Evoked Potentials in Hyperkinetic and Normal Children Under Certainty and Uncertainty: A Placebo and Methylphenidate Study

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ABSTRACT

Differences between hyperkinetic children and normal children and the effects of methylphenidate on hyperkinetic children were investigated under conditions of differential attentional demands. Auditory average evoked potentials were recorded from vertex using a single/double click guessing paradigm under conditions of certainty and uncertainty.

Under conditions of certainty (low attention), in which the subject was told the identity of each stimulus in advance, few significant group differences were found. Under conditions of uncertainty (high attention), in which the subject was asked to guess which stimulus would be presented, large group differences were found. In response to the second click the P200 component was found to be smaller and the N250 component was larger in hyperkinetic children than in normal children. Treatment with methylphenidate "normalized" the evoked potentials of the hyperkinetic children making them more like those of normal children.

The findings are believed: 1) to reflect the deficit in attention observed behaviorally in hyperkinetic children, 2) to support a model of hypoaorusal in hyperkinetic children, and 3) to reflect the behavioral "normalization" observed in hyperkinetic children treated with methylphenidate.

DESCRIPTORS: Evoked potentials, Hyperkinetic children, Attention deficit, Methylphenidate.

The diagnostic label hyperkinetic reaction of childhood has been used without precise definition and so globally that it conveys little clear meaning. Diagnosis of hyperkinesis is largely based on descriptive accounts of the child's behavior and, as such, is subjective in nature. Interestingly, a number of studies show that the most effective means of treating these children is with central nervous system (CNS) stimulants, the drug of choice being methylphenidate (ritalin) and secondly, dextro-amphetamine (see reviews by Conners, 1974; Milli-chap, 1973; Wender, 1971). Improvement under these drugs is defined by the observation of the diminution of those behavioral characteristics which were used to define the syndrome, thus, once again relying on criteria which are subjective and behavioral in nature.

Several investigators have studied the evoked potential in hyperkinetic children in an attempt to find correlates of differential clinical drug response.

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However, the majority of these studies were done under conditions which required minimal attention (i.e., a passive task). While not entirely consistent, results generally show that associated with differential drug response there appears to exist a differential neurophysiological response (Buchsbaum & Wender, 1973; Saletu, Saletu, & Itil, 1973; Saletu, Saletu, Simeon, Viamontes, & Itil, 1975; Satterfield, Cantwell, Lesser, & Podosin, 1972). Evoked potentials of the hyperkinetic children who respond to drug treatment look more like those expected in normal children (i.e., the response is "normalized").

While investigators speak of "normalization," relatively few studies have directly compared the evoked responses of normal children and drug-free hyperkinetic children, and those studies (Buchsbaum & Wender, 1973; Satterfield, 1973) which have made this comparison used a passive task and report results which are not entirely consistent (see Discussion section below). Use of a passive task is probably not an optimal strategy since the behavioral characteristics of hyperkinetic children are most clearly seen in situations which require focusing of attention and inhibition of inappropriate responses.

Conners (1974) found that the manipulation of attention entered as a variable in studying the effect of stimulant drugs in hyperkinetic children. However, his findings cannot be summarized as they varied with drug, electrode locus, modality of stimulation, and component of the evoked potential.

Halliday, Rosenthal, Naylor, and Callaway (1976) studied the relationship between attentional demands and drug outcome in hyperkinetic children. They found that methylphenidate decreased the average trial-to-trial variability in the evoked potentials of nonresponders when they went from a task requiring attention to a passive task—a direction of change which they considered abnormal. The responders showed the normal relationship of greater variability in the passive task and this relationship was unaffected by drug. In addition, for the responders, drug increased the amplitude of the N145-P190 components in the attention condition but not in the passive condition.

