DOCUMENT RESUME

ED 321 457 EC 231 577

AUTHOR Weinberg, Warren A.; Emslie, Graham J.
TITLE Attention Deficit Hyperactivity Disorder: The Differential Diagnosis.
SPONS AGENCY National Inst. of Mental Health (DHHS), Rockville, Md.
PUB DATE Feb 90
NOTE 42p.; Paper presented at the Annual Conference of the Learning Disabilities Association of America (Anaheim, CA, February 21-24, 1990).
PUB TYPE Guides - Non-Classroom Use (055) -- Reports - Research/Technical (143) -- Information Analyses (070)

EDRS PRICE MF01/PC02 Plus Postage.
DESCRIPTORS *Attention Deficit Disorders; Behavior Disorders; *Clinical Diagnosis; Depression (Psychology); Elementary Secondary Education; Emotional Disturbances; Handicap Identification; *Hyperactivity; Learning Disabilities; *Medical Evaluation; Multiple Disabilities
IDENTIFIERS *Differential Diagnosis

ABSTRACT

This paper presents information on the diagnostic criteria and management of disorders that may be wrongly identified as Attention Deficit Hyperactivity Disorder (ADHD) or may coexist with ADHD thus complicating identification and treatment. The disorders discussed are: depression, mania, primary disorder of vigilance, narcolepsy, developmental specific learning disorders, conduct disorders, and acquired neurological deficits. The introduction briefly reviews the history of ADHD identification and treatment. A study in which 100 referred children (ages 3 to 17) were systematically examined found ADHD criteria were met in 63 of the children but only four had ADHD alone. Forty of the children had both developmental specific learning disorders and depression as well as ADHD. Two longitudinal studies reporting relationships between hyperactivity and depression and between learning disability, depression, and ADHD are also briefly reported. Each of the other disorders is discussed including criteria for diagnosis, course of the disorder, biological correlates, the presence of concurrent disorders, and treatment approaches. It is noted that methylphenidate and pemoline, often prescribed for children doing poorly in school, can decrease hypomanic symptoms and improve vigilance but worsen or induce depression. Prognosis for children and adolescents with primary affective illness is reported to be good with appropriate management. Includes 84 references. (DB)

ATTENTION DEFICIT HYPERACTIVITY DISORDER:

THE DIFFERENTIAL DIAGNOSIS

Warren A. Weinberg, M.D.

and

Graham J. Emslie, M.D.

From the Departments of Neurology and Pediatrics (Dr. Weinberg) and Psychiatry (Dr. Emslie), University of Texas Southwestern Medical Center at Dallas, Texas and Children's Medical Center of Dallas

Acknowledgements

This work was supported by contributions from the Caleb C. and Julia W. Dula Educational and Charitable Foundations, Mr. and Mrs. Woody Hunt, Mr. and Mrs. Morton Meyerson (Dr. Weinberg), and by a grant from NIMH, MH 39188 (Dr. Emslie).

This manuscript could not have beer. submitted without the superb diligence and typing of Melody Brummett and the patient and data management of Caryn Harper and Jeanne Rintelmann.

ABSTRACT

Children and adolescents doing poorly in school and/or home are often labelled as suffering from attention deficit hyperactivity disorder (ADHD). Frequently the referred child is not examined systematically for the presence of other disorders. This paper presents the diagnostic criteria and management of disorders that may be wrongly identified as ADHD or may coexist with ADHD thus complicating identification and treatment. The disorders discussed are depression, mania, primary disorder of vigilance, narcolepsy, developmental specific learning disorders, conduct disorders and acquired neurological deficits.

Key Words: attention deficit hyperactivity disorder, depression, mania, vigilance, learning disorders, conduct, children.

INTRODUCTION

Children and adolescents not doing well in school are often referred to physicians for further understanding of their problems. Attention deficit disorder hyperactivity disorder (ADHD) is now a widely used label by primary care physicians and specialists. However, the clinician, when presented with a child or adolescent manifesting difficulties with attention, distractibility, impulsivity, restlessness, and inappropriate excessive motor activity, must go beyond the external behaviors and examine brain and its' functions further than an automatic stamp of ADHD and a prescription for methylphenidate or pemoline. The clinician should consider that ADHD relates to "X" as fever relates to infection or inflammation and their causes[1].

