NEUROPHYSIOLOGICAL EVALUATION OF SENSORY GATING AND SELECTIVE ATTENTION IN SCHIZOTYPAL PERSONALITY.

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A Thesis in the Department of Psychology

Presented in Partial Fulfillment of the Requirements for the Degree of
Master of Arts at Concordia University,
Montreal, Quebec, 1993.

December, 1993

ISBN 0-315-90806-8

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Abstract

NEUROPHYSIOLOGICAL EVALUATION OF SENSORY GATING AND SELECTIVE ATTENTION IN SCHIZOTYPAL PERSONALITY.

Robert M. Roth

Current debate centres around the question of whether schizotypal personality is part of a spectrum of schizophrenia-related disorders. Investigations of biological markers for schizophrenia in schizotypal subjects have tried to test this relationship. Given that there is considerable evidence for both sensory gating and binaural selective attention abnormalities in schizophrenia, as reflected by event-related potentials (ERP), these attention and preattention mechanisms were investigated in students scoring in the top (n = 11) and bottom (n = 12) 10% of scores on the Schizotypal Personality Questionnaire (Raine, 1991). Groups were matched for age, gender composition, handedness and education. Results failed to reveal group differences in suppression of the P50 in the sensory gating task, suggesting that preattentive gating is not disturbed. In contrast, on the selective attention task, predicted patterns of ERP differences were generally found. The schizotypal group was characterized by hyperarousal as indexed by shorter latencies of most ERP components, selective dishabituation of the orienting response to the rare salient stimuli, response-set deficits, intrusion effects, as well as large post-response cognitive processing of stimuli. Results were consistent with ERP abnormalities found in schizophrenics with florid and low formal thought disorders.

This thesis is dedicated to my parents

and the memory of

Michel (Chicho) Farah

Acknowledgments

Upon entering the psychology program at Concordia University, neurophysiology was still something I had only read about. I would therefore like to express my deepest gratitude to Dr. Jacinthe Baribeau, my supervisor, for taking me under her wings and not only showing me the "how to" of ERP research, but also fuelling the passion required to carry out work in this area. This thesis could not have been completed without our many, many hours, of stimulating talks about ERPs and psychopathology.

In addition to the above, I thank Kristina Kandyba, Marc Léger, Nanci Moretti, Hughes Hervé, Aline Szrogosz and Phil Wahba, all of whom assisted me at various times, in a variety of ways, over the course of this project.

A great big thanks also goes out to my parents (Louis and Veronica) and Patricia Farah. Their constant and unswerving support of my endeavors was more than I could have asked for. I salute them, along with various Habibis, for being able to put up with my never ending discussions of the brain and psychopathology.

Last but not least, I would like to thank the members of my thesis committee for their time and helpful comments. Their patience is appreciated.

This research was largely funded by an NSERC grant, and partially by FCAR grant, to the thesis supervisor Dr. Jacinthe Baribeau.

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INTRODUCTION

A great deal of debate in the contemporary psychopathology literature focuses on the question of whether certain disorders, or personality types, form part of a spectrum of schizophrenia-related disorders with a common genetic origin but different phenomenological manifestations. The historical roots of this debate go back almost a century (Kotsaftis and Neale, 1993). Kraepelin took note of the abnormalities of action and thought in the relatives of schizophrenics, and of the possible relationship between dementia praecox and abnormal personality characteristics (Kraepelin, 1904). In 1911, Bleuler made comments pertaining to a continuum of schizophrenic symptoms:

> *"It is extremely important to recognize that ... [the symptoms of schizophrenia] exist in varying degrees and shadings on the entire scale from pathological to normal; also the milder cases, latent schizophrenics, with far less manifest symptoms, are many times more common than the overt, manifest cases" (Bleuler, 1950, p. 130).*

A host of other authors have made reference to the abnormal personalities of relatives of schizophrenics (for a review, see Kendler, 1985). Rado (1953) first proposed the term schizotypal to refer to the "... psychodynamic expression of the schizophrenic genotypes." Following Rado's lead, Meehl (1962) proposed the concept of schizotaxia to refer to the

"integrative neural deficit" he suggested is inherited by some of the relatives of schizophrenics. A relative who inherited schizotaxia was referred to by Meehl as a schizotype, and was believed to be susceptible to developing a syndrome ranging from well adjusted schizotypal to severe schizophrenia.

The concepts of latent and borderline schizophrenia, schizotype, along with evidence for a spectrum of schizophrenic disorders in the families of schizophrenics (Kety, Rosenthal, Wender and Schulsinger, 1968, 1975), played important roles in the development of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III, Third Edition) category of schizotypal personality disorder (SPD; American Psychiatric Association, 1980; Spitzer, Endicott and Gibbon, 1979). It should be noted that it was not the primary goal of Spitzer et al. (1979) to identify individuals genetically related to schizophrenics, but to operationalize diagnostic criteria for personality disorders (Kendler, 1985).

The concept of schizotypal personality used in this thesis is defined according to the criteria for SPD as it now appears in the revised version of the DSM-III (DSM-III-R; American Psychiatric Association, 1987), which consists of the following characteristics: magical thinking/odd beliefs, ideas of reference, constricted or inappropriate affect, unusual perceptual experiences, odd speech, odd or eccentric behavior, no close friends, suspiciousness or paranoid ideation, and excessive social anxiety. This conceptual definition was selected as it has good psychometric properties

(Kotsaftis and Neale, 1993), and is the definition most commonly referred to by researchers in the field. Because of the difficulty in obtaining large samples of subjects with diagnosed SPD, a psychometric approach to subject selection was adopted. The schizotypal personality questionnaire (SPQ, Raine, 1991) was chosen to identify subjects as it is the best available measure of the DSM-III-R criteria for SPD, in terms of its psychometric properties. To ensure convergent validity of the SPQ scale, Research Diagnostic Criteria (RDC; Spitzer, Endicott and Robins, 1975) for schizophrenic thought disorder were applied to subjects participating in the investigation. Finding that subjects scoring high on the SPQ also have prominent thought disorder on the RDC would be relevant since thought disorders are considered central features of schizophrenia.

Genetic Relationship Between Schizophrenia and Schizotypal Personality

A wealth of evidence is now available indicating that the biological relatives of schizophrenics are at high risk for developing schizophrenia themselves (Gottesman and Shields, 1982). Following the theorizing of Meehl (1962), relatives of schizophrenics may be placed at risk for the disorder by virtue of having inherited a neural abnormality which is manifested as a schizotypal personality.

Numerous studies have attempted to ascertain whether there is indeed a genetic relationship between schizophrenia and schizotypal personality, by determining whether there is a greater prevalence of SPD among the biological relatives of schizophrenics than those of control subjects. As predicted, as a number of family (Baron, Gruen, Anis and Kane, 1983; Kendler, Masterson and Davis, 1985; Parnas, Schulsinger and Mednick, 1990), twin (Siever and Gunderson, 1979; Torgersen, 1985), and adoption (Kendler, 1988; Torgersen, 1985) studies have found a higher prevalence of SPD in the biological relatives of schizophrenic probands. Nevertheless, some family (Coryell and Zimmerman, 1988, 1989; Soloff and Millward, 1983), twin (reviewed in Torgersen, 1985), and adoption (reviewed in Kendler, 1988) studies have failed to find significant results.

Discrepancies in the findings have at least partially been attributed to methodological differences in such things as diagnostic criteria for SPD and population sampled (e.g., hospital patients, general population). Of particular intrigue is that evidence suggests that few schizophrenics are found when the probands under study are those with a diagnosis of schizotypal personality (Baron, Gruen, Anis and Lord, 1985; Kendler, 1985; Ritsner, Karas and Ginath, 1993), though some positive results have been emerging (Battaglia et al., 1991; Lenzenweger and Loranger, 1989; Siever et al, 1993; Thaker, Adami, Moran, Lahti and Cassady, 1993).

In may be concluded from the literature that there is a high prevalence of SPD in the biological relatives of schizophrenics when the probands under study are schizophrenics, but that there are few family members who are found to suffer from schizophrenia when the probands are persons with SPD. Some authors have suggested that this conclusion is an artifact produced by sampling limitations whereby samples of SPD probands are too small to detect the relatively rare cases of full-blown schizophrenia, but that the presence of somewhat more readily observable schizotypal characteristics are more likely to be found in families of schizophrenics (McGuffin and Thapar, 1992; Torgersen, 1985).

It should be noted, however, that the presence of SPD or schizotypal characteristics is not limited to the biological relatives of schizophrenics. The prevalence of SPD in the general population is estimated at about 2.3% to 3.0%, depending on the diagnostic instrument employed (American Psychiatric Association, 1987; Baron and Risch, 1987). Some debate has also arisen as to the specificity of SPD to schizophrenia as some studies report the presence of schizotypal characteristics in the family members of patients other than those with schizophrenia or SPD (Silverman et al., 1993). Other evidence, however, suggests that SPD reflects the familial liability to psychotic disorders other than just schizophrenia, but not affective illness (Kendler et al., 1993; Thaker, et al., 1993).

Schizotypal Personality and Biological Markers in Schizophrenia.

Given the arguments suggesting that schizotypal personality may be part of a spectrum of schizophrenia related disorders with a common genetic origin, researchers have assessed schizotypal individuals on putative markers for schizophrenia. Finding that schizotypals are characterized by a given marker for schizophrenia would further support the link between these two conditions. Given the myriad of theories pertaining to schizophrenia genesis, selecting appropriate markers for study becomes at best difficult. Nevertheless, there is growing consensus that schizophrenia results from a biologically based vulnerability that is set into motion by environmental and intrapsychic events (Fowles, 1992; Gottesman, 1991).

Numerous candidates have been proposed as biological markers in schizophrenia. Smooth pursuit eye movement (SPEM; Holzman, 1991; Iacono, 1983), a variety of biochemicals such as monoamine oxidase (Coursey, Buchsbaum and Murphy, 1979), electrodermal orienting (Öhman, 1981), as well as both behavioral and event-related potential indices of information processing dysfunction (Duncan, 1990; Nuechterlain and Dawson, 1984), have been investigated as potential markers in nonsymptomatic relatives of schizophrenic patients. Consistent with the findings for biological relatives of schizophrenics, schizotypal personality has been associated with decreased levels of plasma monoamine oxidase (Baron, Levitt and Perlman, 1980), increased cerebrospinal fluid levels of the

dopamine metabolite homovanillic acid (Siever et al., 1991, 1993), as well as SPEM dysfunction (Lencz et al., 1993; Siever et al., 1990) and neuropsychological deficits (Lyons, Merla, Young and Kremen, 1991; Spaulding, Garbin and Dras, 1989).

Historically, as well as empirically, a great deal of emphasis has been placed on defective information processing as a central etiological abnormality in schizophrenia. Bleuler (1911) suggested that the symptoms of schizophrenia, especially the loosening of associations, may be traced back to a "disconnection of associative threads". This disconnection was hypothesized by Bleuler to be the result of an organically based inability to organize lines of thought via the selectivity of attention. This line of reasoning was supported by the work of Kraepelin (1919).

Since these early reports, numerous clinical observations and self-reports have been published, all pointing towards the importance of information processing disturbance in schizophrenia (Freedman, 1974; McGhie and Chapman, 1961). Matthysse (1978) has noted that deficits in information processing are found more consistently than any neurochemical finding in schizophrenia. Such results along with a plethora of behavioral and neurophysiological studies have led many theorists to view impaired information processing as the core cognitive disturbance in schizophrenia, and has led to the study of neurobiological mechanisms of attention as possible risk markers for schizophrenia (Dawson, 1990; Mirsky and Duncan,

1986; Nuechterlein and Dawson, 1984). It follows from the above that investigations pertaining to information processing may be particularly useful in assessing the relationship between schizotypal personality and schizophrenia.

A Working Model of Normal Information Processing

Attempts at integrating the results of information processing studies of schizophrenics with knowledge of normal information processing have met with variable success. The theories of normal attention developed by Broadbent (1971) and Treisman (1969) have been particularly useful for interpreting the results of both behavioral and neurophysiological studies of attention in schizophrenia. In addition, the Broadbent-Treisman model is the only model of attention that has been cross-validated in normal subjects using neurophysiological techniques (Hillyard et al., 1973; Picton, Campbell, Baribeau and Proulx 1978).

One of the principal goals of research on attention in normal populations has been to determine the stages of information processing at which stimuli are filtered-down or selected for further processing and evaluated in preparation for a response. Broadbent (1971) has suggested that there exists an early selection mechanism labelled stimulus-set that rapidly rejects stimuli in unattended channels[1] before perceptual analysis may be completed at the response-set level. Such a mechanism would help

prevent an organism from being flooded by an overabundant input of unimportant information. Stimuli may be selected on the basis of physical properties or linguistic characteristics. This theory fits in nicely with data obtained from studies of selective attention, in particular dichotic listening studies (e.g., Averbach and Coriell, 1961; cited in Näätänen, 1992; Cherry, 1953).

In contrast to theories of "early selection", a number of authors have proposed that all incoming stimuli receive a complete perceptual analysis prior to being selected for further processing or rejection (Deutsch and Deutsch, 1963, Norman, 1968). This "late selection" theory was based on evidence indicating that subjects would respond to the meaning of significant stimuli (Moray, 1959; Treisman, 1960) presented to the unattended channel.

In an attempt to integrate these "late selection" findings with Broadbent's theory, Treisman (1960) suggested that the filter served as a stimulus attenuator. Kahneman and Treisman (1984) demonstrated that neutral stimuli produce more distraction than highly significant stimuli, but a small percentage of unattended semantic stimuli is actually processed.

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[1]The term "channel" is used here to refer to the ears in which auditory stimuli are presented. This usage of the term is consistent with other selective attention studies (e.g., Hillyard, Hink, Schwent and Picton, 1973; Baribeau, 1986).

Treisman thus suggested that there are at least two stages of stimulus processing: (1) a stage of minimal analysis called channel filtering (or stimulus-set in Broadbent's terminology), and (2) stages of stimulus discrimination such as response selection.

Following Treisman's work (1960), Broadbent (1971) suggested that interference in attentional selectivity may also occur at the level of "response-set". Here, Broadbent was postulating a "pigeon-holing" mechanism in which stimuli that pass the stimulus-set attenuator are compared against memorized "templates" or "models" in memory in order to facilitate the recognition of specific task-relevant signals.

Although there is still some debate as to the relative merits of early and late selection theories, it appears that a majority of authors agree with the proposition that a strict dichotomy between early and late selection is erroneous. Instead, early and late selection are thought to form a continuum (Keren, 1976). The brain uses different mechanisms of selection in terms of the type of task it is carrying out and the amount of attention that is required (Johnston and Holcomb, 1980; Keele and Neill, 1978).

Models of Information Processing in Schizophrenia

The Broadbent-Treisman model of information processing has received a considerable amount of attention over the previous two decades as a means to interpret attention disturbances in schizophrenia. Numerous authors have proposed that schizophrenics have a focal defect in their selective filtering mechanism (stimulus-set), thereby reducing the capacity for the control of attentional selectivity, and resulting in overload of response-set mechanisms (McGhie; 1970; Lang and Buss, 1965).

In contrast, authors such as Claridge (1972) and Venables (1964) have proposed that the primary dysfunction in schizophrenia is that of a defective preattentive sensory gating or arousal mechanism. In other words, controlled aspects of selective attention are normal, but are overloaded by an inability to filter afferent sensory input. A modified gating hypothesis has been proposed by a number of authors suggesting that schizophrenics have a fundamental defect in response-set mechanisms that are either the cause or consequence of abnormal gating and vigilance (Broen and Storm, 1966; Callaway and Stone, 1960).

Based on these theoretical views of information processing disturbance in schizophrenia, and research to be reviewed below, two types of disturbance found in schizophrenics appear particularly well suited for study in schizotypal subjects: the ability to gate sensory input, and the capacity to control the selectivity of attention at the stimulus-set and

response-set levels. Thus the present investigation will seek to determine if there are abnormalities of sensory gating and/or the control selective attention in schizotypal personality.

Sensory Gating deficit in Schizophrenia

As indicated earlier, clinical observations led to the positing of a sensory gating disturbance in schizophrenia. The earliest empirical support for such a disturbance, albeit indirect, came from a study of normal subjects. Gottschalk and coworkers (Gottschalk, Haer and Bates, 1972) subjected volunteers to either overwhelming auditory and visual stimulation or LSD treatment. Both of these manipulations resulted in the cognitive functioning of the normals being acutely driven in a direction consistent with schizophrenia.

Two principal measures have been applied directly to schizophrenics in order to test the deficient gating hypothesis: (1) startle reflex gating, and (2) gating of the P50 component of the event-related potential (ERP). In both these cases, pairs of stimuli are presented with short interstimulus intervals. Presentation of the first stimulus appears to initiate an inhibitory mechanism that attenuates the response to the second stimulus (Merriam, Kay, Opler and Ramirez, 1989). Because the interval between the two stimuli is so short, it is unlikely that voluntarily controlled mechanisms of inhibition are deployed (Hoffman and Ison, 1980; Ison and Hoffman, 1983). Deficits in

sensory gating are therefore suggestive of a preattentive abnormality which may lead to cognitive overload and fragmentation in schizophrenics (Braff, 1993; Geyer and Braff, 1987). Given that the present investigation is concerned with the ERP assessment of gating rather than the startle reflex, research on the startle reflex in schizophrenia with not be reviewed. Readers interested in the startle reflex results in schizophrenics are referred to Braff et al. (Braff, Grillon and Geyer, 1992).

ERPs are extremely useful indices of information processing in that they are time-locked events, and thus allow for the filtering out of random irrelevant neuroelectric potentials. In addition, ERPs are highly attractive for research on schizophrenia because they provide a noninvasive means of assessing cognitive functions of the brain which is not possible with scans or unit recordins (Iacono, 1983, 1985). Deflections of the ERP, also referred to as components, have been associated with particular psychological events (Donchin, Ritter and McCallum, 1978; Hillyard and Picton, 1986; Näätänen, 1992). Following the nomenclature of Donchin and colleagues (Donchin, et al., 1977), N (negative) refers to a component of the ERP with an upward deflection in the waveform, while P (positive) refers to a downward deflection.

ERP Evidence for Gating Deficit in Schizophrenia

Early investigations using ERP technology found support for abnormal gating in schizophrenics in terms of abnormal ERP recovery curves, results being generally interpreted as reflecting underactivation of cortical filtering mechanisms (Butter, 1973; Shagass, 1976; 1977). The most studied component of the ERP in relation to sensory gating in schizophrenia is the P50, as assessed in what has been termed the conditioning-testing paradigm (Eccles, 1969; Freedman, Adler, Waldo, Pachtman and Franks, 1983). In this paradigm paired clicks are presented. Both the first (conditioning) and second (test) stimulus elicit a P50, but the amplitude of the P50 in response to the test stimulus, as measured relative to the preceding negativity, is considerably smaller (i.e., suppressed). In effect, when the interval between the stimuli is 500 ms, suppression may be greater than 80% in normal subjects (Freedman et al, 1987). The decrement of P50 amplitude to the second stimulus is believed to result from activation of inhibitory mechanisms initiated by the first stimulus (Eccles, 1969).

The P50 appears to be particularly attractive as a measure of sensory gating for a number of reasons. Although test-retest reliability of P50 suppression has been questioned (Boutros, Overall and Zouridakis, 1991; Jerger, Beggins and Fein, 1992; Kathmann and Engel, 1990), a number of studies have found the P50 suppression ratio (calculated as the conditioning P50 amplitude/test P50 amplitude, expressed as a percentage value) to be

reliable (Cardenas, Gerson and Fein, 1993; Waldo, Graze, de Graff-Bender, Adler and Freedman, 1987). The consistent finding of high levels of P50 suppression in normal subjects further supports the use of the P50 suppression ratio as an index of gating (Braff and Geyer, 1990; Freedman et al., 1987). Consistent with the view of sensory gating as a preattentive phenomenon, the P50 suppression ratio is also a promising measure because it appears to be unaffected by attentional manipulations (Jerger, et al., 1992; Waldo and Freedman, 1986).

Freedman and colleagues were the first to apply the conditioning-testing paradigm assessment of the P50 gating effect to the problem of schizophrenia. In their first report, auditory stimuli were presented to unmedicated schizophrenics as well as normal controls, at interstimulus intervals of 0.5, 1.0 or 2.0 seconds (Adler et al, 1982). Results revealed that maximal suppression was obtained at the 0.5 second interstimulus interval in normals (mean = 80%), while at this same speed mean suppression was 10% in schizophrenics. This finding has now been replicated in numerous investigations by Freedman's group (Freedman, Adler, Waldo, Patchman and Franks, 1983; Nagamoto, Adler, Waldo and Freedman, 1989; Siegel, Waldo, Mizner, Adler and Freedman, 1984), and independently by others (Boutros, Zouridakis and Overall, 1991; Judd, McAdams, Budnick and Braff, 1992; Schwartzkopf, Lamberti and Smith, 1993). Only one study has failed to replicate the P50 results (Kathmann and Engel, 1990). The recent finding by

Judd et al (1992) indicating that schizophrenia gating deficits are most prominent at a frontal electrode site is particularly interesting given the evidence for frontal lobe involvement in schizophrenia (Goldberg and Weinberger, 1988; Nasrallah, 1991). Previous investigations had measured the P50 solely at the vertex. In addition to the basic finding of poor P50 suppression in schizophrenics as compared to controls, no significant difference in suppression has been found between medicated and unmedicated schizophrenics (Freedman et al, 1983).

Other investigations have demonstrated that the P50 suppression deficit is found in a number of patient populations (Baker et al., 1987; Franks, Adler, Waldo, Alpert and Freedman, 1983). The deficit, however, appears to be a trait phenomenon in schizophrenics unrelated to psychotic state, while being related to clinical "florid" state or medication in other psychiatric conditions (Baker et al., 1987; Franks, Adler, Waldo, Alpert and Freedman, 1983). In addition to such state-trait considerations, biochemical and psychopharmacological studies indicate disparate neurochemical abnormalities underlying deficient P50 gating in different psychiatric populations (Adler et al, 1992; Baker et, 1990; Johnson and Adler, 1993).

It is apparent from investigations comparing schizophrenics with other patient groups that diminished sensory gating is not specific to schizophrenia and therefore may not be used as a diagnostic test. This is not surprising given that a number of authors have observed that at least some

acutely ill patients, not necessarily psychotic, are subject to feelings of being overwhelmed by the environment such that they develop a disturbance in their ability to deal with incoming stimuli (Cummings and Cummings, 1962). Findings that indicate that the P50 suppression deficit is unrelated to psychotic state in schizophrenic, as opposed to being related to clinical state in other psychotic or non-psychotic patient groups, suggests that poor gating of the P50 may be a trait marker for schizophrenia, while being only related to psychotic or other clinical states in other disorders.

Such a proposal has received some support from studies indicating that the asymptomatic relatives of schizophrenics also manifest a deficit in the gating of the P50 (Coon et al., 1993; Siegel et al., 1984; Waldo et al. 1991). In addition, it should be noted that in contrast to the P50 suppression deficit, reduced amplitude and increased latency of the P50 in schizophrenics, as compared to normal controls, appears to be a state phenomenon and thus is of limited use as a biological marker (Freedman et al., 1987).

To date, no study has been conducted in order to determine whether subjects with schizotypal personality have a P50 gating deficit as indicated by a larger P50 suppression ratio than control subjects. Thus the present investigation will employ the conditioning-testing paradigm to study P50 gating in subjects identified psychometrically as having a schizotypal personality.

Selective Attention in Schizophrenia.

In addition to the growing body of literature implicating a sensory gating deficit as possibly etiologically relevant, a considerable amount of evidence has been garnered to suggest that schizophrenics have abnormalities of controlled selective attention mechanisms. While an abnormality of preattentive mechanisms may be reflected in diminished sensory gating, abnormalities of the controlled selective aspects of attention, such as selective filtering, may be ascertained from disturbances of performance on selective attention tasks. Both behavioral and ERP measures of selective attention control have been applied in the attempt to elucidate the pattern of selective attention disturbances in schizophrenia. It should be noted that, since the tasks to be employed in the present investigation are both auditory, and auditory deficits and hallucinations in schizophrenia have been subject to extensive theorizing and exploration, only auditory selective attention will be discussed.

Models of selective attention in normals have been subject to extensive investigation using focused attention tasks. In focused attention (or shadowing) tasks, subjects are required to attend to stimuli being presented to one ear while ignoring stimuli presented to the other ear. Focused attention tasks, therefore, are useful in assessing both the ability to maintain a focus on one source of sensory input, and the ability to ignore distracting stimuli coming from other sources. Slower reaction times to

target stimuli in the attended channel or increased error rates are considered to reflect increased distractibility between channels and/or poor capacity to discriminate targets (Broadbent, 1971).

Behavioral Studies of Selective Attention in Schizophrenia

Payne, Hochberg and Hawks (1970) were the first to apply a selective attention paradigm to schizophrenic patients and normal controls, in the context of testing their "broken filter" theory. This theory was similar to Broadbent's idea of the stimulus set. They presented verbal stimuli to both left and right ears at a rate of 50 words per second. It was hypothesized that schizophrenics will be characterized by increased distractibility as reflected by the incorporation of words from the unattended into the attended channel. Results revealed a greater number of shadowing errors in acute/subacute schizophrenics when distractors were present, but the number of intrusions (words from unattended ear reported) was not significantly different. These results have now been replicated a number of times, though these later studies did report increased intrusions in many groups of acute and subacute schizophrenics (Dykes and McGhie, 1976; Hawks and Robinson, 1971; Spring, Lemon, Weinstein and Haskell, 1989).

Studies by both Wishner and Wahl (1974) and Wahl (1976) suggest that speed of presentation may be an important moderating variable on schizophrenic attention dysfunction. These studies report a greater number of intrusions in the schizophrenic as opposed to control groups, as well as increased shadowing errors and omission errors on recall, as speed of presentation is slowed.

One may conclude from the focused attention studies that clinical observations of increased distractibility in acute/subacute florid schizophrenics are correct. In addition, speed of presentation may be an important moderating variable in assessing attention abnormalities, whether they be at the level of stimulus or response set (Baribeau, 1986; Broen, 1976). The weight of this conclusion must be tempered however by limitations of the behavioral technique. The question of whether distraction occurs at the level of stimulus- or response- set remains unanswered because reaction to distractor stimuli are not assessed directly. A second criticism lies in the observation that there are no methodological controls over degree of arousal or vigilance. One cannot determine whether the abnormally high degree of distractibility seen in schizophrenics is truly due to a filter defect given that a more engaging task usually results in increased arousal and effort (Broadbent, 1971; Eysenck, 1982). In order to be certain that level of arousal is the same during the processing of target and distractor stimuli, attended and ignored stimuli must be assessed in the

same experimental condition. The use of ERPs to simultaneously reflect the fate of attended and unattended stimuli allows for this desired control over arousal. A further limitation is that motor or verbal responses, external to the subject's brain, are used to infer the internal processing of stimuli. The brain processes triggered by a stimulus, leading to a response, remain unknown. Thus while a number of significant of significant findings have been reported, interpretation of exactly which mechanism is most salient remains unclear.

Neurophysiological Study of Selective Attention in Schizophrenia.

ERP components that have received particular attention as potential indices of stages or processes of selective attention in normals, have also been subjected to investigation in schizophrenics. Because of evidence indicating that particular patterns of ERP components elicited by selective attention tasks differentiate schizophrenics from normals, other patient populations, and between schizophrenic subtypes, only components that play a role in this pattern will be assessed in the present investigation.

As the primary focus is on selective attention, the bulk of the following review focuses on components related to early (N1-P2) and late (P3b) aspects of selectivity. In addition, both the N2b-P3a and slow negative wave (SNW) are elicited in binaural selective attention tasks. These components fall within ERP patterns that differentiate schizophrenics from other

populations, and differentiate schizophrenic subgroups (Baribeau, 1986; Pritchard, 1986).

ERP Correlates of Selective Attention in Schizophrenia

Disturbances of selective attention in schizophrenia, as manifested in abnormalities of ERP components, have been demonstrated in a number of investigations. Although the primary concern of the present review are studies employing selective attention paradigms, some mention will be made of investigations employing variants of the standard "oddball" paradigm because: (1) they offer further support for ERP abnormalities in schizophrenia; (2) a channel filtering ERP paradigm has yet to be employed with schizotypal subjects, while oddball studies have been.

Early Selective Attention

Theoretical arguments suggest that schizophrenia may be associated with a deficit in the ability to selectively filter sensory input. Considerable empirical work has gone into determining when and where selective filtering is first reflected in the ERP in normal subjects. Although there is still some debate as to the earliest ERP component subject to attention modulation (eg., Connolly, Aubry, McGillivary and Scott, 1989; Hackley, 1993), numerous investigations have pointed to the N1 component (Baribeau and Laurent, 1993; Näätänen, 1992; Picton et al., 1978).

Picton, Hillyard and colleagues have been credited with the first valid demonstration of a selective attention effect on the N1 (Hillyard, Hink, Schwent and Picton, 1973; Picton and Hillyard, 1974). The paradigm consisted of a signal-detection task in which rare high-pitched tones, in a train of frequent low-pitched tones, served as targets (the so-called "oddball paradigm"). Frequent and rare tones were presented binaurally, in a sequential but random order, to both ears of subjects. Subjects were required to attend to tones in one ear, and simultaneously ignore the other ear. Earlier investigations had failed to present stimuli randomly, thus resulting in artifacts produced by anticipatory evoked-potentials and changes in phasic arousal (Näätänen, 1975; Hillyard and Picton, 1979). Furthermore, unlike previous studies, Picton and colleagues employed very short ISIs to force subjects to focus on relevant channels. Results revealed that both target and nontarget stimuli in attended channels elicited an N1 of significantly greater amplitude and shorter latency than stimuli in unattended channels. This "N1 effect" was interpreted by Hillyard et al. as reflecting Broadbent's (1971) stimulus-set mode of attention. The lower amplitude of the N1 in the unattended channel was taken to signify that ignored stimuli were rejected or attenuated from further processing. These results have now been reliably replicated on a number of occasions (for reviews, see Näätänen, 1982; Näätänen and Picton, 1987; Picton, et al., 1978). In addition, the use of short ISIs has proven to be critical in the elicitation of the N1

effect, whereby N1 is significantly greater with short ISIs and may be small of absent with long ISIs (Parasuraman, 1978; Schwent, Hillyard and Galambos, 1976).