In the present study, in order to maximize the possibility of clarifying differences between normal children, hyperkinetic children under placebo, and hyperkinetic children under drug (methylphenidate), evoked potentials were studied under conditions of varied attentional demands. Under conditions of certainty, the child was told prior to each trial whether a single or a double click would be presented. Under conditions of uncertainty, the child had to guess prior to each trial whether a single or a double click would be presented. In this condition the occurrence of the single or double click informed the child whether his guess was correct or incorrect. Correct guesses were monetarily rewarded. In contrast with the certain condition where attentional demands were minimal, the task in the uncertain condition was attentionally demanding.

Since hyperkinetic children appear to focus attention poorly (Wender, 1971) and have been shown experimentally to be less capable than normal children in responding to differential task demands (Cohen & Douglas, 1972; Halliday et al., 1976), it was expected that in the certain condition, where attentional demands were slight, differences between hyperkinetic children and normal children would be small. However, in the uncertain condition where increased attentional demands were placed on the child, it was expected that differences between groups would be seen. Further, it was predicted that the effects of the drug would be seen under those conditions in which differences were found between hyperkinetic children and normal children. It was expected that these differences would be seen in the components of the evoked potential which have been shown to be related to attention and arousal.

**Method**

**Subjects**

Twenty-four male children, aged 8 to 11, served as subjects. Sixteen of these children were diagnosed as hyperkinetic at the Children's Clinic of the Psychopharmacology Research Unit of the New York State Downstate Medical Center. These children had evidenced problem behavior in the classroom and motoric overactivity which was documented by obtaining a mean score of 1.5 on the hyperactivity factor on the Conners Teacher Rating Scale (Conners, 1969). The hyperkinetic children were divided into two groups: the hyperkinetic drug group [H(D)] which was tested under methylphenidate and placebo, and the hyperkinetic control group [H(C)] tested twice under placebo. The remaining 8 children who served as the normal control group [N(C)] showed no behavioral signs of hyperactivity, did not exhibit behavioral problems in school, and had no history of psychiatric or psychological treatment. Subjects in this group were volunteered by their parents (university and laboratory associates), and were tested twice under placebo.

Subject groups were equated for mean age and age range. No subject showed evidence of neurological or physical disabilities, and all subjects were of normal intelligence (Full Scale WISC IQ greater than 80).

**Instrumentation**

Evoked potentials were elicited by auditory clicks (approximately 55 dB above threshold), delivered through a loudspeaker approximately 1 meter above the subject's head. Single and double clicks (interval of 950 msec) were presented randomly in a 50/50 paradigm under two experimental conditions. In the uncertain condition, the subject guessed prior to each trial whether a single or a double click would be presented. The subject indicated his guess by pressing one of two clearly labeled choice keys. The presentation of the single or the double click
confirmed or disconfirmed the subject's guess. In the certain condition, the subject was told, prior to each trial, whether he would hear a single or double click, and had to press the appropriate key which was followed by the presentation of the single or double click.

Evoked potentials were recorded from scalp at vertex (C3 in the international 10–20 system). A reference electrode was placed on the right earlobe and a ground electrode was placed on the neck. Beckman silver/silver chloride biopotential skin electrodes were used. Amplifier gain was set at 10,000 and bandpass was set between 0.02 and 100 Hz, with a rolloff of 6 dB per octave. Data were recorded on a Sango magnetic FM tape recorder and electronically labeled to enable the sorting and averaging of responses according to different experimental contingencies. Averaging was done off-line with a Xerox Sigma 7 computer.

Since sequential components may have different relationships to experimental variables (Wilkinson & Morlock, 1967; Morrell & Salamy, 1971), all components1 (P50 through N400) were measured relative to baseline rather than peak-to-peak. Baselines for the first and second click (whether present or absent) were established by averaging the 100 msec segment previous to the onset of each "click." In measuring components from baseline, the measure must specify not only distance but whether the component is above or below baseline. In order to establish a meaningful convention for defining polarity, it is assumed that positive-going components will reach their peak at a point which is positive relative to baseline and negative-going components will reach their peak at a point which is negative relative to baseline. Components which do not in fact behave in this way, i.e., positive components which reach their peak negative to baseline (above) or negative components which reach their peak positive to baseline (below), were indicated with a negative sign. It should be noted that, in contrast to positive values, the larger the negative value the smaller the component (Friedman, Hakerem, Sutton, & Fleiss, 1973).

