THE EFFECTS OF RITALIN (METHYLPHENIDATE) ON SELF-REGULATORY PROCESSES OF HYPERACTIVE YOUNG OFFENDERS

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THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY in the Department of Psychology

c Hanna Maria Lysak, 1989
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The effects of Ritalin on self-regulatory processes of hyperactive young offenders

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ABSTRACT

Stimulant medication, especially Ritlaiin, has been a treatment of choice for childhood Attention Deficit Hyperactivity Disorder and has been researched in hundreds of studies. In contrast, the clinical lore pertaining to the efficacy of stimulants in adolescence is based on only three controlled studies. Attention Deficit Disorder is often associated with Conduct Disorder and this mixed diagnostic group is at a higher risk for chronic psychosocial impairment than "pure" ADHD samples. Therefore, the investigation of pharmacological treatment for this mixed diagnostic group was undertaken in the present study. The short-term effects of two doses of Ritalin (0.3 mg/kg and 0.5 mg/kg) were investigated in double-blind, placebo-controlled, crossover trials in a random order. Twenty seven boys between the ages of 13-17 who met the criteria for both Attention Deficit and Conduct Disorders were selected from a population of young offenders who were remanded to a closed forensic assessment unit because of criminal charges or convictions. All subjects were free of other psychiatric disorders, mental retardation, contraindications to
Ritalin, and concurrent pharmacological treatment. They were administered a battery of neuropsychological tests that included the Wisconsin Card Sorting Test, the Word Fluency Test, the Stroop Word-Color Test, the Trail Making Test-B, and the Digit Span and Digit Symbol of the Wechsler Intelligence Scale for Children - Revised. A stepwise Bonferroni procedure yielded significant improvement on the majority of the measures in all treatment conditions relative to the baseline. There was only one significant contrast between the active drug and placebo phases. The differences between two active drug doses were not significant at the Bonferroni-corrected levels of significance. A marginal tendency towards improvement in the active drug phases was observed on timed tests when contingent feedback was not provided. In contrast, a marginally significant, moderate deterioration was observed on measures derived from a more complex, self-paced, and contingently reinforced test. The theoretical and practical implications of these results are discussed within the self-regulation theory.
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Attention Deficit Hyperactivity Disorder (ADHD) refers to a syndrome of developmentally inappropriate inattention, impulsivity, and overactivity (Diagnostic and Statistical Manual of Mental Disorders-3R, 1987). This syndrome has been identified in clinical and research domains for more than 80 years (Still, 1902, cited in Dulcan, 1986). Diagnostically, it has been variously referred to as hyperkinesis, minimal brain damage/dysfunction (MBD), or attention deficit disorder with or without hyperactivity.

In the past, research on hyperactivity was hampered by theoretical assumptions of an underlying minimal brain damage or dysfunction. According to this theory, hyperactivity resulted from an injury to the brain and it was solely determined by the amount of damage, regardless of its site and origin (Pasamanick & Knoblock 1966; Rie & Rie, 1980; Shaffer, Chadwick, & Rutter, 1975). The presence of

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1 The terms ADHD and hyperactivity are used in the present paper interchangeably.
brain damage was assumed despite the absence of history of an injury or any discernible neurological abnormalities (hence the adjective "minimal") (Rutter, 1982). Such a conceptualization of the etiology of hyperactivity was not conducive to generating reliable and valid diagnosis or experimental research as it was unclear how brain dysfunction was to be measured. In particular, within this paradigm, MBD children tended to be compared to children of known brain damage of such a heterogeneity that no meaningful conclusions regarding the etiology of this disorder were permitted (Reitan & Boll, 1973).

The questionable validity and reliability of such an organically-defined syndrome, combined with the accumulation of evidence favoring deficits in attention and concentration (e.g., Douglas & Peters, 1979), resulted in a new label for the syndrome (i.e., Attention Deficit Disorder) within which there is no reference to underlying etiology. Despite the fact that this new atheoretical approach fares better than its predecessor with respect to diagnostic reliability, a dispute over the boundaries of the category continues.
In particular, the core symptoms of ADHD are also very common in Conduct Disorder (CD) which refers to a "persistent pattern of conduct in which the basic rights of others and major age-appropriate societal norms or rules are violated" (DSM-IIIR, 1987, p.53). The criteria for both ADHD and CD are specified in such a way that children tend to receive both diagnoses concurrently. This overlap led some researchers to question the validity and clinical utility of differentiating between these two syndromes (Barkley, 1982; Loney & Milich, 1982; Rutter, 1983) and to emphasize the importance of researching "mixed" ADHD/CD subtypes. The rationale for such a subclassification within the ADHD category is threefold. First, the association between the disorders is meaningful with respect to the long-term outcome. Childhood hyperactivity tends to be associated with adolescent CD and may predispose a development of adolescent CD (Gittelman & Manuzza, 1985; Satterfield, Hoppee, & Schell, 1982; Weiss, 1985). Second, the associated aggressive symptomatology in childhood hyperactivity may be more predictive of the adolescent psychosocial maladjustment than the primary attention deficit (Loney et al., 1982). Consistently, the mixed ADHD/CD symptomatology tends to be associated with the least
favorable course and long-term outcome relative to either of the diagnoses alone (McGee, Williams, & Silva, 1984). Finally, a need for differentiation between "pure" and "mixed" ADHD sub-categories received support from research on external validation of childhood psychopathology. It appears that both disorders are not only similar in their clinical presentation but also they share medical and familial background correlates (e.g., Reeves, Werry, Elkind, & Zametkin, 1987). The rationale for the differentiation of a mixed ADHD/CD category will be discussed more extensively below.

Attention Deficit Disorder in Adolescence

The prevalence of childhood ADHD ranges from 3 to 5%. The sex ratio of males to females ranges from 3-4:1 in community samples to 6-9:1 in clinic samples (Bosco & Robins, 1980; DSM-III-R, 1987; Sandoval, Lambert, & Sassone, 1980; Szatamari, Offord, & Boyle, 1989). The prevalence rates of ADHD in adolescence were investigated in only one community study (Szatamari et al., 1989). In a large sample of Ontario children between ages 4-16 (N not reported), both the prevalence rate (6%) and the male to
female ratio (3:1) were similar in children and adolescents. The findings of this generally well-controlled study must be viewed within its limitations, for the authors failed to incorporate such important diagnostic criteria as the age of onset and duration of the disorder. Such an omission may lead to somewhat inflated prevalence rates.

Traditionally, stimulant medication, especially Ritalin (Methylphenidate), has been a treatment of choice for childhood hyperactivity (Ross & Ross, 1982). Such treatment was typically discontinued before or in early adolescence as the patients were expected to "outgrow" their symptoms after reaching puberty, and to respond adversely to stimulants (Cantwell, 1975). Recent retrospective follow-back and prospective follow-up research of hyperactive probands from childhood to adolescence and/or adulthood challenged the benign prognosis of ADHD and led to the reconsideration of the need for pharmacotherapy past childhood.

Methodologically rigorous prospective studies revealed that only 20 to 50% of hyperactive children were asymptomatic in adolescence (e.g., Lambert, 1988; Weiss,
1985). The remaining 50 to 80% continued to experience some of the core symptoms past childhood. For example, Lambert (1988) identified 43% of previously hyperactive adolescent boys who continued to have the full syndrome of Attention Deficit Disorder with Hyperactivity (ADDH) (DSM-III, 1980) and were receiving pharmacological treatment for this disorder. An additional 37% still had some problems with inattention and impulsivity, although overactivity had diminished (Residual Type in DSM-III, 1980). Similarly, Gittelman et al. (1985) found that the full syndrome persisted into adolescence in a substantial, although smaller proportion of children (24%). Those adolescents who still had two of three core symptoms, however, were not less likely to experience motor restlessness as was purported by Lambert (1988) or others (Weiss, Hechtman, Perlman, Hopkins, & Werner, 1979; Weiss, Minde, Werry, Douglas, & Nemeth, 1971). Conversely, not only was the quantity of teenage overactivity elevated relative to normal controls (Aman, 1984; Gittelman et al., 1985) but also the activity remained unfocused (on goal) and disorganized (Cantwell, 1986).

In addition to ADHD symptoms, a substantial number of adolescents with a childhood history of hyperactivity tended
to demonstrate conduct problems. Lambert (1988) and Lambert, Hartsough, Sassone, and Sandoval (1987) reported that by the age of 14 years, 19% of the hyperactive compared to 3% of the normal control sample was in conflict with the law, and by the age of 17 or 18 years, 9% were adjudicated delinquents. Other authors reported even higher (25-50%) delinquency rates in hyperactive probands (Gittelman, 1982, cited in Cantwell, 1986; Satterfield et al., 1982; Weiss, et al., 1971). Interestingly, in one study which controlled for socio-economic status (SES) of the subjects, the frequency of the offenses was evenly distributed across low, middle, and upper-middle social strata (Satterfield et al., 1982), suggesting that the offences were not simply a reflection of common SES artifacts in criminal behavior.

In conclusion, core ADHD symptoms tend to persist into adolescence and impair psychosocial functioning. Often they are "masked" by secondary problems typically associated with ADHD such as antisocial behavior, school underachievement, or poor peer relations. These secondary symptoms rather than ADHD per se tend to be of primary significance in the referral of adolescents to mental health facilities (Cantwell, 1986; Weiss, et al., 1979). The presence of
only one of the core symptoms at puberty does not seem to differentiate these adolescents from normal controls, suggesting that the syndrome rather than any of the symptoms in isolation is causing psychosocial impairment (Gittelman et al., 1985).

The outcome of the follow-up studies of ADHD children into adulthood appears to be consistent with that of adolescence. Approximately 31% of hyperactive probands compared to 3% of normal controls still had a full ADHD syndrome in early adulthood (Gittelman-Klein, 1982, cited in Weiss, 1985). Fifty percent had some of the core symptoms (Borland & Hechtman, 1976) and about 66% of the hyperactives compared to 10% of controls had at least one core symptom (Weiss et al., 1979; Weiss & Hechtman, 1986). Antisocial personality disorder or antisocial conduct were found in 27% of the samples at the follow-up (Borland et al., 1976; Gittelman-Klein, 1982, cited in Weiss, 1985; Weiss et al., 1979, 1986). Most of the probands in these studies were employed in adulthood but their SES or work status was significantly lower than those of controls, thus indicating an inferior psychosocial adjustment (Borland et al., 1976; Weiss et al., 1986).
In conclusion, the full ADHD syndrome or any two core symptoms in previously hyperactive adolescents and adults tend to persist in a large proportion of the probands and reflect the chronic nature of this disorder. The association of this symptomatology with an impairment in psychosocial functioning is reflected in high rates of antisocial disorders, school underachievement, and poor social relations in adolescents and lower SES and work status in adulthood.

The high incidence of antisocial disorders in "grown-up" hyperactive subjects led some authors to propose that hyperactivity constitutes a pre-disposing factor in the development of conduct disturbance and delinquency (Gittelman et al., 1985; Satterfield et al., 1982; Weiss, 1985). Although grounded in rigorous research, this interpretation is probably erroneous given a common practice of including subjects with symptoms of low frustration tolerance, irritability, temper tantrums, and conduct problems in ADHD samples. The question then, of whether antisocial disturbance is an outgrowth of hyperactivity or an artifact resulting from an undiagnosed early conduct disturbance cannot be unequivocally answered within this research.
paradigm. Using a more appropriate approach, August, Stewart, and Holmes (1983) evaluated adolescent outcome separately for groups of pure ADHD and mixed ADHD/undersocialized aggressive males. Researchers blind to the subjects' childhood psychiatric status found that the antisocial outcome was preceded by childhood conduct disturbance rather than hyperactivity. In this study, none of the purely hyperactive boys, but 37% in the mixed diagnostic group were given a diagnosis of conduct disorder at the follow-up. These findings might very well reflect the "true" outcome of ADHD.

The common practice of an inadvertent and unsystematic inclusion of conduct problems in hyperactive samples was widely criticized for its contribution to the heterogeneity of samples and unreliability of findings in this research area (e.g., Loney, Langhorne, & Paternite, 1978). These criticisms stimulated more systematic investigations of the associative links of ADHD with CD, and recommendations for dividing a category into "pure" and "mixed" subtypes. Comorbidity literature pertaining to this issue consists of comparisons of clinic-referred pure ADHD with mixed ADHD/CD types (August & Stewart, 1982; Loney et al., 1982). Even
more informative studies employed multiple diagnostic groups
such as ADHD, CD, ADHD/CD, anxiety, depression, and
somatization disorders (Munir, Biederman, & Knee, 1987;
Reeves et al., 1987). In such mixed psychiatric groups,
pure ADHD types constituted 21-28% (Stewart, Cummings, &
Singer, 1981; August et al., 1982; Lahey et al., 1988).
Relatively larger proportions of subjects received mixed
ADHD/CD diagnosis: 34 to 75% in hyperactive samples (August
et al., 1982; Biederman, Munir, & Knee, 1987) and approxi-
mately 27% in mixed psychiatric samples (Lahey et al.,
1988).

These studies have also revealed a striking diagnostic
heterogeneity of "hyperactive" samples which may partially
account for highly inconsistent findings across different
areas of outcome. They have further reinforced the need for
a sub-classification within this category according to the
associated psychopathology. For example, in one study 96%
of clinic-referred ADHD children had at least one additional
DSM-III-R (1987) diagnosis (Munir et al., 1987). ADHD was
most commonly associated with CD and Oppositional Personal-
ity Disorder (64% overlap), followed by depression (32%) and
anxiety disorders (27%). A similar heterogeneity within an
ADHD sample was observed in an Ontario community study (Szatamari et al., 1989). Thirty-nine percent of hyperactive children and 47% of hyperactive teenagers had CD as well. The overlap of ADHD with emotional disorders was approximately 19% in childhood and 37% in adolescence. Finally, 30% of the adolescent ADHD sample had also somatization disorders (no data for children were reported).

In conclusion, although it is possible to identify pure ADHD, more often hyperactive children and adolescents are given at least one additional diagnosis, usually that of CD. The high incidence of a combined ADHD/CD diagnosis in most studies has probably resulted in an artifact described by Loney et al. (1982). Somewhat cynically, they said, "we do not have a literature about childhood hyperactivity as such; instead, we have a literature about childhood externalizing behavior problems (hyperactivity and aggression) that we call a literature about childhood hyperactivity" (p. 143). It appears that this artifact could be successfully eliminated by systematically evaluating CD in ADHD probands and differentiating pure from mixed ADHD subtypes (Henker & Whalen, 1989).
The recommendation for systematic evaluation of concurrent CD also stems from the least favorable long-term outcome of this mixed group relative to either of the diagnoses alone (McGee et al., 1984). The joint diagnosis seems to retain the negative characteristics of either disorder, thus magnifying the degree of psychosocial impairment. Further, research on external validation of childhood DSM-III (1980) diagnoses provided additional support. For example, Reeves et al. (1987) found more similarities than differences between ADDH and mixed ADDH/CD samples in sex ratio, clinical presentation of ADDH symptomatology, pre- and perinatal history, cognition, and achievement. Both groups differed from samples of normal controls and anxiety disorders in that they were consistently handicapped with respect to the above mentioned factors. The distinguishing factors were also identified: The ADDH/CD group had an earlier age of onset, significantly more antisocial symptomatology, and severe adverse family background (over 30% had antisocial and alcoholic fathers). In both pure and mixed hyperactive samples, there was no relationship between child and parent ADDH symptomatology. These findings, with the exception of substance abuse in fathers, are generally in agreement with other controlled studies of parental
psychiatric comorbidity of ADHD, CD, and ADHD/CD clinic-referred children (Biederman et al., 1987; Lahey et al., 1988; Stewart, DeBlois, & Cummings, 1980).

