response to the current stimulus at 130 ms, the response to the previous stimulus at 280 ms, the response to the stimulus before that at 430 ms, and so forth. Nonetheless, steady-state ERPs are very useful when high temporal resolution is not needed.

The General Problem of Determining the Process Indexed by a Given ERP Component

I have now discussed a large number of interesting and distinctive ERP components, each reflecting a different aspect of perception, attention, memory, language, emotion, or cognitive control. For most of these components, I have tried to make a link between the component and the specific psychological or neural process that the component reflects. However, I will be the first to admit that most of these links are somewhat tenuous. In particular, the lack of a good theory of the functional significance of the P3 wave seems like a notable failure. If you are interested in reading more about the general problem of linking an ERP component with a specific neural or psychological process, see the online chapter 3 supplement.

work and that he shouldn't try it. I suggested to Vince that he collect some pilot data: if the experiment turned out well, he could show the data to Steve; if it didn't turn out well, Steve would never know. Vince collected the pilot data, and when he showed the data to Steve, Steve realized that it would work, and it turned into a beautiful paper (Clark et al., 1995).

This happened with one of my own graduate students, Weiwei Zhang, as well. One day, Weiwei came to me with an idea for a new way to measure the precision of visual working memory. I told him that there were at least four reasons why it would never work and that he shouldn't waste his time with it. He did the experiment anyway (without telling me), and he came back to me a month later with beautiful data. I was convinced by the data, and that experiment became the beginning of a study that was eventually published in *Nature* (Zhang & Luck, 2008). The conclusion is that you should listen when your mentor suggests adding something to your experiments, but you should feel free to close your ears when your mentor tells you something won't work, especially when you believe in your heart that it will.

The Discovery of the CDA

After completing his dissertation in my lab at the University of Iowa, Ed Vogel went to UCSD to do a postdoc with Steve Hillyard. Ed's dissertation included some behavioral experiments that compared the effects of attention on perceptual encoding and on working memory encoding. In the Hillyard lab, Ed ran an ERP version of these experiments to see whether the P1 was influenced by attention under these conditions. He initially examined the first few hundred milliseconds after the stimulus to look at the P1 wave. The P1 effects were weak, but he saw a big difference between the contralateral and ipsilateral sites toward the end of this time window. As Ed tells the story:

After being underwhelmed by the P1 data, I went back and re-averaged to include the whole retention interval to take a peek at that late effect. At the time it felt like blasphemy to even look at ERP activity that late into the trial. I remember being skeptical about anything that was later than the P3. But there it was. Plain as day.

Ed showed the data to Steve Hillyard, but Steve is a dedicated attention researcher and wasn't interested in pursuing a working memory effect. As Ed told me:

I decided to pocket the effect and chase it down once I had my own lab. Which I did about 6 months later in Oregon. They were the absolute first experiments I ran once my lab was operational.

The first experiment on how CDA was modulated by set size was done in spring 2003. I was going to the Cognitive Neuroscience Society meeting in New York City, and I had my new master's student (Maro Machizawa) collect the data while I was away. Because it was a new experiment and a new student flying solo, I told him to just run one or two subjects while I was gone and that we'd look over the data together when I returned. That way, if there was a problem with the data (e.g., lots of artifacts, missing event codes, etc.) it would be a minimal loss. When I got back to Oregon, I was absolutely irate to find out that he'd run 12 subjects and hadn't looked at any of the data. So for all we knew, the data were noisy or missing event codes and would be unusable. Fortunately the data were clean and showed the first of many demonstrations of the set size modulation and asymptote of the CDA.

I'd like to point out that Ed was right about not running a large number of subjects before checking to make sure everything was okay. There are so many things that can go wrong with an ERP experiment that you need to do a reasonably complete set of analyses on the first few subjects to make sure that you have all the right event codes, that there is nothing weird about the data, and so forth. I have seen many experiments that had to be re-run because of a problem that could have been detected by a closer look at the data from the first few subjects. In this particular experiment, Ed and Maro got lucky and everything turned out okay. In fact, this became the first experiment in a paper that was published in *Nature* (Vogel & Machizawa, 2004). But you should still do a full set of analyses (including behavioral analyses) after the first few subjects to make sure there aren't any problems.

The Discovery of the N400 Component

I asked Marta Kutas to tell me about the discovery of the N400, and here is the story she told me:

The discovery of the N400 was a result of a "failed" P3 experiment that I ran in 1978 when I was a postdoc in Steve Hillyard's lab. We were interested in language, but almost all language ERP experiments up to that point had been oddball experiments with single words, in which participants were asked to make a binary decision about each word (e.g., male versus female). Our goal was to extend the oddball paradigm to sentence materials so that we could use P3 latency to investigate the role of context on word recognition.