Procedure
Each subject participated in 4 sessions, with approximately 2 weeks between sessions. During the first 2 sessions, combined in the data analyses into Period I, children in the hyperkinetic drug group were tested while being treated with methylphenidate, and children in the other two groups were under placebo. During the second 2 sessions, combined into Period II, children in all three groups were tested under placebo. Placebo sessions occurred a minimum of 1 week after the subject was taken off methylphenidate. Psychopharmacological studies provide evidence that methylphenidate is a short-acting drug which is out of the child's system within 24 hours (Andreasen, Peters, & Knott, 1976; Dring, Smith, & Williams, 1970). Each child in the hyperkinetic drug group received the dosage level of methylphenidate prescribed by the clinic as achieving maximum treatment effect for that child (mean dosage level was 33.75 mg/day).

In order to keep motivation and attention at a high level, and frustration at a low level, and to minimize differences between the groups with respect to learning the experimental paradigm, a monetary reward ($0.05) was given for each correct guess and no penalty for incorrect guesses (Freibergs & Douglas, 1969).

A closed circuit television was used to monitor the subject during testing. Head movements, lateral eye movements, and blinks which occurred during a trial could be detected and the trial noted for later deletion. One session (interspersed liberally with rest periods) consisted of 12 blocks of 20 trials, yielding 240 trials and lasting approximately 60 min. Blocks of certain and uncertain trials were alternated.

During a session, the subject was seated and instructed to attend to a red fixation light approximately 0.9 meter in front of him at eye level. In both the certain and uncertain conditions, in order to minimize movement artifact (e.g., position change, eye blink) the subject's choice initiated the trial sequence. One sec later a light flash signaled the subject that the single or double click would follow.

Data Analysis
The P50, N100, P200, N250, P300, and N400 components of the evoked potential in response to the first and second click, and the CNV were identified for each experimental contingency and amplitudes were measured relative to baseline. The CNV, defined as the average amplitude for the 100 msec period surrounding the most negative point prior to the second click (whether present or absent) was referred to two baselines. One baseline was the average amplitude for the 100 msec period prior to the first click, the second baseline was the average amplitude for the 100 msec period prior to the warning signal. All statistical tests for the CNV were done for both sets of measurements.

For the absent second click only the early negative (the most negative going peak following the point in time when the second click would have occurred), and late positive component (the most positive-going peak following the point in time when the second click would have occurred) were identified.

For each response component, comparisons of groups were made separately for conditions of uncertainty and certainty. Statistical comparisons were made in a series of 2 × 2 (Group × Condition) analyses of variance (with repeated measures). In cases where an analysis of variance was not appropriate, t tests for significant differences between means were done.

The hyperkinetic control group which received placebo during both periods, served two purposes. It served as the experimental group when compared with the normal children, and it also served as a control group for the methylphenidate treated hyperkinetic children. In the first set of comparisons the hyperkinetic controls were compared with the normal controls. These analyses tested the hypothesis that the evoked potentials of hyperkinetic and normal children were different. Before proceeding with these analyses, Period II amplitudes were subtracted from Period I and the difference scores were compared between groups, so as to be assured that the hyperkinetic and normal children did not change in different ways over time (between Period I and Period II). No consistent differences were found and, therefore, Period I and Period II were collapsed and all further analyses were based on the average measures.

In the second set of comparisons, change in response between drug and placebo sessions in the drug group was compared to the

1Following Donchin (in press), measured components are distinguished from theoretical components: polarity followed by average latency to refer to measured components (e.g. N250) and polarity followed by theoretical latency which has a bar over it to distinguish a theoretical component (e.g. N250).

