

Neuropsychological Assessment of Attention Problems in Pathological Gamblers

LOREEN RUGLE, Ph.D.,¹ AND LAWRENCE MELAMED, Ph.D.²

Deficits in executive, frontally mediated attention processes have been observed in substance abusers. A significant rate of childhood histories of attention deficit hyperactivity disorder has also been reported in this population, creating the question of whether attention problems predated addiction or were secondary to neurological drug or alcohol effects in pathological gamblers. To address this issue, the current study compared 33 non-substance-abusing pathological gamblers with 33 nonaddicted controls on nine attention measures and childhood behavior questionnaires. Gamblers performed significantly worse than controls on higher order attention measures and reported more childhood behaviors consistent with attention deficits. Results suggest attention deficits may be a risk factor for development of addictive disorders.

—*J Ment Nerv Dis* 181:107–112, 1993

Neuropsychological deficits in substance abusers have generally been assumed to be secondary to the neurotoxic effects of alcohol or drugs (Grant et al., 1978; Ryan and Butters, 1983). However, research has also suggested a link between childhood attention deficit hyperactivity disorder (ADHD) and substance abuse (Cantwell, 1972; Cocores et al., 1987; DeObaldia and Parsons, 1984). Consequently, the neuropsychological deficits characteristic of ADHD may be a risk factor for the development of addictions (Cocores et al., 1987; Tarter et al., 1985).

Although research on addictions has focused on substance abuse disorders, pathological gambling may also be viewed as an addictive disorder with many parallels to chemical dependency (Glen, 1979; Graham and Lowenfeld, 1986; Jacobs, 1986; Peck, 1986). Pathological gamblers without histories of substance abuse are a unique group for the study of neuropsychological deficits in addictive disorders, as there is no evidence that gambling produces neurotoxic effects.

Goldstein and associates (Carlton and Goldstein, 1987; Goldstein et al., 1985) reported that, like substance abusers, pathological gamblers present neurological findings and childhood histories consistent with the ADHD pattern. These authors did not, however, report substance abuse histories of gambling subjects. As other research suggests, a significant percentage of pathological gamblers are likely to be dually addicted

(Ramirez et al., 1983); substance abuse is a potentially confounding variable. In the present study, non-substance-abusing pathological gamblers were compared with non-substance-abusing, nongambling controls on neuropsychological functioning. To obtain additional evidence, retrospective data were collected on childhood behaviors symptomatic of ADHD.

The most consistently reported neuropsychological impairments of substance abusers and ADHD patients are on tests with relatively complex visuospatial stimuli and those measuring abstracting ability (Ackerman, 1977; Brandt et al., 1983; Douglas, 1984; Parsons and Farr, 1981). From the perspective of Posner and Presti's (1987) model of attention, this pattern suggests deficits in higher order attentional capacity.

In Posner and Presti's model, attention is not unitary, but consists of hierarchically ordered functions. In their view, the first level of attention includes alertness and the ability to focus on a stimulus. A second level is selective attention, which involves the flexibility to release attention from one stimulus and re-engage on another. Those lower order attention processes are viewed as being mediated respectively by midbrain, parietal, and temporal cortical mechanisms. At more complex levels of attention, the frontal cortex is seen as having a controlling function. Abilities at this level are described as executive aspects of attention. These include maintaining coherent goal-oriented programs, the capacity to plan and sequence, and the ability to sustain inhibitory control over distracting sensory events.

Impairment in these frontally mediated executive functions has consistently been described in both ADHD (Douglas, 1983, 1984; Weingartner et al., 1980) and substance-abusing patients (Brandt et al., 1983;

¹Brecksville V.A. Medical Center, (116B), 10000 Brecksville Road, Brecksville, Ohio 44141. Send reprint requests to Dr. Rugle.

²Department of Psychology, Kent State University, Kent, Ohio.

The authors would like to acknowledge the valuable editorial assistance of Dr. Milton Strauss and the helpful suggestions of Journal reviewers. An initial version of this paper was presented at the Eighth International Conference on Risk and Gambling, London, England, August 1990.

