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The N1 Wave of the Human Electric and Magnetic Response to Sound: A Review and an Analysis of the Component Structure

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ABSTRACT

This paper reviews the literature on the N1 wave of the human auditory evoked potential. It concludes that at least six different cerebral processes can contribute to the negative wave recorded from the scalp with a peak latency between 50 and 150 ms: a component generated in the auditory cortex on the supratemporal plane, a component generated in the association cortex on the lateral aspect of the temporal and parietal cortex, a component generated in the motor and premotor cortices, the mismatch negativity, a temporal component of the processing negativity, and a frontal component of the processing negativity. The first three, which can be considered "true" N1 components, are controlled by the physical and temporal aspects of the stimulus and by the general state of the subject. The other three components are not necessarily elicited by a stimulus but depend on the conditions in which the stimulus occurs. They often last much longer than the true N1 components that they overlap.

DESCRIPTORS: Auditory evoked potentials, Magnetic responses, Event-related potentials, Components, N1 wave, Mismatch negativity, Processing negativity.

Almost a half-century ago, P.A. Davis (1939) described the sound-evoked changes in the electroencephalogram of the waking human brain. The onset of a tone elicited a negative-positive wave that was larger at the vertex than at the occipital, frontal, or temporal regions of the scalp. A similar evoked potential (EP) occurred following the offset of the tone which lasted "a few seconds." In the published figures, the peak latency of the negative wave varies from 100 to 150 ms. The response was not specific to the auditory modality but could also be elicited by the onset of a light or the onset of a train of electric shocks to the finger (H. Davis, P.A. Davis, Loomis, Harvey, & Hobart, 1939). In sleep the response was much larger and was very different in waveform, adding several other activities to the vertex EP to form a K-complex (Davis et al., 1939). One of the most prominent parts of this complex was a large vertex-negative wave beginning after 100 ms, peaking at 300–500 ms, and followed by an abrupt return to positivity at about 750 ms. The early studies of the vertex potentials (Bancaud, Bloch, & Paillard, 1953; Gastaut, 1953; Roth, Shaw, & Green, 1956) considered the waking and sleeping responses to represent the same process. The widespread scalp distribution of the response suggested to Roth et al. that it resulted from a diffuse projection system mediating arousal; the vertex maximum suggested to Gastaut that it might be generated in the cingulate cortex, a suggestion that received some support from intracranial recordings of the K-complex in monkeys (Hughes & Mazurowski, 1964).

The advent of averaging techniques allowed the early sensory EPs, which varied in their scalp distribution with the modality of the stimulus, to be distinguished from the late vertex potentials which

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appeared to have a similar scalp distribution across modalities (Gastaut, Regis, Lyagoubi, Mano, & Simon, 1967). The vertex response had a prolonged refractory period (Allison, 1962) and there were interactions between the responses to stimuli in different modalities (Walter, 1964). These characteristics suggested that the vertex potential represented a nonspecific response to sensory change mediated by diffuse cortical projections particularly to the frontal cortex. Davis and Zerlin (1966) proposed that this vertex potential may represent a "response related to a change of state rather than a channel for incoming sensory information" (p. 116). Walter (1964) suggested that the vertex potential notified the brain that something was happening, while the specific sensory areas determined what this was.

In recent years, the concepts of the vertex potential have changed. Many other potentials—in the auditory system in particular—are maximally recorded at the vertex. The term "vertex potential" is therefore no longer helpful. Since similarity in scalp distribution does not necessarily entail similar generators (Picton, Woods, Stuss, & Campbell, 1978d), the vertex response may not have the same cerebral source in the different modalities. Nevertheless, the idea that a similar process may be involved in all modalities remains: "the sequence of components beginning with the vertex potential seems similar in all modalities and may reflect similar neural operations, though not necessarily in the same cerebral region" (Allison, Matsumiya, Goff, & Goff, 1977, p. 194).

The vertex response may not represent a single cerebral event since the different waves of the response respond differently to experimental manipulation and have different scalp distributions. Our present nomenclature (Davis & Zerlin, 1966) therefore separately identifies the different waves by polarity and sequence—P1, N1, P2, and N2. In the auditory modality the latencies of the N1 and P2 peaks are about 100 and 175 ms, respectively; in the visual and somatosensory modalities the latencies are 30–40 ms longer. Another nomenclature identifies the waves by their polarity and their usual latency—N100 and P175 would be equivalent to N1 and P2, respectively, in the auditory response.

The EP consists of a sequence of positive and negative waves or peaks. Although these deflections in the waveform provide a convenient point for measurement, they are not necessarily generated by individual cerebral events. At any point in time, multiple cerebral processes may contribute to the EP waveform. In this article we define an EP "component" as the contribution to the recorded waveform of a particular generator process, such as the activation of a localized area of cerebral cortex by a specific pattern of input. A component is therefore related to the "source potential" defined by Scherg and von Cramon (1986a) as "the compound local activity of a circumscribed brain region." Whereas the peaks or deflections of an EP can be directly measured from the averaged waveform, the components contributing to these peaks can usually be inferred only from the results of experimental manipulation. Donchin, Ritter, and McCallum (1978) defined a component in the event-related potential (ERP) as "a source of controlled, observable variability." Our definition is similar with respect to how the components are determined, but it limits the component by referring it back to a localized physiological activity.

In this paper we review the literature on the N1 wave of the sensory EP. Our review concentrates on the auditory modality although we refer occasionally to the visual and somatosensory responses. The goals of the review are to summarize and evaluate the available information on the auditory N1 wave, and to use this information to suggest the cerebral processes which generate this scalp-recorded wave.

As will become evident, many different processes generate negative waves during the latency of the N1—between 50 and 200 ms after the onset of an auditory stimulus. We shall distinguish three "true" or "obligatory" components that are mainly controlled by the physical and temporal features of the stimulus and by the general state of the subject. These components can be distinguished from each other by their different source locations within the brain, and by their different sensitivity to stimulus features and state factors. Unfortunately, most of the N1 literature does not easily permit an analysis or reconstruction of the data in terms of different components. We therefore often refer to the "N1 wave" or "N1 deflection" or "N1 response" without regard to its component structure. Where possible we report baseline-to-peak measures of the amplitude of this wave, although in much of the early literature only N1-P2 measurements were used. Although covarying with the N1 in many dimensions, the P2 may be quite independent (Knight, Hillyard, Woods, & Neville, 1980; Roth et al., 1976; Vaughan, Ritter, & Simson, 1980).

In addition we shall distinguish three other negative components which are not obligatorily evoked by a stimulus but which depend upon the conditions under which the stimulus occurs. These components overlap the true N1 components and often have a considerably longer duration. One of these components, the "mismatch negativity," is not elicited by any particular stimulus but occurs only when a stimulus differs from those preceding it in a ho-

mogeneous sequence. The remaining two components are elicited during selective attention as components of the "processing negativity."

Component Structure and Cerebral Sources

Specific and Nonspecific Responses

The cerebral origins of the auditory N1 wave have generated, in addition to the scalp negativity, much dispute in the literature. In the sixties, the common view was that the N1 represented a nonspecific cerebral process. This view was supported by the similarity of the response across modalities (Gastaut et al., 1967; Goff, Rosner, & Allison, 1962), the widespread scalp distribution centered around or slightly anterior to the vertex (Goff, Matsumiya, Allison, & Goff, 1969; Picton, Hillyard, Krausz, & Galambos, 1974), the interactions between stimuli in different modalities (Davis, Osterhammel, Wier, & Gjerdingen, 1972), and the susceptibility to changes in state (Fruhstorfer & Bergström, 1969; Larsson, 1956, 1960a, 1960b; Näätänen, 1967).

The cerebral source of the scalp-recorded response remained unknown. The general belief was that the response was mediated by nonspecific areas of the thalamus (Jasper, 1960; Lindsley, 1969; Morison & Dempsey, 1942). On the basis of extensive intracranial recordings, Walter (1964) suggested that the N1-P2 represented the widespread activation of the prefrontal regions of the cortex. Fruhstorfer (1971) postulated that the extralemniscal pathways of the bulbar reticular formation and the centrum medianum of the thalamus transmitted the response to the cortex. Large potentials similar to the vertex potential can be recorded from many areas of the thalamus (Ervin & Mark, 1964; Velasco & Velasco, 1986; Velasco, Velasco, & Olvera, 1980, 1985). Since they were able to record the response after hemispherectomy, Saletu, Itil, and Saletu (1971) postulated that the response was actually generated in the brainstem. Another possible nonspecific generator was the cingulate gyrus (Chatrian, Canfield, Knauss, & Lettich, 1975; Gastaut, 1953).

Vaughan and Ritter (1970), however, proposed that the auditory vertex response was generated in the primary auditory cortex on the superior aspect of the temporal lobe. The activation of a region of cortex can separate charges into a dipole layer with a potential gradient perpendicular to the cortical surface. Such a dipole is recordable at a distance from the cortex because the similar orientation of the cortical neurons results in an "open" field (Lorente de No, 1947). A dipole located in the primary auditory cortex would be oriented vertically. The surface potentials generated by such a dipole would be of one polarity at the vertex or frontal regions of the scalp and of the opposite polarity below the level of the auditory cortex. Vaughan and Ritter recorded the N1-P2 waveform along a coronal chain of electrodes using the nose as a reference, and found an inversion of phase over the Sylvian fissure. These responses were evoked by brief 1000 Hz tones presented every 2 s. The responses to tones presented at an irregular interval averaging 45 s were quite different in morphology and scalp distribution. These responses contained a large positive wave with a latency of 300 ms. Since peak-to-peak measures were used it is difficult to determine whether the N1 wave in this response had a similar scalp distribution to that in the response to the more rapidly presented stimuli.

Vaughan and Ritter's interpretation of the N1-P2 generator was soon questioned by Kooi, Tipton, and Marshall (1971) who found no evidence of a polarity reversal across the Sylvian fissure when the potentials were recorded using a noncephalic sternovertebral reference (Stephenson & Gibbs, 1951). They suggested that the nose was not an inactive reference but could pick up activity from the frontal pole. If this activity were similar in morphology to and smaller in amplitude than that picked up from the vertex, an apparent polarity inversion would result when it was subtracted (by the differential amplifier) from the little or no activity picked up in the temporal and mastoid regions. The results of Kooi et al. (1971) were confirmed by Picton et al. (1974), who suggested that the N1-P2 response was mainly generated in the association areas of the frontal cortex. These areas could be activated by projections from the medial thalamus or by corticocortical connections. Several other papers (Picton, Woods, Stuss, & Campbell, 1978; Streletz, Katz, Hohenberger, & Cracco, 1977; Wolpaw & Wood, 1982) have concluded that the sternovertebral reference location is preferable to the nose. Vaughan (1974), however, has argued that an inverted vertex potential can be recorded in attenuated form from the sternovertebral reference which lies below the vertically oriented temporal dipole. Furthermore, it is possible to record some degree of polarity reversal across the Sylvian fissure even with a sternovertebral reference (Lehtonen & Koivikko, 1971; Wood & Wolpaw, 1982).

Using the nose as a reference, Peronnet, Michel, Echallier, and Girod (1974) recorded auditory EPs from a coronal chain of electrodes running from right to left mastoids through the vertex. They reported three findings that were difficult to reconcile with the idea that a nose reference, by picking up an auditory response similar to that at the vertex although smaller in amplitude, was entirely responsible for the polarity reversal over the Sylvian

fissure. First, although the responses were symmetrical for right-ear stimulation, the right-mastoid response was larger than the left when the left ear was stimulated. This asymmetry could not be explained by an active nose reference. Second, the visual response showed no polarity reversal over the Sylvian fissure. If the visual and auditory vertex responses were both nonspecific and recorded in attenuated form at the nose, an apparent polarity reversal should have occurred in both modalities. Third, as we shall discuss later, the topography of the auditory EP showed clear asymmetries in patients with unilateral lesions of the temporal lobe.

In interpreting the scalp distribution of the N1 it is essential to bear in mind the location of the reference electrode. The most commonly used locations are the earlobe and mastoid. An electrode at either of these locations can pick up activity from the lateral and inferior aspects of the temporal lobe. An electrode on the nose can pick up activity from the frontal pole. A chest reference can pick up activity from the base of the brain. Different assumptions about the reference electrode may lead one to infer different sources for a particular scalp-recorded field (Lehmann, 1986). Some knowledge of the intracranial fields may help decide between these alternate interpretations.

Intracranial Recordings

Although intracranial recordings in animals have provided important additional information, they have not completely resolved the location of the sources of the scalp-recorded N1 wave. Depth-electrode recordings in alert rhesus monkeys (Arezzo, Pickoff, & Vaughan, 1975) showed that the late responses (N70 and P110) recorded epidurally over the frontal and central cortex were mainly volume-conducted from the supratemporal plane. These findings support their interpretation of the human scalp recordings, provided that the responses are truly homologous and the intracranial geometries are similar. Two other sources were active at these latencies: the lateral surface of the superior temporal gyrus and a region of the frontal motor cortex near the arcuate sulcus. These monkey responses were evoked by clicks presented every 0.6 s. Similar results were obtained for tones or speech sounds presented every 0.7 s (Steinschneider, Arezzo, & Vaughan, 1980).

Earlier recordings from squirrel monkeys using interstimulus intervals (ISIs) of 10 s (Hardin & Castellucci, 1970) had suggested that the late waves of the auditory and somatosensory EPs originate in the frontal cortex. However, the data presented in their paper are extremely difficult to interpret. They show that transcortical recordings (between the dura and the white matter) of the auditory evoked potential are inverted in polarity from, and approximately equal in amplitude to or slightly larger than, recordings obtained from the white matter using a nose reference. If the potentials were generated completely in the cortex, the transcortical recordings should have been about twice as large as the white matter recordings. The results therefore suggest that there is volume conduction from deeper structures as well as a local origin in the frontal cortex.

Intracranial recordings from human patients have also failed to localize definitively the N1 generator. Intraoperative recordings from the surface of the human temporal lobes (Celesia, 1976) have shown auditory EPs that are not easily related to the vertex N1. In response to clicks presented at a rate of 1/s, Celesia recorded two prominent positive waves from the superior surface of the temporal lobe at latencies of 15 and 32 ms. The later components of the response were smaller and more variable, the most consistent wave being a negative peak at 62 ms. On the lateral surfaces of the temporal, parietal, and frontal lobes in the perisylvian region a different response could be recorded with a prominent positive wave at 40 ms. The later parts of this response showed a positive wave at 98 ms and a negative wave at 142 ms. The laterally recorded response was more susceptible to anesthesia than the response from the superior surface of the temporal lobe.

Depth recordings in human subjects (reviewed by Wood et al., 1984) have demonstrated no clear inversion of the polarity of N1 across the frontal cortex and only occasional inversions as electrodes pass from above to below the plane of the auditory cortex. The striking changes in the waveform between different intracranial locations suggest separate generators with overlapping fields. Overlap can certainly cause significant problems in interpretation. For example, if the supratemporal plane and the frontal cortex are simultaneously active and if their fields are similarly oriented (with negativity above the cortex and positivity below), the field spread from the supratemporal plane could attenuate any evidence of polarity reversal across the frontal cortex. Furthermore, local fields in the region of the thalamus might similarly obscure intracranial evidence of phase reversal across the level of the Sylvian fissure.

Velasco et al. (1985; Velasco & Velasco, 1986) have recently reported subcortical correlates of the vertex responses. They presented auditory, visual, and somatosensory stimuli randomly at an ISI of 4 s. The auditory stimulus was a brief 1000 Hz tone 60dB above a background white noise. Recordings

were obtained during stereotactic operations on patients for Parkinson's disease, epilepsy, or chronic pain, using depth electrodes. Bipolar recordings (Velasco et al., 1985) of the auditory responses indicated rapid changes in potential over very short distances in several subcortical areas: the mesencephalic reticular formation, the medial and dorsal thalamus, the striatum, and the limbic system. These findings suggest sources close to the recording electrodes. No late responses were recorded in the specific sensory nuclei of the thalamus, which showed only early modality-specific waves. The most prominent correlates of the cortical N1 wave to auditory stimulation were located between the upper mesencephalic reticular formation and the ventral lateral (VL) nucleus of the thalamus. Referential recordings using linked mastoids as a reference demonstrated that the waves of the auditory response "attained maximal amplitudes and minimal latencies at the caudal mesencephalic reticular formation (cttc). From here, their amplitude decreased and latency increased with distance along other structures rostrally located" (Velasco & Velasco, 1986, p. 65). There also appeared to be another focal area near the anterior commissure and the gyrus orbitalis. Velasco et al. (1985) postulate that there is a nonspecific sensory system "located in the center of the human brain involving a series of structures with a caudal-rostral arrangement, which extends from the rostral mesencephalon to the striatum and limbic structures of the frontal and temporal lobes" (p. 527).

The existence of such a nonspecific generator for the N1 was hypothesized in early studies (reviewed in preceding sections of this paper) in order to explain such findings as the interactions between sensory modalities and the sensitivity to changes in arousal. Recent evidence supporting such a concept is considered later in this review.

Intracranial recordings have thus indicated that several regions of the brain are active at the time of the scalp-recorded N1. The most important are the superior surface of the temporal lobe, some areas of the frontal lobe, the midbrain reticular formation, and the VL nucleus of the thalamus. Activity in any one of these areas may contribute to the electrical field recorded at the scalp. The amount contributed depends upon the synchronization of the activity, the geometry of the activated area, and the impedance of the brain, skull, and scalp.

Multiple Generators

The idea that multiple generators contribute to the scalp-recorded auditory N1 was first clearly stated by Wolpaw and Penry (1975). The waveform recorded from the temporal regions of the scalp using a noncephalic reference contains a negative peak that occurs about 30 ms later than the vertex N1 (Kooi et al., 1971). Wolpaw and Penry recorded auditory EPs from C_Z, T_3, and T_4 in response to clicks presented at 1/4.7 s. They proposed that a temporal "T-complex," with a positive peak at 105 ms (Ta) and a negative peak at 155 ms (Tb), overlapped a separately generated vertex N1-P2 response. They suggested that the T-complex was generated in the secondary auditory cortex on the lateral aspect of the temporal lobe, and that the N1-P2 was generated in widespread areas of cortex, particularly frontal. As reviewed in the previous section, Celesia recorded from the lateral surface of the temporal lobe (and other perisylvian regions) waves that were of similar polarity and latency (positive at 98 ms and negative at 142 ms) to Wolpaw and Penry's Ta and Tb waves.

Wolpaw and Penry (1977) reported that the peak latency of wave Ta was significantly longer and the Ta-Tb amplitude significantly smaller in the temporal electrodes ipsilateral to stimulation than in contralateral electrodes. In addition they found that the response was significantly larger over the right hemisphere than over the left.

Several other papers have demonstrated a temporal N140 wave that is apparently equivalent to the Tb wave of Wolpaw and Penry (McCallum & Curry, 1979, 1980; Picton, Campbell, Baribeau-Braun, & Proulx, 1978; Picton, Woods, & Proulx, 1978a, 1978b; Picton, Woods, Stuss, & Campbell, 1978; Wood & Wolpaw, 1982). Most papers have suggested that it results from a laterally oriented dipole in the temporal lobe. A concomitant positive wave recorded from the nasopharynx may represent the other side of such a dipole (Perrault & Picton, 1984). Peronnet, Giard, Bertrand, and Pernier (1984) have, however, recently suggested that the precentral motor cortex generates the negative wave that occurs later than the vertex N1 and has its maximum amplitude in the centrotemporal regions of the scalp contralateral to the ear of stimulation.

McCallum and Curry (1979, 1980) identified three separate peaks in the latency region of the auditory N1: N1a, N1b, and N1c. In one experiment the responses were evoked by monaural tones, the frequency of which informed the subject which hand to use in a later response. In a second experiment, the tones could require an immediate response, a delayed response, or a withholding of response. The N1a wave was maximally recorded from temporal and frontal-pole electrodes at a mean peak latency of 75 ms. It was larger in amplitude over the left hemisphere of right-handed subjects and over the right hemisphere of left-handed subjects,

and larger when the stimulus required a response.

The N1b wave of McCallum and Curry was maximally recorded from the central electrodes at a mean peak latency of 106 ms and appeared to correspond to the N1 wave of the vertex response. It was slightly larger contralateral to the stimulated ear, and was larger in the more demanding experimental conditions involving cross-over between the side of stimulation and the side of response. The N1b (and N1a) may have been generated by a source in the supratemporal cortex similar to that postulated by Vaughan and Ritter. However the N1b wave may also have reflected the nonspecific component suggested by the recordings of Velasco and his colleagues. If so, we have to assume that the contralateral predominance resulted from an overlapping with the field of a component that was larger contralaterally, such as the N1c which was recorded maximally from temporal electrodes at a peak latency of 129 ms. This wave was considerably larger over the hemisphere contralateral to stimulation.

Cortical recordings during the same experimental paradigm were obtained from 5 patients. Peaks similar in latency and polarity to N1a and N1c were recorded from electrodes over the temporal and parietal lobes but no component homologous to N1b could be recorded. Over the temporal cortex there was a positive wave at about 100 ms that may have been homologous to the Ta wave of Wolpaw and Penry (1975).

Perrault and Picton (1984) were unable to dissociate the N1a from the N1b component in a variety of experimental manipulations, and suggested that N1a and N1b represented the same cerebral process which was overlapped in the temporal regions by a positive wave such as Wolpaw and Penry's Ta component. Perrault and Picton (1984) demonstrated that the N1c was much larger for contralateral stimulation and found that it was larger when the stimuli were attended to than when they were ignored (and the subject read a book).

Multiple generators can produce complex scalp distributions. Using a sternovertebral reference, Wood and Wolpaw (1982) derived isovoltage topographic maps over the right hemisphere at each millisecond in the response to clicks presented at a rate of 1/3.2 s. In the latency of N1, they found three different and successive electrical fields: one that was frontally negative and temporally positive (field strength maximum at 78 ms); one that was exclusively negative and maximally recorded in the frontal regions (88 ms); and one that was exclusively negative with a temporal maximum (115 ms). These results are illustrated in Figure 1. Wood and Wolpaw pointed out the problems of inferring sources from the scalp-recorded electical fields: "The instantaneous potential field distribution over the scalp is determined by the instantaneous location and configuration of transmembrane current sources associated with active neural elements .... However, the converse is not true. The number, location and configuration of sources are not uniquely determined by the surface potential field. Therefore, hypothesized sources can be rejected if they conflict with empirical scalp distributions, but competing hypotheses that account equally well for empirical distributions must be evaluated using other data (e.g. intracranial recordings, lesion effects, animal studies, etc.)" (Wood & Wolpaw, 1982, p.32). They suggested two hypothetical source configurations that would be compatible with their recordings in the N1 latency: in the first, the vertex N1 is generated in the supratemporal plane as proposed by Vaughan and Ritter (1970), with overlapping activity from an additional undefined source or sources; in the second, the vertex N1 is generated by widespread cortical activation and there is an overlapping temporal complex as described by Wolpaw and Penry (1975).