2When a single click is presented in this kind of experimental paradigm, the point in time when the second click does not occur often yields an evoked potential (Sutton, Tueting, Zubin, & John, 1967). Measurements were made with respect to this response as well.
amount of change between repeated placebo sessions in the control group, and group differences were attributed to the effect of drug. In each comparison before using difference scores, groups were compared on Period II alone, i.e., placebo. No significant differences were found between groups when they were both on placebo.

Due to the fact that time-locking of electrical events to a stimulus may, when poor, change the shape of the waveform, every analysis done for amplitude measures was repeated using a measure of trial-to-trial variability. That is, all comparisons were also made using the standard deviations at the peak of each component in the same manner as was done with amplitude. In addition, an average baseline standard deviation for a 100 msec period prior to the presentation of the warning stimulus was computed for each condition for each child, and all comparisons were also made using this measure.

At the conclusion of this study a systematic error of approximately 5% was discovered in the timers controlling the interval between the warning stimulus and the first click, and the interval between the first and second click. Since it could not be determined at what point the timers had drifted, latency data were not used for the purpose of group comparisons. Component latencies are reported and plotted as averages across all subjects in all experimental conditions over the course of the entire study.

Since the evoked potentials of children are noisier and their shape is less familiar than average evoked potentials of normal adults, some comment should be made with respect to the identification of components. A number of criteria were used, these were: 1) the usual latency for certain components as known from prior work, 2) the presence of a relatively clear peak at a given point in time in other average waveforms for the same subject, 3) knowledge of the behavior of components as a function of experimental conditions, e.g., the well-established fact that P300 becomes larger under uncertainty (Sutton, Braren, Zubin, & John, 1965; Sutton et al., 1967).

In addition, a method was developed for aiding in the discrimination between “noise” peaks, i.e., a peak which is not present with some consistency in the trials constituting an average, and true peaks. Essentially this involved examining the point-to-point (every 10 msec) direction of voltage change in each trial, and only accepting as a true peak a point at which a change in the direction of voltage occurred in a large percentage of the trials which constituted the average.

**Results**

**Group Effects.** In Fig. 1 the average amplitudes (Period I and Period II combined) for all components in response to the first and second clicks in the certain and uncertain conditions are compared for the hyperkinetic controls (dashed line) and the normal controls (solid line). In this comparison both groups were under placebo. It should also be remembered that although in Fig. 1 the data are shown for each group in each condition separately, the main effects for groups were tested across conditions, i.e., in the certain condition analysis for the first click, groups are compared collapsing across single certain and double certain. Likewise, in the uncertain condition analysis for the second click, groups are compared collapsing across double right and double wrong.

In response to the first click the main effects for groups were not significant for either the certain or uncertain conditions. In response to the second click under conditions of certainty the P295 component in the hyperkinetic controls was found to be significantly larger than in the normal group (t(12)=2.28). Since P300 (most probably identical to our P295) would be expected to be relatively small under conditions of certainty (e.g., Roth, 1973; Sutton et al., 1965; Sutton et al., 1967; Tueting, 1968), the fact that hyperkinetic controls have large P300’s in the certain condition suggests that they are responding inappropriately to task demands.

It is in the response to the second click under conditions of uncertainty (whether the guess was right or wrong) that the largest group differences can

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**Fig. 1.** Mean click evoked potential amplitudes are compared for normal controls [N(C), solid lines] and hyperkinetic controls: [H(C), dashed lines] under conditions of certainty (left half) and uncertainty (right half). The upper row shows responses to the first click and the lower row shows responses to the second click.

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8The .05 rejection region was adopted in all statistical evaluations.
be seen. In general the hyperkinetic controls show less "positivity" in their curves—smaller positive components and larger negative components—than do the normal children, especially in the later components, although only statistically significant for the P186 (F(1/12) = 6.55) and N250 (F(1/12) = 6.90) components. With respect to the N250 findings, it should be remembered from the definitions adopted (see Instrumentation section above) that the amplitude of a component which reaches its peak in the unconventional position relative to baseline is larger when the numerical value is smaller.

