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SCALP AND NASOPHARYNGEAL RECORDINGS OF HUMAN EVENT-RELATED POTENTIALS

NORMAND PERRAULT

Thesis presented to the School of Graduate studies of the University of Ottawa in partial fulfillment of the requirements for the degree of Doctor of Philosophy (Psychology).

Ottawa, Canada, 1982

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Perrault, N., and Picton, T.W. Event-related potentials recorded from the scalp and nasopharynx. I. Mesogenous components. In preparation.

Perrault, N., and Picton, T.W. Event-related potentials recorded from the scalp and nasopharynx. II. Endogenous components. In preparation.

ABSTRACT

Mesogenous and endogenous event-related potentials were recorded from the scalp and nasopharynx during a signal-detection task. The mesogenous components were evaluated with respect to the effects of inter-stimulus interval, intensity, frequency, attention and modality. Our results indicate the existence of at least 4 distinct processes occurring in the 75-150 ms latency range following auditory stimuli. The first process is indexed in the vertex-N1b/temporal-N1a component. This component does not reverse in polarity below the Sylvian fissure and is not seen in nasopharyngeal recordings. The location of its generator in not known. Probably there are two or more sources active at this latency. The second process finds reflection in the N1c/PgP120 component. This component is recorded with maximum amplitude on the side contralateral to the ear of delivery. A source in the lateral surface of the temporal lobe is a likely generator. The third process corresponds to Wolpaw and Penry (1975)'s Ta positivity. How much of this represents the underside of a vertically oriented dipole and how much a surface positivity is unknown. Finally, during attention, a lateralized processing negativity seems to overlap these components at the scalp, but not at

the nasopharynx. The auditory vertex N1 component is quite distinct from the visual N1 which is more posteriorly recorded on the scalp and which is associated with a definite nasopharyngeal positive wave. The P2 component is quite similar across auditory and visual modalities. In both modalities it is maximally recorded from the vertex and has no nasopharyngeal concomitant.

The endogenous components were evaluated with respect to the the effects of probability, interstimulus interval, intensity, discrimination difficulty, attention, stimulus omission and modality. Waves of opposite polarity to the scalp N2 and P3 components were recorded in the nasopharynx. The scalp and nasopharyngeal N2 components showed different patterns of variation across experimental conditions. These findings indicate that there are two different cerebral processes occurring at the latency of the scalp N2. The scalp and nasopharyngeal P3 components consistently covaried across conditions, suggesting a single underlying process. The Slow Wave was observed only in the scalp recordings.

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INTRODUCTION

Event-related potentials (ERP) can provide a unique picture of brain electrical activity during information processing in normal human subjects. The use of the evoked potential recording technique to further our theoretical knowledge of human cognition and personality is limited, however, since we do not know much about the component-structure and source generators of the event-related potentials in man. A deeper knowledge of the constituent components of the evoked waveform under given experimental conditions would greatly facilitate the task of establishing relations between brain electrical events and psychological processes. Similar benefits would come from a knowledge of the source generators of the various ERP components, permitting a better understanding of the genesis of the scalp-recorded waveform, which could then be dissociated into physiologically meaningful components. The information gathered in other research areas (animal psychophysiology, neuropsychology, neuropharmacology, etc) could then be used to understand the functional significance of the ERP components.

It is common practice to classify ERP components into exogenous and endogenous (Donchin et al. 1978). Exogenous components are evoked by events extrinsic to the nervous system. Endogenous components are determined mostly by the psychological properties of the eliciting event in interaction with the subjective states of the subject, and can be elicited by the absence of a stimulus. They are related to the use made of stimulus information. Hillyard et al. (1978) have proposed the intermediate "mesogenous" category for those components which are determined jointly by the physical parameters of stimulation and the psychological states of the subject. It is possible that mesogenous components are a result of overlapping endogenous and exogenous processes. The category is useful, however, since it permits distinguishing the vertex N1b-P2 components and the temporal N1a and N1c components from the earlier exogenous components and from the later endogenous N2, P3 and Slow Wave.

The most reliable information concerning the origins of ERP components comes from intra-cerebral recordings in animal and human subjects. Vaughan and colleagues (Vaughan 1974; Vaughan and Ritter, 1970) have used results from animal intra-cerebral studies (Arezzo et al. 1975) to support their hypothesis of a vertically oriented temporal dipole accounting for the N1-P2 components recorded from the

scalp. Recent studies by Halgren and Wood (Halgren et al. 1980; Wood et al. 1980) point to the limbic system as a likely generator for the late endogenous components of the ERP (N2 and P3). This is interesting, in view of current hypotheses linking the endogenous components to mnemonic and affective processes (Donchin 1979-updating of context; Hömberg et al. 1980-incentive value), functions that have traditionally been associated with the limbic system. Depth recordings from the human brain, however, are limited to patient populations were the procedure can be justified on diagnostic grounds. The number of experimental manipulations which can be attempted is very limited due to time constraints. Furthermore, the results that are obtained from the patients are certainly quite different from those that would be obtained in normal subjects. Finally only a few investigators are equipped to deal with the procedures involved, or interested in doing so. For these reasons, topographical studies have been used extensively to distinguish ERP components and infer their cerebral origins. These studies have usually compared waveforms recorded from various locations on the scalp and used the spatial distribution of amplitude to distinguish components. In all these studies, however, an important aspect of the brain is left unexplored, namely, its medio-basal surface.

Nasopharyngeal recordings provide a view from the

medio-basal surface of the brain. The tip of these electrodes rests at the back of the nasopharynx, a location under frontal lobes and the anterior portion of the temporal lobes. This location is diametrically opposed to the midline scalp electrodes and appears ideal to record from the lower end of vertically oriented dipoles. Vaughan and Ritter's (1974) hypothesis concerning the N1-P2 generator would therefore predict an inverted N1 at the nasopharyngeal leads. The nasopharynx would also seem to be a good vantage point to record activity from the limbic system, especially the hippocampus and amygdala. In intra-cerebral recordings, these structures (cf. Wood et al. 1983) are very active during the time of the scalp-recorded N2 and P3 components. It therefore became interesting to see whether we would record these components in the nasopharynx.

A major problem in the identification of ERP components is the possible overlap of several components in the same time period. In such cases, variations in two or more distinct components might be attributed to only one process. There is a lot of evidence for overlapping processes in the period from 50 miliseconds onward. Principal component analyses can be useful to distinguish overlapping components. The presently used techniques of principal component analysis, however, cannot be used if there are changes in latency or if there are components that overlap in both

space and time (Picton and Stuss, 1980). The choice of a method of rotation and the evaluation of component-significance are also still quite problematical (Rösler and Manzey, 1981). Finally, the majority of evoked potential laboratories (including ours) is not properly equipped to deal with the data processing involved. The best means available to distinguish separate components under such circumstances is to use multiple experimental manipulations (cf. Donchin et al. 1978), in conjunction with a careful analysis of scalp-distribution data. It is unlikely that distinct components will react in the same way to several experimental manipulations. Dissociation across experimental manipulations thus provides a further basis for evaluation of the component-structure of the ERP waveform. In the studies reported here we used manipulations of probability, interstimulus interval, intensity, discrimination difficulty, stimulus omissions, attention and modality, in an attempt to maximize the possibility of dissociation between nasopharyngeal and scalp-recorded components. We will now turn to the specific rationale for each one of these manipulations. A brief summary of results is also provided.

RATIONALES AND OUTCOMES

Probability

The effects of probability on the N2 and P3 components have been very well documented (see Picton et al. 1978a for a review). These components increase in amplitude with decreasing probability of the eliciting target. We were interested mainly in seeing whether the inverted N2 and P3 components at the nasopharynx would also decrease with decreasing probability. Fitzgerald and Picton (1983) have recently shown that the endogenous components were primarily determined by the interval of time separating targets (temporal probability), rather than by the probability of occurrence of the targets within the train of stimuli (sequential probability). We were interested to see whether both scalp and nasopharyngeal processes would show a similar dependence on temporal probability. We examined these possibilities in three probability conditions where both types of probability were either jointly manipulated (usual manipulation) or separately manipulated (only sequential or only temporal). The description of the probability conditions will be found on page 82. We hypothesized that the N2 and P3 components would be similarly affected at the scalp

and nasopharynx by our probability manipulations. This hypothesis was confirmed for P3. The scalp and nasopharyngeal N2s appeared differently affected across probability conditions, thus pointing to the possibility of two distinct processes.

Interstimulus interval

Besides its use in determining probability conditions, changes in interstimulus interval (ISI) permit altering the amplitude of components as a function of their refractory period. Long ISIs were used (3.3s and 5.0s) to maximize the amplitude of the mesogenous components and increase the possibility of recording them from the nasopharynx. We hypothesized that the N1-P2 components would be seen with reversed polarity at the nasopharynx, at least in the longest ISI condition. This prediction was not confirmed despite the large amplitude of these components at the scalp. These results were interpreted as not supporting the hypothesis of Vaughan and colleagues concerning a vertically oriented dipole in the temporal lobe, responsible for the generation of the N1-P2 components.

Intensity

Our preliminary experiment (Perrault et al. 1983) did not replicate the findings of Smith et al. (1973) concern-

ing an inverted N1-P2 at the nasopharynx. This discrepancy, however, might have been explained by a lower intensity of the stimuli in our experiment (65 dB). We decided to increase the intensity to 90 dB in the current studies, keeping a 65 dB condition for control purposes. We hypothesized an increase in amplitude of the N1-P2 components at the scalp and hoped to see a reversal of these components at the nasopharynx. Our manipulation increased the amplitude of the N1-P2 to the targets but not to the standards. There were still no components at these latencies in the nasopharyngeal recordings. Somewhat unexpectedly, the intensity manipulation had an effect on the amplitude of the scalp N2, which was not found for the nasopharyngeal N2. This was interpreted as providing further support for the existence of two distinct processes in the N2 latency range.

Discrimination difficulty

This condition was useful in three ways. 1) Increasing the difficulty of discrimination targets from standards is known to increase the latency of both the N2 and P3 components and decrease the amplitude of the P3 (Ford et al. 1976; Fitzgerald and Picton 1983; Campbell et al. 1983). It was therefore important to check if the nasopharyngeal N2 and P3 were similarly affected. We hypothesized similar effects on both the scalp and nasopharyngeal endogenous

components, a prediction that was verified. 2) Since the standards were 2000 Hz in the easy condition and 1050 Hz in the difficult condition, everything else being kept equal, we could study the direct effects of stimulus frequency on the ERP components at both the scalp and the nasopharynx. Following the results of Picton et al. (1978b), we hypothesized no changes in the mesogenous components between these specific frequencies. This hypothesis was confirmed. 3) A comparison of target responses in the easy condition to those of the difficult condition provides information concerning the frequency-specificity of the N1a, N1b, N1c and P2 components. Our only hypothesis concerned the frequency-specificity of the N1 component, which we confirmed.

Stimulus omissions

Stimulus omissions provide a view of the components which can be elicited without external stimulation. The N2, P3 and Slow Wave components remain in the scalp waveform in these conditions and we were interested to see whether the nasopharyngeal components were also truly endogenous. The second omission condition, with a shorter ISI, was introduced to examine and reduce the effects of latency jitter on the scalp components, and again check if their nasopharyngeal counterparts would be similarly affected. We hypothesized covariation of the scalp and nasopharyngeal N2 and P3

components following stimulus omissions. Our results confirm this hypothesis for P3, but again we found indications of two distinct processes in the N2 latency range.

Attention

The attention condition permitted to verify the occurrence of Näätänen's (1979) processing negativity by presumably reducing it in the Ignore condition. Furthermore a P3a component has been reported in response to the targets in Ignore conditions (Squires et al. 1975). We wanted to know whether this component would also appear at the nasopharynx. Finally, Goodin et al. (1980) have reported the existence of a P165 component that can be identified in the waveform following an Attend-Ignore subtraction. We investigated the possibility of a corresponding component at the nasopharynx. We hypothesized a scalp-nasopharynx covariation for the N2 and P3 components. Furthermore, we expected the occurrence of a P3a in the Ignore waveforms, and the appearance of a P165 in the subtraction waveforms, with the P3a reversing at the nasopharynx. Our attention manipulation was not successful in revealing either of these components, preventing consideration of possible generators. We hypothesized an amplitude diminution of P3 at the scalp and nasopharynx, which was confirmed. The N2 results again revealed a dissociation between scalp and nasopharynx.

Modality

We compared results from the auditory and visual modalities, in order to see whether both scalp and nasopharyngeal components would be similarly affected. The scalp-distribution of the N1 component is modality-specific. It could therefore occur at the nasopharynx in the visual modality even though it was not seen in the auditory modality. A similar rationale applies for the N2 component. On the contrary, the P3 and Slow Wave components are not thought to be modality specific. Their representation at the nasopharynx should therefore be the same in both modalities. We hypothesized changes in the Cz/nasopharynx reversal percentages for the N1 and N2 components, since they show different scalp-distributions for the auditory and visual modalities. This hypothesis was confirmed. No changes in reversal percentages were predicted for the P3 and Slow Wave components since these are not affected by stimulus modality. This hypothesis was also confirmed.

THESIS FORMAT

The core of this thesis consists of two articles to be submitted to the Journal of Electroencephalography and Clinical Neurophysiology. For this reason, the format is that required by this publication. An article concerning the results of the preliminary experiments has already been

published (Perrault et al. 1983). It can be found in Appendix A. Appendix B contains tables of mean latencies and amplitudes for the ERP components measured in the core studies.

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EVENT-RELATED POTENTIALS RECORDED FROM THE SCALP AND NASOPHARYNX. I. MESOGENOUS COMPONENTS.

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A distinct negative-positive complex, commonly called N1-P2, can be recorded from the human scalp in response to transient auditory stimuli. The peak latencies of this response are typically 100 and 180 ms. The response is maximally recorded from midline frontocentral electrodes and has therefore often been called the "vertex potential". A response of similar morphology occurs following transient stimuli in the somatosensory or visual modalities (Goff et al. 1969). These waves, which are affected by both the physical parameters of the stimulus and the psychological state of the subject, have been considered as "mesogenous" in nature (Hillyard et al. 1978). This is for the present a convenient terminology since it distinguishes these components of the response from earlier components which are more completely determined by stimulus properties and later components which are more affected by psychological parameters. It is certainly possible that the mesogenous potentials may be composed of overlapping exogenous and endogenous processes but we are at present unable to easily dissociate such subcomponents.

Despite extensive research, the cerebral origins of the auditory vertex potential remain unknown. Vaughan and Ritter (1970) reported that the auditory N1-P2 component measured using a nasal reference reversed in polarity across

the Sylvian fissure. They therefore postulated that these waves were generated by a vertically oriented dipole in the primary auditory cortex on the superior surface of the temporal lobe. These results were criticized by Kooi et al. (1971) who found no evidence of polarity reversal across the Sylvian fissure when the potentials were recorded using a non-cephalic reference. They suggested that Vaughan and Ritter's results were better explained by an active nose reference than by a Sylvian dipole. Other studies supported such conclusions (Picton et al. 1974; Streletz et al. 1977; McCallum and Curry 1980). Vaughan (1974), however, argued that an inverted vertex potential is recorded from the non-cephalic sternovertebral reference, since this location lies in the direction of the maximum potential generated by the vertically oriented temporal dipole. The nose reference is less likely to be contaminated by this potential, since it would lie on the equipotential line of such a dipole. Using even more distant references Streletz et al. (1977-wrist) and Picton et al. (1978c - ankle) found no time-locked activity at the sternovertebral site, whereas a significant amount of activity could be seen at the nose location.

In a recent topographical study, Wolpaw and Wood (1982) attempted to settle the issue of electrical activity at reference sites. Using a non-cephalic sternovertebral

reference, they found voltage gradients to be steepest in the temporal region, becoming extremely shallow at locations on the upper neck and remaining essentially flat below that point. These findings occurred for all subjects, independently of the amount of activity recorded between the nose and sternovertebral reference. Wolpaw and Wood thus conclude that the sternovertebral site is not significantly active during recordings of the auditory evoked potential.

Studies of patients with lesions of the auditory cortex have not produced convincing evidence for the exact cerebral source of the N1-P2 waves. Peronnet and colleagues (Peronnet et al. 1975; Peronnet and Michel 1977), using a nose reference, recorded from patients with unilateral damage to the auditory cortex. They argued that if Vaughan and Ritter were correct, a reversal of the vertex potential should be seen only on the side of their patients' healthy hemisphere. Their recordings support their hypotheses but are difficult to interpret because the phase reversals noted in some of the patients are much larger than those noted in normal subjects. These findings can only be explained by the removal of some overlapping components with opposite polarity to those remaining. Knight et al. (1980) found that the N1-P2 persisted after frontal lobe lesions indicating that the frontal lobe is not their source. They found that unilateral parietotemporal lesions decreased the amplitude

of the response, but the attenuated response was not asymmetrical. This suggests that although the parietotemporal cortices influence the response, they are not its major source. Woods et al. (unpublished manuscript) reported that bilateral lesions of the primary auditory cortex did not remove the N1-P2 response although others have reported definite attenuation of the response after such lesions (Jerger et al. 1969).

Intracerebral recordings have also produced conflicting evidence. During intracerebral recordings from the brain of alert rhesus monkeys, Arezzo et al. (1975) found a reversal of many auditory evoked potentials below the level of the supratemporal plane. It is difficult to determine whether any of these components are homologous to the N1-P2 of the human evoked potential. Furthermore, Arezzo and his colleagues also found that some components of the auditory evoked potential were generated in the frontal cortex. Celesia et al. (1971) did not find any components in human intracerebral recordings of the primary auditory cortex that correlated with the N1-P2 components. Goff et al. (1980) were unable to find any inversion of polarity for the auditory N1-P2 components either across the frontal cortex or across the level of the Sylvian fissure.

Wolpaw and Penry (1975) proposed two overlapping complexes at the time of the N1-P2 components of the

auditory evoked response. They suggested that in the temporal areas there was a positive-negative complex overlapping the vertex N1-P2 components. The positive (Ta) and negative (Tb) peaks occurred at latencies of 105-110 ms and 150-160 ms respectively. They recorded this temporal potential using a complex subtraction procedure, and found latency differences between T3 and T4 that were related to the ear of delivery. Wolpaw and Penry proposed that recording of their Ta positivity in previous experiments by Vaughan and Ritter may have been mistaken for a reversal of the vertex potential. Other researchers have also supported the existence of multiple components in the N1 latency range (Picton et al. 1978c; McCallum and Curry, 1980). McCallum and Curry report two distinct negative peaks in the temporal recordings (N1a-75 ms and N1c-129 ms) in the vertex N1 (N1b) latency range. The amplitude and latency of the temporal N1c component were related to the ear of delivery. In a recent scalp-distribution study, Wood and Wolpaw (1982) provide further evidence for several overlapping potentials in the N1-P2 latency range.

Studies of attention have suggested that there may be other components of the auditory evoked potential at these latencies. In recent years, enhancement of the vertex potential has been associated with an early stage of attentional processing (Hillyard et al. 1973; Picton and Hilly-

ard 1974). NäätÄnen and his colleagues (NäätÄnen et al. 1978; NäätÄnen and Michie 1979) have proposed that such attentional effects may in fact be due to changes induced in a distinct but overlapping "processing negativity". Several of the properties attributed to the auditory vertex response may thus belong to this slower negative process.

Nasopharyngeal electrodes have been extensively used in clinical electroencephalography to record from the mesial surface of the temporal lobe (Mavor and Hellen 1964; deJesus and Masland 1970; see Appendix C for a more precise description of their location). They could therefore provide a view of the event-related potential quite different from that recorded from the scalp. This view from the dark side of the brain might improve our understanding of the component structure of the eventrelated potentials. Smith et al. (1973) reported that the N1 and P2 components of the auditory vertex potential were recorded with opposite polarity from nasopharyngeal electrodes referred to a balanced non-cephalic reference, a finding consistent with Vaughan and Ritter's postulated dipole. On recordings taken between the ear and the nasopharynx (Peters and Reilly 1973), however, the only definitely recognizable evoked potential was a small ear-negative (or nasopharyngeal positive) wave occurring about 35 ms later than the N1 peak at the vertex. In our own nasopharyngeal recordings (Perrault et al. 1983), the only wave recorded in the latency

range of the vertex N1-P2 components was a small positivity occurring at 130 ms. We conjectured that this nasopharyngeal positivity might represent a reversal of the temporal negative peak (Tb) of Wolpaw and Penry (1975). We had not recorded from temporal electrodes, however, and could not confirm this hypothesis.

This paper presents data recorded simultaneously from the midline and temporal locations of the scalp and from the nasopharynx. The terminology introduced by McCallum and Curry (1980) will be used: N1a refers to the temporal negative peak occurring at approximately 75 ms; N1b refers to the negative peak recorded with maximum amplitude from the vertex at a latency of about 95 ms; N1c refers to a second temporal negative peak at approximately 130 ms, that corresponds roughly to Wolpaw and Penry's Tb component. The mesogenous components are evaluated in relation to interstimulus interval, intensity, frequency, attention and modality.

METHODS

Seventeen male and six female subjects ranging in age from 22 to 35 years participated in these experiments. All subjects reported normal hearing and vision. The results from two of these subjects were rejected, one because of a prominent respiratory artifact on the nasopharyngeal re-

cordings, and the other because of very frequent blinking artifacts. Each recording session lasted about 2 1/2 hours. The subjects sat in a comfortable chair in an electrically shielded room and listened to 7-minute trains of auditory stimuli, mentally counting the "target" stimuli that were randomly interspersed among the more frequent "standard" stimuli. All subjects were evaluated in a basic paradigm and in two or more variants thereof. (Each paradigm was repeated for at least two 7-minute periods). At least ten subjects participated in each variant paradigm, several subjects returning for more than one recording session. The presentation of conditions within a recording session was randomized.

In the basic paradigm, tones of 55 ms total duration with 5 ms rise-and-fall times were presented to the right ear through a TDH-49 earphone. The tones were presented at an intensity of 90 dB peak SPL and an interstimulus interval of 1.1 s. Ten percent of the tones were "targets" with a frequency of 1000 Hz, and the other 90% were "standard" stimuli with a frequency of 2000 Hz. At these frequencies and durations the intensity of a 90 dB peak SPL tone was about 75 dB above normal threshold (nHL).

There were five variants of the basic paradigm. In the first, the interstimulus interval was increased to 3.3 s. The target probability was also increased to 30% in order to

maintain the same temporal probability. Ten subjects also participated in an additional study where the ISIs were 0.65 s and 5.0 s. In this study the target probability was kept at 10% across ISI conditions, and stimuli were delivered to the left ear. Results from both ISI manipulations will be presented concurrently. In the second variant, the intensity of the stimuli was decreased to 65 dB peak SPL. In the third, the task was made more difficult by changing the frequency of the standard stimulus to 1050 Hz. In the fourth, the subject was asked to ignore the ongoing train of stimuli presented to the left ear at a rate of 1/0.65 s, and to concentrate on reading a book or magazine instead.

In the fifth variant, stimuli were presented in the visual modality. The timing of the stimuli in the visual paradigm was equivalent to that of the basic auditory paradigm. Flashes from one red and one green light-emitting diode were channeled through the rounded tip of a short glass rod (15 mm in length; 5 mm in diameter), so that both types of stimuli came from the same location. The "targets" had one of the colours (counterbalanced across subjects) and were presented 10% of the time. The display apparatus was located approximately 160 cm from the subject's eyes.

The EEG activity was recorded using Beckman Biopotential Ag/AgCl electrodes affixed to the scalp at Fz, Cz, Pz,

T3 and T4 with adhesive collars and collodion-soaked gauze (Picton et al. 1978a). The electro-oculogram (EOG) was monitored between the middle of the supraorbital ridge and the inferior outer canthus of the left eye. The skin was punctured with a sterile needle under each of the scalp and EOG electrodes to prevent skin potential artifacts (Picton and Hillyard 1972). The nasopharyngeal electrodes obtained from the Montreal Neurological Institute consisted of a doubly bent silver-tipped wire, coated with plastic except at the tip. These electrodes were chlorided following the methodology proposed by Cooper, Osselton and Shaw (1974). All electrodes were referred to a balanced non-cephalic reference (Stephenson and Gibbs 1951).

The EEG and EOG signals were amplified using Beckman R611 polygraph amplifiers with high frequency filters set at 30 cps and a time constant of 0.8 s. Analog-to-digital (A-D) conversion was performed by a PDP MINC-11 computer over a period of 900 ms beginning 50 ms prior to each stimulus. One set of A-D values was obtained every 4.5 ms. Averaging was performed offline from disk-stored single-trial waveforms. Epochs containing EEG or EOG potentials greater than +100 uV were not included in the averages.

The possibility of artifacts produced by the experimental apparatus being recorded in the averaged nasopharyngeal waveforms was investigated in two subjects using three

conditions. The first condition was a replication of the previously described difficult discrimination paradigm. The second condition investigated the possibility of a transient magnetic field generated by the earphone and picked up by induction in the nasopharyngeal electrodes. To eliminate this possibility stimuli were delivered at 90 dB peak SPL to the subject's right ear through a rubber tube 120 cm in length, (0.6 cm inside diameter), fitted at the subject's end with an MX44 rubber cushion similar to that of the TDH-49 earphone. The electro-acoustic transducer was a Dyna Magnetic Devices D-308 phone fitted tightly at the other end of the tube. This phone was secured on the arm of the subject's chair at approximately 80 cm from his right ear and at a minimum of 50 cm from the nearest nasopharyngeal lead. Because of the low pass filter effect of the tube, stimulus frequencies of 1000 Hz (targets) and 1050 Hz (standards) were used. Since the frequencies were close together, any alteration in the spectral properties of the stimuli due to tube conduction would equally affect both stimuli. The subjects did not report any difference between the stimuli delivered through the tube and those delivered through the TDH-49 earphone. The possibility of hardware or software artifacts originating in the experimental apparatus was investigated in a third condition wherein the rubber tube was clamped and the subject's auditory meatus plugged

with wet cotton puffs. The subjects reported hearing no tones during this session.

Amplitude and latency measurements

The mesogenous components of the evoked response were identified in the average waveforms of individual subjects according to the following rules. The N1b component was identified at the Cz electrode as the maximum negativity occurring between 75 and 125 ms. Two distinct peaks were observed in the waveforms from the temporal leads. The first one (N1a) was identified separately at T3 and T4 as the maximum negativity in the latency range from 40 to 125 ms. The second peak (N1c) was also identified separately at T3 and T4, as the maximum negativity between 80 and 160 ms. If one of these components was identifiable as a distinct peak at only one temporal electrode , then the peak latency at this electrode was used to measure the amplitude of the waveform at the opposite temporal lead. A small positive peak (PgP120) was identified in the nasopharyngeal recordings within the same latency range as the N1b component. Its latency was determined at the nasopharyngeal electrode where it was most clearly seen, and its amplitude measured at this latency for both nasopharyngeal waveforms. The P2 component was identified separately at Pz, T3 and T4 as the maximum positive peak occurring between 150 and 275 ms.

In the response to standard visual stimuli, an N1 component was also identified at the midline electrodes as the maximum negativity between 155 and 210 ms. Within this latency range a PgP190 component was identified separately at the nasopharynx. A visual P2 component was identified at the Cz electrode as the maximum positivity between 175 and 310 ms. These components were preceded by a P1 component identified at the Pz electrode as the maximum positivity occurring between 130 and 165 ms. A corresponding positivity was identified at the nasopharynx within the same latency range (PgP145).