Based on this evidence in normals, investigations have been carried out in the attempt to determine whether schizophrenics have an abnormality of N1 attention modulation. Baribeau and colleagues (1983), employing the paradigm developed by Hillyard et al. (1973), were the first to study ERP correlates of auditory selective attention in schizophrenia. Their results revealed that chronic and sub-acute schizophrenics manifest a deficit in the control of selective attention when required to focus their attention, as manifested by a reduced N1 amplitude. Of particular significance was the finding that this N1 abnormality manifested itself in the schizophrenic group when speed of stimulation was slow (mean ISI = 1 second), but normalized when speed of stimulation was fast (mean ISI = 0.5 seconds). These results have now been replicated a number of times in Canadian and French laboratories (Baribeau and Laurent, 1986; Laurent and Baribeau, 1992) as well as in Japan (Hiramatsu et al., 1983; Saitoh et al., 1984), and have been interpreted as reflecting a stimulus-set dysfunction at slow ISIs (Baribeau, 1986; Saitoh et al., 1984). These results indicate that attentional processes in less florid chronic schizophrenics are especially prone to break down when ISI is long, and that this may be due to an inability to maintain selectivity. In contrast, florid subacute and acute schizophrenics loose

channel selectivity when ISI is short (Baribeau, 1986; Pritchard; 1986; Spring et al., 1991).

Research has generally failed to demonstrate a separate or distinct effect of selective attention on the amplitude of the P2 (Hillyard et al., 1973). Nevertheless, the P2 may be somewhat larger in attend than ignore conditions (Näätänen, 1992). Given the subtle attention effect, P2 has often been measured in concert with the N1 as a N1-P2 complex representing stimulus-set type processing (Hillyard and Picton, 1986). The present investigation will follow this use of the N1-P2 complex as its indicator of early selective filtering.

Late Selective Attention

Clearly the most frequently studied ERP component in schizophrenia is the P3b. The P3b is elicited by task-relevant stimuli, such as rare target tones in a train of frequent standard tones (Sutton, Barren, Zubin and John, 1965; Sutton, Tueting, Zubin, John, 1967). P3b amplitude has been argued to reflect the updating of working memory (Donchin, 1981; Donchin and Coles, 1988; Karis, Fabiani and Donchin, 1984). Recent reviews by Johnson (1986, 1992) suggest that P3b amplitude is a function of subjective probability, stimulus meaning (e.g., incentive value), and the certainty with which information presented is perceived by the subject. There is no unitary or invariant significance of the P3b, and no single hypothesis has received

universal acceptance (Pritchard, 1981; Verleger, 1988), while the above factors are reliably shown to affect it.

The vast majority of investigations have reported that schizophrenics manifest reduced P3b amplitude and delayed latency to target stimuli in oddball paradigms (Faux, Torello, McCarley, Shenton and Duffy, 1988; Mirsky and Duncan, 1986; Pfefferbaum, Ford, White and Roth, 1989; Pritchard, 1986). Reduced P3b amplitude appears to be unrelated to medication (Laurent and Baribeau, 1992; Harvey and Pedley, 1989), and is robust under a variety of task demands (Baribeau et al., 1983; 1986; Pfefferbaum et al., 1989; Stranburg, Marsh, Brown, Asarnow and Guthrie, 1984).

Evidence from selective attention research has been interpreted by Hillyard et al. (1973) and others (e.g., Baribeau, 1986; Hillyard and Picton, 1986), as suggesting that P3b is a reflection of Broadbent's (1971) response-set stage of attention. Hillyard and colleagues (Hillyard et al., 1973; Picton and Hillyard, 1974), using binaural listening tasks, reported a large P3b to target tones in channels that were attended to, but only a small or nonexistent P3b to targets in unattended channels.

Based on the selective attention effects on the ERP, it appears that the N1 and P3b form two components of a hierarchical selection mechanism, consistent with the Broadbent-Treisman model of stimulus-set and response-set (Hillyard et al., 1973; Hillyard and Picton, 1986). It should be noted, however, that a hierarchical mechanism of selective attention does not rule

out a parallel processing of stimuli within separate channels (Alain, Richer, Achim and Baribeau, 1991; Baribeau and Laurent, 1993).

Given that response-set has been hypothesized as disturbed in schizophrenics (Broen and Storm, 1966; Callaway and Stone, 1960; Hemsley, 1975), a number of investigations of the P3b elicited during selective attention have been carried out. Results have generally supported this hypothesis, P3b amplitude being diminished in schizophrenics when they are required to selectively attend to one or more channels. In the study of Baribeau et al. (1983), P3b amplitude was smaller and latency longer than for normal controls across speeds of presentation and attention conditions. Given that chronic schizophrenics only manifest N1 abnormalities at slow speeds of stimulation, these results suggest that the N1 and P3b abnormalities seen in schizophrenics are at least to some degree independent disturbances. P3b abnormality in auditory selective attention paradigms has been replicated in a number of samples (Baribeau and Laurent, 1986; Laurent and Baribeau, 1992; Hiramatsu et al., 1983).

Employing similar selective attention paradigms to that applied to schizophrenics, Baribeau and colleagues have demonstrated that, although behavioral performance is disturbed in all groups, the pattern of N1-P3b abnormalities is different under speed and attention conditions for schizophrenics, psychotic depressives (Baribeau and Lesèvre, 1983), sons of alcoholics (Baribeau, Ethier and Braun, 1987), and closed head injury

patients (Baribeau, Ethier and Braun, 1989). These results are important in indicating that selective attention abnormalities found in schizophrenics are not simply the result of motivational differences, and that patterns of normal and abnormal ERPs are more useful for differentiating subject groups, with similar behavioral performance, than single components. The issue of schizophrenia specific signs must be understood in terms of patterns of ERPs rather than single features.

N2b-P3a

The N2b may be elicited in sensory discrimination tasks like the oddball paradigm, in response to deviant stimuli (Ford, Roth, Dirks and Kopell, 1973; Ford, Roth and Kopell, 1976a, 1976b). The N2b has been found to occur prior to overt responding and its latency is affected by the difficulty in discriminating stimuli (Ritter, Simson and Vaughan, 1983).

The N2b appears to reflect a more automatic than controlled detection of a mismatch when an infrequent stimulus occurs after a series of frequent standard stimuli (Loveless, 1983; Näätänen, 1992). The N2b has been found to be sensitive to deviance on a multitude of dimensions that stimuli may take (Polich, McCarthy, Wang and Donchin, 1983; Ritter, et al., 1983), and to reflect deviation from a "centrally maintained expectancy" (Hoffman, 1990) or transient arousal and the orienting response (Loveless, 1983; Näätänen and Gaillard, 1983). Finally, the N2b is much larger in amplitude to attended

stimuli (Näätänen and Gaillard, 1983). This point must however be qualified by the observation that an N2b may be produced in ignored conditions when stimulus deviation is wide, thus reflecting automatic processes (Näätänen, Simpson and Loveless, 1982).

This last observation is supported by evidence indicating that the N2b precedes the P3a elicited by rare or novel stimuli in attend or ignore conditions (Courschesne et al., 1978; Grillon, et al., 1990; Näätánen, 1992; Squires et al., 1975). Numerous studies have observed a close temporal association between the N2b and P3a (Courschesne, Hillyard and Galambos, 1975; Loveless, 1983; Renault and Lesèvre, 1979). Thus some authors (e.g., Näätänen, 1992) have suggested that these two components are part of a N2b-P3a complex reflecting stimulus detection and/or orienting in the Sokolovian sense (Sokolov, 1975).

The N2b-P3a has not received much attention by schizophrenia researchers, but abnormalities have been reported. Baribeau and colleagues (Baribeau and Laurent, 1986, 1992), employing a selective attention paradigm, demonstrated that schizophrenics have reduced amplitude of the N2b in response to stimuli on which they were required to have focused their attention, but that this finding became nonsignificant when the stimuli were actively ignored. This finding is supported by a report indicating reduced amplitude and increased latency of the N2b in chronic schizophrenics during a focused attention auditory oddball task in which subjects were required to

count the target stimuli (O'Donnell et al., 1993). Finally, some studies have reported the P3a amplitude to salient or novel stimuli differs between chronic but not acute schizophrenics and normals (Baribeau and Laurent, 1992; Grillon, Courschesne, Elmasian and Braff, 1990).

SNW

The final component of interest is the slow negative wave (SNW). The SNW is a broad waveform in the ERP that begins roughly at about the same time as the P3, and peaks somewhere around 450 and 750 ms after stimulus onset at frontal sites (Pritchard, 1981). The SNW has been reported to occur in response to attended deviant stimuli in oddball tasks, and has been associated with further processing of stimuli following a response or decision (Rohrbaugh, Syndulko and Lindsley, 1979; Sutton and Ruchkin, 1984).

Baribeau and Laurent (1986; 1992) have demonstrated that chronic and sub-acute schizophrenics with florid content thought disorders manifest an increased SNW at frontal electrode sites during focused and divided attention conditions. This finding was interpreted as suggesting that these schizophrenics are characterized by abnormally long processing of significant stimuli, perhaps due to delusionary pathology. Enlarged late frontal negativities to target tones have been reported in other investigations of schizophrenics (Cohen et al., 1990; Michie, et al., 1990).

The issue subtyping schizophrenics cannot be tested in the present thesis with schizotypals. This is because there is no consistent evidence in the literature to distinguish subgroups of schizotypals, and the sample size employed makes statistical differentiation of potential subgroups unfeasible. Consequently, the strategy used in the present thesis is simply to use dependent variables (ERPs) which may be sensitive to particular subtypes of schizophrenia, and allowing the ERPs to manifest themselves in the results as a pattern of ERP modulations. If results suggest that the pattern of ERP results in schizotypals is similar to that found in a particular subtype of schizophrenia, the pattern of ERPs will qualitatively analyzed on a one-to-one basis for each subject.

Information Processing Links between Schizotypal Personality and Schizophrenia

To date, no ERP study has been conducted to determine whether, like schizophrenics, individuals with schizotypal personality are also characterized by deficient gating of the P50 and/or selective attention deficits. Behavioral studies of information processing in schizotypal personality have, however, provided consistent support of an association with schizophrenia. Like schizophrenics (Braff, 1993; Nuechterlein and Dawson, 1984; Siever, 1985), both college students identified as schizotypes (Merritt and Balogh, 1990; Sterenko and Woods, 1978), and SPD patients

(Braff, 1981; Saccuzzo and Schubert, 1981), have been found to demonstrate deficits in the early stages of visual information processing as reflected in poor backward masking performance. In addition, individuals with schizotypal characteristics, as determined by the MMPI or psychosis-proneness scales, have been found to perform poorly on a number of other information processing tasks including reaction-time crossover (Chapin, Wightman, Lycaki, Josef and Rosenbaum, 1987; Rosenbaum, Chapin and Shore, 1988; Simons, MacMillan and Ireland, 1982), the continuous performance task which assesses various facets of attention (Lezenweger, Cornblatt and Putnick, 1991), and cross-modal attention switching (Wilkins and Venables, 1992). Deficits in the sensory gating of the startle reflex in psychometrically defined psychosis-prone college students (Simons and Giardina, 1992) and SPD patients (Cadenhead and Braff, 1992; Cadenhead, Geyer and Braff, in press) has also been recently reported. This last finding further suggests that investigation of P50 gating may be fruitful.

Application of ERPs to the study of information processing in schizotypal personality has been limited. This situation is in stark contrast to the extensive and growing application of ERPs to the study of the biological relatives of schizophrenics. The vast majority of such genetic high-risk studies have only assessed the P3b component of the ERP as elicited in the oddball paradigm or its variations. Abnormalities of the P3b have indeed been reported in first-degree relatives of schizophrenics, and

has been considered by some as a potential biological marker (Blackwood, St-Clair, Muir and Duffy, 1990; Blackwood, St-Clair and Muir, 1991; Duncan, 1990). However, a small P3b in itself is not schizophrenia-specific, thus the absolute amplitude of P3b cannot be considered a marker. Nevertheless, the relative pattern of speed related attentional modulations of P3b appears to be schizophrenia-specific. Thus a particular pattern may be a marker, not the P3b in itself alone.

An increase in the latency of the P3b has been reported for a group of mixed borderline and schizotypal personality disorder patients (Blackwood, St-Clair and Kutcher, 1986; Kutcher, Blackwood, St-Clair, Gaskell and Muir, 1987). In subjects psychometrically defined as schizotypal or psychosis-prone, abnormalities of the P3b have also been reported (Miller, Simons and Lang, 1984; Simons, 1982; Simons and Miles, 1990). These results, however, appear to depend to some degree on the type of stimuli used and whether subjects are defined as schizotypes or psychosis-prone on the basis of their being anhedonic or some other characteristic(s).

Hypotheses for the Present Investigation

Given the evidence reviewed on schizophrenics, it appears plausible that a population putatively part of a spectrum of schizophrenia related disorders, individuals with schizotypal personality, would manifest a pattern of ERP characteristics similar to that of schizophrenics. Use of potentially medicated patient populations to study SPD leaves one concerned that the secondary effects of medication on behavioral and biological variables may be misinterpreted as pathophysiologically important (Blanchard and Neale, 1992a; Spohn and Strauss, 1989). Furthermore, secondary effects of having a chronic mental disorder may also interfere with the identification of relevant variables (Mednick and Schulsinger, 1968).

The present investigation therefore employed the psychometric method (Simons and Miles, 1990) to identify samples of subjects manifesting high or low degrees of schizotypal personality characteristics. Given that this was the first study to investigate P50 gating and ERP correlates of selective attention in schizotypals, and sample size was not large, this study was considered as exploratory. Nevertheless, the current sample size is consistent with that used in numerous published ERP investigations. Such a sample size is deemed acceptable in ERP research since measures are based on averaging and careful artifact rejection. In addition, the means obtained for each component for each subject in each condition is an average of numerous trials. Such repeated measure designs result in

considerable reduction of error variance (Hillyard and Picton, 1986).

Because finding that schizotypals are similar to schizophrenics on a single ERP component would not be sufficient evidence to suggest an association, given that single components may also be abnormal in other populations, patterns of ERP modulations were sought. Although exploratory, the following hypotheses were posited based on the chronic schizophrenia literature since the most reliable ERP patterns were found in this group:

In the sensory gating task,

Like schizophrenics, the schizotypal group will show significantly less gating of the P50 than the control group, as indicated by larger suppression ratios. This effect will be maximal at the frontal electrodes (Judd et al., 1992), and at the 0.5 second ISI.

In the selective attention task,

1. schizotypal subjects will have slower RTs than controls, a situation that will be aggravated in the slow ISI condition.
2. schizotypal subjects will have a smaller attention modulation of N1-P2, especially at the slow speed of stimulation.
3. schizotypal subjects will have longer latency and smaller amplitude P3bs in attend conditions. P3b may be somewhat larger for ignored than attended

stimuli in schizotypals but not controls, indicating distraction.

4. schizotypal subjects will manifest larger N2b-P3a to target tones in ignored conditions.

5. schizotypal subjects will manifest larger SNWs to target tones in the focused conditions than controls.

Relationship Between Components

Given that both the P50 gating and the N1 filtering effects have been suggested to reflect the early selection of stimuli, the relationship between sensory gating (P50) and controlled filtering (N1) may require clarification. The one study on the topic to date has demonstrated that P50 suppression is not, while N1 amplitude is, modulated by controlled selective attention in normal subjects (Jerger, et al., 1992). This is consistent with the formulation of P50 suppression as an automatic preattentive phenomenon and the N1 effect as related to controlled aspects of filtering.

The schizophrenia-spectrum and schizotypy literature, until now, has not specified the temporal direction of relations in reporting ERP abnormalities. By direction of such relations here we refer to the temporal relation between ERP components, and their known intracerebral relations. The present thesis investigates the relations between P50, N1 and P3, in such a manner as to uncover a pattern which would shed light on a plausible temporal direction of relations.

If indeed, as expected, schizotypals are found to manifest both P50 suppression and N1 effect abnormalities, one must ask, and determine statistically, whether poor P50 gating is significantly related to the N1 results. If one concurs with the hypothesis put forward by authors such as Claridge (1972, 1990) and Venables (1964), then abnormal sensory gating mechanisms are at the root of selective filtering deficits in schizophrenics, and perhaps also in schizotypals. Thus this hypothesis leads to the prediction of a significant relationship between P50 suppression and the N1. If on the other hand, one concurs with McGhie (1970), Lang and Buss (1965) and others, then the primary abnormality lies at the level of attentional filtering (N1) and thus any findings on the N1 should not be significantly related to the subject's sensory gating results. In addition to the relationship between sensory gating and selective filtering, Broen (1966) and Hemsley (1980, 1993) would predict that deficient sensory gating (P50) may have the consequence of producing interference at the level of perceptual response hierarchies (in Broadbentian terms, at the response-set level), and may be the cause of response-set (P3b) deficits. Thus no matter if the primary filtering deficit in schizophrenia lies at the level of P50 or N1, such an abnormality may have consequences for P3b.

METHODS

Screening and Subject Selection

The SPQ (Raine, 1991) was used to assess schizotypal personality for the present investigation. The SPQ is a 74-item forced choice (true or false) self-report inventory specifically designed to provide both an overall measure of schizotypal personality, as well as separate measures of the nine features of schizotypal personality, as defined by the DSM-III-R (American Psychiatric Association, 1987). A copy of the SPQ is presented in Appendix A.

The SPQ appears to have good psychometric properties (Raine, 1991). The SPQ contains two major factors consisting of Cognitive-Perceptual Deficits and Interpersonal Deficits (Gruzelier, Burgess, Stygall, Irving and Raine, 1993; Raine et al., 1992), and is consistent with factor structures reported for psychiatric interview data with schizotypal personality disorder patients (Kendler et al., 1991). Raine et al. (1992) report that internal reliability for the individual subscales range from coefficient alpha of 0.71 (Odd Speech) to 0.81 (Odd Beliefs/Magical thinking), with a total score reliability of 0.91. Test-retest reliability over a two-month interval is reported to be 0.82, which is comparable to the 0.44 to 0.84 reliability range found for the commonly used psychosis-proneness scales (Chapman, Chapman and Miller, 1982), and other self-report schizotypal scales (Eysenck, Eysenck and Barrett, 1985; Rust, 1987; Venables, Wilkins, Mitchell, et al., 1990).

Raine (1991) has also provided evidence for both the convergent and discriminant validity of the SPQ. The SPQ has been found to correlate highly with the STA schizotypal (0.81; Hewitt and Claridge, 1989) and schizophrenism (0.65; Venables et al., 1990) scales, supporting convergent validity. Evidence for discriminant validity is found in the low correlations between the SPQ and both the Anhedonia (0.19; Venebles et al., 1990) and Psychoticism (0.37; Eysenck et al., 1985) scales, measures reflecting characteristics that are not part of the DSM-III-R criteria for schizotypal personality disorder. To establish criterion validity, students with total SPQ scores in the top and bottom 10% of a sample of 497 students were administered the Structured Clinical Interview for DSM-III-R Personality Disorders (SCID-II; Spitzer, Williams and Gibbon, 1987) two months following completion of the SPQ. Results revealed that 55% of those scoring in the top 10% fulfilled DSM-III-R criteria for schizotypal personality disorder, while the remaining 45% of the high scorers were found to present 3-5 schizotypal traits. None of the students in the bottom 10% of the sample fulfilled diagnostic criteria.

A total of 369 students at Concordia and McGill Universities completed the SPQ along with a number of other questionnaires. The cover page of the questionnaire package briefly explained that students are needed for studies concerning various cognitive functions in individuals with a variety of experiences, feelings and beliefs. No mention was made of any specific

personality characteristic. A total of 10-15 minutes was required to complete the questionnaires. A copy of the questionnaire's recruitment strategy is presented in Appendix B.

Means and standard deviations for all nine subscales and the total score, as obtained in the screening sample are presented in Table 1. Given evidence for sex differences on the SPQ (Raine, 1992), scores in Table 1 are also presented separately for males and females. Sex differences on the subscales were analyzed using two-tailed t-tests. Significance level was set at $p \leq .01$. The sample consisted of 129 (34.96%) males and 240 (65.04%) females. Only SPQ's completed by subjects 18 - 26 years of age were only included in the data set.

In the criterion validity study reported by Raine (1991), the ten percent high and low cutoff scores on the SPQ were 41 and 12, respectively. For the present investigation, the ten percent high and low cutoff scores on the SPQ were 40 and 9, respectively. Based on these criteria, 8.4% of the sample scored in the upper ten percent, and 11.11% in the bottom ten percent.

Table 1

Means (M) and standard deviations (SD) for the Schizotypal Personality Questionaire (SPQ) in the screening sample for males (n = 129), females (n = 240) and total sample, and t-test results for sex differences on the SPQ.

		Male	Female	Total	t	P
Ideas of reference	M	3.29	3.81	3.61	2.06	.040
	SD	2.48	2.33	2.39		
Excessive Social Anxiety	M	2.81	3.74	3.38	4.05	.000*
	SD	2.19	2.20	2.24		
Odd beliefs/Magical thinking	M	2.05	2.67	2.43	2.90	.004*
	SD	2.04	1.99	2.03		
Unusual perceptual experiences	M	2.28	2.57	2.46	1.37	.171
	SD	2.04	1.99	2.01		
Odd or eccentric behavior	M	2.68	1.74	2.10	-4.13	.000*
	SD	2.23	2.10	2.20		
No close friends	M	2.54	2.18	2.32	-1.61	.108
	SD	2.34	1.85	2.06		
Odd speech	M	3.42	3.23	3.30	-0.77	.440
	SD	2.60	2.44	2.50		
Constricted affect	M	2.26	1.78	1.96	-2.70	.007*
	SD	1.83	1.83	1.68		
Suspiciousness	M	2.33	2.43	2.39	0.51	.612
	SD	1.98	1.76	1.84		
Total scale score	M	23.69	24.13	23.96	0.35	.725
	SD	12.66	10.59	11.41		
Factor 1 Perceptual/cognitive	M	13.73	14.01	13.90	0.33	.742
	SD	8.29	7.68	7.91		
Factor 2 Interpersonal	M	7.62	7.69	7.66	0.14	.886
	SD	5.18	4.54	4.79		

* $p \leq .01$.

Samples of Subjects

Students fulfilling the criteria established for the SPQ, and who were between the ages of 18 and 26, were first contacted by telephone and described the nature of the study. Subjects were reminded that the study concerned the relationship between personality and cognition but no mention was made of any specific personality characteristic. If the individual was interested in participating they were asked a brief series of screening questions. Individuals reporting that they had experienced a head injury resulting in the loss of consciousness, currently suffer from a serious medical illness, are taking medications that are known to have central nervous system effects that may confound performance evaluation in the study, have poor hearing, or who report excessive drinking or substance abuse, were excluded from participation. To encourage participation subjects who completed the study protocol received a renumeration of $15.00 or $20.00[1].

Based on these criteria a total of 11 subjects composed the schizotypal and 12 the control group, participated in the laboratory testing. There were 7 males and 5 females among the controls, and 6 males and 5 females in the schizotypal group. Although it was not possible to completely

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[1] *Because of the difficulty in obtaining subjects during the summer months, we offered $20.00 to encourage participation.*

match subjects of the two groups on a one-to-one basis, an attempt was made to match subjects as closely as possible on the basis of age, gender and handedness. Comparison of the groups on the SPQ total, subscale and factor scores is presented in Table 2. Significance level was set at $p \leq .01$. Results reveal that the groups are clearly different in terms of schizotypal personality characteristics.

To ensure convergent validity, subjects were scored on RDC (Spitzer et al., 1975) criteria for schizophrenic thought disorder. Of course, the degree of severity and frequency count could not taken into account since we are not dealing with a severely disturbed population. Thus RDC criteria were considered fulfilled at the low range of the frequency scale, with a count of 1 per item. Two-tail t-test analysis indicated a clear difference between the groups on this measure (see Table 2). Thus not only are the groups differentiated in terms of schizotypal personality characteristics according to the full scale score on the SPQ, but also according to two scales reflecting schizophrenic thought disorder, the SPQ odd speech subscale and the RDC criteria.

General Testing Procedure

Upon arrival at the Laboratory of Neuropsychology subjects completed a written informed consent form describing the procedures involved in the study (Appendix C). Subjects were explained in both written and verbal form that they may withdraw from the study at any time they desired by signalling

their desire to do so to the experimenter. Next, audiometric testing was performed with a Lafayette Instruments Co. audiometer (Lafayette, Indiana) in order to determine wether the subject's hearing was adequate to detect tones in the frequency range used in the present investigation. No subjects were eliminated from participation based on this criterion. Following completion of this examination, subjects were administered the Laboratory of Neuropsychology Subject Interview (Appendix D). This interview gathered information pertaining to demographics, personal and family history of medical and psychological disturbance, as well as alcohol and substance use.

Following the interview, a series of self-report questionnaires were administered. These questionnaires were selected to determine wether there existed any group differences in terms of handedness, characterological anxiety, or state anxiety and depression. Table 3 presents the results of two-tail t-tests comparing the two groups on various subject characteristic data obtained from the interview, as well as the self-report measures. Significance level was set at $p \leq .05$ for all variables. Results revealed that groups differed significantly only in terms of depression and state anxiety. Differences in gender composition between the groups was also not significant. The questionnaires employed are briefly described below.

Table 2

Means (M), standard deviations (SD) and t-test results for schizotypal and control groups on the SPQ and the RDC Schizophrenic thought disorder scale.

		Schizotypal	Control	t	p*
Ideas of reference (9)	M	6.72	1.17	-8.04	.000
	SD	1.62	1.70		
Excessive Social Anxiety (8)	M	6.73	1.25	-12.05	.000
	SD	1.19	.97		
Odd beliefs/Magical thinking (7)	M	5.91	.42	-11.02	.000
	SD	1.22	1.17		
Unusual perceptual experiences (9)	M	5.46	.67	-9.33	.000
	SD	1.29	1.16		
Odd or eccentric behavior (7)	M	6.00	.17	-26.36	.000
	SD	.63	.39		
No close friends (9)	M	4.73	.67	-9.28	.000
	SD	1.04	.99		
Odd speech (9)	M	7.18	1.33	-11.10	.000
	SD	1.47	.99		
Constricted affect (8)	M	3.73	1.56	-6.53	.000
	SD	1.55	.67		
Suspiciousness (8)	M	5.00	.58	-7.35	.000
	SD	1.84	.79		
Total scale score (74)	M	51.46	6.67	-31.58	.000
	SD	4.23	2.10		
Factor 1 (41) Cognitive/perceptual	M	31.36	3.75	-19.52	.000
	SD	4.08	2.42		
Factor 2 (25) Interpersonal	M	15.36	2.33	-18.81	.000
	SD	1.43	1.88		
RDC criteria (7)	M	6.18	.75	-13.01	.000
	SD	1.25	.62		

* All group differences significant at $p \leq .001$.

Note. Total score possible for each subscale, total and factor scores are indicated in parentheses.

Edinburgh Handedness Inventory (EHI). The EDI is a self-report measure of handedness. Scores vary from 0 to 100, the greater the number indicating greater right-hand dominance (Oldfield, 1971).

Beck Depression Inventory (BDI). The BDI (Beck, Steer and Garbin, 1988) is a 21-item self-report questionnaire measuring the severity of depressive symptoms. Good reliability (Beck, 1970; Reynolds and Gould, 1981) and validity (Kerner and Jacobs, 1983; Williams, Barlow and Agras, 1973) for the BDI has been established. Scores may range from 0 to 63, higher scores indicating a greater severity of depression.

Speilberger State/Trait Anxiety Inventory-Version 3 (STAI). The STAI (Speilberger, Gorsuch and Lushene, 1970) is a self-report questionnaire purporting to measure the degree of current (state) and characterological (trait) anxiety. Reliability and validity for both state and trait anxiety subscales are good (Speilberger et al., 1970). Scores on both scales may range from 20 to 80, a greater score indicating higher anxiety levels.

Following completion of these questionnaires, electrodes were placed on the subject and the neurophysiological tests administered. All subjects completed both the sensory gating and dichotic listening tasks.

Table 3

Means (M), standard deviations (SD) and t-test results for schizotypal and control groups on subject characteristic data.

		Schizotypal	Control	t	P
Age	M	22.45	22.00	-.54	.596
	SD	2.16	1.86		
Years of education	M	15.55	16.33	1.44	.166
	SD	1.44	1.16		
Handedness	M	80.25	86.31	.98	.341
	SD	17.73	10.72		
Depression (BDI)	M	13.86	2.50	-6.21	.000*
	SD	5.57	2.53		
Trait anxiety (STAI-T)	M	40.73	36.50	-1.62	.120
	SD	6.02	6.49		
State anxiety (STAI-S)	M	40.73	30.67	-2.47	.023*
	SD	7.58	11.67		

* $p \leq .05$.

General Procedures for ERP Recordings

Subjects were seated in a partially sound-attenuated and electrically shielded chamber, in which the interview and questionnaires were completed. Electroencephalographic (EEG) activity was recorded via Grass Instruments gold cup unipolar electrodes affixed to midline (Fz, Cz, Pz), left hemisphere (F3, C3) and right hemisphere (F4, C4) sites, according to the 10-20 International System (American Electroencephalographic Society, 1991; Jaspers, 1958). Linked mastoid electrodes (M1, M2) served as reference. The electrooculogram (EOG) was recorded from electrodes placed on the supra-orbital ridge of the right eye and the outer canthus of the left eye. Pilot work has demonstrated that this EOG setup is useful for recording both lateral and horizontal eye movements. A sternovertebral electrode served as electrical ground. The electrode medium employed consisted of Grass EC2 cream. Prior to placement of the electrodes, the scalp locations for electrode placement were prepared first by cleaning with rubbing alcohol followed by light abrasion. Electrode impedance was maintained below 5 Kohms. Every attempt was made to follow guidelines on disease transmission prevention, as outlined by the SPR ad hoc committee (Putnam, Johnson and Roth, 1992).

