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New Technology: A Biological Understanding of Attention Deficit Hyperactivity Disorder and its Treatment

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Currently, the most common treatment methodology for ADHD is the use of stimulant medication. However, recent advances in computer technology and medical equipment have enabled scientists to study neurological processes more effectively, which has led to alternatives for treating ADHD. This paper describes some of the new technology.

Recent advances in computer technology and medical equipment have enabled scientists to study neurological processes more effectively on living subjects. Such advancement has enabled scientists to gather data that was once only available after the subject was deceased. Magnetic resonance imaging (MRI), positron emission topography (PET), and electroencephalograph (EEG) are methods of monitoring neurological activity that have created vast opportunities to learn how the human brain functions. More importantly, however, is that experiments or interventions can now be conducted more reliably on living subjects and the neurological, cognitive, and/or behavioral results can be monitored upon completion of the intervention.

Attention deficit hyperactivity disorder (ADHD) is a significant problem in the United States, estimated to affect 3-5 percent of the population (Ingersoll & Goldstein, 1993, chap. 1). The American Psychiatric Association recently revised the diagnostic criteria for ADHD in the Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition (1994). The diagnostic criteria for attention problems include, but are not limited to, difficulty sustaining attention, distractibility, and memory problems. Some of the criteria needed for the additional diagnosis of hyperactivity include difficulty sitting still, excessive interruptions, and restlessness.

The correct etiology and treatment methodology of attention deficit hyperactivity disorder (ADHD) has generated significant controversies between researchers, professionals, and parents. Two of the most frequently disagreed upon questions are: what causes ADHD and what is the best method to treat it?

For years the cause of ADHD was linked to numerous social and behavioral sources such as prenatal problems, poor parenting, and improper diet (Ingersoll & Goldstein, 1993, chap 1). Currently, the most common treatment methodology for ADHD is the use of stimulant medication (Klorman, Brumaghim, Fitzpatrick, & Borgstedt, 1990). Until recently the role stimulant medication plays in both reducing attention problems and hyperactivity has not been well understood. What is known, however, is that children diagnosed with ADHD usually respond positively to stimulant medications. For instance, Rapport et al. (1994) found that the use

of methylphenidate accounted for a 46% increase in attention, 32% increase in academic efficiency, and a 27% increase in completed work. Symptoms of hyperactivity were also significantly reduced. Barrickman et al. (1995) found that both bupropion and methylphenidate reduced scores on the Connors Rating Scale for Teachers and Parents significantly on variables measuring hyperactivity.

PET Scans

While there has been limited consistent evidence explaining the neurological effects stimulant medication has on an individual, recent research and new technology may have the answer. The most recent and significant studies regarding the brain functioning in ADHD individuals involves the use of brain imaging techniques. Zametkin et al. (1990), using PET scan technology, discovered that adults with hyperactivity of childhood onset had significantly lower cerebral glucose metabolism levels than the control group while participating in a continuous performance task. The slower metabolism was particularly significant in the premotor cortex and the superior prefrontal cortex. Global cerebral glucose levels of the males tested was 8% lower than that of the controls, while the female ADHD group displayed a 12.7% reduction in comparison to their control group.

It appears as though these results may be generalizable to the public due to the nature of the sample. The individuals selected for the study were screened for both affective and personality disorders using the Schedule for Affective Disorders and Schizophrenia assessment device. They were also administered the Wide Range Achievement Test and the Wechsler Intelligence Test for Adults (Revised) to assess for learning disabilities. In addition, the subjects were screened for a current and/or historical problem with substance abuse. The subjects chosen were functioning socially and occupationally. It appears to be the authors' opinion that more impaired individuals would have even more significant abnormalities. The concluding remarks of the authors indicate that the prefrontal regions may play a vital role in the inhibition of responses, inattentiveness, and distractibility. In another study, Ernst et al. (1994) tried to replicate the work of Zametkin. This study involved the same strict controls as performed in the Zametkin study, however the subjects were adolescent boys and girls diagnosed with ADHD. They did find similar results while conducting PET scans on adolescent girls diagnosed with ADHD. During a continuous auditory performance task, the cerebral glucose metabolism in ADHD girls was 15% lower than the non-ADHD control group. On the other hand, this study was unable to find significant differences in glucose metabolism levels between adolescent males with ADHD and the male control group. Ernst explains this discrepancy: "The less constricting adult environment may be more accommodating to ADHD symptoms, giving the appearance of a reduction in symptoms. It is conceivable, then, that adults who participated in our previous study were the most severely impaired because they had been unsuccessful in adapting to their symptoms in adulthood" (p. 864).

The fact that the results were positive only for the female subjects may suggest that the female adolescent ADHD sample may be more representative of the adult ADHD sample in the previous study. It may also be suggested that "the stability of the ADHD diagnosis was reported much higher for girls than boys" (Ernst et al., 1994, p. 866). Zametkin et al. (1990) supported this finding that adult males with ADHD showed an 8% decrease in glucose metabolism, while adult

females showed a 12.7% decrease in metabolic activity.