Waveforms were smoothed twice (using a binomial smoothing program) prior to plotting and measurement. Amplitudes were measured at each of the identified latencies with respect to the average amplitude of the waveform in the 50 ms preceding stimulus onset. We chose baseline-to-peak rather than peak-to-peak measures, since the latter can always be derived from the former using appropriate additive procedures. Experimental effects were ascertained by pairwise comparisons using one-way repeated measures analyses of variance. Five types of comparisons were made: 1) comparisons of the amplitude or latency of a given component at a given electrode location across two experimental conditions. Such comparisons were always done for the point of maximum amplitude on the scalp and at the corresponding

latency (or component) in the nasopharyngeal waveform. Data from other electrodes were sometimes analysed to check on the spatial extent of experimental effects; 2) comparisons of component latencies across electrodes, within the same experimental condition (e.g. P3-Cz versus P3-Pg); 3) comparisons of the T3/T4 ratios for the N1a and N1c components of the temporal recordings, and of the Pg1/Pg2 ratios for the nasopharyngeal PgP120 component, across ear of delivery. We used ratio limits of 0 and 2. 4) comparisons of the left side (T3) and right side (T4) amplitude values of the N1a, N1c and P2 components to determine possible amplitude asymmetries. Same type of comparison for PgP120 at Pg1 versus Pg2. 5) comparison of Fz/Pz ratios for the N1 component in the visual versus auditory modalities. Differences between means were considered significant at $p<0.01$. The existence of components in particular channels was checked by evaluating the 1% confidence limits of the grand-mean waveform at the latency of the component. All subjects were weighted equally in the grand-mean waveforms.

RESULTS

Basic Auditory Paradigm.

The responses of 20 subjects to the standard stimuli in the basic paradigm are shown in Figure 1. One subject is not included because of data-retrieval problems. The N1b peak had an average latency of 94 ms at Cz. It was maximally recorded over the frontocentral region of the scalp with an average amplitude of -5.6 uV at Fz, -5.7 uV at Cz and -3.3 uV at Pz. The amplitude of the temporal waveforms at the Cz latency was -1.3 uV at T3 and -1.2 uV at T4. There was no evidence of any component being present at this latency in the nasopharyngeal recordings, where the mean amplitude of the waveform (0.2 uV) did not differ significantly from zero. N1b appears to be the only component consistently noted in the EOG channel.

----- Insert Figure 1 about here ----

The P2 component was recorded with maximum amplitude from the Cz electrode (3.0 uV) at a latency of 197 ms. It had an average amplitude of 1.6 uV at Fz and 2.1 uV at Pz.

It occurred at a similar latency at both temporal derivations (198 ms). Its amplitude was significantly larger at the T4 (2.5 uV) than at the T3 electrode (1.8 uV). No significant activity was recorded from the nasopharynx at this latency.

A double negativity could be observed in the temporal recordings. The first negative peak (N1a) occurred with a mean latency of 74 ms at the temporal leads (72 ms at T3 vs 75 ms at T4; not significant). Its amplitude was generally greater at T3 (-1.7 uV) than at T4 (-1.0 uV), but this difference did not reach significance. At the latency of the N1a component the amplitude of the nasopharyngeal waveform did not differ significantly from zero. The second negative peak (N1c) occurred at mean a latency of 130 ms at both T3 and T4. Its amplitude was significantly greater at T3 (-2.3 uV) than at T4 (-1.6 uV), the stimuli in this condition being presented to the right ear.

The only discernible component occurring in response to standard auditory stimuli in the nasopharyngeal recordings was a small positivity peaking at 120 ms (PgP120) with an average amplitude of 0.7 uV at both Pg1 and Pg2 electrodes. This latency did not differ significantly from the latency of the temporal N1b component. Results from our control experiment show that this nasopharyngeal positivity was not caused by an artifact of our experimental or recording

procedures. Figure 2 shows that the nasopharyngeal positivity could be clearly seen with an average amplitude of 1.0 uV at 122 ms using either earphone system and was absent when hearing was prevented.

-----Insert Figure 2 about here-----

Interstimulus Interval (ISI)

Twelve subjects were tested at ISIs of 1.1 s and 3.3 s, with target probabilities of 10% and 30% respectively. Ten subjects were tested at ISIs of 0.65 s and 5.0 s, the target probability being held constant at 10%. The grand-mean waveforms for the 1.1s and 3.3s ISI conditions are superimposed in Figure 3. There were no significant changes in the latency of the N1 component. Its amplitude at Cz increased significantly with increasing interstimulus interval from -5.9 to -9.9 uV. The nasopharyngeal waveforms in the long ISI condition still did not differ significantly from baseline at the latency of the N1b component. Grand-mean waveforms for the 0.65 s and the 5.0 s ISI conditions are presented in Figure 4. The amplitude of the N1 component at Cz again increased significantly from -3.4 uV at 0.65 s ISI to -10.0 uV at 5.0 s ISI. Its latency increased slightly from 104 to 117 ms, but this difference did not reach significance. An examination of N1b amplitude over the 5

subjects common to all four ISI conditions showed average values at the Cz electrode of -3.5, -5.3, -10.0 and -10.8 uV at ISIs of 0.65, 1.1, 3.3 and 5.0 s.

-----Insert Figures 3 and 4 about here-----

The P2 component showed no significant latency differences with increasing ISI from 1.1 s to 3.3 s. Its amplitude increased significantly from 2.8 to 6.5 uV at Cz. This effect was also significant at Pz, but not at Fz, T3 and T4. The corresponding nasopharyngeal values did not differ significantly from zero. The larger ISI variation (0.65 s versus 5.0 s) caused a significant increase in P2 latency from 183 to 214 ms. The amplitude of this component also increased significantly from 1.2 uV to 9.0 uV at Cz. The average P2 amplitudes over the five subjects that were run under all four ISI conditions were 1.2, 2.2, 6.3 and 8.5 uV.

The latency of the N1a component was unaffected by either ISI or electrode location. Its amplitude increased significantly from -1.7 to -2.6 uV at T3 and from -1.3 to -2.6 uV at T4 when the ISI was increased from 1.1 s to 3.3 s (right ear). The amplitude of this component increased from -1.3 to -2.3 uV at T3 and decreased from -1.1 to -0.7 uV at T4 in the larger ISI variation (left ear), but these dif-

ferences did not reach significance. The N1c component (average of T3 and T4) increased significantly when the ISI was increased from 1.1 s (-2.0 uV) to 3.3 s (-3.4 uV). A significant difference was also found when comparing ISIs of 0.65 s (-1.9 uV) and 5.0 s (-3.1 uV). Average amplitude values for the five subjects that participated in all four ISI conditions were -1.9 uV at 0.65 s, -2.0 uV at 1.1 s, -2.6 uV at 3.3 s, and -3.7 uV at 5.0 s. The latency of the N1c showed no significant change (135, 128, 129, and 137 ms with increasing ISI). The ear of delivery effects will be covered later.

The PgP120 component increased from 0.6 to 1.1 uV (average of Pg1 and Pg2) when the ISI was increased from 1.1 to 3.3 s, but this difference did not reach significance. The larger ISI manipulation from 0.65 s to 5.0 s caused a significant increase in the amplitude of this component, from 0.7 to 1.6 uV. The latency showed no significant variation with ISI (121, 120, 136 and 140 ms).

Stimulus Intensity

No significant effects of stimulus intensity were found in the responses to standard stimuli. For target stimuli, the N1b component at the Cz electrode increased significantly from -4.9 to -7.2 uV with increasing intensity. The amplitude of the nasopharyngeal waveform at this

latency was not significantly different from zero at either intensity. The N1c component increased significantly from -2.6 to -4.0 uV at T3 and from -1.8 to -3.2 uV at T4 with increasing intensity. The PgP120 component was not significantly affected (0.9 vs 1.1 uV). The P2 component showed a significant decrease in amplitude with increasing intensity (1.8 vs 0.8 uV), an effect probably due to changes in overlapping endogenous components. The grand-mean waveforms for target stimuli under both intensity conditions are presented in Figure 5 of the second paper (Perrault and Picton, 1983).

Stimulus Frequency

When the frequency of the standard stimuli was lowered from 2000 Hz to 1050 Hz in order to make target discrimination more difficult there were no significant changes the amplitude or latency of any of the measured components of the evoked potential to the standard stimuli.

The target stimuli were presented at a frequency of 1000 Hz in both the easy and the difficult discrimination tasks. No effect was found on N1b latency but its amplitude at Cz decreased significantly from -9.2 to -6.3 uV with increasing discrimination difficulty. This effect was significant at all midline electrodes. No significant effects were found at the nasopharynx at this latency. The

P2 and N1a components were not significantly affected by discrimination difficulty. The N1c component, however, decreased significantly from -4.8 to -2.8 uV with increasing difficulty (average of T3 and T4). There were no significant effects on the nasopharyngeal PgP120 component (1.1 vs 1.3 uV). The grand-mean waveforms for target stimuli are presented in Figure 6 of the second paper (Perrault and Picton, 1983).

Effects of Attention

The grand-mean waveforms for the Attend and Ignore conditions are shown in Figure 5. The only component affected by our attention manipulation in the response to standard stimuli was the temporal N1c component whose amplitude diminished from -2.3 to -1.5 uV at T4 when the stimuli were ignored. The effect was not quite significant ($0.01<p<0.05$). This small effect, however, is replicated in the response to the second stimulus that occurs 0.65 s after the first stimulus in the recordings plotted in Figure 5. In the response to target stimuli, the N1c component diminished significantly ($p<0.01$) from -4.8 to -3.2 uV at the T4 electrode. There were no significant effects of attention on the PgP120 component recorded from the nasopharyngeal electrodes (Attend-Ignore: standards 0.7 - 0.4 uV, targets 0.5 - 0.9 uV; averages of Pg1 and Pg2). The

grand-mean waveforms for targets are presented in Figure 9 of the second paper (Perrault and Picton, 1983).

----- Insert Figure 5 about here -----

We attempted to isolate the processing negativity by subtracting the responses to stimuli in the Ignore condition from those in the Attend condition. No obvious processing negativity could be seen in the difference waveforms outside the latency range and scalp-distribution of the temporal Nlc component.

Ear of Delivery

Ear of delivery effects on the temporal and nasopharyngeal components were assessed in a group of ten subjects who underwent both left and right ear stimulation. Interstimulus intervals were 0.65 s for the left ear and 1.1 s for the right ear. Left and right ear recording sessions did not occur on the same day. In order to compensate for the ISI effects, only T3/T4 and Pg1/Pg2 ratios were evaluated. The Nla component of the temporal complex was unaffected by ear of delivery. The average T3/T4 ratios for ten subjects run under both conditions are 1.1 for the left ear, and 0.8 for the right ear, not a significant difference. The Nlc component was significantly greater on the

side contralateral to stimulation. The average T3/T4 ratios are 0.7 for the left ear and 1.5 for the right ear, the difference being significant. The Pg1/Pg2 ratios for the PgP120 component did not differ across ear conditions (left ear: 0.8; right ear: 0.7). There were no significant latency effects. Similar results were found at ISIs of 3.3 s (right ear) and 5.0 s (left ear), over five subjects run under both conditions.

Responses to Visual Stimuli

The average individual waveforms for this experimental manipulation are presented in Figure 6. The grand-mean waveforms for both standard and target stimuli can be seen in Figure 11 of the second paper (Perrault and Picton 1983). The first peak noted in the response to standard stimuli was a small positivity recorded maximally at Pz with an average latency of 145 ms and a mean amplitude of 1.6 uV. The amplitude of this component (P145) was 0.9 uV at Cz and 0.7 uV at Fz. It was also seen in the recordings from the temporal leads where its mean amplitude was 0.5 uV, and in the nasopharyngeal recordings where it averaged 0.4 uV (no reversal). Although this was not significantly different from baseline, it was quite distinct and was significant in the target responses (Perrault and Picton 1983).

----- Insert Figure 6 about here -----

A visual N1 peak was recorded with maximum amplitude at Pz (-1.0 uV) where it occurred at a latency of 186 ms (N190 or visual N1). At other scalp locations it was either positive (1.2 uV at Fz) or at baseline level, although its morphology was still that of a negative-going peak. At approximately the same latency (190 ms) a significant positivity could be seen at the nasopharynx (PgP190), with an average amplitude of 1.7 uV. In the response to target stimuli, the N1 component was recorded at a latency of 185 ms (Pz). It was positive at all recording locations, averaging 2.2 uV at Fz, 2.0 uV at Cz, 0.6 uV at Pz and 1.0 uV at T3 and T4. At a slightly later latency (197 ms) a significant positive peak was again observed at the nasopharynx, with an average amplitude of 2.3 uV. A comparison of Fz/Pz ratios for auditory (1.7) and visual (-1.2) N1 amplitudes indicated a significantly more posterior distribution for the visual N1 component.

The major component of the visual evoked response to standard stimuli was maximally recorded from Cz as a positive peak reaching 5.5 uV in amplitude at a latency of 237 ms (visual P2 peak). It was equally distributed over the frontal and parietal regions (4.3 uV) and reached its lowest scalp amplitude at the temporal leads (2.2 uV at both T3 and

T4). The average amplitude of the nasopharyngeal waveform at this latency did not depart significantly from baseline (mean of 0.3 uV). In the response to target stimuli, the P2 component occurred at a latency of 230 ms (Cz). Its scalp-distribution was also frontocentral, with values of 8.0 uV at Fz, 8.1 uV at Cz, 6.4 uV at Pz and 3.7 uV at the temporal derivations. The amplitude of the nasopharyngeal waveforms at this latency averaged 1.7 uV, but this is probably due to overlapping endogenous components.

DISCUSSION

These results replicate and extend our previous findings (Perrault et al. 1983). We confirm the absence of any nasopharyngeal concomitants of the N1 and P2 components and demonstrate the existence of a nasopharyngeal positivity at approximately 120 ms. Our various experimental manipulations indicate that there are multiple overlapping components in the mesogenous scalp-recorded waveform. These will be discussed in the following paragraphs.

The Vertex N1b Component

Our experimental manipulations caused no significant changes in the latency of this frontocentral component. Its amplitude increased significantly with increasing interstimulus interval. The recovery function of this component seems to taper off somewhere between 3.3s and 5.0 s. The significant amplitude difference for target responses between easy and difficult discrimination conditions is indicative of a frequency-specific recovery function and agrees well with the results previously reported by Butler (1968, 1972) and by Picton et al. (1978a). The absence of an intensity effect on the amplitude of the N1 component to the standard stimuli seems due to a saturation effect occurring at moderate to high intensities at rapid rates of

stimulus presentation (Moore and Rose 1969; Picton et al. 1970). The N1b to the less frequently occurring targets was increased by increasing intensity. Changing the frequency of the standard stimulus had no effect on the amplitude of the N1 component. Given the frequencies that were used (2000 Hz and 1050 Hz), this finding also agrees with the results previously reported by Picton et al. (1978b), who found little if any effect of stimulus frequency on the N1-P2 components at frequencies less than 2000 Hz. Contrary to what has been previously reported (Picton and Hillyard, 1974), we found no effect of attention of the amplitude of the vertex N1b component. The task did not, however, require selective attention within the auditory modality (Hillyard et al. 1973) and attention to a relatively simple single-channel auditory task may or may not result in an enhanced N1 component depending on the difficulty of the task.

In spite of the high stimulus intensities and long interstimulus intervals used in our experiments, we found no evidence of N1 reversal at the nasopharynx. Smith et al. (1973) have previously reported a reversal of the vertex auditory N1 at the nasopharynx. In their Figure 2, however, the nasopharyngeal positivity occurs at about 120 ms, whereas their vertex N1 peaks at approximately 102 ms. Their nasopharyngeal positivity thus probably corresponds to our PgP120 component. In our recordings, the nasopharyngeal

waveform at the latency of N1b stayed at baseline level through all our manipulations. Nasopharyngeal electrodes appear very well located to record from the lower end of a Sylvian dipole (Vaughan, 1974; Vaughan and Ritter, 1970; Vaughan et al. 1980), or of a dipole located in the frontal cortex (Picton et al. 1974; Kooi et al. 1971; Streletz et al, 1977). Our results thus seem to indicate that the generator of the N1 component is not located in these areas. It is very difficult to postulate a vertical dipole that shows no positivity at either the nasopharyngeal of midtemporal electrodes. It is possible that such a positivity may be overlapped at the nasopharyngeal electrode by a simultaneous negativity (perhaps the underside of a laterally oriented Ta dipole - see next paragraph). This is unlikely since one would have to postulate that both overlapping components are affected in exactly the same way by all our manipulations.

Wolpaw and Penry (1975) have previously reported a "Ta" positivity at the temporal leads in non-cephalic recordings. Wood and Wolpaw (1982) have also reported such a temporal positivity (TP78) which appeared to coincide approximately with the vertex N1 (FN88). Such results are consistent with a vertical dipole in the superior temporal plane, although the vertex negativity and the temporal positivity could also be related to separate generators. The latter explanation

is more likely since most subjects (Figure 1) do not have an actual positive voltage at the temporal regions but just a positive deflection. The existence of a vertically oriented current source in the superior temporal plane at the approximate latency of the N1b seems probable from the recent research on auditory evoked magnetic fields (Bak et al. 1981; Hari et al. 1980; Reite et al. 1982; Zimmerman et al. 1982). As pointed out by Wood and Wolpaw, however, latency and morphology differences between vertex and temporal waveforms as well as the fact that the TP78 component is only a relative positivity in several subjects, indicate that another current source might create overlapping potentials during this time period.

Besides the superior surface of the temporal lobe, several structures have been postulated as possible generators of the N1b component. The findings of Knight et al. (1980) raise the possibility that the parieto-temporal cortex is involved, but this does not easily explain the scalp-distribution of the N1b. The possibility of a callosal generator seems unlikely, in view of the results of Gazzaniga and Hillyard (1973) showing that the N1b component of the auditory evoked response can still be clearly seen in split-brain patients. The hippocampus can also probably be ruled out on the basis of recent intracerebral recordings in humans showing no definite activity in the N1b latency range

(Halgren et al. 1980; Wood et al. 1980, 1983). In the somatosensory modality, generators for the N1 component have been suggested to lie in thalamic structures (Velasco et al. 1980) or in the anterior part of the cingulate gyrus (Chatrian et al. 1975). There are also indications that the centromedian nucleus of the thalamus might be involved in the generation of the N1b component. Ervin and Mark (1964) have recorded activity in the centromedian nucleus following stimulation in the somatosensory, visual and auditory modalities. This activity reached a peak amplitude at about 110 ms. Furthermore, it was markedly reduced when the subjects were distracted from the eliciting stimuli by having them perform a mental task. The hypothesis of a thalamic generator seems consistent with our findings. Such a generator, if oriented vertically with a frontal tilt, might not be picked up at the nasopharyngeal recording sites.

The temporal N1a component

The temporal N1a component covaries with the vertex N1 in all our manipulations. We were unable to replicate the N1a asymmetry (greater over the dominant hemisphere) reported by McCallum and Curry (1980). They do not, however, mention the significance of this asymmetry. An examination of our grand-mean waveforms seems to indicate an identical

onset time for the temporal Nla and the vertex Nl components. It is thus possible that both peaks represent a single process. As with the vertex Nl, the Nla component is not seen in the nasopharyngeal recordings, thus strengthening this hypothesis. This proposition, however, entails the existence of a temporal positivity giving the temporal waveform its two 2 distinct negative peaks. Such a positivity (Ta) has indeed been proposed by Wolpaw and Penry (1975) and measured following a Temporal-Vertex subtraction procedure to remove the Nl overlap at the temporal sites. Wood and Wolpaw (1982) have also recorded a temporal positivity at 78 ms (TP78) for which they suggest an origin in the lateral surface of the temporal lobe.

The temporal Nlc component

The temporal Nlc component appears to represent a distinct component of the auditory evoked potential. It is recorded with maximum amplitude over the temporal area contralateral to the ear of stimulation, a finding already reported by Wolpaw and Penry (1975) for their Tb component, and by McCallum and Curry (1980) for their Nlc component. A temporal negativity at 115 ms (TN115) was also reported by Wood and Wolpaw (1982). It was recorded with maximum amplitude between the C4 and T4 locations. The scalp topography of this component was consistent with a source in

the lateral temporal surface.

The Nlc component increases in amplitude with attention, especially on the side contralateral to stimulation. This might relate to Michie's finding (1983) of lateralized attention effects (at a somewhat later latency) in the somatosensory modality. This was interpreted as suggesting a lateralized processing negativity. The question thus arises whether the temporal Nlc and the processing negativity represent the same process. A broad processing negativity overlapped by Wolpaw and Penry's Ta positivity and by the later P2 component could appear as a discrete negative peak. Inasmuch as the nasopharyngeal positivity to be discussed next represents a reversal of the Nlc component, however, this explanation cannot be sustained since the nasopharyngeal positivity also appears as a discrete positive peak in spite of the absence of any P2 component at the nasopharynx. The Nlc component might be distinct from both Nl and the processing negativity, being related to auditory processing and to the ear of delivery, but unaffected by attention. Attention may result in a lateralized processing negativity that overlaps the scalp-recorded Nlc but does not alter its PgPl20 concomitant.

The nasopharyngeal positivity (PgPl20)

The PgPl20 component appears as a distinct component of

the evoked waveform. Our control studies demonstrate that it is not an artifact of our experimental or recording apparatus. The possibility of muscle artifacts contributing so consistently to the waveform at this latency is unlikely. The PgP120 to corresponds in latency with the temporal N1c component. The temporal N1c and the nasopharyngeal positivity are similarly affected by interstimulus interval, intensity and frequency in the response to standard stimuli. In the Ignore condition, however, the N1c decreases ($p<.05$) whereas a non-significant reverse trend is found for the PgP120 component. The possible overlap of a processing negativity as discussed in the previous paragraph might explain this difference. The responses to target stimuli show some further dissociation between the N1c and PgP120 in the intensity, discrimination difficulty, and attention conditions. In these conditions the N1c increases with intensity, difficulty, and attention whereas the PgP120 is unchanged. These effects may also be due to an overlapping of the processing negativity at the scalp. It is therefore possible that both peaks (N1c and PgP120) are produced by recording from the opposite sides of a common dipole generator. The lack of asymmetry found for the PgP120 could be explained by the proximity of the right and left recording sites in the nasopharynx. A dipole generator on the lateral surface of the temporal lobe (Wolpaw and Penry 1975;

Wood and Wolpaw 1982) would be consistent with these results.

The Vertex P2 component

The P2 component increases significantly with increasing ISI. This component has previously been dissociated from the N1 on the basis of a longer recovery cycle (Roth et al. 1976). Although such a trend is indicated in our data, the difference in amplitude between the 3.3 s and the 5.0 s ISI conditions did not reach significance, presumably because of the small number of subjects available for this analysis. The scalp-distribution of the P2 component is more concentrated on the vertex than that of the N1b component (cf. Picton et al. 1974). A large amount of overlap with the N2 component in the response to target stimuli prevents an accurate evaluation of the frequency specificity of the P2 component. Such an overlap probably also explains the decrease in amplitude with increasing intensity, a result at variance with what has previously been reported (Picton et al. 1978b). As with the N1 peak, P2 does not show up in the nasopharyngeal waveforms. It is therefore also unlikely to be generated in a Sylvian or frontal dipole. Our results are seemingly at variance with those of Smith et al. (1973) who reported a reversal of the vertex P2 component at the nasopharynx. In their Figure 2, however, the peak latency

of the nasopharyngeal negativity is about 235 ms, some 40 ms later than their vertex P2 component. It seems therefore possible that the nasopharyngeal negativity reported by these authors corresponds to an endogenous component such as the P3a of Squires et al. (1975). The asymmetry found at the temporal leads in the basic auditory paradigm seems best explained in terms of overlap with the preceding N1b component.

Visual responses

Three distinct components were identified in the recordings following visual stimuli. These results are similar to those of Smith et al. (1973) although our latencies are longer, probably because our stimuli were less intense. Owing to the lack of experimental manipulations in the visual modality, our inferences concerning the relationships between scalp and nasopharyngeal recordings are based on latency only and must be considered cautiously. Our interpretation is further hampered by the absence of any occipital recordings. The study was mainly concerned with comparisons between the auditory and visual modalities rather than with the component-structure of the visual evoked potential.

The P145 component was seen at both scalp and nasopharynx (PgP145). Since no reversal is seen in the

nasopharyngeal waveform, we must assume that both scalp and nasopharyngeal electrodes are located on the same side of the hypothetical generator. Both an occipital and a subcortical generator (located below the level of the Pg electrodes) could generate a field producing such results.

The visual N1 component reached a scalp maximum at Pz, our most posterior recording site. Its latency corresponds fairly well with those reported previously by Simson and colleagues (Simson et al. 1976, 1977). A reversed peak of greater amplitude was identified at a similar latency in the nasopharyngeal recordings. Several cortical and subcortical generators might lead to similar recordings, most probably a dipole located in the parieto-occipital areas of cortex. The lack of a nasopharyngeal N1 component in the auditory as compared to the visual modality provides further evidence of the difference between visual and auditory N1s. The absence of a clear vertex N1 following visual stimuli in this study is different from the findings previously reported by Goff et al. (1969). Presumably, this is due to the different stimulus parameters used in each experiment. In a more recent paper, Goff et al. (1979) found the visual vertex N1 to appear only under certain conditions.

The P2 component of the visual evoked response has much in common with its auditory counterpart, although it has a longer latency. Simson and colleagues reported latencies

for this component of 235 ms (1976) and 245 ms (1977). This agrees well with our peak latency of 235 ms. The scalp-distribution of the visual and auditory P2s are similar, with slight differences that could be attributed to a larger overlapping vertex N1 in the auditory modality. Furthermore, neither the visual nor the auditory P2 are seen at the nasopharynx. Other studies, however, have reported quite distinct scalp topographies for the visual and auditory P2 components (Simson et al, 1977; Peronnet et al, 1975).

SUMMARY

Mesogenous event-related potentials were recorded from the scalp and nasopharynx during a signal-detection task. Evoked responses were evaluated with respect to the effects of inter-stimulus interval, intensity, frequency, attention and modality. Our results indicate the existence of at least 4 distinct processes occurring in the 75-150 ms latency range following auditory stimuli. The first process is indexed in the vertex-N1b/temporal-N1a component. This component does not reverse in polarity below the Sylvian fissure and is not seen in nasopharyngeal recordings. The location of its generator in not known. Probably there are two or more sources active at this latency. The second process finds reflection in the N1c/PgP120 component. This component is recorded with maximum amplitude on the side contralateral to the ear of delivery. A source in the lateral surface of the temporal lobe is a likely generator. The third process corresponds to Wolpaw and Penry (1975)'s Ta positivity. How much of this represents the underside of a vertically oriented dipole and how much a surface positivity is unknown. Finally, during attention, a lateralized processing negativity seems to overlap these components at the scalp, but not at the nasopharynx. The

auditory vertex N1 component is quite distinct from the visual N1 which is more posteriorly recorded on the scalp and which is associated with a definite nasopharyngeal positive wave. The P2 component is quite similar across auditory and visual modalities. In both modalities it is maximally recorded from the vertex and has no nasopharyngeal concomitant.