Scherg and von Cramon (1985, 1986a, 1986b) have recently developed a technique to model the generator sources for scalp-recorded evoked potentials. They recorded the responses to brief tones presented every 1.5–2.5 s using eleven electrodes symmetrically located on a tilted coronal plane running through both mastoids and a midline electrode 3 cm in front of C_Z. Their technique posits dipole sources that generate a waveform over time and contribute to the scalp recordings on the basis of the location and orientation of the source and the conductivity of the head (modelled as a three-layered sphere). Using reasonable restrictions (triphasic dipole waveforms, four sources, spatial accuracy to within 2 cm), they were able to model the scalp-recorded field with two dipole sources in each temporal lobe. One source was oriented vertically (tangentially to the lateral surface of the skull) and the other horizontally (radially). In normal subjects, the tangential waveform is negative at 100 ms and positive at 180 ms; the radial waveform is positive at 100 ms and negative at 150 ms. These findings are illustrated in Figure 2. Scherg and von Cramon (1985) found similar results when they analyzed the data published by Vaughan et al. (1980) and by Peronnet et al. (1974). The tangential waveform is similar to that proposed by Vaughan and Ritter (1970) and the radial waveform is similar to that proposed by Wolpaw and Penry (1975). Scherg and von Cramon (1986a) found that the temporal P100 wave, like the Ta wave of Wolpaw and Penry, was significantly earlier in the contralateral hemisphere. However, Scherg and von Cramon, unlike Wolpaw and Penry, did not find any significant hemispheric

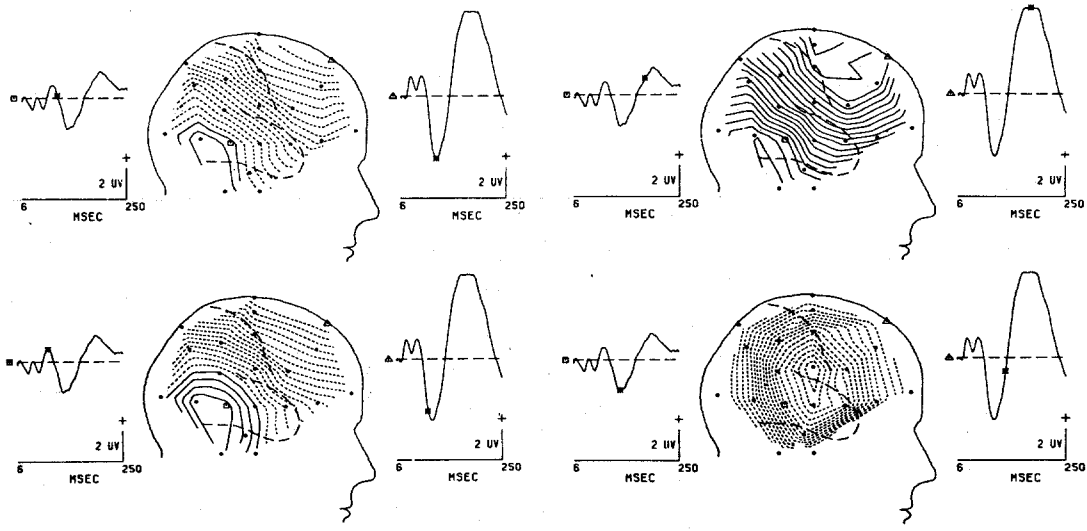

Figure 1. Scalp distribution of the auditory evoked potential to binaural clicks, recorded using a noncephalic reference. Four different scalp distribution maps are shown, each based on a particular latency. The latencies were chosen at peaks or troughs in the midfrontal location (upper figures) or in the midtemporal location (lower figures). The contours of each scalp distribution are plotted with the dotted lines representing negative potentials and the continuous lines representing positive potentials. On the left of each scalp map is shown the ERP recorded from an electrode over the temporal region (small square); on the right is the ERP from an electrode in the midfrontal region (small triangle). The latency at which the scalp distribution map was calculated is shown by the X on the waveform. The waveforms are plotted with scalp positivity upwards. On the left of the figure are shown two scalp distribution maps for the early part of the vertex N1 component, at latencies of 88 ms (top) and 78 ms (bottom). Both of these scalp distributions suggest a dipole with negativity recorded in the frontal regions and positivity recorded in the temporal areas below the Sylvian fissure. In the upper right is shown the scalp distribution at the latency of the P2 component at 170 ms, which shows a widespread positivity over frontal and central regions. In the lower right of the figure is shown the scalp distribution at the latency 115 ms. The scalp distribution shows a negative potential with maximal amplitude over the lateral scalp. From Wood and Wolpaw, 1982, *Electroencephalography & Clinical Neurophysiology, 54*, 25–38. Reprinted with the permission of the authors and the publisher.

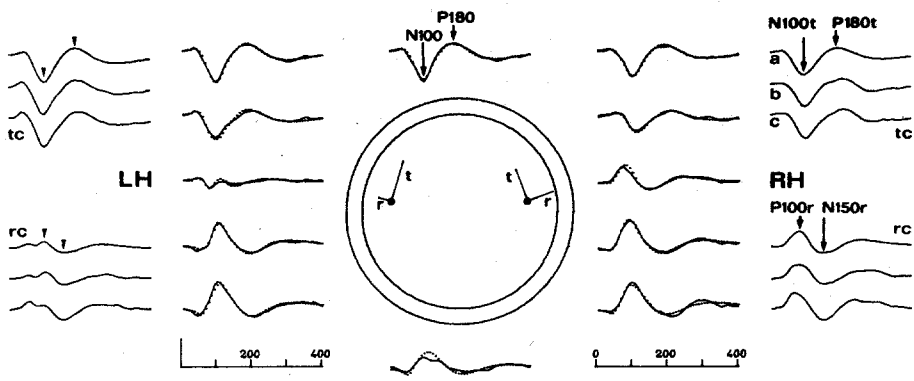

Figure 2. Coronal scalp distribution for the auditory evoked potential to a 1000 Hz tone burst. The recorded potentials (continuous traces around the diagrammatic cross-section of the head) were obtained using an average reference. The superimposed dotted traces are the best-fit waveforms from the spatio-temporal dipole analyses. Within the cross-section are shown the location (dot), orientation (direction of line), and relative strength (length of line) of the tangential (t) and radial (r) dipole source components in both temporal lobes. On the right and left, tangential (top—tc) and radial (bottom—rc) dipole source potentials from three different mathematical analyses (a,b,c) are depicted for the left and right hemispheres (LH, RH). The main peaks in the dipole source potentials are labelled by a polarity-latency-orientation (N/Pxxxt/r) nomenclature. From Scherg and von Cramon, 1986, *Electroencephalography & Clinical Neurophysiology, 65*, 344–360. Reprinted with the permission of the authors and the publisher.

differences in amplitude for the radial component. Furthermore, they reported that the tangential component (N100-P180) was significantly larger and earlier (N100) in the contralateral hemisphere.

The technique used by Scherg and von Cramon may have difficulty in delineating additional sources that contribute to the scalp recordings when these sources generate surface potentials that are similar to those of the initially derived dipoles. For example, the bilateral temporal dipoles may mask a more centrally located dipole, such as may be generated by a nonspecific system. Nevertheless, the accuracy of the modelling in both normal subjects and in patients with lesions of the temporal lobes suggests that the technique is describing the major sources underlying the N1-P2 waves. Further sources may be delineated by experimental manipulation and more extensive scalp recordings.

Magnetic Fields

Recording the magnetic fields that occur at the same time as scalp-recorded potentials can locate possible sources for these potentials. The magnetic field produced by a current dipole differs significantly from the electric field (Cuffin & Cohen, 1979). The magnetic field normal to the scalp is not affected by radial changes in the resistivity of the medium between the source and the recording location (Grynszpan & Gesolowitz, 1973). Since dipoles oriented along radii within a sphere produce zero magnetic field outside the sphere, it has been inferred (Baule & McFee, 1965) that the magnetic fields recorded from the human brain are generated by current sources oriented in a direction that is at least partially tangential to the surface of the head. The location of a tangential dipole in a spherical head (even with concentric inhomogeneities in electrical conductivity) is beneath a point halfway between the field extrema at a depth inversely proportional to the distance between the extrema (Williamson & Kaufman, 1981). Since with increasing dipole depth the magnetic field decreases more rapidly than the electric field, the magnetic response is relatively more sensitive to sources on the surface of the brain than in the depth (Cuffin & Cohen, 1979).

Most magnetic recordings have been interpreted on the basis of a single equivalent dipole underlying the magnetic field. It is, however, quite probable that several current dipoles may be activated at the same time in response to a sensory stimulus (see Kaukoranta, Hämäläinen, Sarvas, & Hari, 1986). The differences between the magnetic fields produced at the surface of a sphere by more than one current dipole and those produced by one dipole can be quite subtle (Nunez, 1986; Okada, 1985).

The first recordings of the magnetic field evoked by sounds were performed by Reite, Edrich, Zimmerman, and Zimmerman (1978). Farrell, Tripp, Norgren, and Teyler (1980), Elberling, Bak, Kofoed, Lebech, and Saermark (1980), and Hari, Aittoniemi, Järvinen, Katila, and Varpula (1980) suggested that the response was generated in the supratemporal plane. In the study of Farrell et al., the responses to clicks were recorded at latencies near 50 ms. Romani, Williamson, Kaufman, and Brenner (1982) recorded the magnetic fields evoked by continuous tones that were amplitude-modulated at rates between 4 and 55 Hz. This type of stimulation produces a response that is periodic at the rate of modulation—a "steady state" response (Regan, 1982). The apparent latency of this response at rates between 15 and 55 Hz was 49 ms. The derived dipole sources were localized to the region of the supratemporal plane. For amplitude-modulated tones with frequencies between 200 and 5000 Hz, the derived dipole sources were tonotopically organized, with the sources for lower frequencies located more superficially and more posteriorly (Romani, Williamson, & Kaufman, 1982).

The magnetic responses showed an anterior-posterior polarity reversal at the latency of the N1 (Elberling et al., 1980; Hari et al., 1980), thus suggesting a vertically oriented dipole at the level of the supratemporal plane. The location of this onset-evoked dipole was similar to that derived for the source of the auditory sustained potential during the continuation of the tone. Either the same cells or different cells located in the same cortical region might therefore generate the onset response and the sustained response. Very recently, however, Hari et al. (in press) have obtained data suggesting that the source of the sustained field is slightly anterior to that of the onset response (a finding corroborated by Mäkelä & Hari, in press), and not separable from the offset response. The offset response to a 550-ms break in a continuous tone originated from a source anterior to the onset response to a 550-ms tone in silence. The onset response also differed from the offset response by being preceded by a magnetic P40m and followed by a magnetic P200m. On the basis of these results and the findings of Sandel and Kiang (1961) that the offset response is more sensitive to anesthesia than the onset response, Hari et al. proposed that offset responses might reflect "the release of spontaneous activity from stimulus-induced inhibition at cortical level." Because of its similarity for many different auditory stimuli (tone pips, noise bursts, frequency changes, speech sounds), the authors suggested that the onset response reflects "cortical activity related to any abrupt change in the auditory environment" (Hari et al., in press).

Elberling, Bak, Kofoed, Lebech, and Saermark (1982) suggested that more than one dipole source might underlie the auditory magnetic fields at the latency of the N1. This could explain why the magnetic fields are not exactly symmetrical anterior and posterior to the postulated vertical source. They proposed a widespread residual magnetic signal, perhaps equivalent to the T-complex of Wolpaw and Penry (1975). However, Pantev, Hoke, and Lehnertz (1986; see also Hoke & Pantev, in press) did not find any evidence for a residual signal. Elberling et al. also reported that the vertically oriented dipole varied in location with the frequency of the tone. This tonotopic organization was not confirmed by Tuomisto, Hari, Katila, Poutanen, and Varpula (1983) or by Pelizzone, Williamson, and Kaufman (1985). Sams, Hämäläinen et al. (1985) observed that, in some subjects, the location and/or orientation of the equivalent current dipole changed during the duration of the N1 deflection. This suggests that more than one process underlies the magnetic N1 or that, as proposed by the authors, the area of activated cortex changes during the period of the N1 deflection. Pelizzone et al. (1985) found that the N1 dipole has a different location from the dipole underlying the auditory steady state response (Romani et al., 1982) and does not show any tonotopic organization (in contrast to the earlier, steady-state dipole). The current dipole at the same latency as the P2 wave is located anterior to the N1 dipole (Hari et al., in press; Pelizzone et al., 1985; Sams, Hämäläinen et al., 1985). Figure 3 illustrates the magnetic fields recorded at the latency of the N1 in response to a 1000 Hz toneburst.

Elberling, Bak, Kofoed, Lebech, and Saermark (1981) found that the magnetic response to 1 kHz tones increased exponentially with increasing intensity. In a very important paper, Bak, Lebech, and Saermark (1985) showed that the magnitude of the current dipole underlying the N1 magnetic response is quite regularly related to the intensity of the stimulus. The square of the intracellular axial current underlying the current dipole varies with the logarithm of the stimulus intensity above the threshold. Elberling et al. (1981) found that the magnetic N1 was larger over the hemisphere contralateral to stimulation than over the ipsilateral hemisphere, but that the two responses had similar intensity relations. Pantev, Lütkenhoner, Hoke, and Lehnertz (1986) reported a similar hemispheric asymmetry. They pointed out, however, that it is difficult to measure the magnitude of this asymmetry when the recordings were obtained at only one location. Elberling et al. (1981) found that the hemispheric asymmetry was greater for the tones presented to the right ear.

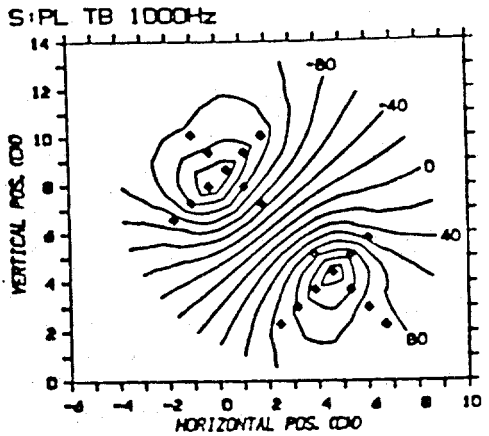

Figure 3. Magnetic fields over the right hemisphere for the 100-ms component of the transient response to a binaural tone burst of 1000 Hz presented at an ISI of 3–4 s and an intensity of 80–90dB SPL. The origin of the coordinate system is the ear canal, with the horizontal position measured toward the outer canthus of the eye (positive or right) and the vertical position measured perpendicular to this line (positive is upward). The contours are in fT units. From Pelizzone, Williamson, and Kaufman, 1985, in Weinberg et al., *Biomagnetism: Applications and theory*, Pergamon Press. Reprinted with the permission of the authors and the publisher.

Since magnetic techniques do not record the effects of dipoles oriented radially to the surface, additional generators of this nature may contribute to the scalp-recorded N1 electric potential and differentiate it from the magnetic response. Several early studies (Reite, Zimmerman, & Zimmerman, 1981; Reite, Zimmerman, & Zimmerman, 1982; Reite, Zimmerman, Edrich, & Zimmerman, 1982) found that the electric potentials differed from the magnetic fields in several stimulus-response relations. It is difficult to be sure that the magnetic field was optimally recorded in these studies since the location of the field maximum was not initially located with multiple recordings.

The different relations of the magnetic fields and of the electric potentials to ISI suggest that there are at least two components contributing to the vertex N1 electrical potential. Hari, Kaila, Katila, Tuomisto, and Varpula (1982) recorded the potentials evoked by brief binaural tones from C_Z, F_Z, T_4, and F_{P2}. They varied the ISIs between 1 and 16 s and maintained a constant interval for each block of recordings. At the shortest interval there were no significant differences in the N1 amplitude recorded at the different scalp locations. At longer intervals the vertex N1 became the largest, and the frontal amplitude was larger than the temporal amplitude. Only the vertex amplitude continued to increase as the interval was increased from 8 to 16 s. At in-

tervals less than 4 s the vertex and frontal amplitudes were equal whereas at longer intervals the vertex amplitude was larger than the frontal amplitude. This could explain why the distribution of the N1 reported by Picton et al. (1974) using a 2-s interval was more frontal than that of Goff, Matsumiya, Allison, and Goff (1977) using an interval of 4 s (and stimuli randomized across three different modalities within the same block). Hari et al. concluded that "evidently the sources activated by frequent and infrequent stimuli are not identical" (p. 566). They also found that the magnetic field recorded at the same time as the N1—N1m—did not increase in amplitude at intervals longer than 4 s. These results are illustrated in Figure 4. Hari et al. proposed that the N1m was generated by current dipoles in the supratemporal planes, and that these dipoles were represented best by the frontal electric recordings. Because of the increase in the vertex N1 at long ISIs they also suggested that "some additional sources are activated during infrequent stimulation . . . . these sources are probably current dipoles perpendicular to the scalp near the vertex. They might be connected to the orienting response" (p. 567).

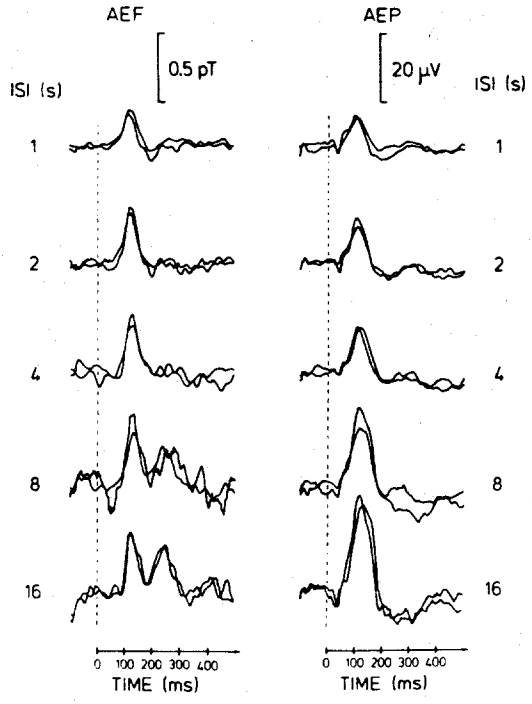

Figure 4. Simultaneous magnetic (AEF) and electrical (AEP) responses from a single subject to tones presented at different ISIs. The magnetic responses were recorded at a location half way between P_4 and T_6, and the electrical responses from derivation C_Z referred to the right mastoid. Positive direction of the magnetic field (into the skull) and negativity of the active electrode upwards. From Hari et al., 1982, *Electroencephalography & Clinical Neurophysiology, 54*, 561–569. Reprinted with the permission of the authors and the publisher.

Magnetic recordings have clearly shown that a current dipole located in the supratemporal plane occurs at the time of the scalp-recorded N1 wave. Such a dipole would generate, in addition to the magnetic field, an electric field at the scalp with a negative polarity maximal in the frontocentral regions and a positive polarity below the level of the Sylvian fissure. This dipole is practically identical to that proposed by Vaughan and Ritter (1970) and to the tangential dipole derived by Scherg and von Cramon (1986a). Unfortunately, magnetic recordings are relatively blind to radial dipole sources. Since the relations of the electric N1 to several stimulus parameters cannot be fully explained by the current dipole derived from magnetic recordings, such radial dipoles almost certainly exist. One such radial dipole—generated in the lateral surface of the temporal lobe—may underlie the T-complex of Wolpaw and Penry (1975). Another—perhaps generated in the region of the vertex by some relatively nonspecific system—may be necessary to explain the electric changes in the N1 at very long ISIs.

Cerebral Lesions

At times, EP recordings in patients with cerebral lesions produce crucial evidence about the location of a generator; at other times, they result in recordings that are difficult or impossible to interpret. Pathology is rarely discrete and often affects regions of the brain other than that responsible for the primary symptoms. Lesions in one area of the brain can alter the function of other areas. Lesions may also alter the conductivity of the brain sufficiently to distort the electric fields produced by an active generator.

The effects of cerebral lesions on the auditory N1 have been variable and complex. Lesions involving both temporal lobes have caused different effects in different patients. Jerger, Weikers, Sharbrough, and Jerger (1969) and Michel, Peronnet, and Schott (1980) were unable to record any N1 in response to sounds from patients with bilateral temporal lobe lesions. Other papers (Parving, Salomon, Elberling, Larsen, & Lassen, 1980; Rosati et al., 1982; Woods, Knight, & Neville, 1984) have, however, reported normal responses in such patients. These differences may have resulted from the variable extent of the lesions in the temporal lobes. The patient reported by Woods et al. (1984) showed approximately normal N1-P2 waves in response to auditory stimuli that the patient could not perceive. One of several possible explanations for this dissociation

between the evoked potential and perception was that "an island of auditory cortex might have remained intact (thus producing the electrophysiological response) but have been isolated from surrounding brain regions necessary for stimulus evaluation and response" (Woods et al., 1984, p. 217). Because of its convoluted geometry, widespread activation of the auditory cortex could cause a large amount of field cancellation. Activation of only an isolated island of cortex could have resulted in less cancellation and may thus have generated a field as large as normal. Another possibility was that the N1-P2 was generated in another region of cortex (such as the angular gyrus) that was spared by the bitemporal lesions.

Woods, Clayworth, Knight, Simpson, and Naeser (1987) have recently reviewed the auditory EPs recorded from 6 patients with bitemporal lesions accurately delineated by computerized tomography. The only patients showing no detectable N1 waves had lesions that extended into the inferior parietal lobule. Patients with lesions restricted to the temporal and/or frontal lobes had N1 waves with normal amplitude. Although these results suggest that the inferior parietal lobule is significantly involved in the generation of the N1, it probably does not contain the actual source since it is difficult to see how the frontocentral negativity could be generated from this location. Part of the N1 is probably generated in some region (thalamus, supratemporal plane, frontal cortex?) that is facilitated by the inferior parietal lobule.