**Group × Condition Interaction.** Significant Group × Condition interactions were found in the certain conditions for the P63 (F(1/12) = 12.27) and N103 (F(1/12) = 5.49) components in response to the first click. While the differences between the means were small, the normal children had a somewhat smaller P63 than the hyperkinetic controls in the single certain condition, and a larger P63 component than the hyperkinetic controls in the double certain condition. For the N103 component, there was no difference between groups in the single certain condition, while the hyperkinetic controls had a larger N103 than the normal children in the double certain condition.

**Uncertain Minus Certain.** Since hyperkinetic children appear to be less able to respond differentially to task demands (e.g., Cohen & Douglas, 1972; Halliday et al., 1976; see above), it was important to look more closely at differences in the response to certainty and uncertainty which were found in hyperkinetic and normal children. As noted, in response to the first click which was noninformational, the main effects for groups were not significant. In Fig. 2 the responses to the second click in the certain and uncertain conditions are compared separately for the hyperkinetic (upper panel) and normal (lower panel) groups. In response to the second click it can be seen that in both groups the later components (P186, N250, P295, N377) are more "positive" in the response to certainty than in the response to certainty, that is, positive components are larger and negative components are smaller. However, the magnitude of this difference (Group × Condition interaction) is smaller in the hyperkinetic controls although only significant for the P186 component for both double right (F(1/12) = 5.18) and double wrong (F(1/12) = 13.14) trials. In other words, the evoked potentials of the hyperkinetic group show a smaller difference between the response to certainty and uncertainty than do those of the normal group.

**Effect of Methylphenidate on Hyperkinetic Children**

Although the statistical analyses were done as differences between Period I and Period II, in order to most clearly illustrate the effect of methylphenidate, the drug and placebo groups are shown in Fig. 3 for Period I only. It is only in Period I that the drug group received methylphenidate. In Fig. 3 the average amplitudes for all components in the response to the first and second click in the certain and uncertain conditions are compared for hyperkinetic children under methylphenidate (solid line) and under placebo (dashed line). Further, as noted above, al-

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**Fig. 2** Mean evoked potentials to the second click are compared under conditions of certainty (solid lines) and uncertainty (right dotted, wrong dashed), separately for the hyperkinetic controls [H(C), upper panel] and normal controls [N(C), lower panel].

**Fig. 3** Mean click evoked potential amplitudes in Period I are compared under drug [H(D), solid lines] and placebo [H(C), dashed lines] for conditions of certainty (left half) and uncertainty (right half). The upper row shows responses to the first click and the lower row shows responses to the second click.
though in Fig. 3 the data are shown for each group in each condition separately, the main effects for groups were tested across conditions.

**Group Effects.** In the certain conditions the main effects for groups (drug vs placebo) were not significant for any component in the response to either the first or second click. Under conditions of uncertainty drug effects were found. In response to the first click, despite the small differences between the means, the amplitudes of the P186 and P295 components were significantly smaller under drug than under placebo (P186, $F(1/13)=10.93$; P295, $F(1/13)=5.16$). The amplitude of the N103 component was significantly larger under drug than under placebo ($F(1/13)=8.31$). However, the largest drug effect was seen in the response to the second click; the same condition in which the largest differences between hyperkinetic and normal children were found. Methylphenidate appears to produce a "positive" shift—particularly in the later components from P186 to N377. Positive components increased in amplitude and negative components decreased in amplitude. This effect of drug was statistically significant for the P186 ($F(1/13)=19.20$) and N250 ($F(1/13)=13.95$) components, the same components which were found to differentiate hyperkinetic and normal children.

Comparing Fig. 3 with Fig. 1, it is clearly seen that the effect of methylphenidate on hyperkinetic children is to "normalize" the evoked response. That is, the curves of both the hyperkinetic children under drug and the normal controls have the same relationship (more "positive") to the hyperkinetic children under placebo.

