PSYCHOPHYSIOLOGY

Vol. 25, No. 1
Printed in U.S.A.

# Electrodermal Lability Predicts Presentation Rate Effects and Stimulant Drug Effects on Paired Associate Learning in Hyperactive Children

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## ABSTRACT

**Hyperactive children were divided into three groups (electrodermal labiles, electrodermal stabiles, and a medium group which fell in between) on the basis of the frequency of spontaneous electrodermal activity. Subsequent tests on a paired associate learning task revealed that the stabiles and medium groups performed poorly when stimulus-response pairs were presented at a relatively slow rate, while for labiles there was no difference across rates. Treatment with stimulant medication abolished the differences across rates for the stabiles and the medium groups, but led to significantly more learning on the fast rate relative to the slow rate for labiles. While the placebo findings were consistent with a low arousal view of hyperactivity, the drug results suggested that stimulant medication corrects an imbalance in the mechanisms which govern sensitivity to task-related stimulation, but induces an imbalance where none is present on placebo.**

**DESCRIPTORS: Hyperactive children, Electrodermal lability, Paired associate learning, Presentation rate effects.**

The construct of arousal has been frequently invoked to account for differences between hyperactive and normal children. Both Satterfield and Dawson (1971) and Zentall and Zentall (1983) have proposed that hyperactives are underaroused, such that high levels of environmental stimulation are needed to raise arousal to an optimal level. However, there is little direct physiological evidence in support of this view. While Satterfield and Dawson (1971) reported lower skin conductance levels in hyperactives than normals, this finding has not been replicated in subsequent work (Satterfield, Atoian, Brashers, Burleigh, & Dawson, 1974; Zahn, Abate, Little, & Wender, 1975; Ferguson, Simpson, & Trites, 1976). Attempts to distinguish hyperactives from normals on the basis of other tonic measures have also yielded inconsistent findings (cf. Solanto & Conners, 1982).

It may be that the inconsistency of findings across studies is partially due to methodological factors. For example, a common practice has been to compare hyperactives and non-hyperactives with respect to a physiological measure (Satterfield & Dawson, 1971; Satterfield et al., 1974). Given the difficulties in diagnosing hyperactives and the possibilities of differences in subject selection procedures, there is a strong likelihood of sampling differences across studies. Perhaps more consistent findings would be obtained if attempts were made to examine psychophysiological measures in relation to task performance, particularly if the task employed were known to be sensitive to the cognitive deficit of hyperactivity. In previous hyperactivity studies when psychophysiological measures have been examined in relation to task performance, a reaction time measure was commonly used (Cohen & Douglas, 1972; Sroufe, Sonies, West, & Wright, 1973; Porges, Walter, Korb, & Sprague, 1975), which is known not to be particularly sensitive to the cognitive deficit of hyperactives (Douglas, 1980).

This research partially satisfied the requirements for the first author's Degree of Doctor of Philosophy at the University of Toronto. The authors thank Committee members Dr. John Furedy and Dr. Kirk Blankstein for their advice and encouragement.

A preliminary report of this research was presented at the Annual Meeting of the Society for Psychophysiological Research, Vancouver, October, 1980.

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In the present study psychophysiological measures were examined in relationship to paired associate learning performance with arbitrary associates. This task was selected on the basis of Douglas' (1980) analysis which indicated that learning arbitrary associates is very difficult for hyperactives relative to normal controls. Further, the event rate at which paired associates were presented was manipulated, since it is well-established that hyperactives, but not normal controls, learn less efficiently when paired associates are presented at low event rates (Conte, Kinsbourne, Swanson, Zirk, & Samuels, 1986; Dalby, Kinsbourne, Swanson, & Sobel, 1977).