ATTENTIONAL DEFICIT: Can’t or Won’t?

Consistent with research findings available in the 1970s (e.g., Douglas et al., 1979), the DSM-III (APA, 1980) and DSM-IIIR (APA, 1987) postulated that problems with inattention are essential to ADHD. Contrary to expectations, this research-based conceptualization of the disorder has not only failed to clarify the confusion with this diagnostic category but it introduced further problems as well. One of the most serious problems rests on the failure to discriminate between an intrinsic cross-situational deficit ("can’t") and situation and task-contingent performance deficit ("won’t"). This shortcoming is partially related to the confusing definition of attention.

Attention is not a unitary concept, but subsumes a multiplicity of psychophysiological processes dependent on motivation and effort. Individuals displaying inattention may have deficits in several cognitive areas. They may be
unable to either sustain attention over an extended period of time (vigilance), focus on relevant stimuli while ignoring distractions (selectivity), withhold responses to nontarget stimuli (impulsivity), or refrain from responding to initially rewarding targets when it is no longer appropriate (flexibility). It was originally unclear whether all of these processes were impaired in ADHD and whether this impairment was consistent across time and situation.

To date, laboratory findings have been equivocal, although an unexpected trend is emerging from rigorous research: An intrinsic attentional deficit may be more apparent than real. Within the original research paradigm, the hyperactive versus normal-control group comparisons yielded differences predominately in the areas of impulsivity, vigilance, and sustained attention (Douglas, 1972; Douglas et al., 1979; Douglas, 1983; Hoy, Weiss, Minde, & Cohen 1978). Hyperactive subjects tended to make errors of omission (failure to respond to target stimuli) and were faster and more inaccurate in their errors of commission (failure to inhibit responses to non-target stimuli). Additionally, their performance tended to deteriorate over time. Deficits have not been consistently found
in selective attention (Drager, Prior, & Sanson, 1986; Prior & Sanson, 1986) or distractibility (Douglas et al., 1979; Douglas, 1983; Tant & Douglas, 1982).

Recently, some researchers postulated that faulty methodology might account for the differences found between hyperactive and normal samples. First, the power of the most frequently utilized tests of vigilance (Continuous Performance Test [CPT], Rosvald et al., 1956) and impulsivity (Matching Familiar Figure Test [MFFT], Kagan, Rosman, Day, Albert, & Philips, 1964) to detect core deficits specific to ADHD is questionable. Poor performance on the CPT was found to be characteristic not only of hyperactivity but also of schizophrenia (Friedman, Erlenmyer-Kimling, & Vaughan, 1982) and CD (Quay, 1986b). Similarly, impaired performance on the MFFT was not unique to ADHD but it correlated with anxiety (Messer, 1970), depression (Schwartz, Friedman, Lindsay, & Narrol, 1982), and CD (Shaffer, McNamara, & Pincus, 1974). It appears that the multitude of differences encountered in the hyperactives versus normals comparisons may be attributable to the "patient" status of hyperactives rather than to the ADHD status per se. This hypothesis is supported by some authors
who claim that attention deficits are generally characteristic of disturbed children regardless of the nature of their psychopathology (Shaffer et al., 1974). Further, a strong emphasis has been placed recently on the need to validate the "fundamental deficit" by including comparisons of hyperactives with other pathological groups in order to support the unique associations of attentional deficits with ADHD. Two rigorous studies of multiple diagnostic categories found that both the CPT and MFFT failed to discriminate between ADDH, CD, mixed ADDH/CD, and anxiety disorders (Koriath et al., 1985; Werry, Elkind, & Reeves, 1987). Additionally, "patients" as a group, and hyperactive patients in particular, were consistently found to be cognitively inferior to normal samples, especially in the area of Full Scale and Verbal I.Q. scores of the WISC-R (Werry et al., 1987).

The fact that the level of intellectual ability can powerfully influence performance of hyperactive samples on attentional tasks has been long recognized but often not well-controlled. In almost all studies, average I.Q. scores of hyperactive and normal groups were compared. Although the differences were typically not statistically
significant, they probably still allowed for clinically meaningful differences. In a study in which the effects of I.Q. were partialled out, the initially inferior performance of both ADHD and mixed ADHD/CD groups relative to anxiety and normal controls disappeared (Werry et al., 1987). Interestingly, following such adjustment for the I.Q. level, one remaining significant finding was a higher number of errors of commission on the CPT performed by mixed ADHD/CD (but not pure ADHD group) relative to controls. This finding suggests that impulsivity in hyperactive samples might be partially related to an undiagnosed CD (Quay, 1986a & b; Taylor, 1980).

Finally, the age of participants was not accounted for in most studies. This appears to be an important shortcoming in light of the fact that the ability to sustain attention or inhibit impulsive responding improves in the course of development (Aman & Singh, 1982; Geffen & Sexton, 1978). Consequently, attentional problems may be modified by the age of the participants (Aman & Turbott, 1986; Werry et al., 1987).
Perhaps one of the most serious methodological problems in studies which found impairment in attention was a failure to discriminate between a pervasive constitutional deficit and situation-contingent performance deficit. In studies where impaired performance was found, the experimental tasks tended to be not only automatic, rigid, boring, or ecologically irrelevant but in addition subjects were monitored by a computer or an experimenter seated behind a one-way mirror. In contrast, hyperactives were able to mobilize their "hidden" skills and were indistinguishable from normals in their performance on attentional tasks when environmental manipulations included self-paced performance (e.g., Sykes, Douglas, Morgenstern, 1972), external contingent feedback (Firestone & Douglas, 1975; Parry, 1973 cited in Douglas, 1983), and the presence of the experimenter during the task (Drager et al., 1986; Milich, Loney, & Landeau, 1982; Nuchterlein, 1983; Prior, Sanson, Freethy, & Geffen, 1985).

It appears that the sensitivity of ADHD patients to environmental manipulations is not consistent with an irreversible pervasive deficit, but it is consistent with a transactional disorder reflecting a child by situation
interaction over time (Henker et al., 1989). It could be argued that the presence of the experimenter during some studies reduced the need for internal control and enhanced children’s motivation and effort. This hypothesis is viable in the light of the common observations that hyperactives tend to be unreliable performers as they have problems mobilizing effort and inhibitory control on dull and repetitive tasks (Barkley, 1982; Douglas, Barr, O’Neill, & Britton, 1986; Douglas, Barr, Amin, O’Neil, & Britton, 1988; Dykman, Ackerman, & Oglesby, 1979; Rosenbaum & Baker, 1984). Douglas (1983) reviewed the relevant literature and concluded that ADHD children notoriously failed to "make consistent use of information and skills that they are known to possess" (p. 283).

In conclusion, the newly emerging view in the literature favors a defective functional self-regulatory mechanisms rather than a constitutional attentional deficit. This departure from a structural medical model is important from a therapeutic standpoint, as optimal situational or pharmacotherapeutic manipulations could be expected to reverse the core handicap through the facilitation of goal-oriented behavior or the inhibition of inappropriate
effort, and bring the performance of hyperactives to the level of normals.

Etiological Hypothesis: Faulty Self-Regulation

Attentional problems associated with ADHD and CD implicate the role of higher cortical inhibitory mechanisms in these disorders. Several authors have drawn striking parallels between the disinhibition of the central regulatory mechanisms presumably located in the anterior area of the brain in both ADHD (Chelune, Ferguson, Koon, & Dickey, 1986; Mattes 1980; Solanto, 1984) and CD (Gornstein & Newman, 1980; Pontius, 1974; Pontius & Ruttiger, 1976; Pontius & Yudowitz, 1980; Rosenthal & Allen, 1978).

According to Luria's (1980) theory of higher cortical regulation and Lezak's (1983) theory of executive functions, frontal lobe activity is essential for programming and executing complex goal-directed behavior due to the intimate connections of this part of the brain with the reticular formation system, motor cortex, and anterior zones of speech area. Clinical evidence (Luria, 1980) suggests that purposeful behavior tends to be disturbed in patients with even
the mildest forms of frontal dysfunction. Such individuals tend to perseverate, be inattentive, disinhibited, impulsive, or unmotivated. Clinical observations are corroborated by a large number of experimental studies comparing frontally and non-frontally damaged psychiatric patients (Kolb & Whishaw, 1985; Lezak, 1982, 1983; Milner & Petrides, 1984). It appears that the dysregulation of behavior manifested in inattention, impulsivity, and poor cognitive flexibility typically found in both ADDH and CD is associated with frontal lobe functions (Kolb et al., 1985; Lezak, 1982, 1983; Milner et al., 1984; Mesulam, 1986; Stuss & Benson, 1984) and may warrant a systematic neuropsychological evaluation.

Within Luria's (1980) theory, the ability to subordinate one's action to a formulated goal and to carry out a plan is formed in the course of development. Some authors proposed that the frontal lobes become functional between the ages of four and seven. They continue to mature until old age (Luria, 1966, 1973), with spurts occurring in adolescence (Yakovlev & Lecours, 1967). Functions associated with frontal lobe maturity such as concept formation, cognitive flexibility, inhibition, planning, or ability to
benefit from the environmental feedback were measured in experimental studies by the Wisconsin Card Sorting Test (Heaton, 1981), the Trail Making Test-B (Reitan, 1979), the Verbal Fluency Test (Benton & Hamsher, 1976), subjects' narratives, or the structure of actual criminal actions committed by subjects (Chelune & Baer, 1986; Chelune et al., 1986; Gaddes & Crockett, 1973; Golden, 1981; Passler & Hynd, 1985). Regardless of the measure used in the studies, an adult-like level of performance was demonstrated by children by the age of twelve.

Stimulant Treatment of Hyperactivity

Childhood hyperactivity has been treated with stimulant medication such as methylphenidate hydrochloride (Ritalin), magnesium pemoline (Cylert), amphetamine (Benzedrine), or dextroamphetamine sulphate (d-amphetamine, Dexedrine) for over thirty years (Barkley, 1981; Walker, 1982). These psychoactive compounds were originally prescribed for a disparate group of "hyperactives" who presented a multitude of additional symptomatology including anxiety, depression, aggression, psychosis, or school phobia.
Until the early 1970s not only were the drug doses recommended by psychopharmacology experts unduly high, but also clinicians tended to prescribe the drugs rather indiscriminately to children with a variety of behavior problems. For example, 10% of school-age children were treated with stimulants in the early 1970s (Ross et al., 1982) despite the fact that the prevalence of ADHD in this population is 3-5% (Bosco et al., 1980; DSM-IIIR, 1987; Szatamari et al., 1989). The wide-spread use of stimulants was criticized by both the public and the research community. Labels such as "chemical straightjackets" and accusations of "drugging" children into submission were put forward (Dulcan, 1986; Ross et al., 1982). These criticisms led to the limitation of the use of stimulants primarily to ADHD populations and to the reduction in the dosage levels by 300-600% (Sprague & Slator, 1975). Finally, they led to the explication of standards for better-controlled efficacy research (Sprague et al., 1975).

To date, a large body of evidence has accrued which supports short-term beneficial effects of stimulants for the behavioral overactivity and performance on simple laboratory tasks in children (see Douglas, 1983, for a review; Douglas
et al., 1986, 1988; Kavale, 1982; Whalen & Henker, 1976). Despite this generally favorable outcome, a number of issues have remained unresolved. First, the effects of stimulants on complex learning and problem solving skills are still controversial (Aman, 1980; Barkley, 1979; Barkley & Cunningham, 1978; Gadow, 1983; Kavale, 1982; Ottenbacher & Cooper, 1983; Rie, Rie, Stewart, & Ambuel, 1976). Some authors asserted that stimulants may have little effect on these skills (Abikoff, 1985; Abikoff & Gittelman, 1985; Gadow, 1983; Tant et al., 1982) or may even cause a deterioration in goal-oriented behavior due to drug-induced "overfocusing" and "perseverativeness" (Fiedler & Ullman, 1983; Margolin, 1978; Robbins & Sahakian, 1979; Sahakian & Robbins, 1977; Sroufe, 1975). Another debated issue is related to the relative input of motivation, compliance, and cognitive ability into the performance of hyperactives and dose-contingent effects of stimulants on these functions (Douglas, 1983; Spraque et al., 1975). Further, the wide heterogeneity of responses to the drug across child, task, time, situation, and dosage has defied any attempts to predict the response of an individual child within a group (Douglas et al., 1986; Pelham, Bender, & Caddell, 1985; Sebrechts et al., 1986; Taylor, 1983; Taylor et al.,
Finally, the evidence for long-term effects of stimulants is lacking (Dulcan, 1986). It appears that drug treatment in childhood may not influence the adolescent outcome (Charles & Schain, 1981; Hechtman, 1985; Satterfield et al., 1982; Sroufe, 1975; Weiss et al., 1975). These findings might be partially related to methodological artifacts such as a failure to control for the proper implementation of the treatment protocol as well as for patients' compliance (Firestone, 1982; Kaufman, Smith-Wright, Reese, Simpson, & Jones, 1981;).

Negative attitudes of consumers towards stimulant medication combined with pervasive myths regarding drug action proved to be a major drawback in the long-term efficacy research. Children tend to dislike taking medication due to feared negative side-effects (e.g., decreased athletic ability) and perceived social stigma associated with mental illness (Henker & Whalen, 1980). Parents prefer nonmedical treatment and tend to alter or discontinue recommended dose without consulting a physician (Dulcan, 1986; Henker et al., 1980). Pediatricians tend to withdraw drug treatment just before or early in adolescence (Weiss, 1981). Such medical practice is seemingly based on a system
of beliefs that children would spontaneously "outgrow" attention problems. More importantly, adolescents were expected to display a "normal" excitatory rather than "paradoxical" calming response to stimulants. A related myth concerns a greater than normal risk of drug abuse expected in children exposed to stimulant medication (Clampit & Pirkle, 1983; Gross & Wilson, 1974; Weiss et al., 1986).

Recently, the need for the psychotropic treatment of adolescents has been recognized. For example, Safer and Krager (1985) reported that 30-50% of patients receiving stimulants in childhood continued their treatment in adolescence. Additionally, the rate of stimulant treatment among adolescents between 12-15 years increased by 158% between 1975-1983 (change from .59 to 1.52%) in a community sample.

The myth of stimulant abuse by adolescents or the alleged predisposing role of stimulants to the development of substance abuse has not been supported by research (Ackerman, Dykman, & Peters, 1977; Henker et al., 1981; Klorman, Coons, & Borgstedt, 1987; Loney et al., 1981; Mendelson, Johnson, & Stewart, 1971). There is only one
case of stimulant abuse reported in the literature (Goyer, Davis, & Rapoport, 1979). Long-term negative side-effects such as growth suppression or cardiovascular changes have not been systematically studied, although transient elevation of heart rate and blood pressure are possible (see Varley, 1985, for a review).

