Event-Related Potential Characteristics in
Children of Alcoholics from High Density Families

Shirley Y. Hill, Ph.D.*
Stuart Steinhauer, Ph.D.**
Jennifer Park, B.A.*
Joseph Zubin, Ph.D.**
Timothy Baughman, B.A.*

* Alcohol and Cognitive Processing Laboratories
  Western Psychiatric Institute and Clinic
  University of Pittsburgh School of Medicine
  3811 O'Hara St.
  Pittsburgh, PA 15213

** Biometrics Research 151R
  VA Medical Center
  Highland Drive
  Pittsburgh, PA 15206

Please send reprint requests to: Shirley Y. Hill, Ph.D.
ERP in High Risk Offspring of Alcoholics

Galley proof should be sent to:
Shirley Y. Hill, Ph.D.
Alcohol and Cognitive Processing Laboratories
Western Psychiatric Institute and Clinic
University of Pittsburgh School of Medicine
3811 O'Hara Street
Pittsburgh, PA 15213

ERP, P300, frontal negativity, high risk, genetic markers, vulnerability to alcoholism
ABSTRACT. Sons and daughters of male alcoholics were compared with sons and daughters of controls employing two auditory paradigms to elicit event-related potentials (ERPs). Enhancement in frontal negativity and deviations in P300 amplitude and latency were seen in the children of the alcoholic fathers from high density families (each father had an average of 3.7 first and second-degree relatives meeting criteria for alcoholism) when compared to controls.
Introduction

Recently a great deal of attention has been focused on event-related brain potentials (ERPs) as possible biological markers for alcoholism risk. It is now well established that chronic alcoholics exhibit deviations in ERP characteristics (Porjesz and Begleiter, 1982; Porjesz et al., 1980). That genetic mediation of risk occurs in at least one type of alcoholism is also now established (Cloninger et al., 1981), though clearly vulnerability to developing alcoholism involves the interaction of both genetic and environmental factors (Hill et al., 1987).

ERP components which are evoked following a stimulus event are typically identified by the polarity of the scalp recorded peak (e.g., "N" for negativity, "P" for positivity) and by the approximate latency at which the peak typically occurs across experiments. Thus, P300 refers to a scalp-positive component which occurs at approximately 300 msec. Components with latencies of 200 msec or less (e.g., P50, N100, P200) tend to be associated primarily with the physical characteristics of the stimuli that are presented, although N100 amplitude also is related to general levels of attention. N250 has been interpreted as the initiation of the process reflecting the discrimination of one of several possible stimulus events (Ritter et al., 1979).

Particular interest has been directed at the examination of the P300 component of the ERP, with reference to such variables as the complexity of the task and modality of information delivery (Donchin, 1979; Sutton and Ruchkin, 1984). P300 has been related to a vast number of processing activities, such as stimulus evaluation, comparison with internal templates, or updating of information to be used in future predictions, and does not seem to be produced by any unitary phenomenon that is consistent across different experiments.

The first report by Elmasian and colleagues in 1982 of ERP abnormalities in
high risk nonalcoholic individuals was an important first step in demonstrating that information processing characteristics might distinguish those at risk from those who were not. In that study, a complex auditory task was used to elicit the P300 component of the ERP both before and after the administration of either a placebo, light or heavy dose of alcohol. In all three conditions, the subjects with a positive family history (FHP) for alcoholism showed a reduced amplitude of the P300 component relative to those without such a history (Family History Negative, FHN).

A number of subsequent studies have provided somewhat varied findings. Neville and Schmidt (1985) reported that no changes in amplitude could be found using the same subject population but without the alcohol administration paradigm. However, the peak latency of the P300 component was found to be longer in the FHP group but not the FHN group. Interestingly, the longer latency was reported to be associated with the typical quantity of alcohol consumed per occasion. In another study employing an auditory paradigm and two levels of task difficulty, no differences in ERP latency or amplitude were apparent as a function of family history of alcoholism (Polich and Bloom, 1987). While the previously mentioned studies have employed adult young men who are assessed in an auditory paradigm, other negative findings have been reported for adult young men, employing a visual paradigm (Polich et al., in press).