For both paradigms, stimulus presentation, monitoring and recording of EEG and EOG activity, as well as averaging, was accomplished using the InstEP Systems (Ottawa, Ontario) evoked- potential program installed on two CIARA 80386 33 MHz computers with SuperSync low radiation 3A monitors (TVM Corporation). A Metraco Diagnostic Instruments polygraph (Houston, Texas) with high and low bandpass filters set at 40 and 0.1 Hz, respectively, was employed. Gain was set at 80 000, programable gain at 2. All data was recorded as single trials and stored on 3M 120 megabyte DC2120 Mini Data Cartridge Tapes (XIMAT format) for later off-line averaging and scoring. All stimuli were presented to the subjects through SONY stereo headphones.

Sensory Gating Paradigm ERP Procedure: Specifics

Stimuli, Instructions and ERP Recording

Auditory stimuli consisted of condensation clicks with a sound pressure level of 100 decibels (dB), equivalent to approximately 65 dB sound pressure level (SPL) in our laboratory environment. Click duration was 0.1 ms. Ten clicks with a ten second intertrial interval were first presented to familiarize the subject with the stimuli. Next, a habituation phase was begun in which 32 single clicks were presented with a ten second intertrial interval. This phase was administered to allow subjects to get used to the stimuli and the manner in which they were presented. Subjects were instructed to relax and pay no attention to the clicks. Following the habituation phase, the test

phase was administered. In the test phase subjects were presented with 96 pairs of clicks which they were instructed to ignore. The interstimulus interval varied randomly between 0.5, 1.0 and 2.0 seconds. Because of evidence indicating that cortical excitability in refractory for up to 8 seconds (Fruhstorfer, Soveri and Järvilehto, 1970; Roth and Kopell, 1969), the intertrial interval was always ten seconds. Total sweep time for each trial in all phases was 2500 milliseconds (ms), including a 50 ms prestimulus baseline. A total of 2048 points per channel were recorded for each trial. Verbatim instructions for the gating study are presented in Appendix E.

In addition to being asked to relax, a number of directives were given in order to reduce artifacts in the data. Subjects were requested to refrain as much as possible from moving their body or tongue, swallowing, and most importantly blinking. In addition, subjects were provided with a "pause" button with which they could press to indicate to the experimenters that their eyes were being stained by the effort of not blinking. This pause system considerably reduced the number of trials lost due to artifacts in pilot testing. Using a SuperSync low radiation 3A monitors (TVM Corporation), during all phases of the gating study subjects were required to stare at a black fixation point centred on a white background. Trials on which the voltage of the EEG or EEG exceeded $\pm$80 μV were eliminated from averaging.

Peak Detection

Given the small amplitude of the P50, it is difficult to measure it relative to prestimulus activity. Consistent with the investigations of Johnson and Adler (1993) and Nagamoto et al (1991), conditioning and test P50's were identified as the most positive peak between 40 and 80 ms after stimulus onset. For one subject in the control group the bulk of P50's clearly occurred between 30 and 40 ms, and was therefore scored within this range. If more than one peak was identified with identical amplitude, the latter one was selected. The amplitude of all P50 responses was measured relative to the preceding negative peak. Measurement of P50 amplitudes was carried out by an investigator blind to group membership and ISI. The amplitude of the test P50 divided by the amplitude of the conditioning P50, expressed as a percentage value termed the P50 ratio, was used as a measure of sensory gating. A higher P50 ratio indicates poorer gating. As with previous investigations (Johnson and Adler, 1993; Nagamoto et al., 1991), any P50 ratio exceeding 200% was eliminated from analyses. The design for the gating study was a 2 (Group) X 3 (ISI) X 7 (Electrode) mixed within-subjects factorial design.

Selective Attention Paradigm ERP Procedure: Specifics

Stimuli and Instructions

Auditory stimuli for the binaural listening signal-detection task consisted of randomized sequences of 1500 Hz target and 1000 Hz standard tone pips, with a 20% and 80% probability, respectively. Targets and standards were identical in duration (50 ms), intensity (50 db HL) and rise and fall time (10 ms). The stimuli were delivered in a sequential and random temporal sequence, both within and between ears, to prevent subjects from anticipating which stimulus would occur next. Such an anticipation may result in an adjustment of alertness levels, the result of which would have be altered amplitudes of the ERPs.

Prior to beginning the selective attention task, subjects were familiarized with the tones. A sequence of 10 standards followed by 10 targets was presented binaurally. Subjects were then questioned as to their ability to discriminate the tones. If subjects could not make a clear discrimination, sound intensity was raised by 5 db HL, and the tone sequence was re-administered. Subjects then completed two practice tasks consisting of a slow, then fast, version of a divided attention task in which subjects were required to detect targets presented randomly to both the left and right ears. Behavioral responses to the second practice task were inspected prior to beginning the selective attention task. If at least 3 incorrect button presses to the stimuli were made, the practice task was

repeated until subjects responding was at minimum 85% accurate.

The selective attention task contained 2 attention conditions. In the focused attention condition, subjects were instructed to focus their attention on one ear and detect the occasional targets from among the frequent standards. The ignore condition was part of the focused condition in that subjects were instructed to completely ignore all stimuli being presented to the unattended ear. ERPs to attended and ignored stimuli were recorded in the same block of trials to ensure that results were not confounded by differential arousal levels. The focused attention/ignore condition was conducted for both left and right ears. Conditions were repeated at both fast and slow speeds of stimulation; a mean interstimulus interval (ISI) of 700 ms (varying from 600 to 800 ms) and 1150 ms (varying from 800 to 1500 ms), respectively. The combination of the 2 attention conditions and 2 ISIs resulted in their being 4 blocks of trials presented to subjects, each lasting between 5 and 7 minutes. Subjects also performed another perceptual task in counterbalanced order that is not pertinent for the present investigation. During all conditions, subjects were required to stare at a black fixation point centred on a white background. See Appendix F for verbatim instructions. Artifact control instructions were identical to those given for the sensory gating task.

Subjects responded to the targets in attended channels by pushing a button on a two button Logitech Serial Mouse (Logitech Inc., Freemont, CA) using their dominant hand. Hits were scored in a time window of 200 to 900 ms. Misses were defined as the absence of response in the hit window. Following the procedure used by Baribeau and Laurent (1986) to keep count of aberrant reaction times, extremely slow responses that fell beyond the hit window, between 900 and 1200 ms, were considered pseudo-misses. False alarms were considered as any response outside the hit and pseudo-miss time windows.

ERP Recording

For each attention, speed, ear condition and electrode, ERPs were averaged separately for attended and ignored targets and standards. Total sweep time for all conditions was 600 milliseconds (ms), which included a 50 ms prestimulus baseline. A total of 256 points per channel were recorded for each trial. Averaging the signal trials, the reject interval for EEG and EOG was set at $\pm$100 μV.

Peak Detection

Peak identification was conducted on paper printouts of the averaged ERPs by an investigator blind to group membership. All scoring of the data was carried out by an individual blind to group membership using the InstEP system. The following neurophysiological indices were measured for both

amplitude with respect to baseline, and latency to peak amplitude, at all electrodes for attended and ignored target stimuli: N1, P2, N2b, P3a, P3b and the slow negative wave (SNW). The N1 was scored as the most negative peak between 80 and 160 ms post stimulus onset. The P2 was considered the largest positive peak following the N1 but before the N2b. The next most negative peak, N2b, was measured between 160 and 350 ms. The P3b was measured as the most positive peak between 250 and 450 ms. The SNW was measured the most negative peak following the P3b. The P3a was measured as the first positive peak following the N2b. Because of the need to reduce the overall number of significance tests conducted, and evidence indicating that the P3a is most likely to appear at frontal electrode sites (Picton, 1992), the P3a was measured only at Fz, F3 and F4.

The design for the ERP data in the selective attention study was a 2 (Group) X 2 (EAR) X 2 (Speed) 2 (attention instruction) mixed within-subjects factorial design. Group (schizotypal, control) was the between factor, while Ear (left ear, right ear), speed of stimulation (fast, slow), and attention instruction (attend, ignore) were repeated factors.

Results

Sensory Gating Study

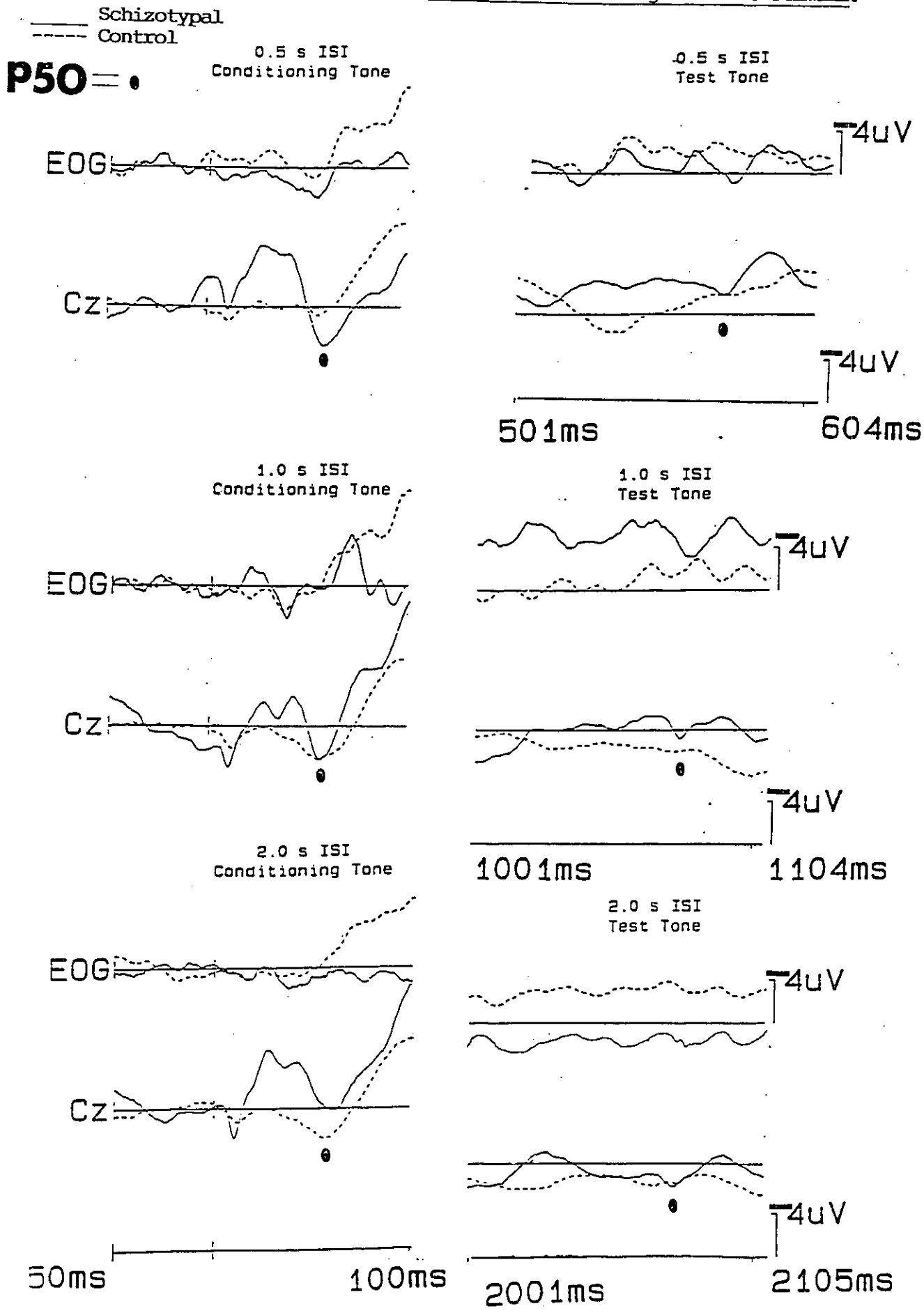
Table 4 presents the means for the P50 data. Data for one subject in the schizotypal, and two controls, was eliminated due to excessive myogenic artifacts. Thus the data analysis was carried out on 10 subjects per group. Analysis of P50 suppression data was carried out using a Group X ISI X Electrode mixed factorial AVOVA using the MANOVA program in SPSSx (Norusis, 1992), ANOVA with repeated measures being a simplified version of MANOVA (Tabachnick and Fidell, 1989). Given that testing multiple comparisons may result in the increased probability of Type I error, significance level was set at $p \leq .01$. No significant results were obtained for either main effects or interactions, indicating that suppression of the P50 by schizotypal was as great as controls. ANOVA summary table for P50 suppression are presented in Appendix G. Figure 1 illustrates the waveforms obtained for the two groups in the sensory gating task.

Table 4

Means (M) and standard deviations (SD) for schizotypal and control groups on P50 suppression ratio as a function of interstimulus interval (in seconds)

		Schizotypal			Control		
		0.5	1.0	2.0	0.5	1.0	2.0
Fz							
	M	76.67	46.23	58.26	40.41	39.69	64.19
	SD	56.72	29.81	20.99	34.62	32.46	49.51
Cz							
	M	72.16	81.12	69.73	62.18	48.73	81.36
	SD	51.23	62.95	43.42	64.05	41.34	60.47
Pz							
	M	67.47	66.05	93.25	33.13	60.73	88.10
	SD	58.79	56.73	47.91	40.38	33.00	79.13
F3							
	M	80.87	64.00	68.00	49.04	46.74	53.91
	SD	70.81	46.56	52.85	43.22	34.83	50.90
F4							
	M	90.12	71.91	73.71	41.10	64.02	81.24
	SD	61.99	33.70	47.62	32.01	36.33	54.60
C3							
	M	86.86	85.75	62.61	60.51	70.30	56.13
	SD	64.48	56.53	57.25	61.49	61.12	28.12
C4							
	M	80.72	59.53	82.45	54.46	51.54	83.18
	SD	49.73	48.39	42.07	44.05	34.61	49.79

Figure 1. P50 obtained at the 3 ISIs for conditioning and test stimuli.



Selective Attention Study

Behavioral Data

Table 5 presents means for reaction time and number of hits, misses, pseudo-misses, and false alarm data. Behavioral data were analyzed using a Group X Speed mixed factorial ANOVA with significance level set at $p \leq .01$ (SPSSx; Norusis, 1992). The only significant results were speed main effects indicating more hits at the slow speed (mean = 24.22 vs 23.55), $F(1, 21) = 11.35$, $p = .003$, and less misses (mean = 0.13 vs 1.13), $F(1, 21) = 32.77$, $p = .000$. ANOVA summary table for behavioral data are presented in Appendix H.

ERP Data

Means and standard deviations for all ERP components elicited by targets are presented in Tables 6 to 11 for latencies, and 12 to 16 for amplitudes, except for P3a which has both it's latencies and amplitudes in Table 9. Given the bulk of these tables, they are presented in Appendix N. Because of the limited sample size and large number of dependent variables, the following strategy was taken in order to reduce the overall number of statistical comparisons performed. N1 and P2 are often considered as one component and are frequently associated conceptually in the ERP literature as reflecting early selective stimulus processing (Näätänen, 1992; Näätänen and Picton, 1987), and N2b and P3a as reflecting detection of stimulus deviance or orienting (Courchesne et al, 1975; Renault and Lesèvre, 1979).

Table 5

Means (M) and standard deviations (SD) for schizotypal and control groups on reaction times (in ms) and number of hits, misses, pseudo-misses and false alarms.

	Schizotypal				Control			
	Left		Right		Left		Right	
	Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Reaction Times								
M	500.14	517.61	482.30	506.63	474.33	464.23	473.76	470.21
SD	59.75	60.26	49.82	72.02	48.12	57.55	49.88	57.03
Hits								
M	22.91	23.73	23.27	23.82	23.92	24.42	24.08	24.92
SD	2.26	1.74	2.05	1.83	1.00	.52	.79	1.83
Misses								
M	1.36	.27	1.09	.27	1.08	.17	1.00	.00
SD	.92	.47	1.14	.47	1.00	.39	.85	.00
Pseudo-misses								
M	.09	.55	.18	.36	.00	.17	.00	.08
SD	.30	.82	.41	.67	.00	.39	.00	.29
False alarms								
M		.55	1.27	.55	.42	1.17	.50	.92
SD	1.51	.52	1.35	.93	.52	1.40	.52	1.44

Thus Group X Ear X Speed X Attention MANOVA's with repeated measures on non-grouping factors were conducted on the N1-P2 and the N2b-P3a. For each of these pairs of components MANOVAs were only conducted at the optimal electrode sites determined by the schizophrenia literature. We will confirm that optimal sites are indeed optimal in our results by presenting midline electrodes where needed for graphic demonstration. In the figures the reader can see that the midline electrode is often maximal in terms of the absolute amplitude of peaks. However please note that our hypotheses are about relative differences between conditions. This is why, in the present study, it was advisable to test only optimal electrodes which were defined as the ones where the most significant differences are predicted in the literature. Numerous investigations (Baribeau, 1986; Baribeau and Laurent, 1986, Laurent and Baribeau, 1992; Hiramatsu et al., 1983) have demonstrated that attention abnormalities are maximal at frontal electrodes and lateral electrodes. Thus in order to minimize the number of tests of significance, only the lateral central and frontal electrodes were tested, and electrode site was not considered a factor. N1-P2 was assessed at lateral electrodes, and N2b-P3a were assessed at frontal (F3, F4) electrodes only.

Given the power of MANOVA with repeated measures to take into account the correlation between dependent variables, and reduction in the probability of Type I error resulting from a smaller number of comparisons, a significance level of $p \leq .01$ was adopted for the analyses. The multivariate index Pillai's criterion was used to assess the null hypotheses given it's robustness when sample sizes are unequal (Tabachnick and Fidell, 1989). Since it is uncommon to associate P3b or the SNW in single analyses with any of the other ERP components assessed in the present investigation, these two components were analyzed using mixed factorial AVOVA (SPSSx; 1992) with significance level set at $p \leq .01$. In order to reduce the number of significance tests, and given that P3b abnormalities in schizophrenics are often reported as maximal at frontal and central electrode sites, P3b was assessed at F3, F4, C3 and C4. SNW was assessed at frontal electrodes (F3, F4) where it is maximal. Given the number of significant findings, and for sake of clarity, only probability values are indicated in the text. Results were considered trends at $p \leq .05$. See MANOVA and ANOVA summary tables for ERP data in Appendices I and J, respectively, for full information. Results reported are only those not confounded by higher-order effects.

Effects not involving Group as a factor

No significant results were obtained for the Ear and Attention main effects, or Ear X Attention and Ear X Speed interactions. This held true for both latency and amplitude data

Speed Main Effects

No significant latency effect emerged. N1-P2 amplitude was larger at F3 (p = .008) and F4 (p = .021) at slow speed (F3, mean = 9.89 μV vs 8.25 μV; F4, 9.28 μV vs 7.75 μV). In addition, N2-P3a amplitude at F3 (p = .002) was larger at fast speed (Fz, 5.72 μV vs 4.65 μV; F3, 5.61 μV vs 4.51 μV).

Speed X Attention Interactions

Generally negativities (N1, N2b and SNW) were faster in latency at fast speed, and more strikingly during attention. SNW latency was found to be shorter to attended targets at F3 (p = .045) when speed was fast (mean = 367 ms vs 383 ms) relative to the ignored condition. No amplitude effects were found.

Ear X Speed X Attention Interactions

No significant effects on latency were found. Trends were found for amplitude data were found for N1-P2 at C3 (p = .026) This interaction indicated that N1-P2 amplitude was larger for attended stimuli in the right ear - slow speed (mean = 10.05 μV vs 8.15 μV), but smaller in right ear at fast speed (mean = 8.97 μV vs 7.64 μV) and left ear - slow speed (mean = 10.44 μV vs 6.86 μV) conditions. Although not tested statistically at all electrodes, the general pattern of N1-P2 modulations were consistent with the literature.

Effects involving Group as a factor

No significant Group X Ear, Group X Attention or Group X Ear X Attention interaction were found. This held true for both latency and amplitude data.

Group Main Effect

A trend for a Group main effect on latency was found for N1-P2 at F4 (p = .041), indicating that schizotypals had shorter latencies than controls irrespective of the level of the repeated factors (mean = 160 ms vs 172 ms). No significant results were obtained for amplitude data.

Group X Speed Interactions

N1-P2 latency was found to be shorter for schizotypals at both F3 (p = .009) and C3 (p = .039), both only at the fast speed of presentation. N2-P3a latency at F3 (p = .012) was also found to be shorter for schizotypals than controls at fast (mean = 263 ms vs 288 ms), but longer at slow (286 ms vs 259 ms) speed. A trend was also found for the SNW at F4 (p = 044). The SNW effect indicates that schizotypals had shorter SNW latency for slow but not at fast speed of presentation. No significant effects were found for amplitude data.

Group X Ear X Speed Interactions

No significant effect on latency was observed. At C3, N1-P2 amplitude was revealed a trend (p = .029) indicating that schizotypals had smaller amplitudes at both fast (mean = 7.83 μV vs 8.78 μV) and slow (mean = 8.56 μV and 9.64 μV) speed to right ear targets. N2-P3a amplitude, was significant at F4 (p = .008) indicating that schizotypals had larger amplitudes to left ear stimuli at both fast (mean = 7.22 μV vs 3.58 μV) and slow (mean = 5.09 μV vs 3.45 μV) speed, and larger amplitudes in the right ear for fast (mean = 8.82 μV vs 5.10 μV) but not slow (mean = 6.45 μV vs 5.66 μV) speed.

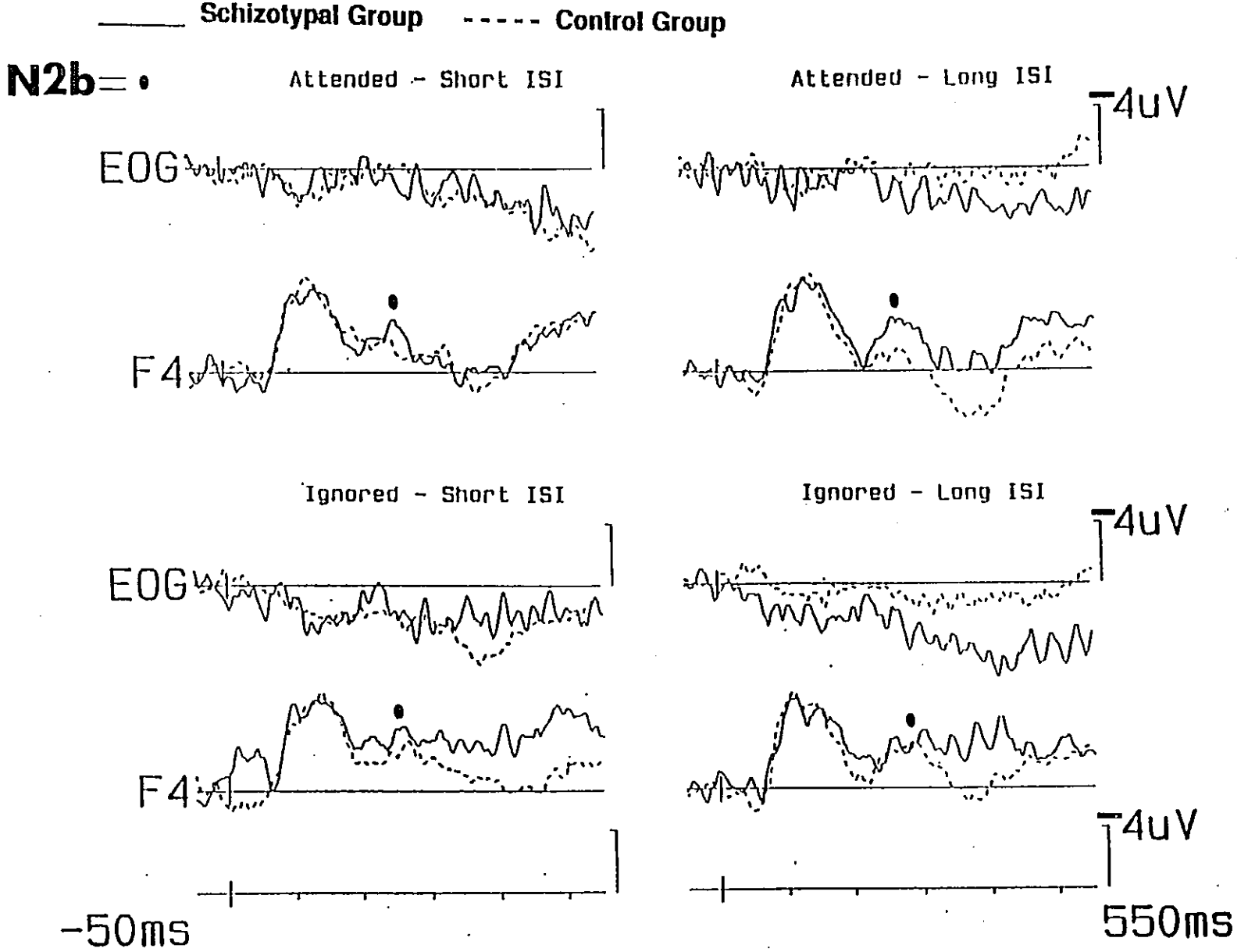
Group X Speed X Attention Interactions

P3b latency at F3 was significantly shorter for schizotypals (p = .007) in the fast attend condition (mean = 343 ms vs 391 ms), but not the fast ignore (mean = 392 ms vs 388 ms), slow attend (mean = 378 ms vs 387 ms) or slow ignore (mean = 379 ms vs 392 ms) conditions. A trend was also found for N1-P2 amplitude at C3 (p = .027) indicating that schizotypals had smaller amplitudes than controls for the fast attend (mean = 6.25 μV vs 7.1 μV) and slow ignore (mean = 8.93 μV vs 10.42 μV) conditions. In addition, N2-P3a amplitude at F4 revealed a trend (p = .035) indicating that schizotypals had larger amplitudes at slow speed for both attend (mean = 5.51 μV vs 2.84 μV) and ignore (mean = 6.03 μV vs 3.33 μV) conditions relative to controls, but not at fast speed for either attend (mean = 6.34 μV vs 5.92 μV) or ignore (5.25 μV vs 4.65 μV) conditions (Figure 2). While both groups showed the expected attention effect on N2b-P3a, the schizotypals also demonstrated an intrusion effect at slow speed, as indicated by larger amplitude in the ignore than attend condition, thus demonstrating distraction to task-irrelevant targets.

Group X Ear X Speed X Attention Interactions

None were found for latency data. In contrast, numerous effects were found for amplitude data. A trend towards significance was found for N1-P2 amplitude at C4 (p = .025). This finding indicates that schizotypals had

Figure 2. **Group differences on N2b-P3a amplitude by function of attention (attend, ignore) and ISI (short, long).**

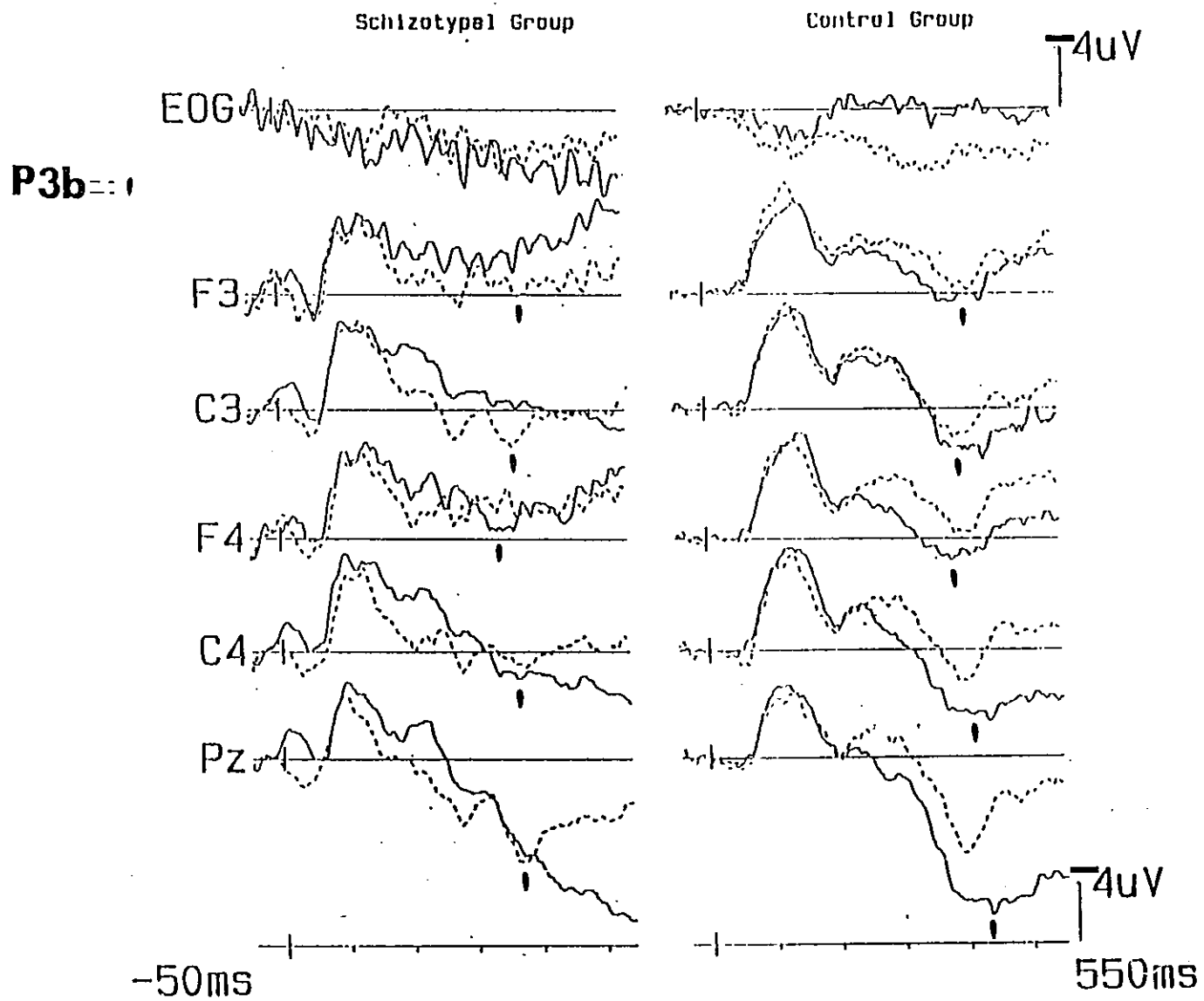


smaller N1-P2's than controls across all factors to right ear stimuli (fast attend, 6.89 μV vs 7.88 μV; fast ignore, 7.85 μV vs 9.25 μV; slow attend, 8.19 μV vs 9.75 μV; slow ignore, 7.09 μV vs 10.44 μV). For left ear stimuli, schizotypals had smaller N1-P2 amplitudes for the fast attend (mean = 7.25 μV vs 8.05 μV) and slow ignore (mean = 8.83 μV vs 11.25 μV) conditions, but not the fast ignore (mean = 7.21 μV vs 8.05 μV) or slow attend (mean = 10.62 μV vs 10.90 μV).