Unilateral lesions involving the temporal lobe have often produced asymmetrical effects on the N1-P2 waves. Peronnet et al. (1974) recorded the auditory evoked potentials from 3 patients with unilateral lesions presumably involving Heschl's gyrus. Using a nose reference they found a prominent polarity reversal over the Sylvian fissure of the healthy hemisphere, but little if any polarity reversal over the damaged hemisphere. These results would be expected from the unilateral loss of a dipole located in the supratemporal plane and directed toward the midfrontal region. There may be little change in the vertex response but only a marked asymmetry in the lateral electrodes below the level of the dipole. Such an asymmetry would not be detected if a linked-mastoid reference were used.

In patients with well-delineated lesions of the frontal lobes or of the temporoparietal regions, Knight et al. (1980) recorded the auditory evoked potentials from the vertex and from temporal, central, and frontal scalp locations using a linked-mastoid reference. The stimuli were brief tones presented at random ISIs of 0.5, 1, and 3 s. The N1 waves of the patients with temporoparietal lesions were much smaller than those of the normal subjects, did not show a vertex predominance, and were not affected by the ISI. On the other hand, their P2 waves were not different from those of normal subjects. These results indicate that the temporoparietal region of the brain is critically involved in the generation of the N1. However, there was no asymmetry of the response related to the side of the lesion. This finding suggests that the temporoparietal region does not generate the response but rather modulates a separate generator. However, the responses in these patients were very small. Because of the field spread from a lateralized generator to the opposite scalp and because of the problems of a linked-mastoid reference, it is difficult to be certain that the response was completely symmetrical. The results, therefore, do not definitely rule out a generator in the temporal lobe.

Knight et al. (1980) found that lesions of the frontal lobe had no effect on the amplitude, scalp distribution, or refractory properties of the N1. These results with frontal lesions suggest that the N1 does not have a generator in the frontal lobes. However, following frontal lesions the N1 was larger to the tones presented contralateral to the lesion. The normal frontal lobes may therefore inhibit the generator of the N1 in response to contralateral sounds.

Using their new dipole-modelling technique, Scherg and von Cramon (1986a, 1986b) demonstrated two patterns of abnormality. Lesions involving the primary auditory cortex severely attenuate the source dipoles in the lesioned temporal lobe for both the middle-latency responses (10–50 ms) and the late waves. This asymmetry of the dipole sources can occur when scalp recordings over the lesioned area show waveforms of normal morphology but attenuated amplitude (volume-conducted responses from the spared contralateral auditory cortex). Patients with unilateral lesions involving the auditory radiations but sparing the auditory cortex showed a second pattern of abnormality: reduced or absent dipoles for the auditory middle latency responses in the lesioned hemisphere but symmetrical dipole sources for the late (N1-P2) waves. In some of these patients the N1 dipole in the lesioned hemisphere was significantly delayed, possibly because it was mediated through callosal pathways. Scherg (personal communication, October, 1986) has recently found evidence for a third type of abnormal auditory evoked potential: in patients with lesions affecting the auditory association areas but sparing the primary cortex, the middle latency responses and the tangential components of the late responses were normal, but

there was a selective attenuation of the radial (P100-N150) component over the side of the lesion.

It is difficult to find a coherent interpretation that takes into account all of the data recorded from patients with cerebral lesions. The simplest explanation is that there are only two sources for the auditory N1 wave and that these are both located in the temporal lobe: the first located in the supratemporal plane and generating a frontocentral N1, and the second located on the lateral surface of the temporal lobe and generating a somewhat later negative wave with maximal amplitude over the temporal scalp. This explanation requires that one attribute the existence of a vertex N1 in patients with bilateral lesions of the temporal lobes to the preservation of some small area of functioning auditory cortex. It also requires that one attribute the lack of asymmetry noted in many patients with unilateral lesions of the temporal lobe to some inadequacy of the recording technique (such as the use of a linked-mastoid reference). Another possible explanation for these discrepancies is a third generator that contributes to the frontocentral N1 and that may be spared in patients with lesions of the temporal lobe. Such a generator, the location of which is not known, may be responsible for the relatively nonspecific N1 component that we proposed in the previous section of this paper to explain the discrepancies between the magnetic and electric recordings at long ISIs.

Summary

The information that we have reviewed so far indicates that the auditory N1 wave does not reflect a single underlying cerebral process and should not therefore be considered as a unitary event. Several aspects of recent research have been important in changing this earlier idea. First, ERP research is beginning to concentrate more on the neurophysiological processes that underlie the scalp-recorded electric fields than on the phenomenology of the fields themselves. Second, we have come to realize that the peaks and troughs of a scalp-recorded waveform, although comfortable landmarks for identification and measurement, are not necessarily any more informative than other points on the ERP curve with respect to the overlapping fields that form this recording (Näätänen, 1975; Wood & Wolpaw, 1982). Third, it is becoming obvious that the use of a limited number of recording sites may seriously impede any evaluation of the component structure of the ERP.

The scalp distribution of the auditory N1, the magnetic fields recorded at the same latency as the N1, and the effects of cerebral lesions on the N1 suggest three different components contributing to this scalp-recorded wave. The first component is a frontocentral negativity generated by bilateral vertically oriented dipole planes in the auditory cortices on the superior aspect of the temporal lobe. This component, originally proposed by Vaughan and Ritter (1970), is strongly supported by the magnetoencephalographic results and by the tangential dipole source potentials derived by Scherg and von Cramon (1986a, 1986b) in normal subjects and patients with cerebral lesions. The second component is the T-complex with a positive wave at 100 ms and a negative wave at 150 ms, as originally described by Wolpaw and Penry (1975). This complex probably originates in the auditory association cortex in the superior temporal gyrus. Due to the radial orientation of the underlying current dipole, this component is not picked up with magnetoencephalographic recordings. Nevertheless, it has been convincingly modelled by Scherg and von Cramon in their analysis of the radial source dipoles for scalp-recorded fields, and it has received support from the intracerebral recordings of Celesia (1976) and of McCallum and Curry (1979, 1980). The third component is one that generates a negative wave at the vertex with a latency of 100 ms. The location of its generator is not known. The justification for this component comes from the intracerebral recordings of Velasco and his associates (1985; Velasco & Velasco, 1986) and the findings of Hari and her associates (1982) concerning the effects of ISI on the scalp topography of the N1. The field of this component is widespread with a maximum that is somewhat posterior to that of component 1, with which it often overlaps.

The existence of such a relatively nonspecific component is more clearly supported in the visual modality. Lehtonen (1973) found that the occipital negative wave evoked by visual stimuli in the N1 latency region (90–120 ms) increased when the ISI changed from 0.5 to 1 s but did not further increase when the ISI changed from 1 to 6 s (a later occipital negative wave at 120–175 ms increasing until the 3-s ISI). On the other hand, the vertex negative wave at 110–170 ms continued to increase in amplitude when the ISI increased to 6 s (see also Gjerdingen & Tomsic, 1970). Lehtonen concluded that the "occipital deflections probably reflect the function of a modality-specific cortical area .... and the vertex waves the function of a modality-nonspecific area" (Lehtonen, 1973, pp. 80–81). Consistently, Vaughan concluded from his topographical analyses that the vertex and occipital waves "result from two distinct generators rather than from volume conduction of the occipital response" (Vaughan, 1969, p. 65).

As we continue with our review, we shall find more evidence supporting these three components, and we shall describe more fully these "true" com-

ponents of the auditory N1 wave. We shall find that they are largely determined by the physical characteristics of the stimulus and by the general state of the subject. We shall also consider other components in the latency region of the N1 which are related more to memory and cognition than to stimulus and state, and which we shall not classify among the "true" N1 components.

Stimulus Parameters

Change

The N1 potential is evoked by a relatively abrupt change in the level of energy impinging on the sensory receptors. Stimuli with very slow onsets do not elicit this response (Clynes, 1969). Sustained stimuli elicit the N1 potential only at their onset, with prolongation of the stimulus increasing the N1 amplitude only up to durations of 30–50 ms (Kodera, Hink, Yamada, & Suzuki, 1979; Onishi & Davis, 1968). There are two possible explanations for an onset response. One is that the response is generated by cerebral systems that respond specifically to the onset. The other is that the neuronal responses are sufficiently synchronized to generate a field potential only at stimulus onset, and that during the continuation of a stimulus the positive and negative potentials generated by unsynchronized neurons cancel. Such an argument is used to explain the compound nerve action potential of the auditory nerve (Elberling, 1976). This explanation could apply in part to the activity evoked in the cortex. However, the majority of cortical neurons, unlike the auditory nerve fibers, respond to the onset and not to the continuation of sensory stimuli (Goldstein, Hall, & Butterfield, 1968). We are therefore probably dealing with a true onset response rather than with some artifact of synchronization.

Gersuni (1971) suggested that the auditory system works through two different mechanisms, one with a short time constant for measuring change and time, and the other with a long time constant for evaluating pitch and intensity. The N1 appears to reflect the short time constant system. It probably indexes a cerebral system that monitors abrupt changes in sensory input and does not record the stable state—"if information about the steady or absolute level of all possible stimuli were transferred to the later stages of the system, these would soon be jammed with irrelevant and useless stores of obsolete data" (Walter, 1964, p. 338).

The response can be elicited by the offset as well as the onset of a stimulus (Davis, 1939; Davis & Zerlin, 1966). An offset response is recognizable only if the stimulus has been on for more than about 0.5 s, and the response increases in amplitude as the stimulus duration is increased (Davis & Zerlin, 1966; Hillyard & Picton, 1978; Onishi & Davis, 1968; Pfefferbaum, Buchsbaum, & Gips, 1971; Rose & Malone, 1965). For equal on-off cycles the offset response is smaller than the onset response. In this comparison, one has to consider that during the offset response there is a superimposed return-to-baseline of the negative "sustained potential" evoked during the continuation of a stimulus (Hillyard & Picton, 1978; Keidel, 1971; Picton, Woods, & Proulx, 1978a, 1978b). In the auditory system the offset response has a latency that is 10–20 ms shorter than that of the onset response (Onishi & Davis, 1968). Unfortunately, it is not possible to determine from available data whether the onset and offset N1 waves have the same or different component structures, although Picton, Woods, and Proulx (1978a) showed that their midline scalp distributions are very similar (cf. the previously discussed magnetic recordings of Hari et al., in press).

Picton, Woods, and Proulx (1978a, 1978b) showed that the N1 response to the onset of a tone differs significantly from the sustained response to the continuation of the tone. The two parts of the response show different relations to the intensity and frequency of the tone. The sustained response is much less sensitive to decreasing ISI than the onset-evoked N1. Furthermore, the refractory effects of the N1 are more widely generalized than those of the sustained potential. Combining rapidly presented clicks with the tones significantly reduced the amplitude of the N1 evoked by tone onset but did not affect the sustained potential. These results show that the sustained potential is more stimulus specific than the N1 wave. As already suggested, the N1 wave appears to contain both stimulus-specific and stimulus-nonspecific components.

The latency and amplitude of the N1 are determined by the slope of the energy change—the rise-time or the fall-time. Several papers (Kodera et al., 1979; Milner, 1969; Onishi & Davis, 1968; Ruhm & Jansen, 1969) have reported that the N1-P2 amplitude of the response decreased as the rise-time or fall-time of the stimulus became longer than 30–50 ms. Furthermore, as already mentioned, the amplitude of the response increased as the duration of a toneburst increased up to 30–50 ms. These times do not relate to psychophysical measurements of the temporal integration time for loudness (about 200 ms). In addition, the effects of ISI on the N1, to be reviewed later in this paper, clearly show that the N1 has little to do with loudness.

The N1 can be elicited by a change in the tonal frequency of a continuous auditory stimulus as well as by a change in intensity (Clynes, 1969; Spoor, Timmer, & Odenthal, 1969). Arlinger et al. (1982) recorded the magnetic fields evoked by brief changes in frequency of a continuous tone. Like the re-

sponse to the onset of a tone, the equivalent dipole underlying the response to a frequency glide was localized to the supratemporal plane.

If a continuous stimulus is already changing, even quite slowly, a further change may not elicit an N1 response. Clynes (1969) recorded the N1-P2 response to changes in a continuous tone. The amplitude or frequency was changed from one value to another over a steady ramp. The onset of a ramp elicited an N1-P2 response but the offset of a ramp, when the tone became constant again, did not elicit the response (Figure 5, top panel). Furthermore, the response to a ramp onset could be markedly reduced or eliminated by preceding the ramp by another ramp in a similar or different direction (Figure 5, bottom panel). These findings were confirmed by Kohn, Lifshitz, and Litchfield (1978, 1980). Furthermore, Clynes reported that a ramp change in one attribute can attenuate the response to a subsequent change in another attribute. Clynes observed such interactions between the pitch and intensity of a continuous auditory stimulus. However, these interactions did not cross modality boundaries. An ongoing pitch modulation did not affect the visual response, although there were similar basic rest-motion findings in the visual (and also somatosensory) modality. Clynes (1969) concluded that the N1-P2 response is initiated by a change from sensory "rest" (a steady stimulus or silence) to sensory "motion" (from one level of a stimulus parameter to another).

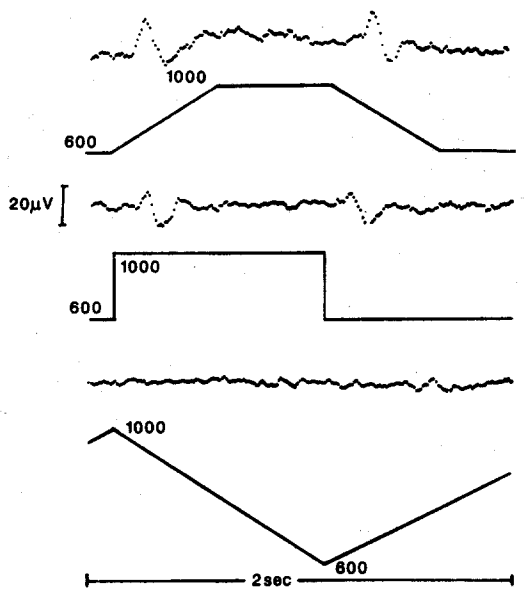

Figure 5. Vertex evoked potentials to changes in the pitch of a sound having constant intensity. The pitch of the sound is shown below each of the three paired recordings. The upper recordings are in response to ramp-changes in pitch. Note the absence of any response at the end of the ramp. The middle tracings show the responses to abrupt changes in pitch. The lower recordings show no clear response when one ramp-change of pitch leads immediately into another. Figure derived from the data of Clynes, 1969, in Donchin and Lindsley (Eds.), *Average evoked potentials*, NASA. Reprinted with the permission of the author and the publisher.

The stimuli most commonly used to evoke auditory EPs are short in duration and separated from each other by silent intervals. An N1 wave is evoked by the onset of the sound—the change from silence to stimulus. Because of the short duration of the stimulus, there is little if any offset response.

In these simple paradigms, another kind of "change" can also occur: the stimulus may change from the preceding stimulus in some parameter such as intensity or frequency. It is therefore important to distinguish between two kinds of change: "change-1" or "level change"—a change from the immediately preceding stable level (usually from silence to sound); and "change-2" or "stimulus change"—a change from some previously presented stimulus. Change-2 cannot occur without a concomitant change-1 and only when successive presentations of change-1 are different (a duration change-2 is an exception). In contrast to the response to a level change, the response to a stimulus change requires that there be some neuronal trace or memory of the previous stimulus (Näätänen, 1985).

The N1 wave is apparently generated by cerebral mechanisms which are primarily sensitive to change-1. This is probably true for all three of the components that we have so far considered. The N1 is triggered by the onset of a change in some physical characteristic from an immediately preceding stable level. The N1 mechanisms may, however, at times appear as if responding to the second kind of change also. When, for instance, an occasional tone of 1500 Hz occurs in a sequence of 1000 Hz tones, the N1 wave in response to the 1500 Hz tone is usually larger than that in response to the 1000 Hz tone (Butler, 1968; Picton, Campbell, Baribeau-Braun, & Proulx, 1978). However, this may be a case of selective refractoriness rather than a specific response to the change. The deviant stimulus apparently activates some "fresh" elements that were not activated by the preceding standard stimuli. As we shall discuss in the next section of this paper, the responsiveness of the N1 generators is decreased for a period of time after the presentation of a stimulus, and this decrease is partially specific to the stimulus. Thus the amplitude decrease is greater when the N1 is reactivated by a similar stimulus than when the N1 is reactivated by a different stimulus, i.e., the neuronal population responding to the

deviant stimulus is less refractory than the neuronal population responding to the standard stimulus.

The amplitude of the N1 is therefore jointly determined by the immediate change in the stimulus level and by the refractory state of the generator mechanism. The refractory state will vary with the time from the preceding stimulus and the similarity between the present and the preceding stimulus (this similarity determining the magnitude of overlap between the the neuronal populations responding to each stimulus). Therefore, when a stimulus signifies change-2 in addition to change-1, the N1 response may be bigger than when change-1 alone occurs. Another component of the response—the "mismatch negativity" (MMN) to be discussed in the following paragraphs—is much more closely related to change-2.

The cerebral response to deviant stimuli that have a lower intensity than standard stimuli provides crucial evidence differentiating the N1 and the MMN and their relations to change-2. Figure 6 illustrates such responses (Näätänen, Paavilainen, Alho, Reinikainen, & Sams, 1987). In this experiment, the standard stimulus had an intensity of 80dB SPL and, in different blocks, the deviant stimulus (p=.10) had an intensity of 57, 70, 77, 83, 90, or 95 dB. The subject read a book and ignored the sequence of auditory stimuli presented at a constant ISI of 460 ms. The responses to the deviant stimuli that had higher intensities than the standard stimulus showed a larger N1 deflection than the responses to the standard stimuli. However, the response to the 77dB deviant stimulus had a smaller N1 deflection than the response to the 80dB standard stimulus. These results are similar to those obtained by Butler (1968). They indicate that the N1 deflection (at least when recorded from the midline) is more related to the physical characteristics of the stimulus than to the change in the stimulus from a preceding stimulus.

The response to the 77dB deviant stimulus revealed a separate negative deflection called the mismatch negativity (MMN). This MMN occurred in the responses to all of the deviant stimuli, both those with higher intensity and those with lower intensity than the standard stimulus. As the difference between the deviant and standard stimuli increased, the MMN became larger and earlier, overlapping the N1 deflection and making it impossible to measure the MMN and the N1 separately. These effects were similar for deviant stimuli that were higher or lower in intensity than the standard stimuli. The MMN therefore appears to vary specifically with change-2 (the difference between successive stimuli) and not with change-1 (the intensity of the stimulus). Since the latency of the MMN may overlap with that of the N1 wave, the MMN must be considered as a possible component of the scalp-recorded N1 wave.

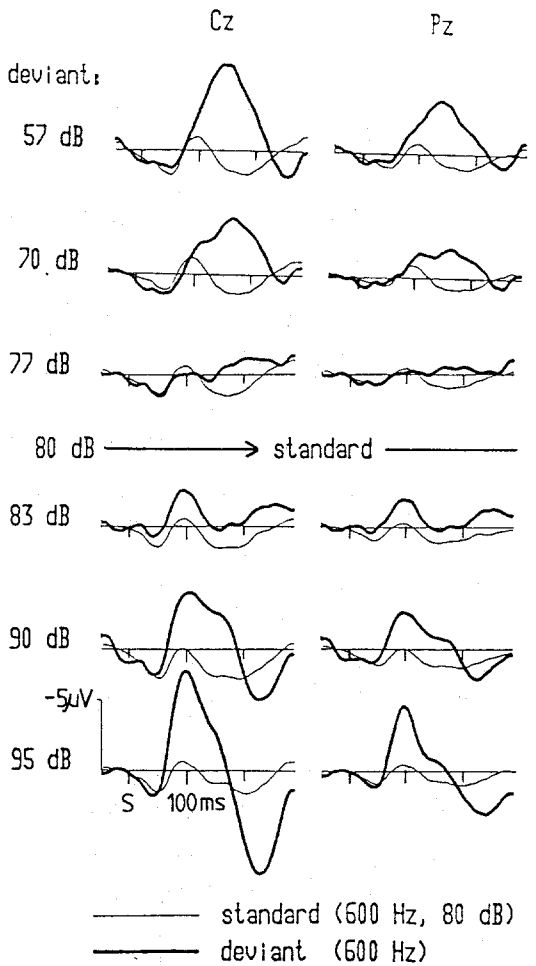

Figure 6. The mismatch negativity to intensity changes. Across-subject average vertex (C_Z) and parietal (P_Z) ERPs to standard stimuli of 80dB (thin-line) and to deviant stimuli of different intensity (thick line) as indicated on the left side of the figure. In each stimulus block, the probability of the standard stimulus was 90% and that of the deviant stimulus was 10%, stimuli being presented in random order. There was only one kind of deviant stimulus in each block. From Näätänen, Paavilainen, Alho, Reinikainen, and Sams, 1987.

This distinction between the N1 response to individual stimuli and the MMN response to the relations between stimuli is further supported by the changes in MMN latency with different degrees of deviance, the N1 latency showing no such variation. As seen in Figure 6, the latency of the MMN decreases with increasing difference in intensity. Figure 7 presents results from an experiment (Sams, Paavilainen, Alho, & Näätänen, 1985) in which the

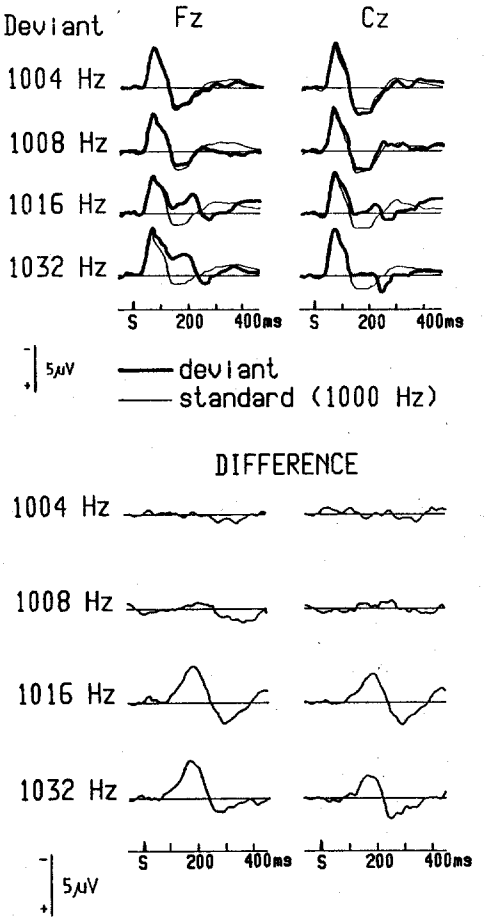

Figure 7. The mismatch negativity to small changes in frequency. The left column shows the frequency of an occasional deviant stimulus in a train of standard stimuli. The middle column shows the EPs recorded from F_Z and the right column shows the EPs recorded from C_Z. The top of this figure shows the across-subject average ERPs to standard stimuli of 1000 Hz (thin line) and deviant stimuli (thick line) of 1004 Hz, 1008 Hz, 1016 Hz, and 1032 Hz. There was only one kind of deviant stimulus in each block. In each stimulus block, the probability of the standard stimulus was 80% and that of the deviant stimulus was 20%, stimuli being presented in random order. The bottom of the figure shows the respective difference waveforms obtained by subtracting the EP to the standard stimuli from the EP to the deviant stimuli. Figure derived from data of Sams et al., 1985, *Electroencephalography & Clinical Neurophysiology, 62*, 437–448. Adapted with the permission of the authors and the publisher.

deviant stimuli (p=.20) differed in pitch from the standard stimuli. The N1 wave in response to the different deviant stimuli (held constant within a block) is similar to that evoked by the standard stimuli and does not vary in amplitude or latency with the degree of deviance. As shown in the difference waveforms on the bottom of Figure 7, the MMN is clearly seen in response to deviant stimuli that could be discriminated from the standard stimuli, i.e. those with frequencies greater than 1008 Hz. There may be a very small MMN at 1008 Hz. The amplitude of the MMN remains relatively stable once the deviance has become clearly recognizable but the MMN peak is earlier for larger deviations in pitch. With very large deviations, the decreased latency of the MMN results in an increasing overlap between the MMN and the N1 wave (Näätänen & Gaillard, 1983). Some of the increase in the N1 wave previously attributed to specific refractoriness in one or more of the N1 generators may be explained on the basis of this overlap.