Similarly, the myth of "paradoxical" effects of stimulants (especially most frequently studied Ritalin) has been dispelled by studies which demonstrate that the same organizing effects are present in all age groups in both hyperactive and normal samples (Rapoport et al., 1978, 1980). Some researchers argued that the only discernible difference in the response to stimulants of hyperactives and normals rests on the more dramatic placebo response of the former group. For example, Zahn and his colleagues (Zahn, Rapoport, & Thompson, 1980) found that in dextroamphetamine-placebo controlled trials, placebo-related improvement was significantly higher (p<.05 to p<.001) in hyperactive relative to normal children on measures of motor activity, focused attention, and impulsivity.
Of particular importance to the present study are findings suggestive of the possible efficacy of stimulants in adolescents (Brown & Sexon, 1988; Coons, Klorman, & Borgstedt, 1987; Lerer & Lerer, 1977; McKay, Beck, Taylor, 1973; Safer & Allen, 1975; Varley, 1983). Similarly, the apparent beneficial effects of stimulant medication for groups of aggressive (Amery, Minichiello, & Brown, 1984; Loney et al., 1980) and delinquent populations (Maletzky, 1974) are relevant to the present research.

To date, despite several open trials of Ritalin in adolescents, only three controlled studies have been reported (Coons et al., 1987; Brown et al., 1988; Varley, 1983). Attention Deficit Disorder in these studies was diagnosed using either the DSM-III - based on (1) structured patient, parent, and teacher interviews (Varley, 1983), (2) semi-structured parental interview corroborated by Conners Teacher Rating Scale (Brown et al., 1988), or (3) retrospective childhood ratings on the Abbreviated Conners Parent Hyperactivity Questionnaire and the Home Activity Scale (Coons et al., 1987). In all cases, probands had a childhood history of Attention Deficit Disorder (the age of onset was not specified) and were free of psychosis, organic brain
disorder, and mental retardation\(^2\). In two studies, the presence of CD was systematically evaluated: No subject with CD was included by Varley (1983), whereas 45% of the Brown et al.'s (1988) sample had an associated CD. Over 60% of the probands were previously treated with Ritalin in two studies (Varley, 1983; Coons et al., 1987).

The design in the three studies was double-blind, methylphenidate and placebo crossover trials in a random order. The drug dosage was either standardized in mg/kg of body weight (Brown et al., 1988; Varley, 1983) or a fixed quantity of the compound was prepared for all subjects (Coons et al., 1987). Regardless of the preparation method, the drug was administered twice daily: one dose in the morning and one in the afternoon.

Negative side-effects were reported only for higher doses: increase in blood pressure (Brown et al., 1988; Coons et al., 1987).

\(^2\)The demographic characteristics of the subjects were as follows: (1) \(N = 22\) (17 males and 15 females); age 13-18, \(M = 14.27\); WISC-R Full Scale I.Q. = 95.1 (SD not reported) (Varley, 1983); (2) \(N = 19\) (sex not reported); age 12-19, M not reported; I.Q. not reported (Coons et al., 1987); (3) \(N = 11\) (black males); age 12-14, \(M = 13\) years 7 months; WISC-R Full Scale I.Q. = 92.91 (SD = 5.28) (Brown et al., 1988).
Varley, 1983), increase in the heart rate (Brown et al., 1988), as well as sleep disturbance and appetite suppression (Varley, 1983). None of the side-effects was serious enough to warrant withdrawal of any of the subjects from the studies.

The results of these controlled studies are as follows. Varley (1983) reported positive drug effects relative to placebo on behavior and school performance measured by parent and teacher narratives and Conners rating scales. In particular, the author reported "significant" contrasts between placebo and .15 mg/kg as well as placebo and .3 mg/kg on all measures. Differences between .15 mg/kg and .3 mg/kg were found only on the parent and teacher narratives. Careful examination of the results, however, revealed that the author failed to control for the family-wise type I error. An introduction of a Bonferroni correction for the number of contrasts by the present author yielded a marginal rather than "significant" improvement due to the drug.

Coons et al. (1987) found that Ritalin compared to placebo enhanced the accuracy and precision of information processing measured by a memory search task and the
Continuous Performance Test (CPT), as well as speed on the CPT. The performance of a small pilot subgroup \( N = 6 \) treated with stimulants in childhood was also compared to an equal number of "never treated" controls matched for age, I.Q., and diagnostic data. The performance of both groups did not differ, suggesting that the prior exposure to stimulants may have little effect on the current response to Ritalin. Similar to the previous study, these results should be viewed within the methodological limitations of this research. The authors did not use the DSM-III (APA, 1980) diagnostic criteria, therefore their sample may not be representative of a hyperactive population as it is currently defined by the psychiatric community. Further, they administered a fixed dose of the drug (15 mg/daily in the first week and 25 mg/daily for subsequent two weeks) to all subjects. This method does not equate therapeutic effects of the drug across subjects of disparate body weights (Sprague et al., 1975). The researchers also combined two different doses of the medicine in their analyses despite a common finding of nonuniform effects of different doses on different aspects of behavior (Sprague et al., 1975).
Finally, Brown et al. (1988) found "significant" improvement on measures of attention and impulsivity on .3 mg/kg and .5 mg/kg compared to placebo and to .15 mg/kg. They also reported "significant" differences between .15 mg/kg and .5 mg/kg for the majority of behavioral, academic, and laboratory measures. The authors concluded that their results support a "significant" linear dose-response relationship where the increased amount of the drug is associated with enhanced performance. Similar to the previously reviewed studies, such conclusions are not justified given the violations of some important statistical assumptions by these authors. First, 36 dependent measures were derived from a small subject sample (N = 11); 75% of these measures were significant at .05 or .01 level. It is likely that some of these results were significant by chance. For each of the 28 significant variables, six pairwise comparisons were performed to ascertain the sources of the significance. Again, p-values for each contrast were reported as either .05 or .01, suggesting a failure to control for a family-wise type I error.

In conclusion, the reviewed short-term Ritalin efficacy studies of adolescent samples met major standards of
contemporary psychopharmacology research. They employed a double-blind design in which an active drug and placebo were investigated in crossover trials in a random order. Nevertheless, the authors had a tendency to draw unwarranted conclusions from their studies. Statistical errors included an excessive number of dependent variables relative to the sample size and a failure to correct for a family-wise type I error. When the present author introduced a Bonferroni correction to control for a familywise type I error, only marginally significant results were found.

**Mechanism of Drug Action: Further Link to the Self-Regulatory System**

As yet the neurochemical effects or the mechanism of the stimulants' action is poorly understood (Barkley, 1981; Solanto, 1984). The psychopharmacological aspect of stimulants, including methylphenidate, is believed to be that of increasing catecholamine (i.e., dopamine and its derivative norepinephrine) activity in the Central Nervous System (CNS) (Margolin, 1978; Solanto, 1984). The actual locus of their action in the CNS is not well studied (Barkley, 1981).
Most recently, evidence from epidemiological, pharmacological, and clinical studies in children, as well as from animal research has led some investigators to suggest that depletion in brain neurotransmitters, particularly catecholamines, may be central to hyperactivity (Conners, Eisenberg, & Sharpe, 1964; Margolin, 1978; Reimherr, Wender, Wood, & Ward, 1987; Shahakian & Robbins, 1977; Shaywitz, Shaywitz, Cohen, & Young, 1984).

In the animal model of hyperactivity, developing rat pups are depleted of brain dopamine via intracisternal administration of the neurotoxin 6-hydroxydopamine (6-OHDA). Such treatment results in rapid and permanent reduction of the brain dopamine to concentrations of 10 to 25% of that in controls. The depletion of dopamine results in attentional and other cognitive difficulties, which are completely reversed by amphetamine or methylphenidate treatment.

Of particular interest to the study of self-regulatory functions in hyperactivity is the finding of high dopamine levels in the prefrontal association cortex of the normal rhesus monkey and its unique role in mediating cognitive functions (Brozoski, Brown, Rosvald, & Goldman, 1979). The
authors reported that dopamine-depleted animals showed a profound deficit in a delayed alteration task which depends on the frontal lobe function. This deficit was nearly as severe as that seen in animals with surgical lesions of the prefrontal cortex. Consistent with the frontal lobe syndrome (Kolb et al., 1985), this cognitive deficit was selective and did not extend to the functions governed by posterior parts of the brain. As expected, general cognitive ability did not deteriorate and was reflected by good performance on a visual discrimination task. The depletion of other neurotransmitters (either norepinephrine or serotonin) in control subjects did not alter performance on the delayed alteration task. The injection of a dopamine precursor, L-Dopa, a substance which can bypass the blood-brain barrier and elevate the levels of brain dopamine, completely reversed the cognitive deficit in the experimental subjects. Importantly, drugs that did not act on the dopaminergic system did not produce significant behavioral recovery.

Shaywitz, Yager, and Klopper (1976) reported similar effects of dopamine depletion via 6-OHDA on motor overactivity and cognitive functioning in rats.
Interestingly, motor overactivity spontaneously abated in rats by the time they reached three to four weeks of life. This constitutes an interesting parallel to hyperactivity in children in which behavioral overactivity somewhat abates by late childhood.

In summary, the reviewed findings suggest that the levels of dopamine found in the prefrontal cortex of normal and surgically depleted subjects might be linked to self-regulatory functions associated with the frontal lobes.

Several studies have addressed the issue of neurochemical abnormalities in residual ADDH in human adults. The effects of the precursors of catecholamine synthesis such as 1-dopa, d,l-phenylalanine, and l-tyrosine were studied. The rationale behind these trials was such that a demonstration of the efficacy of these precursors in Attention Deficit Disorder would suggest that decreased dopaminergic or noradrenergic activity plays a role in the development of this disorder while increased activity ameliorates this disorder.
L-Dopa in low doses produced nausea and sedation in patients. This precluded a trial of higher and presumably optimally effective doses (Wood, Reimherr, & Wender, 1982). D,L-Phenylalanine did decrease overactivity, but did not improve concentration in another study (Reimherr, Wender, Wood, & Ward, 1985). L-Tyrosine produced significant improvement in core symptoms after two weeks of treatment and was effective for subsequent six weeks of treatment. After a total of eight weeks, however, the patients developed tolerance for the drug and experienced a relapse of symptoms. The fact that the precursors produced putative changes only in some target areas for short periods of time are difficult to interpret given the exploratory nature of these studies.

According to the neurochemical hypothesis of hyperactivity (Wender, 1973), if the drug were specifically targeted to the disorder, it should consistently and exclusively alter symptoms in the hyperactive individuals in a way which is qualitatively different from normals (Solanto, 1984). Contrary to this hypothesis, most studies have failed to demonstrate that the effects of stimulants are unique in hyperactive probands. Stimulants tend to produce positive
responses on behavioral and laboratory tasks not only in the ADDH children but also in normal children, adolescents and, adults (Latties & Weiss, 1967; Rapoport et al., 1978; Rapoport et al., 1980; Weiss & Latties, 1962). Improvements were also observed in ADDH children on tasks on which their performance was comparable to normals (Sroufe, Sonies, West, & Wright, 1973; Sykes et al., 1972). These results have led some authors to postulate that stimulants are not specifically targeted to a biochemical cause. Rather, they might enhance motivation and effort and consequently ameliorate behavioral symptoms (Barkley, 1977; Solanto, 1984). Consistent with this hypothesis are findings of facilitative effects of stimulants in normal adults found under conditions of low motivation, fatigue, or boredom (Latties et al., 1967).

Goals of the Present Study

The purpose of the present study was to investigate the short-term efficacy of mild and moderate doses of Ritalin, relative to placebo on self-regulatory processes of hyperactive, conduct disordered young offenders. As previously discussed, the importance of selecting this subgroup of
hyperactive subjects was twofold. First, the co-existence of CD and ADHD is very common. Second, the co-presence of CD tends to aggravate the course and prognosis of ADHD. Consequently, the postulated high risk for chronic psychosocial impairment of this population warrants due investigation of treatment options (McGee et al., 1984; Loney et al., 1982).

To date, little evidence pertaining to pharmacological intervention in the teenage ADHD/CD group is available. Some support for stimulant efficacy can be derived from previously reviewed several open trials and only three controlled studies of hyperactive adolescents. A study of a subgroup of hyperactives identified on the basis of the associated CD might yield greater homogeneity within the present sample. A similar practice, if relatively common among researchers, might improve the consistency of outcome data across studies. Typically, research which failed to systematically evaluate the associated psychopathology in hyperactive subjects yielded extreme heterogeneity of samples and unreliability of findings across studies.
Additionally, as mentioned above, the issue of the existence of a basic constitutional deficit in attention deficit disorder is still hotly debated. A competing hypothesis to a pervasive structural deficit is that of faulty self-regulation and/or a fluctuation of motivation and effort. Although the relative contribution of cognition, motivation, and effort to the performance of hyperactives cannot be separated in the absence of any theoretical guidelines at this time, motivation and effort can be maximized by some environmental manipulations. In previous studies where the experimental task was self-paced, the experimenter was present in the room, and continuous contingent feedback was available, the hyperactives tended to demonstrate their "hidden" skills -- their performance on attentional tasks was indistinguishable from that of normal controls. Hence, the present author expected relatively normative performance under similar environmental manipulations in the present study.

Further, the literature suggests that the amount of the drug tends to interact with the target behavior (Sprague et al., 1975). Drug efficacy also presumably depends upon the magnitude of the discrepancy between the competence and
performance of hyperactives on a given task (Douglas et al., 1988). In particular, within the self-regulation hypothesis (Douglas, 1983; Douglas et al., 1986, 1988), the initial level of performance can influence the effects of stimulants. The greater the gap between the performance and an inherent ability of the subjects, more pronounced drug effects are expected. Conversely, under optimal environmental circumstances enhancing subjects' motivation and effort, the effects of the drug should be limited. Thus, in the present study, improvement due to the drug was expected on tasks on which the organizing effects of the contingent feedback or self-pacing were not available, whereas deterioration was expected where these two enhancing factors were present. Presumably, such deterioration would be caused by "overstimulating" the central regulatory system. This would lead to an excessive self-regulation when flexibility of thinking is required (Dyme, Sahakian, Golinko, & Rabe, 1982; Robbins et al., 1979; Swanson & Kinsbourne, 1979).

Finally, the placebo preparation was expected to be therapeutically efficacious (Ross et al., 1982; Varley, 1983; Werry et al., 1987). The effects of pharmacologically inert substances were found to be particularly
powerful in inpatient settings where expectations of improvement tend to be amplified by the supportive therapeutic milieu (Shapiro & Morris, 1978). The present author expected that even if any response to placebo was found, this finding would have limited implications for the etiology of these disorders. In particular, the relative importance of psychological versus biological factors in the development of ADHD/CD cannot be evaluated on the basis of patients' response to the placebo preparation (Vogel, Goodwin, & Goodwin, 1980). Rather, any improvement in the placebo phase would indicate that the subjects possess significant cognitive resources that can be mobilized under appropriate experimental instructions in order to compensate for any apparent deficits. It is likely that such findings would have important consequences for the prognosis and management of ADHD/CD.
CHAPTER 2

Method

Subject Selection

Subjects were selected from residents remanded to the Inpatient Assessment Unit (I.A.U.) of the Juvenile Services to the Courts located in Burnaby, B.C. The I.A.U. is a closed custody unit where B.C. youths undergo psychological and psychiatric assessments while awaiting trial or sentencing. They are remanded to the Unit for approximately two weeks. The I.A.U. employs psychologists, psychiatrists, social workers, nurses, and health care workers who are involved in these court-ordered assessments.