In the late 1930's it was noted that benzedrine benefited the impulsivity and hyperactivity of so called brain damaged children[2]. In the late 1940's and early 1950's this heterogeneous group of children was called the Strauss syndrome[3]. In the 1960's common labels for this same group of children were Minimal Brain Dysfunction[4] and the Hyperkinetic Impulse Disorder[5]. In the 1970's a commonly used term was the Hyperactive Child Syndrome[6] and now in the 1980's this heterogeneous group of children and adolescents doing poorly at school and/or home are labeled ADHD[7,8]. It is clearly evident that children fulfilling criteria for ADHD have other recognizable conditions[9,10]. These other conditions have biological correlates, familial characteristics and their natural course are being described with specific treatments defined[11-14]. The common disorders presenting as ADHD are 1) depression; 2) mania; 3) primary disorder of vigilance (PDV); 4) narcolepsy; 5) task-dependent

attention disorder (learning disorders); 6) primary disorder of conduct; and 7) acquired focal neurological deficits (Table 1)[1].

Two major problems in the evaluation of children with ADHD are the misdiagnosis of other recognizable disorders as ADHD and the failure to recognize comorbid disorders. When a child presents fulfilling criteria for more than one condition, e.g., depression, learning disorders and conduct disorders, the clinician is faced with the problem of determining the interaction between these disorders and their relative relationship to morbidity. Which one is the primary cause of disturbance at home, school or play? Additionally the temporal relationship of comorbid diagnoses is also important. It is not uncommon for a referred child to evidence ADHD symptoms only when depressed or manifest ADHD symptoms prior to the onset of their depression [15-17]. It is well documented that parents more readily identify and report negative actions and poor cooperation in the child. Schools identify disruptive behavior and poor performance, but children are reliably able to report on their feelings, interests and energy[18,19]. Appropriate diagnosis should include information from all available sources. Diagnosis may be assisted by the use of parent and child self-report instruments. However, a complete diagnostic evaluation must include a semistructured closed-end interview of both the child and his caretakers towards more conditions than the symptom complex of ADHD and must include observations of higher cortical functions (verbal, written and nonverbal communication skills; vigilance, diligence and volition; motor skills; and adaptive intelligence)[11].

Attributing behavioral problems to a biological basis may be comforting to parents as these behaviors might otherwise be blamed on poor parenting or poor schooling with the added notion that the child may

"outgrow it". The label of ADHD is palatable within this context. Physicians when faced with a child failing in one or more of his environments can ask a few questions, review a parent and child-teacher behavior form and offer quickly a prescription for stimulants. This approach to diagnosis and treatment is supported by the pediatric literature on ADHD which is void of studies evaluating additional causes of the symptom complex of ADHD[7,8,20,21]. In this context when ADHD alone is looked for then that is all that is found. However, recent population studies are demonstrating that children doing poorly most often meet criteria for more than one Diagnostic and Statistical Manual, third edition (DSM III)[22] Axis I disorder and infrequently ADHD alone[9,10].

DIFFERENTIAL DIAGNOSIS:

We systematically examined one hundred consecutive children and adolescents using established criteria and methodology previously reported[11]. These children were referred to the Pediatric Behavioral Neurology Program at Children's Medical Center of Dallas. The clinic serves as a tertiary, university based, referral center with referrals mostly from private physicians, including pediatric neurologists and psychiatrists, and psychologists. These 100 patients were all Caucasian and middle to upper middle class. The ages ranged from 3 to 17 years (mean 10.6 $\pm$ 3.4 years); 81 males and 19 females. All were of normal intelligence as judged by adaptive behavior. Excluded were referrals manifesting more fundamental neurologic disease. Table 2 presents the prevalence of ADHD, developmental specific learning disorders (DSLD) and depression. ADHD criteria was met in 63/100 of these children but only 4/100 had ADHD alone. In this population, 40/100 manifested all three conditions at the time of initial evaluation. Of the 63/100 children with

ADHD, 53/63 (84%) had DSLD, 46/63 (73%) were clinically depressed, and 40/63 (63%) evidenced both DSLD and depression. Clearly the majority of patients (76/100) had two or three of these disorders.

To determine which of these conditions is primary or how these conditions interact with each other can only be determined by longitudinal prospective study. For example, does the child treated with stimulants evidence an increased rate of learning and a lifting of depression? Does the learning disabled child show less ADHD symptoms and freedom of depression in a specialized school setting? Does the depressed child evidence less trouble in school performance, improved learning and less ADHD symptoms if the depression is treated? There is some evidence to date that all these states occur.[23-25]

The relationship of depression and hyperactivity has been reported [15,16,17,26]. In an earlier paper of 72 consecutive prepubertal children referred to a school based educational diagnostic center, it was noted, utilizing criteria for depression, 42 of the 72 children with significant developmental specific learning disorders fulfilled criteria for depression and 30 were nondepressed. Of these 72 children 31 fulfilled criteria for hyperactivity. Eleven of the 31 had been hyperactive since infancy or toddler age and free of depression. However, 20 of the 31 hyperactive children were also depressed. Ten of the 20 hyperactive/depressed children were hyperactive only when depressed.[15] A further report of 223 consecutively evaluated normally intelligent learning disabled prepubertal children referred to two educational diagnostic centers showed that 117/223 (52.5%) were hyperactive. Of these hyperactive children, 64/117 (55%) demonstrated clinically significant hyperactivity only during episodes of depression and 38.5% of these 223 children manifested both hyperactivity and depression (Table 3)[16].