Steve and I designed an initial experiment using an oddball paradigm with simple sentences—75% of the sentences were ordinary and meaningful and ended in a relatively predictable way (e.g., "He shaved off his mustache and beard."), but a random 25% of them were oddballs and ended with an interpretable but surprising word (e.g., "He shaved off his mustache and eyebrows."). The standard stimuli were further subdivided into idioms or proverbs (e.g., "A bird in the hand is worth two in the bush.") and facts ("The capital of California is Sacramento.") for which only one final word was acceptable, as well as more open-ended (less contextually constraining) sentences for which multiple endings were possible (e.g., "He returned the book to the library.").

In the long run, the plan was to examine how P3 latency changed as a function of variations in contextual constraint. However, we gave up on that plan because the ERPs elicited by the final words of the oddball sentences were not quite what we expected. Rather than a large P3, oddball words elicited a small negativity followed by a positivity. We thought the negativity might have been an N2, but it was later than usual; the positivity could have been a P3, but we weren't sure. Thus, we went back to the experimental drawing board. After some head scratching, we decided that we should shake the system as much as we could by presenting sentence-final words that were not just improbable and unexpected but semantically anomalous (e.g., "He shaved off his mustache and city." or "I take my coffee with cream and dog."). This was before computers were readily available for presenting stimuli, so it took quite a bit of work to change the experiment. We typed the each word in the middle of a frame the size of a slide, photocopied the words onto clear plastic sheets, cut the sheets into small rectangles, mounted them in slide holders, and then projected them using a slide projector.

The results of this new experiment were much clearer—the anomalous word elicited a large centro-parietal negativity peaking around 400 ms. This negativity (rather than the positivity, whether or not it was a P3) seemed to be where the action was. The rest is N400 history.

An important lesson of this story is that when you find an unexpected result in a complicated experiment, you should conduct a much simpler follow-up experiment so that you can see this new result more clearly. If Marta had just published her original experiment without running the now-classic follow-up experiment, it may have been years before anyone realized that a distinct and important new ERP had been discovered. As Bob Galambos would say, “you’ve got to get yourself a phenomenon” (see box 4.2 in chapter 4).

The Discovery of the ERN

The ERN was independently discovered by Michael Falkenstein and by Bill Gehring. I asked Bill how he happened to discover the ERN, and he told me that he first saw it in the context of a categorization experiment that he ran when he was in graduate school (Gehring, Coles, Meyer, & Donchin, 1995). In this experiment, two words were simultaneously presented, one above the other, and subjects were instructed to squeeze a response device with one hand if the upper word was a member of the category indicated by the lower word (e.g., *robin* above *bird*) and to squeeze with the other hand if not (e.g., *spoon* above *bird*). Although the experiment had been designed to look at the LRP, Bill found some interesting differences in N400 between these trial types. He wanted to know if they were related to task performance, so he did a variety of comparisons between correct trials and error trials. He initially did this for stimulus-locked averages (figure 3.17), but there were not any impressive differences between correct and incorrect trials. In retrospect, the lack of a large ERN in these waveforms probably reflected a large amount of RT variability, causing any activity related to the correctness of the response to be smeared out over a long time period.