Previous reports have been somewhat negative where adult young men have been assessed, in contrast to more positive findings for minor children (Begleiter et al., 1984; Whipple and Noble, 1986). However, unlike these largely negative findings reported for adult subjects, we have reported differences in ERP characteristics of adult men from high risk families when contrasted with low risk (control) subjects (Steinhauer et al., 1987; Hill et al., in press). The subject population studied has included individuals from high density families where multiple cases of alcoholism are found. This is in
contrast to the usual methodology employed in the family history positive/family history negative strategy where a single first degree relative qualifies one for membership in the "high risk" (family history positive) group. Realizing that residual effects of alcohol consumption might have subtle effects on information processing, though correlations between P300 amplitude and latency and recency of alcohol use have not been found (Hill et al., in press), to rule out any confounding effect of alcohol use we have, in this study, begun testing the minor children of subjects previously described (Hill et al., in press) who represent the third generation for whom ERP data is being collected as part of a large scale pedigree study.

Methods

Two groups of children were tested: children of alcoholic fathers and children of normal controls. The alcoholic fathers were selected from treatment facilities if they had a diagnosis of alcoholism and were free of other DSM-III (Axis I) psychopathology. Families were included only if all first degree relatives met criteria for absence of other psychopathology with the exception of sociopathy which was present in some families.

Alcoholism was determined through use of a structured interview which included portions of both the Diagnostic Interview Schedule and the Renard Diagnostic Interview, allowing for determination of whether or not the individual met DSM-III or Feighner Criteria for alcoholism. Additionally, the families of the alcoholic probands were required to meet certain structural characteristics for inclusion: the presence of at least one other alcoholic male sibling and one nonalcoholic male sibling, and at least one parent. For the present study, a child between the ages of 8-16 was also required. Thus, three generations of clinical interview data were available for the families of these
children, yielding an average of 3.7 first and second degree alcoholic relatives in each family.

Control families were selected from among volunteers who responded to an advertisement in local newspapers, which did not indicate that alcoholism was the focus of the study. Adult male social drinkers were selected who had at least one male sibling and parents available for clinical interview and testing. In these families no first or second degree relative met criteria for Axis I psychopathology, including alcoholism.

All of the mothers from both the affected and control families were free of alcoholism and other psychopathology by family report (mothers were not interviewed directly).

Twenty-two Caucasian children between the ages of 8 and 14 were tested. There were seven girls and four boys in the affected group, and eight girls and three boys in the control group. The mean age (+ S.E.) was 10.63 (+ 0.56) for the children of the affected fathers and 10.81 (+ 0.73) for controls. The mean age of the fathers in the two groups did not differ (mean age = 37.38 + 0.49 for the affected group and 37.88 + 0.42 for the controls). The education of the fathers did differ, however (t = 2.44, df = 14, p = .03), with the affected fathers averaging 12.88 years (SE = 0.07) and the controls 14.63 (SE = 0.13).

Procedure

The ERP assessment consisted of presentation of two successive tasks: a Counting task and a Choice Reaction task. Both involved a modified version of the oddball paradigm commonly employed in other ERP studies (Goodin et al., 1978; Donchin, 1979; Pfefferbaum et al., 1984). In the Counting task, subjects were presented with high pitched tones (1500 Hz) or low pitched tones (800 Hz) at 65 dB, 40 msec in duration, through a speaker placed in front of the subject.
who sat in a sound attenuated chamber in a darkened room. In this task, one
tone was presented every 3 seconds for a total of 80 tones (trials) per block.
Subjects were first asked to identify sample tones as "high" or "low" in
pitch. The subject was then instructed to silently count the number of "high"
tones (targets) but not the number of "low tones" (non-targets) at the end of
each block. Subjects were told (1) that there would be fewer high than low
tones, and (2) that there would never be two high tones in succession. To
insure that subjects understood the paradigm, each subject was asked before
recording commenced to relate what would occur next if a high tone was heard.
All subjects reported that the next tone would be a low tone.
The sequence of tones was generated randomly by computer so that the
overall probability of a target (high tone) was 0.25. The random sequence was
restricted only by the requirement that no two high tones occur in succession.
Between 17 and 23 high tones occurred during each block.
A total of six blocks of trials were given each subject, four Counting
tasks blocks and two Choice Reaction task blocks. Approximately 2 minutes was
given for rest between blocks. During the Counting task blocks, subjects
silently counted the number of high tones, reporting the number of targets heard
at the end of each block. In the Choice Reaction task, two blocks of trials
were given in which the subject did not count tones but instead was asked to
make a motor response to indicate what he or she had heard. On one block,
subjects were instructed to press a button with their left thumb if a high tone
was heard and with their right thumb if a low tone was heard. The correct
response was reversed during the other block of trials so the a high tone
signified a right press and a low tone a left thumb press. On the first block,
a high tone was associated with a left thumb press for half of the subjects, the
other half with the right thumb.
Psychophysiological Recording