Two longitudinal studies report the relationship of learning disability, depression, and ADHD [17,23]. The aim of these two studies was to examine the interaction of those three conditions by controlling for the influence of inappropriate stress or failure due to the learning disability. Five consecutive years of children admitted to a school optimally designed for children with learning disabilities were studied. The curriculum of the school was based on principles of true education using bypass compensatory strategies. The educational methodology has been previously reported[17]. All the children (N=227) had normal intelligence with severe learning disabilities determined by multiple psychometric and achievement tests and adaptive behavior[17]. At the time of admission, 81 (35%) were depressed without hyperactivity, 65 (29%) had hyperactivity with depression, 34 (15%) were hyperactive without depression and 32 (14%) had no diagnosable behavioral condition. A prospective study of these students attending school for more than one year showed that 73% of those who only evidenced depression and 71% who evidenced only hyperactivity in addition to their learning disability had no behavioral problems during their tenure in school. However, 66% of those evidencing both depression and ADHD continued to manifest behavioral problems during prospective follow[23]. Most, if not all, of these problems were cyclical as opposed to being persistent.

The paradigm for the clinical evaluation of disorders of higher cortical functioning in school aged children has been published[11]. Specifically, there are four major areas of functioning that must be assessed in each child evaluated. These functions are independent of each other but interact significantly. These functions are a) disturbance of mood and affect, including the vegetative symptoms of depression; b) adaptive intelligence and disorders of learning and communication; c)

disorders of vigilance (alertness and awakefullness), sleep and diligence; d) disorders of conduct and volition. The criteria and a brief discussion for these common disorders in this age group are presented below.

1. Depression.

Depression has been widely studied in children and adolescents and is probably the leading cause of failure in school, play and home in otherwise healthy, normally intelligent young people[23]. Established criteria for depression are available and the various criteria have been compared[15,27,28]. Depression is a symptom complex of disturbed moods and feelings (dysphoria) with self deprecation, beliefs of persecution, poor concentration, lessened interest and energy, loss of the ability to anticipate or experience pleasure (anhedonia), somatic complaints and a change in vegetative functions, e.g., sleep, appetite and affection. The decrease in a child's affectionate behavior may be equivalent to the adult depressive decreased libidinal drive. This symptom complex must be present for more than one month and associated with malfunctioning either at home, school or in play. There must be a change from the child's usual self and not caused by another medical disease.

Table 4 presents an established criteria for depression in children[15,29] and compares it to the original criteria for depression in adults[30]. The criteria for depression in children contains ten major symptom categories. The child must manifest a dysphoric mood and self deprecation (I & II) plus four or more of the remaining eight symptom categories (III - X). In evaluating a child each of these symptoms must be systematically asked about from both the parent and child. Some symptoms will be more reliably reported by children and others by parents. A questionnaire for children and adolescents has been developed based upon

this criteria. The questionnaire requires a fourth grade reading ability and can be used to screen for depression[1,12,31].

The course of depression in children is variable. There are three common courses. Beginning at eighteen months to three years of age there is noted mood swings, ease of frustration, excessive tantrums, and hyperactivity with poor attention. By three to five years of age the child begins to manifest self-deprecation, beliefs of persecution and interval insomnia. During these years this mood and affective instability is manifested in good and bad days and moments per day. With further aging clear episodes persisting for several weeks occur leading to a period of major depression. Another course is hyperactivity in utero, a colicky, irritable, sleep disturbed, demanding unhappy infant becoming a "supermarket" toddler and eventually a clear cycle of major depressive disorder. Thirdly, a child or adolescent with no prior instability or disturbance of mood and affect begins to manifest for no apparent significant reason the symptoms of major depression. There are children that continue to manifest mood swings occurring with good and bad days but no periods of discrete worsening or stable well periods. This is termed dysthymic disorder[22]. We anticipate that a major depressive episode will occur at some time[13].