Measures of electrodermal lability (the frequency of nonspecific electrodermal responses and the rate of habituation) have been shown to be good predictors of performance of normal adults in low event rate situations such as vigilance tests (Sostek, 1978; Coles & Gale, 1971; Crider & Augenbaum, 1975; Siddle, 1972). According to current definitions (Katkin, 1975), electrodermal stabiles produce few nonspecific responses and habituate rapidly, whereas electrodermal labiles produce nonspecific responses at a high frequency and are resistant to habituation. In terms of vigilance performance, stabiles show a large vigilance decrement, whereas labiles tend to show little or no vigilance decrement (Sostek, 1978; Crider & Augenbaum, 1975).

For the purpose of the present research, it was assumed that electrodermal lability provided an index of the arousal construct. This assumption has received support in studies which have indicated a relationship between electrodermal lability and both sleep onset latency during repetitive stimulation (Bohlin, 1972), and the maintenance of cortical arousal during monotonous stimulation (Siddle & Smith, 1974). It has also been established that subjects on the low end of the arousal continuum as measured by electrodermal lability perform similarly to sleep-deprived individuals on vigilance tasks (Wilkinson, 1960).

The principal objective of the present work was to determine whether electrodermal lability could account for individual differences in performance across presentation rate conditions. Given that among normal adults, stabiles show poor performance in the low event rate environment of the vigilance task, we hypothesized that hyperactive subjects who were electrodermal stabiles would perform poorly under slow rates of presentation, whereas electrodermal labiles would show no difference in performance as a function of rate of presentation.

One implication of the low arousal theory of hyperactivity (Zentall & Zentall, 1983) is that stimulant drugs and within-task stimulation both increase arousal within the nervous system. If this is so, then drugs and a high rate of stimulation should have similar effects on performance. We therefore predicted that only those subjects who benefit from increased stimulation on placebo (i.e., show better performance at the fast rate than the slow rate) would also show a drug-induced benefit selectively at the slow rate of presentation.

## Method

### *Subjects*

Thirty-two hyperactive boys ($\overline{X}$ age = 10.8 yrs, SD = 2.1, range 8.2–12.0 yrs) participated in this study which took place in the context of a 2½-day double-blind laboratory drug trial. They were selected on the basis of a teacher referral for hyperactivity, and each child was found by a clinic physician to meet the DSM III criteria for attention deficit disorder with hyperactivity: The child had to show signs of impulsivity, short attention span, and hyperactivity. Also, to be included the children needed average IQ's (WISC-R full scale above 80). None of the subjects had been previously treated with stimulant medication.

### *Paired Associate Learning Procedure*

Each child was tested 5 times on paired associates over 2½ days. The task consisted of a computer-generated display which was presented on a VT-11 graphics processor. It was an array of circles ½″ in diameter. The number of circles corresponded to the number of items in the paired associate task, from 6 to 10. They were arranged in a circular pattern, 3½″ in diameter. Thus, the display at the start of each trial consisted of a large circle composed of a number of smaller circles. Just below the circles was a row of houses which varied in number depending on the number of items (items/2) used in the paired associate task. The houses were numbered from left to right, with one number per house starting from one, and each successive house incremented by one (see Figure 1).

The subject's task was to associate each circle with one house. On each trial a graphically drawn bird appeared in one of the circles and thus indicated the stimulus member on that trial. The bird was in the circle for 3½ s, during which the subject could make an oral response. After this period, feedback was given, i.e., the bird moved to one of the numbered houses. The number of the house indicated the correct response on that trial. When the bird appeared in the house, it remained visible in the circle in which it had first appeared. In this way the subject could study the stimulus-response pair for the trial. Paired associate lists were presented under two conditions. In the fast condition, trials lasted 8 s (.5-s warning, 3.5-s stimulus presentation and response time, 4-s study time). In the slow condition, trials lasted 12 s, and differed from the 8-s condition in that there was 8 s for study instead of 4 s. For both conditions the intertrial interval was 0 s.