RESUME

Potentiels évoqués enregistrés à partir du scalp et du nasopharynx chez l'homme. I. Composantes mésogènes.

Les potentiels évoqués mésogènes ont été enregistrés à partir du scalp et du nasopharynx lors d'une tâche de détection de signaux. Les réponses obtenues furent évaluées quant aux effets de l'intervalle inter-stimulus, de l'intensité des stimuli, de leur fréquence, de l'attention des sujets et de la modalité sensorielle utilisée. Nos résultats indiquent l'existence d'au moins quatre processus distincts durant la période de 75 à 150 ms suivant les stimuli auditifs. Le premier processus se réflète dans les pics N1b du vertex et N1a de la région temporale. Cette composante ne change pas de polarité sous le niveau de la fissure Sylvienne et n'apparaît pas dans les traces du nasopharynx. La source génératrice de cette composante est inconnue. Il est probable que deux ou plusieurs sources soient actives à cette latence. Le deuxième processus se réflète dans le N1c temporal et dans le PgP120 du nasopharynx. Cette composante est enregistrée avec une amplitude maximale sur le coté contralatéral à l'oreille stimulée et pourrait provenir d'une source située dans la

face latérale du lobe temporal. Le troisième processus correspond à l'onde positive Ta de Wolpaw et Penry (1975). On ne sait pas si cette composante représente le dessous d'un dipôle temporal vertical ou une positivité de surface. Finalement, pendant l'attention, une onde négative (processing negativity) latéralisée semble se superposer aux précédentes composantes sur le scalp, mais pas dans le nasopharynx. La composante auditive N1 du vertex se distingue facilement du N1 visuel que l'on enregistre plus postérieurement et qui s'associe à une positivité nasopharyngéale bien définie. La composante P2 est semblable dans les modalités auditive et visuelle, atteignant une amplitude maximale au vertex sans qu'il y ait de concomitant nasopharyngéal.

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FIGURE LEGENDS

Figure 1. Superimposed individual waveforms to standard stimuli from 20 subjects run in the basic auditory paradigm. (90% standards - 90 dB peak SPL - 1.1 s ISI - right ear). Each tracing is the average of between 508 and 900 responses. All recordings were taken using a non-cephalic reference and negativity relative to this reference is indicated by an upward deflection of the tracing.

Figure 2. Results from one subject run in the control experiment. Superimposed waveforms (standard stimuli) are replications from 2 distinct sessions. The first column shows the appearance of the PgP120 component when using the TDH-49 headphones. The second column shows similar results when using a rubber tube in conjunction with a D-308 earphone, thus eliminating the possibility of inductive pickup. The third column shows results when the subject's ears were plugged, thus confirming the physiological nature of the PgP120 component.

Figure 3. Grand-mean waveforms to standard stimuli from 12 subjects run at ISIs of 1.1 s (solid line) and 3.3 s (dotted line). There is no component noted in the nasopharyngeal waveforms at the latency of the N1b (xxx ms), although there is a nasopharyngeal positive wave at 120 ms. The N1c is clearly larger at T3 than at T4, the stimuli being presented to the right ear.

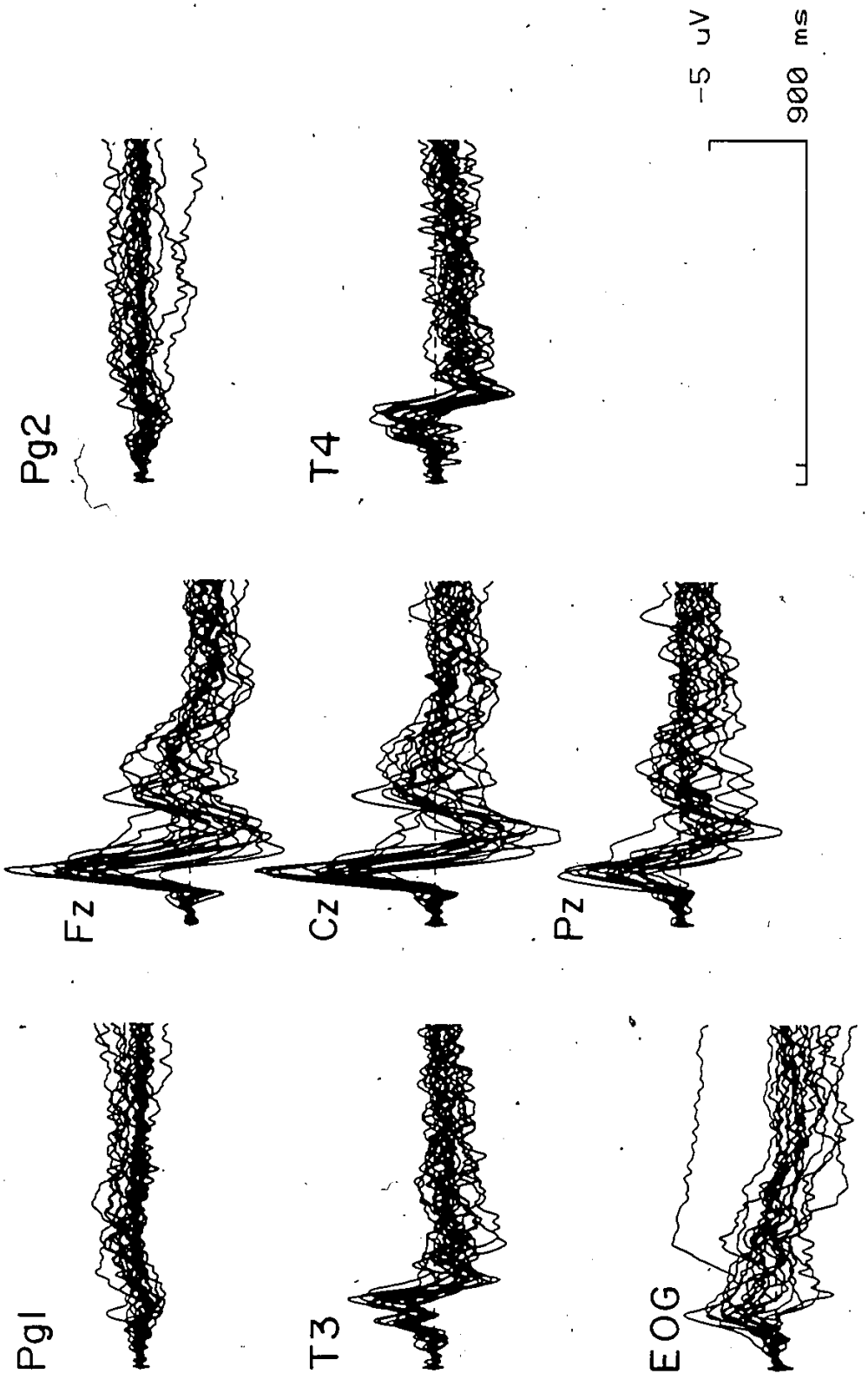
Figure 4. Grand-mean waveforms to standard stimuli from 10 subjects run at ISIs of 0.65 s (solid line) and 5.0 s (dotted line). Note that because of the short ISI there is an additional evoked potential toward the end of the waveform in the 0.65 s condition. The N1c is clearly larger at T3 than at T4, the stimuli being presented to the left ear in both these conditions.

Figure 5. Grand-mean waveforms to standard stimuli from 10 subjects run under Attend (solid line) and Ignore (dotted line) conditions. Stimuli were presented to the left ear at an ISI of 0.65 s, and thus show two responses over the plotted epoch (900 ms). Note the replication of the attention effect on the temporal N1c component at the T4 electrode.

Figure 6. Superimposed individual waveforms to visual stan-

dard stimuli from 10 subjects. Each trace is the average of between 651 and 1124 responses.

Figure 1



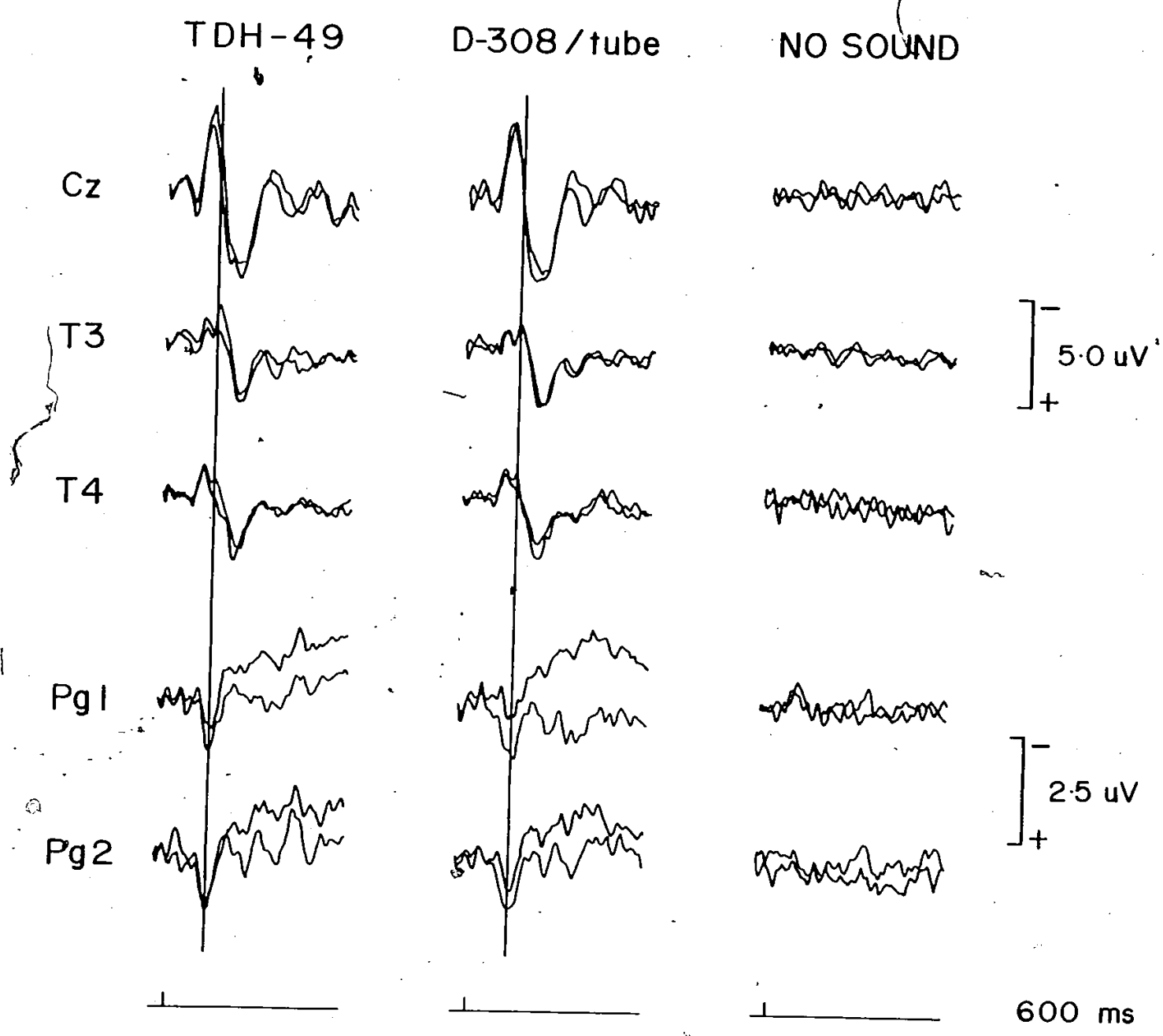


Figure 2

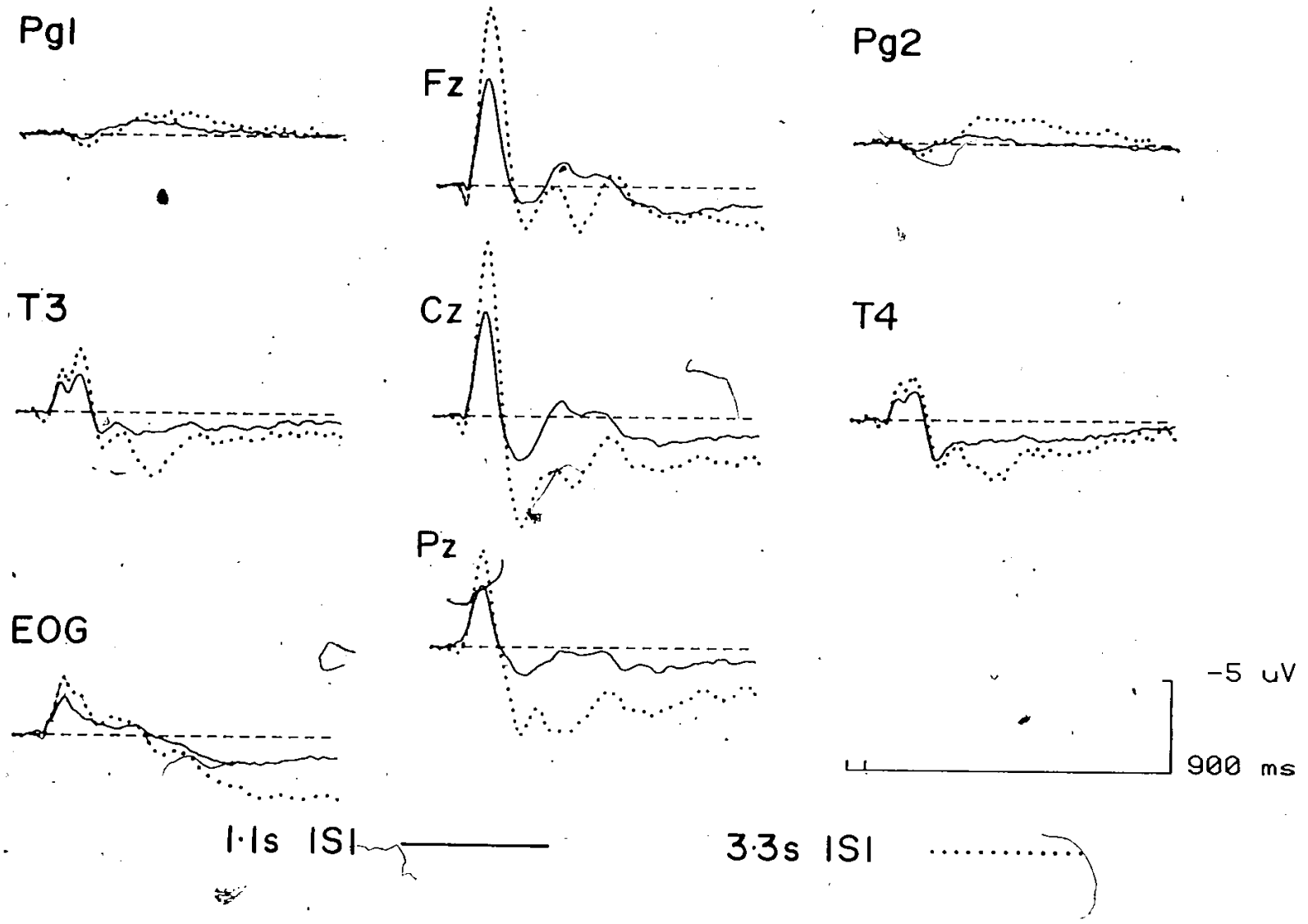
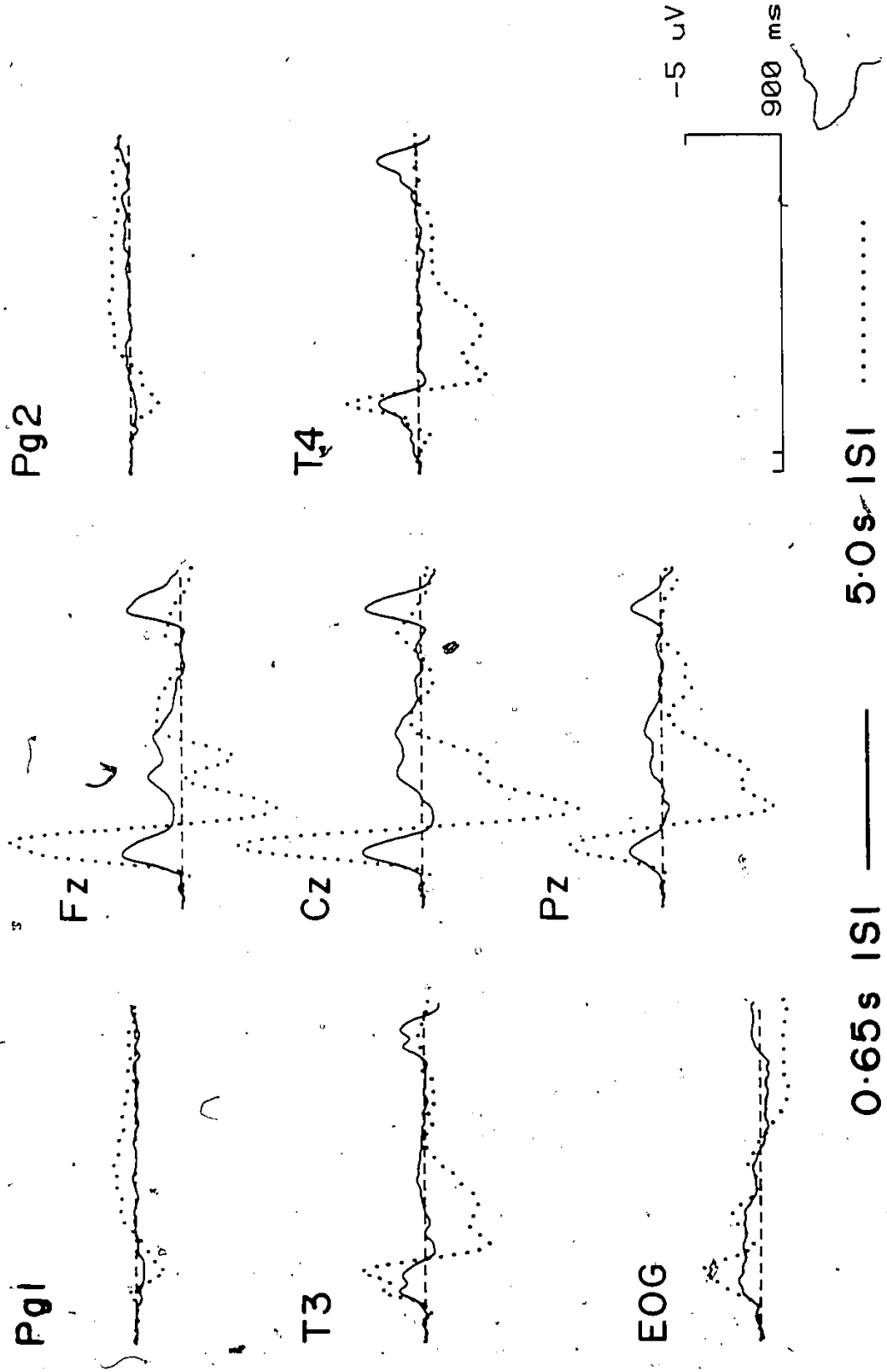


Figure 3

Figure 4



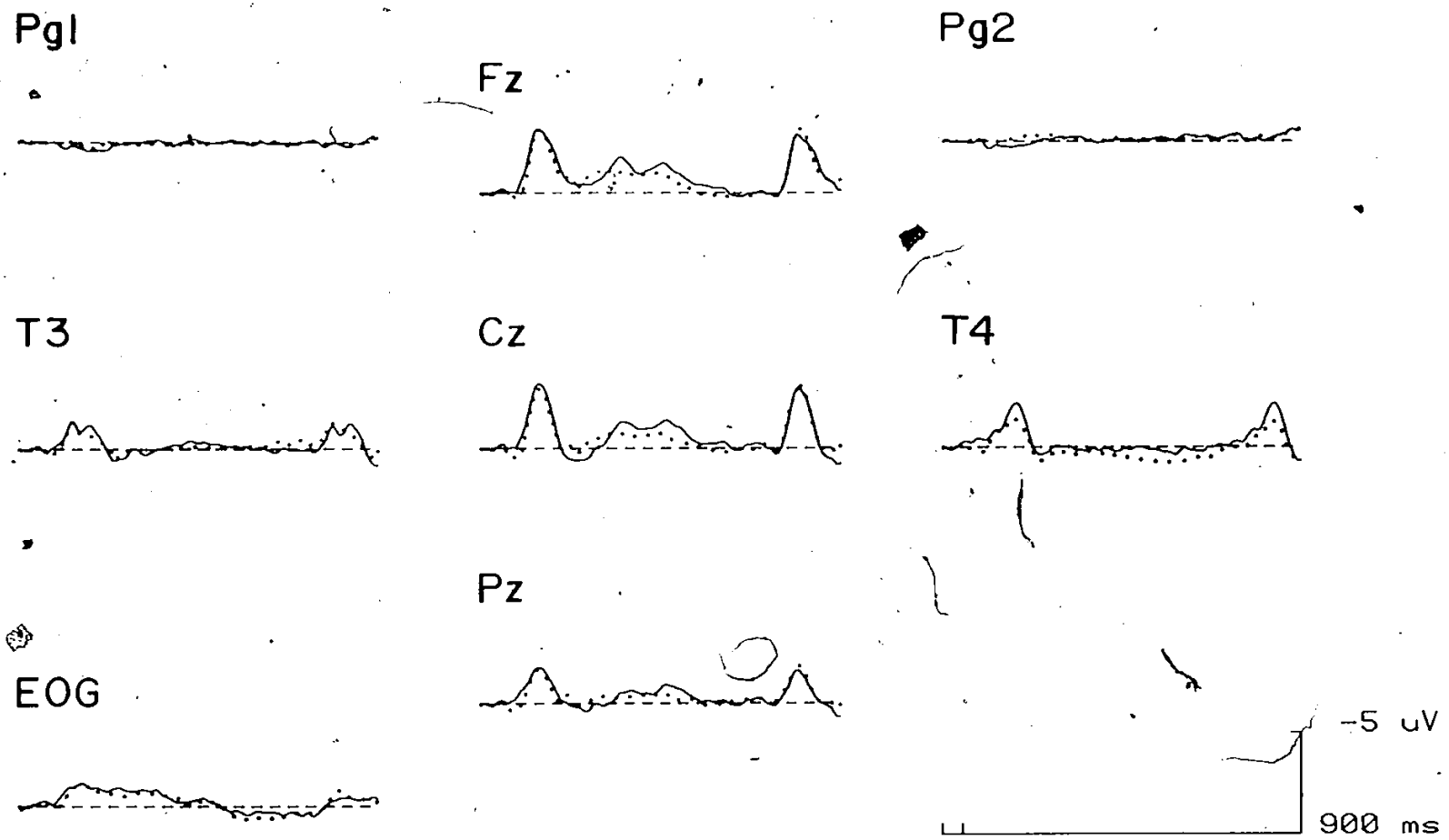
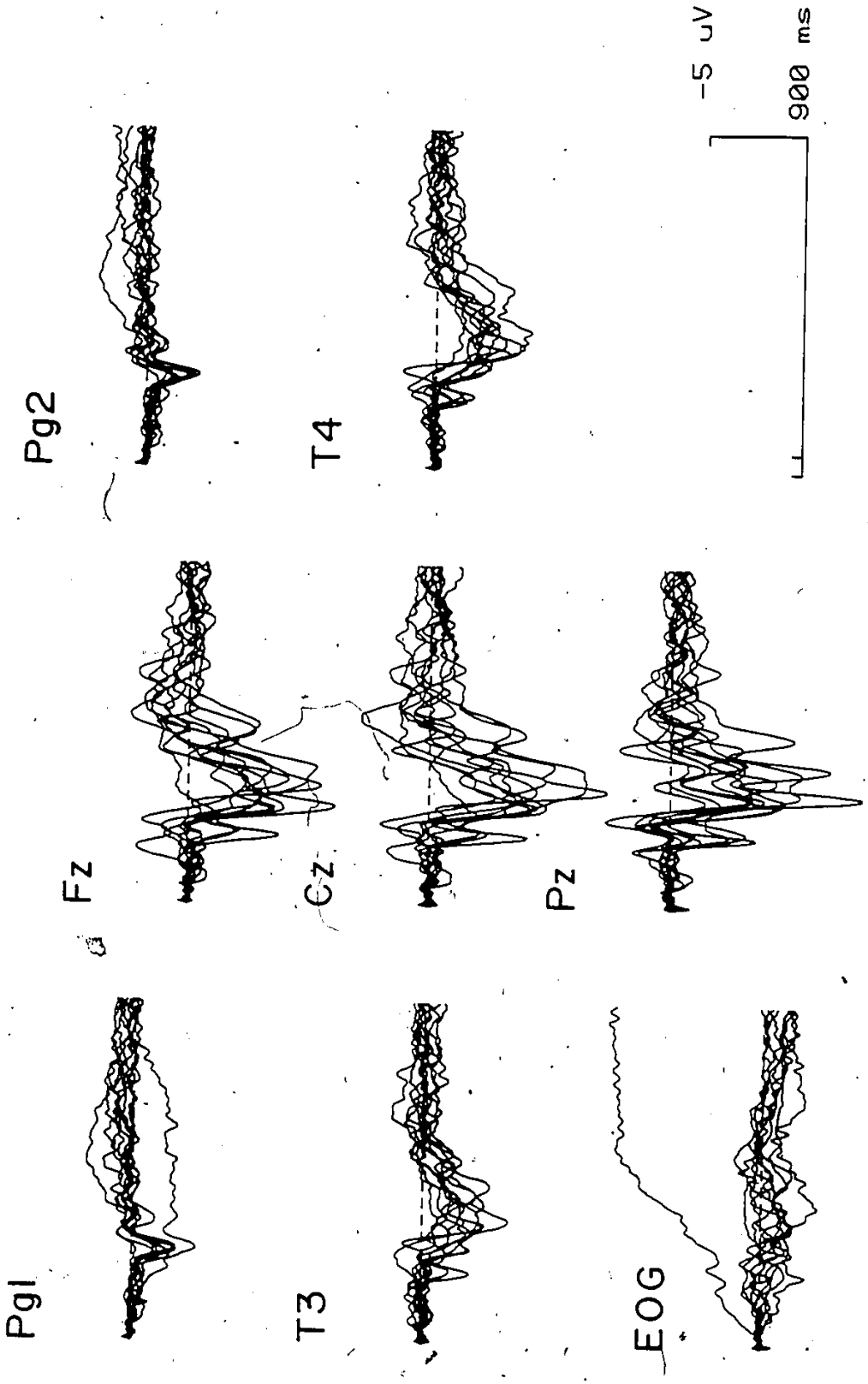


Figure 5

Figure 6



EVENT-RELATED POTENTIALS RECORDED FROM THE SCALP AND NASOPHARYNX. II. ENDOGENOUS COMPONENTS.