The electroencephalogram was recorded with Ag/AgCl electrodes from midline frontal, vertex, parietal and occipital locations (Fz, Cz, Pz, Oz) and modified left and right parietal (P3 and P4) locations, referred to linked ears, using a bandpass of 0.01 to 30 Hz, with digitized samples obtained every 8 msec for a 1200 msec epoch beginning 200 msec prior to stimulus onset. An additional channel monitored eye movement artifacts allowing those trials containing artifacts to be removed. Average ERPs were computed from artifact-free trials. All recording, storage and data analysis was performed by digital computer.

While oddball tasks are most typically analyzed according to rare and frequent events, there are clear effects based on sequences of stimuli (Squires et al., 1977; Tueting et al., 1971). Therefore, data were analyzed by segregating stimuli according to their second order sequence, which is the conditional probability of each event established by the event on the prior trial. High tones are labeled as Targets (T) and low tones as Non-Targets (NT). When a Non-Target was preceded by a Target, then its occurrence was predictable, that is, the conditional probability was equal to 1.00 (labeled NT/t). However, once a Non-Target had occurred, then the next trial was a target one-third of the time, and a non-target two-thirds of the time (conditional probability for NT/nt = .67). Averages based on these three conditional probabilities were calculated for each subject for each task.

Analysis of ERP Data

ERP components were identified using a computer algorithm which initially selected peak latencies and amplitudes (relative to a baseline defined as the median voltage for the 200 msec prestimulus period), with verification by a
rater blind to the diagnosis of the subject. Through this interactive algorithm, P50, N100, P200, N250, and P300 components were identified and checked for each subject in both tasks (these components can also be described by the actual mean latencies observed across subjects and tasks: P61, N107, P180, N257 and P376).

Results

Amplitude data were subjected to analysis of variance with repeated measures over event probability (3 levels) and electrode location (6 levels) for each task condition (modified by the epsilon correction factor for degrees of freedom where appropriate). Latencies were similarly treated using only event probability as the repeated measures factor. Latencies for each component were obtained at the electrode for which the component amplitude was greatest. Interactions between event probability and family type were examined for simple main effects at each of the three probability levels. (Tables 1A & B, 2A & B summarize the results of the ANOVAs for both tasks.)

Insert Tables 1A & B and 2A & B about here

Amplitudes: The analyses indicated significant differences in amplitude for each component across different scalp locations in both tasks (p = .0002 for P50 in the Choice Reaction task, p < .0001 for all other analyses; detailed analyses of the main electrode effects are omitted). For the P50 and P200 components, maximum positivity was observed at the midline vertex (Cz) electrode, where N100 was most negative. N250 showed the greatest negativity at the midline frontal
location (Fz). The P300 component was most positive at the midline parietal location (Pz). Averaged waveforms across the subject groups are presented in Figures 1 and 2.

Insert Figures 1 and 2 about here

The P50 component, which is associated with early processing activity of the auditory stimulus, did not reveal differences for the two groups. The P50 component showed a significant interaction between Probability and Family Type in the Choice Reaction task only (p = .0245). Controls showed greater positivity in the 1.00 compared to the .33 condition, while high risk children showed an opposite effect of slightly higher positivity in the .33 and .67 conditions than in the 1.00 condition. Differences between the two subject groups were not significant at any of the individual probability levels. The functional significance of these differences between groups is not clear.

Because of our interest in determining if there were differences in attentional capacity between the children of alcoholics and controls, amplitude data for N100 were analyzed. No differences in N100 were seen between the two groups of children as a function of family history of alcoholism. However, a trend for greater negativity was seen in the high risk children at the vertex electrode. A main effect for Probability was found for N100 in the Choice Reaction task (p = .0402). Greater negativity occurred in both the .33 and 1.00 conditions than in the .67 condition.