Five different lists of stimulus-response combinations were used for each list length. For each list the

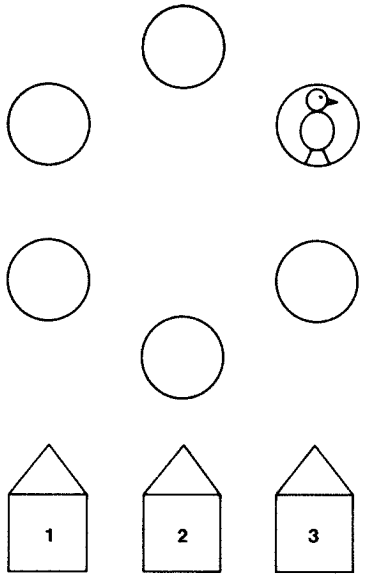


**Figure 1.** Six-item stimulus display for the paired associate learning test.

stimulus-response mappings were assigned randomly with the constraint that each stimulus position had to be used at least once and each response was associated with 2 stimuli. Each list consisted of 12 blocks of items. Each block consisted of the number of items used for that list (6, 8, or 10). Within each block, each stimulus was presented once and the order in which the stimuli were presented within each block was determined randomly. For each subject a list was assigned to a stimulus condition (drug-placebo, fast rate-slow rate) randomly.

Subjects initially visited the laboratory for one-half day to become familiar with laboratory personnel and to practice the experimental tasks. The standard list with 6 items was presented on the initial list presentation. If the child made 20 or more errors, then list length during the experimental tests was 6; for fewer than 20 errors, the number of items was increased by 2 and the practice test was repeated. This procedure was followed until the subject made at least 20 errors. No subject required more than 10 items during testing. During testing, the experimenter, who remained in the experimental room, recorded subjects' oral responses during the task.

On the remaining two experimental days, each subject was tested twice per day, once on the 12-s list and once on the 8-s list. Half the subjects received the 12-s list and half received the 8-s list first. Each testing session took 10–24 min.

### *Electrodermal Recording*

Fifteen minutes prior to the beginning of each session recording electrodes were applied. The volar surfaces of the second and fourth fingers of the non-dominant hand were cleaned with acetone. Grass silver-silver chloride electrodes were filled with a non-commercial paste consisting of a mixture of unibase and .9% saline (Lykken & Venables, 1971). Skin resistance responses were amplified by a Grass Polygraph recorder with a 7P122 DC amplifier. The gain (sensitivity at .5 or 1 mV/cm) and balance voltage settings were adjusted to the individual response by having each subject take a few deep breaths. Then a 5-min period elapsed in order to allow the skin resistance level to recover before the recording session began.

Fifteen minutes after electrode application, the session began with a 7-min recording of electrodermal activity (EDA). Subjects were seated in front of the graphics processor of the PDP-11 computer and were asked to sit quietly for the duration of the resting recording. Electrodermal activity was fed into one channel of a four-channel Hewlett Packard HP 3964A FM tape recorder. The experimenter observed each subject during the recording session and indicated when perceptible body or hand movements occurred by pressing a signal marker which activated a pulse played onto a second tape channel. These segments were excluded in order to minimize the risk of falsely scoring electromyographic data as nonspecific responses. Electrodermal records were subsequently digitized at a rate of 5 Hz, and later processed with another computer program which allowed the experimenter to hand score the data. The data display consisted of electrodermal activity and the channel showing the record of movements. The number of nonspecific responses for each minute was scored by a computer program which enabled the experimenter to move a cursor across each 1-min sweep displayed on the CRT. Segments in which movements occurred were excluded along with any others in which artifacts obscured the records. Less than 1% of the records were deleted because of artifacts. Each subject's score was based on 5 artifact-free minutes of electrodermal activity; i.e., up to 2 min of the 7-min recording session could be deleted because of artifacts. No subject had to be excluded because of excessive artifacts. A nonspecific response was defined as any phasic change in electrodermal activity in excess of 100 ohms which was uncorrelated with stimulus input (Katkin, 1975).