The MMN introduced by these two experiments has been reviewed more extensively elsewhere (Näätänen, 1985, 1986a). We shall discuss in the next few paragraphs some of the specific characteristics of this component and its relation to the N1 wave.

The MMN component was isolated from the N2-P3 and N2-P3a deflections of the EP by Näätänen, Gaillard, and Mäntysalo (1978, 1980), by Näätänen, Simpson, and Loveless (1982), and by Näätänen and Gaillard (1983). Näätänen et al. (1978) suggested that the MMN reflects a pre-perceptual detection of stimulus change. Their subjects performed a selective dichotic-listening task, counting occasional intensity (or pitch) changes presented to one ear. The difference wave obtained by subtracting the EP to standard stimuli from that to deviant stimuli revealed a negative shift with an onset latency of about 100 ms and a duration of 200 ms. This MMN occurred in response to the deviant stimuli in either the attended or unattended inputs and its amplitude was unaffected by attention. The P300 positive wave was much larger to the (target) deviant stimuli in the attended input than to the (non-target) deviant stimuli in the unattended input.

Näätänen et al. (1982; see also Näätänen & Gaillard, 1983) proposed that the N2 deflection elicited by a deviant stimulus in the oddball paradigm consists of a MMN, generated in the specific auditory areas of the cortex, and an N2b wave, a later and sharper component probably of nonspecific origin, that occurred when the subject attended to the stimulus sequence. When the stimuli were ignored, only the MMN occurred, unless the deviant stimuli were widely deviant, in which case some N2b waves were detected in the records of some subjects. In the dichotic listening paradigm, the N2b to targets is much smaller than in the oddball paradigm (Näätänen et al., 1978; Näätänen, Gaillard, & Mäntysalo, 1980). Non-target deviant stimuli that elicit an N2b when

a single attended train of stimuli is presented do not elicit this wave when they belong to the ignored input during a dichotic paradigm (Näätänen et al., 1982).

The MMN is not elicited by change-1 even when preceded by a prolonged period of silence (Näätänen, 1985; Sams, Hämäläinen et al., 1985; Snyder & Hillyard, 1976). A necessary condition for the MMN is that a different stimulus in the same modality has preceded the present stimulus within the last few seconds. This MMN is a reflection of stimulus deviation: what matters is the difference from the preceding stimuli, and not the stimulus parameters per se. If the standard stimulus were omitted, the deviants would elicit no MMN (Sams, Hämäläinen et al., 1985; Snyder & Hillyard, 1976) . In the situation illustrated in Figure 7, exchanging the probabilities of the two stimuli would lead to the 1000 Hz stimulus eliciting the MMN (Näätänen, 1985).

Näätänen (1985, 1986a) suggested that the MMN implies the existence of a precise, rapidly decaying and automatic neuronal representation of the physical features of a stimulus, a representation that may form the neurophysiological basis of auditory sensory or "echoic" memory (Neisser, 1967). When a deviant stimulus of the same sensory modality is presented during the persistence of such a trace, a neuronal mismatch response, reflected by the MMN, occurs, irrespective of the direction of attention, with the assumed biological function of alarming the individual to a change in the environment. The MMN could thus reflect a process of passive attention (James, 1890).

As already mentioned, when a subject actively attends to a train of stimuli in order to detect an occasional change in the stimulus, the EP to this different stimulus contains an N2 wave followed by a P3 wave. Several different cerebral processes—the MMN, the N2b, and perhaps others—probably contribute to the N2 wave. Näätänen, Hukkanen, and Järvilehto (1980) found that the amplitude of this N2 wave varied with the pitch difference between the standard and deviant stimuli when the subjects were attending to these differences. Fitzgerald and Picton (1983), on the other hand, found that the N2 amplitude increased as a function of the difficulty of the discrimination, i.e., N2 was larger the smaller the difference in pitch. In the early part of the N2 wave, however, there was a suggestion of the opposite effect, the waveform being more negative for the greater pitch difference. They interpreted their findings as showing a MMN upon which was partially superimposed a separate effort-related (larger for smaller differences) negative wave. This could be a third component of the N2 wave. Furthermore, Fitzgerald and Picton suggested that the MMN was a continuation of the same processes that generated the usual N1 wave.

The MMN and the N1 wave are indeed similar in some respects. Most importantly, the magnetic field associated with the MMN suggests a current dipole in the supratemporal plane where the N1 dipole occurs (Hari et al., 1984; Sams, Hämäläinen et al., 1985). However, the dipole has a somewhat different location and orientation to the N1 dipole. Furthermore, as already discussed, there are other clearly differentiating characteristics: (1) The relation of the MMN to stimulus deviance is much more precise. The MMN is selectively elicited even when the deviant stimulus differs from the standard stimuli by only a slight amount, while the N1 waves elicited by such stimuli are not recognizably different. (2) The MMN may be evoked by any deviance in the stimulus, even a decrease in intensity, whereas the N1 to a less intense deviant stimulus is smaller. (3) The MMN and the N1 have completely different relations with the ISI. At the beginning of a sequence or after a very long ISI, the MMN is not elicited by either the standard or the deviant stimulus, whereas the N1 to both is very large (Näätänen, 1986b). When the ISI is shortened, the MMN becomes larger but the N1 is attenuated (Mäntysalo & Näätänen, in press; Näätänen, Paavilainen, Alho, Reinikainen, & Sams, in press). (4) The latency of the N1 to a deviant stimulus does not vary with the magnitude of deviation (see also Lawson & Gaillard, 1981), whereas the MMN latency varies dramatically with that magnitude. The evidence therefore suggests that although N1 and the MMN can overlap they reflect distinct cerebral processes.

Intensity, Frequency, and Threshold

With decreasing stimulus intensity the N1 response decreases in amplitude and increases in latency (Beagley & Knight, 1967; Picton, Woods, Baribeau-Braun, & Healey, 1977; Rapin, Schimmel, Tourk, Krasnegor, & Pollak, 1966). The change in amplitude is more variable than the change in latency. The latency change is more prominent with tonal stimuli than with clicks.

There have been many studies relating the amplitude of the N1 response to sensory magnitude. Davis and Zerlin (1966) found that the N1-P2 response to a tone pip (presented every 3.2 s) increased according to a power function with an exponent of 0.12 (compared to the psychophysical power function of 0.3). Keidel and Spreng (1965) reported a higher exponent and a closer correlation to the loudness power function at longer ISIs (30 s), but the N1 that they recorded was later (130–170 ms) than usual. This may have been caused by

their unusual electrode montage: their recordings were taken between the glabella (mid-forehead) and the mastoid. Keidel (1976) reviewed the field and found that the vertex N1 wave shows a power function exponent of between 0.07 and 0.28, the higher values occurring when longer ISIs (30 s) are used. He further reported that the N1 amplitude is more closely related to loudness than the N1-P2 amplitude. Pratt and Sohmer (1977) recorded auditory EPs to clicks at the same time as subjects assessed the loudness of the stimuli. A set of clicks occurred one second after the subject pressed a button to record his or her loudness estimate for the preceding clicks. Pratt and Sohmer found no clear correlation between the amplitude of the late auditory response and the concurrently recorded subjective estimates of loudness.

At high intensities the amplitude of the N1 often levels off or even reduces (Buchsbaum, 1976). This is particularly true when stimuli are presented at intervals of 2.5 s or less and the intensity is held constant within blocks (Picton, Goodman, & Bryce, 1970). This saturation of the N1 amplitude occurs well below any saturation of subjective loudness, and thus indicates a clear dissociation between sensory magnitude and the N1 amplitude. When the ISI is long and stimuli of different intensity are delivered in the same block, the N1 increases in amplitude with increasing intensity even at high intensities (Gille, Böttcher, & Ullsperger, 1986—Figure 8). These findings suggest that part of the N1 wave evoked by a stimulus of high intensity undergoes a more profound and longer lasting refractory period than the rest of the response. This part is probably that which we have tentatively identified as component 3.

The change in the amplitude of the N1 with increasing intensity varies greatly among subjects. Some subjects have an N1 that continues to increase with increasing intensity at all levels, whereas others have an N1 that saturates or becomes smaller at high intensities. This has led to dividing subjects into "augmenters" and "reducers," a classification supposedly reflecting an individual's characteristic response to stimulation (Buchsbaum, 1976). Much research has therefore attempted to relate this EP phenomenon to aspects of personality and psychopathology. Augmentation and reduction have most often been assessed by measuring the vertex P1-N1 amplitudes in the response to visual or auditory stimuli presented at multiple stimulus intensities. Unfortunately, the type of intensity function can change with the sensory modality (Raine, Mitchell, & Venables, 1981), with the ISI (Picton et al., 1970), with the intensity range over which it is measured (Prescott, Connolly, & Gruzelier, 1984), and with the attentive strategy of the

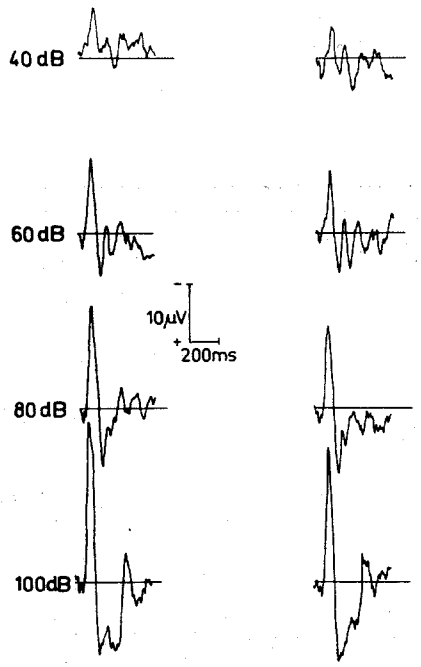

Figure 8. EPs of a typical subject to 40, 60, 80 and 100 dB stimuli, presented in a pseudorandom order at constant 4-s ISIs while the subject was reading a book. The baseline represents the mean of the 200-ms period before stimulus onset. The left side shows data from the 1st session, the right side from the 2nd session. Unpublished figure from the study of Gille, Böttcher, and Ullsperger, 1986, *Zeitschrift für gesamte Hygiene, 32,* 566–568.

subject (Schechter & Buchsbaum, 1973). Moreover, the phenomenon varies with the technique of measurement: individuals who augment at one electrode location for one peak measurement may reduce at another location or for another peak (Prescott et al., 1984). Consequently, a recent review has concluded that the results of augmenting/reducing research have often been "inconclusive or equivocal" (Prescott et al., 1984, p. 32). "Certainly, before the concept is further utilised to distinguish normal from pathological groups there remains a need to demonstrate which EP measures and recording sites will yield reliable, unambiguous and, more importantly, useful measures of an individual's enduring mode of stimulus intensity control" (Prescott et al., 1984, p. 42). The findings of Raine et al. (1981) suggest that "cortical augmenting-reducing is modality-specific and mitigate against the notion of a general mechanism residing in the CNS regulating sensory input" (p. 705).

Even at constant levels of stimulus intensity or perceived loudness, the amplitude of the N1 varies with the tonal frequency of the stimulus (Antinoro & Skinner, 1968; Antinoro, Skinner, & Jones, 1970;

Picton, Woods, & Proulx, 1978b; Stelmack, Achorn, & Michaud, 1977). The N1 decreases with increasing tonal frequency particularly at frequencies greater than 2000 Hz. Some of this effect may be related to the asymmetry of the travelling wave in the cochlea: low-frequency tones activate a much broader region of the basilar membrane than high-frequency tones.

The N1 evoked by binaural tones is slightly larger (about 10%) than that evoked by monaural tones (Davis & Zerlin, 1966; Picton, Woods, & Proulx, 1978b). Nevertheless, the N1 evoked by binaural stimuli increases in amplitude in much the same way as that evoked by monaural stimuli (Butler, Keidel, & Spreng, 1969). The N1 evoked by binaural stimuli is much smaller than what would be expected from the addition of the two monaural responses (Berlin, Hood, & Allen, 1984; Picton, Rodriguez, Linden, & Maiste, 1985). This could result from either occlusion or mutual inhibition between the neuronal populations generating the N1 response. Occlusion occurs when the response elicited by two stimuli presented together is less than the sum of the responses to each stimulus presented separately. Mutual inhibition occurs when the two responses inhibit each other. Some recent evidence from magnetic recordings (Pantev et al., 1986) suggests that there may indeed be some inhibitory interactions between the left and right auditory cortices. The amplitude of the magnetic field response at 100 ms over the temporal cortex contralateral to a monaural stimulus became smaller when the stimulus was made binaural.

At near-threshold levels the N1 amplitude is remarkably larger when a subject counts the auditory stimuli than when reading (Mast & Watson, 1968). This task-related difference is attenuated at higher intensities (Davis & Yoshie, 1963; Gross, Begleiter, Tobin, & Kissin, 1965). Some studies have indicated that the amplitude of the N1 evoked by near-threshold stimuli presented to an attentive subject correlates strongly with sensory magnitude. The N1 amplitude varies directly with the detection of the stimulus and with the subject's confidence in the detection (Squires, Hillyard, & Lindsay, 1973; Squires, Squires, & Hillyard,1975).

Parasuraman and his colleagues (Parasuraman & Beatty, 1980; Parasuraman, Richter, & Beatty, 1982) have recorded auditory EPs during the detection and recognition of a near-threshold tone presented in continuous wideband noise. On half the trials there was a tone and on the other half there was only noise. The subjects reported whether they had heard a tone, using a four-category rating scale, and then chose between two (Experiment 1) or four (Experiment 2) possible frequencies (this choice being made whether or not the subject was sure that the stimulus had occurred). The tones evoked a P300 wave that varied in amplitude with both the confidence in detecting the tone's occurrence and the accuracy in recognizing its frequency. In contrast, the N1 wave, although being larger for the more confident detections, did not vary significantly with the correctness of the recognition. There is some suggestion in the reported data that the N1 amplitude was slightly (but not significantly) larger at higher levels of recognition accuracy. Furthermore, a principal component analysis did not delineate an N1 component as separate from the P300. Nevertheless, the results show that the N1 is much more related to the detection than the recognition of a near-threshold tone.

The latency of the N1 wave increases with decreasing intensity and the N1 evoked near threshold is a small broad wave with a peak latency between 150 and 200 ms. It is difficult to be sure that the cerebral processes underlying this wave are the same as those underlying the N1 at higher intensities. The near-threshold N1 may be overlapped by negative components such as the N2b, which we have already discussed, and the processing negativity, which we shall discuss later.

In subjects who are not particularly attending to the stimuli, the N1 becomes difficult to measure at low intensities. The average difference between the EP threshold and subjective threshold is often less than 20dB (Beagley & Kellogg, 1969; Davis & Zerlin, 1966; Keidel, 1976). This is sufficient for the objective assessment of hearing. However, there are occasional discrepancies between the EP thresholds and subjective thresholds of greater than 60dB (Rose, Keating, Hedgelock, Miller, & Schreurs, 1972). Using interpreters who were blind to the actual stimulus intensity being used, Mendel et al. (1975) measured a mean response threshold in waking adults of 27dB SL with a range of 10 to 45 dB SL. In sleeping subjects, they found the EP threshold to be even higher and more variable. Stapells (1984) reported a mean threshold of 16dB SL and a standard deviation of 17.2 dB in waking subjects. This variability may be partially caused by fluctuations in arousal or attention (Picton et al., 1977). Since it is difficult to control these parameters in young children, EP audiometry with the N1 wave is used only in waking adults. In sleeping subjects and in children the threshold for the N1 response is too high and too variable for clinical use.

Ear of Stimulation

Many studies have investigated asymmetries in the scalp distribution of the late auditory EPs and have attempted to relate these asymmetries to various aspects of the stimulus or to the way in which the stimulus is processed. Prior to reviewing these

findings we shall briefly discuss some physiological explanations for a recorded asymmetry. First, the generators in the two hemispheres, although homologous in function, may be differently located and/or oriented. There are definite asymmetries between the left and right hemispheres in the size and geometry of the auditory cortex (Akesson, Dahlgren, & Hyde, 1975; Geschwind & Levitsky, 1968; Galaburda, Sanides, & Geschwind, 1978). Second, there may be differences in the volume conduction between the two hemispheres. For example, the thickness of bone over one hemisphere may be more or less than over the other hemisphere. These first two explanations are most appropriately used to explain hemispheric asymmetries that persist through various stimulus manipulations, for example, the tendency for the response to be larger over one hemisphere regardless of the ear of stimulation. The third explanation is that there is a change in the amplitude of the response evoked in the different hemispheres caused by different patterns of input to the two hemispheres. This can explain the tendency for the response to be larger over the hemisphere contralateral to the ear of stimulation, since each ear sends a larger number of fibers to the contralateral cortex than to the ipsilateral cortex (Rosenzweig, 1951). Fourth, the response may be larger in one hemisphere not because the input is greater but because there is more processing going on in that hemisphere. This could explain differences in the scalp distribution of the response to the same stimulus when perceived in different ways.

The voltage asymmetries recorded between homologous scalp locations may be much smaller than the actual asymmetries between the hemispheric generators. Because of the broad extent of volume conduction and the high resistance of the skull, and because the auditory EP generators in the two hemispheres are probably similarly oriented, it would take an asymmetry of greater than 5 to 1 at the generator locations in the supratemporal plane before a 2 to 1 asymmetry would be recognizable at the scalp. (These numbers are loosely derived for the C_3-C_4 locations from Figure 1A of Scherg and von Cramon, 1985.)

The N1 wave shows a small but consistent asymmetry when the stimulus is presented monaurally. Price, Rosenblut, Goldstein, and Shepherd (1966), using C_Z–A_1 and C_Z–A_2 electrode montages, recorded the EPs to clicks from 160 normal-hearing subjects. Although the data were variable, they found that the N1 wave was larger in amplitude in the contralateral recording montage than in the ipsilateral recording montage. From their published figure, it would seem that the average asymmetry was on the order of 10%. Because of the montages used in this study, the asymmetry must have been due to some potential difference at the ear electrodes. There are two possible explanations for such a difference. The positivity recorded from below the temporal plane as part of component 1 of the N1 (a dipole with negativity above the temporal plane and positivity below) may have been larger contralateral to the ear of stimulation, because the contralateral cortex is more activated than the ipsilateral. As well, the Ta wave of component 2 or the T-complex, which could be recorded as a positive wave at the earlobe, may have been larger in the temporal lobe contralateral to stimulation.

Using a more extensive array of electrodes, Vaughan and Ritter (1970) found a small but consistent shift toward higher amplitude in the central regions contralateral to the stimulated ear. This response asymmetry was greater over the left hemisphere (7.5%) than over the right (3.8%). Andreassi, DeSimone, Friend, and Grota (1975) recorded the responses to bursts of white noise from C_3 and C_4 using a left earlobe reference. The N1 wave (N2 in their terminology) was larger at C_3 for right-ear stimulation and at C_4 for left-ear stimulation. They also reported that the N1 was larger at C_4 than at C_3 for binaural stimulation. This asymmetry may have been related to their left-sided reference. Picton, Woods, and Proulx (1978b) using a sternovertebral reference found that the N1 evoked by right-ear tones was 20% larger at C_3 than at C_4 whereas the N1 evoked by left-ear tones or by binaural tones was symmetrical between C_3 and C_4.

The asymmetries recorded from the temporal electrodes are usually greater than those recorded from central electrodes. Wolpaw and Penry (1977) found that the T-complex, as well as being earlier and larger over the cortex contralateral to monaural stimulation, was larger over the right hemisphere (about 25%). McCallum and Curry (1980, Figure 1) found a small (10–15%—not reaching statistical significance) contralateral vs. ipsilateral difference for the N1b at central electrodes referred to the contralateral mastoid, and a larger (approximately 25%—statistically significant) difference for the N1c at the midtemporal electrodes. Perrault and Picton (1984) using a sternovertebral reference found that the average T_3/T_4 ratio for the N1c component was 0.7 when tones were presented to the left ear and 1.5 when tones were presented to the right ear, with no significant left-right asymmetry. Connolly (1985) using a linked-earlobe reference found that a temporal negative wave at 120 ms (possibly homologous with the Tb wave of the T-complex) was larger contralaterally than ipsilaterally to the ear stimulated by monaural tones. Furthermore, the left tem-

poral site was more sensitive to this ear effect than the right temporal site. A sternovertebral reference is probably better than the linked-earlobe reference for assessing the asymmetries in the temporal regions since the N1c component can be recorded from the earlobes and since the actual linking of the earlobes can attenuate a field asymmetry (Katznelson, 1981, p. 189).

Scherg and von Cramon (1986a) found that the tangential source dipole underlying the late auditory evoked potentials (component 1) was significantly larger (by about 11%) and earlier (by about 5 ms) in the temporal lobe contralateral to stimulation. They were unable to find any significant ipsilateral vs. contralateral differences for the radial source dipole (component 2) or any significant left-hemisphere vs. right-hemisphere effects for any of their dipole sources. However, there was a fair degree of intersubject variability in the hemispheric effects: it was within the limits of normal for the dipole in one hemisphere to be half the size of the dipole in the other hemisphere.