Analysis of the P200 data revealed no differences between groups. However, a main effect for Probability in the Choice Reaction task (p = .033) was related to increasing positivity associated with increasing event probability. Interactions between Probability x Electrode for both the Counting (p = .0002)
and Choice Reaction tasks (p = .0015) reflected a reduction in positivity for the .33 condition compared to the .67 and 1.00 conditions, with the greatest difference occurring at the vertex electrode.

It was of interest to analyze possible differences in N250 between groups because of reported differences among children and adults (Friedman et al., 1984). The N250 component showed a main effect for Probability (p = .0182) in the Counting task, with greater negativity in the .67 condition compared to .33 and 1.00 conditions. For Counting, an interaction between Probability x Electrode (p = .0373) was due to greater negativity in the .67 condition than the 1.00 condition, with the .33 falling in between at the frontal scalp location, but less negative than the 1.00 condition at other electrodes.

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Insert Figures 3 and 4 about here
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In addition, interactions between Family Type x Probability were seen in both tasks for N250. No differences at any specific probability level were significant for either task; rather, controls were characterized by highest negativity in the .33 condition, while high risk subjects showed the least negativity in the .33 condition.

In general, greater negativity, beginning at approximately N250, can be observed in Figures 1 and 2 at the frontal electrode for the high risk children as compared to controls. Similar negativity for the high risk children also appears at vertex in the Choice Reaction waveforms. A significant interaction between Family Type x Electrode was seen for the Counting task (p = .0083), which reflected the increased negativity for the high risk children at frontal and vertex locations. However, a similar trend (p < .09) was observed for the
Choice Reaction task. The findings for the Counting task were further investigated by running an ANOVA using only the data at the frontal electrode in this task. A main effect for Probability ($F(2,34) = 6.84, p = .005$) reflected the findings described above across all electrodes. Of greater interest was a significant interaction between Family Type x Probability ($F(2,34)= 6.43, p = .006$); simple main effects indicated greater negativity ($p < .001$) for high risk than control children in both the .67 and 1.00 probability conditions.

As expected, the P300 component was increased in amplitude with decreasing event probability for both tasks ($p < .0001$) regardless of subject group. The larger differences in probability at the Pz electrode than at other locations were reflected as significant interactions between Probability x Electrode ($p < .0001$).

Insert Figures 5 and 6 about here

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Significant interactions with Family Type were observed in the Counting task. An interaction between Family Type x Electrode ($p = .0457$) reflected greater negativity in the anterior than posterior scalp locations, with significantly greater negativity ($p < .05$) at the frontal electrode for the high risk children compared to controls. This appeared to be a prolongation of the negativity observed initially with the N250 component for the high risk group.

An interaction between Family Type x Probability in the Counting task ($p = .0129$) indicated a greater rate of decrease among the high risk children in P300 amplitude as event probability increased. In the .33 condition, the high risk children actually showed larger mean P300 amplitudes than controls, but amplitudes for controls were more positive than for the high risk group in the
.67 and 1.00 conditions. In looking only at the maximum amplitude location, the midline parietal electrode, the effect of Probability (F(1,28) = 47.14, p < .0001) and interaction between Family Type x Probability (F(1,28) = 5.19, p = .0142) were maintained. Analysis of the interaction indicated that high risk children showed significantly greater P300 amplitude in the .33 condition but smaller amplitude in the 1.00 condition compared to controls (p < .05); though non-significant, P300 was also smaller in the high risk children for the .67 condition.