Grant et al., 1978; Loberg, 1980; Ryan and Butters, 1983, 1986). Deficits in lower levels of attention (alertness, engaging and selective attention) in ADHD and substance-abusing groups have also been reported (Ackerman, 1977; Elmasian et al., 1982; Garfinkle, 1984; Goldstein and Shelly, 1980). However, the findings of deficits in these lower attentional levels are less consistently found and appear to depend on increasing task demands (Ackerman, 1977; Elmasian et al., 1982; Garfinkle, 1984; Ryan and Butters, 1980). Learning and memory deficits also seem to be found mainly when complex, demanding tasks are used (Douglas, 1983; Ryan and Butters, 1986; Tarter and Ryan, 1983) and when performance is facilitated by spontaneous use of executive functions such as organized strategies for efficient encoding, learning, and retention of material (Brandt et al., 1983; Douglas, 1983; Ryan and Butters, 1980, 1983). Thus, studies of neuropsychological deficits in addictions converge on the hypothesis of executive function or higher order attention capacity limitations. The current study examined the generalizability of this pattern to gamblers. Instruments designed to evaluate the full range of attention were used, with particular focus on measures of executive function.

Method

Subjects

Sixty-six subjects were included in this study (33 gambling and 33 control subjects). Gambling subjects were recruited from the inpatient gambling treatment program ($N = 13$) at Brecksville Veterans Administration Medical Center and from Gamblers Anonymous groups ($N = 20$). Control subjects were recruited from local veterans organizations and other community groups. All subjects were white males because the numbers of minorities and women in Gamblers Anonymous and inpatient treatment are too small to permit their meaningful inclusion.

Subject groups were matched on age and education. Mean age (\pm SD) of gamblers was 41.4 ± 9.5 years, with a range of 26 to 62 years. Mean age of controls was 40.8 ± 10.0 , with a range of 23 to 61 years. Mean years of education for gamblers was 14 ± 2.3 , with a range of 12 to 20 years. Mean education for controls was 14 ± 2.1 years, with a range of 10 to 18 years. Subjects with an estimated IQ below 85 on the Shipley Institute of Living Scale were excluded.

The short form of the Michigan Alcohol Screening Test (SMAST, Selzer, 1971; Selzer et al., 1975) was employed to screen subjects for alcohol. The SMAST was reworded and used to assess for drug abuse (SMAST-D). Subjects were excluded who obtained scores above the conventional cutoff score of 2 on either the alcohol or drug form. Questions were added to both forms to

assess amount of drug and alcohol use. Gambling and control subjects did not differ significantly on these items.

The South Oaks Gambling Screen (SOGS; Lesieur and Blume, 1987) was employed to assess gambling behavior. A cutoff score of 5 as used by Lesieur and Blume was used to eliminate controls. Mean SOGS score for gambling subjects was 17.9 ± 2.4 , with a range of 9 to 20. Mean SOGS score for controls was $.2 \pm .5$, ranging from 0 to 2. Two weeks of abstinence was set as the minimum for inclusion in the study.

A structured interview format was employed with collaterals using the SMAST, SMAST-D, and SOGS to confirm subjects' self-report of substance use and gambling. All collaterals, with the exception of two who were personally seen, were interviewed over the telephone. A total of 55% of these informants were spouses, 42% were other first-degree relatives, and 3% were friends.

A brief medical history was also taken of all subjects to identify conditions or events that might compromise neuropsychological function, *e.g.*, head trauma with significant periods of unconsciousness and/or amnesia, endocrine disorder, cerebral vascular disorder, neurological disorder, liver disorder, history of myocardial infarction or other cardiovascular disorder (other than controlled hypertension), emphysema, diabetes, severe fevers, or complications at birth. Subjects were excluded who reported any such medical conditions or any history of axis I psychiatric diagnosis other than pathological gambling. Subjects were excluded who reported any history of being prescribed antipsychotic, antidepressant, antimanic, sedative, or tranquilizing medication.

Instruments

Neuropsychological Measures

Nine neuropsychological tests were used to assess attention and learning and memory across visual and auditory sensory modalities using both verbal and non-verbal stimuli. To represent the range of attention processes described as higher order or executive functions by Posner and Presti (1987), the following tests were used:

- a) *Embedded Figures Test (EFT)*: a nonverbal visual/perceptual test that invokes discriminative frontally mediated aspects of attention (Douglas, 1984). Total time score was used.
- b) *Wisconsin Card Sorting Test (WCS)*: a visual/perceptual concept formation test that has gained wide acceptance as a test that is particularly sensitive to frontal impairment (Robinson et al., 1980). Total number of trials to traditional criteria of six correct sets was scored.