### *Drug Administration*

A double-blind crossover design was used in which subjects were given a placebo or .3 mg/kg Ritalin ($\bar{X}$ dose = 11.46 mg, SD = 3.24) at 9:00 am, and the same again at 12:00 noon. Half the subjects were given placebo and half Ritalin on day 1. The alternate treatment was given on day 2. Half the subjects in each of the two drug order groups were tested on the 12-s list first, and half were given the 8-s list first.

### *Data Analysis*

In accordance with the procedures used by Dalby et al. (1977), the learning data were evaluated by equating the amount of presentation time across presentation rate conditions. The literature on the total time hypothesis (Bugelski, 1962; Keenan, 1970; Koffman & Weinstock, 1974) indicates that in both adults and children the amount learned per unit of presentation time is invariant as long as total presentation time is held constant across presentation rate conditions. In the present study presentation time across presenta-

tion rate conditions could be equated at 24, 48, 72, and 96 s. For example, at 24 s of presentation time the percent correct on trial 2 at the 12-s rate (12 s/item × 2 trials = 24 s) was compared with the percent correct on trial 3 at the 8-s rate (8 s/item × 3 trials = 24 s). The second point of comparison across presentation rate conditions was at 48 s (the percent correct on trial 4 at the 12-s rate was compared with the percent correct on trial 6 at the 8-s rate). Similar procedures were used to make comparisons across rates at 72 and 96 s.

## Results

### *Time of Day Effects*

Since subjects received a pill at 9:00 am and 12:00 noon, it is possible that the residual effect of the morning dose could carry over to the afternoon testing session. This would have resulted in a significant medication × time of day interaction for one or more of the dependent measures. Analysis of variance indicated that this interaction was nonsignificant for percent correct during paired associate learning, $F(1/30)=.15$, and for nonspecific responses, $F(1/30)=.18$.

### *Presentation Rate Effects*

Analysis of the learning data for the entire group of subjects indicated that there were significant effects of presentation rate, $F(1/30)=12.00$, $p<.01$, and days, $F(1/30)=15.39$, $p<.01$. The presentation rate effect was due to superior performance at the 8-s rate, $\overline{X}=75.5\%$, SD = 17.0, compared to the 12-s rate, $\overline{X}=66.1\%$, SD = 23.5. The effect of days indicated that a higher level of performance was attained on day 2, $\overline{X}=75.7\%$, SD = 20.4, than on day 1, $\overline{X}=66.0\%$, SD = 18.2. There was no interaction of medication status and presentation rate.

### *Nonspecific Response Data*

There were significant effects of medication, $F(1/30)=8.17$, $p<.01$, rate, $F(1/30)=10.57$, $p<.01$, and medication × rate, $F(1/30)=5.54$, $p<.05$. The interaction indicates that medication increased the frequency of nonspecific responses more prior to 8-s tests than prior to 12-s tests. In the placebo condition there was no difference in the number of nonspecific responses prior to the 8-s and 12-s tests, $F(1/26)=1.49$.

### *Presentation Rate Effects in Lability Subgroups*

To examine the relationship between nonspecific responses and presentation rate effects, subjects were divided into three groups on the basis of nonspecific response frequency averaged across the two placebo recording sessions. Subjects falling into the upper and lower thirds of the distribution were categorized as labiles (N = 10) and stabiles (N = 11), respectively. Subjects whose nonspecific response frequencies fell in between these two groups were included in the medium group (N = 11). It should be noted (see Table 1) that there was considerably more variability in the labiles than in the other two groups. Resting skin resistance levels were examined for between-group differences, but there were none, $F(2/26)=.42$. In order to determine the stability of nonspecific responses, Spearman rank order correlations were computed among the values obtained for the four recording sessions. These data (Table 2) indicate that there was only one nonsignificant relationship (between the 12-s placebo test and the 12-s drug test). All others were significant and ranged from .40 to .60.