Significant interactions on P3b amplitude were found for the F3 (p = 012), C3 (p = .007) and C4 (p = .015) electrodes. P3b amplitudes were in general smaller for schizotypals at all electrodes, and for nearly all conditions. Results revealed that schizotypals had larger P3b amplitudes than controls only for the left ear - fast speed ignore (LFI) and right ear - fast attend (RFA) conditions for both F3 (LFI, mean = 2.50 μV vs 1.73 μV ; RFA, 3.67 μV vs 3.00 μV) and C3 (LFI, mean = 3.97 μV vs 3.79 μV; RFA, 5.53 μV vs 5.42 μV), and the RFA condition at C4 (mean = 5.86 μV vs 5.77 μV) electrodes. The larger P3bs in schizotypals in the LFI condition appears to reflect intrusion of ignored stimuli. That is, amplitudes for the ignored stimuli is larger than that for attended stimuli in the same condition as evidenced by the larger ignore versus attend mean amplitudes. Figure 3 illustrates the intrusion effect in the LFI condition as obtained at C3. This was also observable in single schizotypal tracings (Appendix L).

Figure 3 Group Differences on P3b amplitude in attend and ignore conditions

_____ Attended Tone ----- Ignored Tone



Significant interaction effects on SNW amplitude were found for F3 (p = .015), and F4 (p = .035). In general, schizotypals had much larger SNWs than controls across factors at all electrodes. This effect was particularly prominent in the right ear - fast speed ignored (RFI) condition, as indicated by significant simple effects at F3 (p = .000) and F4 (p = .000). Figure 4 illustrates the large SNW in schizotypals as opposed to controls at frontal electrodes.

Figure 5 provides a composite diagram of the ERP effects at optimal electrodes for both short and long ISI, for the two groups. Amplitude effects were found at the same electrodes as latency effects, except for the following two points. Since SNW latency was most prominent at F4 but amplitude at F3, and similarly, since the N2b-P3a latency effect was prominent at F3 but amplitude at F4, data points are presented for both electrodes.

Figure 4. Group differences in SNW amplitude to attended tones in the right ear presented at the short ISI.

_____ Schizotypal Group - - - - - Control Group

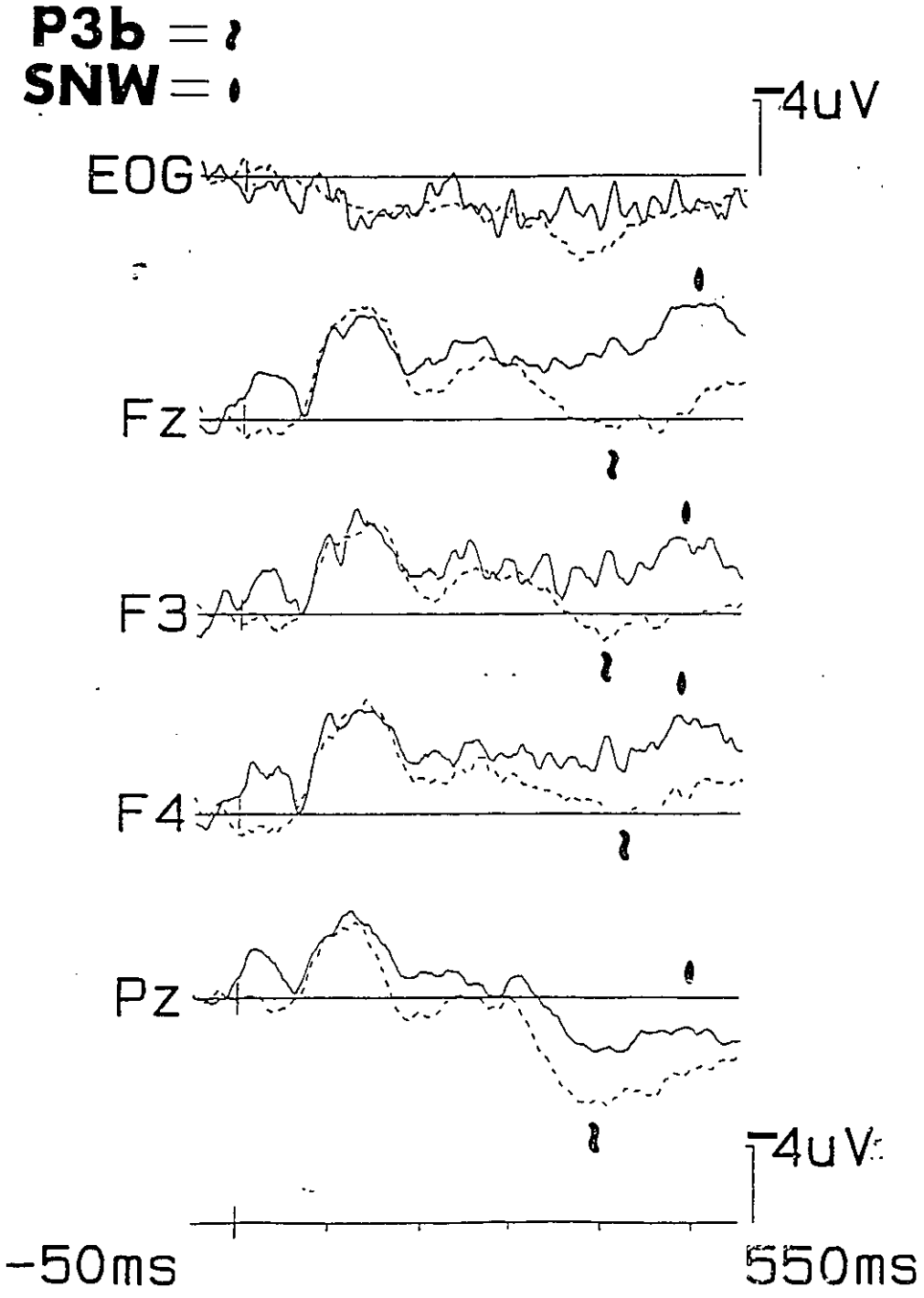
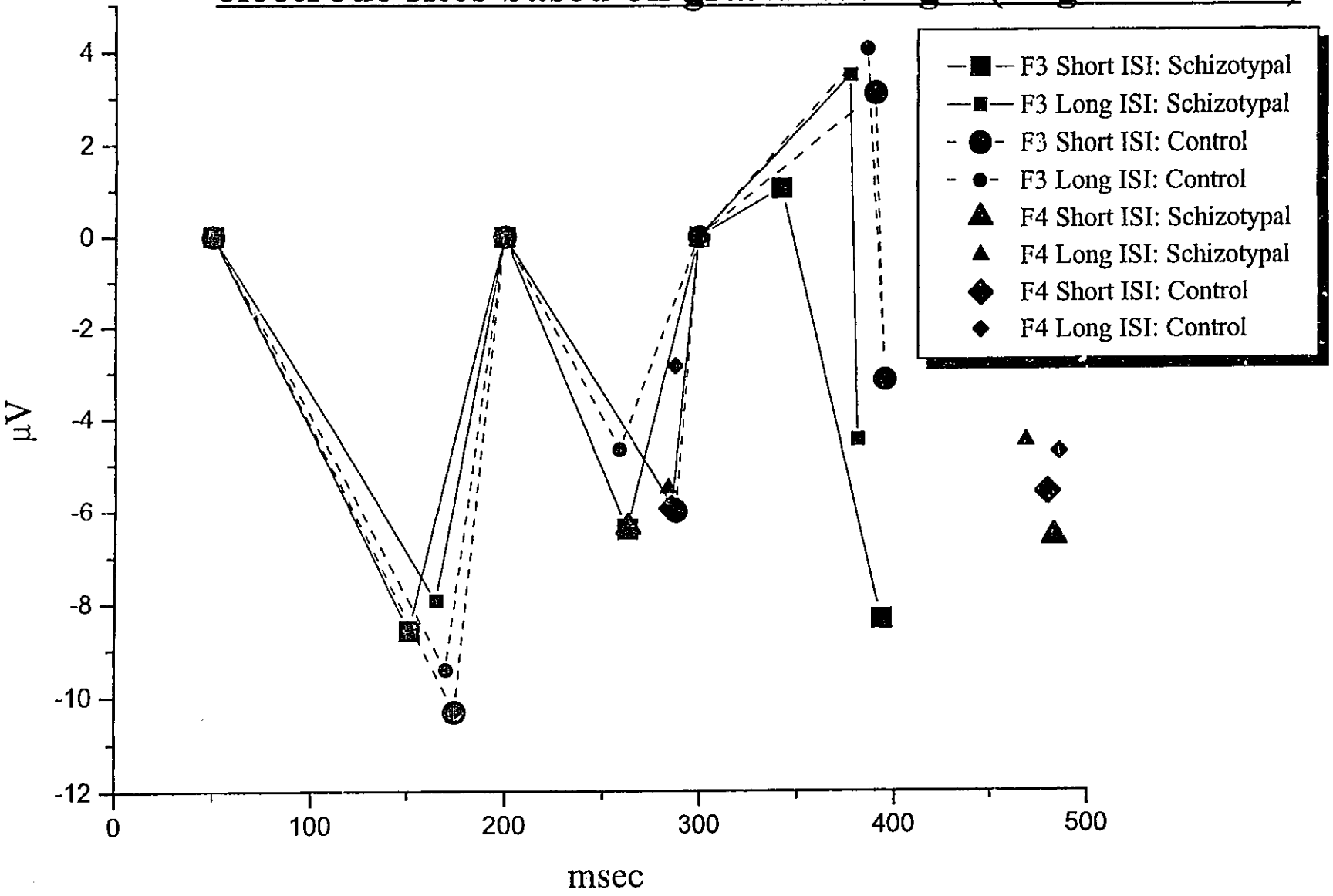


Figure 5. Diagrammatic composite of the pattern of ERPs at optimal electrode sites based on grand averages (Negative down)



Discussion

Schizotypal personality has been argued to be part of a spectrum of schizophrenia related disorders. The present investigation found that individuals with prominent schizotypal personality characteristics manifested one of the key patterns of ERP characteristics found in schizophrenics in the selective attention task. This pattern in our schizotypals is highly consistent with that found in florid schizophrenics with high content thought disorder combined with low levels of formal thought disorder (labelled as ↓FTD in the literature; e.g., Baribeau, 1986; Laurent and Baribeau, 1992).

In contrast, schizotypals did not manifest any behavioral disturbance in the selective attention task, nor any sensory gating deficit at any of the three ISIs nor any of the seven electrodes. The fact that all significant results cluster in only one task (selective attention) and only on attention modulated ERPs indicate a strong dissociation between attentional and preattentional mechanisms. In addition, this combination of results suggests that significant findings are unlikely to be due to Type I error.

Sensory Gating Study

Although a deficit in P50 suppression has been reported in numerous investigations of schizophrenics, we failed to find a deficit in our schizotypal population. The only other study that failed to find weaker suppression in schizophrenics as compared to normals was that of Kathmann and Engel (1990). These authors found that, as expected, schizophrenics demonstrated

almost no suppression. In contrast to previous investigations their controls also failed to produce strong suppression, and had widely distributed suppression ratios or even augmentation in some cases. Freedman (1990) criticized the Kathmann and Engel study stating that no control was exercised over possible muscle artifacts, as no EOG was recorded, and therefore the failure to find a suppression effect may have been due to myogenic contamination of the ERP. In addition, the clicks used by Kathmann and Engel may have been perceived as more intense as they were longer in duration (1.5 ms vs 0.04 ms in other investigations), thus adding to the possibility of introducing artifacts.

In our investigation click duration was 0.1 ms, considerably closer to that used by Freedman's group. Also, our 65 dB SPL click intensity is comparable to the 70 to 90 dB SPL clicks used in other studies. Thus it is highly unlikely that subjects perceived the clicks as louder than subjects in Freedman's studies. We also eliminated from averaging all trials in which the voltage of either EOG or EEG exceeded $\pm$80 μV, a more severe rejection criterion than the $\pm$100 μV employed by other P50 investigators. Thus the lack of significant differences cannot be due to myogenic artifacts.

Given that our methodology is comparable to those studies in which schizophrenics are found to have a suppression deficit in relation to normal controls, our investigation may be interpreted in at least three ways. First, there may be no actual group differences. Second, one may argue that

schizotypals indeed have a gating deficit, but some characteristic(s) in our controls resulted in their also having poor suppression. This is unlikely because comparisons of our controls with Kathmann and Engel's controls (1990), show similar suppression levels while being free from the artifacts that plagued the latter.

While we assessed state and trait anxiety as well as depression, none of which could account for the gating results, no other study has assessed the impact of personality variables. The only variables found to affect the P50 are certain psychiatric diagnoses such as alcoholism, mania, clinical depression and antisocial disorder (Baker et al., 1987), menstrual cycle (Waldo et al., 1987) and levels of certain biochemicals (Baker et al., 1990). There is no reason to expect that any of these, except clinical depression, may be explaining our results. Even this explanation is not supported given that our control subjects were not depressed as indicated by their low BDI scores (2.5 out of 63). Moreover, since variability was equivalent between the groups, and similar to other studies, it is unlikely that it may have accounted for the results. Thus we conclude that the two groups are not different in P50 gating, in clear dissociation with the attention modulated ERPs.

Selective Attention Study

Behavioral Data

Our schizotypals did not manifest behavioral deficts generally found in schizophrenics in vigilance and selective attention tasks (Nuechterlain, 1977; Hemsley, 1975). This is because the behavior of our schizotypals is normal, or because of the possible lack of sensitivity of RTs in reflecting subtle perceptual differences. As predicted by Broadbent (1971), the only significant effect indicated that all subjects were more accurate (more hits, less misses) at slow speed, because of a relatively easier task. Such lack of behavioral differences in attention tasks replicates other reports for samples of subjects with schizotypal characteristics, such as physical anhedonia (e.g., Giese-Davis, Miller and Knight, 1993).

ERP: Effects Unrelated to Grouping

Effects unrelated to grouping factor were generally in line with those reported in the literature. As expected, the longer ISI resulted in larger amplitudes for the N1-P2 (Picton et al., 1978; Teder, Alho, Reinikainen and Näätänen, 1993). P3b amplitude was also affected by ISI, being larger for the slow speed of presentation, as predicted by the memory "context updating" hypothesis (Donchin, 1981; Karis et al., 1984).

Attention modulation of the N2b-P3a and P3b generally followed predictions made by the literature, but were less clear for the N1-P2. For these components, attended stimuli generally produced larger amplitudes than ignored stimuli. Because the attention modulations were more clear on the late components (N2b-P3a-P3b), this data supports the contention that subjects did indeed operate response-set mechanisms to perform the task. The fact that the N1-P2 effect was not clear overall indicates that the stimulus-set mechanisms might not have been needed to perform the task. In retrospect, this makes sense since the present task was quite easy. This was a concern when designing the task. Because we wished to assess schizotypals in identical tasks to those applied to schizophrenics, our task by necessity had to be relatively easy. Finally, consistent with other studies (Pritchard, 1981), the SNW appeared as a large negative peak following P3b (Figure 4), largest at frontal sites, and going under baseline at Pz as predicted (Table 16).

Ear effects.- Single subject analysis and grand means analysis showed that ear effects are inconsistent both within and across components. Since neither the Group main effect nor the Group X Ear interaction for reaction time are significant, it is unlikely that this inconsistency is due to ear preference, since by definition, ear preference requires a consistent direction for it to be called ear preference. Thus the ear effects were random between components and conditions. It was felt that the following interpretations may

proceed without taking ear effects into account. However, since serendipitous and unhypothesized ear effects occurred randomly and with a great deal of variability between electrodes, and since electrode by ear interactions would required to be assessed as factors, and since such a MANOVA on electrodes would require a much larger sample of subjects, we recommend that a future study be addressed to the question of electrode topography and its relation to ear effects in schizotypal subjects. It was not the purpose of the present study to investigate whether ear effects varied in topography between electrodes. This issue was dependent on the observation of behavioral signs of ear preference. We have not found such evidence in the behavioral data. Thus the variability of ERPs in relation to ears has to be interpreted by some other intervening factor than operationally defined "behavioral ear preference" in a future study.

ERP Data: Effects related to Grouping

Overall, from visual inspection of single tracings, schizotypals manifested shorter latencies and larger amplitudes, except for the P3b, which can be seen in figures. This global morphological pattern strikingly resembles the dramatic ERPs recorded in florid high content thought disordered ↓FTD schizophrenics (Baribeau, 1986; Baribeau and Laurent, 1986; Hiramatsu et al., 1983; Laurent and Baribeau, 1992; Pritchard, 1986). We did not observe the flattened low voltage morphology typical of less

florid and more chronic highly formal thought disordered schizophrenics (ibid). Qualitative analysis can best give a feel for the morphological pattern, and is apparent on single tracings of schizotypal versus control subjects, Appendices L versus M respectively, but not as extreme as the contrast between subgroups of schizophrenics. Backing for such morphological differences is found in the following measures taken at discrete points of the tracings.

N1-P2

Latency of the N1-P2 to targets was shorter for schizotypals at F4 irrespective of speed, and at F3 when speed was fast, independent of the focus of attention. This result is consistent with the shorter N1 and P2 latencies reported in studies of unmedicated schizophrenics (Hiramatsu et al., 1983; Roth et al., 1980; Saletu et al., 1971). Shortened N1 latency in schizotypals is consistent with theories positing (Venebles, 1964) and experimental evidence (Baribeau, 1986; Öhman, 1981) for hyperarousal in at least a subgroup of schizophrenics.

N1-P2 amplitudes to attended targets were found to be larger for the schizotypal than control group at F3 at both speeds. This finding is inconsistent with previous work which found smaller N1-P2 amplitude in the lesser florid chronic schizophrenics (↑FTD) at slow speed and no group difference at fast speed (Baribeau et al., 1983; Baribeau and Laurent, 1986), thus suggesting that the schizotypals are not characterized, unlike chronic

↑FTD schizophrenics, as having a deficit in stimulus-set attention. This finding is nevertheless consistent with the shorter N1 latency which usually correlate with larger amplitudes. In our schizotypals, both latency and amplitude effects are consistent with a state of hyperarousal, higher levels of arousal being related to larger N1 amplitudes (Näätänen, 1992). There was no difference between groups in terms of stimulus-set. The very fact that there was no stimulus-set effect allows us to interpret the results for later components as not being due to a stimulus-set deficit. Thus the presumable hyperarousal of schizotypals resulted in neither an enhancement or decrement of behavioral performance nor of stimulus-set filtering.

N2b-P3a

N2b-P3a latency at F3 was found to be shorter for schizotypals relative to controls when speed of presentation was fast, but longer than controls when slow. In addition, N2b-P3a amplitude was found to be larger in the schizotypal than control group at F4 when speed of stimulus presentation was slow, for both the attend and ignore conditions (see Figure 2). In the literature on normals, N2b-P3a is generally faster at faster speed and slower at slower speed. Also, N2b-P3a is generally larger at slower speeds. Thus, here, our schizotypals are just more extreme than the controls. That might be interpreted as a sign of abnormality.

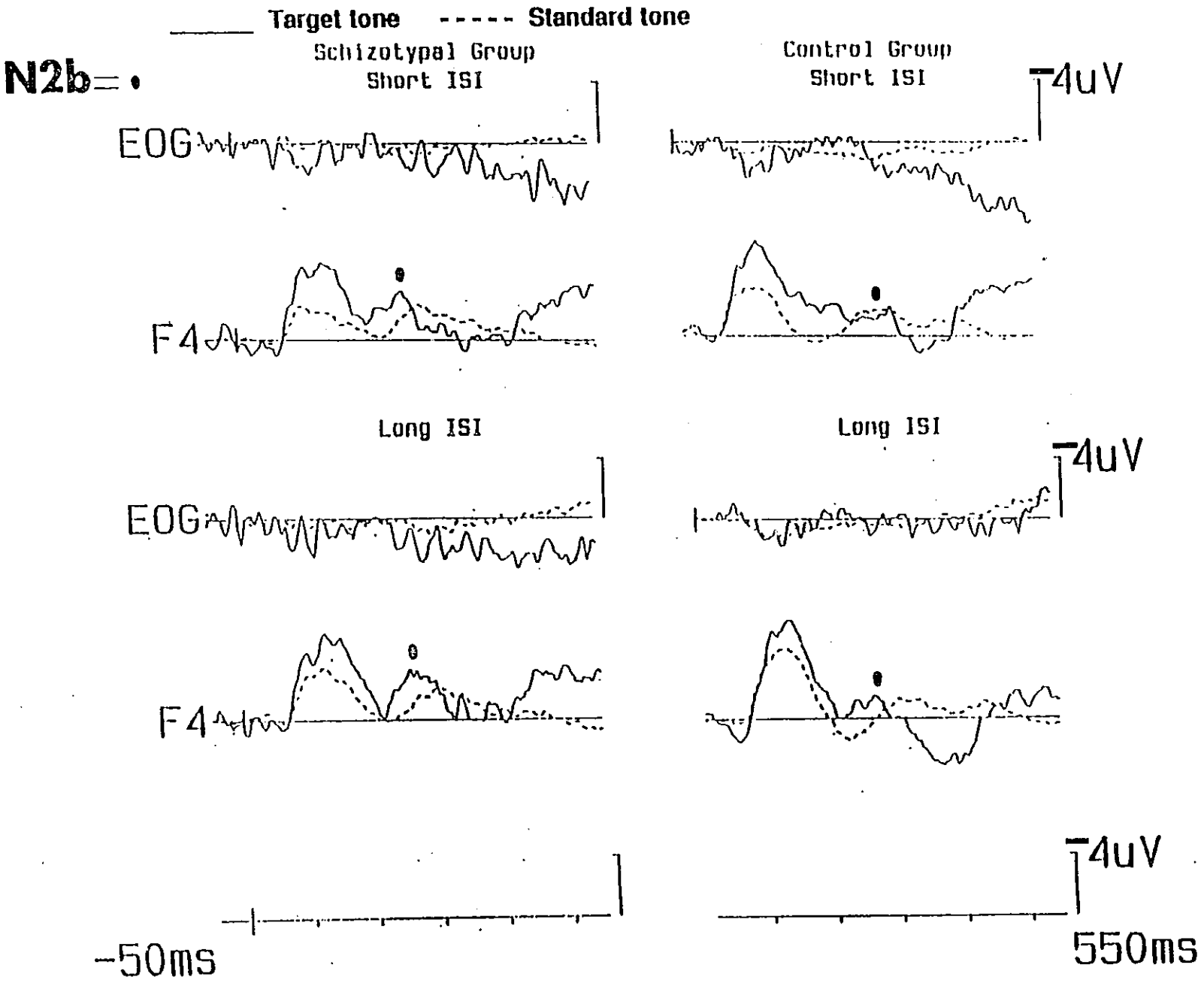
These results can be interpreted by recourse to evidence on the functional significance of N2b and P3a. The N2b has been argued to be associated with transient arousal and the orienting response (Loveless, 1983; Näätänen and Gaillard, 1983). The P3a has been argued to reflect a shift of attention associated with the manifestation of an orienting response, as described by Sokolov (1975). In terms of research on schizophrenia, evidence indicates that schizophrenics with prominent positive symptoms according to Andreasen's full positive score (Zahn, Frith and Steinhauer, 1991) manifest large skin conducting orienting responses (SCOR) to salient stimuli, and that florid ↓FTD schizophrenics but not those with ↑FTD manifest large frontal P3a's to task relevant stimuli (Baribeau and Laurent, 1986; Laurent and Baribeau, 1992). Furthermore, recent data indicates that university students classified as schizotypal based on the SPQ manifest both a larger SCOR and heart rate response to salient stimuli than control subjects (Raine et al., 1992). All these pieces of evidence have been interpreted as manifesting hyperactivity of the orienting response to salient stimuli in at least a subgroup of schizophrenics. Thus it seems plausible that our enhanced N2b-P3a also reflects hyperactivity of the orienting response, with consequent "hypernormal" speed effects as described above.

Moreover, that N2b-P3a amplitude was generally larger in our schizotypals when speed was slow rather than fast suggests that hyperactivity of orienting is a better explanation of the finding than hyperarousal. This argument is consistent with evidence indicating that at slow speed there is more likely to be selective dishabituation of the orienting response to targets, such that detection of match-mismatch is heightened (Sokolov, 1975), while unselective or nonspecific arousal is more likely to be heightened when speed is fast (Broadbent, 1971; Näätänen, 1992). This unselectivity of arousal should appear in response to standard tones. Because our results show it does not, the nonspecific arousal interpretation is dismissed. As seen in Figure 6, N2b-P3a is considerably larger to target than standard tones, qualitatively illustrating the mismatch effect on the N2b-P3a complex, and supporting the hyperorienting interpretation. Future studies are needed to assess other ERP components that have been argued to index orienting.

P3b

Consistent with the N1-P2 and N2b-P3a results, P3b latency at F3 was found to be shorter for schizotypal than control subjects at fast speed when attended. This is in line with the hyperarousal interpretations provided above for the faster N1-P2 and N2b-P3a reported above.

Figure 6. N2b-P3a amplitudes to target and standard tones for the schizotypal and control groups.



That P3b amplitude was found to be reduced in the schizotypal group, across speed and attention conditions, and at all electrodes, is consistent with the bulk of the literature on schizophrenia (all subtypes) indicating response-set deficits (Baribeau et al., 1983; Hiramatsu et al., 1983; Pfefferbaum et al., 1989; Pritchard, 1986). In addition, schizotypals often had larger P3bs to the ignored than attended stimuli, particularly at fast speed, unlike controls. Larger ignore than attend P3b's may be interpreted as reflecting response-set interference and distraction. This type of distractibility (not to be confused with stimulus-set or filtering failure) has been reported for heterogeneous groups of schizophrenics (Grillon et al., 1991; Spring et al., 1991). Such interference, restricted to fast speed of stimulation has been reported for florid schizophrenics with ↓FTD but not for less florid ↑FTD schizophrenics (Baribeau, 1986; Baribeau and Laurent, 1986; Laurent and Baribeau, 1992). That distractibility was found for schizotypals at fast but not slow speed suggests that their hyperarousal may have resulted in what we label here as an intrusion effect. Such an intrusion effect has previously been operationally defined in Baribeau and Laurent (1986).

Smaller P3b amplitudes found in schizotypals even though targets were detected as accurately as controls, across attention conditions, may be interpreted as reflecting a general inefficiency in obtaining information from significant or task-relevant stimuli. That the P3b amplitude deficit is no more severe at fast than slow speed of presentation suggests that it is unrelated

to arousal effects (Baribeau, 1986), though the shorter P3b latency at the fast speed may be. According to Donchin et al. (1984), the factors modulating P3b amplitude can be distinct from those modulating P3b latency.

The response-set deficit implicated by small P3b's does not appear to be a secondary effect of stimulus-set (as explained above) since schizotypals did not manifest a loss of channel selectivity (as indicated by the N1-P2). Comparable behavioral results suggest that the groups were equally motivated in performing the task. Given that P3b latency is taken as an index of how quickly a stimulus event is evaluated (Kutas et al., 1977; Pritchard, 1981), our results would suggest that schizotypals are excessively rapid in evaluating a target stimulus for decision, the consequence being a significantly more inefficient evaluation for decision. This inefficiency may be being compensated for somewhere along the chain of information processing given the lack of group differences on behavioral performance.

SNW

The SNW appeared consistently and clearly in schizotypal subjects. It was generally larger and faster frontally for schizotypals, most prominently at slow speed (Figure 3). This is consistent with the above hyperarousal interpretation. These results are consistent with previous work with schizophrenics (Cohen et al., 1990; Michie et al., 1990), though increased SNW are particularly characteristic of more florid schizophrenics with ↓FTD (Baribeau and Laurent, 1986; Laurent and Baribeau, 1992).

It is unlikely that the large SNW in schizotypals is a contingent negative variation (CNV) because of the randomized sequence of stimuli. Moreover, the CNV explanation is also discountable because schizotypals were found to be more state-anxious than controls, and anxiety has been found to reduce CNV amplitude (Knott and Irwin, 1973). This is consistent with the high anxiety and large SNW found in florid ↓FTD schizophrenics (Laurent and Baribeau, 1992).

In line with the literature, increased amplitude of the SNW in schizotypals may be interpreted in light of the relationship between P3b and the SNW. These two components bear an opposite relation to perceptual sensitivity/evaluation for decision in signal detection tasks (Sutton and Ruchkin, 1984), with larger P3b and smaller SNW indicating more sensitivity/better evaluation. Given that under most task conditions P3b was found to be smaller and SNW larger in schizotypals, this suggests that schizotypals have poor perceptual sensitivity/evaluation for decision, and consequently, as indicated by the large SNW, must continue to carry-out task-irrelevant cognitive processing of the stimuli to a much greater degree than controls (Sutton and Ruchkin, 1984). This is compatible with Posner's frontal mechanism of attentional disengagement that is abnormal in schizophrenics (Nestor et al., 1992).