c) *Porteus Maze Test, forms XII-Adult*: a visual, non-verbal test measuring the capacity to plan, attend, and make appropriate decisions (Porteus, 1965) and to focus attention while engaged in sequential problem solving (Douglas, 1984). It has been found to be responsive to medication in groups of hyperkinetic children and sensitive to frontal lesions in adults (Porteus, 1965). Age scores were employed.

d) *Trail Making Test, part B* (Trails B): a visual/verbal test of attention that also has been viewed as including elements sensitive to frontally mediated aspects of attention (Reitan, 1986). However, it seems to be mediated more by the engaging/disengaging dimension rather than the conceptual/planning aspect observed in the WCS or Porteus (Corrigan and Hinkley, 1987). Total time score was used.

e) *List Learning with Categorical Clustering* (List): a selective reminding task based on the type employed by Buschke and Fuld (1974). This type of verbal learning task is enhanced by spontaneous use of frontally mediated organization strategies and has been found to discriminate addicted and attention deficit disorder groups from controls (Douglas, 1984; Ryan and Butters, 1980). Total words correct over three trials was the score used.

The remaining four tests were included as measures of lower order attention functions:

f) *Symbol Digit Substitution Test* (SDST), forms 1 through 3: a visual, nonverbal measure of sustained attention, requiring encoding, memory storage, and visual search among alternatives (Royer et al., 1981). Total of three forms was the score used.

g) *Knox Cube Test* (Knox): a visual, nonverbal attention span measure (Stone and Wright, 1980). Total correct score was used.

h) *Primary Memory with Interference Test* (Prime): a Brown-Peterson type distraction task which is a visual/verbal task measuring the ability to inhibit distracting sensory and mental input and the capacity to engage and maintain attention (Peterson and Peterson, 1959). Total letters correct was score used.

i) *Seashore Rhythm Test*: a measure of alertness and focused attention at a basic level (Reitan, 1986). Total correct was scored.

Self-Report Measures

The childhood Self-Control Rating Scale (SCS; Kendall and Wilcox, 1979) was employed as a measure of cognitive-behavioral self-control. This test has been found to correlate significantly with children's performance on tests of impulsivity and attention, such as the Matching Familiar Figures Test and Porteus Maze Test, and with behavioral observations. This questionnaire was also used to interview collateral informants. On this instrument, 95% of informants were first-degree

TABLE I
Means and Standard Deviations for Substance Use and Gambling

Variable	Gambler (N = 33)	Control (N = 33)
SMAST		
Self	.06 ± .24	.03 ± .17
Collateral	.03 ± .17	.00 ± .00
SMAST-D		
Self	.03 ± .17	.03 ± .17
Collateral	.03 ± .17	.03 ± .17
SOGS*		
Self	17.88 ± 2.38	.15 ± .51
Collateral	16.88 ± 2.58	.00 ± .00
SCS*		
Self	122.52 ± 34.02	98.33 ± 29.64
Collateral	108.97 ± 39.70	83.58 ± 29.75

* $p < .005$.

relatives and the remaining 5% were friends or grandparents.

The Beck Depression Inventory (BDI; Beck et al., 1961) was employed to assess possible effects of depression on neuropsychological test performance (DeOaldia et al., 1983).

Procedure

All potential subjects were informed of the general requirements of the study. Participating subjects gave informed consent after receiving this information. They were then administered Shipley, SOGS, SMAST, SMAST-D and a medical history questionnaire. Those subjects who met criteria and were willing to participate were scheduled for neuropsychological assessment. All subjects with the exception of one gambler and one control completed neuropsychological assessment in one session.

Neuropsychological measures were randomized for each subject to control for order effects. All subjects were given a break of approximately 10 minutes after completion of five of the nine neuropsychological tests.

Results

As indicated in Table I, there were no significant differences between gambling and control subjects on self-report or collateral substance abuse measures (SMAST and SMAST-D).

There were significant differences between gamblers and controls on the SOGS on both self- and collateral report. Furthermore, gamblers, as was hypothesized, obtained significantly higher ratings than controls per both self- and collateral report on the SCS, indicating greater number and intensity of childhood behaviors consistent with ADHD. Correlation between self- and collateral report on SCS was significant ($r = .513$, $p < .002$). The BDI scores were also significantly higher

TABLE 2
Means and Standard Deviations for Attention Measures

Variable ^a	Gambler	Control
EFT ^{b,c}	983.73 ± 474.17	629.85 ± 392.78
WCS Total ^{b,c}	107.33 ± 22.35	88.88 ± 18.99
Porteus ^d	15.33 ± 2.16	16.16 ± .86
Trails B ^b	69.51 ± 27.03	68.33 ± 29.67
List Learning	20.97 ± 2.26	21.42 ± 2.45
Prime	35.76 ± 8.14	37.09 ± 6.78
Seashore	26.27 ± 2.50	27.12 ± 2.56
Knox Total	12.67 ± 3.39	13.27 ± 2.76
SDST Total	126.51 ± 21.66	131.58 ± 26.58

^aWCS Total, total correct score; SDST total, total across three forms.