**Table 1**

*The effect of stimulant medication on nonspecific response frequency in the stabile, medium, and labile groups*

| | | Nonspecific Response Frequencies (SDs in Parentheses) | |
|---|---|---|---|
| Groups | N | Placebo | Medication |
| Stabile | 11 | 2.2 (1.0) | 6.5 (4.4) |
| Medium | 11 | 4.9 (0.8) | 6.7 (4.9) |
| Labile | 10 | 11.1 (5.9) | 11.5 (5.7) |

The purpose of the initial analysis was to determine if there were differences in presentation rate effects across the three lability groups in the placebo condition. The stabile and medium groups learned more at the faster rate of presentation, whereas the labiles learned equally well across rates (see Figure 2). A 3(group: stabile, medium, labile) × 2(drug order: pd, dp) × 2(presentation rate: 8, 12) split-plot analysis of variance indicated a significant group × rate interaction, $F(2/30)=5.30$, $p<.01$. Decomposition of this effect indicated that the stabile and medium groups learned significantly less at the 12-s compared to the 8-s rate, $F(1/10)=5.79$, $p<.05$, and $F(1/9)=4.83$, $p<.05$, respectively. For the

**Table 2**

*Correlations between nonspecific response frequency in the placebo and medication conditions*

| | Correlations | | |
|---|---|---|---|
| Conditions | Placebo 12-s | Placebo 8-s | Medication 12-s |
| Placebo 8-s | .47** | | |
| Medication 12-s | .25 | .48** | |
| Medication 8-s | .40* | .55** | .60** |

*$p<.05$, **$p<.01$.

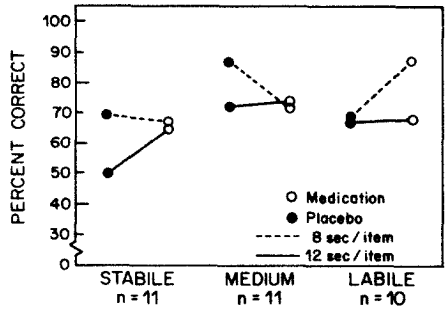


**Figure 2.** Presentation rate effects in the stabile, medium, and labile groups as a function of medication condition.

labile group there was no difference across rates on placebo, $F(1/9)=.61$.

To examine the pattern of drug effects on learning in the three lability groups, a 3(group) × 2(drug order) × 2(medication) × 2(presentation rate) split-plot analysis of variance was performed. This analysis revealed a significant group × medication × rate interaction, $F(2/26)=4.64$, $p<.02$, suggesting that there were differential effects of the medication on performance as a function of presentation rate condition and group. Post-hoc analyses indicated that after treatment with stimulant medication there was no difference across presentation rate conditions for the stabile and medium groups, $F(1/10)=.05$, and $F(1/9)=1.05$, but there was significantly more learning at the 8-s rate than the 12-s rate for the labiles, $F(1/9)=18.84$, $p<.01$. There was a notable difference in the form of the drug effect in the medium and stabile groups. In the medium group there was a significant decline in performance at the 8-s rate from placebo to medication, $F(1/9)=11.79$, $p<.01$, while there was a marginally significant improvement in performance from placebo to drug at the 12-s rate for the stabile group, $F(1/9)=3.90$, $p<.07$.

### *The Effect of Medication on Nonspecific Responses in Lability Subgroups*

The effect of medication on nonspecific responses in labiles and stabiles was examined in order to determine if a differential physiological response could account for the different behavioral effect of the drug across the three groups. Analysis of variance on the data shown in Table 1 indicated that there was a greater effect of medication on nonspecific responses in the stabile than the labile group as evidenced by a significant medication × group interaction, $F(1/19)=4.62$, $p<.05$. However, this interaction was nonsignificant in comparisons of the medium group with the labile and stabile groups, $F$'s$<1$. As shown in Table 1, the frequency of nonspecific responses increased in stabiles after drug treatment, $F(1/10)=13.9$, $p<.01$, but there was no change in the medium or labile groups, $F(1/10)=1.4$, and $F(1/9)=.11$, respectively.