Twenty-seven subjects entered the study but only 21 completed all aspects of it. Two subjects had to be taken off the moderate drug dose due to adverse side-effects. The additional two subjects missed the placebo phase and two other subjects missed the mild dose phase due to lack of
cooperation, transfer to another forensic facility, or assaultive behavior requiring a seclusion placement.

One hundred eighty six males were screened for the study in the period of 18 months. They constituted 49% of the total of 378 admissions to the I.A.U. during that time period. One hundred six boys met the diagnostic criteria of both ADHD and CD. Twenty-three possible candidates (22%) refused to enter the study and nine (8.5%) had clinical contraindications to Ritalin treatment (cardiovascular disease or substance abuse). The remaining forty seven (44%) were not included because they were either remanded for an insufficient period of time (and they would require a court-ordered re-remand in order to complete the study), transferred elsewhere due to overcrowding, or the forensic nature of their assessment ("raise" transfer or fitness issues) precluded them from entering the study.

Subject characteristics

All subjects were boys aged 13-17 years (\(M = 14.2, \ SD = 1.38\)). Twenty-four (89%) were Caucasian, two were Native Indians, and one was East Indian. Information about the
background characteristics of the boys was collected from the reports of psychologists, psychiatrists, social workers, probation officers, as well as from discharge reports from hospitals and residential treatment centres. All boys were of normal intelligence, although the Full Scale I.Q. scores of two boys were three points below the 80-point criterion which is considered the lower bound of normal intelligence (Wechsler, 1974). The assessing psychologists believed that the subjects' problems with sustained attention and effort resulted in the underestimation of their intellectual level. The results of the WISC-R (Wechsler, 1974) or Wechsler Adult Intelligence Scale-Revised (WAIS-R) (Wechsler, 1981) were available for 26 subjects. Table 1 presents means, standard deviations, and range for Verbal, Performance, and Full Scale I.Q. scores separately for the WISC-R and WAIS-R.
<table>
<thead>
<tr>
<th></th>
<th>Verbal I.Q.</th>
<th>Performance I.Q.</th>
<th>Full Scale I.Q.</th>
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<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
<td>WISC-R</td>
<td>96.0 (12.90)</td>
<td>102.52 (13.6)</td>
<td>99.0 (12.98)</td>
</tr>
<tr>
<td>WAIS-R</td>
<td>105.5 (10.70)</td>
<td>113.80 (8.98)</td>
<td>108.8 (9.42)</td>
</tr>
</tbody>
</table>

**NOTE:**
- WISC-R: N = 21, range of Full Scale I.Q. = 77 - 123
- WAIS-R: N = 5, range of Full Scale I.Q. = 94 - 117
Despite normal intellectual ability, only four boys (15%) did well at school. The remaining 23 subjects (85%) were underachieving academically (repeated grades, low marks, school suspension, or drop-out). Early childhood symptoms of both ADHD and CD were largely responsible for poor school performance in all boys.

Only four boys (14.8%) had an uninterrupted family involvement since childhood (three were raised by both parents and one by a single mother). The remaining 85% had a chaotic and disruptive family background. Youths tended to relocate between their natural or adoptive parents and multiple foster and group homes. This transiency resulted from abuse or neglect on the part of the caretakers and/or behavioral unmanageability of the child. Eleven boys (41%) were abused physically and seven (26%) sexually. Alcohol or drug abuse was identified in 22% of the primary caretakers of these boys.

Five subjects (18.5%) reported regular use of alcohol and eight (29.6%) reported use of illicit drugs on at least one occasion (marijuana, hashish, cocaine, heroin, L.S.D. or sniffing glue or gasoline). These self-reports were not
always validated by an independent data source and are likely to underrepresent the rate of substance abuse in this group. Two weeks prior to the admission to the I.A.U., two boys reported using alcohol on one or two occasions and two used marijuana or hashish once or twice.

Prior corrections history was available for 23 (85%) of the subjects. The subjects had a record of 1 to 20 offenses ($M = 6.48$, $SD = 5.33$). A first offense was committed by one boy as young as 12 years 1 month of age, whereas the oldest boy was 16 years 7 months at a time of first offence ($M = 13.5$, $SD = 1.21$). There were 3 categories of offenses: (1) against persons such as assault, intimidation, threat to cause bodily harm, sexual assault; (2) against property such as robbery, break and enter, theft, arson, possession of stolen property; and (3) "other" offenses which were neither against persons or property. The category of "other" offenses included impaired driving, breach of probation, escape from lawful custody, a failure to appear in court, or a failure to comply with disposition. Table 2 presents means, standard deviations, range, and number of subjects engaging in each type of offense.
<table>
<thead>
<tr>
<th>Offences against:</th>
<th>N(%)</th>
<th>M(SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Persons</td>
<td>10 (43%)</td>
<td>2.3 (1.42)a</td>
<td>1 - 5</td>
</tr>
<tr>
<td>Property</td>
<td>20 (87%)</td>
<td>4.2 (3.69)</td>
<td>1 - 14</td>
</tr>
<tr>
<td>Other</td>
<td>13 (48%)</td>
<td>2.54 (1.61)</td>
<td>1 - 5</td>
</tr>
</tbody>
</table>

**NOTE:** a- Include 6 sex offences committed by 3 subjects (range 1 -3)
Prior to their assessment at the I.A.U., fifteen subjects (65%) had been sentenced to probation and/or community service work whose sentence durations ranged from 3 months to 54 months 12 days ($M = 27.3$, $SD = 16.7$). Eleven subjects (48%) were remanded to or received detention sentences. The range of time served in custody was 7 to 402 days. Two subjects had unusually long sentences (194 and 402 days). These outlying statistics were therefore eliminated from the analysis. The remaining sample of $N = 9$ (33%) was previously sentenced to 7 to 28 days of institution time ($M = 17.22$ days, $SD = 6.72$).

**Diagnosis**

All participants were given diagnoses of both ADDH and CD determined by a DSM-3-based standardized interview (Reznick & Murphy, 1986) administered by an I.A.U. psychiatrist (see Appendix A for the interview format). Reliability was assessed for 143 (77%) of 186 interviewed males. The Kappa coefficients for three reliability raters for the ADDH diagnosis were .965 (98%), .959 (98%), and .908 (95%). Reliability ratings for CD were 1.0 (100%) for all three
raters. All subjects were free of organic brain involvement, other psychiatric diagnoses, and had no contraindications to methylphenidate treatment such as tics or cardiovascular disease. Subjects were not receiving any concurrent medication.

An attempt was made by a social worker to administer a similar standardized interview by telephone to a parent or guardian of the child who was a prospective candidate for the study. The interview information was available for only 18 subjects (67%). The relatively low success rate stemmed from the fact that these boys had multiple caretakers throughout their lives (sometimes as many as 15) who were not able to provide necessary historical background. All of the successfully completed interviews corroborated an early onset of both disorders in the probands. A perusal of medical and probation reports of boys whose guardians were not able to provide information about the history of the difficulty revealed that the remaining sample had displayed symptoms of ADDH and CD prior to entering school or shortly thereafter. Thus it is apparent that all of the subjects had experienced core symptoms of both disorders in early childhood.
Procedure

Adolescents and their parents or guardians were informed about the potential advantages and side-effects of Ritalin (see Appendix B for patient information) and signed consent forms to participate in a large ADD project (see Appendix C for a consent form). The present study was a part of this project.

Ritalin was prepared in gelatine capsules of .3 mg/kg and .5 mg/kg for body weights ranging from 50 to 85 kg. The active drug and placebo capsules were identical. Neither the experimenter, nursing staff, or the subjects were aware of the content of the capsules. The active drug or placebo was administered to the subjects randomly in 2-day phases interspersed with 1-day washouts 30 minutes before the morning and afternoon meals. The nursing staff ensured that capsules were swallowed.

Tests were administered by two research assistants on the second day of each phase, one hour after the second ingestion of the capsule. The total testing time did not
exceed 40 minutes. Thus, the testing was done during the peak period of presumed effects of Ritalin.

Side-effects were monitored by nursing staff who completed the Conners Side-Effect Questionnaire (Barkley, 1981) daily across all conditions (Appendix D). Side-effects were observed most frequently in the moderate dose condition. The symptoms experienced by patients included decreased appetite (31% on the drug versus 11.5% during the washout); stomach aches (15% on the drug versus 0% off the drug); anxiety and "talking less with others" (11.5% on the drug versus 0% off the drug for both symptoms). Fifteen percent of the sample was rated as severely irritable when on the moderate dose of the drug. However, the same percentage of the sample experienced this symptom also when on placebo and off the drug. Eight percent had headaches, drowsiness, and proneness to crying on the moderate dose comparing to 0%, 4%, and 4% respectively when off the drug.

The side-effects in the moderate dose resulted in a discontinuation from the study of only two subjects: one experienced decreased appetite, stomach aches, and drowsiness, whereas another was euphoric, energized, overactive,
and very much enjoyed this state. Incidentally, the latter subject admitted to abusing Ritalin in the past. The psychiatrist recommended against future treatment with higher doses of stimulants of these boys.

Upon completion of the study the subjects and guardians received feedback and a recommendation for treatment contingent on the drug response was included in the psychiatrists' reports to the courts.

**Experimental design**

The design was a 10-day, double-blind, cross-over trial of placebo and methylphenidate in a random order. The doses of methylphenidate were standardized in mg/kg of body weight (Sprague et al., 1975). Three-tenths (.3) of mg/kg was administered in the low dose condition and .5 mg/kg in the moderate dose condition. There were random 2-day drug phases interspersed with 1-day washouts. Such a design was possible due to a very rapid half-life of Ritalin (2 to 5 hours following the oral ingestion with peak behavioral effects occurring within 1 to 2 hours) (Barkley, 1981; Walker, 1982). Each subject was tested first in the
baseline, and then in the low dose, moderate dose, and placebo conditions that were administered in one of the six randomly determined orders.

The inclusion of a placebo phase was important from a methodological (Breuning & Ackles, 1985) as well as a therapeutic perspective (Ross et al., 1982). The value of the placebo preparation stems from the need to differentiate the effects of a chemical compound from a "nonspecific" effect of a "pill-taking ritual" (White, Tursky, & Schwartz, 1986). Moreover, researchers postulated that placebo may play a powerful therapeutic role in the management of hyperactivity (Ottenbacher et al., 1983; Ross et al., 1982; Varley, 1983; Werry et al., 1987; Zahn et al., 1980), particularly in an inpatient setting (Shapiro et al., 1978).

**Measures**

A battery of neuropsychological tests, presumably sensitive to the impairment of attention and self-regulation was administered to the subjects. Self-regulation is not a unitary process but comprises a multitude of cognitive skills, such as goal formulation, planning, choosing and
rejecting an alternative, persisting in successful behavior, shifting and inhibiting actions when they are no longer appropriate, utilizing environmental feedback, and self-monitoring (Lezak, 1982, 1983). Incidentally, some of the measures in this study are also sensitive to frontal lobe pathology, typically associated with disinhibition and dysregulation of behavior. In this study, an attempt was made to identify any functional deficits rather than localizing them to a specific area of the brain. The hypothesis of frontal lobe involvement in hyperactive behavior and of the effects of stimulants on the frontal lobe activity has been entertained by researchers in the past and is viable in the present study. Nevertheless, the localization of the function is beyond the scope of this study as it would require validation from other independent data sources (Johnston, 1986).

In order to enhance the subjects' motivation and effort in the present study, the experimenter was present throughout the evaluation, and on some tasks self-pacing and continuous feedback were available. An additional motivating manipulation might have resulted from the fact that the subjects were informed that treatment recommendations based
on the study would be made to the court. Informal observations of the young offenders at the pre-trial stage suggest that they might prefer treatment recommendations in the hopes that they might attenuate the severity of the court disposition. Overall, this author expected that the motivation and effort exerted by the subjects would be optimal due to the above factors and that it would lead to more normative performance in this group than has been typically found in studies on hyperactivity.


The Wisconsin Card Sorting Test (WCST) is a measure of the ability to derive abstract principles and a tendency to perseverate (Heaton, 1981; Lezak, 1982, 1983). Important-ly, it is a self-paced task where continuous contingent feedback is provided. The subject is given two decks of 64 cards in each deck. There are 1 to 4 symbols -- a triangle, star, cross, or circle in red, green, yellow, or blue -- printed on each card. The task is to place each card under one of four stimulus cards: one red triangle, two green stars, three yellow crosses, and four blue circles, according to a principle deduced from the feedback provided by the
examiner. There are three possible sorting principles: Color, Form, and Number. For example, when the principle is Color, the subject should place a card with a red sign on it under the stimulus card "one red triangle" regardless of the form and number of elements on the card. The examiner tells the subject whether or not his response is correct after each card is placed. After ten consecutive correct responses the examiner shifts to another sorting principle by changing the pattern of "right" and "wrong" statements. At no point during the testing does the examiner make it explicit that a shift in the sorting principle is to be expected. The subject must deduce it from the pattern of feedback.

Several scores can be derived from this test. First, Categories Achieved and Conceptual Level Responses (Insight) are two measures of the patients' ability to discover the sorting principles which presumably reflects abstract concept formation ability and learning from the environmental feedback. Second, a measure of Perseverative Errors refers to mental flexibility and the ability to shift a conceptual set. Perseverations occur when the subject continues to respond to an initially guessed incorrect
principle or to the previously successful principle despite
the feedback that it is no longer appropriate. Finally,
*Failures to Maintain Set* is a measure of the ability to
sustain correct behavior in order to successfully complete
the discovered category and the ability to inhibit incorrect
responding. Studies have shown that the WCST is sensitive
to the faulty integrative functions of the brain commonly
associated with the frontal area. Consistently, neurologi-
cal patients with frontal damage tend to perform signifi-
cantly worse than normal controls on this test (Bornstein,
1986; Drewe, 1974; Kolb et al., 1985; Lezak, 1982, 1983;
Malmo, 1974; Milner et al., 1984; Nelson, 1976;
Pendelton, Heaton, & Lehman, 1982; Robinson, Heaton,
Lehman, & Stilson, 1980; Wedding, Horton, & Webster, 1986).

Only one study used the WCST to evaluate its sensitivi-
ty to cerebral dysfunction in adolescence (Chelune & Thomp-
son, 1987). The authors compared a group of 62 heteroge-
neous cerebral patients (neurologic disease/trauma, seizure
disorder, learning disability) aged between 10 and 15.75
years (M = 13.4) to a normal quasi-control group of 42
children from the senior author’s previous study (Chelune et
al., 1986a). The control group was younger than patients
(range 10-12.8, $M = 11.13$) thus providing a conservative basis for a comparison. Patients performed significantly worse than controls on measures of Categories Achieved, Perseverative Responses, and Perseverative Errors ($p < .005$ for all measures). These results are suggestive of the sensitivity of the WCST to cerebral dysfunction in adolescence.