^bPoorer performance indicated by higher scores. On all other test poorer performance is reflected in lower scores.

^c $p < .005$.

^d $p < .06$.

among gamblers. Mean BDI for gamblers was 10.79 and for controls was 5.82 ($T = 2.68$, $p = < .01$). Table 2 presents means and standard deviations for attention measures. As can be seen, gamblers had average scores consistent with poorer attentional performance on all measures.

Multivariate analysis of variance indicated that the groups differed significantly on the set of measures $F = 2.136$ (9,56; $p < .04$). Univariate follow-up tests indicated that gamblers used more trials on the WCS ($F = 13.064$ [1,64], $p < .001$) more time on the EFT ($F = 10.901$ [1,64], $p < .002$) and tended to make more errors on the Porteus mazes ($F = 3.803$ [1,64], $p < .06$). These three measures reflect executive, frontally mediated attention functions. Differences on lower order attention functions were not significant (Trails B: $F = .029$, $p < .866$; List: $F = .615$, $p < .436$; SDST: $F = .523$, $p < .472$; Knox: $F = .634$, $p < .429$; Prime: $F = .719$, $p < .400$; Seashore: $F = 1.854$, $p < .178$).

Correlations between SCS rating and neuropsychological measures were computed for each group. Among gamblers, only Prime (Brown-Peterson Interference task) was significantly correlated with SCS ($r = .380$, $p < .03$); no correlations were significant for controls.

The BDI scores were correlated with neuropsychological measures for gambling and control subjects. Among gamblers, the BDI correlated significantly with Prime only ($r = .358$, $p < .04$); no other correlations were significant.

Discussion

The main objective of this study was to determine whether pathological gamblers differed from nonaddicted controls on measures of attention, particularly those that assess executive, frontally mediated aspects of attention. Based on prior research on neuropsychological

deficits in alcoholics and ADHD subjects (Ackerman, 1977; Douglas, 1984; Ryan and Butters, 1983; Tarter and Ryan, 1983), it was anticipated that tests assessing the more executive aspects of attention would differentiate between the groups. The data obtained support the hypothesis that pathological gamblers would display greater attention deficits in the area of executive functions.

The EFT, WCS, and Porteus mazes that are most consistently sensitive to the executive aspects of attention were significant in differentiating gamblers from controls. Trails B and List, although involving frontal functions, did not discriminate between groups. These tests may not have been sufficiently perceptually complex and demanding to measure group differences. The differences on the other neuropsychological tests were quite small and did not approach significance.

The finding that gamblers obtained significantly higher BDI scores is consistent with reports of depressive symptoms among pathological gamblers (McCormick et al., 1984), particularly as the group of gamblers included hospitalized subjects. The BDI scores did not correlate significantly with those attention measures that differed significantly between gamblers and controls. Therefore, the presence of depression among gamblers would not seem to account for performance differences between the groups on neuropsychological measures.

The results are also consistent with the hypothesis that gambling subjects would endorse more childhood behaviors consistent with ADHD than would controls. Although retrospective reports are problematic, the similar pattern of differences in collateral report, and the significant correlation between self-report and collateral reports lends some credence to the finding. It is important to note that the SCS focuses on a dimension of cognitive/behavioral control, rather than aggressiveness/conduct disorder (Kendall and Wilcox, 1979). This offers support for the contention that differences in childhood history between gamblers and controls are related to overactivity, distractibility, and impulsivity, rather than to angry, acting out behaviors.

The third hypothesis, that childhood history would correlate with current neuropsychological functioning, was not supported. As with most negative findings, this is difficult to interpret. It may reflect insensitivity of either set of measures or that neuropsychological and SCS assess independent risk factors for development of addictive behavior.