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The evoked potential to a detected improbable signal contains a series of late components, the most prominent of which are N2, P3 and the Slow Wave (Sutton et al. 1965; Ritter and Vaughan 1969; Roth 1973; Picton and Hillyard 1974; Squires et al. 1975; Rorhbaugh et al. 1978; Ritter et al. 1979). The N2 component appears to be modality-specific (Simson et al. 1977) and has been associated with sensory discrimination (Ritter et al. 1979) and mismatch detection (Näätänen 1981). The P3 component varies in amplitude as a function of stimulus probability (Duncan-Johnson and Donchin 1977; Campbell et al. 1979). Picton and Stuss (1980) distinguished between sequential probability (probability of a particular stimulus occurring within a number of stimuli) and temporal probability (the probability of a particular stimulus occurring within a period of time). Fitzgerald and Picton (1981) have proposed that the amplitude of the P3 component might be more related to the temporal than the sequential probability of the eliciting targets. Increasing the difficulty of detecting the targets increases the latency of both the N2 and P3 components of the evoked response and reduces the amplitude of the P3 (Ford et al. 1976; Fitzgerald and Picton 1983). The slow-wave (Squires et al. 1975; Rorhbaugh et al. 1978, 1979) is usually negative over the frontal scalp and becomes positive

over parietal regions. Like P3 it is recorded only after detected targets and it varies in amplitude with the probability of the target. The N2, P3 and Slow Wave can also be recorded to the absence of a stimulus, if this absence is important to the task (Klinke et al. 1968; Weinberg et al. 1970; Picton and Hillyard 1974; Simson et al. 1976; Renault and Lesevre 1978, 1979). These components are more related to the meaning attributed to a stimulus than to the physical characteristics of the stimulus, and have therefore been considered "endogenous" (Sutton 1969; Donchin et al. 1978).

The cerebral origins of the endogenous event-related potentials that are recorded from the scalp are not known. Human intracerebral recordings (Halgren et al. 1980; Wood et al. 1980, 1983) have indicated that deep temporal lobe structures are quite active during the scalp-recorded endogenous potentials. These limbic regions have been traditionally associated with memory and emotion. Several current hypotheses about the nature of the P3 component also involve such mnemonic and affective functions as the updating of context (Donchin 1979), or incentive (Hömberg et al. 1980). Furthermore, the hippocampus has been associated with dishabituation and memory-mismatch processes (Vinogradova 1970), as has the N2 component of the evoked potential (Näätänen 1981).

Until recently, the endogenous event-related potentials

in normal subjects have only been evaluated with scalp electrodes. Recordings from the nasopharynx can provide a different viewpoint and might therefore increase our understanding of both the component-structure of the endogenous event-related potentials and the role of limbic structures in human cognition. In preliminary studies (Perrault et al. 1983) we recorded nasopharyngeal components at similar latencies but with opposite polarity to the scalp-recorded N2 and P3 waves. We could not, however, determine whether the scalp and nasopharyngeal waveforms reflected the same or different components. This paper describes further experimental studies of the relations between scalp and nasopharyngeal endogenous components. Donchin (1978, p.353) has proposed a definition whereby "a component is a set of potential changes that can be shown to be functionally related to an experimental variable or to a combination of experimental variables." Our general approach was therefore to change the scalp-recorded components using accepted experimental manipulations, and to determine if the simultaneously occurring nasopharyngeal components were similarly or differently affected by these manipulations.

METHODS

The methodology of most of the experiments has already been described in the preceding paper (Perrault and Picton,

1983). Twenty-three subjects participated in a basic paradigm where tones of 55 ms duration, with an intensity of 90 dB peak SPL were presented to the subjects right ear at a rate of 1/1.1 s. The results from two of the subjects were rejected because of artifacts. Ten percent of the stimuli were 1000 Hz "targets" and the remaining 90% were 2000 Hz "standards". The subjects were asked to keep a mental count of the number of targets. Several variants of this basic paradigm were used to investigate different aspects of event-related potentials. A minimum or ten subjects were used for any experiment.

The effects of temporal and sequential probability were studied in three experiments. The first experiment investigated the well known effect of probability on the amplitude of the endogenous components. Targets were presented at a probability of either 10% or 30% in a train of stimuli occurring every 1.1 s. In this experiment, both the temporal and the sequential probability of the targets were changed. In the second experiment, the temporal probability of the targets was kept constant while the sequential probability was varied. Targets were presented at probabilities of 10% and 30% in trains where stimuli occurred every 1.1 s and 3.3 s respectively. In both manipulations, the target stimuli occurred every 10 s, on average. In a third experiment, the sequential probability of the targets was

kept constant, while their temporal probability varied. Targets were presented at a sequential probability of 10%, in trains where the stimuli occurred either every 0.65 s or every 5.0 s. The temporal probability of the targets was 1/6.5 s in the first instance, and 1/50 s in the second case.

There were two major variants of the basic paradigm. The effects of intensity were studied by comparing the responses to stimuli presented at either 90dB peak SPL or 65 dB peak SPL. The effects of discrimination difficulty were investigated by comparing the responses to targets of 1000 Hz occurring among standard stimuli of either 2000 Hz or 1050 Hz.

Missing stimulus potentials were studied in two separate omission paradigms. In the first one, missing targets were presented at a sequential probability of 10% in a train of 2000 Hz standards presented to the right ear at the rate of 1/1.1 s. In the second paradigm, missing targets occurred at a sequential probability of 10% in a train of standards presented to the left ear at the rate of 1/0.65 s. By shortening the ISI, we hoped to achieve better time-locking of the missing stimulus potentials.

The effects of attention were assessed by comparing responses obtained in a condition where the subjects were actively engaged in a signal-detection task to those obta-

ined when the subjects were reading a book or magazine and ignoring the incoming stimuli. In this manipulation target stimuli (2000 Hz) were presented 10% of the time in a train of standard stimuli (1000 Hz) coming every 0.65 s. All stimuli had an intensity of 90 dB peak SPL and were presented to the left ear. Finally, the effects of stimulus modality were investigated by comparing the responses to visual stimuli to those obtained in the basic auditory paradigm.

Amplitude and latency measurements

Only the late components of the event-related potentials will be considered in this paper. The N2, P3 and Slow Wave components were identified on the average waveforms for each individual subject. The N2 peak was identified separately at Cz, T3, T4, Pg1 and Pg2 electrodes as the maximum scalp negativity or nasopharyngeal positivity between 150 and 300 ms. The P3 component was identified at Cz, Pz, Pg1 and Pg2 electrodes as the maximum scalp positivity or nasopharyngeal negativity between 250 and 500 ms. The SW component was identified at the latency of the peak negativity following the P3 at Fz. Amplitudes were measured at each of the identified latencies with respect to the average amplitude of the waveform in the 50 ms preceding stimulus onset. The data were analyzed using repeated

measures analyses of variance and confidence intervals, as described in the preceding paper (Perrault and Picton, 1983). For descriptive purposes, the nasopharyngeal components have been labelled according to their scalp equivalents. Thus, the nasopharyngeal positivity at about 200 ms is called "N2" despite its polarity, and the negative nasopharyngeal wave occurring at the same latency as the scalp P3 component is called "P3". This seems preferable to introducing a PgP200 and PgN400 nomenclature, since these components can vary in latency, especially with discrimination difficulty.

RESULTS

Basic Auditory Paradigm

The individual waveforms of the responses to the targets from 20 of the 21 subjects evaluated in the basic paradigm are shown in Figure 1. (The waveforms of one of the subjects were unavailable because of a disk malfunction). The N2 peak had an average latency of 205 ms at Cz. It was maximally recorded in the frontocentral regions with an average amplitude of -6.7 uV at Fz, -6.0 uV at Cz, and -3.1 uV at Pz. At the temporal leads the N2 component

had an average amplitude of -2.7 uV at T3 and -2.4 uV at T4, with no significant asymmetry noted. A simultaneous positivity (212 ms) was recorded at the nasopharyngeal electrodes with an average amplitude of 2.6 uV. This represents a 43 % reversal from Cz. No significant asymmetry was noted between nasopharyngeal leads, where the amplitude of the N2 component was 2.4 uV at Pg1 and 2.8 uV at Pg2. The P3 wave was maximally recorded from the parietocentral region with an average amplitude of 10.0 uV at Fz, 14.0 uV at Cz and 14.2 uV at Pz. Its average latency was 318 ms at Cz and 348 ms at Pz, not a significant difference. A corresponding negativity was observed at the nasopharynx at a latency of 366 ms with an average amplitude of -3.3 uV. Again, no lateral asymmetry was noted (7.1 uV at T3; 6.8 uV at T4; -3.4 uV at Pg1; -3.2 uV at Pg2). The nasopharyngeal latency for P3 was significantly longer than the vertex latency, but was not significantly different from the Pz latency. The SW component was recorded as a negative wave at all electrodes except for Pz. It was maximally recorded at Fz with an average amplitude of -7.0 uV and an average peak latency of 539 ms. At this latency, the amplitude at Pz was +0.5 uV. The SW component at the nasopharyngeal electrodes was small (-0.9 uV) and did not differ significantly from zero.

----- Insert Figure 1 about here -----

Stimulus Probability.

There were significant changes in the amplitude of the late components when the probability of targets was increased from 10% to 30% (ISI of 1.1 s under both conditions). The grand-mean waveforms are shown in Figure 2. The average amplitude of the N2 component at Cz decreased significantly from -4.3 to -1.2 uV with increasing target probability. The simultaneous positivity recorded at the nasopharyngeal electrodes also diminished significantly from 2.2 to 1.3 uV although the relative change was not as large as that at the vertex. There were no significant differences in N2 latency between the different probabilities.

-----Insert Figure 2 about here-----

The average amplitude of the P3 peak decreased significantly from 13.2 to 8.3 uV at Cz, and from -3.1 to -1.7 uV at the nasopharynx. There were no significant probability-related changes in latency. The frontal slow wave showed a small decrease in amplitude with increasing target probability at the scalp (-5.5 vs -3.3 uV at Fz; $0.01<p<0.25$). No significant differences were found in the nasopharyngeal waveforms at this latency (-1.1 vs -0.7 uV;

average of Pg1 and Pg2).

In the second experiment, targets were presented at sequential probabilities of 10% and 30%, while the temporal probability was kept constant at 1/10 s by changing the ISI from 1.1 s to 3.3 s. Grand-mean waveforms for this experiment are presented in Figure 3. The N2 component decreased significantly from -5.3 to +0.3 uV at Cz. Similar trends were found at other scalp electrodes. The change at the nasopharynx was also significant, the amplitude of the positive wave decreasing from 2.7 uV at 10% to 0.5 uV at 30%. The latency of the N2 peak varied significantly from 201 (10%) to 247 (30%) ms at Cz. A similar trend was found at the nasopharynx (213 vs 234) but did not reach significance. The amplitude of the P3 component decreased slightly at Cz from 9.9 uV (10%) to 8.2 uV (30%), but this effect did not reach significance. A reverse trend was observed at the nasopharynx where the average (Pg1 and Pg2) amplitude increased from -2.4 to -3.5 uV. Again, however, this effect was not quite significant ($p.01<p<0.05$). The latency of the P3 peak was not significantly affected by sequential probability at either scalp or nasopharynx (337 vs 347 at Cz; 376 vs 383 at the nasopharynx). The amplitude of the frontal slow wave decreased slightly from -7.5 uV (10% - 1.1 s) to -5.9 uV (30% - 3.3 s), not a significant effect. Its latency was also unaffected (527 vs 525 ms). At this

latency the amplitude of the nasopharyngeal waveforms increased from -0.7 (10% - 1.1 s) to -2.6 uV (30% - 3.3 s). This change was not quite significant ($0.01<p<0.05$) although in the 30% - 3.3 s ISI condition the SW measurement at the nasopharynx was significantly different from baseline.

----- Insert Figures 3 and 4 about here -----

In the third experiment, targets were presented at a sequential probability of 10%, while the ISI was changed from 0.65 s to 5.0 s, for corresponding temporal probabilities of 1/6.5 s and 1/ 50 s. The grand-mean waveforms for this experiment are presented in Figure 4. The amplitude of the N2 component decreased significantly from -5.3 to -0.5 uV at the Fz lead with decreasing temporal probability. Similar trends were observed at other scalp electrodes. A reverse trend was found in the nasopharynx where the amplitude increased from 2.5 to 3.4 uV with decreasing temporal probability (not a significant effect). The latency of the N2 peak increased significantly from 194 to 265 ms at Fz with decreasing temporal probability. The nasopharyngeal N2 also occurred somewhat later (236 vs 251 ms) without this difference reaching significance. The amplitude of the P3 component increased from 9.4 to 11.1 uV at Fz, from 13.0 to 15.5 uV at Cz and from 10.9 to 16.0 uV

at Pz with decreasing temporal probability. None of these effects, however, reached significance. The amplitude of the P3 component at the nasopharynx was not affected by this manipulation (-2.1 vs -2.1 uV). The latency of the P3 component was significantly longer with smaller temporal probability at both Cz (338 vs 378 ms) and nasopharyngeal electrodes (345 vs 417 ms). No significant effects were found on the amplitude of the frontal slow wave at either scalp (-4.5 vs -5.5 uV at Fz) or nasopharynx (-0.3 vs 0.8 uV; average of Pg1 and Pg2). The amplitude of the nasopharyngeal waveforms at this latency did not differ significantly from baseline. The latency of the frontal slow wave increased significantly from 517 to 628 ms with decreasing temporal probability.

Endogenous components were sometimes seen in the waveforms to standard stimuli during the probability manipulations. In the first experiment (90% vs 70% standards; ISI of 1.1 s), the grand-mean waveform to the 70 % standard stimuli contained a small positive deflection at approximately the latency of the P3 component recorded in response to the target stimuli. Its amplitude was 0.8 uV at Fz, 1.1 uV at Cz and 0.9 uV at Pz (versus -0.5, -0.4 and 0.0 uV respectively in response to the 90 % standard stimuli). No significant activity was recorded from the nasopharynx at these latencies. There was no evidence of N2 or Slow Wave

components at any lead. In the second experiment, a distinct P3 component could be observed in the response to the standard stimuli during the long ISI condition (3.3 s), as can be seen in Figure 3 of the preceding paper (Perrault and Picton, 1983). The temporal probability of the standards in this condition was 1/4.7 s. The P3 latency was 345 ms at Cz. The amplitude of the grand-mean waveform at this latency reached a maximum of 4.5 uV at Pz (3.9 uV at Cz and 2.3 uV at Fz). This component could also be seen at the temporal leads and at the nasopharynx where its amplitude reached 3.3 and -1.2 uV respectively. The P3 component to standard stimuli can be seen even more clearly in Figure 4 of the first paper, where the ISI was 5.0 s (temporal probability of the standards was 1/5.56 s). It occurred at a Cz latency of 339 ms (versus 378 ms for the target stimuli) and reached an amplitude of 4.2 uV. A corresponding measurement at the nasopharynx yielded a value of -1.5 uV.

Stimulus Intensity

In the response to targets the amplitude of the scalp-N2 component increased significantly with increasing intensity from -3.4 uV to -5.7 uV at Cz. This change was significant at all scalp electrodes. There was no corresponding increase in the nasopharyngeal positivity which was on average 2.5 uV at the high intensity and 2.4 uV at

the low intensity. Changing the stimulus intensity had no significant effect on the P3 or SW components. The grand-mean waveforms are shown in Figure 5. The standard stimuli elicited no significant late response in this condition.

----- Insert Figure 5 about here -----

Discrimination Difficulty

The grand-mean waveforms for the target-evoked potentials in this experiment are shown in Figure 6. Increasing the difficulty of target discrimination increased the latency of both the N2 and P3 components of the response. The N2 component increased from 213 to 248 ms at the vertex. A similar change was found for the nasopharyngeal positivity (208 ms vs 251 ms). The P3 peak latency also increased in latency with difficulty at both scalp and nasopharyngeal sites (319 vs 387 ms at Cz; 358 vs 433 ms at the nasopharyngeal electrodes). The SW component increased in latency from 542 to 595 ms but this effect did not reach significance.

-----Insert Figure 6 about here-----

There were no significant effects of increasing the difficulty of target discrimination on the amplitude of the N2 component either at the scalp (-6.8 uV for easy vs -6.3 uV for difficult at Fz; -6.3 vs -5.9 uV at Cz; -2.4 vs -3.3 uV at Pz) or nasopharynx (2.1 vs 2.2 uV). It was possible that the increased discrimination difficulty lowered the actual detection of the signals. The absence of a change in N2 amplitude could therefore have resulted from the mixing of responses to detected and non-detected targets in the difficult condition. As a check on this possibility we reanalyzed our data using only those sessions where the counts were 100% accurate. Again there was no significant difference in N2 amplitude with increasing difficulty of target discrimination. The amplitude of the P3 component decreased significantly from 16.0 to 11.1 uV at Cz, and from -4.0 to -3.1 uV at the nasopharynx. The amplitude of the slow wave component showed no significant effect of discrimination difficulty. There were no significant effects of discrimination difficulty in the response to standard stimuli within the latency range of the late components.

Omission Paradigms

Definite endogenous potentials were recorded in re-

sponse to the stimulus omissions under both ISI conditions. The grand-mean waveforms are plotted in Figure 7 (1.1 s ISI) and Figure 8 (0.65 s ISI). The latency of the N2 peak in the response to the omitted stimuli (ISI of 1.1 s) was found to be significantly longer than that in the response to target stimuli at the nasopharynx but not at the scalp (208 vs 261 ms at the nasopharynx; 213 vs 227 ms at Cz). Similar trends were also found in the 0.65 s ISI experiment (226 vs 237 ms at the nasopharynx; 216 vs 210 ms at Cz), with the difference reaching significance at the scalp but not at the nasopharynx.

-----Insert Figures 7 and 8 about here-----

At the 1.1 s ISI, stimulus omissions elicited much smaller N2 components at the scalp electrodes than the target stimuli (-2.0 uV for omissions vs -5.0 uV for targets at Cz). There was no significant effect found at the nasopharyngeal leads where the amplitude of the positive peak was 1.9 uV for the omitted stimulus and 2.1 uV for the target stimulus. At the 0.65 s ISI, the N2 amplitude diminution at the scalp did not reach significance (-3.0 uV for omissions vs -4.9 uV for targets at Cz). Again, no amplitude change was found at the corresponding nasopharyngeal positivity (2.3 uV for targets vs 2.5 uV for

targets). In the 1.1 s ISI experiment, the amplitude of the P3 peak was reduced at all leads for the omitted stimulus (16.0 vs 6.2 uV at Cz; -4.0 vs -1.3 uV at the nasopharynx). Similar effects were found at 0.65 s ISI (13.0 vs 8.8 uV at Cz; -2.1 vs -1.1 uV at the nasopharynx; average of Pg1 and Pg2; in the nasopharynx the amplitude reduction was significant at Pg2 only where the amplitude was -2.3 uV for targets vs -1.0 uV for omissions). The frontal slow wave showed no significant change in amplitude in either experiment.

At the 1.1 s ISI, the P3 component for the omitted stimuli occurred significantly later than the P3 component to the target sitmuli at both scalp and nasopharyngeal sites (319 vs 381 ms at Cz; 351 vs 411 ms at Pz; 358 vs 493 ms at the nasopharynx). Results from 0.65 ISI experiment show similar trends (338 vs 367 ms at Cz; 345 vs 372 ms at Pz; 345 vs 406 ms at the nasopharynx), but the Cz difference did not reach significance. In the 1.1 s ISI manipulation, the slow wave component was also significantly later in response to stimulus omissions (543 vs 641 ms). Again, a similar trend was found in the 0.65 s ISI experiment (517 vs 565 ms), without this difference reaching significance.

Effects of Attention

Grand-mean waveforms for the Attend and Ignore condi-

tions are presented in Figure 9. The N2 component generally decreased in amplitude at the scalp during the Ignore condition, but these effects were significant only at the Fz (-5.3 vs -2.1 uV) and T4 electrodes (-2.5 vs -0.7 uV) electrodes. The N2 component does not show up very clearly in the grand-mean waveforms. This seems due to an increased inter-subject variability in the latency of this peak during the Ignore condition. The mean latencies were 216 ms in the Attend condition (standard deviation of 28 ms) and 237 ms in the Ignore condition (standard deviation of 52 ms). At the nasopharynx, the amplitude of the positive peak diminished from 2.5 to 1.6 uV across these conditions, but this effect did not reach significance. The latency of the nasopharyngeal N2 was 226 ms in the Attend condition and 217 ms in the Ignore condition. The P3 component was reduced significantly at the scalp (13.0 vs 2.2 uV at Cz). A corresponding change was found at the nasopharynx (-2.0 vs -0.9 uV), but this difference did not reach significance. The Slow Wave component also decreased in amplitude (-5.5 vs -2.3 uV at Fz) but this effect did not reach significance. No changes were found in the nasopharyngeal recordings at this latency.

-----Insert Figure 9 about here-----

Responses to Visual Stimuli

Superimposed waveforms following target stimuli can be seen in Figure 10, and the grand-mean waveforms for both standard and target stimuli are presented in Figure 11. The N2 component of the response to visual target stimuli was widely distributed. It reached its maximum scalp amplitude at Cz (-1.4 uV; versus -1.1 uV at Fz and Pz) where its peak latency was 283 ms. It occurred at a somewhat later latency at the temporal leads (298 ms), where its amplitude was -1.2 uV (average of T3 and T4). By comparison, the amplitude of the auditory N2 at the temporal leads was about one half of the Cz amplitude. At a latency of 280 ms a clear posivity could be seen in the nasopharyngeal recordings, with an amplitude of 3.2 uV. This represents a 230% reversal from the Cz amplitude (as opposed to the 33% reversal for the auditory N2 in the 10 subjects that participated in both auditory and visual manipulations).

-----Insert Figures 10 and 11 about here-----

The visual P3 component occurred at a latency of 412 ms at Cz, significantly later than its auditory counterpart. Its amplitude was also maximum parietally (17.8 uV). Corresponding amplitudes at Fz and Cz were 11.2 and 16.7 uV

respectively. At the temporal leads, it averaged 6.5 uV. At a somewhat later latency (436 ms; not a significant difference) a negative peak was observed in the nasopharyngeal waveforms with an amplitude of -4.3 uV (a percentage reversal from Cz of 25%, as compared to 25% for the auditory P3).

The visual slow wave was recorded with maximum amplitude from Fz (-3.4 uV) at a latency of 602 ms. Its amplitude was near baseline at Cz (-0.5 uV) and positive at Pz (3.4 uV). The average amplitude of the temporal recordings at this latency was -1.6 uV. At the nasopharynx, the waveforms did not differ significantly from zero (mean of -1.0 uV).

DISCUSSION

The results of these experiments replicate our previous findings (Perrault et al. 1983). At the latencies of the N2 and P3 components there were definite nasopharyngeal waves with opposite polarity to those recorded from the scalp. In the basic auditory paradigm the nasopharyngeal positive wave was on average 43% or the vertex N2 component (cf. 51% in the previous study) and the nasopharyngeal negativity 23% or the vertex P3 component (cf. 19% in the previous study). Again, we found no nasopharyngeal concomitant of the slow-wave. The following paragraphs will discuss the implications of our results for the component structure and cerebral origins of the scalp-recorded N2, P3 and Slow Wave components.

The N2 component

The results of the probability studies suggest that the N2 recorded from the scalp and the concomitant nasopharyngeal positivity are different. Both waves decreased in amplitude when the probability of the target was decreased from 10% to 30% and the ISI kept constant. In our second probability experiment, the nasopharyngeal positive wave and

the scalp N2 both decreased in amplitude when the interstimulus interval was increased without altering the temporal probability of the targets. At the scalp this might be due to an overlapping P2 component that increased in amplitude at the longer interstimulus intervals, particularly at the high intensities used in this experiment. Fitzgerald and Picton (1981) found no significant change in the scalp N2 component when they increased interstimulus interval while maintaining constant the temporal probability of the targets. Their results are based on target-standard subtraction waveforms, however, and the effects of ISI on P2 may have been cancelled out in the subtraction. Fitzgerald and Picton's raw waveforms (their Figure 2) show a reduced N2 amplitude at longer temporal probabilities. The attenuation of the nasopharyngeal positivity cannot be explained by any overlap with a P2 component, since the P2 is not recorded from this location. There thus seems to be a definite change in the nasopharyngeal N2 despite constant temporal separation between targets. This might indicate two distinct processes occurring at this latency. The results from our third ISI manipulation provide evidence for this interpretation. When the temporal probability is varied (from 1/6.5 s to 1/50 s) without altering the sequential probability of the targets (10%), the scalp N2 again decreases whereas the nasopharyngeal N2 is unchanged.

It is possible that there are two separate processes occurring in the N2 latency range, one recorded from the scalp and varying with temporal probability, and the other best recorded from the nasopharynx and affected primarily by sequential probability. Although the probability-relations for the nasopharyngeal N2 are fairly clear, the effects of the different probabilities of the scalp N2 may be related to concomitant effects on the overlapping P2 or P3 components. Thus, in the absence of subtraction waveforms, our results must be considered cautiously.

Increasing the difficulty of target discrimination caused a significant increase in the latency of both the scalp N2 and its nasopharyngeal counterpart without altering the amplitude of either. This lack of any significant change in N2 amplitude falls between the decrease in amplitude reported by Näätänen et al. (1980) and the increase reported by Fitzgerald and Picton (1983). Ford et al. (1976) also found no significant effect of target discriminability on N2 amplitude. This wide variety of results is probably due to different degrees of attentional engagement in the discrimination task (Fitzgerald 1982).

Further evidence for two different N2 processes come from the intensity manipulation, where the scalp N2 is significantly reduced at the lower intensity whereas the nasopharyngeal positivity remains the same. The effect of stimulus intensity on the scalp N2 cannot be explained by an

overlapping P2 component since the latter is known to decrease at low intensities (Keidel 1976; Picton et al. 1978). In the present experiment, the P2 component to standard stimuli showed no change across intensity conditions. The N2 effect cannot be explained by a differential superimposition of the following P3 wave, since this component remained unchanged by stimulus intensity. It is unlikely that an early portion of the negative slow wave might contribute to greater N2 amplitudes at high intensities since there was no significant difference in slow wave amplitude across intensity conditions. We are therefore left with the conclusion that there are two distinct components at the N2 latency. One component is affected by stimulus intensity and is best recorded from the scalp; the other is unchanged by the intensity manipulation and is best recorded from the nasopharynx.

The response to stimulus omissions when the ISI was 1.1 s shows a significant decrease of N2 amplitude at the scalp, whereas the nasopharyngeal N2 remains unchanged. The change in the scalp N2 could be related to the intensity relationship described in the preceding paragraph, or to some jitter in its occurrence. When the ISI was shortened to 0.65 s, neither N2 process was significantly different between the omitted stimuli and the targets, although a definite decrease in the scalp N2 may have been obscured by

waveform variability. If there was a latency effect, it did not affect the nasopharyngeal N2. These results indicate a greater dependence of the scalp N2 on either the timing or the intensity of the stimulus, and further support the existence of two distinct processes in this latency range.

Our ignore condition caused a significant decrease in N2 amplitude at Fz and T4, but not at other scalp or nasopharyngeal locations. These results are consistent with the existence of a processing negativity recorded primarily over the fronto-central and contralateral regions of the scalp (Näätänen in press). We reported a contralateral attention effect on the temporal Nlc component in the preceding paper (Perrault and Picton, 1983). Our findings might thus be caused by a fairly broad processing negativity overlapping or composing both the Nlc and N2 components at the scalp.

The N2 component shows evidence of modality-specificity. Its scalp amplitude is more evenly distributed in the visual condition than in the auditory condition where it has a clear fronto-central maximum. The difference in amplitude between the auditory and visual conditions is significant at the frontal and central locations, but not at the temporal sites or in the nasopharynx. The present data would be consistent with the further specification of a modality-specific scalp N2 and a non-modality-specific

nasopharyngeal N2. Lack of manipulations in the visual modality, however, do not permit further support of this hypothesis.