Finally, as with the P3b, SNW amplitudes were more often larger in ignore than attend conditions for schizotypals than controls (see Table 16). This suggests that schizotypals not only were more susceptible to intrusion of the to-be-ignored targets, but that they continued to conduct a large amount of task-irrelevant cognitive processing of the stimuli. This phenomenon is very compatible with the cognitive style, the overgeneralization, the elusive and loose associations of individuals who rate high on the SPQ, particularly the odd speech subscale. Given that the SPQ thought disorder items do not differentiate content from formal ones, further investigations will be needed to determine whether the results reflect one or the other, or both.

Conclusions

While this thesis sought to determine whether schizotypal personality is like schizophrenia, the results suggest that some qualification of the schizotypal personality - schizophrenia relationship is due. Some parallels and some differentiations from the mainstream schizophrenia literature are apparent. The at times incongruent nature of our findings with such mainstream research may however be due to the problem of relating schizotypal personality to a unitary concept of schizophrenia, which appears to be a heterogeneous disorder. Thus schizotypal personality may only be related to a particular subtype of schizophrenia.

Our pattern of results in the schizotypal group is highly consistent with the pattern demonstrated by highly florid ↓FTD schizophrenics, but not the less florid ↑FTD schizophrenics (Baribeau, 1986; Baribeau and Laurent, 1986; Laurent and Baribeau, 1992). ERP studies of schizophrenics categorized according to other criteria do not conform with our pattern of results (Pritchard, 1986)

Summary.- Like more florid ↓FTD schizophrenics, results revealed that schizotypals are characterized: by (1) hyperarousal consistent with the input dysfunction model of schizophrenia (Claridge, 1972; McGhie; 1970; Venables, 1964); (2) enhanced frontal N2b-P3a suggestive of selective dishabituation of the orienting response to task-relevant stimuli at slow speed of stimulation; (3) a response-set deficit to attended stimuli as well as response-set interference and intrusion/distraction; and (4) large frontal SNW indicating more task-irrelevant post-response cognitive processing.

Although the results of this investigation are interpretable in terms of work with schizophrenics, it remains unclear as to whether our schizotypals were totally free from any other nonclinical form of disorders since full diagnostic interviews were not carried out. Our lab questionnaire could only rule out major psychiatric or neurological conditions. The concern that samples of schizotypal subjects are contaminated by the presence of borderline personality features has been highlighted by a number of authors (for a review, see Kotsaftis and Neale, 1993). Others have, however,

provided evidence indicating that schizotypal but not borderline features are predictive of vulnerability to schizophrenia (Fenton and McGlashan, 1989), and that information processing disturbances in schizotypal and borderline patients are distinct (Schubert, Saccuzzo and Braff, 1985). Nevertheless, further investigations using the SPQ should carry out comprehensive screening of subjects through diagnostic interviews prior to participation in cognitive testing sessions. In addition, comparison of schizotypal and borderline subjects on the tasks used in the present investigation may help clarify their relations to each other and schizophrenia. Finally, schizotypals in this investigation were found to be more depressed and anxious at the time of testing than controls, thus introducing a possible confound. It is unlikely, however, that these affective variables would significantly alter the pattern of results obtained since ERP studies involving depressed or anxious subjects do not reveal such patterns with large N2b-P3a and SNW amplitudes (Baribeau and Lesèvre, 1983; El Massioui and Lesèvre, 1988; Towey et al., 1990). Nevertheless, further studies should investigate the contribution of affective variables to ERP findings in schizotypal subjects, and compare schizotypals to depressed and anxious patient populations.

Given the wealth of evidence for subtyping schizophrenia, it may prove elucidating to determine whether there are salient subtypes of schizotypal personality. Preliminary investigations of this sort have separated subjects who score high on the cognitive/perceptual factor from

those who score high on the interpersonal deficits factor of the SPQ (Gruzelier et al., 1993). In addition, a number of studies have observed ERP differences between student samples with and without signs of schizophrenic thought disorder (McConaghy et al., 1993; Ward, Catts, Armstrong and McConaghy, 1984). Given cur results, further investigations may attempt to differentiate schizotypals in terms of signs of formal and content thought disorders.

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Appendix A

Schizotypal Personality Questionnaire (SPQ).....

SRQ (RAINE, 1991)

Please answer each item by circling Y (Yes) or N (No). Answer all items even if unsure of your answer. When you have finished, check over each one to make sure you have answered them.

Y N 1. Do you sometimes feel that things you see on the TV or read in the newspaper have a special meaning for you ?

Y N 2. I sometimes avoid going to places where there will be many people because I will get anxious.

Y N 3. Have you had experiences with the supernatural ?

Y N 4. Have you often mistaken objects or shadows for people, or noises for voices ?

Y N 5. Other people see me as slightly eccentric (odd).

Y N 6. I have little interest in getting to know other people.

Y N 7. People sometimes find it hard to understand what I am saying.

Y N 8. People sometimes find me aloof and distant.

Y N 9. I am sure I am being talked about behind my back.

Y N 10. I am aware that people notice me when I go out for a meal or to see a film.

Y N 11. I get very nervous when I have to make polite conversation.

Y N 12. Do you believe in telepathy (mind-reading) ?

Y N 13. Have you ever had the sense that some person or force is around you, even though you cannot see anyone ?

Y N 14. People sometimes comment on my unusual mannerisms and habits

Y N 15. I prefer to keep to myself.

Y N 16. I sometimes jump quickly from one topic to another when speaking.

Y N 17. I am poor at expressing my true feelings by the way I talk and look.

Y N 18. Do you often feel that other people have got it in for you ?

Y N 19. Do some people drop hints about you or say things with a double meaning ?

Y N 20. Do you ever get nervous when someone is walking behind you ?

Y N 21. Are you sometimes sure that other people can tell what you are thinking ?

Y N 22. When you look at a person, or yourself in a mirror, have you ever seen the face change right before your eyes ?

Y N 23. Sometimes other people think that I am a little strange.

Y N 24. I am mostly quiet when with other people.

Y N 25. I sometimes forget what I am trying to say.

Y N 26. I rarely laugh and smile.

Y N 27. Do you sometimes get concerned that friends or co-workers are not really loyal or trustworthy ?

Y N 28. Have you ever noticed a common event or object that seemed to be a special sign for you ?

Y N 29. I get anxious when meeting people for the first time.

Y N 30. Do you believe in clairvoyancy (psychic forces, fortune telling) ?

Y N 31. I often hear a voice speaking my thoughts aloud.

Y N 32. Some people think that I am a very bizarre person.

Y N 33. I find it hard to be emotionally close to other people.

Y N 34. I often ramble on too much when speaking.

Y N 35. My "non-verbal" communication (smiling and nodding during a conversation) is poor.

Y N 36. I feel I have to be on my guard even with friends.

Y N 37. Do you sometimes see special meanings in advertisements, shop windows, or in the way things are arranged around you ?

Y N 38. Do you often feel nervous when you are in a group of unfamiliar people ?

Y N 39. Can other people feel your feelings when they are not there ?

Y N 40. Have you ever seen things invisible to other people ?

Y N 41. Do you feel that there is no-one you are really close to outside of your immediate family, or people you can confide in or talk to about personal problems ?

Y N 42. Some people find me a bit vague and elusive during a conversation.

Y N 43. I am poor at returning social courtesies and gestures.

Y N 44. Do you often pick up hidden threats or put-downs from what people say or do ?

Y N 45. When shopping do you get the feeling that other people are taking notice of you ?

Y N 46. I feel very uncomfortable in social situations involving unfamiliar people.

Y N 47. Have you had experiences with astrology, seeing the future, UFOs, ESP or a sixth sense ?

Y N 48. Do everyday things seem unusually large or small ?

Y N 49. Writing letters to friends is more trouble than it is worth.

Y N 50. I sometimes use words in unusual ways.

Y N 51. I tend to avoid eye contact when conversing with others.

Y N 52. Have you found that it is best not to let other people know too much about you ?

Y N 53. When you see people talking to each other, do you often wonder if they are talking about you ?

Y N 54. I would feel very anxious if I had to give a speech in front of a large group of people.

Y N 55. Have you ever felt that you are communicating with another person telepathically (by mind-reading) ?

Y N 56. Does your sense of smell sometimes become unusually strong ?

Y N 57. I tend to keep in the background on social occasions.

Y N 58. Do you tend to wander off the topic when having a conversation.

Y N 59. I often feel that others have it in for me.

Y N 60. Do you sometimes feel that other people are watching you ?

Y N 61. Do you ever suddenly feel distracted by distant sounds that you are not normally aware of ?

Y N 62. I attach little importance to having close friends.

Y N 63. Do you sometimes feel that people are talking about you ?

Y N 64. Are your thoughts sometimes so strong that you can almost hear them ?

Y N 65. Do you often have to keep an eye out to stop people from taking advantage of you ?

Y N 66. Do you feel that you are unable to get "close" to people ?

Y N 67. I am an odd, unusual person.

Y N 68. I do not have an expressive and lively way of speaking.

Y N 69. I find it hard to communicate clearly what I want to say to people.

Y N 70. I have some eccentric (odd) habits.

Y N 71. I feel very uneasy talking to people I do not know well.

Y N 72. People occasionally comment that my conversation is confusing.

Y N 73. I tend to keep my feelings to myself.

Y N 74. People sometimes stare at me because of my odd appearance.

Appendix B

Recruitment Strategy.....

"If you decide not to fill out this questionnaire please return the unused portion, as we would like to recycle it".

LABORATORY OF NEUROPSYCHOLOGY
DEPARTMENT OF PSYCHOLOGY - CONCORDIA UNIVERSITY
Loyola Campus, 7141 Sherbrooke W., Drummond Sciences Bldg, Rm.413.
Montreal, Quebec, H4B 1R6. Tel: 848-2244.

We are currently in need of participants for diverse investigations on the attention, memory and problem solving abilities of individuals with various experiences, feelings and beliefs, as well as billinguals and musicians. To do so, we require the administration of a the following questionnaires to at least 800-900 students. The questionnaire booklet is 7 pages and takes aproximately 60 minutes to complete. Because we wish to reduce needless paper usage and time, we ask that you complete these questionnaires only if you are interested in coming to our laboratory later on in the current or next semester, to participate in a short study. That study will involve the completion of various psychological tests, and can be done at your convenience.

You will only be contacted for later investigations if you fulfill our study's criteria based on the questionnaires. You may of course refuse to participate any further, at any time, as **completion of the questionnaires places you under no obligation** whatsoever to participate in our studies. All information you provide in this questionnaire booklet will be kept **strictly confidential** by the study team.

Individuals participating in the laboratory study will be entered into a cash prize drawing. If you are interested in participating in our study, complete the questionnaires and return them to the member of our study team who will be there to pick them up at you next class (or return to the address given above). Please answer all questions asked, do not leave any questions blank. If you have any questions about this questinnaire ot the laboratory study please feel free to contact us.

We thank you for your time.

Dr. Jacinthe Baribeau & Robert M. Roth

Name (please print): _____
Telephone: _____ AGE: _____ Sex: M F
Handedness: LEFT RIGHT BOTH. ETHNICITY: _____
Birthdate: DAY _____ MONTH _____ YEAR _____
When is the best time to reach you ? morning / afternoon / evening / weekend
What do you consider to be your first language?_____
What other languages do you speak? _____
Do you play a musical instrument or sing? NO YES
If YES, which? _____

If YES, indicate approximate level: novice / serious amateur / artist level

Appendix C

Informed Concent Form.....

PERSONALITY/COGNITION STUDY
LABORATORY OF HUMAN NEUROPSYCHOLOGY AND NEUROPHYSIOLOGY
Informed Consent Form

I (please print clearly) _____, age _____, consent to participate in a study conducted by Robert M. Roth under the supervision of Dr. Jacinthe Baribeau, at Concordia University, Montreal, Quebec.

I understand that my participation will involve completing a number of questionnaires and a brief interview pertaining to demographics, personal and family history of psychological and medical problems.

I understand that my participation will also involve the recording of my brain's electrical activity while I perform various tasks. Small plastic caps will be placed at a number of locations on my head such that recordings of my brain activity may be obtained. To place these caps such that recordings may be properly made, it will be necessary to slightly abbraid the skin under the electrodes. **The procedure is safe, there is no risk of harm whatsoever.**

I understand that the entire testing session will take approximately 2 1/2 hours to complete. I understand that I may withdraw from the experiment at any time by indicating my desire to do so to the experimenter.

I understand that all my results will be kept strictly confidential by the experimental team and that I may have access to my results and the results of the study as available. I also understand that I will be paid $10.00 for my participation in this study.

Participant Signature: _____

Witness Signature: _____

Date: ___/___/_____
Day Month Year

Appendix D

Laboratory of Neuropsychology Interview.....

CONCORDIA UNIVERSITY RESEARCH PROGRAM
LABORATORY OF NEUROPHYSIOLOGY AND NEUROPSYCHOLOGY
SUBJECT INTERVIEW (3 pages)
Robert M. Roth & Jacinthe Baribeau

Current Date: Day _____ Month _____ Year _____
Interview Conducted By: _____

DEMOGRAPHICS

Name (please print): _____
Home Phone Number: _____
Home adresse: _____
Date of Birth: Day _____ Month _____ Year _____
Gender: Male _____ Female _____
In what religion were you raised? _____
Rate the strength of your religous belief: 0 1 2 3 4 5
(0 = none to 5 = very strong)
Years of Academic Education Completed: _____
Highest level of academic attainment: _____
Current Occupation (describe job): _____
Income (previous year): $ _____
Marital Status (circle): single divorced separated marri
cohabitating

PSYCHIATRIC & MEDICAL HISTORY

Were there any problems with your own birth? Yes ___ No ___
If Yes, explain: _____

Have you ever had an injury to your head resulting in loss consciousness? Yes _____ No ____
If Yes, how old were you (in years)? _____
how long were you unconscious (in days)? _____

Are you currently suffering from any medical problems? Yes _____ No ___
Yes, describe: _____
_____

Have you, in the past (pre 1 year ago), suffered from a medical problem? Yes ____ No _____
If Yes, describe: _____
_____

Are you currently using an nonprescription drug such as marijuana? Yes ____ No _____
If Yes, list type(s) and frequency (per week): _____
_____

Do you drink alcohol? Yes ____ No ____
If Yes, how many drinks: Per Day ____ Per Week ____ Per Month ____
[n.b. 1 drink = 1 beer, one glass of wine of a shot of spirits]

Do you smoke? Yes ____ No ____
If Yes, how many cigarettes: Per Day ____ Per Week ____

Are you currently receiving any treatment for a psychological problem? Yes ____ No ____

If Yes, list type(s): type(s): _____

_____

Have you received, in the past, any treatment for a psychological proble Yes ____ No ____

If Yes, list type(s): _____

_____

_____

_____

Interviewer! If subject indicates that he/she has a psychologic problem(s), ask age of onset for each one.
Has subject indicated a psychiatric problem? Yes ___ No _
If Yes, list disorder and age:
Disorder 1: _____ Age: ___
Disorder 2: _____ Age: ___
Disorder 3: _____ Age: ___

FAMILY HISTORY

Now I would like to ask you some questions about your family. I interested in your parents, brothers, sisters, paternal and matern grandparents, children, well as any uncles and aunts.

Are you adopted? Yes ____ No __
(if adopted, for the questions below, indicate if adotive or biologic family member is in question. Ask about both.)

How many years of education has your father completed? ____
What is your fathers current occupation? _____
Is your father left, right or mixed handed? _____

How many years of education has your mother completed? ____
What is your mothers current occupation? _____
Is your mother left, right or mixed handed? _____

Do any of your family members suffer from a medical problem? Yes ____ No ____

If Yes, please list member and describe problem:

_____

_____

_____

Do any of your family members currently suffer from a psychologi
problem? Yes ____ No _
If Yes, list member and problem: _____

_____

_____

_____

_____

Have any of your family members suffered from a psychological problem
the past (pre 1 year ago)? Yes ____ No _
If Yes, list member and problem: _____

_____

_____

_____

Are any of your family members currently using any medication?
Yes ____ No _
If Yes, list member, type and use of medication: _____

_____

_____

Do any of your family members suffer from epilepsy? Yes ____ No _
If Yes, list member: _____
At what age did the seizures begin? ___
Did it follow some form of trauma? Yes ___ No
If Yes, explain: _____

_____

Appendix E

Instructions for the Sensory Gating Study.

Familiarization Task: You are going to hear a short series of clicks through the headphones, so you can get used to how they sound. Although at first you may have trouble ignoring the clicks, try to relax and not pay any attention to them.

Baseline Task: Now you are going to hear the same clicks an number of times, but this time the task will be somewhat longer. Again, try to relax and not pay any attention to the clicks.

Test Task: Now you are going to hear the same clicks again, but this time they will come in pairs. Also the this task will be even longer than the previous one. Once again, try to relax and not pay any attention to the clicks.

Appendix F

Instructions for Binaural Listening Task

Tone Familiarization Task: You are going to hear a series of tones through the headphones. There are two kinds of tones, one of low pitch and one of high pitch (experimenter makes verbal simulation of the two tones). During the practice trials and the test blocks you will be asked to respond only to the high pitched tone. This tone is what we call the target. Now you are going to hear first ten of the low followed by 10 of the high pitched tones. This is done to make certain that you can clearly discriminate them.

Practice Task:

Practice block #1: Now I am going to give you a practice block in which you will hear a random series of the two tones presented. Remember that you will never hear two tones at the same time in two different ears. Instead, the ear that the tone is presented to, and the type of tone that is presented, will be random. What I would like you to do is the following: when you hear the high pitched tone in your right ear, press the right button on the mouse; when you hear the high pitched tone in your left ear, press the left button on the mouse. Respond as quickly as possible, but try not to make any mistakes.

Practice block #2: Now I would like you to do a second block of practice trials. But this time you may notice that the tones will come a bit faster. Again, same as the first practice, when you hear the high pitched tone in your right ear, press the right button on the mouse; when you hear the high pitched tone in your left ear, press the left button on the mouse. Respond as quickly as possible, but try not to make any mistakes.

Test Task: Now we will begin the actual testing. The idea is generally the same as the practice blocks, but there are three different types of instructions. I will give you one of these instructions prior to each block:

Focus left: When you hear the high pitched tone in your left ear press the left button on the mouse. Try to completely ignore the tones being presented to your right ear. Respond as quickly as possible, but try not to make any mistakes.

Focus right: When you hear the high pitched tone in your right ear press the right button on the mouse. Try to completely ignore the tones being presented to your left ear. Respond as quickly as possible, but try not to make any mistakes.

Appendix G

ANOVA (mixed within-subjects factorial design) summary table[1]

Source	Sum of Squares	df	Mean Square	F	P
G	22116.78	1	22116.78	2.12	.16
Error	218901.24	21	10423.87		
E	13760.49	6	2293.42	1.12	.35
Error	220265.85	108	2039.50		
S	10278.78	2	5139.39	.96	.39
Error	192684.79	36	5352.36		
E X S	25167.37	12	2097.28	1.25	.25
Error	361544.45	216	1673.82		
G X E	1110.22	6	185.04	.09	.99
Error	220265.85	108	2039.50		
G X S	16430.83	2	8215.42	1.53	.23
Error	192684.79	36	5352.36		
G X E X S	7793.53	12	649.46	.39	.97
Error	361544.45	216	1673.82		

1. G = Group, S = Speed, E = Ear.
* $p \leq .01$.

Appendix H

ANOVA (mixed within-subjects factorial design) summary tables[1]

Source	Sum of Squares	df	Mean Square	F	P
Reaction times					
G	22116.78	1	22116.78	2.12	.16
Error	218901.24	21	10423.87		
E	785.73	1	785.73	1.01	.33
Error	16346.74	21	778.42		
S	1136.95	1	1136.95	1.09	.31
Error	21841.06	21	1040.05		
E X S	257.80	1	257.80	.32	.58
Error	16668.29	21	793.73		
G X E	1681.60	1	1681.60	2.16	.16
Error	16346.74	21	778.42		
G X S	4411.53	1	4411.53	4.25	.05
Error	21841.06	21	1040.05		
G X E X S	.13	1	.13	.00	.99
Error	16668.29	21	793.73		
Hits					
G	18.66	1	18.66	2.86	.11
Error	137.21	21	6.53		
E	1.80	1	1.80	3.49	.08
Error	10.85	21	.52		
S	10.44	1	10.44	11.35	.003*
Error	19.30	21	.92		
E X S	.01	1	.01	.01	.92
Error	11.21	21	.53		
G X E	.06	1	.06	.12	.73
Error	10.85	21	.52		
G X S	.00	1	.00	.00	.97
Error	19.30	21	.92		
G X E X S	.53	1	.53	.99	.33
Error	11.21	21	.53		

1. G = Group, S = Speed, E = Ear.
* $p \leq .01$.

Source	Sum of Squares	df	Mean Square	F	P
Misses					
G	.81	1	.81	1.09	.31
Error	15.56	21	.74		
E	.39	1	.39	.81	.38
error	10.11	21	.48		
S	21.00	1	21.00	32.77	.001*
Error	13.46	21	.64		
E X S	.05	1	.05	.15	.70
Error	7.27	21	.35		
G X E	.00	1	.00	.00	.97
Error	10.11	21	.48		
G X S	.00	1	.00	.00	.99
Error	13.46	21	.64		
G X E X S	.18	1	.18	.53	.48
Error	7.27	21	.35		
Pseudo-misses					
G	1.25	1	1.25	3.50	.08
Error	7.47	21	.36		
E	.04	1	.04	.63	.44
Error	1.46	21	.07		
S	1.13	1	1.13	4.55	.05
Error	5.20	21	.25		
E X S	.18	1	.18	1.68	.21
Error	2.27	21	.11		
G X E	.00	1	.00	.00	.97
Error	1.46	21	.07		
G X S	.21	1	.21	.87	.36
Error	5.20	21	.25		
G X E X S	.05	1	.05	.48	.50
Error	2.27	21	.11		
False alarms					
G	.11	1	.11	.04	.83
Error	50.05	21	2.38		
E	.06	1	.06	.09	.77
Error	13.05	21	.62		
S	.01	1	.01	.01	.94
Error	29.14	21	1.39		
E X S	.70	1	.70	1.35	.26
Error	10.80	21	.51		
G X E	.40	1	.40	.65	.43
Error	13.05	21	.62		
G X S	7.31	1	7.31	5.27	.03
Error	29.14	21	1.39		

Source	Sum of Squares	df	Mean Square	F	P
False alarms - continued					
G X E X S	.00	1	.00	.00	.96
Error	10.80	21	.51		

Appendix I

MANOVA summary tables[1,2,3]

Source	Approx. F	Pillai	P
N1-P2 latency to targets			
F3			
S	4.85	.326	.02
G X S	6.05	.377	.01
F4			
G	3.76	.273	.04
C3			
G	3.93	.282	.04
G X S	3.85	.278	.04
S X A	6.97	.412	.005
N1-P2 amplitude to targets			
F3			
S	6.22	.384	.01
G X E X S	3.33	.250	.06
F4			
S	4.04	.287	.03
C3			
S	13.00	.565	.001
E X S	5.93	.372	.01
E X A	4.01	.286	.03
S X A	6.80	.405	.01
G X E X S	4.25	.298	.03
G X S X A	4.36	.304	.03
E X S X A	4.42	.307	.03

1. G = Group, S = Speed, A = Attention condition, E = Ear.
2. Degrees of freedom for all effects are 2, (error) 20.
3. Because of space limitations, the source table contains only those effects that are trends at $p \leq .05$ and significant at $p \leq 0.01$.

Source	Approx. F	Pillai	P
N1-P2 amplitude to targets - continued			
C4			
S	17.82	.641	.001
A	6.49	.394	.01
G X A	4.31	.301	.03
E X S	4.00	.286	.03
E X A	3.69	.270	.04
G X E X S A	4.48	.309	.03
N2-P3a latency to targets			
F3			
S	4.17	.295	.03
G X S	5.54	.356	.01
N2-P3a amplitude to targets			
F3			
S	8.34	.455	.002
F4			
S	3.98	.285	.04
G X E	9.61	.490	.001
E X S	3.51	.260	.05
G X E X S	6.15	.381	.01
G X S X A	3.99	.285	.04

Appendix J

ANOVA (mixed within-subjects factorial design) summary tables[1,2]

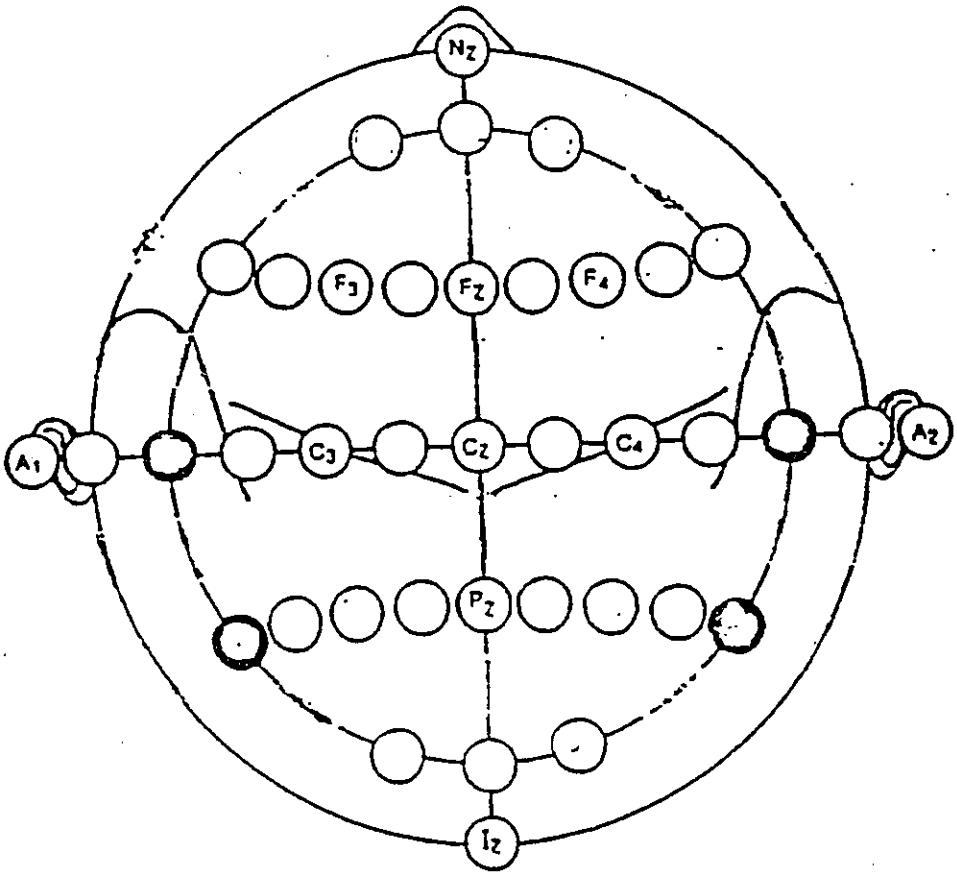
Source	Sum of Squares	df	Mean Square	F	P
P3b latency to targets					
F3					
A	7975.23	1	7975.23	4.69	.04
Error	35698.00	21	1699.90		
S X A	4508.12	1	4508.12	4.55	.05
Error	20816.79	21	991.28		
G X S X A	9040.03	1	9040.03	9.12	.01
Error	20816.79	21	991.28		
P3b amplitude to targets					
F3					
A	58.08	1	58.08	4.63	.04
Error	263.51	21	12.55		
G X E X S X A	64.21	1	64.21	7.55	.01
Error	178.60	21	8.50		
F4					
A	170.09	1	170.09	19.74	.001
Error	181.00	21	8.62		
C3					
S	89.38	1	89.38	11.91	.002
Error	157.62	21	7.51		
E X S X A	23.22	1	23.22	4.29	.05
Error	113.71	21	5.14		
G X E X S X A	48.64	1	48.64	8.98	.01
Error	113.71	21	5.14		
C4					
S	58.59	1	58.59	8.19	.01
Error	150.20	21	7.15		
A	297.23	1	297.23	14.57	.001
Error	428.37	21	20.40		
G X E X S X A	51.07	1	51.07	6.97	.02
Error	153.80	21	7.32		

1. G = Group, S = Speed, A = Attention condition, E = Ear.
2. Because of space limitations, the source table contains only those effects that are significant at $p \leq 0.01$ and trends at $p \leq 0.05$.