Chelune et al. (1986a) administered the WCST to a group of 24 hyperkinetic and 24 normal children between the ages of 6 and 12. The groups were matched for I.Q. measured by the Peabody Picture Vocabulary Test ($M = 96.58$, $SD = 12.86$ in the hyperactive sample; $M = 100.33$, $SD = 12.57$ in the normal sample). Hyperactives were impaired on Categories Achieved, Perseverative Errors, and Failures to Maintain Set ($p < .025$, .01, and .05 respectively). The authors concluded that the WCST was diagnostic of a problem-solving style characterized by poor cognitive flexibility, disinhibition, dysregulation of goal-directed activity, and decreased responsivity to feedback in hyperactives.
The Word Fluency Test (WFT) evaluates the subjects' ability to generate words starting with the letters "F", "A", and "S" within the time limit of one minute per letter. The score of Total Number of Words is useful in assessing the capacity to initiate, maintain, and stop an intended activity in favor of an instructional set. It requires mental flexibility, the capacity to suppress habitual behavior, and adaptation to the changing environmental demands, thus it is sensitive to problems with self-regulation (Lezak, 1982, 1983; Perret, 1974). Research evidence is suggestive that this test is sensitive to integrative pathology of the brain, particularly associated with the frontal region (Benton, 1968; Bornstein, 1986; Crockett et al., 1986; Lezak, 1983; Milner, 1964; Milner et al., 1984; Pendelton et al., 1982).

Gaddes et al., (1973) provided norms for the WFT for Western Canadian normal children of both sexes between the ages of 6 and 13. Children as young as 11-13 tended to reach the level of performance indistinguishable from that
of normal adults. No information about I.Q. levels of these subjects was obtained in the study, however, the children's academic performance was average (the majority maintained C marks in the basic academic subjects; a few obtained B- and B marks in a minority of subjects). Therefore, these norms seem appropriate for individuals of an average intellectual level.

**Stroop Color and Word Test (Golden, 1978)**

The 100-item version of the Stroop Color and Word Test (SCWT) is a measure of the ability to shift cognitive set according to the demands of the task as well as to maintain a desired course of action by suppressing interference from other competing irrelevant data sources (Comalli, Wapner, & Werner, 1962; Golden, 1976; Lezak, 1983). The test consists of three tasks: word reading, color naming, and suppressing word reading in favor of color naming within the time limit of 45 seconds per task. The critical ability involved in this task is the inhibition of a highly overlearned and automatized activity (i.e., reading words depicting colors) in favor of naming a perceptual property (i.e., color of the ink in which the word is printed). For
example, the word "red" can be printed in blue or green and the subject is required to attend to the color of the ink while ignoring to read the word. A pure Interference Score is computed according to the manual by subtracting a raw score from a predicted interference score based on age-corrected reading and color naming scores.

**Trail Making Test - B (Reitan, 1979)**

The Trail Making Test-B (TMT-B) is a measure of visual scanning, conceptual flexibility, and rapid visual-motor processing of information (Lezak, 1983). Importantly for the self-regulation hypothesis, this test appears to be one of the better measures of the integrative functions of the brain as it places demands on the abilities to plan, choose between alternatives, and subordinate a series of actions to a desired goal (Lezak, 1983). The task is to connect as quickly as possible consecutive numbers and letters, alternating between both sequences. The examiner points out any errors to the subject and encourages him to look for the correct solution while the timing of the performance continues uninterrupted. The scores are based on Total Time. The child version is administered to subjects up to 14 years,
whereas a slightly longer version is used with children 15 years and older. One study that used this test with 27 hyperactive boys aged 13 years, reported that they performed worse (but not significantly so using the Bonferroni criterion of $p < .005$) than 62 matched normal controls. The authors reported the WISC-R Performance I.Q. scores of $M = 105.8$ ($SD = 16.4$) in the ADD group and $M = 107.9$ ($SD = 12.9$) in the control group.

**Digit Span of the WISC-R (Weschsler, 1974)**

Digit Span is comprised of two different tests, *Digits Forward* and *Digits Backward*, which involve different attentional abilities. Both tests consist of seven sets of increasingly longer strings of random number sequences that are read to the subject at the rate of one digit per second. Subjects are required to repeat the numbers either in the order presented to them or backward. *Digits Forward* measures auditory attention of a passive quality, whereas *Digits Backward* is an effortful activity in which concentration, memory, and reversing operations are required (Lezak, 1983; Wechsler, 1974).
Digit Symbol of the WISC-R (Wechsler, 1974)

Digit Symbol is a time-limited symbol-substitution task in which the subject is asked to fill in blank spaces with the symbol that is paired to the number printed above the blank space. This task is sensitive to right frontal pathology (Lezak, 1983). Motor persistence, sustained attention, response speed, and visual-motor coordination play important roles in this test.

Moderate improvement was observed on tests of short-attention span and concentration such as Digit Span and Digit Symbol in studies of stimulant efficacy (Barkley, 1976; Kavale, 1982). However, the issue whether the abilities measured by these tests are relevant to the basic dysfunction in ADD is debated. For example, Lambert et al. (1987) compared 59 hyperactive and 58 normal control children 14 years of age on the WISC-R Digit Span and a double-length version of Digit Symbol. The authors did not find significant differences between the samples on these measures. These findings might be consistent with previously reviewed studies which suggest that short-term attention span may not be impaired in hyperactives (see Douglas, 1983,
for a review). Additionally, the subjects in Lambert et al.'s (1987) study were closely supervised which may have normalized their performance.

Test-Retest Reliability of the Proposed Battery

Test-retest reliabilities of the Stroop Color-Word Test covering periods from one minute to ten days for word reading, color naming, and color/word reading ranged from .71-.88 (Golden, 1975; Jensen, 1965). Lezak (1983) reported a test-retest reliability coefficient of .67 for three consecutive administrations of the Trail Making Test-B. Although this coefficient was moderately high, the cumulative practice effects expressed in total time changes were not significant. Similarly, Bornstein, Baker, and Douglas (1987) obtained a three-week-retest reliability coefficient of .70 for a sample of 23 normal adults on the TMT-B. The change in performance time over two trials was small (9%) and nonsignificant. Wechsler (1974) reported reliability coefficients for Digit Span and Digit Symbol based on N = 104 subjects aged between 14.5-15.5 years. The Digit Span subtests' first administration (M = 9.8, SD = 3.3) and second administration (M = 10.3, SD = 3.1) correlated at r =
.77 when correction was made for the variability within the sample. The Digit Symbol subtest yielded a corrected $r = .71$ ($N = 10.3$, $SD = 3.2$ in the first administration; $M = 11.2$, $SD = 3.6$ in the second administration). The stability coefficients for the remaining tests of the proposed battery are not available.

In order to control for the possibility of practice effects which are likely to arise from a multiple administration of the proposed battery, the present author attempted to collect data for a no-treatment control group of ADHD/CD individuals. This attempt proved unsuccessful due to the lack of subjects. It was hypothesized, however, that any possible practice effects should not influence the conclusions regarding the effects of mild and moderate drug doses relative to placebo due to the counter-balanced nature of the design.
CHAPTER 3

Results

Univariate analyses of variance (ANOVA) performed on a repeated battery of 11 tests were highly significant for 9 of the dependent measures \((p - \text{values ranged from .0076 to .0000})\). The means of the remaining nonsignificant dependent measures (WCST-Failures to Maintain Set and Digits Backward) were relatively high and yielded little variability across experimental phases. This finding probably reflects ceiling effects. Table 3 presents univariate main effects of drug phases expressed in \(F\) and \(p - \text{values, whereas Appendix E presents sources of variance for these effects. Table 4 presents means and standard deviations obtained in the baseline, placebo, mild dose, and moderate dose conditions.}

Univariate 6(drug order) x 4(experimental phase) analyses of variance revealed significant interactions for three dependent variables: Stroop Interference, Trail Making Test-B (adult version), and Digits Forward. The results of other dependent variables were not modified by
TABLE 3

UNIVARIATE MAIN EFFECTS OF EXPERIMENTAL CONDITIONS

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>df</th>
<th>F-value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
<td>3,60</td>
<td>11.77</td>
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</tr>
<tr>
<td>WCST-Perseverative Errors</td>
<td>3,60</td>
<td>12.84</td>
<td>.0000</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>3,60</td>
<td>17.18</td>
<td>.0000</td>
</tr>
<tr>
<td>WCST-Failures to Maintain Set</td>
<td>3,60</td>
<td>.41</td>
<td>.7443</td>
</tr>
<tr>
<td>WFT-Total Words</td>
<td>3,60</td>
<td>11.00</td>
<td>.0000</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>3,57</td>
<td>5.21</td>
<td>.0003</td>
</tr>
<tr>
<td>TMT-B-Child-(sec)</td>
<td>3,33</td>
<td>4.79</td>
<td>.0076</td>
</tr>
<tr>
<td>TMT-B-Adult-(sec)</td>
<td>3,21</td>
<td>7.16</td>
<td>.0009</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>3,60</td>
<td>5.22</td>
<td>.0029</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>3,60</td>
<td>.21</td>
<td>.8876</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>3,60</td>
<td>10.70</td>
<td>.0000</td>
</tr>
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TABLE 4
MEANS AND STANDARD DEVIATIONS OF DEPENDENT VARIABLES

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Experimental Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td>5.82</td>
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<td>WCST-Perseverative Errors</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>19.00</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>56.30</td>
</tr>
<tr>
<td>WCST-Failure to Maintain Set</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>1.52</td>
</tr>
<tr>
<td>WFT-Words Total</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>25.37</td>
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<tr>
<td>Stroop Interference</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>56.90</td>
</tr>
<tr>
<td>Child TMT-B -</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>36.67</td>
</tr>
<tr>
<td>Adult TMT-B</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>92.75</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>7.22</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>5.96</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>M</td>
</tr>
<tr>
<td></td>
<td>57.57</td>
</tr>
</tbody>
</table>

NOTE. Frequency of cases per condition is 23-27.
WCST - Wisconsin Card Sorting Test. WFT - Word Fluency Test.
TMT - Trail Making Test.
the drug order factor. Table 5 presents F and p-values for 2-way drug order x drug phase interactions. Appendix F presents sources of variance for these interactions. Main effects of the drug order factor and sources of variance are presented in Appendix G.

Further, 2-way 2(examiner) x 4(drug phase) ANOVAs revealed a significant interaction for only one nonsignificant variable. Table 6 presents F and p-values for these analyses. Appendix H presents sources of variance for these interactions, whereas Appendix I presents main effects and sources of variance for the experimenter factor.

Finally, a possibility of carry-over effects due to multiple test administrations was investigated. The fact that each subject served in all of the treatment phases created a possibility that his performance in any of the conditions was partially dependent on the previous participation in other conditions. In order to control for this potential artifact, the drug order was completely counterbalanced (i.e., all of the six possible drug sequences were represented) and administered randomly. In addition, the counterbalancing effect was analyzed statistically. A
### TABLE 5

<table>
<thead>
<tr>
<th>Variable</th>
<th>df</th>
<th>F-Value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
<td>15,45</td>
<td>1.15</td>
<td>.341</td>
</tr>
<tr>
<td>WCST-Perseverative Errors</td>
<td>15,45</td>
<td>.71</td>
<td>.760</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>15,45</td>
<td>.66</td>
<td>.809</td>
</tr>
<tr>
<td>WCST-Failure to Maintain Set</td>
<td>15,45</td>
<td>1.00</td>
<td>.4691</td>
</tr>
<tr>
<td>WFT-Total Words</td>
<td>15,45</td>
<td>1.39</td>
<td>.951</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>15,45</td>
<td>2.00</td>
<td>.039</td>
</tr>
<tr>
<td>Child TMT-B Child (sec)</td>
<td>12,21</td>
<td>.43</td>
<td>.934</td>
</tr>
<tr>
<td>Adult TMT-B (sec)</td>
<td>12,9</td>
<td>3.20</td>
<td>.045</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>15,45</td>
<td>2.02</td>
<td>.035</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>15,45</td>
<td>.78</td>
<td>.697</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>15,45</td>
<td>1.33</td>
<td>.224</td>
</tr>
</tbody>
</table>

**NOTE.**  
WCST - Wisconsin Card Sorting Test.  
WFT - Word Fluency Test.  TMT - Trail Making Test.
### TABLE 6

**UNIVARIATE EXPERIMENTER X EXPERIMENTAL CONDITION INTERACTIONS**

<table>
<thead>
<tr>
<th>Variable</th>
<th>df</th>
<th>F-Value</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
<td>3,57</td>
<td>2.39</td>
<td>.4361</td>
</tr>
<tr>
<td>WCST-Perservative Errors</td>
<td>3,57</td>
<td>1.30</td>
<td>.3722</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>3,57</td>
<td>.23</td>
<td>.8528</td>
</tr>
<tr>
<td>WCST-Failure to Maintain Set</td>
<td>3,57</td>
<td>3.20</td>
<td>.0299</td>
</tr>
<tr>
<td>WFT-Total Words</td>
<td>3,57</td>
<td>.15</td>
<td>.8991</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>3,54</td>
<td>.18</td>
<td>.8881</td>
</tr>
<tr>
<td>TMT-B Child-Total Time</td>
<td>3,30</td>
<td>.84</td>
<td>.4810</td>
</tr>
<tr>
<td>TMT-B Adult-Total Time</td>
<td>3,18</td>
<td>.31</td>
<td>.8710</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>3,57</td>
<td>.49</td>
<td>.7288</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>3,57</td>
<td>.81</td>
<td>.4093</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>3,57</td>
<td>.28</td>
<td>.8750</td>
</tr>
</tbody>
</table>

**NOTE.** WCST - Wisconsin Card Sorting Test.  
WFT - Word Fluency Test.  
TMT - Trail Making Test.
3(position of the drug phase in the administration sequence) x 2(baseline versus one drug phase) 2-way analysis of variance was performed for each of the six significant dependent measures not modified by the experimenter or drug order factors. The consecutive time of administration of the treatment phases served as a between-group factor, whereas one drug phase versus the baseline phase served as a within-group factor. For example, in order to examine the effects of repeated testing on performance in the moderate dose phase, the performance of three subgroups of subjects (i.e., those who received the dose either immediately after the baseline, after one other, or two other drug administrations) was evaluated against their respective baseline level. Similar procedures were employed in the mild dose and placebo phases, with the baseline serving as a control.

The analyses of variance revealed no significant interactions between the position of the drug in the administration sequence and drug phase factors. Stated differently, the differences between the baseline and either the placebo, mild, or moderate drug dose phases were not modified by the number of the preceding test administrations. Appendices J, K, and L present \( F \) and \( p \) values for the
position in the sequence x drug dosage interactions separately for the placebo, mild, and moderate phases. Interestingly, the main effects of the drug position in the administration sequence were also not significant in the placebo, mild, and moderate drug phases. This suggests that there were no appreciable practice effects between the second, third, and fourth administrations of the tests in either of the drug dose phases. Appendices M, N, and O present main effects expressed in $F$ and $p$ values for the administration sequences in the placebo, mild, and moderate dose.