It seems possible to integrate the results previously discussed concerning the N2 by postulating the existence of two distinct processes in this latency range. A first process is picked up mostly at the scalp and is similar to Näätänen's processing negativity. The amplitude of this process is affected by attention, temporal probability, modality and by the physical characteristics of the eliciting event. As such, it might index a stimulus evaluation procedure carried out in the neo-cortical areas of the brain. The second process is picked up primarily in the nasopharynx as a positive wave. This process is less dependent on attention, is affected by the sequential probability of events, is not affected by the physical parameters of stimulation and might be independent of modality. This process might index a "pure" mismatch-detection procedure, whereby events are compared to a memory template created by previous events. Subcortical limbic regions are a plausible source of this component.

The P3 component

A large parieto-central P3 wave was recorded in the response to improbable targets. The P165 component (Goodin

et al. 1980) was not recognized in our waveforms, even following the Attend-Ignore subtractions. No clear P3a component (Squires et al. 1975) could be recognized, even in the response to ignored targets. The following discussion applies therefore only to the usual "P3b" component (Squires et al. 1975).

Our results concerning the scalp-P3 component generally agree with the previous litterature on the effects of intensity (Hillyard and Picton 1979), discrimination difficulty (Fitzgerald and Picton, 1983; Campbell et al. 1983) stimulus omissions (Picton and Hillyard 1974; Simson et al. 1976), attention (Hillyard et al. 1973; Picton and Hillyard, 1974) and modality (Simson et al. 1977). The first probability experiment shows an expected decrease of P3 amplitude with increasing probability. The second experiment, where the sequential probability of the targets was increased but the temporal probability was kept constant, showed no significant change in P3 amplitude. This result supports Fitzgerald and Picton (1983)'s hypothesis that temporal probability is more important than sequential probability in determining P3 amplitude. In the third experiment, however, the temporal probability of the targets was increased from 1/6.5 s to 1/50 s without producing a significant increase in P3 amplitude. There may be a saturation of P3 at very low temporal probabilities.

There is strong support in our data for the hypothesis that the scalp-P3 and the concurrent nasopharyngeal negativity reflect a single cerebral process. All of our experimental manipulations which led to significant changes in the scalp-P3 had similar effects on the nasopharyngeal negative wave. Both waves decrease in amplitude with increasing temporal probability, with increasing discrimination difficulty, with stimulus omissions and during the Ignore condition. Their latencies increase with discrimination difficulty and for stimulus omissions. Conversely, those manipulations which caused no change in the scalp-P3 did not affect the nasopharyngeal negative wave. Both waves remain unaffected by stimulus intensity, and by sequential probability when the temporal probability is kept constant. The scalp-distribution and reversal percentages from Cz to nasopharynx of the P3 component were not affected by stimulus modality. One is therefore tempted to conclude that there is a single P3 generator with an equivalent dipole located between the scalp and the nasopharynx. This would be compatible with the results of intracerebral recordings (Halgren et al.,1980; Wood et al. 1980, 1983).

There is, however, the problem of a significant latency difference between the vertex-P3 and the nasopharyngeal negative wave. This difference is about 35 ms following the target discriminations but is 91 ms following the stimulus

omission at an ISI of 1.1 s. Similar results were found at an ISI of 0.65 s. (29 ms and 61 ms respectively). One way of reconciling this latency difference with the concept of a single generator is to postulate an overlapping negative slow wave at the vertex. This negative slow wave could cut short the vertex P3, giving it an earlier peak latency. This would not occur at the nasopharynx where there is little if any slow wave component. Such a hypothesis would also explain why the P3 latency at Pz is longer than at Cz, since there is no negative slow wave at Pz in most conditions. The only condition in which there is a negative slow wave at Pz is following the stimulus omission, and in this condition the P3 latency at Pz is significantly shorter than the latency of the nasopharyngeal negative wave. This interpretation is supported by a recent principal component analysis of the event-related potential which suggests a large amount of overlap between P3 and slow wave components at the scalp (Ruchkin et al. 1980; McCallum and Curry 1981).

Our results therefore suggest that there is a single process underlying the P3 component. They do not indicate where in the brain this process is occurring, nor indeed that it is generated in one particular region alone. There is, however, a very distinct cerebral process occurring at this latency and generating a large and widespread dipole field.

The Slow Wave Component

The Slow Wave component covaries with P3 under all experimental conditions. Although the nasopharyngeal waveform at the latency of the frontal slow wave turned out to be significantly different from baseline in the 3.3 s ISI condition, this effect was not replicated in the 5.0 s ISI condition. The representation of the slow wave at the nasopharynx is minimal, thus indicating different generators for these two components.

SUMMARY

Scalp and nasopharyngeal recordings of the N2, P3 and Slow Wave components were compared in a target-detection task. The effects of probability, interstimulus interval, intensity, discrimination difficulty, attention, stimulus omission and modality were evaluated. Waves of opposite polarity to the scalp N2 and P3 components were recorded in the nasopharynx. The scalp and nasopharyngeal N2 components showed different patterns of variation across experimental conditions. These findings indicate that there are two different cerebral processes occurring at the latency of the scalp N2. The scalp and nasopharyngeal P3 components consistently covaried across conditions, suggesting a single underlying process. The Slow Wave was observed only in the scalp recordings.

RESUME

Les composantes N2, 'P3 et l'onde lente du potentiel évoqué ont été enregistrées à partir du scalp et du nasopharynx lors d'une tâche de détection de signaux. Les effets de la probabilité des signaux, de l'intervalle inter-stimulus, de l'intensité, de la difficulté de discrimination, de l'attention, de l'omission des signaux et de la modalité sensorielle furent évalués. Des ondes correspondant aux composantes N2 et P3 du scalp furent observées dans les enregistrements à partir du nasopharynx, mais ayant une polarité inverse. Les composantes N2 du scalp et du nasopharynx semblent s'associer à des processus distincts, puisqu'elles réagissent différemment à nos manipulations expérimentales. Les composantes P3 du scalp et du nasopharynx réagissent parallèlement aux diverses conditions, ce qui semble indiquer un processus sous-jacent unique. L'onde lente ne fut enregistrée que sur le scalp.

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FIGURE LEGENDS

Figure 1. Superimposed individual waveforms to target stimuli from 20 subjects that participated in the basic auditory paradigm. (10% targets - 90 dB peak SPL - 1.1 s ISI - right ear). Each tracing is the average of between 50 and 110 responses. All recordings were taken using a non-cephalic reference and negativity relative to this reference is indicated by an upward deflection of the tracing.

Figure 2. Grand-mean waveforms to target stimuli from 10 subjects in two target probability conditions (10% - solid line; 30% - dotted line). Targets of 1000 Hz were presented among standard stimuli of 2000 Hz. All stimuli had an intensity of 90 dB peak SPL and were presented to the right ear with an ISI of 1.1 s. Note the decrease in amplitude of the N2 and P3 components at the scalp in the 30% condition, and the covariation in their nasopharyngeal concomitants.

Figure 3. Grand-mean waveforms to target stimuli from 12

subjects in two sequential probability conditions (10% - 1:1 s ISI - solid line; 30% - 3.3 s ISI - dotted line) when the temporal probability of targets was kept constant at 1/10 s. Targets of 1000 Hz were presented among standard stimuli of 2000 Hz. All stimuli had an intensity of 90 dB peak SPL and were presented to the right ear. The P3 amplitude differences were not significant.

Figure 4. Grand-mean waveforms to target stimuli from 10 subjects in two temporal probability conditions (1/6.5 s - 0.65 s ISI - solid line; 1/50 s - 5.0 s ISI - dotted line) when the sequential probability of targets was kept constant at 10%. Targets of 1000 Hz were presented among standard stimuli of 2000 Hz. All stimuli had an intensity of 90 dB peak SPL and were presented to the left ear. The P3 differences in amplitude were not significant. Note that because of the short ISI, there is an additional evoked potential to the standard stimuli occurring toward the end of the tracing in the 0.65 s condition.

Figure 5. Grand-mean waveforms to target stimuli from 10 subjects in two intensity conditions (90 db peak SPL - solid line; 65 dB peak SPL - dotted line). Targets of 1000 Hz occurred 10% of the time among standard stimuli of 2000 Hz. Stimuli were delivered to the right ear with

an ISI of 1.1 s in both conditions. Note the clear reduction in N2 amplitude at the scalp and the absence of this effect in the nasopharyngeal concomitant.

Figure 6. Grand-mean waveforms to target stimuli for 10 subjects in two discrimination difficulty conditions. In the Easy condition (solid line) targets of 1000 Hz were presented among standard stimuli of 2000 Hz. In the Difficult condition (dotted line) targets of 1000 Hz were presented among standard stimuli of 1050 Hz. Targets occurred 10% of the time. All stimuli had an intensity of 90 dB peak SPL and were presented to the right ear with an ISI of 1.1 s. Note the increased latencies of the N2 and P3 components in both scalp and nasopharyngeal recordings.

Figure 7. Grand-mean waveforms to targets for 10 subjects in a condition where the target stimuli were 1000 Hz tones occurring among 2000 Hz standards (solid line) and in a condition where the target stimuli were omitted (dotted line). All presented stimuli had an intensity of 90 dB peak SPL and were delivered to the right ear with an ISI of 1.1 s.

Figure 8. Grand-mean waveforms to targets for 10 subjects

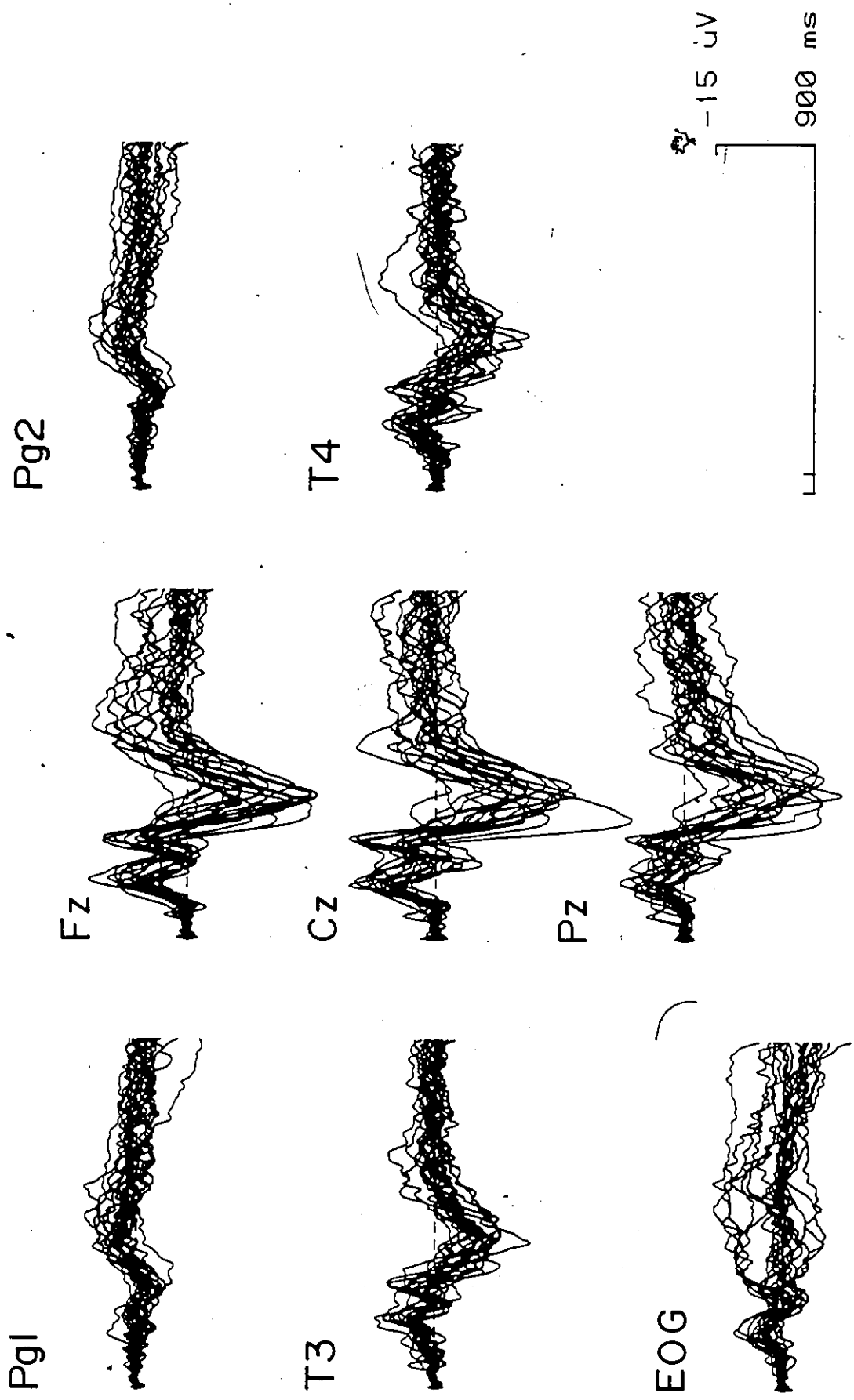
in a condition where the target stimuli were 1000 Hz tones occurring among 2000 Hz standards (solid line) and in a condition where the target stimuli were omitted (dotted line). All stimuli had an intensity of 90 dB peak SPL and were delivered to the left ear with an ISI of 0.65 s. Note that because of the short ISI, an additional evoked potential is present toward the end of the waveform.

Figure 9. Grand-mean waveforms to target stimuli for 10 subjects that participated in Attend (solid line) and Ignore (dotted line) conditions. During the Attend condition, the subjects had to keep a mental count of the number of targets. In the Ignore condition they concentrated on reading a book or magazine. Targets of 1000 Hz were presented among standard stimuli of 2000 Hz. All stimuli had an intensity of 90 dB peak SPL and were delivered to the left ear with an ISI of 0.65 s. Note that because of the short ISI, an additional evoked potential is present toward the end of the waveform.

Figure 10. Superimposed individual waveforms to target stimuli from 10 subjects in a condition where stimuli were presented in the visual modality. Targets occurred 10 % of the time with an ISI of 1.1 s and were dif-

ferentiated from the standards by color (red or green). The N2 and P3 components also reverse in the nasopharyngeal recordings. Each trace is the average of between 78 and 129 responses.

Figure 11. Grand-mean waveforms to targets (solid line) and standard stimuli (dotted line) from 10 subjects that participated in a condition where the stimuli were presented in the visual modality. Targets were presented 10% of the time. The ISI was 1.1 s. Reversed N2 and P3 components can be seen at the nasopharynx in response to target stimuli. In addition a positive N1 can be seen in the nasopharyngeal waveforms for both standard and target stimuli. As in the auditory modality, the P2 component (at approximately 235 ms) is not seen in the nasopharyngeal recordings.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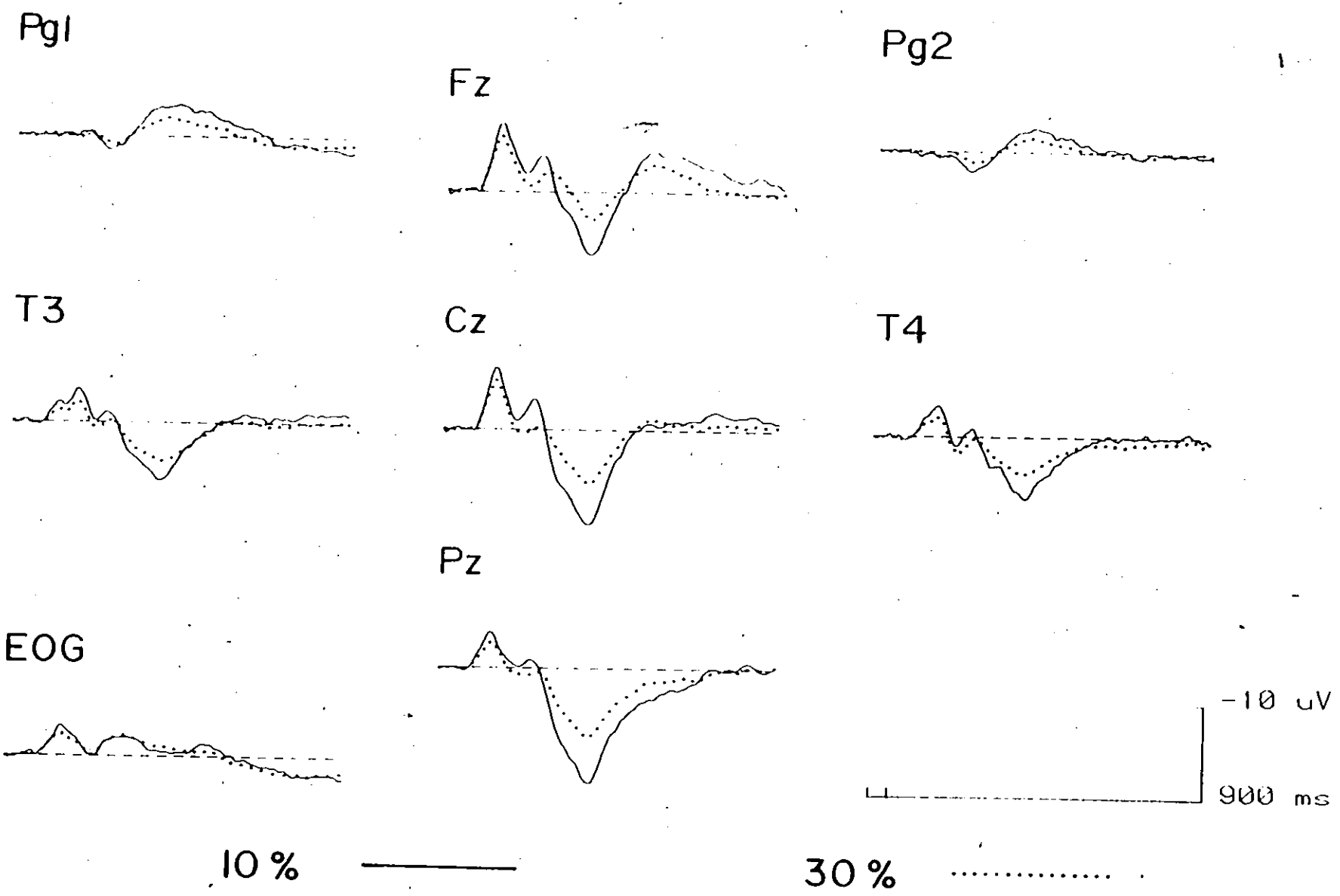
T4
-15 uV
900 ms
Fz
Cz
Pz
Pgl
T3
EOG



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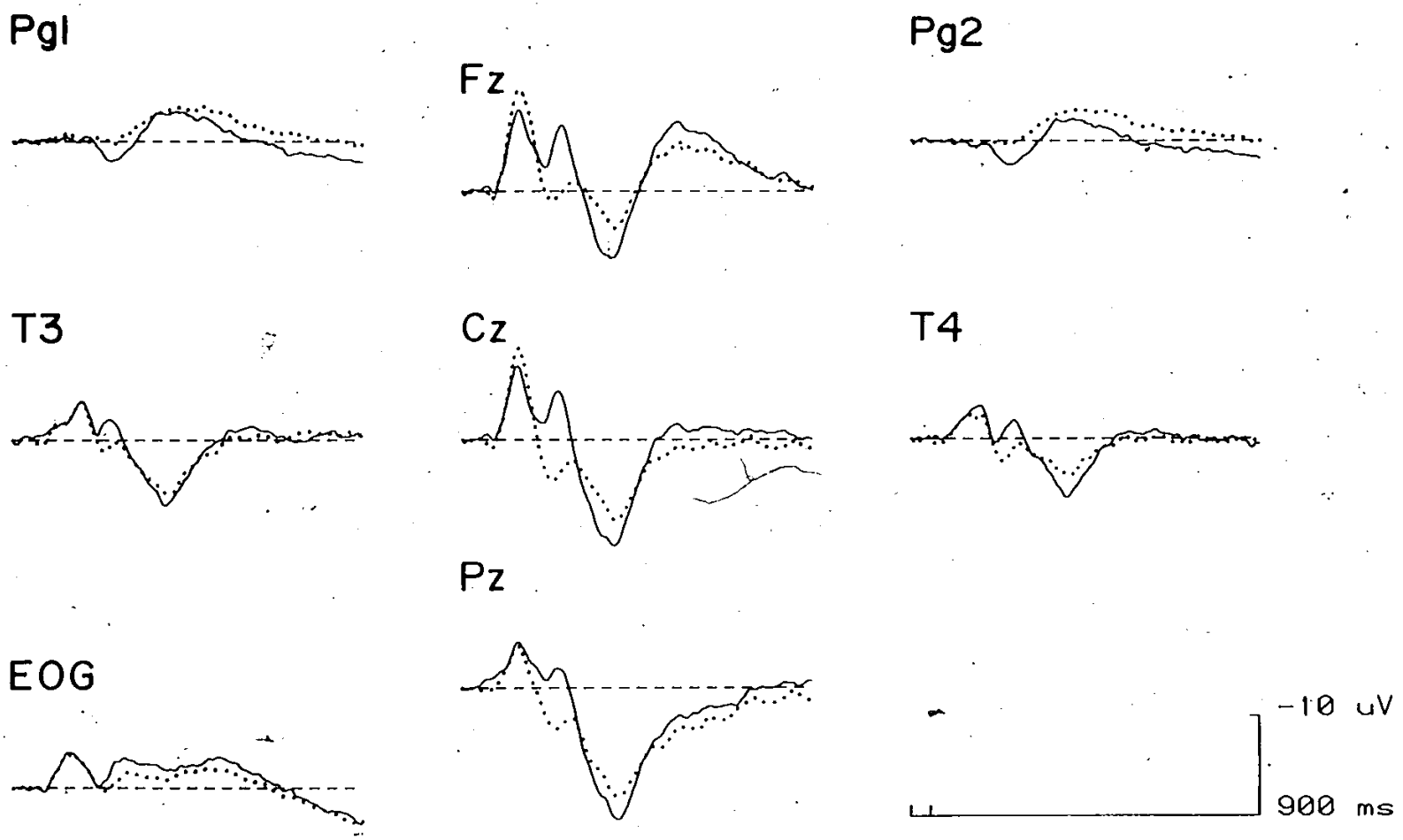

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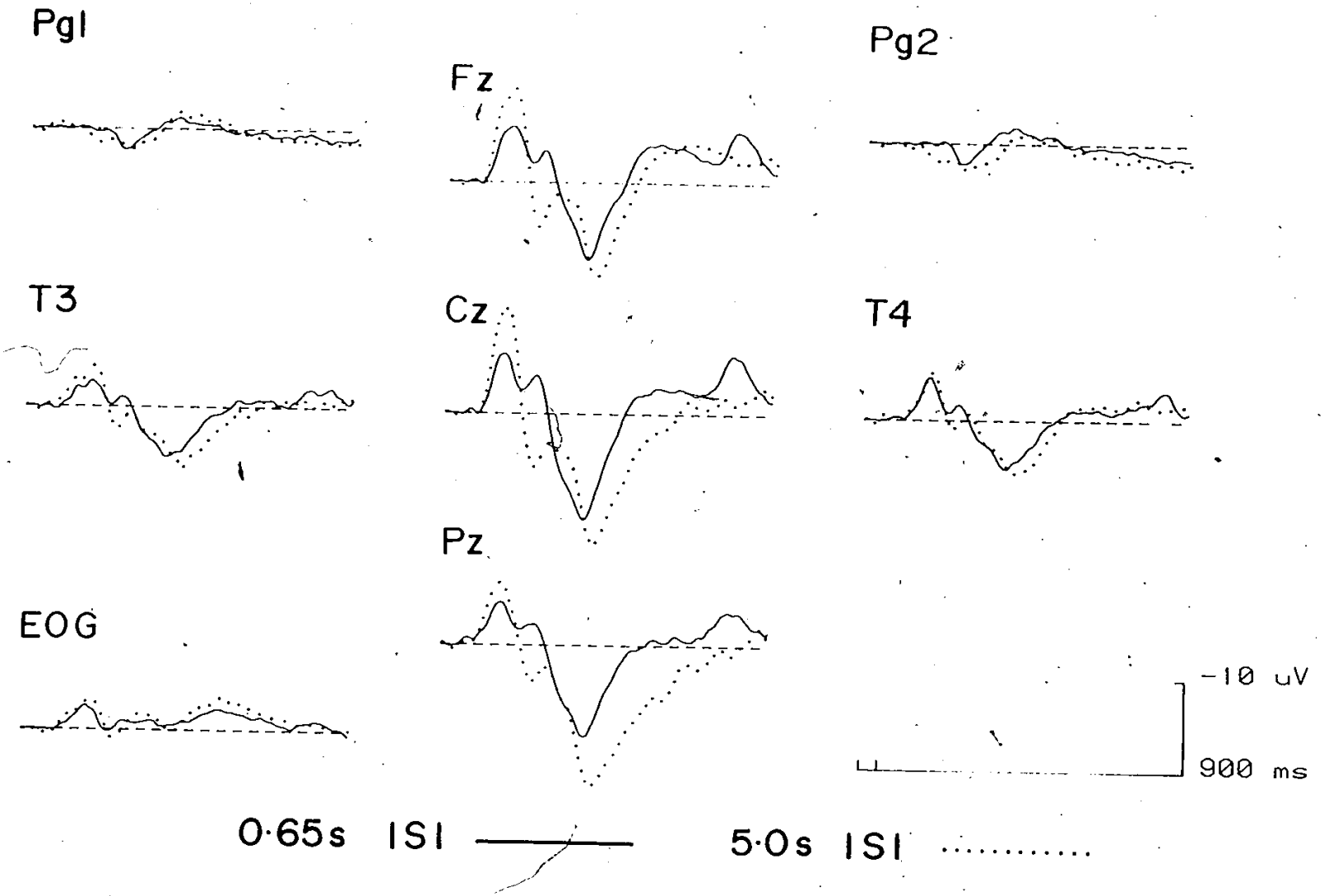
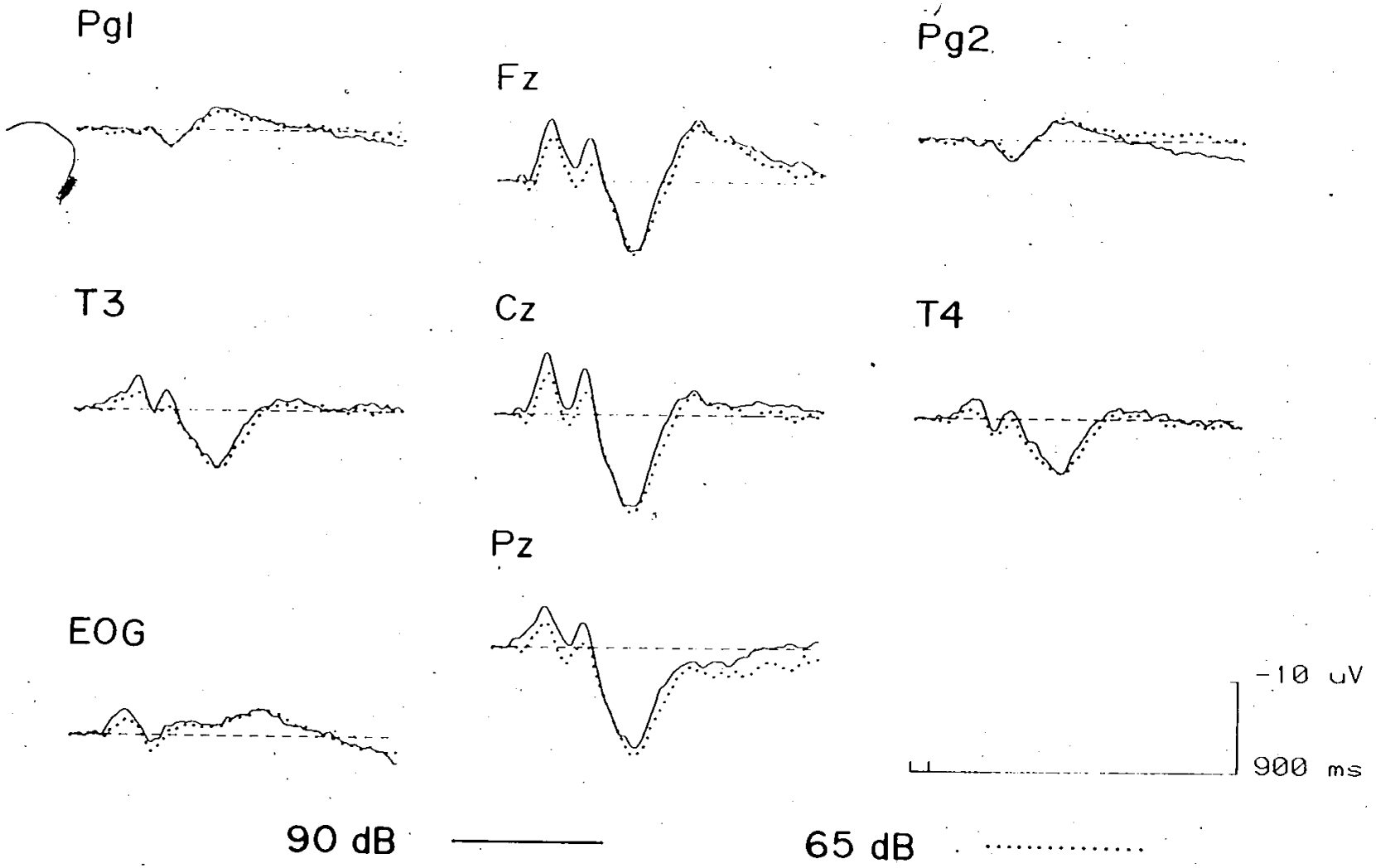