In conclusion, there are definite asymmetries in the N1 wave related to the ear of stimulation. Component 1 is definitely larger contralaterally—probably by about 10%. Although not conclusive, the magnetic recordings discussed in a preceding section of this paper support this asymmetry. This asymmetry is probably explained by an asymmetry of the input to the auditory cortices. Component 2 is probably larger contralaterally although not all studies have found a significant difference. There is no consistent asymmetry between left and right hemispheres, although there is a fair amount of intersubject variability in the left-right ratios. This may be related to individual differences in the anatomy of the temporal lobe in each hemisphere.

Speech Sounds

Many papers have attempted to relate the asymmetry of the late auditory response to the known functional asymmetry of the hemispheres with respect to speech processing. Unfortunately, the findings have been small and very difficult to replicate (Picton & Stuss, 1984). There are two basic kinds of experiments that have related hemispheric asymmetries of the response to speech processing.

The first type of experiment evaluates the asymmetry of a response to a speech sound. Morrell and Salamy (1971) reported that the response to speech sounds had a larger N1 wave over the left temporoparietal region than over the right. It is difficult, however, to determine how much of this difference was related to speech, since no recordings were made of the response to non-speech stimuli using the same electrode derivations. Grabow, Aronson, Rose, and Greene (1980) were unable to replicate the asymmetry reported by Morrell and Salamy.

A second kind of experiment uses the same stimuli but has the subject attend to either the phonemic or the acoustic aspects of the stimuli. Wood, Goff, and Day (1971) presented syllables, each consisting of a stop consonant and a vowel, at a rate of one syllable every 5 s. In one condition the subjects were requested to discriminate between two equiprobable stop-consonants /ba/ and /da/. In a second condition, the subjects were asked to discriminate between two different fundamental frequencies for the same syllable /ba/. The /ba/ stimulus with a fundamental frequency of 104 Hz was exactly the same in both conditions. The left-hemisphere response to this stimulus differed between the two conditions whereas the right-hemisphere response showed no change. The main difference was that the left-hemisphere N1 peak was larger during the discrimination of stop-consonants than during the discrimination of fundamental frequency. Wood (1975) replicated these results but Grabow, Aronson, Offord, Rose, and Greene (1980) did not.

As well as investigating hemispheric asymmetries, the ERPs may be helpful in delineating the processes involved in the perception of speech stimuli. Lawson and Gaillard (1981), using consonant-vowel (CV) syllables, found that the N1 latency and the RT were considerably shorter for plosive (short consonant-duration) than for non-plosive CVs, the plosive CVs evoking N1 waves with similar latency to those evoked by tones. The authors concluded that "since no differences were found between the tone and the short consonant duration syllables, it appears that the analysing mechanism as reflected by these ERP components was the same for the phonetic and acoustic stimuli. This supports the conclusion reached by Schouten (1980), upon reviewing evidence whether or not a special mechanism for speech perception would exist, that speech and non-speech stimuli are probably perceived in the same way" (Lawson & Gaillard, 1981). Results consistent with those of Lawson and Gaillard have been reported by Woods and Elmasian (1986), who found that the N1 waves evoked by speech or by tones showed similar scalp distributions, and by Hari et al. (in press), who found no difference in the N1m evoked by speech or by non-speech auditory stimuli.

Rate Effects and Short-Term Response Decrements

The N1 wave is exceptionally sensitive to the rate at which the stimuli are repeated. This sensitivity contrasts with the relative resistance to rate shown by earlier (Picton, Stapells, & Campbell, 1981) and later (Woods, Hillyard, Courchesne, &

Galambos, 1980) auditory EPs. Davis, Mast, Yoshie, and Zerlin (1966) presented the first clear description of these temporal effects for the auditory N1. They found that the N1-P2 amplitude of the response to tone pips presented at regular ISIs betweeen 0.5 and 6 s increased with increasing ISI. They estimated that the amplitude would continue to increase to ISIs of more than 10 s. These findings have been confirmed in many other papers (Nelson & Lassman, 1968; Milner, 1969; Picton et al., 1977). Milner (1969) found that the N1-P2 amplitudes to 80dB tones presented at intervals between 0.75 and 13.5 s could be fitted with an exponential curve having a time constant (the ISI at which the amplitude reached 63% of maximum value) of 4.3 s. Although not reported, the curve shown for 60dB tones appears to have a shorter time constant. Picton et al. (1970) and Nelson and Lassman (1973) also showed that the N1-P2 amplitude reaches a maximum value at shorter intervals when the stimulus is less intense. These data suggest that for intense stimuli the recovery period of the N1-P2 amplitude must be very long. More recently, Hari et al. (1982) observed that the amplitude of the vertex N1 evoked by 80dB SPL tones increased when the ISI was prolonged from 8 to 16 s. We therefore do not know at what interval the vertex N1 to loud stimuli becomes saturated.

Davis et al. (1966) also presented pairs of stimuli (cf. Allison, 1962) at ISIs of 0.5 s and inter-pair intervals of 3.0 s. They found that the N1-P2 amplitude in response to the second stimulus was one third the amplitude of the response to the first stimulus. These temporal effects were similar to those obtained by using constant ISIs of 0.5 or 3.0 s.

These effects may be caused by habituation since habituation is greater when the rate of stimulation is faster. Habituation is the progressive decrease in a response with repetition of the stimulus; it differs specifically from other causes of reduced responsiveness by being susceptible to "dishabituation" (Thompson & Spencer, 1966; Thompson, Groves, Teyler, & Roemer, 1973). Dishabituation is the recovery or partial recovery that occurs in a response without any change in the stimulus eliciting the response (or in the ISI). Dishabituation is caused experimentally by inserting a dishabituating stimulus into the train of habituating stimuli. Spontaneous dishabituation is presumably a response to some internal stimulus or change in state. The recovery of the response with a change in some characteristic (for example, pitch) of the stimulus eliciting the response does not unequivocally demonstrate habituation since the reduced responsiveness may be specific to that particular stimulus characteristic (Loveless, 1983; Graham, 1973; Öhman & Lader, 1977).

Habituation is usually evaluated by observing the decrease in a response with stimulus repetition. Because of the requirements of averaging, the change in an ERP from one stimulus to the next can be observed only if one presents multiple blocks of stimuli (separated by long inter-block intervals) and then averages the ERPs across the blocks separately for each position in the block. This technique was first used by Ritter, Vaughan, and Costa (1968) who presented "runs" of brief tones separated by inter-run intervals of several minutes. When the ISI within a run was 2 s, the N1-P2 amplitude decreased rapidly over the first few stimuli and then stabilized at about half the amplitude of the first response by the second, third or fourth stimulus, depending on the subject. At ISIs of 10 s, there was no significant change in amplitude across the run. When the frequency of the tone presented at an ISI of 2 s was changed from 1000 to 2000 Hz, the response to the first stimulus having the new frequency contained as its most striking aspect a large positive wave with a peak latency of about 350 ms. A similar positive wave occurred in response to the first stimulus in the run. From the published data (their Figures 3 and 4), it is not possible to determine whether the N1 amplitude to the stimulus initially deviating in frequency was larger than for the preceding stimuli. The subsequent stimuli evoked responses that were not significantly different from those preceding the stimulus change. Ritter et al. concluded that the decrement in the N1-P2 over time did not represent genuine habituation, since it could not be reversed by "appropriate dishabituation procedures," but rather reflected "refractoriness" within the auditory system (cf. the previously mentioned study of Davis et al., 1966). Fruhstorfer, Soveri, and Järvilehto (1970), however, claimed that this result "only demonstrates that generalization of habituation to different types of auditory stimulation has occurred" (p. 159).

The decrease in the N1 during a brief train of auditory stimuli was soon confirmed by other investigators. Roth and Kopell (1969) found that the N1-P2 amplitude of the response to the first stimulus in a train of five was significantly larger than that to the following stimuli, with the difference being greater for more rapid rates and for more intense stimuli. Öhman and Lader (1972) found a response decrement over a brief train with ISIs of either 3 or 10 s.

Fruhstorfer et al. (1970) presented trains of 8 clicks using ISIs of 1 or 3 s and inter-train intervals of 100 s. They recorded the auditory EPs between an electrode near the vertex and one on the forehead. The N1 deflection to the first stimulus of a train was very large, about 10 times that elicited by the second stimulus for trains with the 1-s intervals

and more than 2 times for the trains with the 3-s intervals. The amplitude decreased further until the third or fourth stimulus and then stabilized. The authors interpreted this reduction in terms of short-term habituation but did not try to demonstrate dishabituation. In a following paper, Fruhstorfer (1971) found that a somatosensory stimulus inserted into the train of clicks evoked an N1 wave that was larger than that evoked by the clicks and larger than what would have been evoked if it had been preceded by a train of somatosensory stimuli. These results are illustrated in Figure 9. The next standard stimulus after the deviant stimulus showed an N1 that was not significantly different from the N1 prior to the deviant stimulus. Similar results were obtained when an auditory stimulus was inserted into a train of somatosensory stimuli.

The increase in amplitude of the N1 when the modality of the stimulus is changed does not clearly demonstrate dishabituation since it may be explained on the basis of specific refractoriness. The attenuation of the N1 with stimulus repetition is to some extent specific to the precise characteristics of the repeating stimulus and to some extent generalized to other stimuli. This generalization is demonstrated by Fruhstorfer's (1971) findings that the deviant stimulus evoked an N1 that was not as large as the N1 it would have evoked if not preceded by the repeating stimuli in the other modality. Cross-modal generalization of the refractoriness of the N1 clearly occurs. Many studies have shown that the N1 response to an auditory stimulus can be reduced by preceding it by a stimulus in another modality (Davis et al., 1972; Gjerdingen & Tomsic, 1970; Hay & Davis, 1971; Kevanishvili, Pantev, & Khachidze, 1979; Larsson, 1956, 1960a, 1960b; Rothman, Davis, & Hay, 1970). This intermodal refractory effect may explain some of the findings of Öhman and his colleagues (MacLean, Öhman, & Lader, 1975; Öhman & Lader, 1972). They presented successive trains of 10 acoustic stimuli with inter-train intervals of 30 s. In contrast to the results of Fruhstorfer, the response to the first stimulus was not much larger than the response to the subsequent stimuli. This was probably due to intermodal effects of a red lamp that was switched on shortly before the first stimulus of each train.

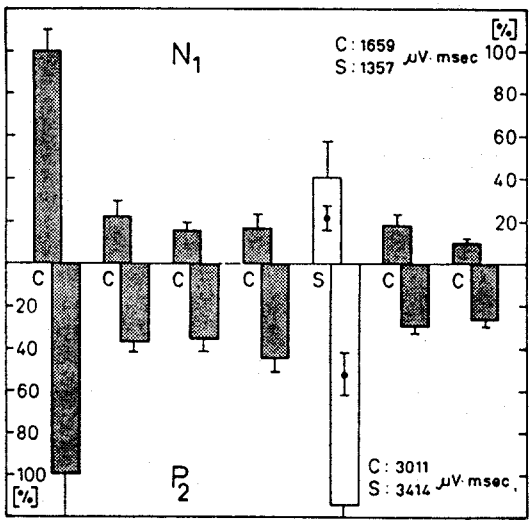

Figure 9. Measurements of the EPs to clicks (C) presented at a rate of 1/s in trains of 8 stimuli with inter-train intervals of 100 s. Both the N1 and P2 waves (measured as the area under the wave in μV-ms and expressed as a percentage of the response to the initial stimulus) decreased dramatically after the first stimulus. The response to a somatosensory stimulus occurring at the fifth position in the train (unshaded bars) was larger than the click EPs and larger than the somatosensory EP if it had been preceded by a train of somatosensory stimuli rather than clicks (the mean standard deviations within the unshaded bars). From Fruhstorfer, 1971, *Electroencephalography & Clinical Neurophysiology, 30*, 306–312. Reprinted with the permission of the author and the publisher.

Within the auditory modality there is both specificity and generalization of the refractory effects. Butler (1968) found that changing the stimulus could increase an N1 that had been attenuated by stimulus repetition. He recorded the EP to a 1000 Hz 600-ms tone presented every 5 s. The intervals between these 1000 Hz "test" tones contained either no tones or 3 "intervening" tones. The amplitude of the response to the test tone was largest when there were no intervening tones. When these did occur, the amplitude of the response to the test tone varied with the frequency of the intervening tone, being smallest when this frequency was identical to that of the test tone. This selective adaptation has been replicated (Butler, 1972a; Picton, Woods, & Proulx, 1978b). Butler proposed that the decrement in the N1-P2 response was specific to the frequency of the stimulus: "When the frequency of the intervening stimuli was progressively removed from that of the test stimuli, . . . the population of neural units activated by the two categories of stimuli became more and more disparate. Hence, each time the test stimulus was presented, neural units not activated by the preceding three intervening stimuli were brought into play" (p. 949). Butler (1972a) suggested that the response was generated in the auditory cortex and that, although the cortex was tonotopically organized, each frequency had a widespread neural representation.

Very recently, however, Näätänen, Sams et al. (1987) have shown that under some conditions the N1 can be highly stimulus specific. Test tones of 1000 Hz subjectively located in the center of the head were presented with equiprobable intervening

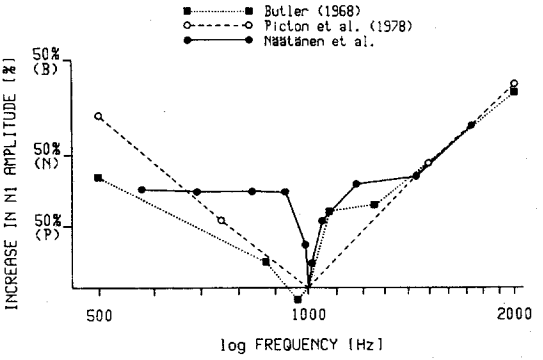

Figure 10. A comparison of the relative frequency specificity of the N1 obtained by Butler (1968), Picton, Woods, and Proulx (1978a), and Näätänen, Sams et al. (1987). The figure shows the increase in amplitude of the response as a function of the frequency separation between the test and intervening tones. The increase was calculated in relation to the amplitude when the test and intervening stimuli were both 1000 Hz. Three different types of measurement were used (N1-P2 by Butler, baseline-N1 by Picton et al., and P1-N1 by Näätänen et al.). In order to facilitate comparisons, three different scales are used (as shown on the vertical axis by B, N, and P). Butler's (1968) amplitudes were from his Table II; the other amplitudes were from the experimental records.

tones at a constant ISI of 460 ms. The frequency of the intervening stimulus varied between different blocks from 578 to 1728 Hz. Figure 10 presents the vertex P1-N1 amplitudes of the test-stimulus EP as a function of the frequency of the intervening stimulus. The amplitudes are expressed as a percentage of the amplitude at 1000 Hz and compared to the results of Butler (1968) and Picton, Woods, and Proulx (1978a). The sharp stimulus-specificity curve for frequency in the Näätänen et al. data may be related to the very short ISI and the equiprobability of the test and intervening stimuli. The frequency specificity is even sharper when measured at the midfrontal electrode.

In a very important study involving periodicity pitch, Butler (1972b) demonstrated that the activation of frequency-specific neurons during the N1 wave does not underlie the perceived pitch of the auditory stimulus. When a tone of a particular carrier frequency is amplitude modulated at a lower frequency, the perceived pitch corresponds to the modulation frequency rather than the carrier frequency (Ritsma, 1970). Butler used a test stimulus with a carrier frequency of 1000 Hz and modulated its amplitude at 200 Hz. Perceptually, the pitch of this stimulus was in the neighbourhood of 200 Hz. The N1-P2 amplitude to these test stimuli was considerably reduced when the intervening stimuli were pure tones of 1000 Hz but was little affected when the intervening stimuli were pure tones of 200 Hz.

Butler (1972c) also studied the effects of spatial location on the auditory N1-P2 response. He observed that the response to sounds alternating between two different loudspeakers was larger than that elicited by sounds always originating at the same location. This amplitude enhancement was greater when the two loudspeakers were located in the horizontal plane than when they were equally widely separated in the vertical plane.

In an elegant study, Butler, Spreng, and Keidel (1969) showed that the stimulus specificity of the N1-P2 rate effect involved the central rather than the peripheral auditory system. They presented a monaural test stimulus with intervening stimuli that were either monaural (to the same ear as the test stimulus) or binaural. The actual physical stimuli presented as intervening stimuli to the test ear were exactly the same in both conditions: the binaural stimulus consisted of an additional and equal stimulus in the opposite ear. The response to the test stimulus was larger when the intervening stimulus was binaural. These results have been recently replicated by Picton et al. (1985). Butler et al. concluded that the reducing effect of short ISIs is maximum when the neural patterns of excitation triggered in the central nervous system by the repeating stimuli are the same. These results may be related to the previously mentioned finding that a binaural stimulus elicits a smaller N1 than would be expected from the sum of the monaural responses. There may be some binaural inhibition that would reduce the N1 response in the cells responding to one ear and thus make them less fatigued when a subsequent monaural stimulus is presented. Whatever the mechanism, it must occur within the central nervous system where the inputs from the two ears can interact.

The effects of changing the intensity of the stimulus can indicate the type of process underlying the response decrement. Butler (1968) found that, when the intervening stimulus was higher in intensity than the test stimulus, the response to the test tone was either the same or smaller than when all stimuli had the same intensity. On the other hand, when the intervening stimulus was lower in intensity than the test stimulus, the response to the latter was larger than when all stimuli had the same intensity. Butler concluded that new neural units were activated by the test stimulus when its intensity exceeded that of the intervening stimuli, whereas no new neural units were activated by the test stimulus when its intensity was the same or less than that of the intervening stimuli.

Megela and Teyler (1979) evaluated the change in N1-P2 amplitude over brief trains of tones presented at ISIs of 1 s and inter-train intervals of 5–10 s. A test tone with a different intensity from the

repeating tone was given randomly after 5–9 tones. They found that the N1-P2 amplitude for a loud test tone was larger when it followed a series of soft tones than when it followed a series of loud tones. The opposite effect was not true, the response to a soft tone was no larger when it followed a series of loud tones than when it followed a series of soft tones. These results suggest that the process underlying the decrement in N1-P2 with stimulus repetition involves decreased synaptic efficiency (cf. Thompson & Spencer, 1966) and not a comparison of incoming information to a neuronal model (Sokolov, 1963, 1975). A soft stimulus would activate only a subset of the synapses activated by the loud stimulus and thus the generalization of the decrement from soft to loud would be less than the generalization from loud to soft. Decreasing the intensity of the stimulus should be similar to increasing the intensity in a neuronal model. It is difficult, however, to determine the specific significance of these results for the N1 wave. Megela and Teyler obtained significant findings on their N1-P2 measurement but not on their baseline-N1 measurement.

Woods and Elmasian (1986) obtained data which they interpreted as suggesting that "short-term habituation is a function of the acoustic resemblance of successive stimuli." They presented sequences of 6 identical auditory stimuli but in half of these sequences, the fifth stimulus was replaced with a different stimulus. The stimuli used were different kinds of tones and speech sounds, administered in different combinations of standards and deviants. The N1 recorded with maximal amplitude at the vertex decreased rapidly with stimulus repetition to 35% of the response to the first stimulus in the sequence for ISIs of 0.5 s, and to 60% for ISIs of 1 s. The amplitude of the temporal negativity measured some 50 ms later than the vertex N1 showed a similar decrement. The decrement in the vertex N1 amplitude with stimulus repetition was greater for speech than for non-speech sounds. The N1 was larger to the deviant stimuli than to the standard stimuli occurring in the 5th position. When the deviant stimulus differed from the standard stimuli in phonetic structure (but was more similar acoustically), "cross-habituation" was stronger (the amplitude recovery was smaller) than when the two stimuli were different tones (less similar acoustically). The N1 amplitude to the standard stimulus immediately following the deviant showed no amplitude recovery (dishabituation) in comparison with the response to the standard stimulus immediately preceding the deviant.

Woods and Elmasian suggested that three different processes contribute to the "short-term habituation" of the auditory evoked potential: 1) a modality nonspecific process revealed by the intermodal interactions; 2) a stimulus-specific process, reflected in the more marked amplitude reduction when identical auditory stimuli are repeated than when succeeding auditory stimuli differ in some physical characteristic; and 3) a process that results in a memory of the repeating stimulus and causes the generation of a MMN when a deviant stimulus occurs.

Roth (1973) presented a sequence of auditory stimuli at constant ISIs of 1 s. In some runs a 1000 Hz tone was the frequent stimulus and a burst of white noise was the infrequent stimulus. In other runs the two stimuli exchanged their roles. The experiment used three different probabilities for the deviant stimulus. There was no evidence for dishabituation in the ERP to the standard stimulus following a deviant stimulus. Sams, Alho, and Näätänen (1984) obtained similar results. In these cases, the prolonged interval between the stimulus that follows the change and the stimulus that precedes the change should be taken into account when assessing whether dishabituation has occurred. Because of generalization effects, the insertion of a different stimulus into a train of stimuli at short ISIs without changing the temporal parameters of the repeating stimuli would almost certainly decrease rather than increase the response to these stimuli.

As is evident from this section, the literature on the short-term decrement in the N1 often confuses the testing for dishabituation with evaluating the degree of generalization of the decrement. True dishabituation occurs when a decremented response returns toward its original value without any change in the stimulus evoking the response, that is, this return is caused by some additional stimulus or by a change in a preceding stimulus. Since dishabituation has not been demonstrated, and since it is probable that it will not be demonstrated, we prefer to consider these short-term decrements in the auditory N1 as evidence of a prolonged refractory period unrelated to habituation as originally suggested by Ritter et al. (1968). Webster (1971) proposed that such short-term changes in an EP might be considered simply as "rate effects." The recovery of the N1 amplitude varies only with the time elapsed from the preceding stimulus and the dissimilarity between the stimuli, there being no apparent mechanism for expediting this process.

It is difficult to determine the specific relations of these short-term decrements to the different components of the N1 wave since the research has for the most part considered the N1 a unitary process. Both component 1 and component 2 appear to show significant rate effects and significant intramodal generalization. It is possible that the particularly

large N1 occurring after a long interval (for example, as the first stimulus in a train) is the result of a superimposition of component 3 on the field generated by component 1. Component 3, mediated as it is by multimodal brainstem pathways, may also be the component that is especially susceptible to intermodal refractory effects. Component 4, the mismatch negativity, is related to ISI in an opposite manner to the other three components. It is larger when the interval between the deviant stimulus and the preceding standard stimulus is shorter.