These effects are illustrated in the waveforms for the individual subjects in response to the second click for the double right condition shown in Fig. 4. It can be seen in the bottom panel [H(D)] that the drug caused increased positivity in the late components of the evoked response in 7 out of 8 hyperkinetic children in the drug group. By comparison the children in the hyperkinetic control group (middle panel) and the normal control (top panel) showed no consistent differences between Period I and Period II.

**Group X Condition Interactions.** Significant Group X Condition interactions were found in the P63 component of the response to the first click under conditions of certainty ($F(1/13)=5.55$) and uncertainty ($F(1/13)=6.34$). In the certain condition, the hyperkinetic drug group showed a larger decrease in amplitude (Period I minus Period II) in the single certain condition than in the double certain condition, as compared to the hyperkinetic control group which showed a larger decrease in amplitude in the double certain condition than in the single certain condition. In the uncertain condition, the difference between the hyperkinetic drug and hyperkinetic control groups in the amount of change in amplitude across periods (i.e., drug effect) was larger in the guess double condition than in the guess single condition. The significance of these interactions is somewhat unclear in view of the fact that little is known about the P50 (most probably identical to our P63) component, nor are these findings under drug particularly consistent with the differences between hyperkinetic and normal children under placebo. While the interaction in the P63 component was also present for comparison of hyperkinetic and normal controls under conditions of certainty, it was only seen in the drug comparisons under conditions of uncertainty.

**Uncertain Minus Certain.** Since in the compari-

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*With respect to this finding (N250 component) see conventions for amplitude discussed in Instrumentation section.
son of hyperkinetic controls to normal children it was found that there were smaller differences between the response to certainty and uncertainty in the hyperkinetic children than in the normal children, it was of interest to see the effect of drug on the hyperkinetic children's ability to respond differentially to these task demands. It was found that the difference between certain and uncertain conditions was larger under drug than under placebo, significant for the P186 component in the double wrong/double certain comparison (F(1/13)=5.59). Thus, once again the drug had a “normalizing” effect, making the response of the hyperkinetic children more like that of the normal children.

**Contingent Negative Variation**

There were no significant differences between hyperkinetic and normal controls in the CNV; nor was there any significant effect of methylphenidate on the CNVs of the hyperkinetic drug group.

**Emitted Potentials**

There were no significant differences between hyperkinetic and normal controls for the emitted potential (at the point in time when the second click was not presented); nor was there any significant effect of methylphenidate on the emitted potential of the hyperkinetic drug group.

Presumably, the emitted potential reflects the point in time of the non-occurrence of a stimulus (Sutton et al., 1967). For example, in the present study, the inter-click interval (slightly less than 1 sec) must carry over and be "estimated" in trials in which the second click does not occur. Further, the consistency with which the subject can estimate time is critical in determining the degree to which an emitted potential will be seen when one is using an averaging procedure. Investigators have proposed that variation in time estimation leads to a wide distribution in peak latencies of the components of the emitted potential and consequently to a broad duration, low amplitude waveform (Ruchkin & Sutton, in press; Sutton et al., 1967; Sutton, Ruchkin, & Tueting, in press).

In the present study the late positive component (P300) which is the major feature of the emitted potential was of extremely broad duration. Thus, it would suggest that children (both hyperkinetic and normal) are poor time estimators. However, no differences between groups were found in the emitted potential.

**Trial-to-Trial Variability**

Due to the fact that poor time-locking of electrical events to a stimulus may change the shape of the average waveform, variability cannot be overlooked when comparing amplitude differences in evoked potential components. This is particularly the case when one is comparing evoked potentials in two groups, one of which might be expected a priori to have greater trial-to-trial variability. In the present study no significant group differences were found in intra-individual trial-to-trial variability.

**Discussion**

**Effects of Uncertainty and Methylphenidate**

The major findings in the present study were seen under conditions of uncertainty in the response to the informational (second) click. The P186 component was significantly smaller (less positive) and the N250 component significantly larger (more negative) in the hyperkinetic children than in the normal children. More generally, it can be seen in Fig. 1 that all components from P186 to N377 are less positive in the hyperkinetic children than in the normal children. That is, positive components are smaller and negative components are larger.