The robust dependent variable in biological psychiatry is family history[30]. The evaluation of children for depression requires obtaining a detailed family history for affective illness (depression and mania), alcoholism, sociopathy and hysteria (Briquet's Syndrome) in first and second order relatives. Depressed children will have a positive family history for depression[15,17] (one first order or 2 or more second order biological relatives). Likewise 30 to 40% of depressed children have a parent who is actively depressed at the time of the initial

evaluation[15]. Positive family histories will fall into two groups: families positive for only affective disease ("pure familial"), and those with affective illness plus one or more of either alcoholism, hysteria (Briquets Syndrome) and sociopathy ("depressive spectrum biogeny")[12,32].

Biological correlates for depression in children similar to those in adults have been reported including polysomnographic[33] and hypothalamic pituitary axis differences[29]. The neurology of depression is being defined[34,35]. Seemingly the right cerebral hemisphere is important for the manifestation of the vegetative and dysphoric symptoms of depression[34-37]. Specifically, temporal and anterior parietal regions are malfunctioning during a depressed state[36]. However, the literature from adult stroke patients has suggested depression occurring from left frontal lesions [38-40]. Whether or not the malfunctioning of the brain is a primary cortical condition or depression is a metabolic encephalophathy secondary to a more basic disorder at the locus ceruleus or medium raphe brain stem nuclei and its' bioamine products remains unknown. Our bias is that depression is a primary problem of right temporal-parietal cerebral cortex. The bioamine correlate to depression is well established in relationship to the two neuro-transmitters, serotonin and norepinephrine. The bioamine correlate most reproducible remains the excessive reuptake of serotonin and/or norepinephrine at the cortical presynaptic terminals[41].

Neurological examination is very important in elucidating evident neurological signs in young people manifesting primary affective illness. In depression the child and young adolescent manifest left limb abnormalities of tone, posturing and reflexes[25,35]. These abnormal motor signs disappear during well states.

It should be stated that anticonvulsant drugs used to control seizures can induce or promote depression. Phenobarbital is notorious for inducing

symptoms of agitation, hyperactivity and depression, especially in children with a family history of affective disorders [42,43]. In our experience primidone, carbamazepine and the benzodiazepines also promotes depression[44]. Migraine is often functionally significant only during periods of depression[45]. When treated with periactin and/or propranolol, the headaches may improve but depression often worsens.

Concurrent diagnoses are common in children with major depressive disorder[46,47]. Recently, we examined sixty-five children and adolescents admitted to the Children's Medical Center Psychiatric Unit who fulfilled DSM III criteria for major depressive disorder. ADHD criteria was met by 21 out of 65 of these subjects. The ADHD preceded the onset of the major depressive disorder and most of these young people had been previously treated for ADHD with methylphenidate and pemoline.

The hallmark and proven treatment for depression in adults are the tricyclic antidepressant medications (Table 5)[41]. It is not possible by laboratory study at this time to predict which antidepressant might be best for an individual depressed person. It is hypothesized that a depressed patient might be right brain deficient in serotonin, norephinephrine, both or neither. Amitriptyline is more of a serotonergic agonist at the presynaptic terminal as compared to desipramine which is a norephinephrine agonist. Imipramine seems to be equally serotonergic and noradrenergic. The other tricyclic antidepressants vary, some being more serotonergic and others noradrenergic. Based upon clinical studies in adults and empirical observation in children and adolescents, if one tricyclic antidepressant is not effective then one must try other tricyclics. If a first order biological relative has been successfully treated with a tricyclic antidepressant, the child most often responds to that same drug. Even though the tricyclic antidepressants are not FDA

approved for children less than 12 years of age (except for imipramine for enuresis) we believe these drugs to be safe and effective in lifting depression in children[1,12,17,48,49].

There are observations that assist the clinician in choosing the sequence of tricyclic antidepressants. Prepubertal children who are not excessively obese are offered a trial on amitriptyline. If not effective, then imipramine followed by desipramine. For depressed children not obese but with significant anxiety and/or phobias, doxepin is offered followed by amitriptyline and then nortriptyline. For pubertal adolescents, imipramine is offered first followed by desipramine, then nortriptyline; if not effective, amitriptyline. Obese depressed children and adolescents are first offered a trial on imipramine followed by desipramine, then nortriptyline, a metabolite of amitriptyline. We avoid in obese young people amitriptyline and doxepin which seems to cause polyphagia with subsequent weight gain. Imipramine seems to lower appetite, thus lower weight. Desipramine has a more neutral effect on appetite and weight. For children and young adolescents manifesting both depression and primary disorder of vigilance, protriptyline is often the drug of choice.

Equally important is cognitive coaching and counseling of the individual and his primary caretakers. Cognitive coaching is assisting the individual and his caretakers, on an ongoing basis, to come to know that actions should dictate feelings, rather than feelings promoting actions. The statement offered is "learning to use ones' intelligence to override ones' feelings" (Table 6)[12].