Each of the six dependent variables which yielded overall significance were analyzed in more detail. The remaining five dependent variables were dropped from further analyses (two were nonsignificant and three were modified by the drug order factor), as they were uninterpretable. A stepwise Bonferroni procedure was used in order to control for a familywise type I error. A .05 criterion was adopted per family of all pairwise comparisons for each dependent variable. Five of the six dependent variables yielded significant differences between the baseline and the placebo, mild, and moderate drug phases respectively. All of the
changes were consistent with improvement on any of the inactive and active drug phases relative to the baseline. The sixth variable (the child version of the Trail Making Test-B) yielded significant contrasts between the baseline and the mild and moderate drug phases respectively. Only one contrast (WFT - Total Number of Words) yielded a significant difference between the placebo and mild drug dose phases. Table 7 presents the discussed contrasts.

Several of the examined contrasts yielded marginally significant differences between placebo and active drug phases. The mild dose phase resulted in improvement on the child version of the TMT-B Total Time \((p < .10)\). In contrast, there was a tendency towards a deterioration on the WCST-Insight variable on the mild dose \((p < .06)\). The moderate dose of Ritalin enhanced performance on the WFT - Total Words \((p < .05)\), but it led to a marginally significant deterioration on the WCST-Categories Achieved \((p < .06)\) and WCST-Perseverative Errors \((p < .08)\). The comparisons of two doses of the active drug revealed a marginally significant deterioration on the moderate drug dose relative to the mild dose on the WCST-Categories Achieved \((p < .09)\). Table 7
<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>Placebo vs. Baseline</th>
<th>.3mg/kg vs. Baseline</th>
<th>.5mg/kg vs. Baseline</th>
<th>Placebo vs. .3mg/kg</th>
<th>Placebo vs. .5mg/kg</th>
<th>.3mg/kg vs. .5mg/kg</th>
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</thead>
<tbody>
<tr>
<td>WCST - Categories Achieved</td>
<td>.0003*</td>
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<td>.0001*</td>
<td>.2431</td>
<td>.0634</td>
<td>.0900</td>
</tr>
<tr>
<td>WCST - Perseverative Errors</td>
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<td>.0037*</td>
<td>.0001*</td>
<td>.3198</td>
<td>.0795</td>
<td>.7686</td>
</tr>
<tr>
<td>WCST - % Insight</td>
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<td>.0000*</td>
<td>.0001*</td>
<td>.0643</td>
<td>.1225</td>
<td>.4715</td>
</tr>
<tr>
<td>WFT - Total Words</td>
<td>.0030*</td>
<td>.0000*</td>
<td>.0001*</td>
<td>.0116</td>
<td>.0534</td>
<td>.6806</td>
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<tr>
<td>Child TMT-B (sec)</td>
<td>.0753</td>
<td>.0002*</td>
<td>.0102</td>
<td>.1018</td>
<td>.3825</td>
<td>.9716</td>
</tr>
<tr>
<td>Digit Symbol</td>
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<td>.0005*</td>
<td>.0000*</td>
<td>.8268</td>
<td>.1495</td>
<td>.2352</td>
</tr>
</tbody>
</table>

**Note.** * Contrasts significant at the Bonferroni p < .008.

presented $p$-values for the marginally significant contrasts as well.

The magnitude of change was determined for all contrasts significant at $p < .10$ by computing Effect Sizes (ES) based on difference scores between placebo and either of the two doses of the active drug and between the baseline and placebo. All ES-values were computed using custom-made denominators based on standard deviations of the difference scores. ES-values for all significant contrasts ranged from .64 to 1.37 standard units. The magnitude of effects approaching significance ranged from -.43 to -1.01 when a deterioration was present, whereas they ranged from .45 to .47 in the case of improvement. Table 8 presents ES-values for the significant and marginally significant contrasts. ES-values of .50 are considered moderate, whereas ES-values of .80 are large (Howell, 1986).

The baseline performance of the subjects on the majority of the tests was compared to the performance of normal children in other studies on hyperactivity using the Satterthwaite-$t$ formula. The subjects in the present study were nondistinguishable from quasi-control normals on all
but one variable. On the WCST—Failure to Maintain Set, quasi-controls performed significantly better than this sample \((p < .005)\). Table 9 presents means, standard deviations, group sizes, and relevant demographic characteristics of quasi-controls.
TABLE 8

EFFECT-SIZE VALUES FOR CONTRASTS SIGNIFICANT AT P < .10

<table>
<thead>
<tr>
<th>Variable</th>
<th>Placebo vs. Baseline</th>
<th>.3mg/kg vs. Baseline</th>
<th>.5mg/kg vs. Baseline</th>
<th>Placebo vs. .3mg/kg</th>
<th>Placebo vs. .5mg/kg</th>
<th>.3mg/kg vs. .5mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST - NUMBER OF CATEGORIES</td>
<td>.90*</td>
<td>.88*</td>
<td>.90*</td>
<td>ns</td>
<td>-.43</td>
<td>-.62</td>
</tr>
<tr>
<td>WCST - NUMBER OF PERSEVERATIVE ERRORS</td>
<td>1.17*</td>
<td>.64*</td>
<td>.93*</td>
<td>ns</td>
<td>-.40</td>
<td>ns</td>
</tr>
<tr>
<td>WCST - INSIGHT (%)</td>
<td>1.27*</td>
<td>1.00*</td>
<td>.94*</td>
<td>-1.01</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>WFT - TOTAL NUMBER OF WORDS</td>
<td>.69*</td>
<td>1.26*</td>
<td>.92*</td>
<td>.57</td>
<td>.45</td>
<td>ns</td>
</tr>
<tr>
<td>CHILD TMT-B (SEC)</td>
<td>.52</td>
<td>1.19*</td>
<td>.80</td>
<td>.47</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>DIGIT SYMBOL</td>
<td>.77*</td>
<td>.81*</td>
<td>1.37*</td>
<td>ns</td>
<td>ns</td>
<td>ns</td>
</tr>
</tbody>
</table>

NOTE. ns - Contrasts were not significant at p ≤ .10 (probabilities provided in Table 7)

* Contrasts significant at Bonferroni p < .008; ES = .50 (moderate) ES = .80 (large).

Positive ES values indicate improvement of the first condition relative to the second, whereas negative values indicate deterioration.
TABLE 9

BASELINE PERFORMANCE COMPARED TO RESULTS OF NORMAL SAMPLES FROM OTHER STUDIES

<table>
<thead>
<tr>
<th>Dependent Variables</th>
<th>Baseline</th>
<th>Quasi- Controls</th>
<th>df</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST - CATEGORIES ACHIEVED</td>
<td>M 5.82</td>
<td>5.70&lt;sup&gt;a&lt;/sup&gt;</td>
<td>28</td>
<td>.29</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD (2.57)</td>
<td>(.95)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PERSEVERATIVE ERRORS</td>
<td>M 19.00</td>
<td>12.30&lt;sup&gt;a&lt;/sup&gt;</td>
<td>21</td>
<td>1.17</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD (9.05)</td>
<td>(16.94)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FAILURES TO MAINTAIN SET</td>
<td>M 1.52</td>
<td>.70&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24</td>
<td>2.83</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>SD (.93)</td>
<td>(.68)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WORD FLUENCY (TOTAL WORDS)</td>
<td>M 25.37</td>
<td>28.80&lt;sup&gt;b&lt;/sup&gt;</td>
<td>18</td>
<td>1.26</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD (6.04)</td>
<td>(8.3)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STOOP INTERFERENCE (T-SCORE)</td>
<td>M 56.73</td>
<td>50&lt;sup&gt;c&lt;/sup&gt;</td>
<td>ns</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>SD (6.28)</td>
<td>(10)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHILD TMT-B (SEC)</td>
<td>M 36.67</td>
<td>38.9&lt;sup&gt;d&lt;/sup&gt;</td>
<td>36</td>
<td>.63</td>
<td>ns</td>
</tr>
<tr>
<td></td>
<td>SD (13.76)</td>
<td>(14.5)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE:  

- t and df values based on the Satterthwaite- t formula
- a - Chelune et al. (1986): N=10, age = 12.25, males and females; I.Q. = 111.90 (15.7) on the Peabody Picture Vocabulary Test
- b - Gaddes et al. (1973): N=12, age = 13, boys; average school achievement
- c - Golden (1978): age-corrected T-Scores. Scores must differ by at least 10 points in order for the difference to be significant.
- d - McGee et al. (1989): N=62, age = 13, boys; Performance I.Q. = 107.9 (12.9) on the WISC-R.
The objective of this study was to assess the acute effects of mild and moderate doses of Ritalin relative to placebo on the "executive" control processes of hyperactive, conduct disordered young offenders. Within the proposed self-regulation hypothesis (Douglas, 1983; Douglas et al., 1986, 1988), the fundamental deficit in hyperactivity rests on a transient disruption in both the facilitation of goal-directed activity and in the inhibition of goal-irrelevant responding, rather than on a pervasive structural deficit. Consistent with this view, some authors proposed that the causes of the often impaired performance of hyperactives should not be sought in a child himself, but rather in a child x situation interaction over time (Henker et al., 1989). Stated differently, the proposed deficit rests on the less-than-optimal performance rather than a lack of competence per se. Accordingly, hyperactives are expected to possess "hidden" knowledge which can be
mobilized by appropriate environmental or therapeutic manipulations.

Within this theory, the role of stimulants is limited to the reduction of a performance-competence discrepancy by activating the central self-regulatory mechanism often associated with the frontal lobes (Lezak, 1983). The magnitude of the drug effect should be contingent on subjects' initial output. In the present study, significant facilitation effects were expected only if the subjects demonstrated an initial subnormal level of performance relative to their actual ability. Conversely, limited positive effects of the drug were expected in the case when other nonpharmacological manipulations have already facilitated patients' "executive" control. In particular, it was hypothesized that the presence of the experimenter, contingent feedback, and self-pacing on some tasks might yield relatively good performance even prior to the administration of the drug. A possibility of a deterioration was expected upon the introduction of the active drug, when the initial performance on self-paced and contingently reinforced tasks was relatively good. Such deterioration would be presumably
caused by "overstimulating" the subjects and inducing excessive self-control.

In order to interpret the results of this study, a distinction between drug response and drug effect is in order (White et al., 1986). Drug response refers to the drug-baseline performance differences. It has been recognized in the psychopharmacology literature that the major component of the drug response is not only the action of the drug itself, but also non-specific factors (placebo), repeated testing, and any uncontrolled fluctuation in the disorder (Ross et al., 1986). Conversely, drug effect refers to any change above the placebo level. In the present study, the baseline phase served as a control for drug response, whereas the placebo phase was a control for drug effect. Further, baseline was a control for placebo response. In order to determine the placebo effect, a no-treatment control group was necessary. An attempt to collect data for such a group has failed in this study due to the lack of subjects. The placebo effect then cannot be evaluated in the present study.
The reviewed results demonstrated highly significant active drug and placebo response using baseline as a control for 9 out of 11 dependent measures. Additional 2-way analyses of variance revealed significant order by phase interactions for three significant variables (Stroop Interference, the adult version of the Trail Making Test-B, and Digits Forward). These variables were dropped from further analyses as they rendered uninterpretable results. As expected, the effects of the position of the drug phase in the administration sequence were counterbalanced, and there were no appreciable practice effects between the second, third, and fourth administration of either of the active drug doses or placebo.

Pairwise comparisons between the baseline and inactive and active drug phases controlled by a stepwise Bonferroni procedure revealed that the subjects significantly improved on the placebo and the active drug relative to the baseline on self-paced and contingently reinforced tasks which measured the ability to deduce an abstract principle utilizing the environmental feedback. In particular, they improved on the Wisconsin Card Sorting Test - Categories Achieved and Conceptual Level Responses (Insight). Similar
improvement was also observed on the measure of the ability to shift mental set according to external demands: the Wisconsin Card Sorting Test - Perseverative Errors. The magnitude of improvement on these measures was moderate to large (Howell, 1986) and ranged from .64 to 1.27 of the standard score.

Consistent with the above mentioned results, improvement was also observed on measures requiring speed of information processing. The subjects' performance was enhanced in the placebo and active drug phases relative to the baseline on timed measures of mental flexibility (the Word Fluency Test) and short-term attention span (Digit Symbol). The Effect Size-values for these measures were moderate to large and ranged from .69 to 1.37 of the standard score. The child version of the Trail Making Test-B, a timed measure of flexibility and rapid conceptual tracking, resulted only in the mild and moderate dose response. The size of the changes was large (ES=1.19 and .80 respectively).

As stated previously, drug response (as opposed to drug effect) tends to result from several sources of variance:
the drug itself, nonspecific factors associated with taking the pill (placebo), repeated testing, and spontaneous fluctuation in patients’ symptomatology. In order to determine the drug effect, these confounding factors had to be ruled out by introducing appropriate controls.

Drug effects of the mild and moderate doses were evaluated against placebo. The relative efficacy of mild and moderate levels of the drug were also compared. Only one significant contrast was obtained: the WFT—Total Number of Words improved on the mild dose relative to the placebo ($p < .01$). This warrants a conclusion that neither mild nor moderate doses yielded significant drug effect for all but one measure, despite their pronounced drug responses.

Nevertheless, some of the investigated contrasts yielded marginally significant drug effects. There was a trend towards improvement on the Word Fluency Test in the moderate drug phase relative to placebo ($p < .05$, $ES = .47$). Similarly, performance on the child version of the Trail Making Test-B improved on the mild dose of Ritalin ($p < .10$, $ES = .47$). The magnitude of the above changes was moderate.
The remaining marginally significant trends indicated **deterioration** on the active drug relative to placebo. Interestingly, the deterioration in performance was observed on three measures derived from the Wisconsin Card Sorting Test. The level of **Insight** deteriorated in the mild drug phase (p < .06, ES = -1.01). **Categories Achieved** deteriorated on moderate drug dose when compared to both placebo (p < .06, ES = -0.43) and mild dose (p < .09, ES = -.62). The number of **Perseverative Errors** has also increased in the moderate dose phase (p < .08, ES = .40). It appears that these three conceptually and statistically related measures (the correlations for the **Categories Achieved** with **Perseverative Errors** and **Insight** were -.64 and -.74 respectively) tended to show marginally significant, moderate deterioration on either dosage of the drug.

In conclusion, it appears that the Word Fluency Test (WFT) and the Wisconsin Card Sorting Test (WCST) yielded marginally significant drug **effects** in the opposite directions. This suggests that the examination of the task parameters involved in these tasks might be worth further research. The WFT performance marginally improved on both mild and moderate doses, whereas the various measures of the
WCST tended to marginally deteriorate on both doses. Both tests presumably measure mental flexibility and adaptation to external demands. They both involve processes of initiating, maintaining, modifying, and inhibiting actions in accordance with the task demands. The major difference between these tests rests on the fact that the WCST is more complex, self-paced, and contingently reinforced. Additionally, the WCST is considerably longer and involves learning of new abstract concepts. In contrast, the WFT is timed, nonreinforced, and utilizes previous knowledge of high frequency words. It appears that both mild and moderate doses of Ritalin tended to facilitate the simpler timed task, whereas they tended to impair the more complex, self-paced, and contingently reinforced tasks. Importantly, self-pacing and contingent feedback have been identified as factors enhancing information processing in hyperactives (see Douglas, 1983, for a review). It could be speculated that in the present study, these factors have probably facilitated optimal performance on the WCST prior to the introduction of the active drug. Consequently, the subsequent introduction of the active drug has probably caused "overdosing" and excessive self-regulation in the subjects. This hypothesis requires, however, further investigation.
The effect of the placebo preparation cannot be unequivocally evaluated in the absence of a no-treatment group which would control for both the spontaneous fluctuation in the patients' symptoms and repeated testing. Attempts to collect such data proved futile due to the lack of subjects.