**Table 3**

*Correlations between the effects of medication on paired associate learning (percent correct-medication minus percent correct-placebo) and the effect of medication on nonspecific response frequency (medication minus placebo)*

| Groups | N | Correlations | |
|---|---|---|---|
| | | 12-s Rate | 8-s Rate |
| Stabile | 11 | −.15 | −.55* |
| Medium | 11 | −.16 | −.11 |
| Labile | 10 | .08 | .01 |

*$p<.05$.

As an alternative method of examining this issue, correlations between the drug effect on learning (percent correct on medication minus percent correct on placebo) and the drug effect on nonspecific response frequency (drug minus placebo) were computed. These correlations were calculated separately for the stabile, medium, and labile groups and for each rate of presentation. The only significant correlation was for the stabile group at the fast rate, $r=-.55$, $p<.05$, but it indicated that the largest changes in nonspecific response frequency were associated with the smallest changes in performance (Table 3).

## Discussion

The principal finding of the present study was that electrodermal lability predicted the effects of presentation rate on placebo. The stabile and medium groups both learned less at the 12-s rate than the 8-s rate, whereas there was no difference across rates for labile subjects. Assuming that electrodermal lability provides an index of arousal, these data provide evidence that the arousal construct may be useful in explaining the learning performance of hyperactive children. The fact that both the stabile and medium groups showed significantly greater learning at the faster rate of presentation also suggests that rate of presentation can compensate for the low arousal condition, whereas a fast rate has no appreciable effect on the higher arousal labile subjects.

Since electrodermal lability predicts responsivity to external stimuli (Katkin, 1975), one way of conceptualizing the present results is to assume that differential performance across rates in the three groups of subjects is due to variation in reactivity to task-related stimulation. Thus, there is a greater density of stimulation at the faster rate of presentation, and presumably, low arousal subjects (sta-

bile and medium) require a higher level of stimulation to function optimally. High arousal subjects (the labiles) do not benefit. This interpretation is consistent with the findings reported by Conte et al. (1986). In that study attention disordered subjects, unlike normal subjects, learned less efficiently on the slow rate items when the effects of rate were assessed across different lists of items, i.e. a 6-s fixed rate list vs. a 12-s fixed rate list. When fast and slow items were intermixed within the same list, attention disordered and normal controls learned equally well across rates. This result indicated that the attention span limitation was distributed across blocks of trials and did not occur within a trial.

One prediction made from the arousal theory proposed by Zentall and Zentall (1983) is that stimulant medication and task-related stimulation have similar effects. In the context of the present study, these arousal sources would presumably interact with the dimension of electrodermal lability. For the sake of simplicity, one could propose that lability, task-related stimulation, and stimulant medication are all equivalent sources of arousal, and that the effects of each source are additive with the others. The data from the stabile group fits this model since stimulant medication and a fast rate of presentation had similar effects on performance such that performance at the 8-s rate on placebo and the 12-s rate on medication was identical. Stimulants also abolished the difference in performance across rates in the medium group, but it did so by means of a significant decline in performance at the 8-s rate. Arousal theory can account for this finding if it is assumed that arousal level was optimal in the placebo/8-s rate condition, and that arousal level in the drug/8-s rate condition exceeded the optimal level, leading to performance decline. However, the performance of the labiles at the 8-s rate appears to be at variance with the predictions of arousal theory. Since the labiles have a higher tonic arousal level than the medium group, then one would predict that drug would lead to a significant performance decline also in this group at the 8-s rate. However, these subjects showed a significant drug-induced increase at the 8-s rate. Thus, while arousal theory is consistent with some of the drug findings, there are inconsistencies with the simple additive model, i.e., that medication, lability, and a fast rate of presentation are all equivalent sources of arousal.