In treatment resistant depressed children newer antidepressants (e.g. fluoxetine, trazodone, maproptyline) are occasionally effective. Other treatment strategies, as in adults, have included the addition of lithium, stimulants and thyroid hormone. The effectiveness of these strategies, in

this age group, is unclear at this time. Methylphenidate commonly induces or promotes depression in susceptible children, unlike reports in adult depressives.

2. Mania

Table 7 lists the criteria for mania in children[50] and compares it to the original criteria for mania[30] in adults.

Mania is euphoria, defined as elated, expansive mood and feelings with inappropriate cheerfulness, intrusiveness, disruptiveness, push of speech and flight of ideas. Marked inattention and difficulty concentrating and staying on task are prominent symptoms. Also evident are sexual hyperactivity and lessened need for sleep. The hallmark of mania is hostile anger (rages) and denial, "nothing is wrong with me", as the young person is tearing up the world. This symptom complex must be present for at least two weeks.

Although the classic symptom complex of mania is relatively infrequent in children, a course consistent with chronic hypomania as a cause of ADHD is common. Hypomania is characterized by hyperactivity, silliness, giddiness, cheerfulness, distractibility, inattention, moments of rudeness and crudeness and fluctuating cheerful to irritable moods[22]. Hypomania can have its onset at any period from infancy on and is one cause of developmental hyperactivity.

Cyclothymia is characterized by moments per day, with good and bad days of mixed brief states of both dysthymia and hypomania[22]. This condition persists for years in children. In our experience and that of others, many of these children will develop manic-depressive disease, either rapid cycling bipolar disease or discrete episodes of depression and mania[13,51,52].

The biology of mania is less well understood. Mania can not be explained by a unitary bioamine theory, either hyper-excitability of the post-synaptic terminals of serotonin and norephinephrine or by the neurotransmitter dopamine, either at its' pre or post-synaptic membrane. During manic episodes abnormal motor signs are noted in the right limbs[12]. These findings suggest mania to be a left hemisphere abnormality, possibly left mesial temporal cortex or left limbic system. The abnormal neurological signs of tone, posturing and reflexes disappear during the well state.

The treatment for mania and bipolar disorder is lithium[53] which has been used safely and effectively in children [54,55]. However, few children and adolescents are completely controlled by lithium alone. Many children requiring lithium continue with prominent depressive symptoms and require the addition of a tricyclic antidepressant. Those with persisting hypomanic symptoms may require the addition of clonidine, methylphenidate, and/or thioridazine. If hostile anger is still symptomatic then carbamazepine is helpful.

Children manifesting affective illness (depression or bipolar disorder) with a "pure familial" family history usually respond well to treatment. The complex difficult to control children come from a "depressive spectrum biogeny" family history. Any drug used to treat depression can either induce or promote mania and any drug used to control mania can induce or promote depression. Another complexity is the concept that a malfunctioning right brain can cause the left brain to act manic and vice versa. In unusual cases a manic state will respond to a tricyclic antidepressant medication. Similarly, a depressed state will be worsened by all the antidepressants only to respond to lithium. Hypomania often responds to a stimulant (methylphenidate; pemoline). However, in hypomanic

young children treated only with stimulant medication, rapid cycling bipolar symptoms are commonly noted.

3. Primary Disorder of Vigilance

Vigilance is steady state alertness or steady state awakefullness. The body of scholarly scientific psychological data defines vigilance as sustained attention[56]. Neurological literature defines a neural network for directed attention[57]. The arousal state is dependent upon the intactness of that network with predominance of the right cerebral hemisphere[57,58] (right posterior parietal cortex). One cause of trouble with sustained attention is the inability to stay awake, or alert, on tasks requiring continuous mental performance. Tests for measuring vigilance are not usually measuring pure vigilance but are measuring sustained attention. As such there is no laboratory test for vigilance. Clinically there are people of all ages who manifest difficulty staying awake and alert in activities requiring continuous mental performance. We have labelled this condition the Primary Disorder of Vigilance[1,11,12]. It is our opinion that this condition is a leading cause of ADD without hyperactivity or only minimal hyperactivity and is an inherited disorder of the right posterior parietal cortex.