There is sparse data base relevant to dose-response drug studies of hyperactive adolescents which would provide an evaluative context for the present study. Consistent with the present study, the previously discussed controlled studies of adolescents (Brown et al., 1988; Coons et al., 1987; Varley, 1983) provided only weak support for short-term efficacy of Ritalin. Similarly, a controlled dose-response study of children (Douglas et al., 1988) yielded marginally significant results on a task developed by Petrides and Milner (1982, cited in Douglas et al., 1988) to assess planning and self-monitoring skills in patients with frontal lobe dysfunction. It could be speculated that this test was conceptually relevant to the self-regulation hypothesis proposed in this study and it appears consistent with the marginal trends discovered by the present author.
Theoretical Implications

The results of the present study appear to support the self-regulation hypothesis of Douglas and her colleagues (Douglas, 1983; Douglas et al., 1986, 1988). Briefly, these authors proposed that hyperactives do not demonstrate a stable cognitive deficit. Rather, they fail to make use of their inherently intact skills due to faulty self-control. Accordingly, environmental manipulations or stimulant drug action should have similar facilitatory effects on these processes. In particular, the presumed role of the drug is to reduce the gap between the inherently possessed ability and the performance deficiency. Consequently, the drug effects should be contingent on the magnitude of such competence-performance discrepancy. In the present study, the highly significant placebo and drug responses are suggestive of the fact that the subjects' initial performance was less than optimal, hence the improvement in the experimental phases. However, the drug effect was only marginally significant for some of the variables. This is likely due to the fact that the subjects might have
approached their optimal level of performance during the
placebo phase against which the drug effects were evaluated.

The directions of the positive and negative marginally
significant trends provide weak support for the self-regula-
tion hypothesis. While performance on the relatively
simple, timed, and non-reinforced tasks tended to improve on
both doses of the drug, it tended to deteriorate on the more
complex, self-paced, and contingently reinforced tasks. The
environmental manipulations presumably enhanced motivation
and effort to the optimal level in the placebo phase,
therefore the active drug "overstimulated" the subjects.
The regulatory central mechanisms presumably overstimulated
by the drug might have caused excessive self-control,
"overactivation", or "overfocusing" when flexibility was
required (Dyme et al., 1982; Robbins et al., 1979; Swanson
et al., 1979).

A hypothesis of the lack of a stable constitutional
deficit, as proposed within the self-regulation theory, is
further supported by the generally normal baseline perfor-
mance of the present sample relative to normal, quasi-
control groups selected from other hyperactivity studies.
Table 9 presented means and standard deviations for the present and comparison samples. Subnormal performance was observed only on the Wisconsin Card Sorting Test - \textbf{Failure to Maintain Set} (p<.005). Incidentally, this measure was impervious to the experimental manipulations. The \textbf{Failure to Maintain Set} is presumably sensitive to the impairment in the ability to sustain appropriate effort, and such a deficit has been identified in the performance of hyperactives (Douglas, 1983). However, the present results cannot be unequivocally evaluated because this measure interacted with the experimenter variable. Future investigations of this concept might be warranted.

\textbf{Use of Ritalin in clinical practice}

The greatest challenge for clinicians is to predict stimulant response for individual subjects. Typically, group studies tended to have limited value for the practicing clinician due to the previously discussed methodological flaws, confusion regarding the core deficits, and the great heterogeneity of the etiology and drug response. A comprehensive review of the literature by Barkley (1976) revealed that neither psychophysiological, neurological, demographic,
psychological, or diagnostic data were reliable predictors for groups of subjects. The most likely candidates were attention span and concentration measures. The utility of these variables was also emphasized by Ross et al. (1982) in their more recent review. Although these predictors might have fared well for groups, their value may still be limited for smaller subgroups or individual subjects.

No attempts to predict an individual subject's response were made in this study in the absence of any theoretical guidelines. Post-hoc analyses were employed to investigate the value of attention span measures (Barkley, 1977). The present sample was divided into two subgroups according to their Freedom From Distraction score (Wechsler, 1974) obtained from the WISC-R protocols. A combined mean scaled score of Digit Span, Arithmetic, and Digit Symbol subtests was computed for each subject. The group had naturally separated into "low" scorers of subjects who obtained scores one-half to one standard deviation below the mean (N=13) and into "high" scorers who scored one-half to one standard deviation above it (N=14). Two-way 2(attention) x 4 (experimental phase) analyses of variance performed for six significant dependent measures revealed no significant main
effects of the attention factor or attention by drug phase interactions for any of the dependent measures. This finding suggests that attention span as a proposed predictor had limited value in this study.

The large F-values in the present analyses, however, revealed that the error variance within the sample was small and indicative of the fact that the subjects responded relatively uniformly to the experimental manipulations. The visual examination of the patterns of raw scores for each subject has further confirmed the relative homogeneity of responses. Stated differently, it appears that the findings apply to the entire sample.

The self-regulation hypothesis investigated by the present author might be worth considering by the individual clinician. It was proposed that the drug response is contingent on the size of the discrepancy between the inherent competence and the actual performance of the subjects. The greater the discrepancy, the greater the likelihood of drug-related facilitation of the target behavior. With smaller discrepancies, the drug might cause limited improvement on simpler reaction-time tasks, whereas
it could cause deterioration on self-paced and contingently reinforced tasks requiring flexible self-regulation. It appears that the repeated evaluation of the off-drug performance under environmental conditions conducive to self-regulation such as continuous supervision or contingent feedback would be a valuable prognostic sign in treatment planning.

Further, a trial of placebo might facilitate the prediction of the drug response. In this study, the subjects have likely reached the optimal level of performance during the placebo phase. Therefore, the drug effect above this level was rather limited. Although the magnitude of the effect strictly due to the expectations of improvement (placebo) cannot be evaluated in this study, it is reasonable to assume that they contributed to the enhancement of tested behavior. Such a conclusion is consistent with the self-regulation hypothesis, within which the fluctuation in both motivation and deployment of effort are the core factors in Attention Deficit Disorder. Henker et al. (1980) conducted a study of expectations and attributions of children and adolescents receiving stimulant treatment and reported that the subjects expected to "calm down", "concentrate", and improve their performance following the
ingestion of the drug. In accordance with these findings, the heightened expectancy to improve on the drug might lead to increased motivation to cope with the task and subsequently greater output of energy.

Support for the prognostic value of placebo response comes from other stimulant outcome studies as well. Werry (1977) found that approximately 40% of hyperactive children responded favorably to placebo. Approximately 30% of improvement in attention and impulsivity due to stimulants was attributed to the placebo effect in the meta-analysis of 61 outcome studies performed by Ottenbacher et al. (1983). Gualtieri, Hicks, Mayo, and Schroeder (1984) also argued that the effects of Ritalin are contingent on placebo response. In that study, placebo accounted for 25-99% of the improvement due to the drug on various cognitive measures.

In conclusion, it seems feasible that the repeated evaluation of the patient’s competence-performance discrepancy and a trial of placebo may have a prognostic utility for a clinician in planning treatment for individual patients.
Future Research Directions

This research represents a first systematic attempt to study the effects of Ritalin on self-regulation in hyperactive, conduct disordered adolescents. Present findings should be conceptually replicated on both inpatient and outpatient populations using similar measures of self-regulation. The present sample was rather stringently defined, therefore it is reasonable to expect that the generalizability of these findings may be restricted to only similarly defined populations. However, given the formidable heterogeneity of investigated hyperactive samples and related inconsistency of findings across these samples, the issue of internal validity of findings is of primary importance at this time. The subsequent step would involve an attempt to generalize conclusions derived from this study to less stringently defined samples.

It appears that the lack of subjects to form a no-treatment control group prevented this author from drawing potentially important conclusions regarding the therapeutic effects of the placebo preparation independent of spontaneous fluctuations in the symptoms and repeated testing.
Within the traditional model of psychopharmocotherapy, the placebo preparation was regarded as a therapeutically "inert" nuisance variable (White et al., 1986) and it was merely employed as a minimum efficacy standard that "legitimate" treatment had to surpass. Contrary to this biological model, it is possible that the biochemically inert placebo might be a potential therapeutic agent worthy of systematic investigation.

SUMMARY

To date, the clinical lore pertaining to the acute effects of stimulants in hyperactive adolescents is based on only three controlled studies. Years of neglect of this population stemmed from pervasive myths that the childhood disorder would undergo a spontaneous remission prior to puberty and that the action of stimulants would shift from "paradoxical" calming effects to normal "excitatory" (i.e., detrimental) effects in adolescence. Further, drug efficacy research was complicated by a high incidence of associated psychopathology in hyperactive probands, especially conduct disorder. Lack of agreement regarding the fundamental deficit in hyperactivity has also impeded the development of
appropriate dependent measures. Some most commonly used
tests of attention and impulsivity lack discriminant validi-
ty and tend not to differentiate hyperactives from patients
with schizophrenia, conduct disorder, anxiety, or even
normal controls.

In the present study, dependent measures were derived
from a theory of central self-regulation and administered
during controlled random drug trials. In general, signifi-
cant, moderate to large improvement was observed in the
placebo, mild, and moderate dose phases relative to the
baseline. These findings suggest that initially the sub-
jects did not perform optimally relative to their ability.
Active drug effects relative to placebo were moderate to
large in magnitude but significant for only one measure and
marginally significant for several other measures. The
magnitude and the direction of change on the drug relative
to placebo was consistent with the self-regulation hypothe-
sis. Improvement was observed on timed, nonreinforced tasks
of lesser complexity, whereas deterioration was present on a
complex, self-paced, and contingently reinforced task where
flexible control was required. It was concluded that the
basic deficit in ADHD rests on a transient situation- and
task-contingent disruption of attention and effort. Therefore, the efficacy of the drug is limited by the magnitude of initial competence-performance discrepancy and the presence of other non-pharmacological treatment provided by the milieu in which a study is conducted. A relatively small gap between the subjects' inherent ability and output on the tests, the presence of a therapeutic milieu, and the patients' expectations of improvement likely limited the efficacy of Ritalin in this study and led the present author to conclude that alternative nonpharmacological avenues of treatment might be viable. An exploration of alternative treatment options is further reinforced by the finding that drug treatment might induce excessive self-regulation in the subjects and consequently impair their performance. The present conclusions may only hold for similarly selected and stringently defined samples equipped with a comparable level of motivation and the availability of a therapeutic milieu. Further research should attempt to replicate these findings and subsequently generalize them to less stringently defined inpatient and outpatient populations.
APPENDIX A

PATIENT INTERVIEW SCHEDULE

Ask the patient the following questions and please circle the answer.

1. What street drugs (including alcohol) have you used in the last 2 weeks?


Frequency


2. Has a doctor ever told you or your parents that you were hyperactive (or overactive, or "hyper" or as having Attention Deficit Disorder)?

Yes No Unsure

3. Have you ever been prescribed methylphenidate which is a drug also known as Ritalin?

Yes No Unsure

(If child indicates "no" or "unsure", please inquire as to whether a physician has ever prescribed amphetamine or caffeine for hyperactive symptoms. If the patient remembers being prescribed a drug, but does not recall its brand name, inquire as to how many times that he took the drug daily and what the drug or pill looked like.

If the answer is YES to any of the above, ask how long a period of time the patient was on the drug.


4. Have you ever been brought to a mental health professional for problems with overactivity, being "hyper", or for difficulty paying attention for extended periods of time?

Yes No Unsure
The child displays, for his mental and chronological age, signs of developmentally inappropriate inattention, impulsivity and hyperactivity. Because the symptoms are typically variable, they may not be observed by the clinician. Symptoms typically worsen in situations that require self-application, as in the classroom. Signs of the disorder may be absent when the child is in a new or a one-to-one situation.

When assessing ADD(H), always keep in mind the following:

THE NUMBER OF SYMPTOMS SPECIFIED IS FOR CHILDREN BETWEEN THE AGES OF 8 AND 10. IN YOUNGER CHILDREN, MORE SEVERE FORMS OF THE SYMPTOMS AND A GREATER NUMBER ARE USUALLY PRESENT. THE OPPOSITE IS TRUE OF OLDER CHILDREN. THE MANIFESTATIONS OF SYMPTOMATOLOGY MAY CHANGE WITH AGE, THEREFORE DEVELOPMENTALLY AppROPRIATE QUESTIONS MAY NEED TO BE ASKED.

SEVERITY OF THE BEHAVIOUR should be rated in terms of how much it poses a problem for the patient, his/her parents or family, and/or the degree of disruption in performance at school.

SEVERITY SCALE:  
0  Was not a problem  
1  Mild problem  
2  Moderate problem  
3  Severe problem

For each item, rate the severity in the corresponding column in terms of the patient’s CURRENT symptomatology.

ATTENTION:  At least 3 of the following

............ 1. Fails to finish things he or she starts.  
............ 2. Often doesn’t seem to listen.  
............ 3. Easily distracted.  
............ 4. Difficulty concentrating on schoolwork or other tasks requiring sustained attention.  
............ 5. Has difficulty sticking to a play activity.

IMPULSIVITY:  At least 3 of the following

............ 1. Acts before thinking.  
............ 2. Shifts excessively from one activity to another.  
............ 3. Difficulty organizing work (not due to cognitive impairment).
4. Needs a great deal of supervision.
5. Frequently calls out in class.
6. Problems awaiting turn in games/group situations.

**HYPERACTIVITY:** At least 2 of the following

1. Runs about or climbs on things excessively.
2. Difficulty sitting still or fidgets excessively.
3. Difficulty staying seated.
4. Moves about excessively during sleep.
5. Is always "on the go" or acts as if "driven by a motor."

ONSET before the age of 7.

Yes No Unsure

DURATION of at least 6 months in childhood.

Yes No Unsure

NOT DUE to schizophrenia, affective disorder, or mental retardation.

Yes No Unsure

**SUBJECT DIAGNOSIS**

Meets criteria for ADD without Hyperactivity -- Present ........
Meets criteria for ADD with Hyperactivity -- Present ........

ADD(H) Diagnosis: Mild ............
Moderate ............
Severe ............

Please proceed to the DSM-III criteria for CONDUCT DISORDER on the following page.

**DSM-III CRITERIA FOR ASSESSMENT OF CONDUCT DISORDER**

The essential feature is a repetitive and persistent pattern of conduct in which either the basic rights of others or major age-appropriate societal norms or rules are violated. The following 4 specific subtypes are based on the presence or absence of adequate social bonds and the presence or absence of a pattern of aggressive conduct.
SEVERITY SCALE:  
0 Was not a problem  
1 Mild problem  
2 Moderate problem  
3 Severe problem  

AGGRESSIVE TYPE: At least 1 of the following  

............ 1. Physical violence against persons or property (not defend oneself) (e.g. assault, vandalism)  
............ 2. Thefts outside the home involving confrontation with the victim (e.g. extortion, purse-snatching)  

NONAGGRESSIVE TYPE: At least 1 of the following  

............ 1. Chronic violations of a variety of important rules (that are reasonable and age-appropriate) at home or at school (e.g. persistent truancy, substance abuse)  
............ 2. Repeated running away from home overnight  
............ 3. Persistent serious lying in and out of the home  
............ 4. Stealing not involving confrontation with the victim  

FOR EACH ITEM MARK A CHECK IN THE CORRESPONDING COLUMN IF THE BEHAVIOUR IS PRESENT.  