In summary, this study presents evidence to support the contention that specific forms of attention deficit will discriminate gamblers as a non-substance-abusing addicted group from nongambling, nonaddicted controls. Furthermore, the results of this study indicate higher rates among gamblers of childhood behaviors

of a form associated with attention dysfunction. This suggests that attention deficits and the behavior problems associated with them have been a long-term problem in this addicted group, and supports the hypothesis that deficits in neuropsychological function are a risk factor for development of addictive disorders.

This is not to say that these results are definitive in establishing deficits in the set of skills described as executive attention functions as operative in addictive disorders. It should be acknowledged that variables including motivation, interest, test sensitivity, or other nonspecific factors may have contributed to group differences. Certainly, future research needs to more clearly assess any effect such nonspecific factors may have had on current findings.

Along with clarifying more precisely the nature of deficits observed in gamblers in this study, additional research could expand this design to compare substance abusing gamblers and other substance abusers with non-substance-abusing gamblers. This would allow evaluation of whether substance abuse contributes to a different pattern or degree of neuropsychological deficits. Also, future research should be designed to include female gamblers and non-Caucasian gamblers to determine whether the patterns observed in this study generalize to other groups of gamblers.

Furthermore, if such deficits are a risk factor in developing an addictive disorder, perhaps they also contribute to higher risk for relapse. Therefore, further research needs to be conducted to identify individuals who may be at higher relapse risk due to such attentional dysfunction and to assess relapse rates in this group. Treatment interventions to meet the needs of this potentially higher risk group need to be formulated and evaluated.