An alternative view is that stimulant drugs have properties such that the direction of the drug effect is dependent on the base-state or placebo level of performance. The literature on rate-dependence (Kelleher & Morse, 1968; Grossman & Sclafani, 1971) indicates that the direction of the stimulant drug effect is often dependent on response rate during placebo. A high rate of response on placebo is associated with a drug-induced decrease in performance, whereas a low rate of response on placebo is associated with a drug-induced increase in performance. It is becoming apparent that such effects occur frequently in humans, particularly in response to stimulant administration (Robbins & Sahakian, 1979; Hicks, Gualtieri, Mayo, Schroeder, & Lipton, 1985; Kinsbourne, 1985). In the present data there is evidence that the relative level of performance across presentation rate conditions on placebo may have had a strong influence on the drug effects across rates. Perhaps the difference in performance across rates on placebo is a manifestation of an imbalance in mechanisms which regulate sensitivity to environmental stimulation. When there is an existing imbalance, drug corrects it, but when there is no imbalance (as in the labiles) one is produced such that the pattern of presentation rate effects for the labiles on medication is similar to the stabiles and medium groups on placebo.

It could be argued that the relationship between the placebo findings and the drug effects on learning are due to regression to the mean; i.e., those subjects (labiles) who do well at the slow rate on placebo do not benefit from drug at the slow rate, whereas those subjects (stabiles) who do poorly at the slow rate on placebo, improve after drug treatment. But at the very least this does not describe the findings for labiles. For them, there was no difference across rates on placebo, and yet, although they showed no drug-induced improvement at the slow rate, they did improve at the fast rate. The cumulative weight of evidence tends to support the view that attention deficient children have unstable mechanisms of behavioral control (Kinsbourne, 1985). Stimulant administration may correct an existing imbalance (as in the stabile subpopulation in the present study) but may induce imbalance where it is absent (as in the labiles).

In summary, the results of the present study are of relevance to the understanding of both hyperactivity and stimulant drug action. The findings for placebo are consistent with a low arousal view of hyperactivity, insofar as the performance enhancing effect of a rapid rate of presentation occurs only in hyperactives with low rates of autonomic activity (nonspecific responses). The most comprehensive explanation of the drug findings is that different levels of performance across rates may be an indication of an imbalance in mechanisms which control sensitivity to environmental stimulation. When there is an existing imbalance, drug corrects it, but when there is none on placebo, drug induces an imbalance.

## REFERENCES


Bohlin, G. (1972). Susceptibility to sleep during a habituation procedure as related to individual differences. *Experimental Research in Personality, 6*, 255–258.

Bugelski, B.R. (1962). Presentation time, total-time, and mediation in paired associate learning. *Journal of Experimental Psychology, 63*, 409–412.

Cohen, N., & Douglas, V. (1972). Characteristics of the orienting response in hyperactive and normal children. *Psychophysiology, 9*, 238–245.

Coles, M.G.H., & Gale, A. (1971). Physiological activity as a predictor of performance on a vigilance task. *Psychophysiology, 8*, 594–595.

Conte, R., Kinsbourne, M., Swanson, J.M., Zirk, H., & Samuels, M. (1986). Presentation rate effects on paired associate learning in attention deficit disordered children. *Child Development, 57*, 681–687.

Crider, A., & Augenbaum, C.B. (1975). Auditory vigilance correlates of response habituation speed. *Psychophysiology, 12*, 36–40.

Dalby, J.T., Kinsbourne, M., Swanson, J.M., & Sobel, M. (1977). Hyperactive children's underuse of learning time. *Child Development, 48*, 1448–1453.

Douglas, V. (1980). Higher mental processes in hyperactive children: Implications for training. In R.M. Knights & D.J. Bakker (Eds.), *Treatment of hyperactive and learning disordered children* (pp. 65–91). Baltimore: University Park Press.

Ferguson, H., Simpson, S., & Trites, R. (1976). Psychophysiological study of methylphenidate responders and nonresponders. In R. Knights & D.J. Bakker (Eds.), *Neuropsychology of learning disorders* (pp. 89–98). Baltimore: University Park Press.

Grossman, S.P., & Sclafani, A. (1971). Sympathomimetic amines. In E. Furchtgott (Ed.), *Pharmacological and biophysical agents and behavior*. New York: Academic Press.