UNDERSOCIALIZED TYPE: No more than 1 of the following  
SOCIALIZED TYPE: At least 2 of the following  

............ 1. Has one or more peer-group friendships that have lasted over 6 months  
............ 2. Extends himself for others even when no immediate advantage is likely  
............ 3. Apparently feels guilt or remorse when appropriate  
............ 4. Avoids blaming or informing on companions  
............ 5. Shows concern for the welfare of friends or companions  

106
DURATION of pattern of conduct of at least 6 months.

Yes
No
Unsure

DOES NOT MEET the criteria for Antisocial Personality Disorder, if 18 years of age or older.

Yes
No
Unsure

SUBJECT DIAGNOSIS

Patient meets the criteria for Conduct Disorder?

Yes
No

Patient meets the criteria for which of the following subtypes?

Very Mild: Socialized Nonaggressive .........
Mild: Undersocialized Nonaggressive .........
Moderate: Socialized Aggressive .........
Severe: Undersocialized Aggressive .........
Patient Information

Dr. Emlene Murphy of Juvenile Services to the Courts, Erica Reznick, and Hanna Lysak, doctoral candidates in clinical psychology at Simon Fraser University, are conducting clinical drug trials to examine the effectiveness of a drug called Ritalin in treating adolescents with behaviour problems associated with a history of childhood attention deficit disorder with hyperactivity.

Children or adolescents with hyperactivity or attention deficit disorder usually have difficulty in the following areas: Paying attention and concentrating; Controlling impulsive behaviour - acting before they stop to think; Controlling their activity level. The Ritalin medication you will be receiving is prescribed by your doctor at the Inpatient Assessment Unit and is helpful in treating persons who have difficulties in the above described areas. The medication will be administered by a nurse twice a day, one half-hour before the breakfast and noon meals. Ritalin medication is not addictive. The possible side-effects are stomachaches and fatigue, decreased appetite and sleep problems. If you do experience any of the above side-effects, we ask you to notify the nurse. Do not be alarmed, as most side-effects are eliminated when the dosage is decreased. If you have any further questions about the medication, please ask the nurse or doctor.

The study will also involve the administration of some psychological tests, none of which are harmful or invasive. You will be asked to complete a questionnaire and to participate individually in some computer games. Depending upon your performance on the computer tasks, you could win up to $10. The money you win will be paid to you upon your release from the Inpatient Assessment Unit.

At the end of the study, the doctor and Ms. Reznick will discuss the results with you. The findings may be included in the doctor's recommendations to the court.
Please note that should you consent to participate and later have serious complaints such that you would like to withdraw from the study, the above described procedures will be terminated at the time of your request.

Thank you for your time and cooperation.
APPENDIX C

JUVENILE SERVICES TO THE COURTS
INPATIENT ASSESSMENT UNIT

CONSENT TO PARTICIPATE

IN

RESEARCH STUDY: HYPERACTIVITY - ATTENTION DEFICIT DISORDER

(YOUTH)

I,______________________, presently on remand at the
(Name of Youth)

Inpatient Assessment Unit of Juvenile Services to the Court,

declare as follows:

(a) THAT I have read the attached information sheet which de-
    scribes the research study on hyperactivity and the use of
    the drug RITALIN and I have had the contents explained to me
    by______________________;
        (Name of Psychiatrist)

(b) THAT I understand that the assessment indicates that I have a
    history of hyperactivity in my childhood;

(c) THAT I understand the possible side effects that I may
    experience if I take the drug RITALIN;

(d) THAT the drug will be administered for a period of nine (9)
    days under the supervision of a doctor with nursing super-
    vision on a twenty-four hour basis;

(e) THAT I consent to participate in this research study and
    authorize the administration of the drug RITALIN.

    DATE:_______________

Signature of Youth__________________  Signature of Psychiatrist__________________
### APPENDIX D

**SIDE-EFFECTS QUESTIONNAIRE**

<table>
<thead>
<tr>
<th>BEHAVIOUR</th>
<th>ABSENT</th>
<th>SERIOUS</th>
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</thead>
<tbody>
<tr>
<td>Insomnia</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Nightmares</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Stares a lot/daydreams</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Talks less with others</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Uninterested in others</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Irritable</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Stomach aches</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Headaches</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Drowsiness</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Sad/unhappy</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Prone to crying</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Anxious</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Bites nails</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Euphoric</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td>0 1 2 3 4 5 6 7 8 9</td>
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</table>
# APPENDIX E

**ANOVA SOURCE TABLE FOR MAIN EFFECTS OF EXPERIMENTAL CONDITIONS**

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Square</th>
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</thead>
<tbody>
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<td></td>
<td>Between</td>
</tr>
<tr>
<td>WCST-Categories Achieved</td>
<td>27.44</td>
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<tr>
<td>WCST-Persevative Errors Errors</td>
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<tr>
<td>WCST-% Insight</td>
<td>1836.62</td>
</tr>
<tr>
<td>WCST-Failures to Maintain Set</td>
<td>.76</td>
</tr>
<tr>
<td>WFT-Total Words</td>
<td>240.65</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>223.65</td>
</tr>
<tr>
<td>Child TMT-B (sec)</td>
<td>406.24</td>
</tr>
<tr>
<td>Adult TMT-B (sec)</td>
<td>3368.28</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>6.94</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>.39</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>906.56</td>
</tr>
</tbody>
</table>

Note. WCST - Wisconsin Card Sorting Test. WFT-Word Fluency Test. TMT-Trail Making Test. 
*df, p, and F-values are presented in Table 3*
### ANOVA Source Table for Experimental Condition x Drug Order Interactions

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Square</th>
<th>Between</th>
<th>Within</th>
</tr>
</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
<td>2.59</td>
<td></td>
<td>2.24</td>
</tr>
<tr>
<td>WCST-Perserative Errors Errors</td>
<td>22.10</td>
<td></td>
<td>31.08</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>76.97</td>
<td></td>
<td>116.90</td>
</tr>
<tr>
<td>WCST-Failures to Maintain Set</td>
<td>1.85</td>
<td></td>
<td>1.85</td>
</tr>
<tr>
<td>WFT-Total Words</td>
<td>26.81</td>
<td></td>
<td>19.34</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>68.05</td>
<td></td>
<td>33.95</td>
</tr>
<tr>
<td>Child TMT-B (sec)</td>
<td>44.58</td>
<td></td>
<td>113.38</td>
</tr>
<tr>
<td>Adult TMT-B (sec)</td>
<td>558.63</td>
<td></td>
<td>184.10</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>2.14</td>
<td></td>
<td>1.06</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>1.52</td>
<td></td>
<td>1.96</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>104.17</td>
<td></td>
<td>78.21</td>
</tr>
</tbody>
</table>

*Note.* WCST - Wisconsin Card Sorting Test. WFT-Word Fluency Test. TMT-Trail Making Test.

df, p, and F-values are presented in Table 5.
APPENDIX G

ANOVA SOURCE TABLE FOR DRUG ORDER MAIN EFFECTS

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Square</th>
<th></th>
<th></th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Between</td>
<td>Within</td>
<td>F</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>WCST-Categories Achieved</td>
<td>5.73</td>
<td>12.98</td>
<td>.44</td>
<td>.813</td>
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</tr>
<tr>
<td>WCST-Perserative Errors Errors</td>
<td>18.73</td>
<td>132.40</td>
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<td>.980</td>
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</tr>
<tr>
<td>WCST-% Insight</td>
<td>94.78</td>
<td>574.80</td>
<td>.16</td>
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</tr>
<tr>
<td>WCST-Failures to Maintain Set</td>
<td>5.38</td>
<td>3.33</td>
<td>1.62</td>
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<tr>
<td>WFT-Total Words</td>
<td>143.28</td>
<td>182.54</td>
<td>.78</td>
<td>.576</td>
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<tr>
<td>Stroop Interference</td>
<td>187.77</td>
<td>93.15</td>
<td>2.02</td>
<td>.139</td>
<td></td>
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<tr>
<td>Child TMT-B (sec)</td>
<td>323.27</td>
<td>209.67</td>
<td>1.54</td>
<td>.289</td>
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<tr>
<td>Adult TMT-B (sec)</td>
<td>733.77</td>
<td>1889.88</td>
<td>.39</td>
<td>.809</td>
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<td>Digits Forward</td>
<td>9.92</td>
<td>17.17</td>
<td>.58</td>
<td>.717</td>
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<tr>
<td>Digits Backward</td>
<td>12.90</td>
<td>18.81</td>
<td>.69</td>
<td>.641</td>
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<td>Digit Symbol</td>
<td>408.90</td>
<td>793.84</td>
<td>.52</td>
<td>.761</td>
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</tr>
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</table>

Note. df = 5,15. WCST - Wisconsin Card Sorting Test. WFT - Word Fluency Test. TMT - Trail Making Test.
APPENDIX H

ANOVA SOURCE TABLE FOR EXPERIMENTAL CONDITION x EXPERIMENTER INTERACTIONS

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Square Between</th>
<th>Mean Square Within</th>
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</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
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<td>2.41</td>
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<tr>
<td>WCST-Perserative Errors Errors</td>
<td>21.04</td>
<td>29.25</td>
</tr>
<tr>
<td>WCST-% Insight</td>
<td>22.51</td>
<td>111.36</td>
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<tr>
<td>WCST-Failures to Maintain Set</td>
<td>5.32</td>
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</tr>
<tr>
<td>WFT-Total Words</td>
<td>2.34</td>
<td>22.20</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>9.46</td>
<td>44.79</td>
</tr>
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<td>Child TMT-B (sec)</td>
<td>76.82</td>
<td>91.12</td>
</tr>
<tr>
<td>Adult TMT-B (sec)</td>
<td>143.51</td>
<td>460.55</td>
</tr>
<tr>
<td>Digits Forward</td>
<td>.43</td>
<td>1.38</td>
</tr>
<tr>
<td>Digits Backward</td>
<td>2.04</td>
<td>1.84</td>
</tr>
<tr>
<td>Digit Symbol</td>
<td>19.21</td>
<td>88.14</td>
</tr>
</tbody>
</table>

Note. WCST - Wisconsin Card Sorting Test. WFT-Word Fluency Test. TMT-Trail Making Test.
F, df, and p-values are presented in Table 6.
## APPENDIX I

### ANOVA SOURCE TABLE FOR THE EXPERIMENTER MAIN EFFECTS

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Square</th>
<th>Between</th>
<th>Within</th>
<th>F</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>WCST-Categories Achieved</td>
<td>9.92</td>
<td>11.23</td>
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<td>.88</td>
<td>.359</td>
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<tr>
<td>WCST-Perserative Errors Errors</td>
<td>2.58</td>
<td>109.32</td>
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<td>.879</td>
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<td>WCST-% Insight</td>
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<td>.38</td>
<td>.543</td>
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<tr>
<td>WCST-Failures to Maintain Set</td>
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<td>3.99</td>
<td></td>
<td>.27</td>
<td>.609</td>
</tr>
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<td>WFT-Total Words</td>
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<td>181.31</td>
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<td>.05</td>
<td>.821</td>
</tr>
<tr>
<td>Stroop Interference</td>
<td>117.58</td>
<td>118.08</td>
<td></td>
<td>1.00</td>
<td>.332</td>
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<tr>
<td>Child TMT-B (sec)</td>
<td>766.80</td>
<td>199.39</td>
<td></td>
<td>3.85</td>
<td>.091</td>
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<tr>
<td>Adult TMT-B (sec)</td>
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<td>1294.11</td>
<td></td>
<td>.65</td>
<td>.451</td>
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<td>12.16</td>
<td></td>
<td>6.26</td>
<td>.022</td>
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<tr>
<td>Digits Backward</td>
<td>120.14</td>
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<td>Digit Symbol</td>
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<td>656.48</td>
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<td>2.25</td>
<td>.149</td>
</tr>
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</table>

**Note.** df = 1,19. WCST - Wisconsin Card Sorting Test. WFT-Word Fluency Test. TMT-Trail Making Test.
## APPENDIX J

### REPEATED TESTING x DRUG PHASE INTERACTIONS IN THE PLACEBO PHASE

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>F-Value</th>
<th>P</th>
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</thead>
<tbody>
<tr>
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<td>.149</td>
</tr>
<tr>
<td>WCST - Perseverative Errors</td>
<td>.48</td>
<td>.629</td>
</tr>
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<td>1.61</td>
<td>.225</td>
</tr>
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<td>WFT - Total Words</td>
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<td>.754</td>
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<td>Digit Symbol</td>
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</table>

**Note.** df = 2,20
APPENDIX K

REPEATED TESTING x DRUG PHASE INTERACTIONS IN THE MILD DOSE PHASE

<table>
<thead>
<tr>
<th>Dependent Variable</th>
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<td>WCST - Perseverative Errors</td>
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<td>.280</td>
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<td>WCST - % Insight</td>
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<td>.911</td>
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<td>Child TMT-B (sec)</td>
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<td>Digit Symbol</td>
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Note. df = 2,22
### APPENDIX L

**Repeated Testing x Drug Phase Interactions in the Moderate Dose Phase**

<table>
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<tr>
<th>Dependent Variable</th>
<th>F-Value</th>
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<tbody>
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<td>.591</td>
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<tr>
<td>WCST - % Insight</td>
<td>.97</td>
<td>.397</td>
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<td>WFT - Total Words</td>
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<td>.707</td>
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</table>

*Note.* $df = 2,19$
APPENDIX M

UNIVARIATE MAIN EFFECTS OF REPEATED TESTING IN THE PLACEBO PHASE

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>F-Value</th>
<th>P</th>
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</thead>
<tbody>
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<td>WCST - Perseverative Errors</td>
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<td>.994</td>
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<tr>
<td>WCST - % Insight</td>
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<tr>
<td>WFT - Total Words</td>
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<td>.738</td>
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<td>.235</td>
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<td>Digit Symbol</td>
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<td>.399</td>
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Note. df = 2,20
## APPENDIX N

### UNIVARIATE MAIN EFFECTS OF REPEATED TESTING IN THE MILD DRUG DOSE PHASE

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>F-Value</th>
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</tr>
</thead>
<tbody>
<tr>
<td>WCST - Categories Achieved</td>
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<td>.167</td>
</tr>
<tr>
<td>WCST - Perseverative Errors</td>
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<td>.714</td>
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<td>WFT - Total Words</td>
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**Note.** df = 2,22
APPENDIX O

UNIVARIATE MAIN EFFECTS OF REPEATED TESTING IN THE MODERATE PHASE

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<tr>
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<tr>
<td>WCST - Perseverative Errors</td>
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<td>.845</td>
</tr>
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</tr>
<tr>
<td>WFT - Total Words</td>
<td>1.96</td>
<td>.169</td>
</tr>
<tr>
<td>Child TMT-B (sec)</td>
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<tr>
<td>Digit Symbol</td>
<td>1.36</td>
<td>.280</td>
</tr>
</tbody>
</table>

Note. df = 2,19
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Golden, C.J. (1981). The Luria-Nebraska Children's


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