Primary Disorder of Vigilance is manifested by inappropriate sleepiness with yawning, stretching, fidgeting, moving and restlessness as efforts to stay awake. This leads to difficulty attending to tasks and appearing restless, inattentive, "bored", and sleepy. These individuals often resist such tasks knowing the discomfort it causes. Symptoms of this disorder is particularly evident in reiterative tasks such as reading, listening to lectures, composing an essay and drill requiring continuous mental concentration. People with this condition will fall

asleep or doze while reading or listening to lectures, riding in a car or sitting in an office, or classroom. They often complain of being "bored" rather than sleepy. Repetition rapidly becomes boring, monotonous and tiring. In such activities, adults fall asleep while children become restless, inattentive, hyperactive and "daydream". This observation is consistent with Zentall's optimal stimulation theory of hyperactivity[59]. It is the opinion of the authors that children who are hyperactive only in class or when having to be still and concentrate are manifesting Primary Disorder of Vigilance. For this group of children ADHD is symptomatic of a disorder of continuous arousal and steady state awakefullness. These children are actually "sleepy", more so when inactive and having to concentrate.

Vigilance is worsened during a period of depression and when properly evaluated it is not unusual for a child failing and misbehaving in school to be manifesting this condition plus DSLD and affective illness[11]. Of the 100 consecutive children previously described, 63 fulfilled criteria for ADHD. Forty-seven of these sixty-three children (74.6%) manifested Primary Disorder of Vigilance or a trait for this disorder as a factor in their difficulties with attention.

Treatment is often successful with methylphenidate or pemoline when free of depression. If depression is evident, then a tricyclic antidepressant is used, preferably without methylphenidate or pemoline, until the depression has lifted. Protriptyline is beneficial in those manifesting both depression and primary disorder of vigilance. On occasion, stimulant and anti-depressant drugs will be needed concomitantly. Sedative drugs including antihistamines and anticonvulsants worsen vigilance in this group of children.

Very important in management is the offering of instruction, practice and experiences through context of randomization rather than repetition, reiteration and excessive drill. Parents and teachers should provide children with Primary Disorder of Vigilance environments that randomize the occurrence of events. Frequent changes in daily living schedules should be provided. Classroom instruction should be varied, avoiding structured sameness. Vigilance may improve or worsen with aging and/or be relatively task dependent.

4. Narcolepsy

Narcolepsy is an uncommon cause of ADHD but must be considered in the differential diagnosis. Narcolepsy is characterized by inappropriate sleep attacks in association with hypnogogic hallucinations, sleep paralysis and/or cataplexy. These are symptoms of Rapid Eye Movement (REM) sleep states occurring inappropriately during the day, on entering sleep, on arousal during the night or in the morning. Narcolepsy can be considered an abnormality of the brain stem property of REM sleep and overrides the right brain function of vigilance (awakefullness).

Narcolepsy appears to be a dominantly inherited condition in children[60]. The inappropriate sleep attacks and hypnogogic hallucinations often begin in childhood. Sleep paralysis usually occurs during mid adolescence. Cataplexy has a later onset. The relationship to HLA-DR2 has been demonstrated[61] but is not diagnostic. Sleep polysomnography may show early onset REM activity during night time recordings or during daytime multiple sleep latency tests (MSLT) in association with sleep attacks. Narcolepsy continues to be a clinical diagnosis. The clinician must ask specifically for these symptoms because they are rarely spontaneously volunteered by the child or adolescent.

Individuals fulfilling criteria for the primary disorder of vigilance or their relatives do not have the clinical manifestations of abnormal REM activity such as hypnogogic hallucinations, sleep paralyses and cataplexy. Also, their excessive day-time sleepiness is not associated with electrical REM activity. Whether or not narcolepsy, primary disorder of vigilance, and idiopathic hypersomnolance are a spectrum of one disorder or discrete entities remains unknown.

Treatment for narcolepsy is the stimulant medications [62,63] and for those with depression, protriptyline. On occasion other tricyclic antidepressants and a stimulant in combination are effective.

5. Task Dependent Attention Disorder (Developmental Specific Learning / Communication Disorders)

For a child with learning disabilities, school is an unpleasant, often noxious environment. The brain has great capacity to remove itself from unhealthy environments if allowed. Therefore, it stands to reason that children and adolescents with various specific learning disabilities will manifest poor attention, distractibility, day dreaming, restlessness, and limited diligence in tasks requiring use of weak facilities. Based upon the work of Sherrington[64], select impairment of cortical functioning in response to an epileptic focus[65], and well described focal and neurological disorders in adults[66], we have postulated that continuous stimulation of a developmentally (genetic) different or damaged area of brain makes that area, the surrounding cortex, and its' homologous area function less well[12]. Language impaired and dyslexic children (left brain) become moody and irritable when asked to speak, read or recall words and some will cry (right brain). On occasion some will express hostile anger and become oppositional (left brain). In our experience it

is common for poor spellers practicing each evening for the Friday spelling test to become increasingly dysphoric, anxious and disattentive (right brain) as the year progresses. The subsequent conflict and failure has lead to physical abuse by parents. Commonly, the dyscalculic young girl becomes more disorganized with resultant overt depression (right brain). With the powerful influence of right brain, especially right posterior parietal cortex in attention (arousal), it is not surprising for learning disabled children to become progressively disattentive. This can be considered a good example of the interaction of environmental stress and biological dysfunction of the cerebral hemispheres[67] (indeed, a metabolic encephalopathy). Children with learning disabilities, even in their early school years, attempt to avoid focus on their deficits of communication, excessive time on task, drilling to improve weak symbol skills, and not beneficial repetition.