The physiological mechanisms underlying these short-term decrements are not known. It is unlikely that they are caused by actual refractory periods in simple cellular mechanisms. Synapses do not usually get tired that quickly or recover that slowly. Complex neuronal circuits are probably responsible for these effects. Reduced temporal uncertainty about the next stimulus has been considered as one explanation for the prolonged refractory period of the N1. We shall consider this later in this paper. Picton, Campbell, Baribeau-Braun, and Proulx (1978) suggested that the N1 "might reflect the activation of the processes necessary to the evaluation of incoming information, such processes remaining active for a period of time equivalent to the relative refractory period . . . . The analysis of a second similar stimulus occurring during this period would only require partial reactivation of these processes" (p. 207).

Long-Term Response Decrements

The decrement in the auditory EP that occurs over a longer period of time may be more closely related to genuine habituation. Bogacz, Vanzulli, and Garcia-Ausst (1962) presented 90dB clicks at constant ISIs of 1 s and found that the N1 waxed and waned in amplitude and slowly decreased over several minutes. The EEG was monitored to ensure that the subjects remained awake. The response appeared to dishabituate when flashes were inserted into the train of auditory stimuli. No statistics were used to support this conclusion, however, and the data of only one subject were shown. Interestingly, the dishabituation effect was reversed when the flashes were made very intense (perhaps due to some cross-modal refractory effect).

The long-term decrement in the N1 has been confirmed by many other reports (reviewed by Callaway, 1973; Loveless, 1983). Roeser and Price (1969) maintained alertness by having the subjects count the number of stimuli occurring between occasional visual signals, and measured the N1-P2 response to 500 Hz tones presented at a rate of 1/2.3 s over a period of 2 hrs. They found that the response decreased over the first half hour and then remained stable. Weber (1970) reported that the habituation was greater for tones of longer duration. Bothe, Zahn, and Elfner (1974) found that the N1-P2 amplitude decreased over a period of 10 min, with the decrement being greater for simple stimuli than for complex stimuli. Picton, Hillyard, and Galambos (1976) reported habituation over the first 10–30 min of stimulation in subjects who stayed alert by reading. The response could not be dishabituated by giving irrelevant shock stimuli, but the response could be "dishabituated" by asking the subject to attend to the stimuli in order to perform a difficult frequency discrimination.

Salamy and McKean (1977) found that the N1 evoked by 55dB SL clicks presented at a rate of 1/s decreased in amplitude over a period of several minutes, the rate of decrease being greater when the subject was reading than when the subject "did nothing." They reported being able to dishabituate the response, averaged over 50 or 100 consecutive trials, by increasing the intensity of the stimulus or by either increasing or decreasing the ISI during these trials. However, only an amplitude recovery occurring during the period of decreased ISI would provide unequivocal evidence for dishabituation since ERPs tend to be larger when the ISI or intensity is increased. Unfortunately, the data involving the ISI decrease were illustrated only for one subject. Nevertheless, the increase in amplitude of the response when the intensity was increased was considerably larger than could be expected on the basis of the 3dB increase in intensity.

Rust (1977) compared the habituation of the auditory EP and autonomic responses using a 95dB SPL tone presented at an ISI of 33 s. He found that the N1-P2 amplitude decreased over the first 5 min of stimulation but not thereafter. The N1-P2 amplitude correlated with the magnitude of the galvanic skin response and the heart rate response to the tone, suggesting a relation to the orienting response.

The long-term and short-term decrements of the N1 can be examined together by repeating brief trains of stimuli and comparing the responses across the trains as well as across the positions within a train. Fruhstorfer (1971) reported that the large N1 to the first stimulus in a train, presented after a silent interval of 54 s, decreased as the session progressed. Öhman, Kaye, and Lader (1972) found a slow decrease in the N1-P2 amplitude across trains. Similar results were obtained by Öhman and Lader (1972) and by Woods and Elmasian (1986). In the Öhman and Lader study, the decrease occurred both when the subjects paid attention and when they did not, and the decrease was not associated with any change in skin conductance. In the Öhman et al.

(1972) study, however, the decrease in the N1-P2 amplitude was accompanied by a decrease in the skin conductance. Very recently, Woods and Courchesne (1986) have provided evidence that the long-term and short-term decrements are independent, thus suggesting that two separate mechanisms are involved.

We conclude that the N1 decreases in amplitude during a long sequence of repeating stimuli. Although some of this effect may be due to a change in the subject's state caused by monotonous stimulation, not all studies employing some measure of arousal, such as skin conductance, have observed such a change (Öhman & Lader, 1972), and in other studies the time course of such a change has differed from the time course of the N1 change (Bogacz et al., 1962; MacLean et al., 1975). Moreover, behavioral performance and endogenous potentials such as the CNV can remain at a constant level during the long-term decrement of the N1 (Näätänen & Gaillard, 1974; Woods & Courchesne, 1986). There is some evidence that the N1 decrement can be reversed by dishabituation, but this is not fully convincing. Nevertheless, the time course of the decrement in these long sessions suggests that it may represent a genuine habituation. The component of the N1 most affected during this process is probably component 3.

Subject Factors

Temporal and Event Uncertainty

In 1973 Schafer and Marcus reported that the EP to an auditory or visual stimulus that was triggered by the subject pressing a button was smaller than that evoked by a stimulus presented by a machine. They attributed this effect to temporal uncertainty (Klemmer, 1956), since "the subjects possessed complete foreknowledge of stimulus timing when they stimulated themselves" and "no foreknowledge when the machine delivered the stimuli randomly in time" (p. 176). Furthermore, the size of this effect appeared to vary with the intelligence of the subject, a more intelligent subject showing a greater reduction in amplitude under the self-stimulation condition. The N1 reduction with time certainty probably stems from some dampening of the relatively nonspecific component 3, since the N1 under time-uncertainty conditions, unlike the N1 under time-certainty conditions, was attenuated by moderate doses of ethanol and was vulnerable to other changes in state. Moreover, the visual ERP showed the self-stimulation effect when recorded over the vertex but not when recorded over the occiput, this again suggesting that the nonspecific component of N1 was affected.

The self-stimulation results were corroborated and extended by McCarthy and Donchin (1976), Braff, Callaway, and Naylor (1977), and Schafer (1982). McCarthy and Donchin showed that the EPs recorded during self-stimulation contained a slow negative shift prior to the stimulus, but that the reduction in the EP with self-stimulation was not caused by any overlap with motor-related potentials. By using two different auditory stimuli, one of which occurred more frequently than the other, they were able to distinguish the effects of temporal uncertainty (when an event occurs) from event uncertainty (which event occurs). They found that the N1 was increased under conditions of either temporal or event uncertainty but that the P3 wave occurred only under conditions of event uncertainty and only in response to the more infrequent stimulus.

Schafer, Amochaev, and Russell (1981) evaluated the effect of a subject knowing when a stimulus would occur (temporal uncertainty) independently of any self-stimulation effect. Subjects were asked to press a button in response to a tone that occurred regularly every 10 s either coincident with a visually displayed counter reaching zero or without any temporal relation to the counter. The N1 amplitude was larger and the N1 latency longer in the time-uncertainty condition. However, in the time-certainty condition, the task was less demanding and the subject did not need to attend to the auditory stimuli for response initiation. The results may therefore be confounded by effects of task difficulty and attention. Furthermore, considering the long intervals between the auditory stimuli, the N1 amplitudes in this study were very small, on the order of only 1–2 μV. This suggests strong intermodal refractory effects of the visual stimuli upon the auditory N1 response (probably the nonspecific component).

Wastell, Kleinman, and MacLean (1982; Wastell, 1980) have suggested that diminished temporal uncertainty may explain the reduction in the N1-P2 waves of the EP at short stimulus intervals. The idea is that it is much more difficult to predict the moment when a stimulus occurs if the ISI is long. Näätänen, Muranen, and Merisalo (1974) evaluated the ability of subjects to predict time intervals by having them press a button after an estimated duration of 0.5, 1, 2, or 4 s. For short durations, the timing of the button-presses was quite accurate, but for the 4-s duration the timing was very variable. With long ISIs the subject probably seldom experiences "peaks of expectancy" just prior to the stimulus. Time uncertainty is large when the first stimulus of a train occurs after a long inter-train interval and according to Wastell, this could ex-

plain the very large N1 to the first stimulus of a sequence—the "first stimulus effect."

Similarly, Loveless (1983) concluded that "the temporal information content of the evoking stimuli is the critical variable underlying fast habituation as well as temporal recovery" (p. 76). According to him, event uncertainty is relatively low when the subject is exposed to repeated stimuli of the same kind but it is their precise moment of occurrence that is imperfectly known. Therefore the occurrence of a stimulus resolves temporal rather than event uncertainty. If the subject has to construct and maintain a model that predicts the stimulus timing, then, when a train of stimuli is presented, "temporal uncertainty associated with the first stimulus is necessarily greater than that associated with later stimuli, so that the model is then maximally deficient and there will be a large first stimulus effect. As a train progresses, the model is increasingly refined until temporal uncertainty reaches a minimal value . . ." (p. 76).

Öhman et al. (1972) reduced temporal uncertainty by switching on a red lamp about 3 s before the first stimulus of a train of clicks presented at ISIs of 3 s. Under these conditions the amplitude of the response to the first stimulus of the train was greatly reduced and was not much larger than that to the subsequent stimuli. This reduction, however, could also be explained, as mentioned earlier, by cross-modal refractory effects between the light and the first click.

In order to dissociate the effects of ISI from temporal uncertainty, Wastell (1980) provided subjects with visual cues about when an auditory stimulus could occur. A spot on an oscilloscope revolved around a circular course once every 3 s. One or three stationary spots above the rotating spot indicated whether a brief tone would occur, when the rotating spot reached the fixation point after one or three revolutions. The subjects pressed a button in response to the tone, the timing of which varied between ± 150 ms from the moment that the rotating spot reached the fixation point. In an unclocked condition there were no visual stimuli. The N1 amplitude was significantly smaller in the clocked condition than in the unclocked condition. Furthermore, the N1-P2 was not significantly larger when the preceding interval was 9 s rather than 3 s in the clocked condition but was so in the unclocked condition. Sample results from 2 subjects are shown in Figure 11. The average vertex N1 amplitudes at 3- and 9-s intervals were 4.1 and 4.1 μV, respectively, in the clocked condition, and 14.7 and 20.1 μV in the unclocked condition (Wastell, personal communication, December, 1985). In a "control" experiment a rotating spot was present but its

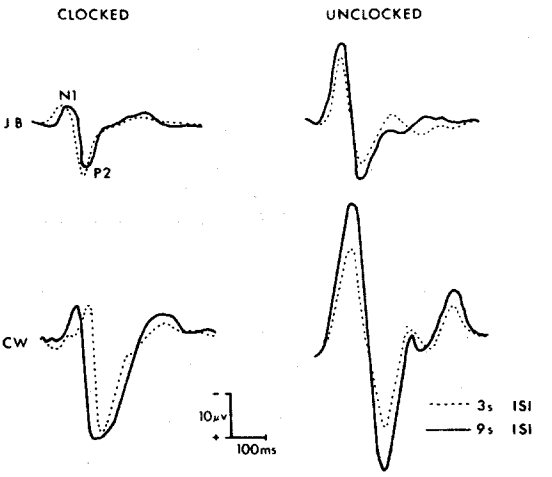

Figure 11. Vertex evoked potentials for 2 typical subjects for the 3- and 9-s ISIs under "clocked" and "unclocked" conditions. From Wastell, 1980, in Barber (Ed.), *Evoked potentials*, MTP Press. Reprinted with the permission of the author and the publisher.

cycle (6.7 s) was not related to the timing of the tone. In this condition the N1 amplitudes at the 3- and 9-s intervals were 5.6 and 9.5 μV respectively (Wastell, personal communication, September, 1986). These findings indicate two separate processes. First, there were significant cross-modal refractory effects causing the auditory N1 to be much smaller when there were concomitant visual stimuli. Second, the results suggest that the larger N1 amplitudes at longer ISIs are actually due to increased temporal uncertainty, since when this was eliminated by the clock there was no ISI effect.

If the effects of ISI on the auditory N1 wave are due to the concomitant changes in the subject's uncertainty about the timing of a stimulus, the response to stimuli presented irregularly should be larger than the response to regularly presented stimuli. Unfortunately, the data reported in the literature are equivocal on this point. Nelson, Lassman, and Hoel (1969) found no differences in the N1-P2 amplitudes for tones presented regularly every 2 s and for tones presented at intervals between 1 and 4.5 s with a mean of 2 s. These findings were confirmed in later studies (Nelson & Lassman, 1977). The results of both studies suggest that temporal uncertainty does not necessarily increase the amplitude of the response. Rothman et al. (1970) found a small but apparently insignificant enhancement of the N1-P2 response to tones when they were presented at an irregular ISI. The amplitude of the response varied directly with the immediately preceding interval, and to a very small extent with the interval before that. They suggested that the am-

plitude was more affected by a prolonged recovery of excitability than by any unpredictability of timing. Öhman et al. (1972) found a small increase (about 10%) in the N1-P2 amplitude when the ISI was made irregular. Irregularity in stimulus interval may therefore increase the N1 response but not as much as might be expected if the interval effect were caused only by the subject's ability to predict stimulus timing.

It is, of course, essential not to change the overall stimulus rate when making the ISI irregular or random. The results of Tyberghein and Forrez (1969) have been quoted as showing that the auditory EP is larger when the stimuli are presented randomly than when presented periodically. However, their overall stimulus rate was quite different between conditions: the ISI varied between 1 and 3 s during their random condition and was constant at 1 s for their periodic condition. Furthermore, since the N1 amplitude is not linearly related to ISI, the perfectly designed protocol for irregular stimulation would use a non-uniform distribution of ISIs.

Refractory effects determine the amplitude of the response to stimuli that occur with equally predictable (or unpredictable) timing. Roth et al. (1976) recorded the EPs to tones presented at randomized ISIs of 0.75, 1.5, and 3.0 s. In a reaction-time (RT) condition the subject pressed a button in response to an occasional softer tone, and in a reading condition he read a book. The N1 increased in amplitude as the immediately preceding interval increased. These results cannot be explained by the subject's greater time uncertainty when 3 s than when 0.75 or 1.5 s have elapsed from the preceding stimulus. Several studies have shown the shortest RTs for the longest of different randomized equiprobable intervals (Elithorn & Lawrence, 1955; Näätänen 1970a, 1971; Nickerson & Burnham, 1969; for a review, see Niemi & Näätänen, 1981). This effect, which is opposite to the ISI-RT relationship for (within-block) constant intervals, is due to the informative nature of the flow of time after the preceding stimulus, which results in an increasing "conditional probability" for the next stimulus (Elithorn & Lawrence, 1955; Nickerson & Burnham, 1969). Consequently, toward the end of the 3-s ISI, there was, presumably, a decrease in the time uncertainty of Roth et al.'s subjects. However, as already mentioned, the N1 amplitudes were largest for this interval. A further aspect of their data supporting the refractoriness interpretation was that the same pattern of EP results occurred in both reading and RT conditions. While reading, the subject presumably does not form expectancies, at least not consciously, concerning the moment of presentation of the irrelevant auditory stimuli.

Butler (1973) measured the N1-P2 amplitude in the response to brief tones at a constant ISI of 5.5 s. Each of these test stimuli was preceded by a constant silent period of 930 ms. The rest of the interval contained no stimuli, a block of tones at a rate of 1, 5, or 10/s, a continuous tone, or a "conditioning" stimulus immediately before the silent period. The response to the test stimulus was most reduced by the intervening repetitive stimuli at 5 or 10/s. These temporal effects cannot be explained by time uncertainty. The latter was probably smallest for the 1/s condition with all the ISIs in the stimulus block having approximately the same duration.

The results of Rothman et al. (1970) also support the refractoriness explanation. They used three mean rates of auditory stimulation: 2, 4, or 8 stimuli/10 s. One of the ISIs in each condition equalled 2.5 s and EPs after that ISI were compared between the blocks of differing mean rates. The N1-P2 amplitude was smallest in the block with the fastest rate. In that block the 2.5-s ISI alternated with three consecutive ISIs of 0.83 s which certainly made the stimulus presented at the end of the 2.5-s ISI much less temporally predictable than when the stimuli were in the block with the regular rate of 1/2.5 s (4 stimuli/10 s).

Ford and Hillyard (1981) recorded the auditory EPs to an occasional noiseburst that occurred prematurely after an interval of 300 ms instead of the usual interval of 600 ms. These noisebursts evoked a negative-positive response when the subject either attended to or ignored the stimuli. The peak latency of the negative wave was 20–30 ms longer than the latency of the regular N1. These waves, called N_E-P_E by the authors, are perhaps similar to the N2-P3a waves evoked by unpredictable changes in the intensity or frequency of regular stimuli (Näätänen et al., 1982; Ritter et al., 1968; Snyder & Hillyard, 1976; Squires et al., 1975). They may represent a temporal mismatch process. (Similar responses to "too early" vibratory stimuli and to "too early" visual stimuli have been observed by Klinke, Fruhstorfer, and Finkenzeller (1968), and by Loveless (1986), respectively.) Due to these large waves, it was not possible to measure separately the N1 to the early stimulus, whereas to the unexpectedly late stimulus the N1 was enhanced to the size observed when this interval was the regular interval of a sequence. There was, however, a latency reduction in response to the late stimulus.

In conclusion, many studies clearly demonstrate that temporal refractoriness can affect the amplitude of the N1 component of the auditory EP independently of temporal uncertainty. Moreover, the studies reviewed in the preceding part of this paper have demonstrated that the decrement in N1 am-

plitude with stimulus repetition depends on parameters of the event that have nothing to do with its timing: the physical similarity between the repeating stimuli and the intensity of the stimuli. On the other hand, time-uncertainty factors must also play an important role in explaining stimulus-repetition effects on the N1. Reduced time uncertainty also decreases other physiological responses (Lykken & Tellegen, 1974). The skin conductance response (Grings, 1973) and the cardiac acceleration (Lykken, Macindoe, & Tellegen, 1972) elicited by an electric shock are smaller with more predictable timing. Epstein (1973) found a smaller galvanic skin response to a "punishing sound" when the timing of the sound was predictable than when not predictable. Whereas the subjects rated the loudness of the sound as the same in both conditions, they rated their reactivity as having been higher under time uncertainty.

The evidence supporting the effect of time uncertainty on the N1 wave derives mainly from studies of the response occurring after silent intervals of more than 3 s. It is therefore possible that refractoriness is the major determinant of the amplitude of components 1 and 2 but that both temporal uncertainty and refractoriness determine the amplitude of component 3.

Selective Attention

Several early experiments (reviewed by Näätänen, 1967, 1975) suggested that the N1 wave of the auditory EP was larger when the subject was attending to the stimuli than when ignoring them. There were two basic kinds of studies. Those belonging to the first category usually managed to demonstrate that the N1 amplitude was larger when the eliciting stimuli were attended to than when the identical stimuli were ignored in a separate experimental condition. In the second kind of study, the relevant and irrelevant stimuli were presented in the same block. Spong, Haider, and Lindsley (1965) alternated flashes and clicks at 1-s intervals. When the subject's task was to attend to the clicks and to ignore the flashes, the clicks elicited much larger EPs than those elicited by the same clicks when the subject was attending to the flashes. In turn, the flash-elicited potentials were larger when the task involved the visual stimuli. Such a paradigm, however, did not dissociate the effects of selective attention from those due to nonspecific arousal. The subject could predict the timing of the stimuli to be attended and become phasically more aroused before these stimuli than before the equally predictable irrelevant stimuli. Näätänen (1967, 1970b) provided evidence that arousal-related changes (a decrease in the EEG amplitude and the development of the CNV) preceded the relevant but not the irrelevant stimuli. Consistent results were obtained by Donchin and Smith (1970) and by Wilkinson and Ashby (1974).

On initial examination, it appears somewhat contradictory that, in these studies of attention, predicting the order of relevant and irrelevant stimuli increased the N1 amplitude for the relevant stimuli, whereas, in the studies reviewed in the previous section, eliminating time uncertainty reduced the N1 amplitude. However, the enhancing effect of temporal predictability on the N1 stems from comparisons between relevant and irrelevant stimuli that occur together in the same block, whereas the data supporting a reducing effect derives from comparisons between different blocks. A block of unpredictable stimuli may be associated with a higher degree of arousal or nonspecific reactivity than a block of predictable stimuli. Furthermore, the tasks involved in the time uncertainty studies were relatively simple and it is probable that the effects of prior preparation are manifest only when the task is particularly demanding (a difficult sensory discrimination rather than a simple button press). There are thus two probable effects that may increase the N1 amplitude: any prior uncertainty about stimulus timing and any prior preparation for performing a demanding task.

If, in the selective attention experiments, the timing of the attended and ignored stimuli was made unpredictable (thereby eliminating the possibility of selective prior preparation), there were no attention-related changes in the N1 (Hartley, 1970; Näätänen, 1967; Wilkinson & Ashby, 1974). However, these experiments used a rather slow rate of stimulus delivery, and it is possible that the subject was able to attend to both types of stimuli despite instructions to the contrary (Hartley, 1970). Hillyard, Hink, Schwent, and Picton (1973), following Wilkinson and Lee (1972), increased the rate of stimulation to eliminate this possibility. Hillyard et al. found that the N1 wave of the auditory EP was increased by selective attention. Subsequent studies (reviewed by Hillyard & Picton, 1979) demonstrated that this N1 effect occurred only when the rate of stimulation was rapid (Schwent, Hillyard, & Galambos, 1976a) and when the intensity of the stimuli was not too loud (Schwent, Hillyard, & Galambos, 1976b). Schwent and Hillyard (1975) reported that the magnitude of the N1 effect varied with the amount of attentional resources allocated to different incoming stimuli.

Näätänen et al. (1978; Näätänen, Gaillard, & Mäntysalo, 1980) and Näätänen and Michie (1979) pointed out that the attentional effect could be dissociated in time from the N1 wave and often ex-

tended for several hundred milliseconds beyond the N1 peak. The attentional effect could be best evaluated in the "difference waveform" obtained by subtracting the EP to stimuli being ignored from the EP to the same stimuli when they were being attended. Such a subtraction shows a broad negative wave that Näätänen and his colleagues called the "processing negativity" and Hansen and Hillyard (1980) called "Nd". Figure 12 illustrates these difference waveforms. The processing negativity has been reviewed extensively elsewhere (Näätänen, 1982; Hillyard & Kutas, 1983). It will be considered here only as it relates to the N1 wave.