Figure 4



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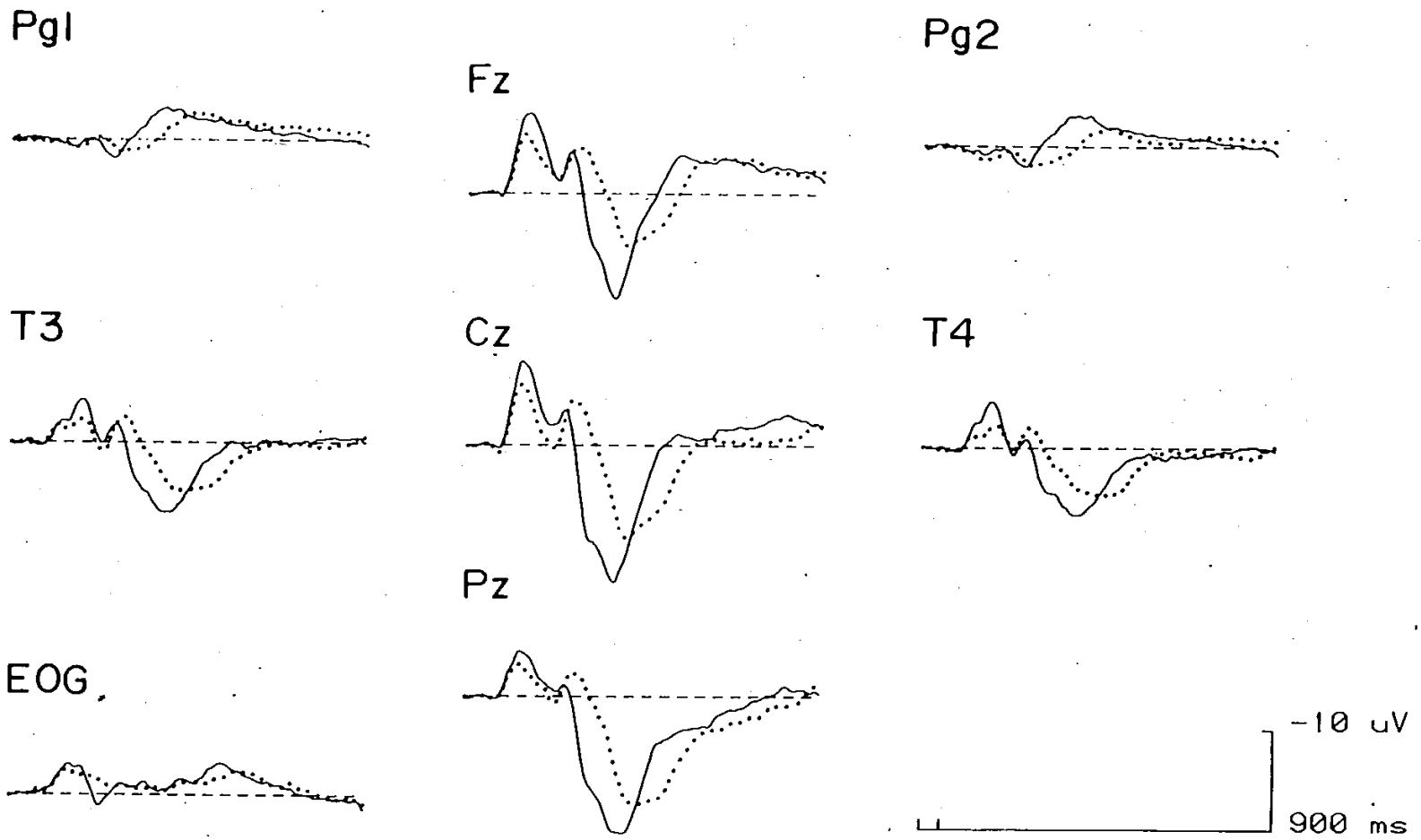

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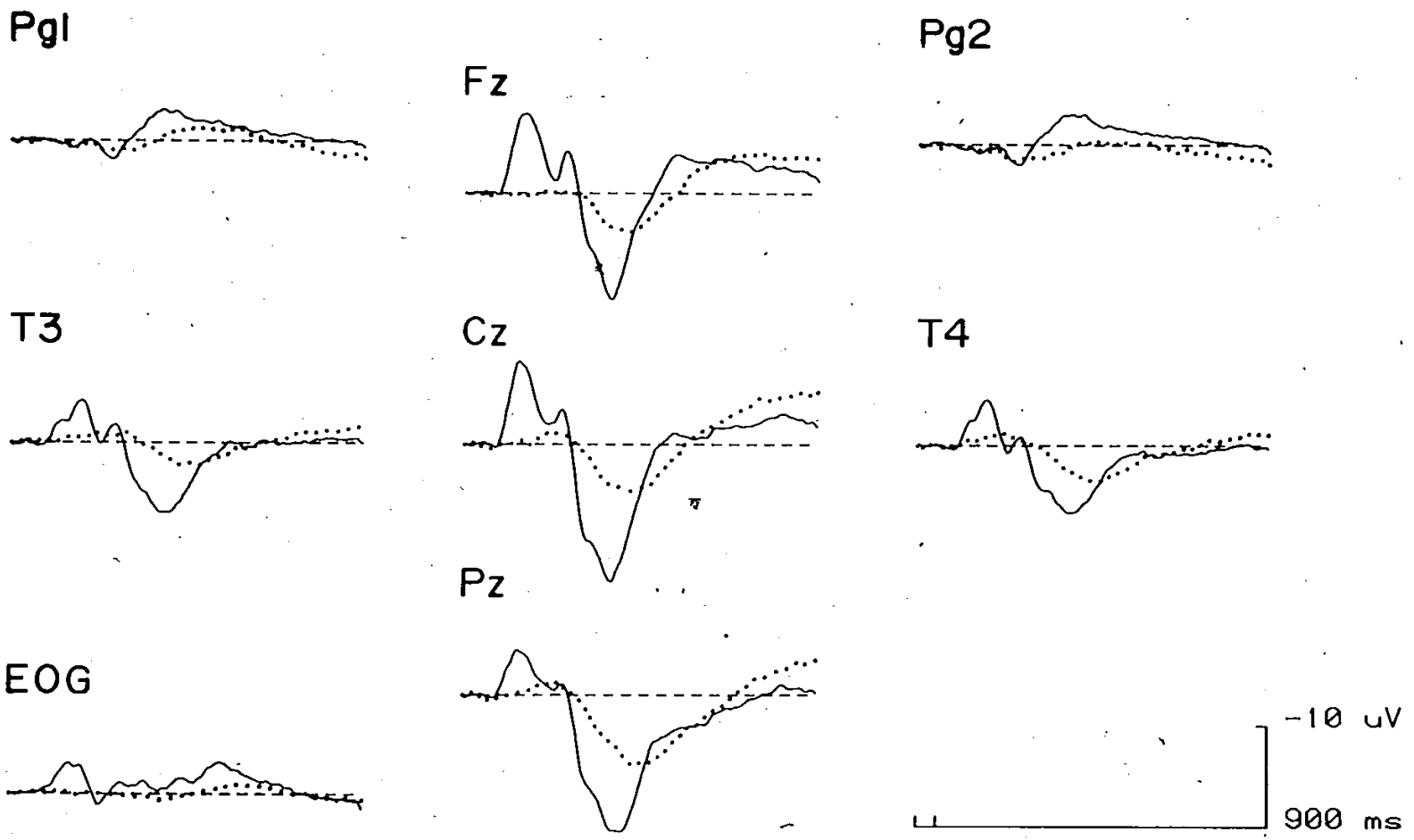

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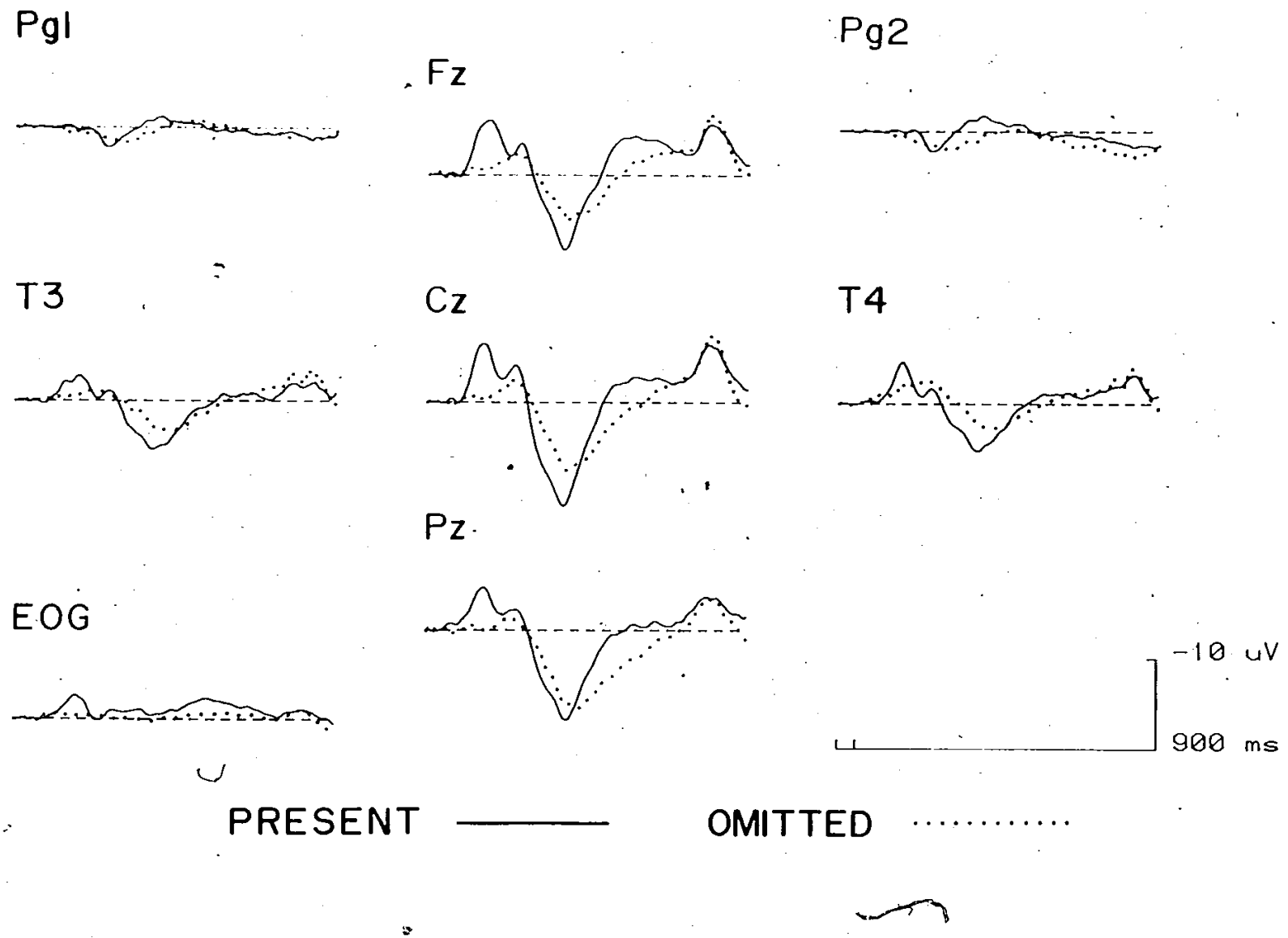


Figure 8

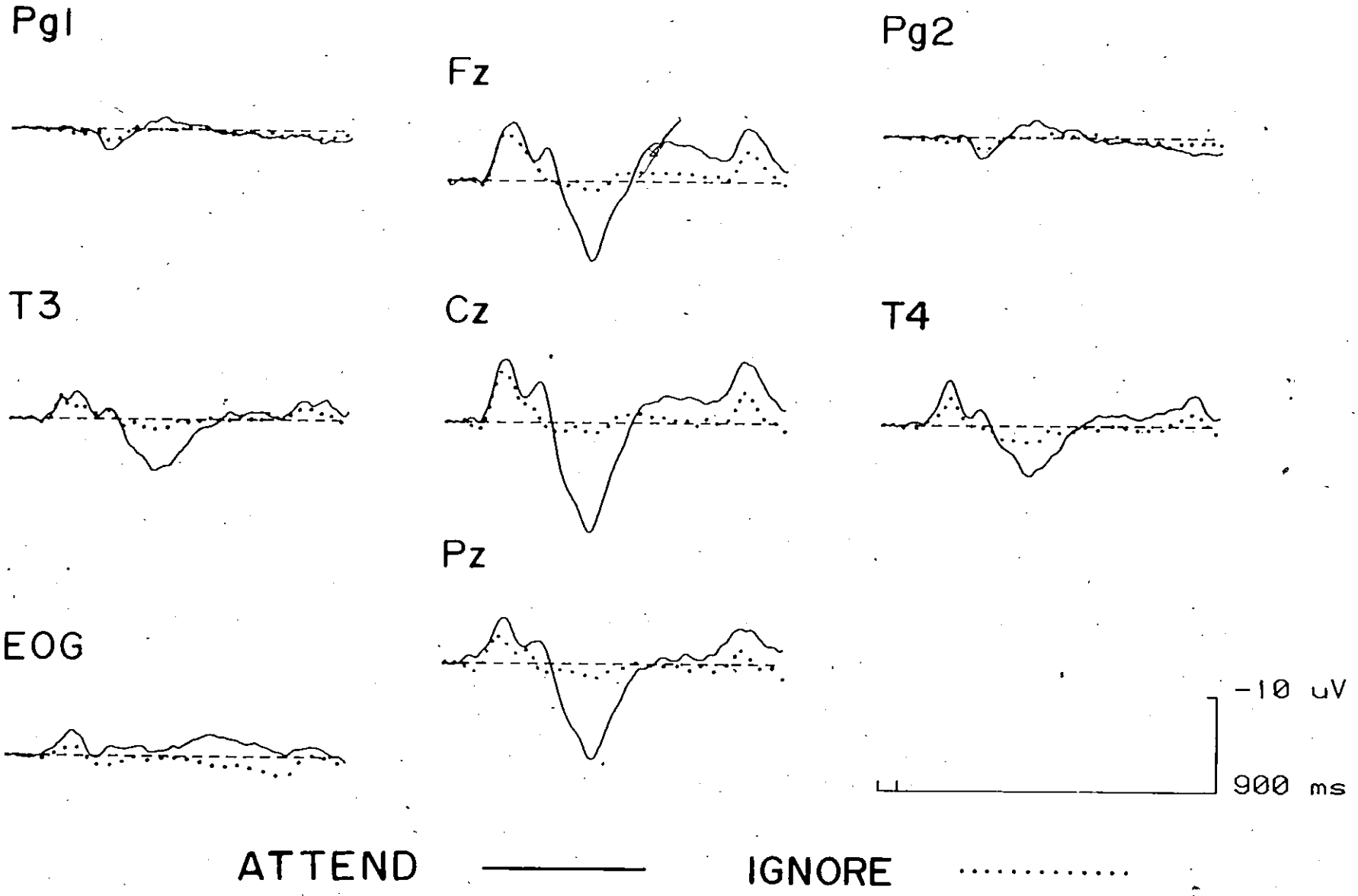
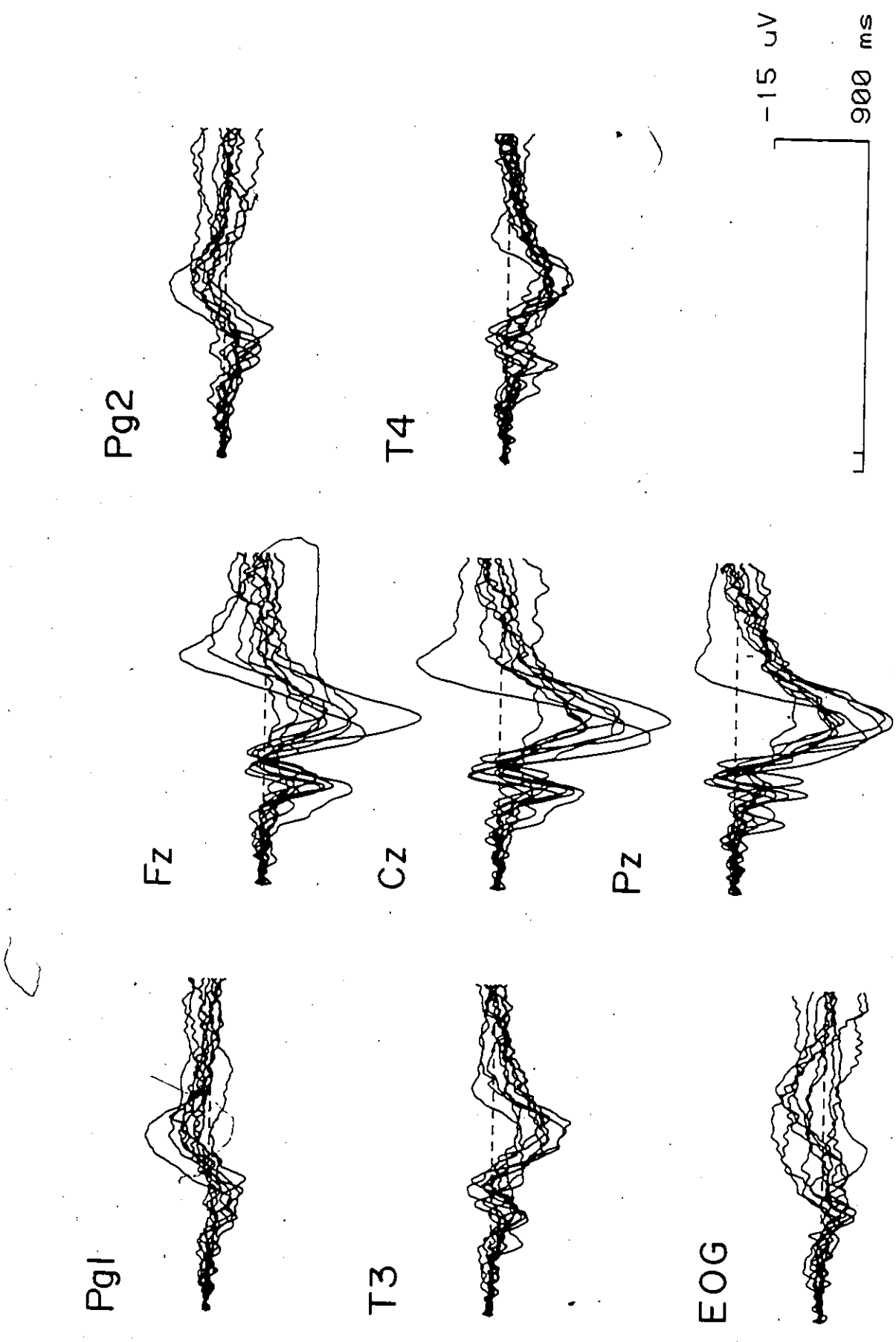


Figure 9

Figure 10



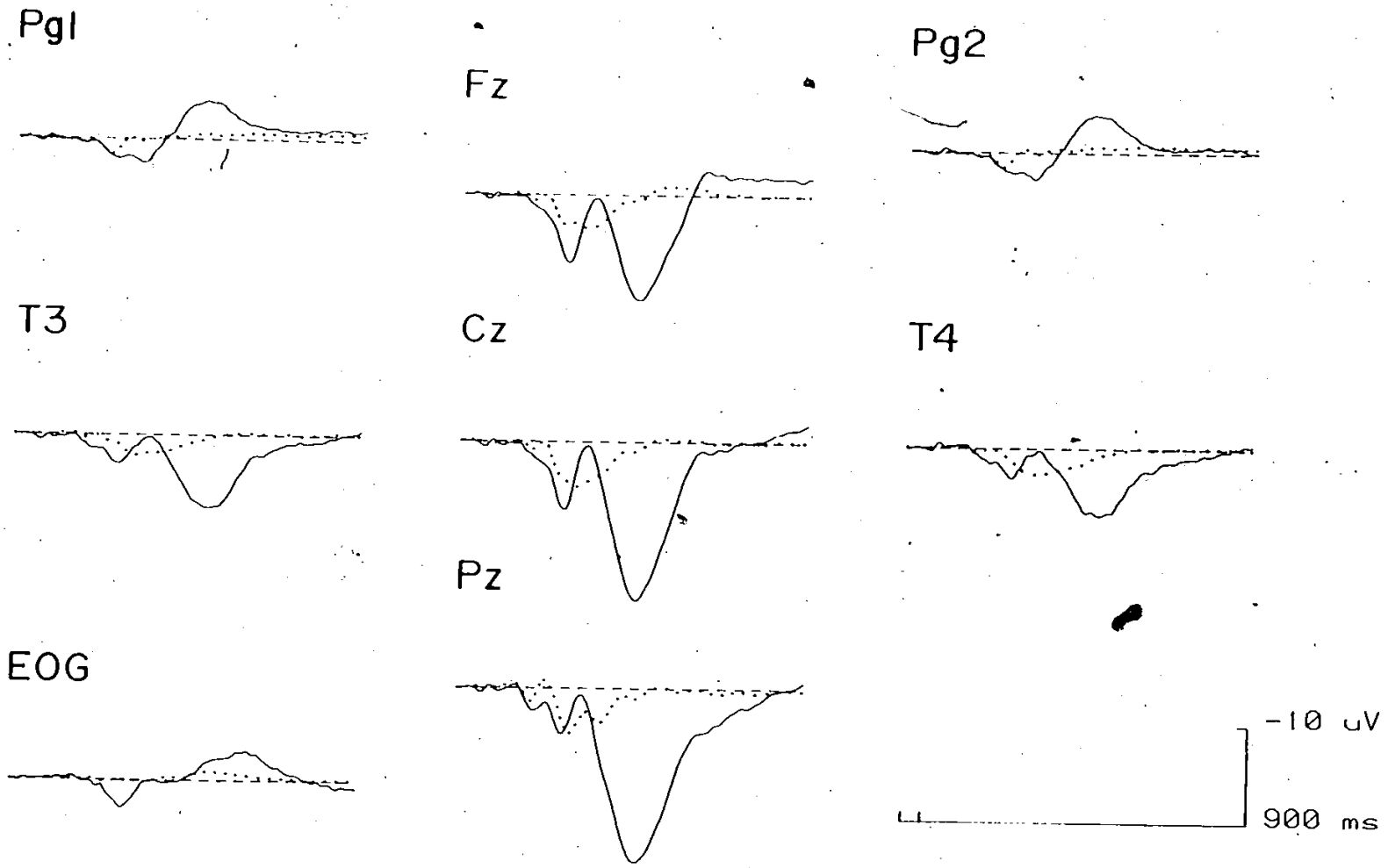


Figure 11

APPENDIX A: Nasopharyngeal recordings of endogenous event-related potentials.

From the Proceedings of the Sixth International Conference on Event-related Slow Potentials of the Brain. Chicago, Illinois, 1982.

NASOPHARYNGEAL RECORDINGS OF ENDOGENOUS EVENT-RELATED POTENTIALS

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In response to a task-relevant and improbable signal, there can be recorded from the human scalp, in addition to the "exogenous" or stimulus-bound evoked potentials, a series of "endogenous" potentials related to the perceptual detection of the stimulus (Donchin, Ritter & McCallum, 1978). The most prominent of these potentials is the late positive component, "P3" or "P300", that is maximally recorded from the centroparietal scalp. On the basis of this scalp-distribution it has been suggested that the P3 wave is generated in parietal association cortex (Vaughan & Ritter, 1976; Picton, Campbell, Baribeau-Braun & Proulx, 1978; Vaughan, Ritter & Simson, 1980).

Recently the endogenous components of the human evoked potential have been recorded from depth electrodes placed in the brains of epileptic human patients. Wood, Allison, Goff, Williamson and Spencer (1980) recorded from multiple-contact electrodes directed toward the temporal lobes from frontocentral and parieto-occipital locations. They found that there was no phase reversal of the P3 across the cortex, that the endogenous components were recorded with maximum amplitude subcortically, and that there was a shifting of peak latencies between the different recording locations on the electrode. These results suggested the possibility of multiple generators - perhaps both cortical and subcortical. Halgren, Squires, Wilson, Rohrbaugh, Babb and Crandall (1980) have recorded from depth electrodes placed in the hippocampus and amygdala. Large event-related potentials occurred in these areas at the same time as scalp-recorded endogenous potentials and with similar relationships to attention and probability. These potentials reversed in polarity over small distances and were associated with concurrent changes in unit activity. These results indicated definite neural activity in the limbic system during the scalp-recorded endogenous potentials, but left open the question whether part of the scalp-recorded activity is volume-conducted from the limbic system.