References

- Ackerman PT (1977) Teenage status of hyperactive and nonhyperactive learning-disabled boys. *Am J Orthopsychiatry* 47:577-596.
- Beck AT, Ward CH, Mendelson M, Erbenger JK (1961) An inventory for measuring depression. *Arch Gen Psychiatry* 4:561-571.
- Brandt J, Butters N, Ryan C, Bayog R (1983) Cognitive loss and recovery in long-term alcohol abusers. *Arch Gen Psychiatry* 40:435-442.
- Buschke H, Fuld PA (1974) Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology* 24:1019-1025.
- Cantwell DP (1972) Psychiatric illness in the families of hyperactive children. *Arch Gen Psychiatry* 70:414-417.
- Carlton PL, Goldstein L (1987) Physiological determinants of pathological gambling. In T Galski (Ed), *A handbook of pathological gambling* (pp. 111-122). Springfield, IL: Charles C Thomas.
- Cocores JA, Davies RK, Mueller PS, Gold MS (1987) Cocaine abuse and adult attention deficit disorder. *J Clin Psychiatry* 48:376-377.
- Corrigan JD, Hinkley NS (1987) Relationships between parts A and B of the Trail Making Test. *J Clin Psychol* 43:402-409.
- DeOlbaldia R, Parsons OA (1984) Relationship of neuropsychological performance to primary alcoholism and self-reported symptoms of childhood minimal brain dysfunction. *J Stud Alcohol* 45:386-392.
- DeOlbaldia R, Parson OA, Yohman R (1983) Minimal brain dysfunction symptoms claimed by primary and secondary alcoholics: Relation to cognitive functioning. *Int J Neurosci* 20:173-182.
- Douglas VI (1983) Attentional and cognitive problems. In M Rutter (Ed), *Developmental neuropsychiatry* (pp. 147-162). New York: Guilford.
- Douglas VI (1984) The psychological processes implicated in ADD. In LM Bloomingdale (Ed), *Attention deficit disorder: Diagnostic, cognitive, and therapeutic understanding* (pp. 280-329). New York: Spectrum Publications.
- Elmasian R, Neville H, Wood D, Schuckit M, Bloom F (1982) Event-related brain potentials are different in individuals at high and low risk for developing alcoholism. *Proc Nat Acad Sci USA* 79:7900-7903.
- Garfinkle BD (1984) Neuroendocrine and cognitive responses to amphetamine in adolescents with a history of ADD. In LM Bloomingdale (Ed), *Attention deficit disorder: Diagnostic, cognitive, and therapeutic understanding* (pp. 163-178). New York: Spectrum Publications.
- Glen AM (1979, August) *Personality research on pathological gamblers*. Paper presented at the American Psychological Association Annual Convention, New York.
- Goldstein G, Shelly C (1980) Neuropsychological investigation of brain lesion localization in alcoholism. In H Begleiter (Ed), *Biological effects of alcohol* (pp. 731-743). NY: Plenum.
- Goldstein L, Manowitz P, Nora R, Swartzburg M, Carlton PL (1985) Differential EEG activation and pathological gambling. *Bio Psychiatry* 20:1232-1234.
- Graham JR, Lowenfeld BL (1986) Personality dimension of the pathological gambler. *J Gambling Behav* 2:58-66.
- Grant I, Adams KM, Carlin AS, Rennick PM, Judd LL, Schoef K (1978) The collaborative neuropsychological study of polydrug users. *Arch Gen Psychiatry* 35:1063-1074.
- Jacobs DF (1986) A general theory of addictions: A new theoretical model. *J Gambling Behav* 2:15-31.
- Kendall PC, Wilcox LE (1979) Self-control in children: Development of a rating scale. *J Consult Clin Psychol* 47:1020-1029.
- Lesieur HR, Blume SB (1987) The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. *Am J Psychiatry* 144:1184-1188.
- Loberg T (1980) Alcohol misuse and neuropsychological deficits in men. *J Stud Alcohol* 41:119-128.
- McCormick RA, Russo AM, Ramirez LF, Taber JI (1984) Affective disorders among pathological gamblers seeking treatment. *Am J Psychiatry* 141:215-218.
- Parsons OA, Farr SP (1981) The neuropsychology of alcohol and drug use. In S Filskov, T Boll (Eds), *Handbook of clinical neuropsychology* (pp. 320-365). New York: John Wiley & Sons.
- Peck CP (1986) Risk-taking behavior and compulsive gambling. *Am Psychol* 41:461-465.
- Peterson LR, Peterson MJ (1959) Short-term retention of individual verbal items. *J Exp Psychol* 58:193-198.
- Porteus SD (1965) *Porteus Maze Test*. Palo Alto: Pacific Books.
- Posner MI, Presti DE (1987) Selective attention and cognitive control. *Trends Neurosci* 10:13-17.
- Ramirez LF, McCormick RA, Russo A, Taber JI (1983) Patterns of substance abuse in pathological gamblers undergoing treatment. *Addictive Behav* 8:425-428.
- Reitan RM (1986) Theoretical and methodological bases of the Halstead-Reitan neuropsychological test battery. In I Grant, KM Adams (Eds), *Neuropsychological assessment of neuropsychiatric disorders* (pp. 3-30). New York: Oxford University Press.
- Robinson DL, Heaton RK, Lehman RAW, Stilson DW (1980) Utility of Wisconsin Card Sorting Test in detecting and localizing lobe lesions. *J Consult Clin Psychol* 48:605-614.
- Royer FL, Gilmore GC, Gruhn JJ (1981) Normative data for the symbol digit substitution task. *J Clin Psychol* 87:608-614.
- Ryan C, Butters N (1980) Learning and memory impairments in young and old alcoholics: Evidence for the premature aging hypothesis. *Alcoholism* 4:288-293.
- Ryan C, Butters N (1983) Cognitive deficits in alcoholics. In B Kissin (Ed), *The pathogenesis of alcoholism, biological factors: The biology of alcoholism* (Vol 7, pp. 485-538). New York: Plenum.
- Ryan C, Butters N (1986) The neuropsychology of alcoholism. In

- D Wedding, AM Horton, J Webster (Eds), *The neuropsychology handbook: Behavioral and clinical perspectives* (pp. 376-409). New York: Springer.
- Selzer ML (1971) The Michigan Alcoholism Screening Test: The quest for a new diagnostic instrument. *Am J Psychiatry* 127:1653-1658.
- Selzer ML, Winokur A, vanRooijen L (1975) A self-administered short alcoholism screening test (SMAST). *J Stud Alcohol* 36:117-126.
- Stone MW, Wright BD (1980) *Knox Cube Test instruction manual*. Chicago: Stoelting.
- Tarter RE, Alterman AI, Edwards KI (1985) Vulnerability to alcoholism in men: A behavior-genetic perspective. *J Stud Alcohol* 46:329-356.
- Tarter RE, Ryan CM (1983) Neuropsychology of alcoholism: Etiology, phenomenology, process, and outcome. In M Galanter (Ed), *Recent developments in alcoholism* (Vol 1, pp. 449-469). New York: Plenum.
- Weingartner H, Rapoport JL, Buchsbaum MS, Bonney WE Jr, Ebert MH, Mikkelsen EJ, Caine ED (1980) Cognitive processes in normal and hyperactive children and their response to amphetamine treatment. *J Abnorm Psychol* 89:25-37.