Disorders of learning and communication must be considered when ADHD has its onset at the start of school. A taxonomy and method of clinical evaluation for DSLD have been reported[11]. Left brain and its' dysfunctions of verbal and written language skills (dysphasia and dyslexia) are more readily identified. The right brain functions of prosody, order, praxias, humor and social competence are more historical and observational. Dysfunctions of the right brain[68-71] often go unrecognized. The two major areas of difficulty evidenced by children with right brain dysfunction are: order (seeing the world as a series of continuous events) and social competence (out of context in word usage and gestures, either overly expressive or underexpressive)[11,12]. Children manifesting dysfunctions of right brain also have a primary deficit in attention[72] (arousal).

The diagnostic criteria for task dependent disorder of attention are listed in Table 8. The treatment is removal of the inappropriate stress[11,73,74]. The most common inappropriate stress is the offering by educators of remediation, drill, repetition and excessive emphasis on the communicative skills of reading, spelling, writing and arithmetic. In junior high years, overloading this group of young people with reading or writing assignments, tasks requiring "fill in the blank" (naming and, more specifically, nominal recall), and tasks requiring high level organizational skills (ordering-sequencing) continue to promote "ADD without Hyperactivity". The treatment is bypass strategies: multi-media for input and output, multiple choice tests, calculator, and computer as a word processor for reports.

The clinician must avoid the inappropriate notion that more time on task, excessive emphasis, drill or various teaching-training programs will accelerate the development of symbol skills. There is absolutely no evidence to support those offerings[11]. Likewise, cortical anomalies are now being reported in developmentally dyslexic [75] and dysphasic brains[76]. Fundamental parietal lobe functions might well be genetic codes and develop at their own predetermined, predictable rate[12]. If education is the acquisition and utilization of meaningful information, the identification and assistance in the pursuit of one's talents and assets, and culturing socialization skills, then bypass strategies can be implemented in the regular classroom. Clinically, this resolves "ADD without hyperactivity" in otherwise normally healthy, vigorous brains.

6. Conduct Disorder

Conduct disorder covers a heterogeneous group of children and adolescents who manifest persistent violation of age appropriate rules

with or without aggressive behavior. Their attentional problems arise from persistent rule breaking with disruptive behavior. In a recent population study of children examined for the presence of affective disorder, conduct disorder, ADD and anxiety disorders, 53% of children with ADD fulfilled DSMIII criteria for conduct disorder[9].

Does sociopathy manifest itself in childhood and early adolescent years? Using Research Diagnostic Criteria (RDC)[77], primary sociopathy cannot be recognized in this young age group. However, there are children who seemingly have very little to no regard or intention to abide by age appropriate rules and regulations. They are unable to form a meaningful and bonded relationship with others and are often cruel to animals. Their misdeeds become more disruptive and destructive with aging. Interestingly, this group of children and adolescents manifest no problems with reading or spelling, attention or vigilance or other temporal-parietal lobe functions. This suggests primary malfunctioning of the frontal lobes, right possibly more than left[78,79,80]. Neurologically, these childrens findings are quite normal as a group but have lowered tolerance for pain and discomfort. They are excessively modest and difficult to physically examine. Attention deficit disorder with or without hyperactivity is primarily volitional and the family history of these children is strongly positive for characterological disorders.

A second group of inattentive and impulsive children with conduct disorder are undersocialized children. These children often come from more chaotic environments or under-ruled environments. They present with histories of repeated early abuse or neglect, multiple caretakers but inadequate or absent parenting. On entrance to school, a highly rule-oriented setting, the symptom complex of ADHD is what is recognized. Treatment of undersocialized children is the continuation of consistent

limits both at home and school and identification of treatable conditions in the child and primary caretakers. These children are in need of structured, consistent, rule-orientated environments in order to learn social skills.