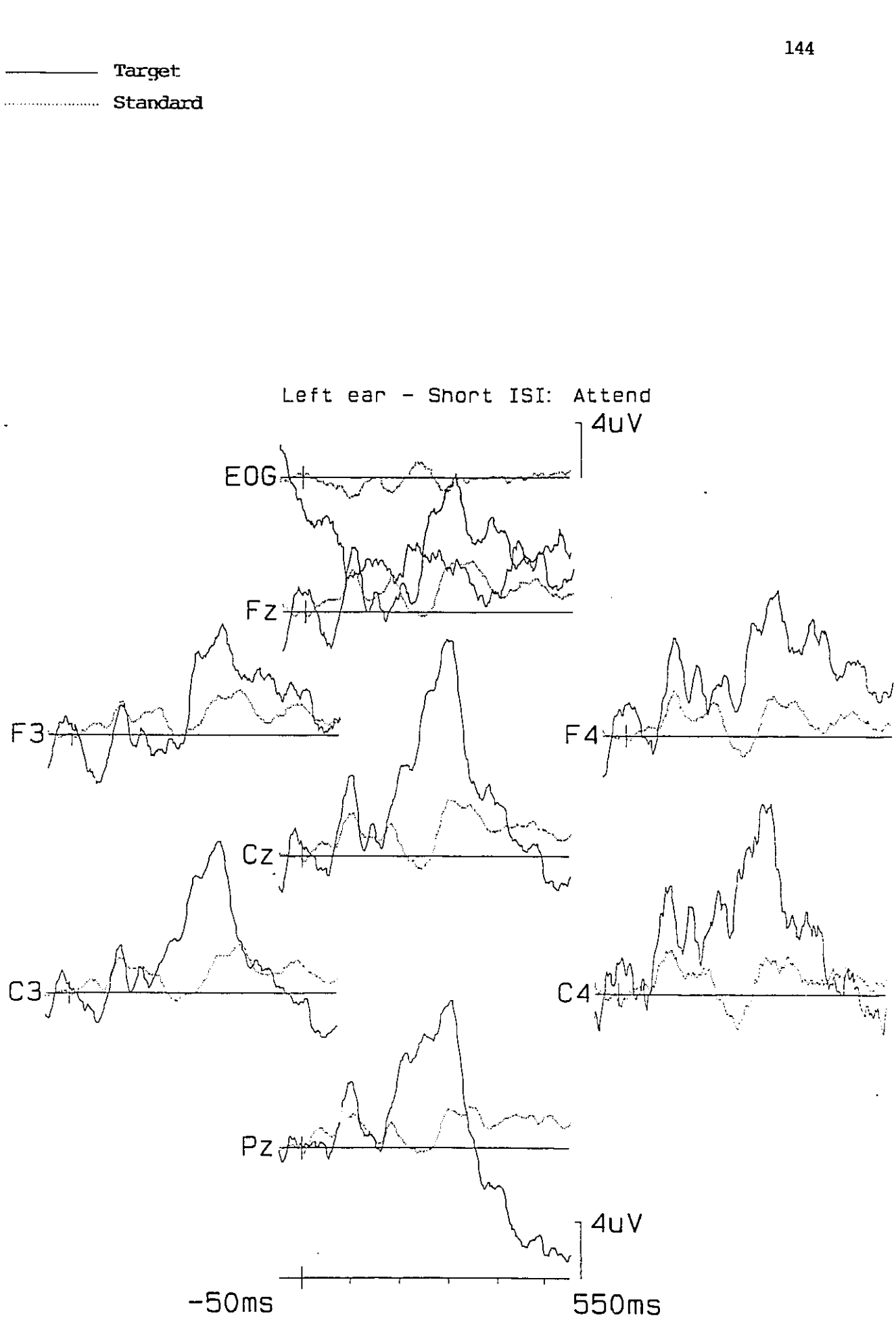
Source	Sum of Squares	df	Mean Square	F	P
Slow Wave latency to targets					
F3					
A	7975.23	1	7975.23	4.69	.04
Error	35698.00	21	1699.90		
S X A	4508.12	1	4508.12	4.55	.05
Error	20816.79	21	991.28		
F4					
G X S	4783.18	1	4783.18	4.58	.04
Error	21909.09	21	1043.29		
Slow Wave amplitude to targets					
F3					
G	241.00	1	241.00	15.98	.001
Error	1001.63	21	47.70		
G X E X S X A	105.22	1	105.22	6.95	.02
Error	317.78	21	15.13		
F4					
A	43.59	1	43.59	4.48	.05
Error	204.45	21	9.74		
G X E X S X A	56.91	1	56.91	5.10	.04
Error	234.22	21	11.15		

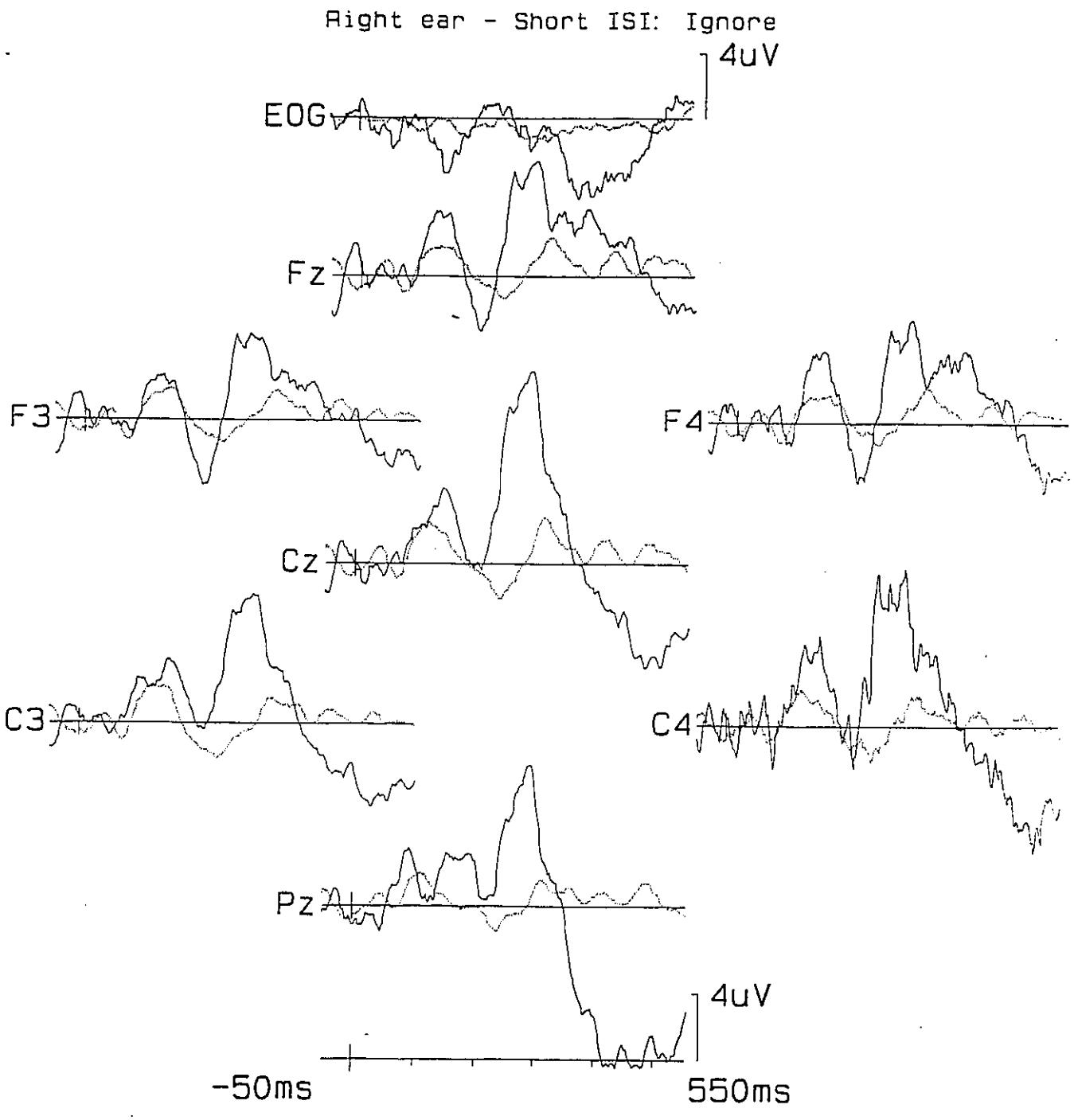
Appendix K

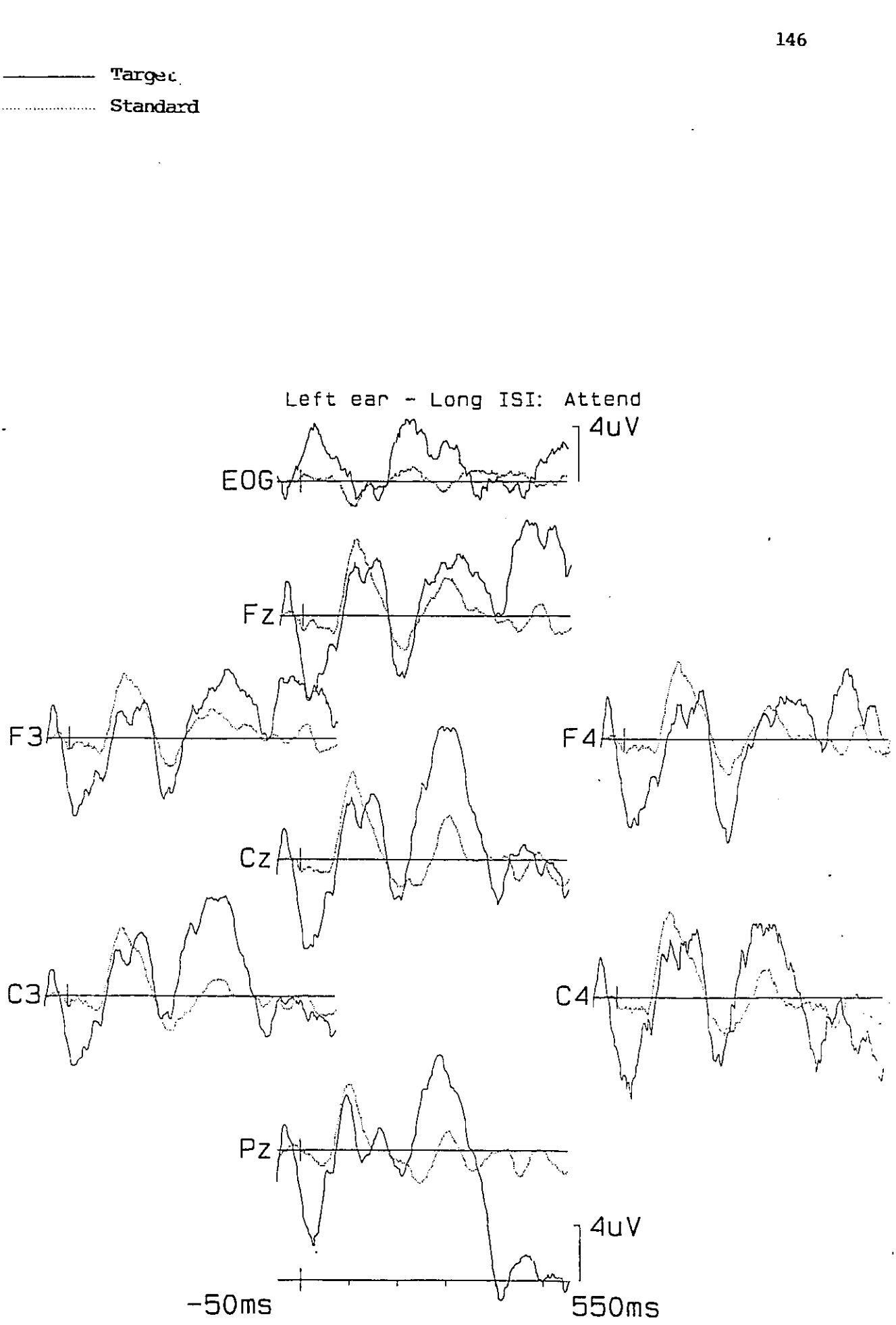
Electrode Placement.

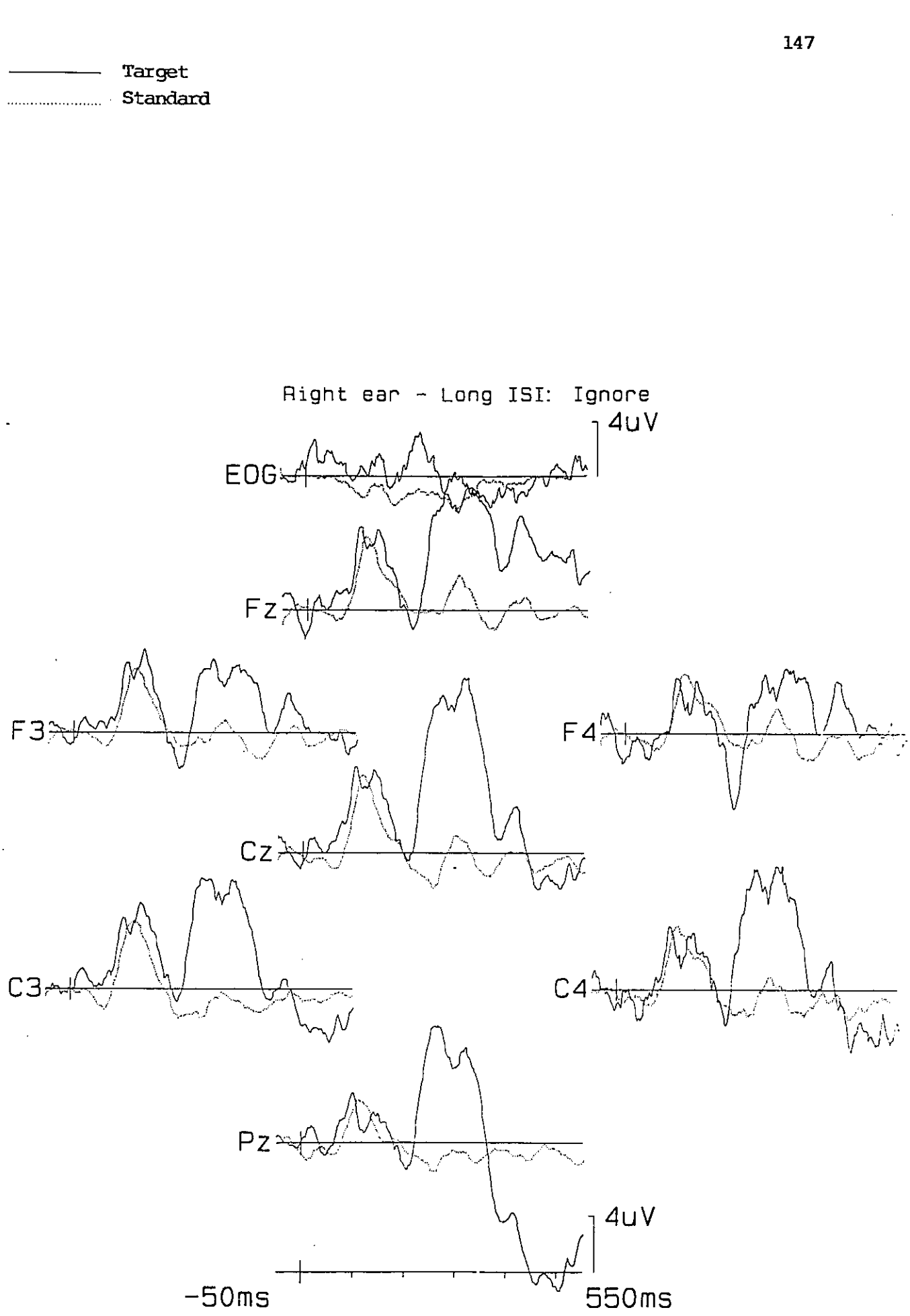


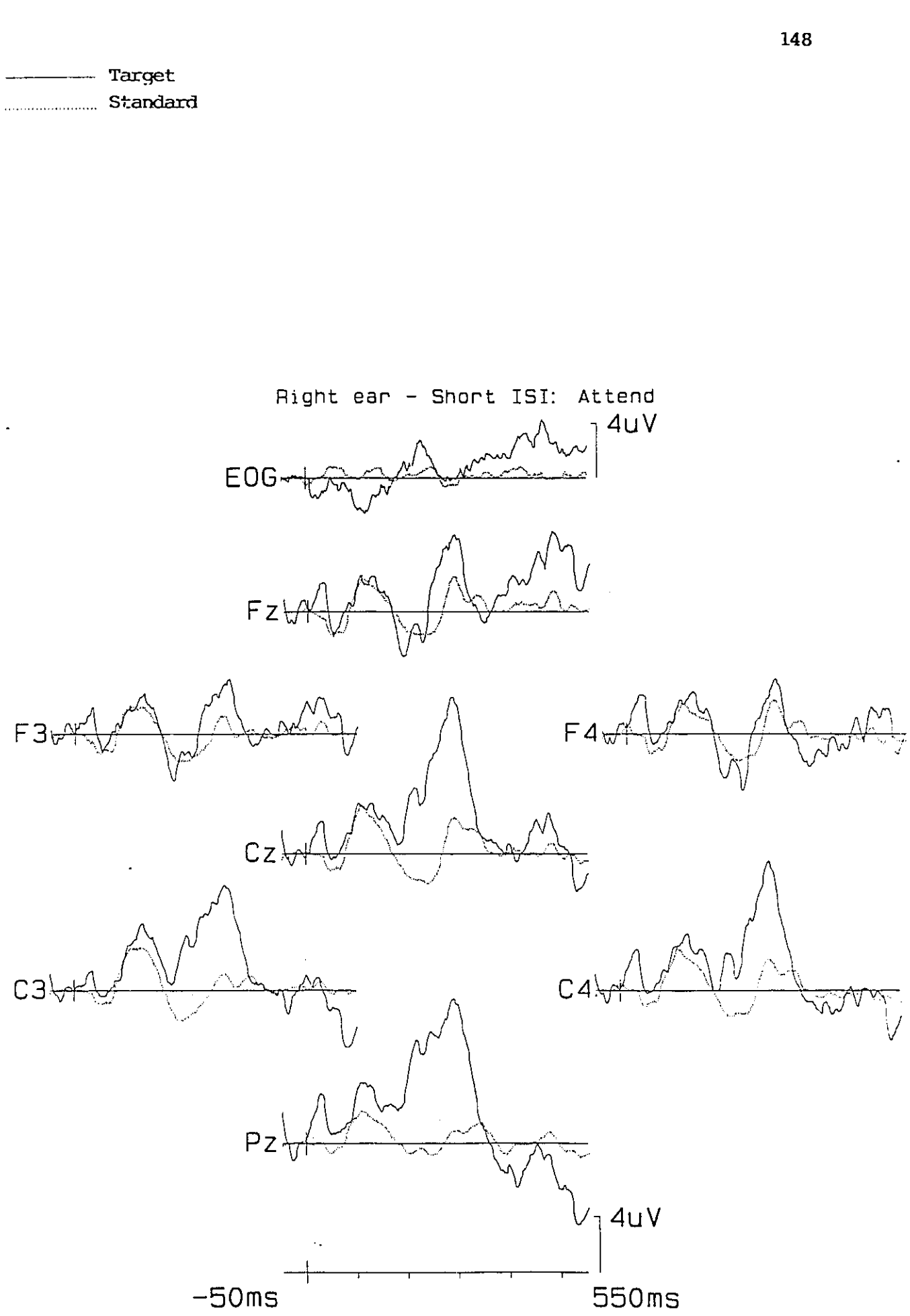
Modified combinatorial nomenclature.


Target
Standard
Left ear - Short ISI: Attend
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

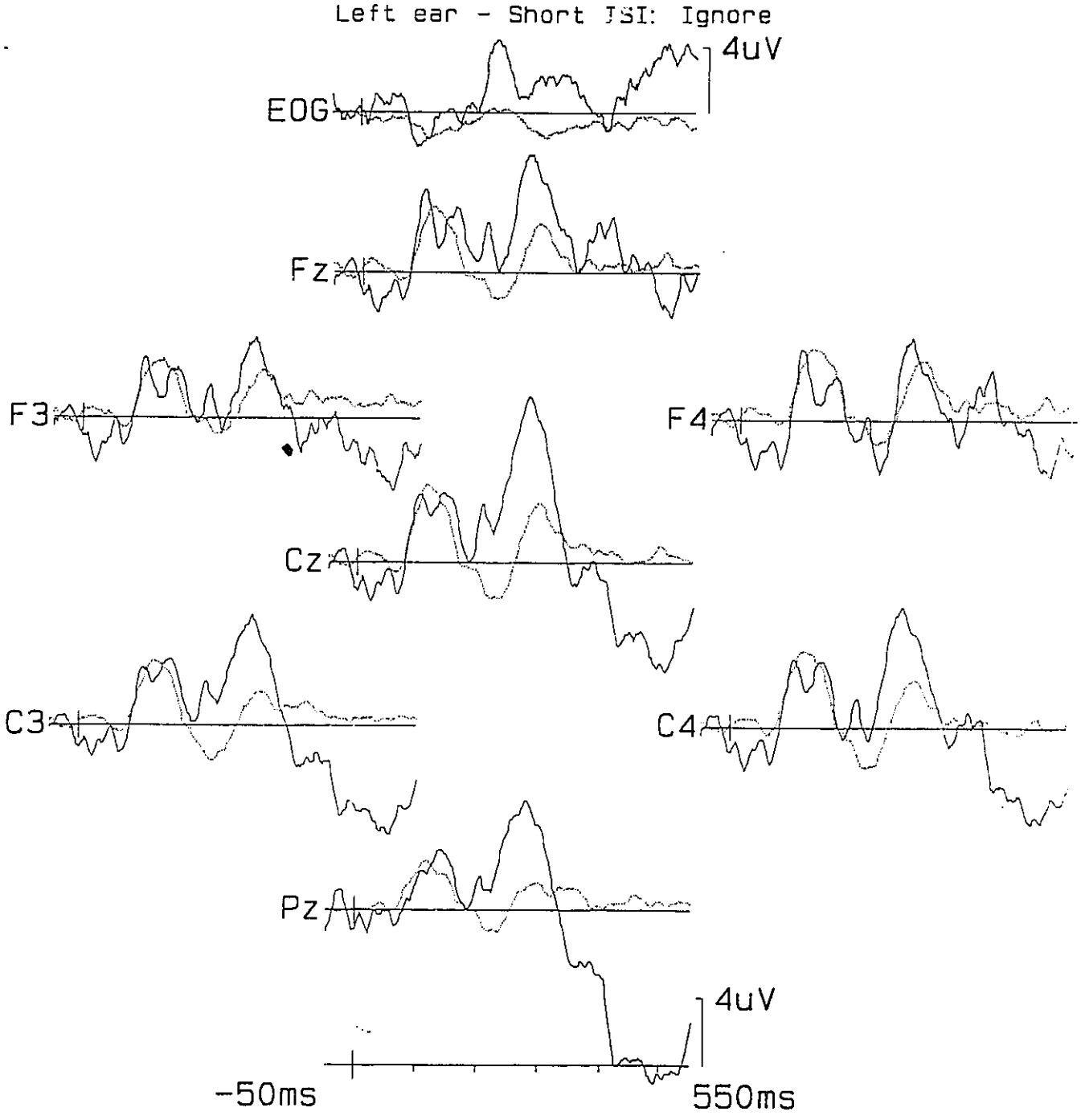

Target
Standard
Right ear - Short ISI: Ignore
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

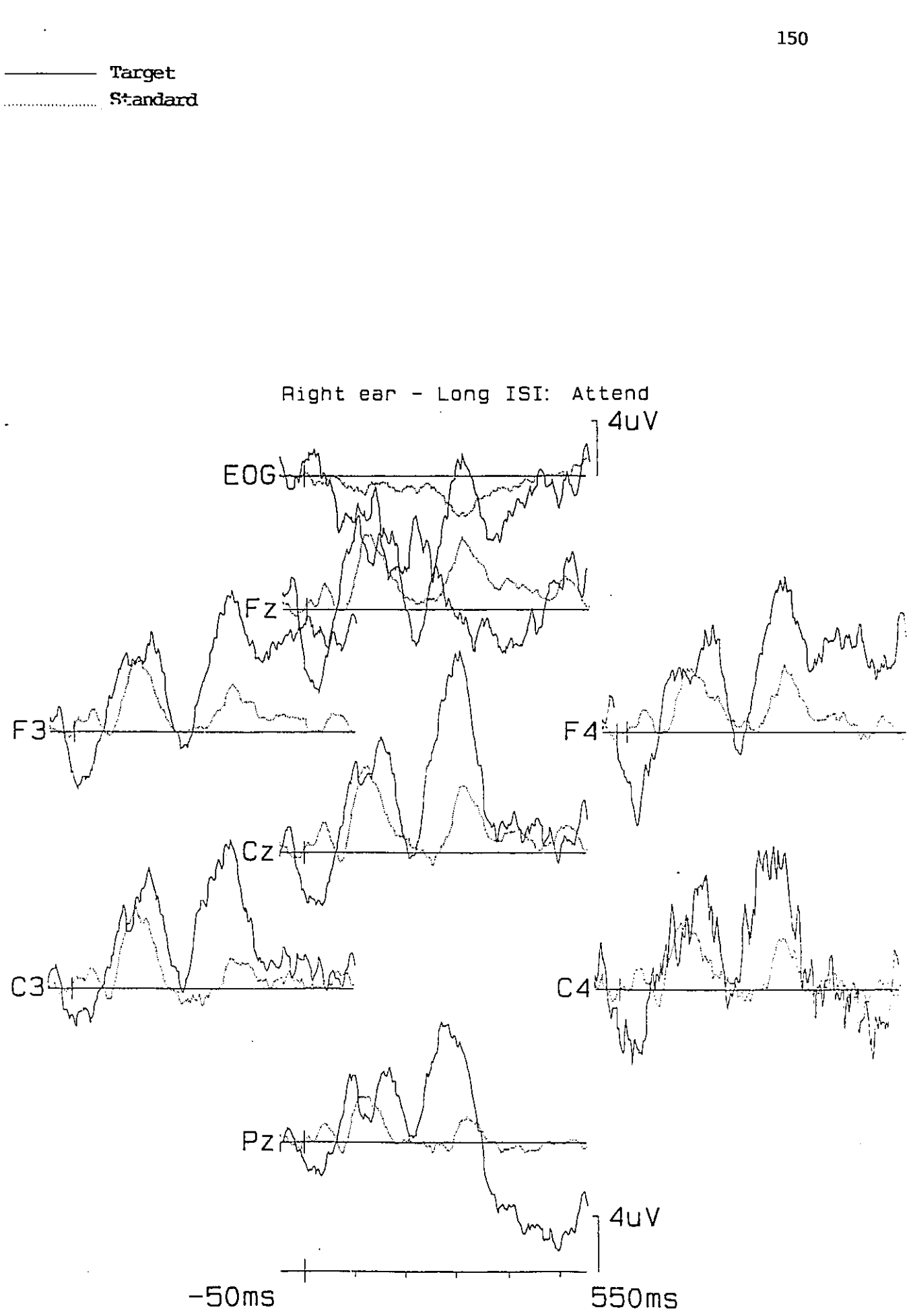

Target
Standard
Left ear - Long ISI: Attend
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms


Target
Standard
Right ear - Long ISI: Ignore
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

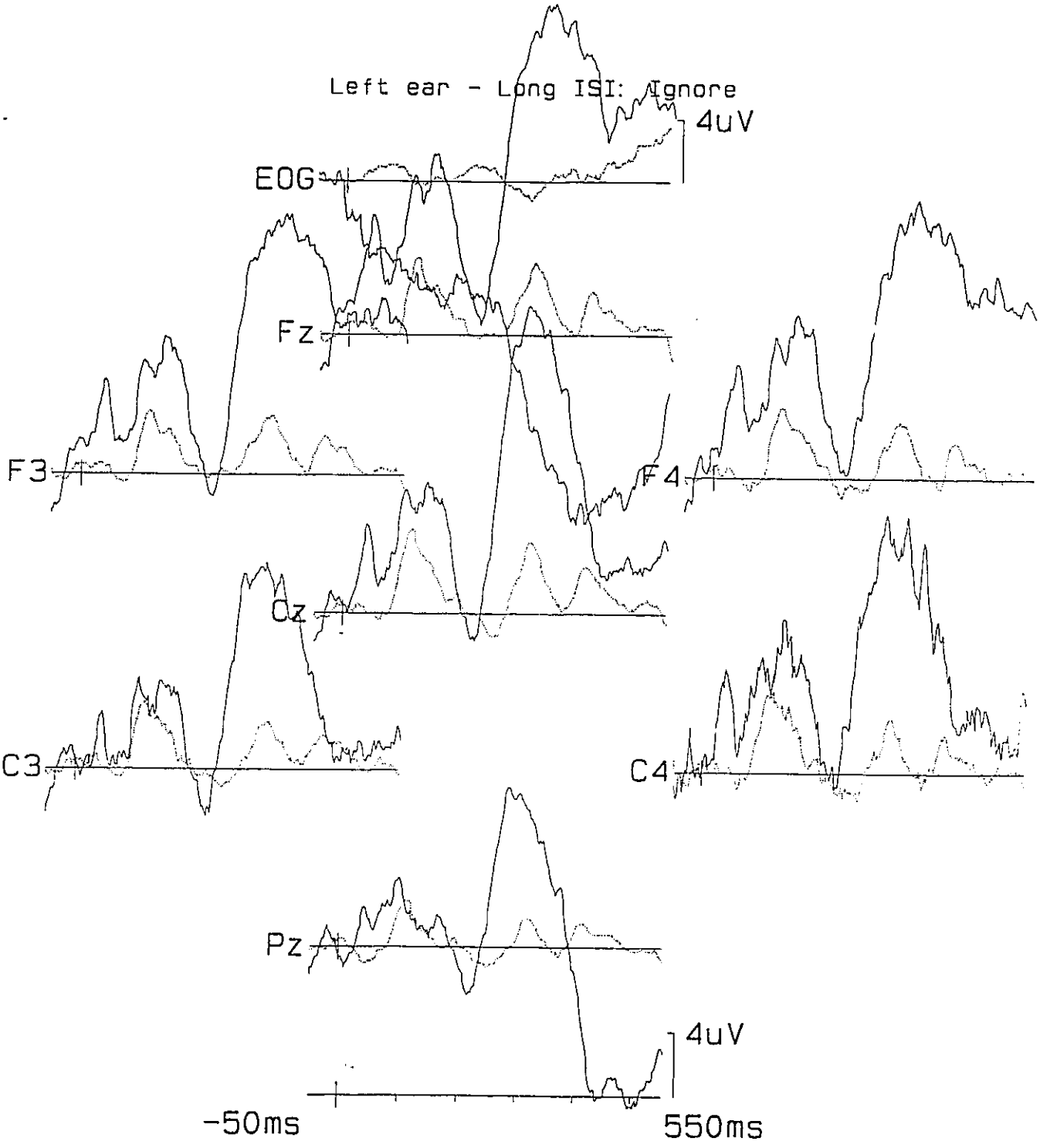

Target
Standard
Right ear - Short ISI: Attend
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