The scalp distribution of the processing negativity indicates that it may arise from more than one cerebral generator. Hansen and Hillyard (1980) demonstrated that the early portion of the Nd wave has a frontocentral scalp distribution similar to that of the N1 wave, whereas the later portions are somewhat more frontal. This result was confirmed by later studies (Hansen & Hillyard, 1983, 1984; Maiste & Picton, in press; Näätänen, Gaillard, & Varey, 1981; Okita, Konishi, & Inamori, 1983) but in Okita's (1979) paper such a topographical shift did not appear to be present in the published figures. Maiste and Picton (in press) suggested that the frontal negativity is present only when the within-channel task is quite difficult. In a detailed topographical study, Woods and Clayworth (in press) obtained evidence for two components, but the earlier component also had a scalp distribution that differed from that of the N1 in response to the ignored stimulus. Moreover, the early Nd wave differs from the N1 by not showing a phase reversal over the Sylvian fissure when recorded using a nose reference (Alho, Paavilainen, Reinikainen, Sams, & Näätänen, 1986). Thus perhaps neither of the two components of the processing negativity represents an enhancement of some obligatory component of the N1 wave. These two components would then be components 5 and 6 of the present review.

Under certain conditions, however, the Nd effect recorded magnetically does indicate a current source in the supratemporal plane which may be identical to the N1 source in that plane (Kaufman & Williamson, in press). Furthermore, in some data (e.g., Hillyard et al., 1973; Hillyard, Woldorff, Mangun, & Hansen, in press; Donald, 1983) the time match between the auditory N1 wave and the selective-attention effect is so good as to suggest a direct effect of selective attention on some obligatory component of the N1 generator. Since the selective attention effect is obtained at midline scalp electrodes, this component could be either component 1 or component 3. Due to the short onset latency of the selective attention effect (reviewed by Näätänen, 1982) and to some evidence for its lateralization to the scalp contralateral to the attended ear (Michie, personal communication, August, 1986), the relatively nonspecific component 3 does not appear as plausible as the supratemporal component 1. Consequently, we consider it possible that the original "N1 effect" of Hillyard et al. (1973) and the processing negativity described by Näätänen and his associates are different processes, and that whether either or both occur during selective attention de-

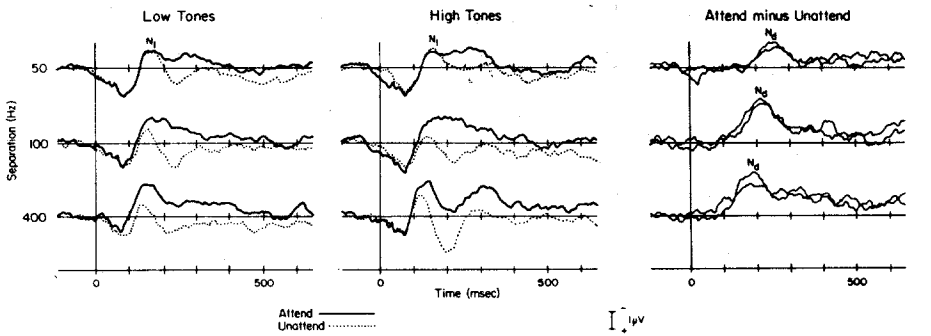

Figure 12. The processing negativity or difference wave during attention to channels characterized by different frequencies. The subject's task was to attend either to a sequence of 300 Hz (low frequency) tones or to a sequence of higher frequency tones in order to detect an occasional tone of longer duration. These waveforms represent the grand average ERPs to the shorter duration tones for 12 subjects at three different interchannel frequency separations. The tracings on the right show the difference waves between the attended and the unattended ERPs for the high tones and the low tones that are plotted on the left and center of the figure. Recordings were taken from the vertex with negativity being represented by an upward deflection. In the difference recordings, there is an Nd wave that has a longer latency when the channels become closer together in frequency. From Hansen and Hillyard, 1980, *Electroencephalography & Clinical Neurophysiology, 49*, 277–290. Reprinted with the permission of the authors and the publisher.

pends upon the stimuli, their timing, and the subject's strategy. This state of affairs for the auditory modality would then be analogous to findings in the visual modality. Visual selective attention can produce processing negativities (for a review, see Harter & Aine, 1984; Hillyard & Mangun, 1986; and Näätänen, 1986c), and also enhance exogenous components (Hillyard & Münte, 1984). The latter effect, however, occurs only in connection with spatial visual attention (Hillyard, Münte, & Neville, 1985).

In sum, it appears that in most conditions auditory selective attention causes the superimposition on the N1 wave of a processing negativity, consisting of two components (5 and 6 in the present review) that overlap the true N1 components. It is possible that under certain conditions attention may selectively enhance a true N1 component, as suggested by Hillyard et al. (1973). In that case, this enhanced component would probably be the supratemporal component (component 1).

States of Arousal and Levels of Performance

Although the early attention experiments failed to establish a correlate of selective attention, they could be interpreted as suggesting that attention is accompanied by a general and nonspecific increase in cerebral excitability which might increase the amplitude of the N1 wave. Näätänen (1967) studied the auditory EPs when attention was directed toward visual stimuli. An imperative flash (S2) requiring a rapid key press, was delivered randomly either 1, 2, or 3 s after a warning flash (S1). On one third of the trials an irrelevant click was presented during the S1-S2 interval so that its timing was irregular in relation to the occurrence of S1 and S2. This click elicited a larger vertex N1 than an identical control click delivered during the S2-S1 interval (11 s). Somewhat analogous results with relevant and irrelevant stimuli both in the auditory modality were obtained by Hermanutz, Cohen, and Sommer (1981). Näätänen (1967) concluded that "It might be that all kinds of stimuli, even of a completely irrelevant sense modality, elicit EPs with enhanced amplitudes, if presented during attention directed to one sense modality" (p. 60).

The responses to probe stimuli presented during tasks other than foreperiod RT paradigms have also suggested that arousal enhances the N1 amplitude. When probe stimuli are presented during tasks such as mental arithmetic, EPs to the probe stimuli are larger than when delivered during relaxation (Eason, Aiken, White, & Lichtenstein, 1964; Eason & Dudley, 1971) or during less demanding performance (Nash & Williams, 1982). In the latter study, the N1 to auditory probe stimuli (requiring a switch press) presented under high-speed instructions in the visual primary task was larger than under moderate-speed instructions. Loveless (1977) found that the N1 response to a visual imperative stimulus of a go/no-go RT task was larger in blocks in which the auditory no-go stimulus was very loud (97dB) than in blocks in which it was soft (57dB). Arousal may not always enhance the N1, however. Schafer (1978) found no difference in the N1 amplitude to a probe stimulus (a momentary slight increase in the brightness of the TV picture) when the subjects watched a dull TV program (Dick and Jane Talk) and when they watched a program they regarded as considerably more interesting (Dick and Jane Make Love).

The N1 evoked by unattended auditory stimuli is larger at higher levels of alertness, as estimated on the basis of the prestimulus EEG (Fruhstorfer & Bergström, 1969). Since the mean ISI in this experiment was 12 s and because the response was recorded between the vertex and the forehead, it is possible that the nonspecific N1 component is mainly responsible for this relationship.

It is possible that the effects of arousal are mediated in the brain by the same processes that underlie the enhancement of the N1 with selective attention. Picton, Ouellette, Hamel, and Smith (1979) pointed out that it is "probably impossible to change levels of arousal in the waking state independently of any attentional change." During heightened states of arousal subjects usually (although not always) increase their alertness or general attentiveness to the external world.

The late components of the EP are dramatically affected by sleep (Anch, 1977; Fruhstorfer & Bergström, 1969; Osterhammel, Davis, Weir, & Hirsh, 1973; Paavilainen et al., in press; Picton et al., 1974; Weitzman & Kremen, 1965; Williams, Tepas, & Morlock, 1962). One of the most striking effects is the large negative wave with a peak latency of 300 ms. This wave is often visible in the unaveraged EEG as the "vertex sharp wave" or "sleep N2." The nature of this N2 wave is not known: "it might possibly represent an excitatory phenomenon associated with an unsuccessful attempt at arousal or an inhibitory wave preventing awareness or memory of the incoming information" (Picton, Campbell, Baribeau-Braun, & Proulx, 1978).

The waveform at the latency of N1 shows complex changes during sleep. Most reports have indicated a decrease in N1 amplitude with sleep (Anch, 1977; Kevanishvili & von Specht, 1979; Paavilainen et al., in press) while others have reported no change (Buchsbaum, Gillin, & Pfefferbaum, 1975). Recent work by Bell and Campbell (personal communication, February, 1986) has indicated that these

differences may be partially related to the measurement technique. The peak of N1 measured relative to a baseline decreases significantly during sleep whereas the N1-P2 measurement may be less affected. In the data of Bell and Campbell and of Paavilainen et al., sleep appears to remove a negative wave lasting through the latencies of the waking N1 and P2 waves, or to add a positive wave of similar duration. Figure 13 shows the pattern of auditory EPs recorded during sleep from midline scalp electrodes. Bell and Campbell (personal communication, October, 1986) have also found that the temporal negative wave at a latency of about 150 ms (apparently component 2) decreases in amplitude during sleep.

Many studies suggest that the N1 amplitude recorded during wakefulness correlates with task performance, being larger on those trials which are associated with a higher level of performance (other factors being constant). We have already reviewed studies showing that the N1 is larger during better detection of threshold auditory stimuli. Working with visual stimuli, Haider, Spong, and Lindsley (1964) found that the accuracy of discriminating occasional dim flashes in a sequence of brighter

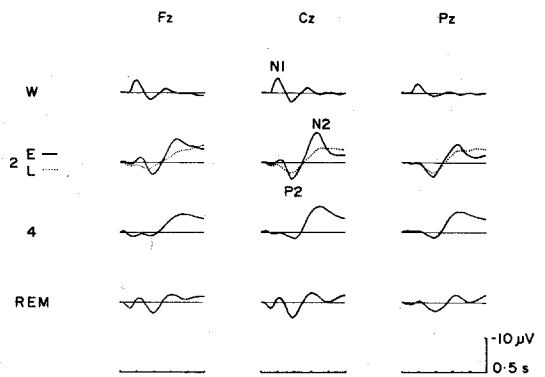

Figure 13. Auditory evoked potentials during sleep. Grand average waveforms from 10 young adult subjects recorded during overnight sleep. The evoked potentials were elicited by 80dB SPL, 1000 Hz tonebursts with a duration of 55 ms presented once every 1.1 s. The tonebursts were transduced through a hearing aid device to ensure constancy of the stimulation during sleep. Recordings were made during wakefulness (W), and during stages 2, 4, and REM sleep. The recordings during stage 2 are separated into those recorded early in the night (E) and those recorded late (L). The evoked potentials were recorded from mid-frontal, vertex, and mid-parietal electrodes using a noncephalic, sternovertebral reference. The major findings are the attenuation of the N1 wave during sleep and the addition of a large N2 wave during the non-REM stages of sleep, particularly early in the night. Unpublished data obtained by Ian Bell and Kenneth Campbell and reprinted with their permission.

flashes correlated with the amplitude of the vertex N1-P2 elicited by both the target and the non-target flashes. Several studies have shown that the auditory N1 amplitude is larger when the simple reaction time to the stimuli is shorter (Bostock & Jarvis, 1970; Dustman & Beck, 1965; Näätänen & Gaillard, 1974). Wilkinson and Morlock (1966), however, did not find such a correlation. Bostock and Jarvis (1970) demonstrated that the correlation between the N1 amplitude and the reaction time is largely (but not entirely) due to similar time-on-session effects on both measures. Interestingly, a later negative deflection, "N2", did not change with time-on-session but strongly correlated with the reaction time, being larger for slower reaction times. (For consistent data, see Wilkinson, Morlock, & Williams, 1966; Wilkinson & Morlock, 1966.) Some subjects even fell asleep during the session with the consequence that the N2 grew very large. It appears, therefore, that the N2 observed in association with slow reaction times is identical to the "sleep N2" which, when occurring during wakefulness, indicates drowsiness and is in this way related to weak performance.

Increasing motivation by making the amount of monetary reward dependent on performance (Wilkinson & Morlock, 1966) has resulted in enhanced N1 amplitudes and better performance, but again, it is not possible to conclude with certainty that increased arousal enhanced the N1 amplitude.

Considering all of the evidence reviewed in this section, there is some evidence for task- or attention-induced stimulus-nonspecific increase in the excitability of some neuronal population contributing to the N1 deflection. This increase causes the N1 amplitude to any input, relevant or irrelevant, to be larger when the subject is engaged in some task rather than relaxing, and larger when performing a more rather than less involving task. Similarly, while performing a continuous task, if he or she can predict the moments of delivery of the relevant events above the chance level, these moments tend to be preceded and coincided by an excitability increase which causes the N1 amplitude to the relevant stimuli to be bigger than that elicited by the irrelevant stimuli.

It is possible, however, that this nonspecific excitability increase should not be exclusively interpreted in terms of increased arousal but rather as also being due to a general increase in sensory sensitivity. This might be independent of arousal, since a subject who is highly aroused when attending to his or her internal thoughts may not notice events in the external world at all. It is therefore possible that the brain possesses a general gain control over its own sensory input. This is proposed, for in-

stance, in the orienting-response theory (Sokolov, 1963) and there is strong evidence for that proposal (for a review, see Lynn, 1966). When an unpleasant stimulus is anticipated, the sensory sensitivity is decreased ("negative preception"; Lykken & Tellegen, 1974) whereas, when a pleasant, important or interesting stimulus is expected, sensitivity is increased ("positive preception"). A similar basic assumption underlies the "directional-fractionation" hypothesis (Lacey, 1967) which proposes that the heart rate changes differentially depending on whether the subject prepares for stimulus intake (heart rate deceleration) or for stimulus rejection (acceleration).

The EP literature provides some support for interpreting the nonspecific excitability increase in terms of modulation of sensory sensitivity. Young and Horner (1971) obtained smaller EPs (although there is some uncertainty as to whether the N1 was reduced) in response to affective than non-affective verbal stimuli in those subjects to whom the emotionality of the affective word was great (such as the word "rape" presented to an unmarried female by a male experimenter). "Since both the affective and non-affective words were presented sequentially (i.e. in separate lists) those subjects who gave reduced N1-P1 peak amplitudes for the affective stimuli possibly realized at some early point in the affective list that all of the words had been emotional. They would expect, therefore, the next word to be emotional, and it would be this expectancy, or anticipation, that could create the decrease of the N1-P1 peak amplitude" (pp. 299–300). Consistently, there was no reduction of the EP to affective words in subjects who did not find the words particularly emotional.

The auditory N1 has also been recorded from subjects who have taken drugs that affect the nervous system. Sedative drugs that bring on sleep cause changes in the auditory EP that are characteristic for sleep. When the subject is under the influence of a sedative medication but remains awake, the N1 is often decreased in amplitude. It is tempting to speculate that this reduction in amplitude varies with a drug-related decrease in alertness. The effect of ethanol has been evaluated extensively since Gross, Begleiter, Tobin, and Kissin (1966) initially reported an ethanol-induced reduction in the N1 and P2 deflections of the auditory EP. Fruhstorfer and Soveri (1968) showed that ethanol had a much greater effect on the N1 wave evoked by the first stimulus in a brief train than on the later responses. Interestingly, Järvilehto, Laakso, and Virsu (1975) found that the hangover state had a similar effect to ethanol intoxication on the response to the first stimulus in a train. Wolpaw and Penry (1978) reported that ethanol decreased the amplitude of the vertex N1-P2 waves but did not change the amplitude of the Ta-Tb complex recorded from temporal electrodes (although there were some latency changes). The data from these last three studies suggest that the nonspecific component of the N1 wave is the most sensitive to ethanol. Pfefferbaum, Roth, Tinklenberg, Rosenbloom, and Kopell (1979) found that the ethanol-induced reduction in N1 was similar across different intensities. Campbell, Marois, and Arcand (1984) found that the N1 reduction caused by ethanol could be reversed by requiring the subjects to attend to the stimuli in order to discriminate changes in their frequency.

Concluding Discussion

Some Anatomical and Physiological Considerations

The human auditory cortex is located on the superior and lateral aspects of the temporal lobe. Early descriptions of the human cortex proposed that primary projection areas received sensory input and connected to the surrounding primary association areas. These primary association areas then connected to secondary association cortex. Since practically all regions of the cortex have subcortical connections, there is really no clear distinction between "projection" and "association" areas on the basis of subcortical input. Modern neuroanatomy distinguishes different regions of the cortex on the basis of their cellular structure. The primary projection areas, since they are densely packed with granule cells, are called "koniocortex".

Galaburda and Sanides (1980) have recently shown that the human auditory cortex is somewhat more extensive than previously described. The primary projection areas AI (or lateral auditory koniocortex—KAlt) and AII (or medial auditory koniocortex—KAm) are located on Heschl's gyri. The AII area is located medial to AI and is richer in callosal connections. Animal studies show that both regions are tonotopically organized with the higher frequencies located caudal and medial to the lower frequencies (Brugge & Merzenich, 1973). Positron-emission tomography indicates a similar tonotopic organization in human subjects (Lauter, Herscovitch, Formby, & Raichle, 1985). Surrounding these areas of koniocortex are several areas of parakoniocortex that receive input from the primary auditory area and from the medial geniculate body. These cortical areas extend onto the lateral aspect of the temporal lobe and onto the parietal operculum. There are extensive interconnections between these auditory areas (Fitzpatrick & Imig,

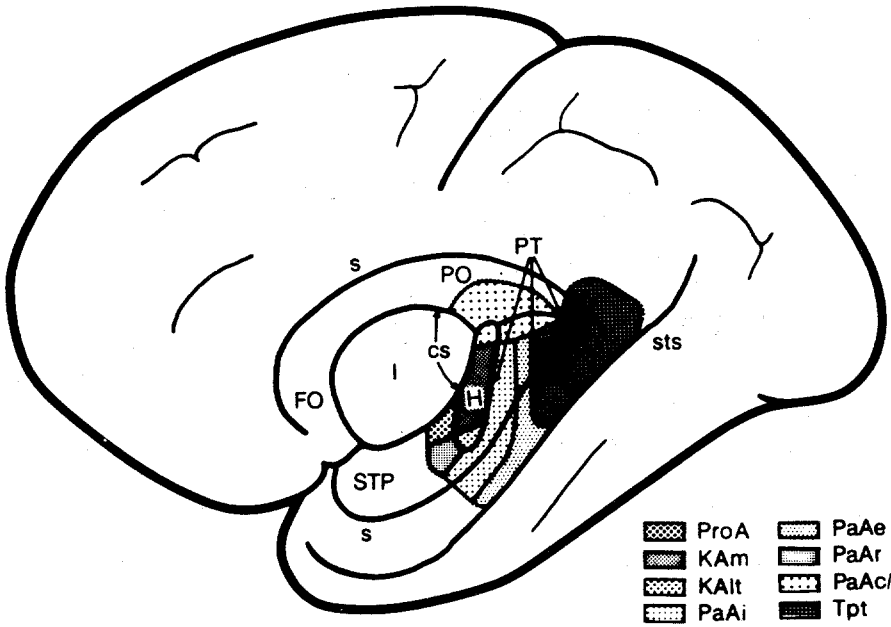

Figure 14. The human auditory cortex. This diagram exposes the Sylvian fossa to show the extent of the auditory areas. The edges of the Sylvian fissure are labelled "s". The frontal and parietal opercula (FO and PO) form the upper boundary of the fissure, and the supratemporal plane (STP) forms its lower boundary. The insula (I), delimited by the circular sulcus (cs), is the floor of the fissure. On the middle of the supratemporal plane are the transverse temporal gyri of Heschl (H). Posterior to these gyri is the planum temporale (PT). The auditory areas are located on the supratemporal plane, the parietal operculum, and the superior temporal gyrus (on the lateral surface of the temporal lobe above the superior temporal sulcus—sts). The shading shows eight different kinds of cortex. This figure is adapted from Galaburda and Sanides, 1980, *Journal of Comparative Neurology, 190*, 597–610, with the permission of the authors and the publisher.

1982). Posterior to these areas on the supratemporal plane and extending onto the lateral surface of the temporal lobe and onto the anterior portion of the parietal lobe is a large area of temporoparietal or Tpt cortex. The left hemisphere often has a much larger Tpt cortex than the right (Galaburda et al., 1978). The arrangement of the different auditory areas varies both between individuals and between hemispheres (Galaburda & Sanides, 1980, p. 603). Figure 14 provides a diagrammatic representation of the human auditory cortex.

Pandya and Seltzer (1982) have recently described the connections of the auditory sensory and association areas in the monkey. They distinguish two auditory association areas—AAI and AAII—on the lateral surface of the superior temporal gyrus, with AAI located more posteriorly. Primary auditory cortex AI projects to the AAI area of association cortex. This connects back to the AII primary sensory cortex, and projects to the AAII association area and the premotor cortices of the frontal lobe. The AAII region projects to the paralimbic area of the anterior temporal lobe, to the polymodal association areas in the parietal and temporal lobes, and to the prefrontal cortex. Similar patterns of connections can be traced in the visual and somatosensory modalities, with each primary cortex projecting to a region of association cortex which then connects to multi-modality regions of the frontal and temporoparietal cortex (Jones & Powell, 1970).

There are complex connections among the primary auditory cortex, subcortical structures, and the polysensory regions of cortex in the frontal, temporal, and parietal lobes. Irvine and Phillips (1982) have suggested that an "adjunct" auditory system (involving the reticular formation and the medial and intralaminar regions of the thalamus) projects directly to the polysensory association areas independently of cortico-cortical connections. Bignall and Imbert (1969) recorded from the frontal cortex click EPs showing clear polarity reversals between the cortical surface and the subcortical white matter. Since these responses were not abolished after bilateral ablation of the auditory cortex, they were probably mediated through thalamic projections. Like Arezzo et al. (1975) they also recorded

responses that did not show any polarity reversal and probably originated in the auditory cortex, but these were less common. This difference may have been related to the different species of monkey (the auditory cortex of squirrel monkeys is more laterally oriented), the chloralose anesthesia, or the long ISI (not less than 7 s compared to the 0.6 s of Arezzo et al.). Bignall (1969) stimulated the auditory association cortex of the squirrel monkey and recorded responses from the frontal lobes. Lesion studies indicated that these responses were mediated through cortico-cortical connections. The polysensory cortex of the frontal lobe therefore receives auditory input from both the thalamus and the auditory association areas. Newman and Lindsley (1976) described the responses of units in the frontal cortex to auditory stimuli. These units responded to a wide range of frequencies and to many different types of stimuli. They were particularly sensitive to species-specific vocalizations, a bias not shown in the units of the primary auditory cortex. Newman and Lindsley therefore suggested that the frontal units were more affected by the motivational significance of the stimulus than by its physical nature.