The effect of methylphenidate on hyperkinetic children was to increase the amplitude of the P186 component and decrease the amplitude of the N250 component moving both components in the direction of the normal children. In Fig. 3 methylphenidate appears to shift all components beginning with P186 (N250, P295, and N377) in a more positive direction, i.e., in the direction of the normal children.

Another way in which differences between groups were considered was by comparing the magnitude of the differences in amplitude between the condition of uncertainty and the condition of certainty. Here it was found that the difference in P186 amplitude between conditions of uncertainty and certainty was significantly smaller in hyperkinetic children than in normal children. In the hyperkinetic children this difference was increased under methylphenidate, thus, the change brought about by drug was again in the normalizing direction.

While in the present study the response to uncertainty was different from the response to certainty in all groups, the magnitude of this difference was larger in normal children than in hyperkinetic children and was increased in hyperkinetic children under drug. Using electrodermal measures, the findings of Cohen and Douglas (1972) were consistent with those of the present study. Skin conductance level in normal children was found to increase with increased task demands from non-signal (passive attention) to signal (active attention) condi-

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9In fact, drug normalizes these two components in the sense that no significant differences were found when one compared the hyperkinetic children on drug (Period 1) to the normal children on placebo (Period 1).
tions, whereas no such differences were found in untreated hyperkinetic children. However, Cohen, Douglas, and Morgenstern (1971) reported inconsistent findings with respect to the effect of methylphenidate on hyperkinetic children in response to varied task demands.

A number of studies in which attention was manipulated in a variety of ways, found the P200 component of the evoked potential to be larger when more attention is directed to a stimulus (Davis, 1964; Eason & Harter, 1969; Picton & Hillyard, 1974; Picton & Low, 1971; Satterfield, 1965; Spong, Haider, & Lindsley, 1965). Therefore, the finding in this study of smaller P186s (most probably identical to P200) in hyperkinetic children is perfectly consistent with the behavioral observation of attentional deficit in this group. Similarly, the increase in the amplitude of P200 under drug is consistent with the behavioral observation of improvement in ability to attend. Furthermore, the fact that no P200 differences were found under conditions of certainty, emphasizes the critical role of attention in these findings.

It was somewhat surprising not to obtain corresponding group differences in the P295 component (most probably identical to P300) since P300 and P200 tend to behave similarly in the uncertainty vs. certainty experimental paradigm (Tueting, 1968; Friedman et al., 1973). Secondly, since hyperkinetic children are less able to respond differentially to task demands, and since the amplitude of P300 is strongly linked to the presence or absence of uncertainty, one would have expected smaller differences in P300 amplitude between uncertain and certain conditions in the hyperkinetic group. It should be noted, however, that all the trends are in the expected direction (see Figs. 1, 2 and 3).

As in the present study, Satterfield (1973) reported that the P200 component of the evoked potential was significantly smaller in amplitude in hyperkinetic children than in normal children. Further, in the present study close to half of the hyperkinetic children had P200 amplitudes which were smaller than in any of the normal children. Satterfield (1973) also found that the N100–P200 component was significantly smaller in hyperkinetic children than in normal children. However, since he did not report N100 findings, this leaves open the question of whether N100 was the same or different. Contrary peak-to-peak findings were reported by Buchsbaum and Wender (1973) who found hyperkinetic children to have significantly larger N100–P200 components than normal children. Buchsbaum and Wender also did not report results for individual components. In the present study peak-to-peak measurements of the N103–P186 (most probably identical to N100–P200) component did not yield significant group differences. Another factor which makes comparisons across studies difficult is that the age of the groups studied varies and age has been shown to affect the amplitude of these components (Buchsbaum & Wender, 1973).