Nasopharyngeal electrodes are used in clinical electroencephalography to record from the mesial surface of the temporal lobe (Mavor & Hellen, 1964). Smith, Lell, Sidman and Mavor (1973) have reported that the N1 and P2 of the vertex potential are recorded with opposite polarity from nasopharyngeal electrodes referred to a balanced non-cephalic reference (Stephenson & Gibbs, 1951). On recordings taken between the ear and nasopharynx (Peters & Reilly, 1973), however, the only definitely recognizable evoked potential was a small ear-negative wave occurring about 35 ms later than the N1 peak at the vertex.

It is possible that nasopharyngeal recordings might allow an evaluation of limbic activity during normal human cognition. We therefore decided to record from nasopharyngeal electrodes the event-related potentials associated with the detection of improbable target stimuli.

Methods

Twelve subjects participated in the experiment. In each of two subjects only one nasopharyngeal electrode gave stable recordings, and the data analysis was therefore limited to 10 subjects. Each subject kept a running mental count of the number of 2000 Hz "target" tones presented unpredictably in 7-minute trains of 1000 Hz "standard"

tones. The probability of the targets during different blocks of stimuli was either 15% of 25%. The tones - 55 ms in total duration with rise and fall times of 5 ms each - were presented at an intensity of 65 dB peak SPL (about 50 dB SL) and a rate of once a second to the right ear.

Electroencephalographic signals were recorded from Fz, Cz, Pz, Pg1 and Pg2 relative to a balanced noncephalic reference electrode (Stephenson & Gibbs, 1951). An electro-oculogram was recorded between the upper and lower orbital ridges of the left eye. The signals were amplified using a bandpass of 0.16 - 100 Hz and averaged on a TN1500 Signal Analyser. Trials containing potentials of greater than 100 uV were excluded from the averaging. N1 (70-140 ms), P2 (130-230 ms), N2 (180-270 ms) and P3 (250-400 ms) peaks were identified in the vertex recording. The peak amplitudes were measured relative to the baseline at the onset of the evoked potential sweep and at the peak latency of the vertex component. A slow wave component consisting of a frontal negativity and a parietal positivity was measured at the peak latency of the frontal negativity.

Results

The grand-average waveforms for all electrode montages are plotted in Figure 1 together with the target-standard

subtraction waveforms. The left side of the figure shows the waveforms when the target had a probability of 15% and the right side shows the responses during the 25% target condition. The recordings from the nasopharyngeal electrodes are plotted at twice the amplitude of the scalp and EOG recordings.

The N1 component was maximally recorded at the midfrontal and vertex electrodes with an average peak latency of 103 ms. It was larger in response to the standard stimuli - 5.1 vs 3.6 uV at Cz. There were no significant potentials recorded at either of the nasopharyngeal electrodes at the peak latency of the vertex N1. There was, however, a small positivity with an average peak amplitude of 0.6 uV in the nasopharyngeal responses at a somewhat longer latency - 130 ms. This is identified in the average waveforms by the arrows. The P2 component was maximally recorded at the vertex with an average peak latency of 180 ms. It was small in amplitude (1.0 uV) and not significantly different between target and standard stimuli. There were no significant potentials recorded from either nasopharyngeal electrode at the vertex P2 peak latency.

The N2 component was maximally recorded from frontocentral electrodes in response to the target. There

was no significant N2 in the response to standard stimuli. The average peak-latency of the N2 component was 214 ms. At this latency there was a significant positive wave recorded from the nasopharyngeal electrodes with an amplitude equal to 51% of the vertex negativity. This positive wave was slightly but not significantly larger in the left nasopharyngeal recordings. The scalp N2 was larger for the 15% target than for the 25% target (2.9 vs 1.9 uV at Cz) but this difference was not quite significant ($0.05<p<0.10$). There was no significant difference between the two probabilities for the nasopharyngeal positive wave (1.4 vs 0.9 uV).

The P3 component was maximally recorded at the parietocentral electrodes with an average peak latency of 346 ms. At this latency there was a significant negative peak in the target-evoked potential recorded from the nasopharyngeal electrodes. This was symmetrical between Pg1 and Pg2 and on average 19% of the vertex P3 amplitude. This percentage was significantly smaller than that for the N2 component. The scalp P3 was larger at the vertex to the 15% target than to the 25% target (9.7 vs 8.4 uV) but this difference was not significant ($0.05 < p < 0.10$). The negative component recorded at the P3 latency from the nasopharynx showed no change with the different target probabilities

(1.6 vs 1.6 uV).

The slow wave component was recorded as a negative wave at Fz and as a positivity at Pz. These were significantly greater for targets than for standards but there were no significant effects of target probability. The average peak latency of the Fz negativity was 527 ms. No significant response was recorded from either nasopharyngeal electrode at this latency.

Discussion

There was no significant potential recorded from the nasopharyngeal electrodes at the latency of the scalp N1 component. There was, however, a somewhat later positive wave. This agrees well with the data presented in the figures of Smith and his colleagues (Smith et al., 1973) and with the measurements of Peters and Reilly (1973). It is possible that this component may occur at the same time as the temporal negative wave reported by several authors (Wolpaw and Penry, 1975; Picton, Woods, Stuss and Campbell, 1978; McCallum and Curry, 1980) as occurring with a peak latency between N1 and P2. The large nasopharyngeal negativity recorded at the same time as the scalp P2 component by Smith and his colleagues (1973) was not recognized by us or by Peters and Reilly (1973). This difference may be

related to the higher intensity of the stimulus used by Smith.

There were distinct components in the nasopharyngeal recordings at the same latencies as the endogenous N2 and P3 components recorded from the scalp. Similar though less clearly defined components have been noted in sphenoidal recordings (Halgren, personal communication). The three endogenous components were picked up in the nasopharynx with different amplitudes relative to the scalp components. The N2 was about one half the scalp amplitude, the P3 about one fifth and the slow wave virtually nonexistent. This is further evidence that the cerebral processes underlying these components are quite different.

The nasopharyngeal peaks were of opposite polarity to the scalp-recorded N2-P3 endogenous waves. This indicates that an equivalent dipole source for the potentials recorded at these peak latencies would be located between the scalp and the nasopharynx. Because little is known about volume conduction in the base of the skull, and because there are probably multiple concurrent generators, it is difficult to hypothesize about the source or sources of these potentials. The nasopharyngeal potentials were similar to the scalp-recorded potentials in relation to task-relevance, being

virtually non-existent in the response to standard stimuli. Unfortunately, our probability manipulation did not create a sufficiently large difference in the scalp-recorded endogenous components to determine whether the scalp and nasopharyngeal potentials were similarly or differently related to probability.

In conclusion, definite endogenous potentials can be recorded from nasopharyngeal electrodes. The exact relations of these potentials to the processes of cognition and to the scalp-recorded endogenous components remain to be determined.

Acknowledgements

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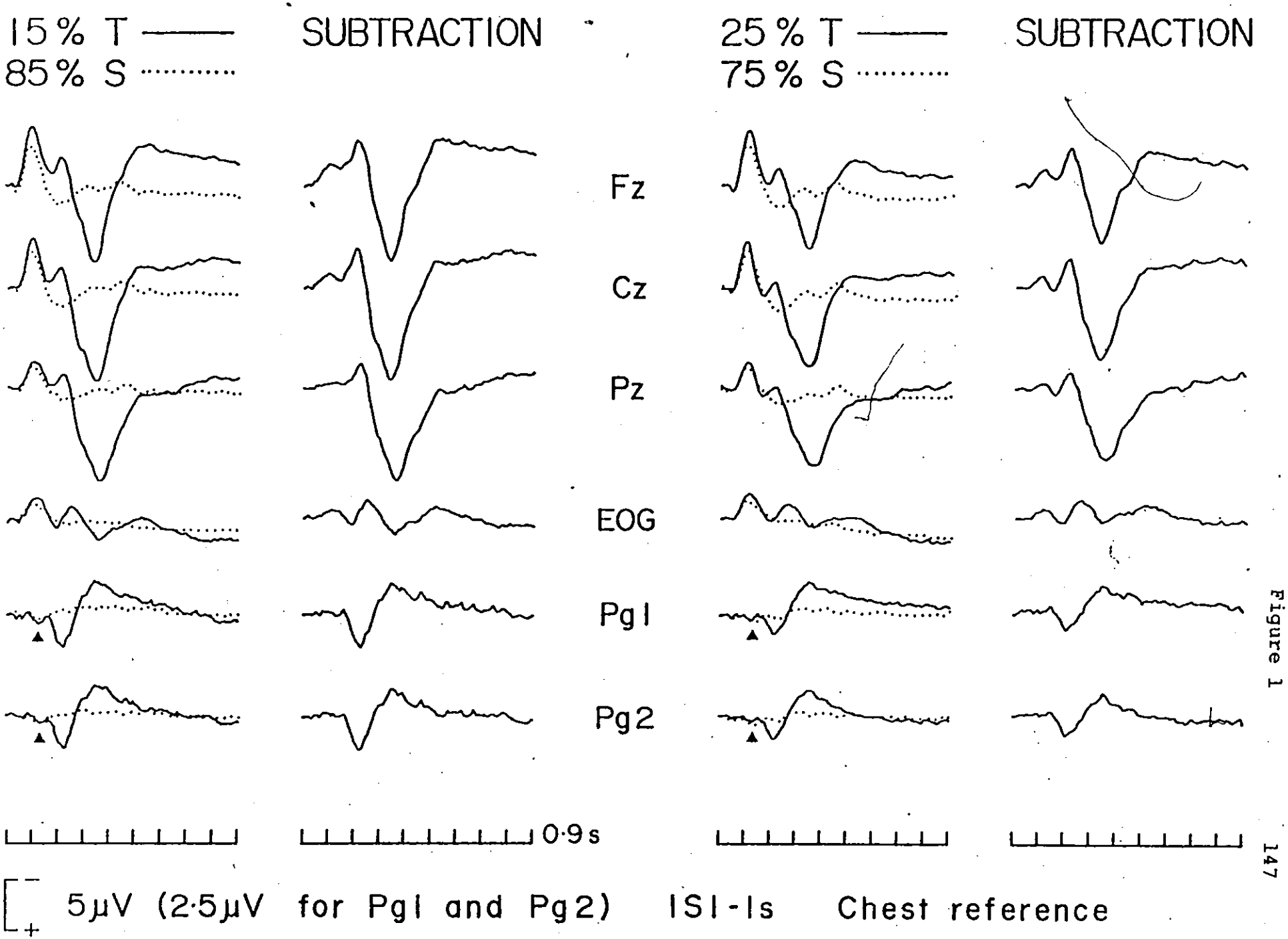

15% T
85% S
SUBTRACTION
25% T
75% S
SUBTRACTION
Fz
Cz
Pz
EOG
Pg1
Pg2
0·9s
5μV (2·5μV for Pg1 and Pg2) ISI-1s Chest reference

Figure 1

APPENDIX B: Tables of mean latencies and amplitudes.

EXPLANATORY NOTE

The tables are organized as follows:

Tables I, II, III-a and III-b are tables of latency. The labels above the latency figures refer to the component and the electrode at which the peak latency was determined (thus, for example, N1-Cz refers to the N1 peak in the waveform recorded from the Cz electrode). Experimental parameters are given at the beginning of each row, in the following order: sequential probability of the eliciting stimulus (%), interstimulus interval (ISI), stimulus intensity (Int.), number of subjects (N), and ear of delivery (right (R) or left (L)). Conditions can be compared to other conditions as indicated following the word "COMP:" (e.g. for purposes of determining intensity effects on latency, the second row can be compared to row number eight in Table I). The name of the condition has been put between parentheses in those cases where it cannot be derived from the experimental parameters given. Standard deviations are given between parentheses below the latency figures.

Tables IV to XII give the means and standard deviations of amplitude for indicated components at indicated electro-

des. They are otherwise organized as previously described. Tables XIII and XIV are self-explanatory.

Latencies are in miliseconds (ms) and amplitudes in microvolts (uV).

*****NOTA BENE*****

Due to a conversion error, all amplitudes must be multiplied by 1.22 in the following tables. Values given in the text are correct.

LIST OF TABLES

TABLE I

STANDARD STIMULUS / LATENCIES / AUDITORY

%	ISI	Int.	N	N1a-T3	N1a-T4	N1b-Cz	N1c-T3	N1c-T4	P2-Cz	PgP120
90	1.1	90	21 R	72	75	94	129	130	197	120
Basic Aud. Paradigm				(8)	(12)	(9)	(11)	(10)	(32)	(19)
90	1.1	90	12 R	71	73	92	127	130	191	120
COMP: 3.3 s ISI				(8)	(14)	(8)	(12)	(8)	(33)	(24)
90	1.1	90	10 R	70	72	91	124	126	187	120
COMP: 65 dB				(8)	(14)	(8)	(11)	(10)	(23)	(19)
90	1.1	90	10 R	73	79	90	128	129	191	117
COMP: 70% standards				(7)	(9)	(6)	(14)	(10)	(33)	(20)
90	1.1	90	10 R	77	77	96	129	132	205	117
COMP: Difficult Omitted 1.1s Visual				(8)	(10)	(10)	(10)	(12)	(31)	(16)
70	1.1	90	10 R	76	80	92	126	127	194	129
				(8)	(9)	(5)	(12)	(11)	(23)	(10)
70	3.3	90	12 R	73	73	96	128	130	196	136
				(9)	(17)	(8)	(12)	(14)	(14)	(27)
90	1.1	65	10 R	78	77	97	132	129	195	129
				(9)	(9)	(8)	(10)	(12)	(31)	(25)
90	1.1	90	10 R	73	70	93	130	126	200	127
(Difficult)				(7)	(15)	(7)	(12)	(2)	(29)	(14)
90	1.1	90	10 R	70	69	98	130	130	204	131
(Omitted)				(11)	(13)	(7)	(10)	(16)	(29)	(11)
90	.65	90	10 L	77	76	104	133	137	183	132
COMP: 5.0 s ISI Ignore				(19)	(19)	(15)	(22)	(9)	(35)	(19)
90	5.0	90	10 L	88	80	117	139	136	214	141
				(6)	(11)	(11)	(8)	(6)	(17)	(20)
90	.65	90	10 L	77	82	100	132	132	193	124
(Ignore)				(16)	(28)	(11)	(22)	(17)	(24)	(29)

TABLE II

TARGET STIMULUS / LATENCIES / AUDITORY

%	ISI	Int.	N	N2-Cz	N2-Pg	P3-Cz	P3-Pz	P3-Pg	SW-Fz	PgP120
10	1.1	90	21 R	208	212	318	348	366	539	110
Basic Aud. Paradigm				(20)	(13)	(45)	(30)	(42)	(74)	(22)
10	1.1	90	12 R	201	213	337	358	376	525	109
COMP: 3.3 s ISI				(11)	(13)	(33)	(26)	(51)	(74)	(22)
10	1.1	90	10 R	208	216	323	339	359	504	105
COMP: 65 dB				(25)	(11)	(26)	(28)	(34)	(61)	(26)
10	1.1	90	10 R	206	211	337	353	375	534	114
COMP: 30% targets				(26)	(14)	(39)	(32)	(53)	(80)	(28)
10	1.1	90	10 R	213	208	319	351	359	543	109
COMP: Difficult Omitted 1.1s Visual				(28)	(12)	(36)	(29)	(20)	(81)	(16)
30	1.1	90	10 R	218	219	333	338	364	506	117
				(24)	(15)	(54)	(60)	(33)	(57)	(28)
30	3.3	90	12 R	247	234	347	358	383	527	134
				(35)	(29)	(42)	(58)	(52)	(64)	(23)
10	1.1	65	10 R	212	214	335	345	352	507	122
				(25)	(11)	(34)	(30)	(46)	(60)	(23)
10	1.1	90	10 R	248	251	388	411	433	595	118
(Difficult)				(30)	(26)	(48)	(41)	(55)	(67)	(16)
10	1.1	00	10 R	227	261	382	411	493	641	146
(Omitted)				(44)	(45)	(45)	(42)	(68)	(83)	(40)
10	.65	90	10 L	216	226	338	345	345	517	121
COMP: 5.0 s ISI Omitted 0.65s Ignore				(28)	(25)	(27)	(25)	(42)	(40)	(21)
10	5.0	90	10 L	257	251	378	394	417	628	—
				(28)	(30)	(51)	(45)	(53)	(139)	—
10	.65	00	10 L	210	237	367	372	406	565	107
(Omitted)				(42)	(42)	(32)	(33)	(41)	(48)	(22)
10	.65	90	10 L	237	217	341	343	362	501	108
(Ignore)				(52)	(12)	(42)	(44)	(56)	(63)	(52)

TABLE III-a

STANDARD STIMULUS / LATENCIES / VISUAL

%	ISI	Int.	N	P1-Pz	N1-Pz	PgP190	P2-Cz
90	1.1	—	10	145	186	190	237
	(Visual)			(4)	(8)	(8)	(27)

TABLE III-b

TARGET STIMULUS / LATENCIES / VISUAL

%	ISI	Int.	N	N1-Pz	PgP197	P2-Cz	N2-Cz	N2-Pg	P3-Cz	P3-Pz	P3-Pg	SW-Fz
10	1.1	—	10	185	197	230	283	279	412	415	436	602
	(Visual)			(16)	(15)	(18)	(17)	(16)	(19)	(16)	(27)	(57)

TABLE IV

STANDARD STIMULUS / AMPLITUDES / AUDITORY Nla / Nlc

%	ISI	Int.	N	T3		T4		Pg1		Pg2	
				Nla	Nlc	Nla	Nlc	Nla	Nlc	Nla	Nlc
90	1.1	90	21 R	-1.4	-1.9	-0.8	-1.3	0.0	0.2	0.0	0.3
Basic Aud. Paradigm				(0.5)	(0.8)	(0.7)	(0.8)	(0.4)	(0.5)	(0.4)	(0.4)
90	1.1	90	12 R	-1.4	-1.9	-1.1	-1.4	-0.1	0.1	-0.1	0.3
COMP: 3.3 s ISI				(0.5)	(0.7)	(0.5)	(0.8)	(0.4)	(0.5)	(0.4)	(0.4)
90	1.1	90	10 R	-1.4	-1.7	-0.6	-1.0	-0.1	0.2	-0.1	0.2
COMP: 65 dB				(0.6)	(0.7)	(0.7)	(0.9)	(0.4)	(0.4)	(0.4)	(0.4)
90	1.1	90	10 R	-1.6	-1.6	-0.9	-0.9	0.0	0.2	-0.1	0.2
COMP: 70% targets				(0.7)	(0.5)	(0.9)	(0.9)	(0.4)	(0.6)	(0.3)	(0.4)
90	1.1	90	10 R	-1.3	-2.0	-0.9	-1.4	0.0	0.2	0.1	0.5
COMP: Difficult Omitted 1.1s Visual				(0.4)	(0.8)	(0.6)	(0.6)	(0.5)	(0.6)	(0.3)	(0.4)
70	1.1	90	10 R	-1.6	-1.8	-1.2	-1.3	-0.2	0.1	0.0	0.7
				(0.5)	(0.4)	(0.9)	(0.7)	(0.7)	(0.9)	(0.3)	(0.4)
70	3.3	90	12 R	-2.1	-3.1	-2.1	-2.5	-0.2	0.3	-0.2	0.6
				(1.1)	(1.2)	(1.3)	(0.9)	(0.8)	(1.0)	(0.4)	(0.9)
90	1.1	65	10 R	-1.2	-1.2	-0.9	-0.9	-0.1	0.1	0.0	0.2
				(0.4)	(0.5)	(0.7)	(0.8)	(0.4)	(0.3)	(0.4)	(0.6)
90	1.1	90	10 R	-1.5	-2.1	-0.9	-1.5	0.2	0.3	0.3	0.8
(Difficult)				(0.6)	(1.0)	(0.7)	(0.8)	(0.4)	(0.5)	(0.3)	(0.7)
90	1.1	90	10 R	-1.3	-2.3	-1.1	-1.7	0.0	0.4	-0.2	0.2
(Omitted)				(0.5)	(1.2)	(0.7)	(1.1)	(0.4)	(0.5)	(0.3)	(0.7)
90	.65	90	10 L	-1.1	-1.2	-0.9	-1.9	—	—	—	—
COMP: 5.0 s ISI Ignore				(0.5)	(0.6)	(0.4)	(0.8)	—	—	—	—
90	5.0	90	10 L	-1.9	-2.8	-0.6	-3.2	—	—	—	—
				(1.0)	(1.2)	(1.6)	(0.7)	—	—	—	—
90	.65	90	10 L	-1.0	-0.9	-0.8	-1.2	—	—	—	—
(Ignore)				(0.6)	(0.4)	(0.7)	(0.5)	—	—	—	—

TABLE V

STANDARD STIMULUS / AMPLITUDES / AUDITORY

N1b-Cz

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
90	1.1	90	21 R	-4.6	-4.7	-2.7	-1.1	-1.0	0.1	0.2
Basic Aud. Paradigm				(1.5)	(1.4)	(1.1)	(0.6)	(0.8)	(0.5)	(0.3)
90	1.1	90	12 R	-4.9	-4.8	-2.8	-1.1	-1.2	0.1	0.2
COMP: 3.3 s ISI				(1.1)	(1.2)	(1.0)	(0.5)	(0.7)	(0.6)	(0.5)
90	1.1	90	10 R	-4.0	-3.8	-2.6	-1.1	-0.8	0.2	0.2
COMP: 65 dB				(1.5)	(1.9)	(0.9)	(0.7)	(0.8)	(0.5)	(0.3)
90	1.1	90	10 R	-4.5	-4.2	-2.6	-1.2	-1.1	0.0	0.2
COMP: 70% targets				(1.6)	(1.2)	(1.0)	(0.7)	(0.8)	(0.6)	(0.3)
90	1.1	90	10 R	-4.3	-4.7	-2.5	-1.2	-1.0	0.1	0.3
COMP: Difficult Omitted 1.1 s Visual				(2.0)	(1.5)	(1.3)	(0.5)	(0.6)	(0.6)	(0.4)
70	1.1	90	10 R	-4.5	-4.1	-2.4	-1.1	-1.3	-0.2	0.2
				(1.7)	(1.2)	(0.6)	(0.7)	(0.9)	(0.9)	(0.4)
70	3.3	90	12 R	-7.5	-8.2	-4.4	-1.7	-1.9	0.1	0.2
				(2.9)	(2.7)	(1.6)	(1.1)	(1.3)	(1.3)	(0.6)
90	1.1	65	10 R	-3.7	-3.7	-2.4	-1.0	-0.8	0.0	0.0
				(1.0)	(1.0)	(0.5)	(0.7)	(0.8)	(0.3)	(0.4)
90	1.1	90	10 R	-4.4	-4.6	-2.8	-1.2	-1.0	0.3	0.5
(Difficult)				(1.5)	(1.5)	(1.2)	(0.7)	(0.8)	(0.6)	(0.6)
90	1.1	90	10 R	-4.2	-4.3	-2.6	-1.2	-1.1	0.3	0.1
(Omitted)				(1.0)	(1.0)	(1.2)	(0.8)	(1.1)	(0.5)	(0.5)
90	.65	90	10 L	—	-2.8	—	—	—	—	—
COMP: 5.0 s ISI Ignore				—	(0.8)	—	—	—	—	—
90	5.0	90	10 L	—	-8.2	—	—	—	—	—
				—	(1.5)	—	—	—	—	—
90	.65	90	10 L	—	-2.6	—	—	—	—	—
(Ignore)				—	(1.0)	—	—	—	—	—

TABLE VI

STANDARD STIMULUS / AMPLITUDES / AUDITORY

P2-Cz*

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
90	1.1	90	21 R	1.3	2.5	1.7	1.5	2.0	-0.2	0.0
Basic Aud. Paradigm				(1.4)	(1.4)	(1.0)	(0.7)	(0.7)	(0.4)	(0.5)
90	1.1	90	12 R	1.1	2.3	1.5	1.5	2.0	-0.2	-0.1
COMP: 3.3 s ISI				(1.5)	(1.4)	(0.9)	(0.7)	(0.7)	(0.5)	(0.4)
90	1.1	90	10 R	2.0	3.0	1.8	1.3	2.1	0.0	0.0
COMP: 65 dB				(1.3)	(1.2)	(0.7)	(0.6)	(0.7)	(0.4)	(0.5)
90	1.1	90	10 R	1.1	1.9	1.2	1.5	2.2	-0.1	0.1
COMP: 70% standards				(1.4)	(1.2)	(0.6)	(0.9)	(0.5)	(0.5)	(0.4)
90	1.1	90	10 R	1.6	2.7	2.0	1.4	2.1	-0.3	0.1
COMP: Difficult Omitted 1.1 s Visual				(1.0)	(1.2)	(1.1)	(0.7)	(0.6)	(0.5)	(0.5)
70	1.1	90	10 R	1.1	2.1	1.7	1.4	2.4	0.1	0.4
				(1.8)	(1.6)	(1.1)	(1.0)	(0.9)	(0.9)	(0.3)
70	3.3	90	12 R	2.2	5.3	4.4	2.0	2.5	0.1	0.4
				(2.6)	(2.8)	(2.2)	(1.3)	(1.1)	(0.7)	(0.8)
90	1.1	65	10 R	2.1	2.7	1.8	1.2	1.6	0.1	0.1
				(0.9)	(0.9)	(0.6)	(0.5)	(0.6)	(0.3)	(0.6)
90	1.1	90	10 R	0.9	2.0	1.5	1.6	2.3	-0.3	-0.2
(Difficult)				(0.9)	(1.2)	(1.2)	(0.9)	(0.7)	(0.3)	(0.7)
90	1.1	90	10 R	2.1	3.5	2.5	1.9	2.1	-0.2	-0.3
(Omitted)				(1.2)	(1.4)	(1.7)	(0.9)	(0.7)	(0.6)	(0.6)
90	.65	90	10 L	—	1.0	—	—	—	—	—
COMP: 5.0 s ISI Ignore				—	(1.1)	—	—	—	—	—
90	5.0	90	10 L	—	7.4	—	—	—	—	—
				—	(2.7)	—	—	—	—	—
90	.65	90	10 L	—	0.5	—	—	—	—	—
(Ignore)				—	(0.8)	—	—	—	—	—

* measured separately at T3 and T4 peak latencies.