Pandya and Seltzer (1982) describe a general pattern of connectivity whereby sensory information processed in the primary and association areas "is conveyed to polymodal zones for cross-modal interchange of information, to paralimbic and limbic areas for investment with emotional tone and placement in memory, and to the frontal association areas where both sensory and limbic data are integrated in preparation for the organism to respond to sensory stimuli by an appropriate action" (p. 390).

As well as receiving input from them, the prefrontal cortex projects back to the sensory association areas so that there is a two-way communication between these regions (Nauta, 1971). The prefrontal cortex also projects to the reticular regions of the thalamus where it might, according to Scheibel (1980), exert some gating control over the input to the primary sensory cortices.

Sensory information begins to reach the primary auditory cortex of a human subject about 10 ms after being received in the cochlea (Celesia, 1976). It is difficult to determine what subsequent processes result in negative waves peaking at approximately 100 ms in several regions of cortex.

The extent of cortical excitation will depend on three parameters: the actual anatomy of the connections, the nature of the incoming sensory information, and the degree to which the interneuronal connections have been facilitated or inhibited. The connectivity can be modulated either generally through arousal mechanisms or more specifically through the priming or inhibition of interneurons mediating particular connections.

An unattended auditory stimulus probably activates two major areas of cortex: the supratemporal plane (component 1) and the superior temporal gyrus (component 2). The effects of an auditory stimulus may remain for a period of several seconds as continuing neuronal activity or as some neurochemical change. If the stimulus occurs again during this period the response evoked by the second stimulus is less than the response to the first, with a recovery function that lasts for several seconds. This time may be related to the duration of auditory sensory or echoic memory (Näätänen, 1984; Picton, Campbell, Baribeau-Braun, & Proulx, 1978).

The motor and premotor regions of frontal cortex may be activated by cortico-cortical connections from the auditory cortices and by less specific connections from the brainstem. This activation probably occurs only if the stimulus reaches a threshold that varies with the interval from the preceding stimulus, being very high during the first few seconds after that stimulus. The motor and premotor cortices would therefore be activated by stimuli occurring infrequently, this activation being considerably stronger for louder stimuli.

When incoming information is not the same as in previous stimuli, two different processes will occur. First, extra activity may be immediately evoked and seen in the increase of the N1 amplitude (cf. Butler, 1968). The enhancement of the N1 will vary with the difference from the preceding stimulus. Second, the sensory system detects the difference from the previous stimulus and generates a MMN. This will be larger the greater amount of activity remaining from the previous stimulus, that is, the shorter the interval between the stimuli. The MMN will be earlier when the difference between the stimuli is larger.

Attention to auditory stimuli may act by facilitating interneuronal connections to increase the extent and degree of cortical activation by the stimuli. When it is possible to predict the nature and timing of an upcoming stimulus, the brain may activate the neuronal patterns necessary for evaluating the stimulus prior to its occurrence. The activation and maintenance of these templates may be manifest in the sustained negative waves recorded prior to the stimulus. When the stimulus occurs it need not fully activate the neuronal patterns by itself, and the N1 response is therefore much smaller. The map of this facilitated activity across the cortical surface may be evident in the scalp distribution of the processing negativities and in the areas of increased cerebral blood flow reported by Roland (1981, 1982) and by Roland, Skinhøj, and Lassen (1981). These studies (recently reviewed by Näätänen, in press-a) have

shown that attention to a sensory stimulus increases blood flow in both the specific sensory cortex and the frontal lobe.

During sleep the functional organization of cortical activity is quite different from during wakefulness. The major difference may be the lack of interneuronal facilitation. The N1 activation of the cortex is thus restricted in time and space. An underlying positive wave is revealed, perhaps related to depolarization of the deeper regions of cortex rather than to depolarization of the surface dendrites.

Component Structure of the N1 Wave

Our review of the literature has convinced us that several different cerebral processes contribute to the N1 wave of the scalp-recorded auditory EP. These "component" processes occur in different cerebral locations and subserve different psychophysiological functions. They are distinguished by their characteristic electrical and/or magnetic fields and by their specific relationship to various experimental manipulations. The following paragraphs describe six components that we hypothesize as contributing to the scalp-recorded N1 wave. The first three can be considered as true N1 components, whereas the other three are components that often exist in the latency region of the N1 wave but may occur independently.

Component 1 is generated in the cortex of the supratemporal plane, as originally proposed by Vaughan and Ritter (1970). This component has a a peak latency at 100 ms, is maximally recorded from the frontocentral scalp, and has a scalp field that is slightly greater over the hemisphere contralateral to stimulation. The justification for this component derives from the magnetic recordings (Elberling et al., 1980; Hari et al., 1980) and the scalp-distribution analysis of Scherg and von Cramon (1985, 1986a).

The component is probably generated over a much wider region of the supratemporal plane than that occupied by the primary auditory cortex on Heschl's gyri. This is suggested by the finding that bilateral lesions of the temporal lobe must extend into the temporoparietal region before the N1 is abolished (Woods et al., 1987). Because of the variable orientation of the supratemporal plane among individuals, the degree of frontal spread and of left-right asymmetry will vary from one subject to another. It is possible that hemispheric asymmetries in the response may be related to asymmetries in the size and orientation of auditory cortex on the supratemporal plane.

The amplitude of this component probably changes with intensity according to the rules proposed by Bak et al. (1985) for the current dipole generating the magnetic fields that they recorded. If so, with increasing intensity the component increases in amplitude and the slope of this change decreases. Judging from the data of Hari et al. (1982), we suggest that the relative refractory period for this component is 4 s or slightly longer. It is possible that this component may be enhanced by attention through some thalamocortical gating mechanism.

Component 2 is a biphasic component with a positive wave at about 100 ms and a negative wave at approximately 150 ms, as originally proposed by Wolpaw and Penry (1975). It is probably generated on the superior temporal gyrus and is recorded from the scalp with maximum amplitude at the midtemporal electrodes. The justification for this component derives from the cortical recordings of Celesia (1976) and of McCallum and Curry (1979), and from the scalp-distribution analysis of Scherg and von Cramon (1985, 1986a). This component has a radially oriented generator and therefore is not picked up magnetically.

This component would be generated in the auditory association areas, activated by connections from the primary auditory cortex and also possibly from the thalamus. The response may be larger if the association cortex is primed by some expectancy mechanism. This would explain the results of Arezzo et al. (1975) who found that the lateral surface of the temporal lobe was activated only in monkeys who had previously used auditory stimuli in behavioral tasks. Furthermore, Perrault and Picton (1984) found that the component was enhanced when subjects attended to a train of monaural stimuli compared to when the subjects ignored the stimuli.

The component is much larger, and slightly earlier, over the hemisphere contralateral to the ear of stimulation than over the ipsilateral hemisphere. There are at present no data concerning the effects of intensity or ISI on this component.

Component 3 is a vertex negative wave with a peak latency of approximately 100 ms. The location of the generator of this component is not known. We suggest that this component is generated in the frontal motor and premotor cortex under the influence of the reticular formation and the VL nucleus of the thalamus, which projects to the precentral gyrus, to the adjacent regions of the superior, middle and inferior frontal gyri, and to the supplementary motor area on the mesial surface of the frontal lobe. These areas may also receive auditory input from the auditory association cortices. Component 3 is recorded on the scalp with maximal amplitude at the vertex and the lateral central electrodes. The justification for this component derives from Hari et al. (1982), who found that at long ISIs the vertex potential increased independently of the magnetic

response, and from Velasco et al. (1985; Velasco & Velasco, 1986) who found the reticular formation and the VL nucleus of the thalamus to be very active during the auditory vertex potential. Arezzo et al. (1975) found that some areas of the monkey frontal cortex were active during the scalp-recorded N1. Hari (1983) speculated that the supplementary motor area (on the mesial frontal cortex above the cingulate gyrus) may be involved in this component. Libet, Alberts, Wright, Lewis, and Feinstein (1975, their Figure 3) reported that the human auditory evoked potential recorded from the supplementary motor area in the latency region of 135–220 ms showed polarity reversal between the surface and the depth.

We cannot rule out the possibility of another generator location for this component. We doubt that the scalp recordings pick up fields with amplitudes on the order of 10 μV that were generated in the reticular formation or thalamus, as suggested by Loveless (1983), because of the small area, the relative lack of dipole structure, and the large depth of these regions. The largest local fields recorded from subcortical areas by Velasco et al. (1985; Velasco & Velasco, 1986) were on the order of 50 μV. If these fields were indeed contributing to the scalp recording one might expect local subcortical fields that were some hundred times larger (cf. Nunez, 1981, p. 168). It is possible that component 3 may be generated in the supratemporal plane with a dipole orientation that is tilted somewhat more posteriorly than the dipole underlying component 1 (personal communication, Michael Scherg, October, 1986). Such a tilt in the dipole may not have been recognizable in the single-channel magnetic recording of Hari et al. (1982). However, the possibility of both these components originating in the supratemporal plane is difficult to reconcile with the persistence of an N1 wave in bilateral lesions of the temporal lobe (Woods et al., 1987).

We therefore suggest that this response is the cortical projection of a reticular process that facilitates motor activity. The early work of Larsson (1956, 1960a, 1960b) related the vertex potential to components of the startle reflex. Rossignol and Jones (1976) measured the H-reflex following 110dB SPL tones presented at a rate of 1/15 s. They found an enhancement of the reflex that began at 80 ms, peaked between 110 and 130 ms, and lasted for about 200 ms. This period of enhancement may have been mediated by descending influences from the reticular formation and the frontal cortex. Hazemann, Audin, and Lille (1975) found that the N1 wave of the auditory EP is reduced during voluntary self-paced movements. This finding further supports the relations between the N1 and the motor system. We suggest that component 3 may be largely generated in areas of the cortex mainly responsible for motor activity.

This component is most easily recorded in response to auditory stimuli presented at intensities of greater than 60dB SPL and at ISIs of greater than 4–5 s. The relative refractory period for the response lasts for at least 30 s. A similar response would be elicited by intense and infrequent stimuli in other modalities and there would be definite intermodal refractory effects. Much like the startle reflex, this response would be attenuated by knowledge of the timing of the stimulus.

Component 4 is the mismatch negativity. It is generated in the same regions of the brain that generate the first component, although probably by somewhat different neuronal processes. This MMN reflects the results of an automatic comparison between the present stimulus and those preceding it.

Component 5 is the sensory-specific processing negativity. This begins at approximately 50–100 ms and lasts during the processing of an attended auditory stimulus. This component is probably generated in the auditory sensory and association areas on the supratemporal plane and on the lateral aspects of the temporal lobe. Its scalp distribution may vary with the relative amounts of processing in the different areas.

Component 6 is the "attentional supervisor," a second component of the processing negativity. This wave has a longer time span than the sensory-specific processing negativity. We propose that it is generated in the anterior frontal cortex since it receives information from the auditory association cortex and since it feeds back to these sensory areas in order to bias particular kinds of auditory processing. The justification for proposing this component derives from the scalp-distribution studies of Hansen and Hillyard (1980), which show an Nd wave, the later part of which is more frontal than the early part, and from the results of Roland (1981, 1982) and of Roland et al. (1981), who found patterns of enhanced blood flow in the frontal lobes during attention to auditory stimuli.

Functional Significance of the N1 Components

This section will discuss the role that the first three components—the true N1 components—might play in auditory perception. The functional significance of components 4, 5, and 6 has been examined elsewhere (Hillyard & Kutas, 1983; Näätänen, 1982, 1985, 1986a, 1986b, 1986c; Picton, Stuss, & Marshall, 1986).

All three N1 components respond to a steep change in a level of physical energy that has remained constant for at least a short time. The N1

components are thus sensitive to the transient aspect (Graham, 1979; Loveless, 1983; MacMillan, 1973) of stimulation. When these transient aspects are controlled, rather similar N1 responses are elicited by very different kinds of acoustic stimuli such as clicks, tones, speech, and animal sounds. This suggests that the N1 generators are not stimulus specific. However, these recordings are unfortunately blind to the possibility that different auditory stimuli may elicit very specific micropatterns of neuronal activity since at a distance these micropatterns would result in a similar electrical field.

The techniques of selective adaptation may provide a better view of the stimulus specificity of the N1 generators. The results of Butler (1968, 1972a, 1972b) and Picton, Woods, and Proulx (1978b) suggest a loose stimulus specificity for the generators recorded from midline electrodes (components 1 and 3). However, Näätänen et al. (1986) have recently shown (Figure 10) that, with very short ISIs, the refractoriness in N1 generation can be highly specific to the auditory frequency. At high stimulus rates auditory neurons with wide or relatively wide receptive fields may become refractory whereas the very specific neurons, those with narrow receptive fields, continue to respond. A second possibility is that at high stimulus rates the receptive fields of the auditory neurons change so that they respond only to a very narrow range of frequencies. Furthermore, at fast rates, the nonspecific neurons responsible for component 3 are relatively silent and cannot swamp the specificity of component 1.

Several neuronal processes may underlie the effects of selective adaptation. There is probably adaptation of the neurons responding to the repeating stimulus. In addition, the neurons that respond to a stimulus of one frequency may inhibit neurons that respond to stimuli of other frequencies and this lateral inhibition may last for several seconds. These inhibitory interneurons may undergo their own time-course of adaptation. Whatever the underlying processes, the recent results of Näätänen, Sams et al. (1987) nevertheless indicate that some of the neurons contributing to the N1 wave respond quite specifically to a very narrow range of frequencies.

Although at least some neuronal populations in the N1 generators can perform highly precise stimulus coding, this coding does not appear to underlie perception in the case of perceived pitch. Butler (1972b) found that a 1000 Hz tone amplitude-modulated at 200 Hz (and with a perceived pitch similar to that of a 200 Hz pure tone) affected the N1 evoked by a 1000 Hz tone much more than the N1 evoked by a 200 Hz tone. This dissociation between the frequency specificity of the N1 generators and the perceived pitch demonstrates that the N1 generators are not closely related to the perception of pitch. The amplitude of N1 is also not closely related to the perception of loudness: at short ISIs the N1 is much reduced although loudness remains the same; loudness summation occurs over longer stimulus durations than those that affect the N1 amplitude; the N1 for equally loud tones diminishes with increasing frequency; and the N1 may saturate at high intensities but the perception of loudness does not. These dissociations between the N1 and perception are consistent with Parasuraman et al. (1982) reporting that the N1 amplitude correlated with the detection of a threshold-level auditory stimulus but not with the recognition of its pitch. These results are consistent also with the suggested transient-detector character (Davis & Zerlin, 1966; Graham, 1979; Loveless, 1983; MacMillan, 1973; Walter, 1964) of processes underlying some of the N1 components. Davis and Zerlin suggest that "the mechanisms that generate the potential and determine its magnitude do not lie on the direct path, so to speak, to psychological sensation but rather on a parallel path with other functions" (Davis & Zerlin, 1966, p. 116). In serving transient detection, the N1 amplitude may correspond to the "obtrusiveness" of the stimulus. This is consistent also with reduction or total disappearance of the N1 wave in sleep. The N1-generator neurons may then not play a "specific" role in perception. Näätänen (1986d) suggests that some generator processes (those corresponding to components 1 and 3 of the present review) underlying the N1 act as nonspecific attention-triggering processes, causing the brain to become conscious of "silent" stimulus information extracted by other earlier mechanisms.

It is also possible that the process generating the N1 is the result of such an attention trigger rather than the trigger itself. In this view the N1 would represent the initial readout of information from the sensory analyzers. The readout could be triggered at a set time after the stimulus by some relatively nonspecific signal from the reticular formation. Although the trigger of the sensory cortices may be quite nonspecific, the pattern of activity evoked by the trigger may be highly specific. Unfortunately, the resolution available to EP recordings is not sufficient to tease out this possible highly specific contribution to N1 (components 1 and 2). The perceptual processes may use all or part of the information contained in the N1 readout. Thus there need not be a close correlation between the N1 and perception. For example, the perception of pitch may be based on the periodicity of the stimulus and not on the frequency structure of the stimulus, despite both sets of information being available in the N1 readout.

Another way to interpret the functional significance of component 1 is in relation to sensory memory. The component may reflect the formation of a trace for the eliciting stimulus. In this hypothesis the neurons underlying auditory sensory memory are involved in the generation of both component 1 of the N1 wave and the MMN (component 4). The latter would be generated by a comparison between the activation pattern or trace evoked (or being evoked) by the sensory input and the trace remaining from previous stimuli. Component 1 of the N1 wave would then be determined by two factors: 1) the physical properties of the stimulus (explaining the dependence of the N1 on stimulus intensity, frequency, location, etc.), and 2) the timing and physical properties of previous stimuli, which determine the degree and pattern, respectively, of refractoriness in the N1 neurons. A "neuronal model" of the stimulus may thus be represented in the pattern of refractoriness prevailing in the generator mechanism of component 1. In response to a stimulus identical to the stimulus represented by the trace, only a small N1 is generated because the stimulus affects mainly neurons that are very refractory. This response further increases the refractoriness (and thus improves the stimulus trace). On the other hand, when a different stimulus is delivered in the sequence, the N1 amplitude may be increased because this stimulus affects neurons whose refractoriness is less. The trace-formation process elicited by the stimulus is stronger, and a new trace is developed in the system. The smaller is the difference between the two stimuli, the less the neuronal trace-formation process triggered by the new stimulus since the overlap between the two traces is greater.

This memory-trace hypothesis can well account for the ISI effects on the N1 wave (component 1). Judging from the data of Hari et al. (1982), we suggest that component 1 is fully recovered when the ISI is greater than 4 s. This means that the pattern of refractoriness produced by a stimulus would dissipate in about 4 s and the whole system would then regain its full reactivity. Since there now is no previous trace, each stimulus produces maximal trace-formation activity. This duration of the stimulus trace is consistent with most estimates for the duration of the auditory sensory memory (Cowan, 1984; Kroll, 1975).

In this hypothesis, the comparison stage of sensory memory is reflected by the MMN. The MMN generator process is initiated by an input which does not perfectly match with the trace, whereas when a perfect match occurs (an identical stimulus is repeated), no comparison process is triggered. Consistently, there is no MMN to the first stimulus or to repeating stimuli in a sequence but only to a deviant stimulus. Furthermore, the MMN is earlier and usually larger for larger stimulus deviations. The deviant stimulus also elicits N1 (component 1), in keeping with the suggestion that the trace-formation stage, or at least its initiation, occurs before the comparison stage reflected by the MMN. Support is provided by Sams et al.'s (1984) finding that a deviant stimulus not only elicits a MMN but, as suggested by the MMN elicited by a standard stimulus following a deviant, also forms its own trace. The onset latencies of the N1 and the MMN are consistent with this dual-process proposal. The close relation between component 1 of the N1 wave and the MMN is further supported by both being generated from approximately the same cerebral regions in the auditory cortex according to the magnetoencephalographic recordings of Hari et al. (1984) and of Sams, Hämäläinen et al. (1985). Perhaps most importantly, there appears to be a reciprocal relationship between the ISI dependence of the MMN and N1 (component 1) in that when component 1 is fully recovered no MMN can be elicited, and when component 1 is not fully recovered there can be an MMN (Näätänen, 1984). The 4–5 s time (or slightly longer) for full recovery of component 1 seems to match the estimates of the ISI at which the MMN can no more be elicited (Mäntysalo & Näätänen, in press; Näätänen et al., in press).

In earlier sections of this paper, we have reviewed the evidence associating the N1 processes to the stimulus itself and the MMN to stimulus comparison or discrepancy. Crucial evidence for this characterization of N1 is provided by the attenuation of the N1 amplitude with decreased stimulus intensity even when this decrease means an increased stimulus deviation (Figure 6). Moreover, the N1 latency remains constant when the magnitude of the stimulus deviation is varied whereas the MMN latency is considerably longer for smaller differences (Figure 6). These facts are consistent with the hypothesis that component 1 of the N1 wave reflects the amount of neuronal activity occurring in trace formation whereas the MMN reflects the comparison between consecutive stimuli.

We have thus considered three possible functions for the neurons that are activated during the generation of component 1 of the N1 wave. These neurons may act to call attention to the availability of stimulus information, to read out sensory information from the auditory cortex, or to form a sensory memory of the stimulus within the auditory cortex. It is possible that different subpopulations of the N1 neurons may serve each of these functions.

Due to the scarcity of component-specific data, we can propose no distinct function for component 2, the T-complex, generated in the auditory association cortex on the lateral aspect of the temporal

lobe. This component may function similarly to component 1, or it may be more concerned with information processing than sensory memory. It is also possible that components 1 and 2 may relate to different types of auditory information. Component 1 may code the auditory stimulus in terms of tonal frequency and spatial location, and component 2 may use some other coding mechanisms. There is a great need for experiments that attempt to distinguish different controlling mechanisms for the two components.

The N1 wave is susceptible to three different modulations of the general state of the individual: 1) arousal changes associated with the sleep-wakefulness dimension, with certain drugs and alcohol, with circadian rhythms, and with involvement in task performance; 2) a sensory acceptance-rejection factor which may enhance responses to all sensory inputs during expectancy for important, interesting or pleasant stimuli, and attenuate responses elicited during expectancy for irrelevant, uninteresting or unpleasant stimuli; and 3) the degree of time uncertainty with regard to the next significant stimulus. These nonspecific influences probably have more effect on component 3 than on the first two components.

The generators of component 3 may belong to an extensive cerebral mechanism that functions to produce a widespread transient arousal of the organism. This transient arousal response appears to facilitate sensory and motor responses to the eliciting stimulus (as well as the associated central integrative processes) and also to shift the organism to a more efficient functional state. In the previous section of this paper we mentioned the evidence relating the N1 wave (apparently component 3) to a transient increase in spinal excitability. The long refractory period, the vigorous response when recovered, and the multimodal nature of this component support its interpretation as a transient arousal response with widespread facilitatory influences. Component 3 may also serve some aspects of detection and perception by alerting sensory association and motor cortex when a stimulus occurs after a period of quiescence.

Implications

The N1 wave of the human auditory evoked potential should not be considered a unitary process. Any investigation of its relations to physical and psychological parameters should therefore attempt to break it into its component parts. We have hypothesized six different component processes contributing to the N1 wave, the first three of which are true N1 components. This structure is heuristic and should be improved by further research.

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