——— Target
..... Standard



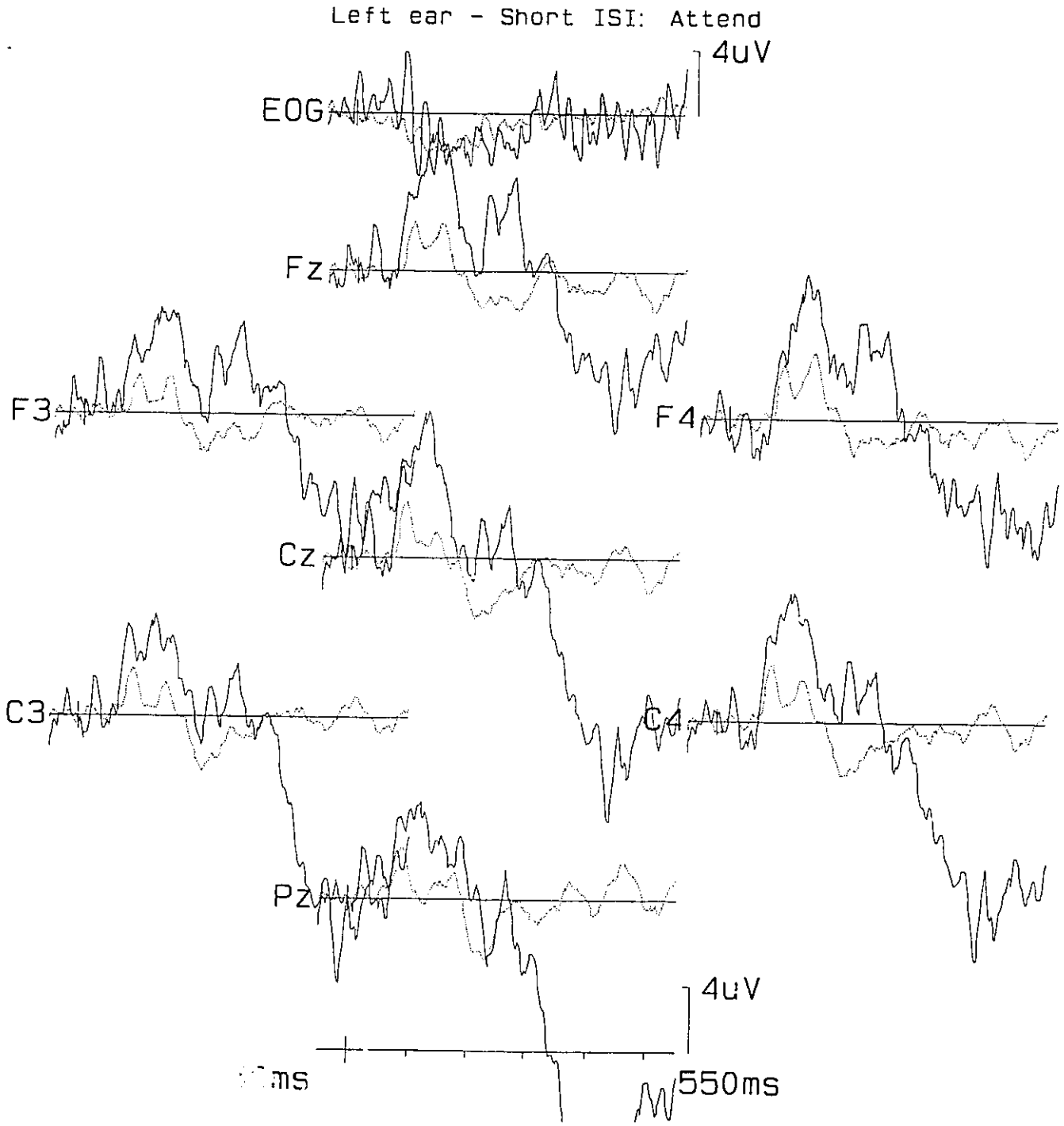

Target
Standard
Right ear - Long ISI: Attend
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

Target
Standard



Target

Standard



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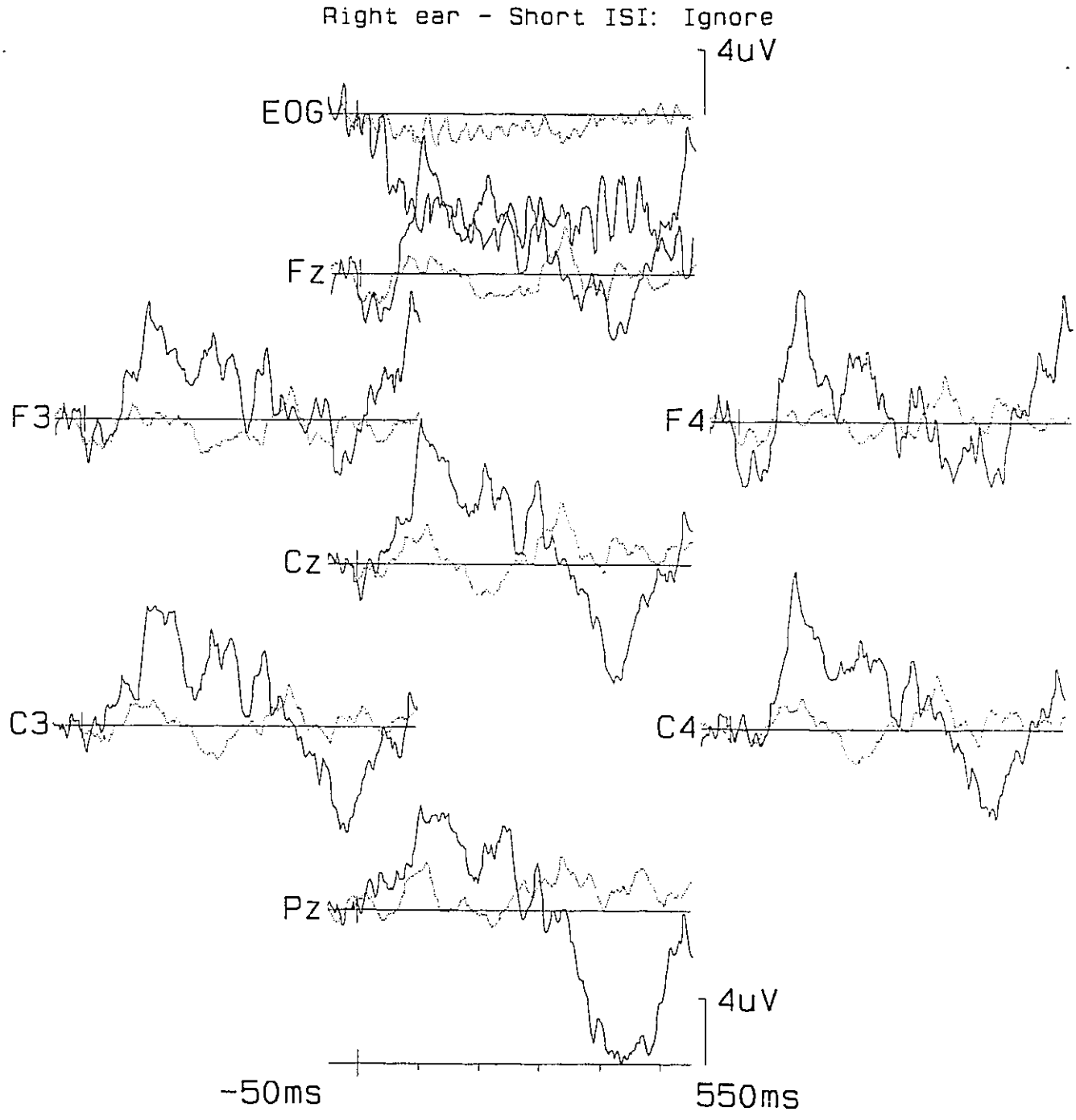

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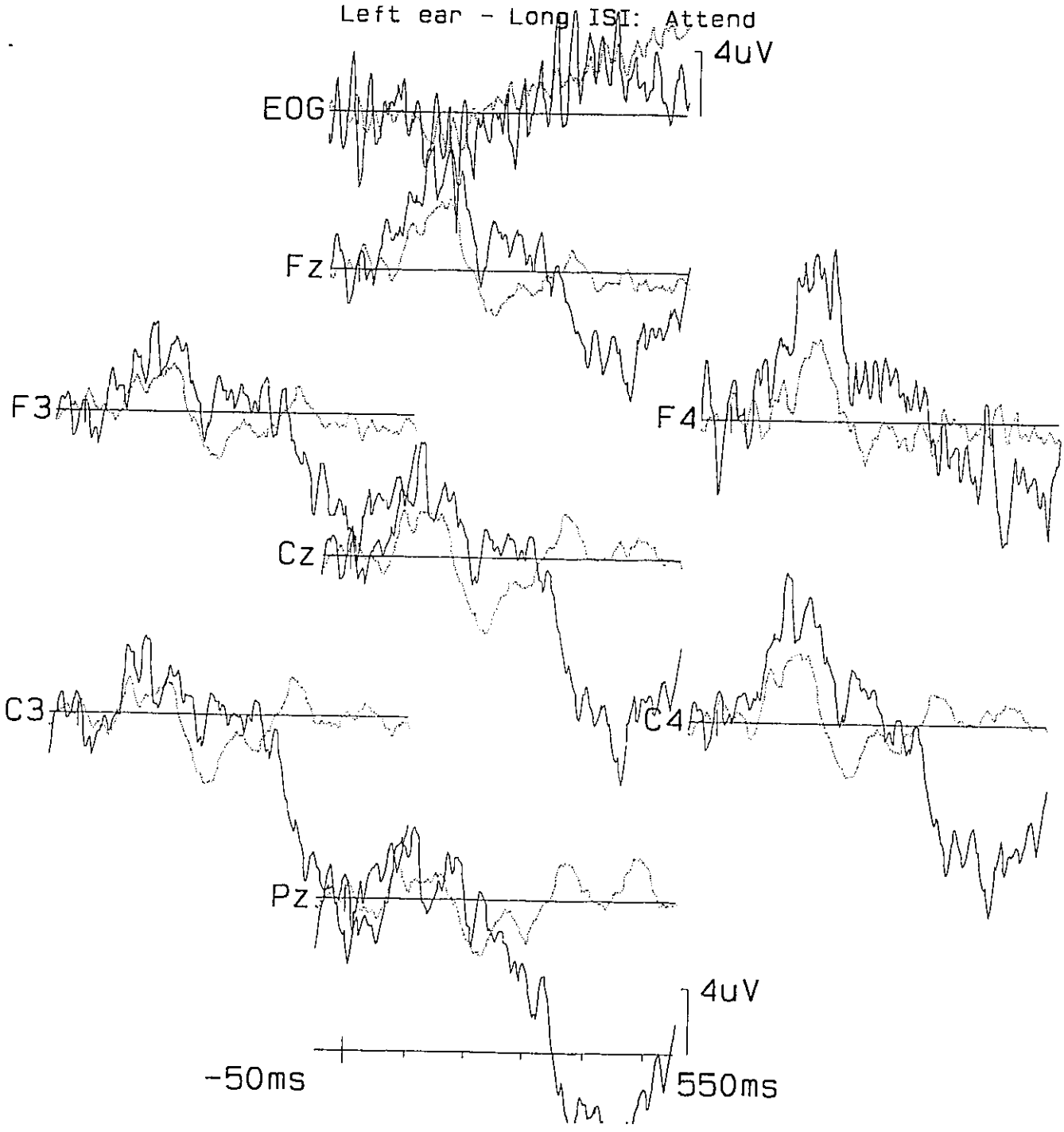

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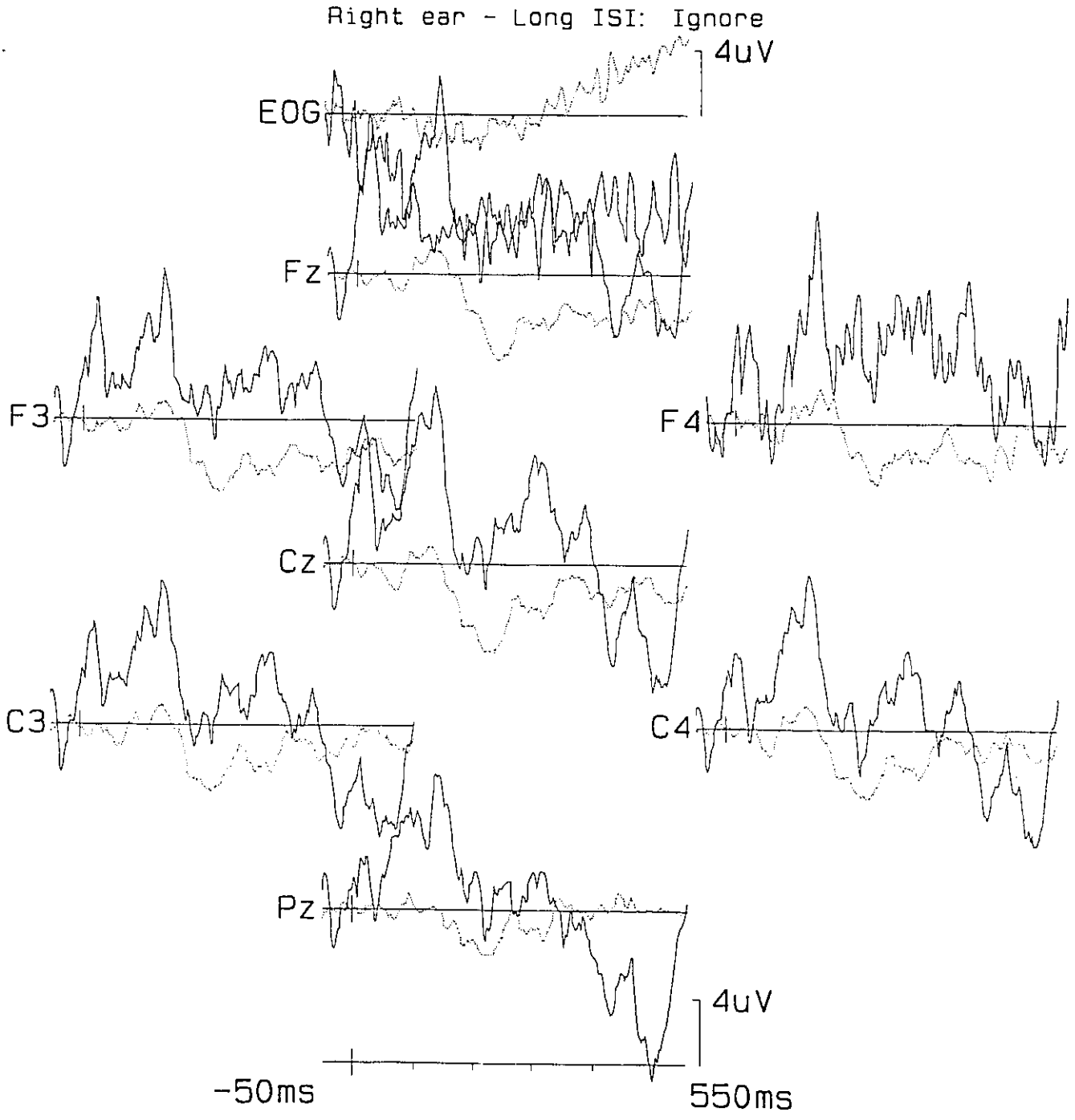
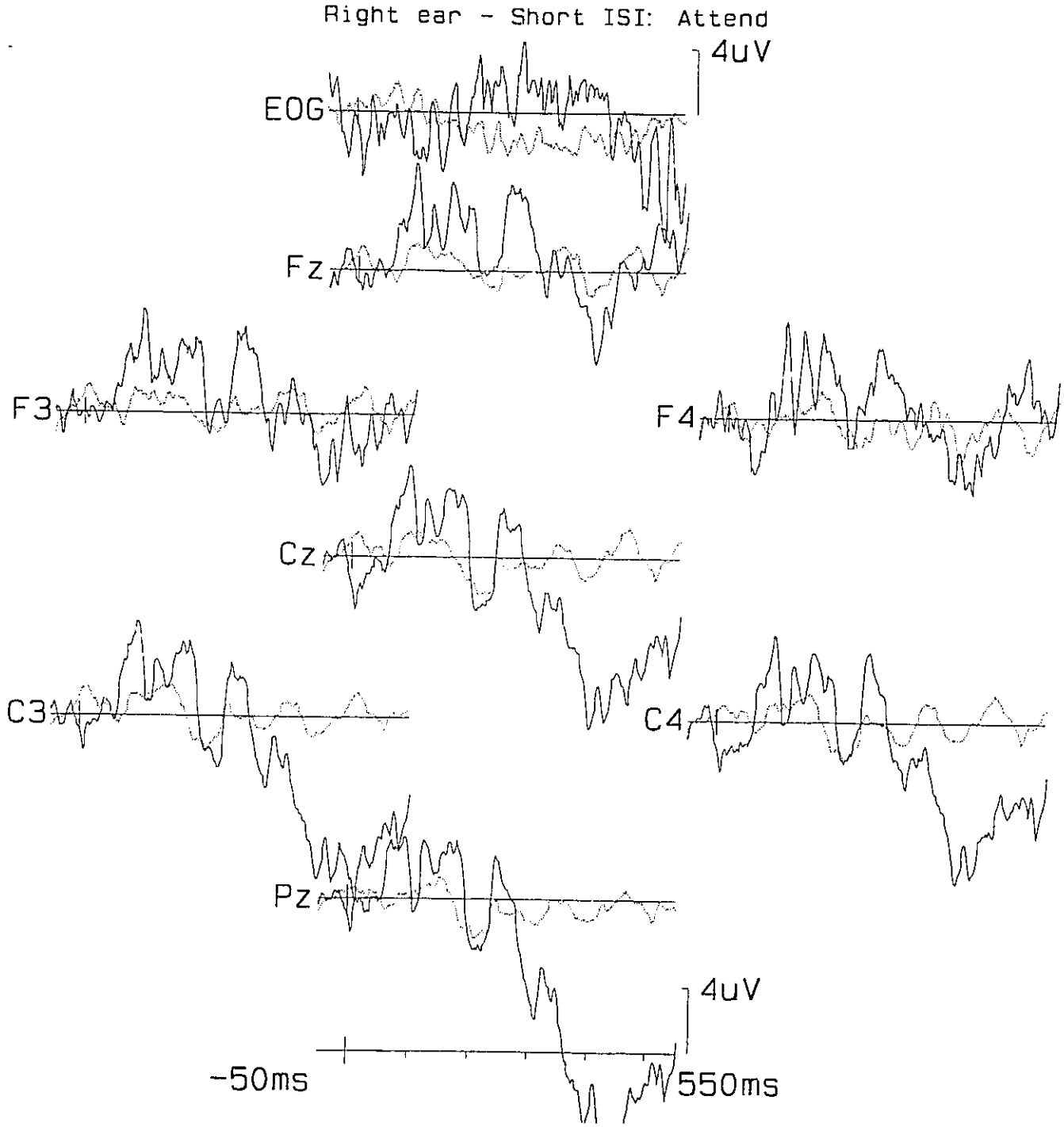


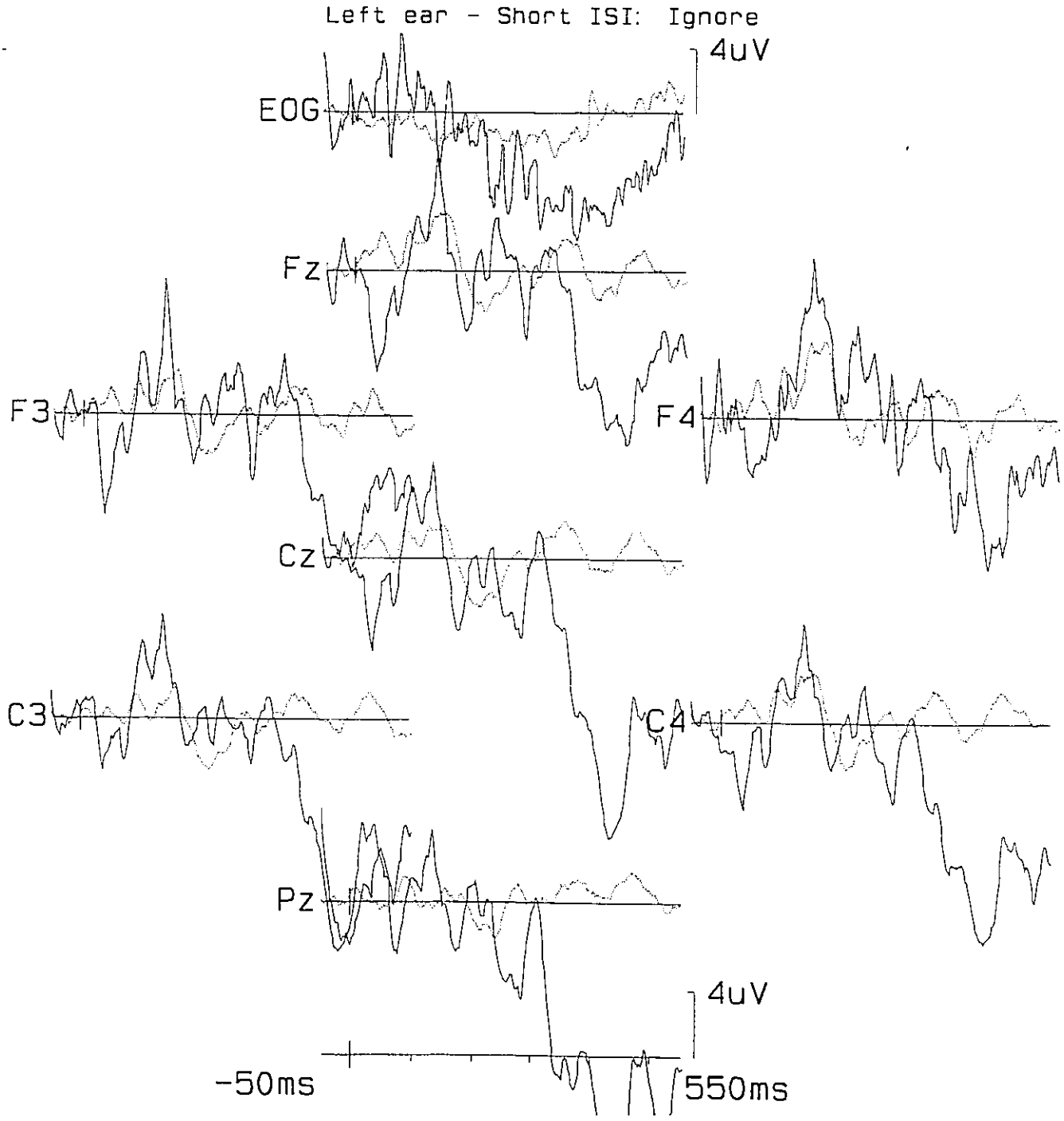
Target

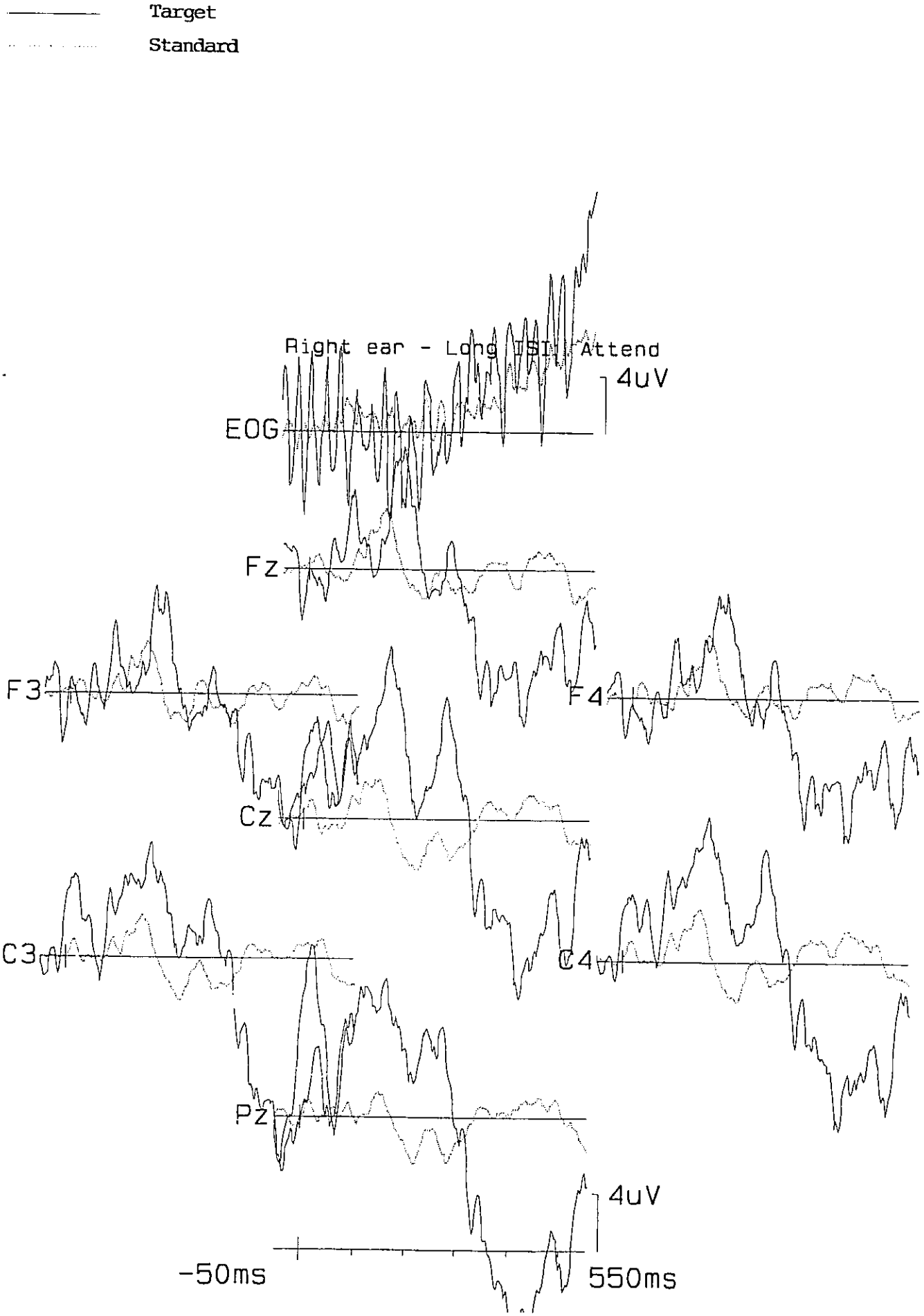
Standard



Target

Standard




Target
Standard
Right ear - Long ISI, Attend
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

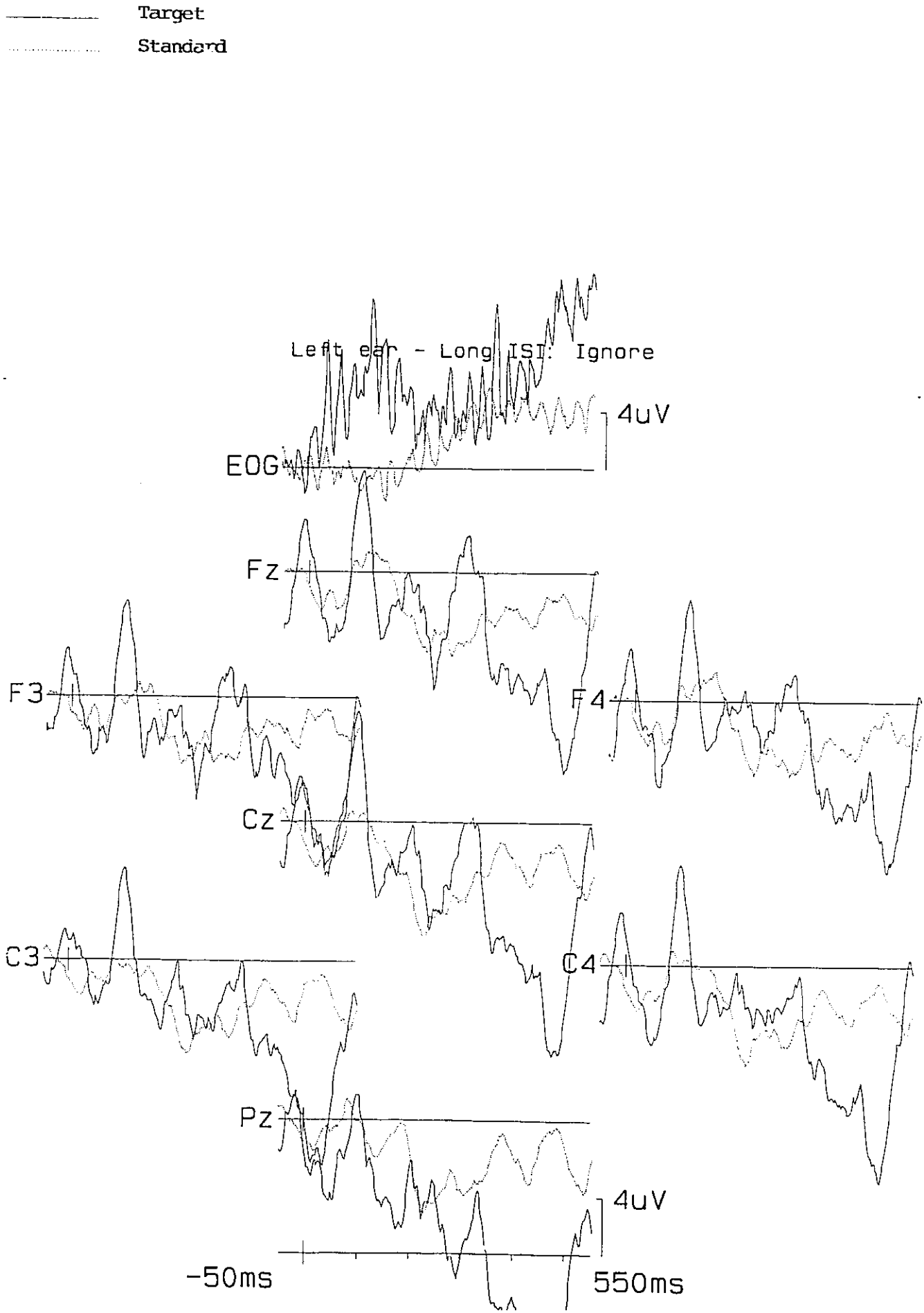

Target
Standard
Left ear - Long ISI: Ignore
4uV
EOG
Fz
F3
F4
Cz
C3
C4
Pz
4uV
-50ms
550ms

Table 6

Means (M) and standard deviations (SD) for schizotypal and contol groups on N1 latency to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	115.76	133.90	119.66	128.60	127.14	123.26	124.09	126.18
	SD	23.39	23.97	17.14	19.24	22.89	22.05	21.61	23.50
Ign	M	118.29	114.86	120.36	116.37	127.71	127.16	133.60	17.78
	SD	19.15	18.86	23.02	15.87	17.41	23.50	15.08	19.12
Cz									
Att	M	113.63	133.45	117.77	119.60	118.73	121.30	121.74	123.44
	SD	21.62	22.37	15.06	17.44	24.18	17.82	23.33	17.28
Ign	M	108.05	123.02	116.63	112.96	125.41	128.13	131.63	116.22
	SD	16.74	22.73	18.85	17.65	16.70	23.58	15.99	18.46
Pz									
Att	M	111.85	120.98	113.96	117.70	121.68	117.38	118.59	125.39
	SD	23.68	25.24	17.25	11.42	22.56	17.93	17.87	23.33
Ign	M	107.04	111.52	113.03	111.94	121.10	119.73	124.23	115.82
	SD	22.28	26.76	18.71	18.62	21.02	21.74	16.10	20.53
F3									
Att	M	118.32	133.28	127.14	124.56	123.45	125.78	129.49	130.27
	SD	24.21	22.63	16.74	22.27	23.62	25.26	24.33	20.07
Ign	M	116.65	121.30	117.03	120.84	126.95	127.15	137.90	116.62
	SD	20.96	20.05	21.02	27.07	17.98	25.48	16.35	23.11
F4									
Att	M	118.54	133.91	127.78	130.09	136.53	124.81	127.00	133.99
	SD	24.71	20.32	19.47	18.76	21.47	22.49	22.91	20.65
Ign	M	117.01	118.33	121.55	119.39	125.21	137.90	136.37	117.41
	SD	20.71	21.03	21.46	23.84	20.45	21.26	13.63	20.36
C3									
Att	M	116.76	132.70	121.81	121.30	131.65	126.37	137.80	134.36
	SD	25.20	22.58	16.30	18.43	15.09	20.95	27.78	18.41
Ign	M	114.27	125.35	118.14	119.85	129.09	128.75	125.81	115.83
	SD	15.06	22.44	19.16	22.35	15.54	22.96	16.36	22.83
C4									
Att	M	119.86	133.25	118.84	129.63	128.33	122.28	123.08	133.22
	SD	28.12	21.20	15.67	18.79	21.67	17.75	25.02	20.92
Ign	M	112.36	121.70	117.67	114.93	124.20	133.39	129.12	114.67
	SD	19.02	26.79	25.93	22.97	18.10	19.98	15.31	20.47

Note: Att = attended tone, Ign = ignored tone.