Latencies: Among the earlier components no significant effects were found for P50 or P200. For N100 mean latencies for the high risk children were shorter than for controls in both the Counting task (p = .0298; affected = 99 msec, controls = 110 msec) and Choice Reaction task (p = .0346; affected = 100 msec, controls = 118 msec). The shorter latencies for the high risk children were not observed in any subsequent components. No differences by group were seen for N250 in either task. In the Counting task, the latency of N250 for the .33 condition (246 msec) was significantly shorter than for the .67 and 1.00 conditions (both averaging 260 msec) (p = .0253)

For P300 latency, main effects for Probability were seen in both tasks (Counting: p < .0001; Choice Reaction: p = .0017). Latency was longest for the .33 condition, decreasing as event probability became more certain (Figures 7 and 8). In the Counting task, an interaction between Family Type x Probability (p = .0083) reflected a greater decrease in latencies going from the .33 to the 1.00 condition for the control than for high risk children. While P300 latency for controls was longer in the .33 condition and shorter in the .67 and 1.00 conditions than for the high risk group, the differences were significant only for the .67 and 1.00 conditions (p < .05). Though no significant differences were seen between groups for the Choice Reaction task, a similar direction of
changes was observed at the .33 and 1.00 probability levels.

Insert Figures 7 and 8 about here

Discussion

Evaluation of event-related potentials in children of alcoholics compared to children of controls revealed two major findings: an enhancement of frontal negativity in the children of alcoholics, and deviations in P300 amplitude and latency under conditions of minimal task engagement among the high risk children.

Increased frontal negativity in high risk children compared to controls has not been reported previously. This was a robust finding apparent in both tasks. While a significant difference in negativity began with N250, the negativity was prolonged and still showed greater anterior scalp differentiation between groups even at the latency at which P300 was observed. This negativity appears to be the classical N250 described by Ritter et al. (1979). As noted by Fitzgerald and Picton (1983), N250 tends to be more closely related to discriminability between stimuli, but is not particularly responsive to monotonic changes in event probability. In the present study, an increased N250 was observed in the .67 condition, but there was no monotonic relationship between event probability and N250, which is therefore consistent with previous reports. While negative shifts are often seen to begin earlier in the temporal sequence of events, no significant difference in negativity was found for the earlier N100. However, a more negative though nonsignificant N100 component
could be observed for the high risk children at the vertex electrode. The likelihood that the difference at N100 is related to the later negativity at N250 seems minimal given the clear lack of differentiation between the groups for the intervening P200 component.

The frontal maximum N250 for children has previously been reported by Friedman et al. (1984). While N250 has a more typical vertex maximum in adults, Friedman et al. noted the prominence of the frontal N250 in children, which decreased in negativity not only through adolescence, but which they also observed in other data (e.g., Pfefferbaum et al., 1980) to continue to decrease in negativity throughout the life span. This frontal negativity has been viewed as being associated with the development of more posterior (parietal) Slow Wave, and its decrease has been suggested as reflecting maturational development (Friedman et al., 1984). Consequently, the findings for greater negativity in the children of alcoholics in the present study suggest an interesting hypothesis: the high risk children may be showing an electrocortical sign of maturational lag. While the groups did not differ in age, however, it would be most critical to examine larger groups of high risk children and controls at each of several ages. Larger samples are also needed to control for possible sex differences: Friedman et al. (1984) noted greater negativity for N250 among males than females, although in the present study, almost equal numbers of males and females comprised each group.

P300 was clearly characterized in both groups and for both tasks as increasing in amplitude and latency as the conditional probability associated with each event decreased, an inverse relationship. This is similar to findings with adult populations (Steinhauer and Zubin, 1982; Steinhauer et al., 1987; and Hill et al., in press). The effects of probability for P300 are not accounted for by differences in earlier components. For example, P200 showed reduced positivity for the .33 condition in both tasks, and N250 showed reduced latency
for the .33 condition in the Counting task. Other significant effects on probability for earlier components were not monotonically related to event probability. It is interesting to note that while P300 amplitude was maximal for both groups at Pz, a greater absolute difference between groups can be observed at Cz. This may be due in part to overlap from the frontal negativity described above.

The high risk children showed decreased P300 amplitudes and increased P300 latencies in the .67 and 1.00 conditions of the Counting task, with similar trends for the Choice Reaction task. These are the conditions which require the least overall engagement in processing allocation. The subjects were not required to either count the stimuli or to initiate an overt motor response. In contrast, the presentation of the target in the .33 condition of the Counting task involved active attention and memory updating. In the latter situation, the high risk children were actually seen to show significantly greater amplitudes than the controls.

Similar findings for P300 latency in these tasks is seen among the affected fathers of these children (Hill et al., in press). We have suggested that at low levels of task demand, the adults are showing signs of greater delay in physiologically processing an event and as task demand is increased, normalization of processing activity occurs.