Matochik et al. (1993) found that stimulant medications do affect cerebral metabolism levels in adult individuals diagnosed with ADHD. The results of his study suggest that the effects of stimulant medication on neurophysiological processes are not actually increasing the metabolic activity of certain regions of the brain, but rather they are governing the use of glucose throughout the brain. Before and after PET scans were conducted on 27 adult outpatient subjects diagnosed with ADHD. After analyses of their data, it was found that 9 of 16 regions actually decreased in metabolic activity after the administration of stimulant medication, while the other 7 areas increased in metabolic activity. Matochik et al. found that there were significant increases in metabolic activity in the following regions: anterior medial frontal, right anterior temporal, posterior temporal, and middle temporal; and sub-cortical regions, right thalamus, and caudate.

While Matochik's hypothesis that stimulant medication's primary function was to increase cerebral metabolic activity was not entirely correct, it is apparent that stimulants indirectly increase metabolic activity in certain regions of the brain. It could be speculated then, that the decrease in metabolic activity that stimulant medications cause in other regions of the brain may have more of an inhibitory effect on an individual's cognitions and behaviors.

In conclusion, PET scan technology has shown that there are decreased levels of cerebral metabolic activity in the frontal region of both adolescent and adult individuals with ADHD. It is possible that this has a significant effect on both the premotor cortex and superior frontal cortex. This may explain the symptomology that is characterized by an inability to inhibit motor activity, sustain attention, and memory difficulties. The gender and age differences give some understanding of the complexity and depth of this problem. While it was hypothesized that stimulants increase global glucose metabolism, it is apparent that stimulants alter the use of glucose throughout the brain. It seems obvious that the recent development of these technologies may lead one to the answers, however it may first have to forego many new questions.

Power Spectral Analyses of EEG

Another means to measure levels of neurophysiological activity is by means of EEG recordings. While the use of EEG data has been around for more than 20 years, the computerization of this instrument has given scientists the ability to topographically analyze individual results and statistically compare them to group results.

The neural frequencies measured using power spectral analyses (PSI) include delta, theta, alpha, and beta. Delta frequencies (Bestak, 1984) are typically produced when an individual is in a deep sleep. Theta frequencies are produced when an individual is in a rather light state of sleep. Alpha activity is usually the highest amplitude neural frequency, produced when an individual is awake and in a fairly relaxed state. Beta activity increases as the individual becomes more focused or is involved in more complex cognitive processes.

By means of a 16-channel multi-electrode EEG, Mann et al. (1992) achieved 80% diagnostic accuracy of individuals meeting the criteria for ADHD. His results confirmed those of Zametkin's, finding evidence that during cognitive tasks there is hypoactivation of the frontal

neocortex. That is, in the frontal region, individuals with ADHD tend to produce higher amplitude theta activity during cognitive tasks which is usually only produced during a light sleep with limited awareness. During the same cognitive task, ADHD subjects were found to produce low amplitude beta activity that is typically enhanced during more difficult cognitive tasks. When involved in complex cognitive processes the typical individual responds quite differently, with beta activation and suppression of theta activity creating a more alert and focused state of mind, rather than an inattentive state similar to day dreaming.

Recent event-related potential studies have also generated interesting results regarding the neurological activity of those diagnosed with ADHD. Event-related potential studies conducted by Robaey et al. (1992) hypothesized that the difficulties ADHD children have may be due to their reduced ability to relay information regarding controlled cognitive processes. He continued to hypothesize that most of their impulsive and distractible behaviors are a result of their tendency to function in neural frequencies that tend to involve automatic cognitive and behavioral processes. His experiment involved testing ADHD children with a stimuli screen that presented two different types of tasks. During the first task, the child was asked to differentiate between repeated and novel visual images, thus tapping into the more automatic type of processing. The second task involved the children identifying novel words that did not correlate with the selected classification, to measure more controlled cognitive processes of reading and category differentiation. The results suggested that the ADHD children actually performed better at recognizing or detecting novel stimuli of a basic shape or form. The evoked-related potentials on these tasks showed the P250 in the centrofrontal area was significantly larger in the ADHD group. In fact, they were larger regardless if it was a repeated or a novel stimulus, suggesting that the children's orientation to differences between environmental stimuli be enhanced regardless of its relevance. However, their ability to produce and/or sustain P300 event-related potentials in the centro-frontal area was limited during the reading tasks. Thus, while the ADHD children were superior in their ability to detect stimuli, their ability to interpret the stimuli's meaning was more limited than that of the control group.