Table 7

Means (M) and standard deviations (SD) for schizotypal and contol groups on P2 latency to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	183.73	209.10	181.67	213.86	207.59	207.83	205.88	215.42
	SD	21.52	19.45	16.30	21.41	33.65	33.29	34.69	26.40
Ign	M	195.67	198.23	189.49	199.51	224.47	219.53	215.82	210.93
	SD	27.69	28.35	39.37	27.13	45.91	27.48	32.54	29.54
Cz									
Att	M	182.70	208.45	183.15	210.14	197.08	206.26	201.23	215.83
	SD	29.60	22.38	17.46	15.45	38.86	34.20	39.92	27.36
Ign	M	189.72	197.80	183.32	191.40	220.33	216.07	219.33	213.28
	SD	29.08	29.35	31.14	33.28	37.83	30.38	38.33	24.78
Pz									
Att	M	184.58	212.69	189.62	221.02	203.91	198.65	205.08	213.30
	SD	40.13	29.77	23.15	27.56	33.74	34.63	42.41	28.63
Ign	M	199.30	196.73	180.37	185.10	209.39	207.08	213.69	221.67
	SD	35.70	36.16	34.13	41.56	44.71	17.13	43.08	36.35
F3									
Att	M	176.92	215.91	186.55	219.13	201.94	211.73	218.95	214.47
	SD	28.22	27.74	23.25	18.30	43.09	35.11	31.21	31.11
Ign	M	199.93	205.03	177.76	200.35	222.08	217.18	218.58	220.69
	SD	37.71	33.89	42.84	35.88	44.22	27.22	35.35	28.24
F4									
Att	M	190.35	197.49	195.92	213.13	213.10	215.03	207.26	216.21
	SD	26.57	16.77	29.66	17.56	30.60	31.65	40.02	26.76
Ign	M	198.23	200.80	182.87	202.01	219.34	217.98	219.93	213.28
	SD	30.98	21.48	34.12	34.26	35.32	34.51	34.20	29.75
C3									
Att	M	172.20	212.08	180.08	224.00	206.05	210.76	212.34	212.28
	SD	37.08	29.28	26.62	27.75	24.16	34.07	32.67	24.47
Ign	M	200.36	194.61	186.72	193.11	229.30	217.38	224.82	216.21
	SD	37.56	27.82	35.86	36.61	38.60	33.92	34.98	26.39
C4									
Att	M	185.00	210.55	179.93	206.74	204.49	217.25	206.48	212.30
	SD	24.72	26.60	26.10	28.50	31.33	37.42	37.84	31.22
Ign	M	191.84	195.25	183.12	192.47	220.90	217.58	212.70	213.67
	SD	26.34	37.35	33.62	37.23	43.04	31.07	29.00	29.70

Note: Att = attended tone, Ign = ignored tone.

Table 8

Means (M) and standard deviations (SD) for schizotypal and control groups on N2b latency to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	242.97	272.16	244.49	268.14	263.67	262.69	254.29	269.72
	SD	37.48	40.59	41.42	43.14	52.80	41.71	46.02	26.08
Ign	M	248.29	260.45	255.53	268.75	280.47	288.09	274.03	279.52
	SD	48.90	44.57	36.34	44.18	35.53	24.06	35.57	33.68
Cz									
Att	M	238.49	262.77	236.86	271.54	269.72	251.75	252.14	262.93
	SD	48.38	46.84	43.63	35.00	52.19	47.82	42.52	38.09
Ign	M	245.95	270.04	252.57	270.24	266.87	282.99	281.65	277.74
	SD	53.28	30.68	46.51	42.40	50.85	27.18	33.85	40.57
Pz									
Att	M	244.43	262.63	243.72	255.97	259.38	262.63	246.71	262.30
	SD	52.70	45.06	44.85	28.62	44.05	42.74	52.78	44.33
Ign	M	243.82	250.39	247.67	262.92	279.46	272.43	281.15	282.88
	SD	55.55	43.37	42.75	49.72	48.67	33.96	35.37	40.91
F3									
Att	M	231.07	281.25	252.40	279.82	266.40	266.95	262.49	269.13
	SD	48.13	40.98	44.39	43.96	51.24	47.01	41.71	40.17
Ign	M	246.39	261.29	249.36	265.78	276.97	280.47	282.76	269.73
	SD	52.08	40.64	47.82	49.31	39.10	29.30	31.87	44.23
F4									
Att	M	239.99	261.12	246.64	272.17	264.65	266.40	260.97	270.31
	SD	48.91	47.89	41.80	39.07	46.13	46.85	57.61	26.34
Ign	M	248.71	269.17	257.00	258.09	280.08	281.05	281.25	278.53
	SD	50.82	47.10	41.33	49.67	38.27	26.14	33.34	34.98
C3									
Att	M	230.60	265.13	229.60	278.76	264.84	265.47	267.58	266.83
	SD	55.92	48.72	47.78	44.03	42.94	39.30	38.53	37.86
Ign	M	242.53	255.98	254.68	268.29	275.39	283.89	277.84	285.16
	SD	52.98	35.79	43.64	41.66	35.62	26.14	31.89	38.99
C4									
Att	M	235.73	262.83	235.35	257.94	262.35	259.39	259.02	268.76
	SD	46.31	53.08	44.28	27.84	42.24	40.11	35.55	27.18
Ign	M	244.87	250.23	250.00	266.84	275.38	275.98	268.05	278.73
	SD	50.71	51.39	39.14	57.03	38.26	27.10	39.30	41.53

Note: Att = attended tone, Ign = ignored tone.

Table 9

Means (M) and standard deviations (SD) for schizotypal and control groups on P3a to target tones.

Amplitude

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	-0.71	-0.68	0.21	-2.11	-1.55	0.48	-1.61	0.09
	SD	3.86	5.26	3.55	7.53	4.98	3.48	5.26	4.25
Ign	M	-0.11	-1.00	-2.63	-0.93	-2.34	-1.48	-0.25	-2.04
	SD	6.23	4.23	4.72	4.45	4.85	3.12	2.46	4.09
F3									
Att	M	-0.86	-0.68	0.[illegible]	-0.41	-1.55	0.14	0.35	-0.05
	SD	3.04	3.38	2.9[illegible]	5.73	4.14	4.21	4.03	4.57
Ign	M	1.47	-0.52	-0.63	0.45	-1.04	-0.49	-0.48	-1.45
	SD	4.38	3.48	4.13	3.36	4.08	3.09	3.71	3.15
F4									
Att	M	-0.21	-0.11	-0.96	-0.59	-2.05	-0.83	-0.71	-1.67
	SD	2.94	5.32	3.64	6.30	4.54	4.15	4.64	4.11
Ign	M	-0.20	0.61	-3.07	0.51	-2.42	-1.73	0.08	-1.27
	SD	4.67	3.84	3.35	2.78	3.64	2.76	2.95	2.66

Latency

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	299.99	300.58	291.78	298.65	282.60	303.06	293.70	307.96
	SD	53.84	51.23	48.01	30.38	43.99	38.15	42.35	39.48
Ign	M	301.77	317.78	315.43	315.43	288.77	289.06	293.65	305.60
	SD	39.92	28.08	44.45	31.36	44.03	45.72	34.58	36.35
F3									
Att	M	267.69	307.75	291.34	310.70	297.21	298.45	299.23	296.23
	SD	45.30	37.47	50.47	39.75	55.99	47.41	49.84	45.36
Ign	M	288.57	287.06	280.26	300.07	302.18	310.16	319.73	297.85
	SD	41.47	37.97	47.57	43.76	43.48	34.38	37.71	47.23
F4									
Att	M	288.14	297.09	279.68	304.06	288.28	293.94	286.41	298.06
	SD	45.25	35.24	45.79	34.15	53.95	51.18	54.88	25.14
Ign	M	285.36	314.98	281.68	296.02	310.98	302.34	310.98	311.13
	SD	43.98	60.00	40.13	40.52	36.30	29.91	29.45	38.35

Note: Att = attended tone, Ign = ignored tone.

Table 10

Means (M) and standard deviations (SD) for schizotypal and control groups on P3b latency to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	368.67	374.43	364.07	377.62	391.00	378.87	377.94	376.29
	SD	52.29	35.90	34.74	40.27	37.72	31.45	35.05	31.92
Ign	M	372.52	373.60	378.70	380.70	392.78	392.18	379.91	386.13
	SD	46.84	36.28	37.60	50.33	33.77	33.82	43.96	31.04
Cz									
Att	M	369.53	373.80	372.15	385.29	387.70	373.44	375.34	378.69
	SD	65.19	48.83	61.20	40.25	35.49	35.07	27.34	26.35
Ign	M	369.96	379.54	386.15	386.28	391.92	381.46	382.07	389.65
	SD	40.90	31.45	42.07	40.11	42.21	26.83	43.42	33.78
Pz									
Att	M	400.86	381.72	384.95	388.30	415.63	364.66	391.24	391.38
	SD	62.05	37.56	48.55	37.20	35.37	51.55	30.82	28.14
Ign	M	386.15	377.65	393.19	394.59	392.78	395.38	383.59	399.62
	SD	37.26	32.77	37.23	40.33	26.14	37.96	40.67	25.51
F3									
Att	M	336.94	377.00	349.92	380.40	390.63	389.98	393.00	384.97
	SD	59.11	47.05	53.41	49.41	36.91	43.63	36.28	34.49
Ign	M	391.10	377.80	393.81	381.89	392.18	389.63	385.75	396.11
	SD	41.75	25.31	41.21	44.18	33.94	35.93	47.19	33.02
F4									
Att	M	365.88	373.04	372.29	372.12	393.18	381.23	379.17	372.87
	SD	51.29	53.68	37.57	45.47	42.85	32.72	40.27	26.85
Ign	M	374.13	366.19	370.17	370.11	386.13	369.15	384.02	386.01
	SD	50.61	42.96	37.63	43.44	32.09	41.27	49.53	46.61
C3									
Att	M	362.70	377.74	383.17	378.05	398.83	389.64	390.68	385.71
	SD	53.79	48.02	41.77	38.45	35.69	36.43	31.33	25.16
Ign	M	374.71	370.39	388.50	395.70	388.29	391.78	374.03	388.82
	SD	35.18	26.98	37.23	37.23	34.03	31.59	41.31	48.92
C4									
Att	M	380.45	348.61	382.01	378.48	408.01	385.16	387.42	388.65
	SD	71.45	124.40	45.35	39.58	35.23	29.00	20.64	22.19
Ign	M	382.54	372.76	386.80	389.35	385.74	377.18	386.91	385.35
	SD	41.02	39.33	38.91	40.81	29.80	48.53	43.15	30.10

Note: Att = attended tone, Ign = ignored tone.

Table 11

Means (M) and standard deviations (SD) for schizotypal and control groups on SNV latency to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	514.42	490.34	498.49	495.08	487.49	491.22	489.94	504.49
	SD	19.69	41.50	39.69	28.78	32.03	26.46	35.73	25.41
Ign	M	479.48	468.96	487.36	453.48	481.89	482.81	491.80	479.10
	SD	48.21	43.94	36.11	37.52	34.66	37.33	39.71	39.65
Cz									
Att	M	479.05	465.49	448.49	462.26	484.53	481.72	490.07	501.14
	SD	45.79	41.87	63.21	34.92	35.72	35.17	39.90	44.55
Ign	M	453.05	458.59	479.27	461.24	483.61	476.18	485.74	493.29
	SD	42.37	38.73	38.36	36.71	32.48	46.64	35.69	34.29
Pz									
Att	M	400.86	381.72	384.95	388.30	415.83	364.66	391.24	391.38
	SD	62.05	37.56	48.53	37.20	35.37	51.55	30.82	28.14
Ign	M	386.15	377.65	393.19	394.59	392.78	395.38	383.59	399.62
	SD	37.26	32.77	37.23	40.33	26.14	37.96	40.67	25.51
F3									
Att	M	336.94	377.00	349.92	380.40	390.63	389.98	393.00	384.97
	SD	59.11	47.06	53.41	49.42	36.91	43.63	36.28	34.49
Ign	M	391.10	377.80	393.81	381.89	392.18	389.63	385.75	396.11
	SD	41.75	25.31	41.21	44.18	33.94	35.93	47.19	33.02
F4									
Att	M	473.08	476.94	489.36	489.66	475.19	493.48	485.13	498.75
	SD	57.94	42.11	47.35	39.33	38.06	32.83	36.10	29.25
Ign	M	494.83	460.50	478.42	450.53	487.66	475.99	475.20	478.55
	SD	41.98	35.74	42.07	40.72	34.67	39.36	34.34	38.90
C3									
Att	M	448.22	459.69	474.66	466.06	490.09	496.44	481.08	484.17
	SD	41.97	45.87	44.71	33.07	38.59	41.68	47.45	47.29
Ign	M	459.02	453.90	484.17	459.88	494.15	462.49	491.56	495.12
	SD	43.69	41.16	36.55	34.60	36.59	44.84	35.05	40.18
C4									
Att	M	469.46	467.50	478.10	445.45	477.09	491.61	471.53	487.08
	SD	61.14	44.39	43.91	27.49	37.20	28.75	34.09	42.84
Ign	M	474.16	458.61	486.31	467.34	485.16	484.54	493.95	476.00
	SD	47.60	35.30	39.68	32.36	32.78	38.19	34.90	37.66

Note: Att = attended tone, Ign = ignored tone.

Table 12

Means (M) and standard deviations (SD) for schizotypal and control groups on N1 amplitude to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	-7.81	-8.87	-8.17	-9.30	-7.98	-9.57	-8.96	-9.04
	SD	3.02	3.77	2.49	2.84	3.88	4.14	3.95	4.99
Ign	M	-7.22	-8.08	-8.35	-6.98	-8.32	-10.05	-8.62	-10.04
	SD	3.35	3.97	2.58	3.88	3.36	4.06	3.05	3.62
Cz									
Att	M	-7.70	-8.83	-8.41	-8.98	-8.72	-9.53	-8.18	-8.67
	SD	2.85	4.79	3.15	3.69	4.20	4.74	4.49	4.71
Ign	M	-6.96	-9.01	-8.45	-6.08	-7.39	-10.24	-8.60	-9.53
	SD	3.96	4.66	2.19	4.80	3.86	4.15	3.54	4.04
Pz									
Att	M	-6.25	-6.07	-6.76	-6.61	-6.56	-6.56	-5.65	-6.71
	SD	2.83	3.50	2.93	2.67	2.82	3.64	3.74	4.66
Ign	M	-5.06	-6.30	-6.47	-4.17	-5.31	-6.98	-5.56	-5.75
	SD	3.03	2.90	1.90	2.85	2.86	3.29	2.86	3.86
F3									
Att	M	-7.32	-8.22	-9.06	-9.27	-7.20	-7.84	-7.83	-8.76
	SD	2.23	3.02	2.98	2.51	3.50	4.13	3.95	4.75
Ign	M	-6.49	-8.13	-8.42	-7.23	-8.24	-8.48	-7.27	-8.70
	SD	3.26	3.50	2.82	3.02	3.42	5.02	3.39	3.39
F4									
Att	M	-8.05	-8.55	-7.67	-6.33	-7.96	-9.63	-7.72	-7.75
	SD	2.23	3.62	2.56	6.69	4.03	3.82	3.95	4.22
Ign	M	-6.85	-7.37	-8.78	-7.30	-8.15	-8.69	-8.20	-8.84
	SD	2.54	4.00	3.14	3.43	2.97	4.01	2.84	3.97
C3									
Att	M	-7.07	-8.29	-8.65	-8.69	-7.41	-7.44	-7.73	-8.97
	SD	2.74	3.38	3.06	3.56	3.22	3.68	3.27	4.59
Ign	M	-6.63	-9.02	-7.54	-5.87	-7.39	-8.83	-7.52	-7.86
	SD	3.82	4.01	2.09	3.97	3.01	3.61	3.08	3.22
C4									
Att	M	-8.35	-8.48	-7.61	-8.23	-7.86	-8.80	-7.48	-7.44
	SD	2.46	3.53	2.55	3.16	2.99	3.51	2.96	4.37
Ign	M	-5.82	-7.04	-8.26	-6.01	-7.33	-8.09	-7.21	-8.32
	SD	3.10	3.98	2.30	3.32	2.58	2.99	2.67	2.69

Note: Att = attended tone, Ign = ignored tone.

Table 13

Means (M) and standard deviations (SD) for schizotypal and control groups on P2 amplitude to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	-1.30	1.84	-0.29	0.19	-0.17	0.25	-0.63	1.75
	SD	2.98	5.27	5.37	3.67	5.32	4.80	6.25	4.56
Ign	M	-0.06	1.37	0.41	-0.48	-0.75	1.26	1.14	1.09
	SD	4.15	3.69	3.72	3.20	3.14	4.41	2.72	4.55
Cz									
Att	M	-0.33	2.76	-0.68	2.08	1.15	1.98	0.76	2.94
	SD	3.47	5.44	4.17	3.88	6.46	6.50	6.64	5.56
Ign	M	2.15	2.01	0.42	2.33	0.26	3.58	3.14	3.46
	SD	5.44	4.32	3.72	4.52	4.49	5.61	4.25	4.34
Pz									
Att	M	0.48	4.81	0.91	3.48	2.33	4.50	1.64	3.81
	SD	1.97	4.14	3.58	2.72	4.21	3.42	3.69	3.86
Ign	M	3.79	2.92	1.65	3.00	1.04	3.84	3.51	4.33
	SD	4.39	4.84	3.15	3.73	2.78	3.87	4.25	3.69
F3									
Att	M	0.11	2.69	0.62	1.70	-0.01	0.99	0.11	1.03
	SD	2.64	3.43	4.68	2.67	4.92	4.65	5.04	4.17
Ign	M	1.43	2.52	0.84	1.53	-0.54	1.39	1.62	0.50
	SD	4.44	3.71	4.72	2.40	3.04	4.81	3.31	4.22
F4									
Att	M	-0.91	1.49	0.50	0.66	0.03	0.83	-0.25	1.84
	SD	3.20	4.62	4.52	2.38	5.24	4.89	5.98	3.78
Ign	M	0.52	2.69	-0.67	0.63	0.26	0.69	1.03	0.93
	SD	3.97	4.58	2.95	2.48	3.83	4.05	2.93	3.45
C3									
Att	M	-1.77	2.31	-1.45	1.38	-0.04	1.71	0.35	1.06
	SD	2.70	4.75	4.16	2.50	4.55	3.49	3.77	3.58
Ign	M	0.53	1.10	0.92	1.18	-0.35	1.92	1.95	1.37
	SD	4.67	4.51	3.19	3.39	2.68	3.51	3.59	2.83
C4									
Att	M	-1.10	2.14	-0.72	-0.04	0.19	2.10	0.40	2.31
	SD	3.23	4.07	4.26	3.84	5.23	4.89	4.97	4.71
Ign	M	1.39	1.79	-0.41	1.08	0.25	3.16	2.04	2.12
	SD	3.73	3.58	2.67	2.97	3.28	4.70	3.53	3.01

Note: Att = attended tone, Ign = ignored tone.

Table 14

Means (M) and standard deviations (SD) for schizotypal and contol groups on N2b amplitude to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	-7.39	-5.00	-6.87	-7.37	-6.46	-5.05	-6.47	-3.72
	SD	1.85	4.44	3.86	6.04	5.24	4.28	4.68	5.09
Ign	M	-5.90	-5.24	-7.97	-6.18	-6.90	-5.75	-6.76	-6.60
	SD	4.97	2.79	3.63	3.57	2.76	2.65	4.22	4.43
Cz									
Att	M	-6.40	-4.69	-6.24	-5.13	-5.62	-3.19	-5.23	-3.09
	SD	3.60	4.90	4.10	5.99	5.89	4.84	5.06	6.41
Ign	M	-4.68	-5.18	-7.51	-4.68	-2.87	-4.34	-5.08	-4.56
	SD	5.92	4.70	3.81	4.47	7.86	3.58	4.63	5.31
Pz									
Att	M	-4.03	-1.05	-3.87	-1.64	-3.52	-0.86	-3.27	-1.06
	SD	2.69	4.77	3.60	4.15	4.94	4.68	3.88	5.08
Ign	M	-2.85	-2.54	-4.89	-1.58	-3.11	-0.86	-3.02	-2.24
	SD	4.00	3.79	2.92	3.68	4.54	3.42	4.67	3.75
F3									
Att	M	-6.80	-5.21	-5.70	-6.84	-5.62	-3.94	-4.94	-3.57
	SD	3.19	3.00	3.20	5.27	4.16	3.86	3.79	4.17
Ign	M	-5.06	-4.70	-8.25	-5.44	-5.53	-3.94	-5.48	-5.34
	SD	4.57	3.62	4.84	2.95	3.87	2.87	2.80	2.81
F4									
Att	M	-7.48	-5.11	-6.37	-6.61	-5.37	-4.67	-5.13	-3.51
	SD	1.46	3.17	3.96	5.34	4.85	4.43	5.13	4.60
Ign	M	-6.54	-4.57	-7.23	-6.37	-6.47	-4.79	-5.71	-4.87
	SD	4.02	2.94	3.92	3.67	4.43	2.49	3.73	4.00
C3									
Att	M	-6.56	-3.84	-5.14	-4.40	-6.33	-3.41	-5.45	-3.88
	SD	1.81	4.81	3.26	4.03	4.44	3.67	3.00	4.26
Ign	M	-3.91	-4.36	-5.97	-3.69	-5.20	-3.80	-4.69	-4.13
	SD	4.81	3.51	3.56	3.88	3.85	2.45	3.45	3.79
C4									
Att	M	-6.70	-3.56	-5.90	-5.12	-5.07	-2.62	-4.71	-3.23
	SD	2.27	3.41	2.86	4.42	4.16	4.10	4.26	5.51
Ign	M	-4.39	-4.23	-6.28	-3.56	-5.12	-3.56	-4.54	-3.57
	SD	4.34	3.38	3.06	4.16	3.57	2.57	3.51	3.67

Note: Att = attended tone, Ign = ignored tone.

Table 15

Means (M) and standard deviations (SD) for schizotypal and control groups on P3b amplitude to target tones.

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	1.47	3.03	2.91	1.47	4.53	4.70	4.22	6.85
	SD	4.02	5.52	2.80	6.20	5.29	5.30	6.59	4.43
Ign	M	0.68	1.29	-0.89	0.85	-5.79	3.03	3.69	3.49
	SD	4.03	5.12	2.39	3.74	6.51	3.55	3.48	4.15
Cz									
Att	M	3.07	5.70	5.51	4.42	7.22	9.26	6.83	9.98
	SD	3.13	7.05	6.08	6.15	7.71	6.86	7.19	6.00
Ign	M	3.61	2.66	0.82	4.74	4.81	6.21	5.69	7.60
	SD	4.09	5.53	2.34	4.87	4.87	4.49	4.12	4.38
Pz									
Att	M	9.65	10.67	10.05	11.15	11.75	12.83	10.00	12.22
	SD	4.92	6.51	5.01	6.47	8.61	6.43	6.06	6.44
Ign	M	8.35	8.25	5.51	9.20	7.17	9.28	7.84	10.22
	SD	3.97	5.73	1.97	5.26	5.54	4.91	5.62	4.74
F3									
Att	M	1.05	3.50	3.67	2.57	3.11	4.06	3.00	4.53
	SD	3.65	3.91	2.50	5.06	4.25	4.60	4.17	3.72
Ign	M	2.50	0.64	0.78	2.30	1.73	3.36	2.73	2.38
	SD	4.18	3.71	3.10	2.35	3.11	3.20	1.80	4.15
F4									
Att	M	2.70	3.32	3.48	2.04	3.78	4.23	2.84	5.17
	SD	3.54	4.28	3.80	5.27	4.64	4.46	5.31	4.10
Ign	M	1.20	0.98	-0.07	1.40	1.34	1.72	2.73	2.84
	SD	3.47	4.33	1.94	3.24	3.03	2.78	3.87	3.53
C3									
Att	M	2.28	4.93	5.53	4.63	5.20	7.11	5.42	7.31
	SD	4.16	6.64	4.13	5.55	6.75	5.83	5.29	5.44
Ign	M	3.97	3.60	1.65	4.70	3.79	5.90	4.68	5.50
	SD	3.90	5.54	2.40	3.83	4.03	3.32	3.89	4.07
C4									
Att	M	4.58	7.10	5.86	4.64	6.77	7.84	5.77	8.21
	SD	2.78	6.24	4.73	4.30	6.10	4.83	5.96	4.82
Ign	M	3.31	2.52	1.58	4.21	3.65	4.80	4.56	5.80
	SD	3.52	5.50	2.57	3.77	3.60	2.79	4.00	3.35

Note: Att = attended tone, Ign = ignored tone.

Table 16

Means (M) and standard deviations (SD) for schizotypal and control groups on SNW amplitude to target tones

		Schizotypal				Control			
		Left		Right		Left		Right	
		Fast	Slow	Fast	Slow	Fast	Slow	Fast	Slow
Fz									
Att	M	-8.29	-6.61	-7.09	-8.68	-4.73	-5.75	-6.57	-5.07
	SD	3.58	7.61	4.47	4.79	4.32	5.23	4.89	4.63
Ign	M	-6.58	-7.10	-9.74	-5.30	-5.53	-5.07	-4.20	-3.95
	SD	3.70	4.78	2.52	3.71	1.72	3.56	2.35	4.58
Cz									
Att	M	-3.34	-2.84	-0.26	-3.41	-0.76	-1.56	-2.09	-1.53
	SD	5.25	10.37	7.26	7.19	6.77	6.71	5.63	5.20
Ign	M	-4.33	-4.58	-6.06	-1.36	-3.40	-2.91	-2.08	-0.89
	SD	4.84	4.59	3.20	6.34	3.12	3.16	3.70	5.30
Pz									
Att	M	4.39	4.60	3.68	5.45	6.26	4.91	4.27	3.92
	SD	4.92	6.76	6.33	6.88	7.06	5.71	4.92	4.76
Ign	M	0.70	2.31	-1.07	3.59	-0.24	2.38	1.98	4.38
	SD	3.53	4.56	3.06	5.10	3.56	3.11	4.93	6.25
F3									
Att	M	-7.84	-5.95	-5.83	-7.36	-4.03	-4.38	-5.41	-4.84
	SD	4.30	4.59	4.00	3.84	4.40	4.82	4.05	4.60
Ign	M	-4.57	-6.82	-8.32	-4.45	-4.80	-3.10	-3.15	-3.10
	SD	4.19	5.10	2.99	3.01	1.85	2.58	1.80	4.20
F4									
Att	M	-5.69	-4.63	-5.68	-6.48	-3.05	-3.26	-5.42	-3.47
	SD	3.29	6.18	4.03	3.94	3.08	2.96	3.78	3.77
Ign	M	-5.90	-6.13	-9.04	-5.12	-5.85	-5.13	-4.10	-4.15
	SD	4.35	4.42	2.49	3.19	1.96	3.24	1.86	3.83
C3									
Att	M	-2.83	-1.26	-1.13	-2.29	-1.36	-0.59	-2.18	-1.99
	SD	4.06	7.80	5.47	4.50	5.80	4.91	4.40	4.34
Ign	M	-2.39	-3.27	-4.30	-0.88	-2.96	-1.39	-0.56	-0.69
	SD	4.07	4.40	2.02	4.30	1.69	2.68	2.77	3.83
C4									
Att	M	-1.45	0.65	-0.70	-1.79	1.03	0.91	-0.32	0.71
	SD	3.97	8.05	4.89	4.46	4.47	3.73	4.49	4.43
Ign	M	-2.76	-2.99	-5.50	-1.14	-3.36	-2.06	-1.53	-0.73
	SD	3.48	3.84	2.82	3.85	1.54	2.35	2.60	3.87

Note: Att = attended tone, Ign = ignored tone.