A parallel but more complicated interpretation may be applied to the children's data. Under minimal task demand where both increased latency and decreased amplitude of P300 were seen, greater time was also required for the completion of stimulus evaluation (i.e., the latency increase). Furthermore, the attentional significance of these events appears to have been reduced, as indexed by decreased P300 amplitude. Thus, increasing task demand apparently tended to normalize P300 amplitude for the high risk children, suggesting that demand provided sufficient motivation or focusing of attention to alter the
response. In fact, in the Counting task which is less demanding relative to the Choice Reaction task, this process (increased focused attention) resulted in an overcompensation leading to a significant increase in P300 at the .33 probability condition.

One of the implications of these findings is that the high risk children are not processing increasingly complex information along a continuum. Instead, they appear to show a larger jump in processing activity with initial increases in task demand. It is conceivable, then, that they show a curvilinear function relating task demand and neurophysiological performance. In this case one would anticipate that under even more demanding (complex) tasks high risk children would begin to show increasing deficits after some optimal level of performance had been exceeded. Thus, this conceptualization could account for the decreased P300 amplitude seen for high risk children in the perceptual identification task of Begleiter and colleagues (1984). In their task the subjects were required to identify the correct lateralization of a figure whose orientation in space could be changed. This is a relatively complex visuospatial task for children to perform. This task would lie at the extreme end of our hypothesized inverted U-shaped function. The ideal test of this hypothesis would be to compare the present two auditory tasks (Counting, Choice Reaction) with the visuo-spatial paradigm of Begleiter et al. (1984) within the same group of subjects to determine if our hypothesis provides a meaningful model to describe how these children are utilizing information. The focus of our on-going pedigree study will include this comparison.
ACKNOWLEDGEMENT

We greatly appreciate the help of the parents who permitted their children to take part in the study, transported them to our laboratory and patiently waited for them to complete the assessments. A special thanks to the children for volunteering to be in our study. Also, we want to thank Linda Wastyn for her excellent editorial assistance.

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REFERENCES


LIST OF FIGURES

Figure 1. ERP waveforms recorded from children of alcoholics and children of controls -- the Counting task. Positivity at the scalp is indicated by a downward deflection of the waveform. Stimulus onset coincides with the beginning of the .5 sec. time calibration.

Figure 2. ERP waveforms recorded from children of alcoholics and children of controls -- the Choice Reaction task

Figure 3. Changes in N250 amplitudes as a function of the conditional probability of stimulus in the Counting task

Figure 4. Changes in N250 amplitudes as a function of the conditional probability of stimulus events in the Choice Reaction task

Figure 5. Changes in P300 amplitude as a function of the conditional probability of stimulus events in the Counting task

Figure 6. Changes in P300 amplitude as a function of the conditional probability of stimulus events in the Choice Reaction task

Figure 7. Changes in P300 latencies as a function of the conditional probability of stimulus events in the Counting task

Figure 8. Changes in P300 latencies as a function of the conditional probabilities of stimulus event in the Choice Reaction task
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### Table 2 - ANOVA Summary for Latencies -- Counting Task

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### Table 2A - ANOVA Summary for Latencies -- Choice Reaction Task

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**Figure 2**

T/NT $p = 0.33$

NT/NT $p = 0.67$

NT/T $p = 1.00$

---

Frontal [Fz]

Vertex [Cz]

Parietal [Pz]

Occipital [Oz]

Left Parietal [P3]

Right Parietal [P4]

---

.5 sec

15 $\mu$V

---

Children of Control

Children of Affected
FIGURE

N250 Amplitudes – Choice Reaction Task

-20
-15
-10
-5
N250 Amplitude at Fz (µV)

Conditional Probability

.33
.67
1.00

Children of Control

Children of Affected
P300 Amplitudes – Counting Task

- Children of Controls
- Children of Affected

P300 Amplitude at P2 (μV)

Conditional Probability

0.33

0.67

1.00
P300 Amplitudes – Choice Reaction Task

- Children of Control
- Children of Affected

P300 Amplitude at Pz (μV) vs. Conditional Probability.
FIGURE 7

P300 Latencies – Counting Task

- Children of Control
- Children of Affected

P300 Latency at Pz (Msec)

Conditional Probability