MRI Results

A recent MRI study conducted by Semrud-Clikeman et al. (1994) indicates that children with ADHD have a less developed splenial area of the corpus callosum. While her sample size was small and this is the first study of its type, the author speculates that there is a possibility of a relationship between her findings and that of Zametkin and his colleagues. She believes that the lowered metabolism found in the posterior cortical regions that Zametkin's study identified may be related to a smaller splenium with fewer fibers for interhemispheric communication.

EEG Training as a Treatment

The results from these new technologies has created significant insight into a previously misunderstood disorder of early childhood. It has also begun to increase the options available when considering treatment of ADHD. Lubar (1992), a pioneer in the new field of neurotherapy, has found that by training individuals to reduce excessive slow wave theta activity and increase the more intense beta activity in the frontal lobe, they are able to reduce symptoms of

hyperactivity, and increase their levels of focus and concentration. Lubar reports that after 30-40 sessions of neurotherapy individuals with ADHD show significant academic improvement and score significantly higher on intelligence testing.

In conclusion, it is obvious that all of the preceding research is in its early stages and that more research is needed prior to making firm conclusions. The effect of this new technology represents significant advances for the field of psychology. While most of the research in this article is relatively new, it seems evident that the cerebral glucose metabolism levels of those diagnosed with ADHD seem to identify both underactivity and overactivity in different regions of the brain. This is most significant in the frontal lobe. Both electroencephalogram and evoked potential studies tend to support this conclusion. Given that the effects of stimulant medication for ADHD have been proven to be significant for many years, it is encouraging that other treatment options such as neurotherapy and diagnostically accurate devices such as power spectrum analyses are becoming available.

While behavioral elements will continue to play a role in the understanding of ADHD, it is obvious that more biological explanations will be available in the near future. Based on the results of this research regarding new technology on the etiology and treatment of ADHD, it seems apparent that the confusion about its cause is related to the diffuse nature in which it affects the brain. Variables of attention, memory, and motor activity are all affected and it does not seem possible that any one region of the brain will ever be identified as responsible for ADHD.

References

American Psychiatric Association. (1994). Diagnostic and Statistical Manual of Mental Disorders (4th ed.). Washington, D.C.: American Psychiatric Association.

Barrickman, D. O., Perry, P., Allen, A., Kuperman, S., Arndt, S., Herrmann, K., & Schumacher, E. (1995). Bupropion versus methylphenidate in the treatment of attention deficit hyperactivity disorder. Journal of the Academy of Adolescent and Child Psychiatry.

Bestak, R. (1984). The Brain. New York: Bantam Books.

Ernst, M., Liebenauer, L. L., King, A. C., Fitzgerald, G. A., Cohen, R. M., & Zametkin, A. J. (1994). Reduced brain metabolism in hyperactive girls. Journal of The American Academy of Child and Adolescent Psychiatry, 33, 858-868.

Ingersoll, B., & Goldstein, S. (1993). Attention Deficit Disorder and Learning Disabilities (pp. 34-35). New York: Doubleday.

Klorman, R., Brumaghim, J., Fitzpatrick, P., & Borgstedt, A. (1990). Clinical effects of a controlled trial of methyl-phenidate on adolescents with attention deflct disorder. Journal of the American Academy of Adolescent and Child

Psychiatry, 29, 702-709.

Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention deficit/hyperactivity disorders. Biofeedback and SelfRegulation, 16, 201-225.

Mann, C. A., Lubar, J. F., Zimmerman, A. W, Miller, C. A., & Muenchen, R. A. (1992). Quantitative analyses of EEG in boys with attention deficit hyperactivity disorder: A controlled study with clinical implications. Pediatric Neurology, 8, 3036.

Matochik, J. A. et. al. (1993). Effects of acute stimulant medication on cerebral metabolism in adults with hyperactivity. Neuropsychopharmacology, 8, 377-386.

Rapport, M. D., Denney, C., DuPaul, G. J., & Gardner, M. J. (1994). Attention deficit disorder and methylphenidate: Normalization rates, clinical effectiveness, and reponse prediction children. Journal of the American Academy of Child and Adolescent Psychiatry, 33, 882-893.

Robaey, P., Breton, D., Dugas, M., & Renault, B. (1992) An event-related potential study of controlled and automatic processes in 6-8 year-old boys with attention deficit hyperactivity disorder. Electroencephalography and Clinical Neurophysiology, 82, 330-340.

Semrud-Clikeman, M., Filipek, P. A., Biederman, J., Steingard, R., Kennedy, D., Renshaw, P., & Bekken, K. (1994). Attention deficit hyperactivity disorder: Magnetic resonance imaging morphometric analyses of the corpus callosum. Journal of The American Academy of Child and Adolescent Psychiatry, 33, 875-881.

Zametkin, A. J., Nordahl, T E., Gross, M., King, A. C., Semple, W. E., Rumsey, J., Hamburger, S., & Cohen, R. M. (1990). Cerebral glucose metabolism in adults with hyperactivity of childhood onset. The New England Journal of Medicine, 323, 1361-1366.

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