Hicks, R.E., Gualtieri, C.T., Mayo, J.P., Schroeder, S.R., & Lipton, M.A. (1985). Methylphenidate and homeostasis: Drug effects on the cognitive performance of hyperactive children. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and treatment rationale* (pp. 131–141). New York: Spectrum Publications.

Katkin, E.S. (1975). Electrodermal lability: A psychophysiological analysis of individual differences in response to stress. In I. Sarason & C.D. Spielberger (Eds.), *Stress and anxiety* (Vol. 2, pp. 141–176). New York: Academic Press.

Kelleher, R.T., & Morse, W.H. (1968). Determinants of the specificity of behavioral effects of drugs. *Ergebnisse der Physiologie, 60*, 1–56.

Keenan, V. (1970). Presentation method and learning time constancy in serial learning. *Canadian Journal of Psychology, 24*, 311–320.

Kinsbourne, M. (1985). Base-state dependency of stimulant effects on the cognitive performance of hyperactive children. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and treatment rationale* (pp. 143–154). New York: Spectrum Publications, Inc.

Koffman, E.C., & Weinstock, R.B. (1974). Total-time hypothesis in low meaningful serial learning: Task, age, and verbalization instructions. *Journal of Experimental Psychology, 103*, 1210–1213.

Lykken, D.T., & Venables, P.H. (1971). Direct measurement of skin conductance: A proposal for standardization. *Psychophysiology, 8*, 656–671.

Porges, S.W., Walter, G.F., Korb, R.J., & Sprague, R.L. (1975). The influence of methylphenidate on heart rate and behavioral measures of attention in hyperactive children. *Child Development, 46*, 727–733.

Robbins, T.W., & Sahakian, B.J. (1979). "Paradoxical" effects of psychomotor stimulant drugs in hyperactive children from the standpoint of behavioral pharmacology. *Neuropharmacology, 18*, 931–950.

Satterfield, J.H., Atoian, G., Brashers, G.C., Burleigh, A.C., & Dawson, M.E. (1974). Electrodermal studies in minimal brain dysfunction children. In C. Conners (Ed.), *Clinical use of stimulant drugs in children*. Amsterdam: Excerpta Medica.

Satterfield, J.H., & Dawson, M.E. (1971). Electrodermal correlates of hyperactivity in children. *Psychophysiology, 8*, 191–197.

Siddle, D.A.T. (1972). Vigilance decrement and speed of habituation of the GSR component of the orienting response. *British Journal of Psychology, 63*, 191–194.

Siddle, D., & Smith, D. (1974). Effects of monotonous stimulation on cortical alertness in fast and slow habituation groups. *Journal of Research in Personality, 8*, 324–334.

Solanto, M., & Conners, C.K. (1982). A dose-response and time-action analysis of autonomic and behavioral effects of methylphenidate in attention deficit disorder with hyperactivity. *Psychophysiology, 19*, 658–666.

Sostek, A.J. (1978). Effects of electrodermal lability and payoff instructions on vigilance performance. *Psychophysiology, 15*, 561–568.

Sroufe, L.A., Sonies, B., West, W., & Wright, F. (1973). Anticipatory heart rate deceleration and reaction time in children with and without referral for learning disabilities. *Child Development, 44*, 267–273.

Wilkinson, R. (1960). The effect of lack of sleep on visual watchkeeping. *Quarterly Journal of Experimental Psychology, 7*, 36–40.

Zahn, T.P., Abate, F., Little, B.C., & Wender, P.H. (1975). Minimal brain dysfunction, stimulant drugs, and autonomic nervous system activity. *Archives of General Psychiatry, 32*, 381–387.

Zentall, S.S., & Zentall, T.R. (1983). Optimal stimulation: A model of disordered activity and performance in normal and deviant children. *Psychological Bulletin, 94*, 446–471.


(Manuscript received August 6, 1985; accepted for publication July 20, 1987)