**Arousal Level**

Not reported in previous studies is the finding of larger N250 components in hyperkinetic children than in normal children. Methylphenidate decreased the amplitude of the N250 component in hyperkinetic children, once again appearing to normalize their response. Satterfield (1973) reported smaller P200–N250 components in hyperkinetic children. Again, it is difficult to compare his results directly to those of this study since he did not report the N250 component separately.

Wilkinson, Morlock, and Williams (1966) reported that as accuracy decreased over time in a vigilance task the amplitude of P200 declined while the amplitude of N250 increased. They interpreted these findings to mean that the decline in amplitude for P200 represented a distraction process and that the increase in N250 was correlated with declining arousal (see also Fruhstorfer & Bergström, 1969; Picton, Hillyard, Krausz, & Galambos, 1974; Tueting, 1968; Weitzman & Kremen, 1965; Williams, Tepas, & Morlock, 1962). The amplitude of the N250 component has further been shown to decrease with states demanding heightened attention, such as conditions of increased uncertainty (Tueting, 1968).

The fact that in the present study N250 was found to be significantly larger and P200 significantly smaller in hyperkinetic children than in normal children has several implications. In terms of attention (P200), these findings may be considered to represent additional physiological evidence of the attentional deficit in hyperkinetic children which is reversed under drug treatment. Of even greater interest, however, is the implication for an arousal model of hyperkinesis. The finding of increased N250 amplitude suggests strongly that hyperkinetic children are in a state of low arousal. Methylphenidate decreased the amplitude of the N250 component, increasing the arousal level, and making the response of hyperkinetic children more like that of the normal children. The fact that CNS stimulant drugs are found to be therapeutically effective is entirely consistent with a low arousal model for hyperkinesis.

Supporting evidence for low arousal level in hyperkinetic children has been reported in several studies of electrodermal activity (Cohen & Douglas, 1972; Satterfield & Dawson, 1971; Spring, Greenberg, Scott, & Hopwood, 1974). In addition, while results of the studies of electrodermal activity
were not entirely consistent, all three found that stimulant drugs increased at least one measure of autonomic arousal. Also consonant with a low arousal model of hyperkinesis are the findings of increased slow wave activity in the electroencephalograms of hyperkinetic children (Capute, Niedermeyer, & Richardson, 1968; Grünwald-Zuberbier, Grünwald, & Rasche, 1975; Satterfield et al., 1972; Wikler, Dixon, & Parker, 1970). Yoss and Moyer (Note 1) found evidence of hypoarousal in the pupillogram of approximately 25% of the hyperkinetic children they tested, and further reported normalization of the pupillogram in these children under stimulant drugs.

Some caution must be exercised, however, in invoking a generalized or tonic arousal explanation based on the evidence of the present study. The differences in N250 found between hyperkinetic and normal controls were limited to the uncertain condition. There were no N250 differences found in the certain condition. In the same vein, methylphenidate was found to decrease the amplitude of N250 in hyperkinetic children in the uncertain condition but had no effect on N250 in the certain condition. The data therefore, while supporting a theory of under-arousal in hyperkinetic children clearly only offer support for phasic or task-specific underarousal.

While an underarousal model may appear to be in conflict with clinical reports of motor overactivity, attempts to document a gross increase in motor activity in hyperkinetic children using objective measuring devices have been largely unsuccessful (see review by Wender, 1971). However, increases were documented in activities such as fidgeting and restlessness; and time spent in any single activity was found to be greatly decreased. Thus, the description of increased activity in hyperkinetic children probably reflects their decreased attention span, as represented by the decreased time spent in any single activity, therefore leading to the appearance of overactivity. An explanation offered by Satterfield and Dawson (1971) suggests that the activity observed in hyperkinetic children constitutes an attempt to increase proprioceptive and exteroceptive sensory input through self-stimulation. Zentall (1975) noted that descriptions of hyperkinetic behavior resemble the behavioral effects of sensory deprivation and suggests that both can be explained in terms of a theory based on an optimal level of stimulation.

REFERENCES


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REFERENCE NOTE


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