TABLE VII

STANDARD AND TARGET STIMULI / AMPLITUDES / AUDITORY PgP120

%	ISI	Int.	N	STANDARD Pg1	STANDARD Pg2	TARGET Pg1	TARGET Pg2
10† Basic Aud. Paradigm	1.1	90	21 R	0.5 (0.4)	0.6 (0.4)	0.9 (1.1)	1.0 (0.6)
10† COMP: 3.3 s ISI	1.1	90	12 R	0.5 (0.5)	0.5 (0.3)	0.6 (1.0)	1.0 (0.5)
10† COMP: 65 dB	1.1	90	10 R	0.6 (0.4)	0.6 (0.4)	0.9 (1.0)	0.9 (0.8)
10† COMP: 30% targets*	1.1	90	10 R	0.6 (0.5)	0.8 (0.3)	0.5 (1.3)	0.8 (0.8)
10† COMP: Difficult Omitted 1.1s	1.1	90	10 R	0.4 (0.4)	0.7 (0.3)	0.8 (1.3)	0.9 (0.5)
30©	1.1	90	10 R	0.4 (0.7)	0.8 (0.4)	0.3 (0.9)	0.6 (0.3)
30©	3.3	90	12 R	0.9 (0.8)	0.9 (0.7)	0.5 (1.0)	0.8 (1.0)
10†	1.1	65	10 R	0.3 (0.4)	0.4 (0.5)	0.7 (0.7)	0.8 (0.5)
10† (Difficult)	1.1	90	10 R	0.5 (0.4)	1.0 (0.7)	0.9 (0.9)	1.2 (0.8)
10† (Omitted)	1.1	00	10 R	0.6 (0.5)	0.4 (0.4)	— —	— —
10† COMP: 5.0 s ISI Omitted 0.65s Ignore	.65	90	10 L	0.6 (0.6)	0.6 (0.5)	0.4 (1.2)	0.4 (1.4)
10†	5.0	90	10 L	1.2 (0.9)	1.4 (0.6)	— —	— —
10† (Omitted)	.65	00	10 L	— —	— —	0.9 (0.7)	0.8 (0.6)
10† (Ignore)	.65	90	10 L	0.4 (0.6)	0.3 (0.5)	0.7 (0.9)	0.7 (0.8)

† or "90%" for STANDARD;
* or "70% standards" for STANDARD
© or "70%" for STANDARD

TABLE VIII

TARGET STIMULUS / AMPLITUDES / AUDITORY

Nla / Nlc

%	ISI	Int.	N	T3		T4		Pg1		Pg2	
				Nla	Nlc	Nla	Nlc	Nla	Nlc	Nla	Nlc
10	1.1	90	21 R	-1.8	-3.8	-1.5	-3.5	-0.2	0.1	0.0	0.3
Basic Aud. Paradigm				(0.9)	(1.4)	(1.1)	(1.6)	(0.8)	(1.1)	(0.7)	(0.7)
10	1.1	90	12 R	-1.8	-3.7	-1.8	-3.2	-0.5	-0.1	-0.2	0.3
COMP: 3.3 s ISI				(0.7)	(1.4)	(1.0)	(1.3)	(0.9)	(1.2)	(0.6)	(0.5)
10	1.1	90	10 R	-2.0	-3.3	-1.3	-2.6	-0.5	-0.1	-0.5	0.1
COMP: 65 dB				(1.1)	(1.5)	(0.9)	(1.0)	(0.6)	(0.9)	(0.7)	(0.6)
10	1.1	90	10 R	-1.9	-3.6	-1.7	-3.0	-0.2	-0.1	0.0	0.4
COMP: 30% targets				(1.0)	(0.8)	(1.1)	(1.3)	(0.8)	(1.1)	(0.5)	(0.7)
10	1.1	90	10 R	-1.8	-3.8	-1.5	-4.0	0.0	0.1	0.1	0.2
COMP: Difficult Visual				(0.9)	(1.7)	(1.3)	(1.2)	(0.8)	(1.3)	(0.6)	(0.7)
30	1.1	90	10 R	-1.2	-2.4	-0.9	-1.9	-0.2	0.1	0.0	0.4
				(0.6)	(0.7)	(0.6)	(0.9)	(0.5)	(0.9)	(0.4)	(0.4)
30	3.3	90	12 R	-2.1	-3.6	-2.3	-2.0	-0.6	-0.2	-0.5	0.3
				(1.1)	(1.1)	(1.6)	(2.0)	(1.0)	(1.0)	(0.6)	(1.1)
10	1.1	65	10 R	-1.4	-2.1	-1.1	-1.5	0.2	0.1	0.0	0.0
				(0.4)	(1.2)	(1.3)	(1.5)	(0.6)	(0.7)	(0.6)	(0.8)
10	1.1	90	10 R	-1.6	-2.1	-1.1	-2.5	0.2	0.4	0.5	0.9
(Difficult)				(0.6)	(0.8)	(0.6)	(1.2)	(0.8)	(1.1)	(0.8)	(0.9)
10	.65	90	10 L	-1.1	-1.2	-0.9	-1.9	—	—	—	—
COMP: 5.0 s ISI Ignore				(0.5)	(0.6)	(0.4)	(0.8)	—	—	—	—
10	5.0	90	10 L	-1.9	-2.8	-0.6	-3.2	—	—	—	—
				(1.0)	(1.2)	(1.6)	(0.7)	—	—	—	—
10	.65	90	10 L	-1.0	-0.9	-0.8	-1.2	—	—	—	—
(Ignore)				(0.6)	(0.4)	(0.7)	(0.5)	—	—	—	—

TABLE IX

TARGET STIMULUS / AMPLITUDES / AUDITORY

N1b-Cz

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
10	1.1	90	21 R	-6.7	-6.7	-4.0	-2.0	-2.4	0.0	0.2
Basic Aud. Paradigm				(2.4)	(2.0)	(1.7)	(1.6)	(1.4)	(1.2)	(0.8)
10	1.1	90	12 R	-6.8	-6.5	-3.9	-2.0	-2.2	-0.5	0.0
COMP: 3.3 s ISI				(1.9)	(2.1)	(1.7)	(1.9)	(1.0)	(1.1)	(0.6)
10	1.1	90	10 R	-5.9	-5.9	-3.6	-1.8	-1.8	-0.1	-0.1
COMP: 65 dB				(1.9)	(2.3)	(2.0)	(2.1)	(1.1)	(0.6)	(0.8)
10	1.1	90	10 R	-6.1	-5.8	-3.2	-2.0	-1.9	-0.4	0.0
COMP: 30% targets				(2.0)	(1.6)	(1.5)	(1.2)	(1.0)	(1.2)	(0.5)
10	1.1	90	10 R	-7.3	-7.6	-4.7	-2.7	-3.1	0.2	0.3
COMP: Difficult Omitted 1.1s Visual				(2.8)	(1.7)	(1.3)	(1.2)	(1.8)	(1.5)	(0.9)
30	1.1	90	10 R	-5.1	-4.6	-2.4	-1.3	-1.5	-0.3	0.1
				(1.5)	(1.0)	(0.5)	(0.8)	(0.8)	(1.0)	(0.3)
30	3.3	90	12 R	-9.2	-5.9	-3.9	-1.8	-2.2	-0.7	-0.2
				(2.5)	(2.9)	(2.0)	(1.5)	(1.4)	(1.5)	(0.8)
10	1.1	65	10 R	-4.1	-4.0	-2.6	-1.1	-0.8	0.1	0.1
				(1.7)	(2.1)	(2.0)	(1.1)	(1.1)	(0.5)	(0.6)
10	1.1	90	10 R	-5.1	-5.2	-2.8	-1.6	-1.2	0.5	0.8
(Difficult)				(2.3)	(2.3)	(2.1)	(0.9)	(1.3)	(1.2)	(1.1)

TABLE X

TARGET STIMULUS / AMPLITUDES / AUDITORY N2-Cz* and N2-Pg†

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
10	1.1	90	21 R	-5.4	-5.0	-2.5	-2.2	-2.0	2.0	2.3
Basic Aud. Paradigm				(2.4)	(3.0)	(2.3)	(1.9)	(2.1)	(1.1)	(.09)
10	1.1	90	12 R	-5.4	-4.4	-2.4	-2.2	-2.0	2.0	2.4
COMP: 3.3 s ISI				(2.3)	(2.8)	(2.1)	(1.7)	(1.9)	(1.0)	(0.8)
10	1.1	90	10 R	-5.0	-4.7	-2.3	-2.1	-1.8	1.7	2.2
COMP: 65 dB				(2.7)	(2.8)	(2.2)	(1.6)	(1.9)	(1.4)	(1.2)
10	1.1	90	10 R	-4.2	-3.5	-1.9	-1.7	-1.3	1.5	2.0
COMP: 30% targets				(1.8)	(1.8)	(1.8)	(1.1)	(1.2)	(1.0)	(0.8)
10	1.1	90	10 R	-5.6	-5.2	-2.0	-2.4	-1.6	1.6	1.8
COMP: Difficult Omitted 1.1s Visual				(2.8)	(3.2)	(2.9)	(2.4)	(2.6)	(1.8)	(1.1)
30	1.1	90	10 R	-2.3	-1.0	-0.1	-0.6	-0.4	1.0	1.2
				(2.0)	(1.5)	(1.9)	(1.3)	(1.3)	(0.8)	(0.7)
30	3.3	90	12 R	-2.0	0.3	-2.0	0.2	-0.2	0.3	0.4
				(3.2)	(2.7)	(0.8)	(1.7)	(1.9)	(1.2)	(1.7)
10	1.1	65	10 R	-2.5	-2.8	-0.8	-0.9	-0.4	1.9	2.1
				(2.7)	(3.4)	(1.8)	(1.9)	(1.8)	(1.2)	(1.2)
10	1.1	90	10 R	-5.2	-4.9	-2.5	-2.7	-2.4	1.5	2.0
(Difficult)				(1.6)	(2.7)	(2.0)	(1.1)	(2.1)	(1.1)	(1.1)
90	1.1	00	10 R	-0.8	-1.6	-0.9	-1.2	-1.1	1.3	1.7
(Omitted)				(1.7)	(2.5)	(2.2)	(1.4)	(1.6)	(1.2)	(1.1)
90	.65	90	10 L	-4.4	-4.0	-2.8	-1.4	-2.0	2.0	2.0
COMP: 5.0 s ISI Omitted 0.65s Ignore				(2.5)	(4.3)	(3.6)	(1.5)	(1.5)	(1.1)	(1.7)
90	5.0	90	10 L	-.04	1.0	1.2	0.1	-1.0	2.2	3.1
				(4.2)	(4.1)	(2.6)	(2.3)	(3.6)	(2.2)	(2.3)
90	.65	00	10 L	-2.3	-2.4	-1.8	-1.4	-2.4	1.7	2.0
(Omitted)				(1.7)	(2.0)	(1.9)	(1.1)	(1.6)	(1.0)	(1.5)
90	.65	90	10 L	-1.8	-1.5	-1.1	-1.1	-0.6	1.1	1.3
(Ignore)				(2.1)	(2.5)	(1.7)	(1.0)	(1.2)	(1.4)	(1.3)

* measured separately at T3 and T4 peak latencies.
† at Pg1 and Pg2 electrodes only.

TABLE XI

TARGET STIMULUS / AMPLITUDES / AUDITORY

P3-Cz* and P3-Pg†

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
10	1.1	90	21 R	8.2	11.5	11.6	5.8	5.6	-2.8	-2.6
Basic Aud. Paradigm				(5.1)	(5.2)	(4.2)	(2.1)	(2.6)	(1.6)	(1.7)
10	1.1	90	12 R	6.3	9.9	11.1	5.5	5.3	-2.8	-2.0
COMP: 3.3 s ISI				(4.4)	(4.0)	(3.5)	(1.5)	(2.8)	(1.7)	(1.3)
10	1.1	90	10 R	7.7	10.4	10.3	5.5	5.1	-2.3	-2.0
COMP: 65 dB				(4.0)	(4.5)	(4.0)	(1.9)	(2.9)	(1.5)	(1.5)
10	1.1	90	10 R	6.5	9.5	10.1	5.6	6.1	-2.9	-2.1
COMP: 30% targets				(4.9)	(4.7)	(3.9)	(2.2)	(2.6)	(1.8)	(1.5)
10	1.1	90	10 R	9.4	13.1	12.8	5.8	5.5	-3.1	-3.4
COMP: Difficult Omitted 1.1s Visual				(5.4)	(5.5)	(4.4)	(2.3)	(2.2)	(1.4)	(1.9)
30	1.1	90	10 R	3.1	5.8	6.8	3.9	3.7	-1.6	-1.2
				(2.3)	(2.8)	(3.2)	(1.9)	(1.4)	(1.6)	(1.1)
30	3.3	90	12 R	4.1	8.2	10.6	5.0	3.6	-3.6	-3.3
				(3.0)	(2.9)	(3.3)	(2.0)	(2.4)	(2.4)	(1.8)
10	1.1	65	10 R	7.6	10.3	11.4	5.6	5.1	-2.5	-2.7
				(2.0)	(3.5)	(3.7)	(1.8)	(2.6)	(1.4)	(1.4)
10	1.1	90	10 R	5.2	9.1	10.4	4.3	4.3	-2.9	-2.0
(Difficult)				(4.6)	(4.5)	(3.1)	(1.6)	(2.0)	(1.3)	(1.7)
10	1.1	00	10 R	3.8	5.1	6.5	2.1	2.9	-2.0	-1.1
(Omitted)				(3.1)	(2.9)	(2.9)	(1.3)	(1.6)	(1.3)	(1.3)
10	.65	90	10 L	7.7	10.7	8.9	—	—	-1.4	-1.9
COMP: 5.0 s ISI Omitted 0.65s Ignore				(4.7)	(4.1)	(3.2)	—	—	(1.0)	(1.3)
10	5.0	90	10 L	9.1	12.7	13.1	—	—	-2.0	-1.3
				(6.7)	(5.4)	(5.7)	—	—	(2.6)	(2.9)
10	.65	00	10 L	4.8	7.2	8.1	—	—	-1.0	-0.8
(Omitted)				(2.5)	(2.8)	(2.6)	—	—	(1.5)	(1.2)
10	.65	90	10 L	1.3	1.8	2.0	—	—	-0.5	-0.9
(Ignore)				(1.2)	(1.3)	(1.2)	—	—	(1.2)	(1.2)

* measured separately at Pz peak latency.
† at Pg1 and Pg2 only.

TABLE XII

TARGET STIMULUS / AMPLITUDES / AUDITORY

SW-Fz

%	ISI	Int.	N	Fz	Cz	Pz	T3	T4	Pg1	Pg2
10	1.1	90	21 R	-5.8	-2.8	0.5	-0.7	-0.4	-0.8	-0.7
Basic Aud. Paradigm				(3.1)	(2.8)	(3.2)	(1.8)	(2.1)	(1.6)	(1.5)
10	1.1	90	12 R	-6.1	-2.1	0.8	-0.9	-0.8	-0.6	-0.4
COMP: 3.3 s ISI				(3.5)	(2.8)	(3.8)	(2.1)	(2.6)	(1.5)	(1.3)
10	1.1	90	10 R	-5.9	-2.5	0.7	-0.8	-1.1	-0.3	-0.3
COMP: 65 dB				(3.6)	(2.4)	(2.7)	(1.6)	(2.5)	(1.9)	(1.6)
10	1.1	90	10 R	-4.5	-1.8	0.6	-0.6	0.5	-1.2	-0.7
COMP: 30% targets				(2.6)	(2.4)	(3.7)	(2.0)	(1.4)	(1.7)	(1.4)
10	1.1	90	10 R	-5.0	-2.7	1.2	-0.3	0.2	-1.5	-1.6
COMP: Difficult Omitted 1.1s Visual				(3.3)	(3.3)	(2.4)	(1.6)	(1.2)	(1.7)	(2.2)
30	1.1	90	10 R	-2.7	-1.1	0.8	-0.1	0.6	-0.7	-0.3
				(1.4)	(1.2)	(1.8)	(1.1)	(0.9)	(0.9)	(0.9)
30	3.3	90	12 R	-4.9	-0.9	2.2	-0.7	-0.6	-2.2	-2.0
				(3.1)	(2.5)	(3.1)	(1.6)	(2.3)	(1.5)	(1.9)
10	1.1	65	10 R	-5.7	-1.9	1.7	-1.1	-0.7	-0.8	-0.7
				(3.9)	(2.5)	(2.1)	(1.2)	(2.7)	(1.4)	(1.3)
10	1.1	90	10 R	-4.4	-1.8	1.5	-0.2	0.3	-1.2	-0.7
(Difficult)				(2.8)	(3.5)	(2.5)	(1.1)	(1.4)	(1.4)	(1.8)
10	1.1	00	10 R	-4.3	-3.8	-0.5	-0.9	-0.3	0.0	0.5
(Omitted)				(2.7)	(4.8)	(3.9)	(1.8)	(1.7)	(1.6)	(1.2)
10	.65	90	10 L	-4.5	-3.2	-0.9	—	—	-0.1	-0.4
COMP: 5.0 s ISI Omitted 0.65s Ignore				(3.1)	(2.6)	(2.3)	—	—	(1.8)	(2.7)
10	5.0	90	10 L	-5.4	-1.8	1.5	—	—	0.4	1.1
				(3.9)	(2.2)	(2.4)	—	—	(3.5)	(5.2)
10	.65	00	10 L	-2.4	-0.9	1.6	—	—	0.1	1.0
(Omitted)				(1.7)	(2.6)	(2.5)	—	—	(1.6)	(1.1)
10	.65	90	10 L	-1.9	-1.5	-0.5	—	—	-0.2	-0.3
(Ignore)				(0.8)	(0.9)	(0.8)	—	—	(1.1)	(1.4)

TABLE XIII
STANDARD STIMULUS / AMPLITUDES / VISUAL

	P1-Pz	N1-Pz	PgP190	P2-Cz
Fz	0.6 (1.6)	1.0 (1.1)	1.4 (1.4)	3.6 (1.6)
Cz	0.8 (1.6)	0.6 (1.2)	1.2 (1.6)	4.5 (1.6)
Pz	1.3 (1.3)	-0.8 (1.1)	-0.4 (1.2)	3.4 (2.1)
T3	0.4 (0.7)	0.2 (0.6)	0.5 (0.9)	1.8 (0.9)
T4	0.4 (0.8)	-0.2 (0.5)	0.1 (0.7)	1.8 (1.0)
Pg1	0.3 (0.5)	1.3 (0.7)	1.4 (0.7)	0.4 (0.7)
Pg2	0.3 (0.3)	1.2 (0.5)	1.4 (0.6)	0.2 (0.5)

TABLE XIV

TARGET STIMULUS / AMPLITUDES / VISUAL

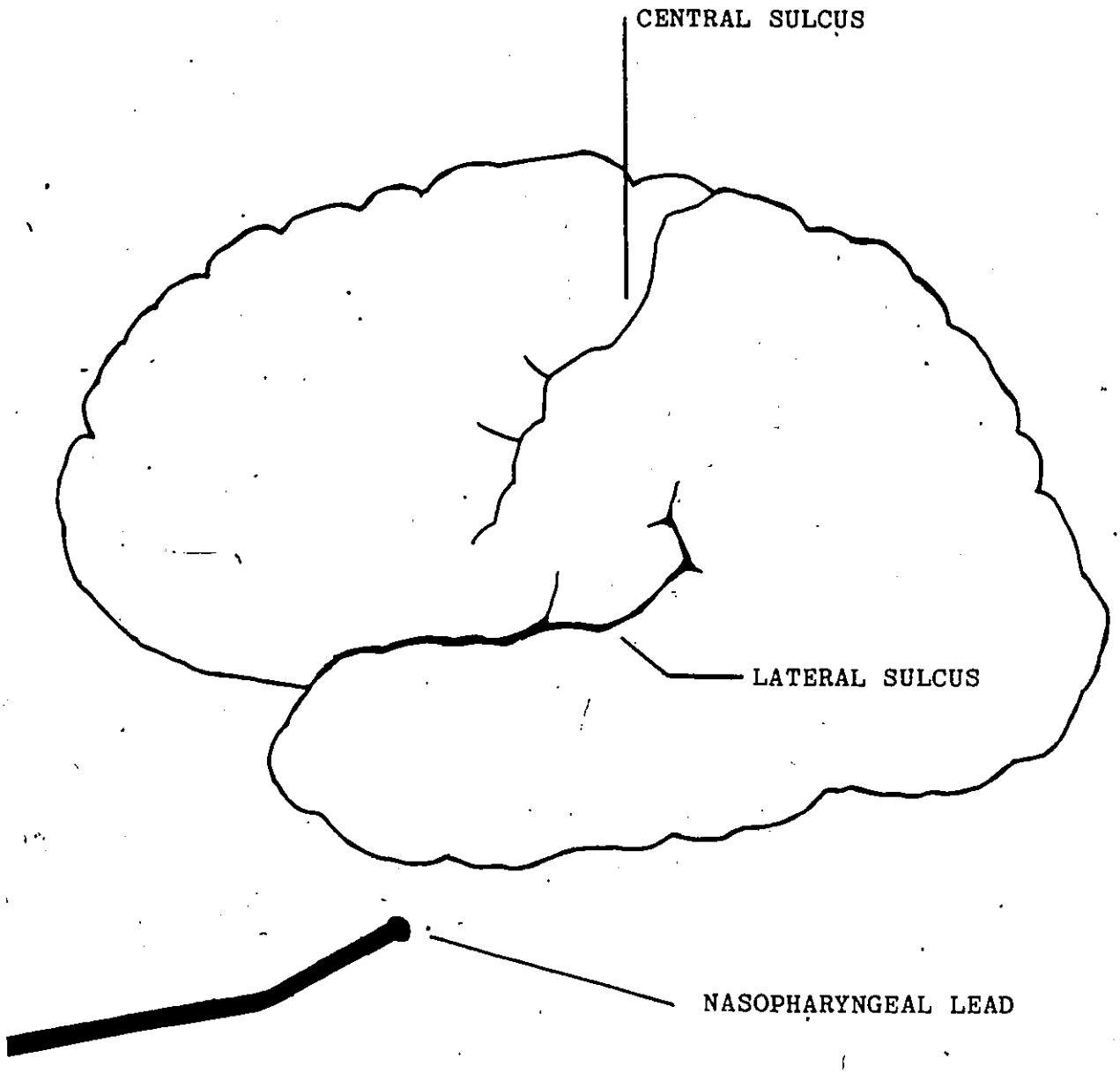
	N1–Cz	PgP190	P2–Cz	N2–Cz	N2–Pg	P3–Cz	P3–Pg	Sw–Fz
Fz	1.8	—	6.6	-0.9	—	9.2	—	-2.8
	(2.6)	—	(2.5)	(1.3)	—	(4.9)	—	(3.8)
Cz	1.6	3.3	6.6	-1.2	—	13.7	—	-0.4
	(2.4)	(2.5)	(2.3)	(2.1)	—	(4.7)	—	(4.1)
Pz	0.4	—	5.3	-0.9	—	14.7	—	2.8
	(0.8)	—	(2.4)	(2.7)	—	(3.9)	—	(3.2)
T3	0.8	—	3.0	-0.9	—	5.4	—	1.2
	(0.9)	—	(1.2)	(1.6)	—	(2.2)	—	(1.8)
T4	0.8	—	2.9	-1.0	—	5.2	—	1.5
	(1.2)	—	(2.1)	(1.3)	—	(1.5)	—	(1.5)
Pg1	1.4	2.0	1.3	2.3	2.5	-2.8	-3.5	-1.2
	(1.2)	(1.3)	(1.2)	(1.5)	(1.5)	(2.0)	(2.4)	(1.7)
Pg2	1.4	1.9	1.4	2.5	2.7	-2.9	-3.5	-0.5
	(1.2)	(1.3)	(1.3)	(2.0)	(2.1)	(1.6)	(1.8)	(2.1)

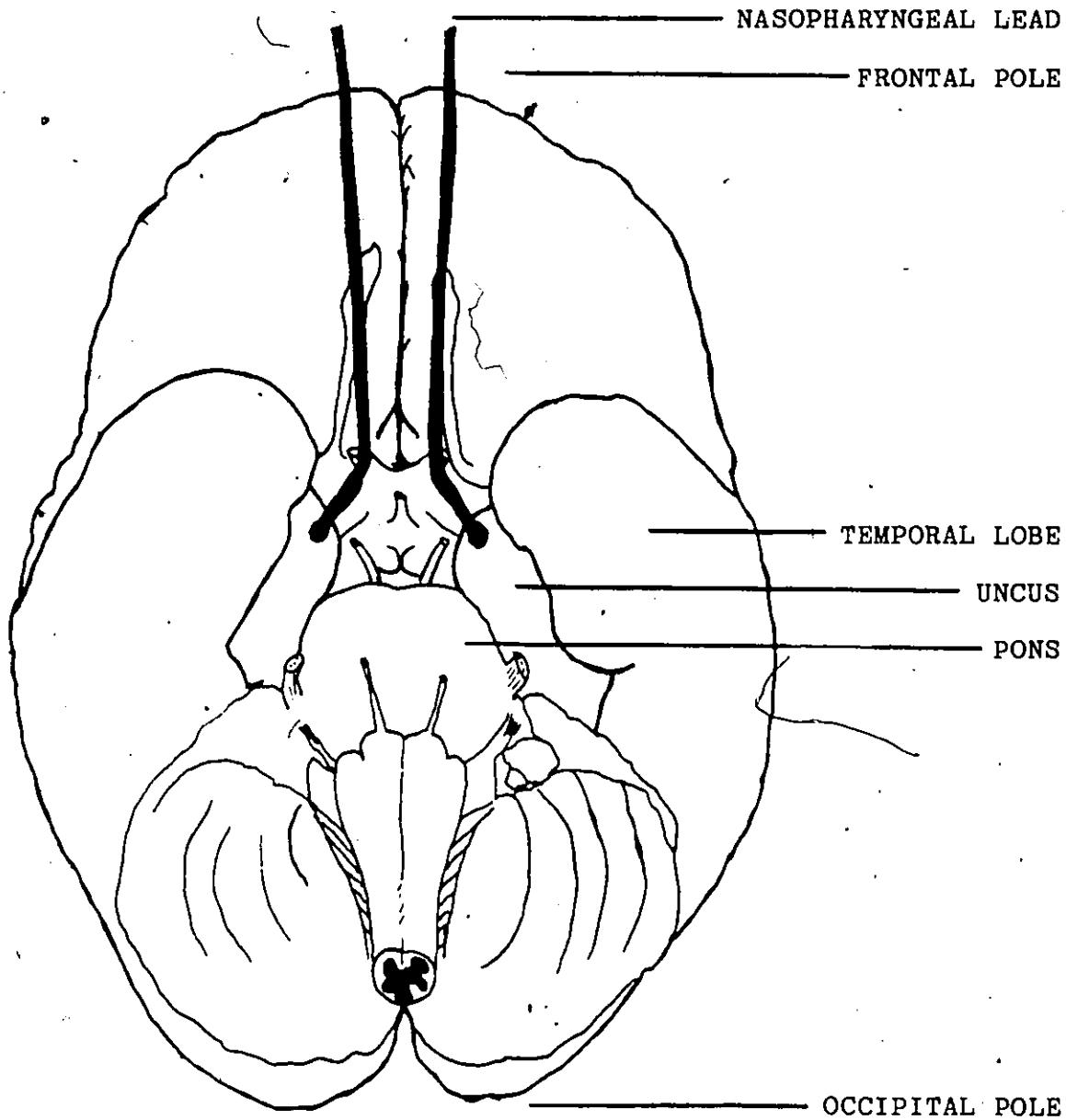
APPENDIX C: Anatomical localization of nasopharyngeal electrodes.

FIGURE LEGENDS

Figure 1. Lateral view of the left hemisphere. The location of the nasopharyngeal electrode has been estimated from the X-rays of two different subjects. The tip of the electrodes is located approximately 0.5 cm from the basal surface of the temporal lobe and about 4.5 cm below the lateral sulcus. Scale is 5/6.

Figure 2. Basal view of the brain. The location of the nasopharyngeal electrodes has been estimated from the X-rays of two different subjects. The tip of the electrodes is located approximately 6.5 cm from the frontal pole in the antero-posterior dimension and about 1.5 cm from midline, laterally. Scale is 5/6.


CENTRAL SULCUS
LATERAL SULCUS
NASOPHARYNGEAL LEAD


NASOPHARYNGEAL LEAD
FRONTAL POLE
TEMPORAL LOBE
UNCUS
PONS
OCCIPITAL POLE