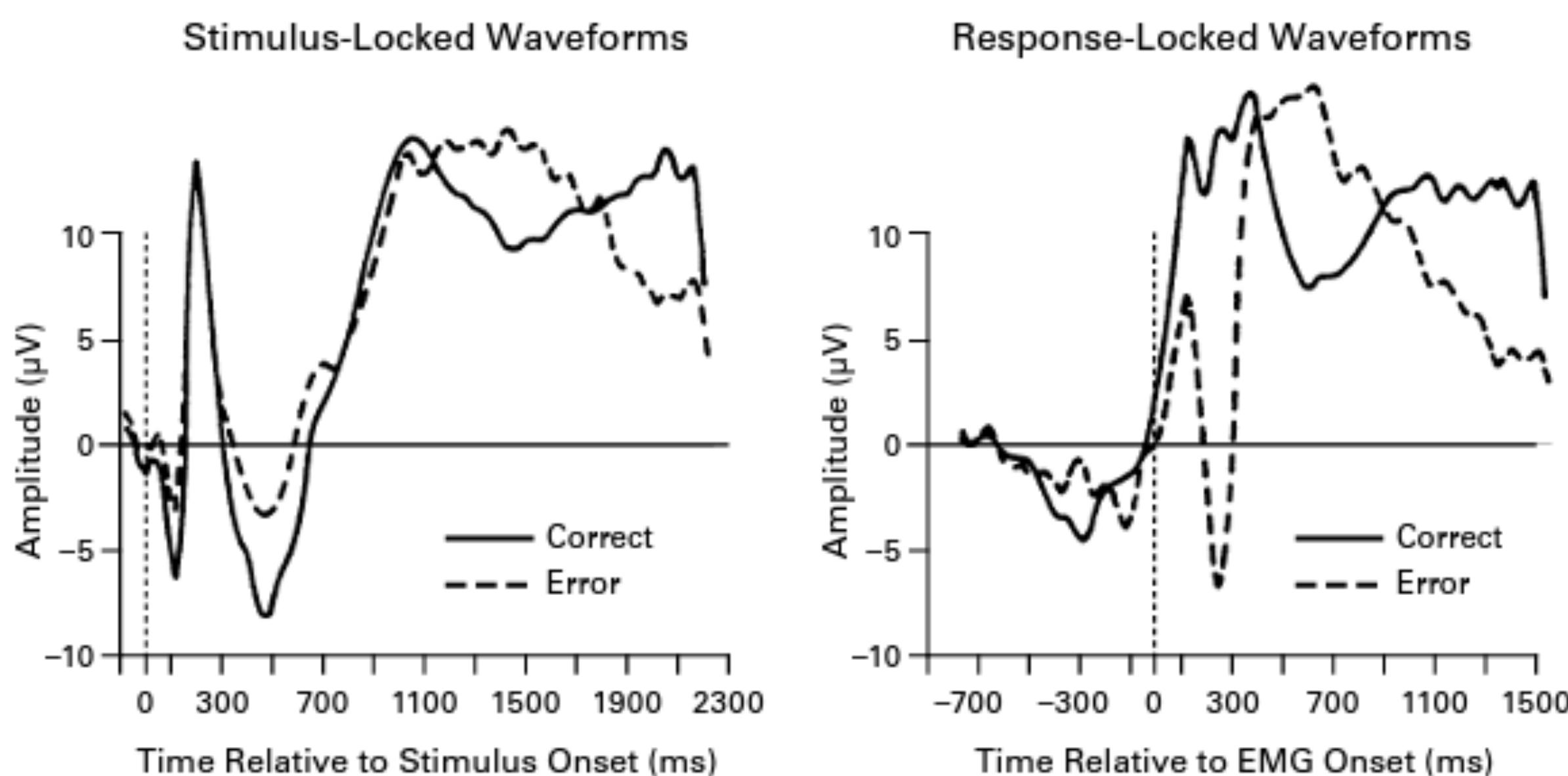


Figure 3.17

Stimulus-locked and EMG-locked ERPs from the experiment in which Bill Gehring first observed the error-related negativity (ERN). Courtesy of Bill Gehring.

Bill might have given up on the ERN at that point except for a conversation he had with Marta Kutas. Bill was a graduate student in the famous Cognitive Psychophysiology Laboratory (CPL) at the University of Illinois, where many classic ERP studies have been conducted over the decades. Marta Kutas had done her Ph.D. work at the CPL many years before, and she had coffee with Bill one day when she was back at the CPL for a visit. As Bill told me:

She suggested looking at response-locked data: that N400/performance relationships might come out better if we looked at N400 in the response-locked waveforms. So that's where the ERN popped up.... I remember vividly when I first saw the ERN: our Harris computer green CRT displays plotted the waveforms out one pixel at a time, so you could see it draw the waveform in bright green from left to right. It sort of looked liked an oscilloscope plotting out the waveform in green against a black background. First the correct waveform emerged, and then the error trials were plotted out, and at the time of the response a huge peak happened—it looked just like a big blip on an oscilloscope. (For a while in the lab we nicknamed the ERN the “blunder blip.”) I’m sure I had a big P3 at just that moment. The ERN was so huge, in fact, that we were worried it was some kind of artifact, so I spent the better part of the next couple of years re-analyzing other data sets to see if the ERN was also in those data.... One of the reasons it took us so long to publish the data was because we had decided to look at all of the data we could to make sure we were dealing with something real.

The response-locked waveforms are shown in figure 3.17, and you can see that the ERN was quite impressive in these waveforms.

I love this story because a graduate student made an important discovery after following a suggestion from a more senior researcher, and I also love it because the results were not published for several years because the graduate student wanted to make sure it was real. This is just what happened with the discovery of the N2pc.

Suggestions for Further Reading

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