Thirdly, there is clearly a group of secondary conduct disorders. These are children and adolescents who only manifest behavioral disturbance during episodes of affective illness. Puig-Antich[46] reports that in 43 prepubertal boys with major depression 16 (37%) also met criteria for conduct disorder. Thirteen of these sixteen were treated with imipramine and 11 evidenced improvement in both depressive symptoms and conduct disorder. In a longitudinal study of depression, comorbid conduct disorder was present in 23% and in most cases occurred as a complication of depression. Depressed girls who also had attention deficit disorder seemed at higher risk for developing conduct disorder during the study period. Several subjects with depression and episodic conduct disorder subsequently developed bipolar disorder[14]. It is possible that in some, episodic conduct disorder, ADD and affective instability is a precursor of manic-depressive disease. Children with secondary conduct disorders will have a positive family history for affective illness, alcoholism and either or both sociopathy and hysteria (depressive spectrum biogeny). The trait for sociopathy seemingly is being induced by a treatable disease.

In summary, in the great majority of children presenting with misconduct, one must search for treatable causes, eg, depression, mania and learning disabilities. In these young people conduct disorder is a secondary phenomenon.

7. Acquired Focal Neurological Deficits

Impairment in directed selective attention as a neglect syndrome in

children with right hemisphere lesions has been reported [81]. A small group of children with subtle but definite right hemisphere motor findings and attention deficit disorder demonstrated neglect [72]. Neglect is more evidently associated with right hemisphere dysfunction, particularly right posterior parietal lobe lesions[58]. It is possible that neglect is a manifestation of hypoarousal of the right posterior cerebral hemisphere. In addition, inattention and poor school performance as a phenomenon of primary deficiency in planning, organization and motivation suggesting frontal lobe dysfunctioning has been reported[82]. This supports the adult case of similar findings by Eslinger and Damasio[83].

Treatment of these focal deficits are their recognition and the development of creative ways to circumvent requirements and expectations of attention. This group probably will function best in the "free field" allowing their other abilities to operate in an independent manner.

CONCLUSION

The label ADHD engulfs a heterogeneous group of children and adolescents. ADHD is frequently a manifestation of affective illness[84], primary disorder of vigilance (PDV) and/or DSLD ("asking brain to do what it is not so gifted to do"). The clinician must examine the young patient for: (a) depression and mania utilizing well established criteria; (b) communication and developmental specific learning disorders; and (c) ability to stay alert and awake in tasks requiring contin us mental performance. Methylphenidate and pemoline, often prescri. e an automatic response by the clinician to children doing poorly in school, can lower hypomanic symptoms and improve vigilance but worsen or induce depression.

Our bias considers depression, the primary disorder of vigilance and communication problems with order and prosody to be right hemisphere dysfunctions with these problems often interacting negatively with each other. Mania might be a left hemisphere disorder, probably left mesial temporal cortex or left limbic system.

Prognosis for children and adolescents with primary affective illness should be good with appropriate management. Long-term continuous treatment might be indicated for some of these young people. It is important to offer cognitive counseling described as learning to use intelligence to override emotions: "Actions must dictate feelings rather than feelings promoting actions". Important is the consideration that basic school skills, order, and naming might be genetic codes that will develop with aging and at their own rate. The goals of education should be the acquisition and utilization of meaningful information, assisting the individual in coming to know his or her assets and pursuing those assets and talents, and culturing socialization within the individual. With these educational goals there should be noted less inappropriate use of stimulant medications for task-driven attention deficit disorder.

In examining a child in the four major areas: (a) mood and affect, (b) learning and communication, (c) alertness and awakefullness (vigilance), and (d) conduct, it is common for an individual child to manifest problems in more than one area. At the time of initial examination, most children will meet criteria for more than one disorder. Only through ongoing observation will one be able to determine which condition or conditions are clinically significant. Delineating the clinically significant condition should allow the understanding of prognosis with appropriate anticipation and management.

References

1. Weinberg W, Emslie G: Attention deficit disorder: A form of childhood depression or other disorders of brain. Int Pediatr 1987; 2:135-145.
2. Bradley C: The behavior of children receiving benzedrine. Am J Psychiatry 1937; 94:577-585.
3. Strauss AA, Lehtinen LE: Psychopathology and Education of Brain-injured Child. New York, Grune and Stratton Inc, 1947.
4. Clements SD, Peters JE: Minimal brain dysfunction in the school-age child. Arch Gen Psychiatry 1962; 6:185-197.
5. Menkes M, Rowe J, Menkes J: A twenty-five year follow-up study on the hyperkinetic child with minimal brain dysfunction. Pediatrics 1967; 39:393-399.
6. Stewart M, Pitts, F, Jr., Craig A, Dieruf W: The hyperactive child syndrome. Am J Orthopsychiatry 1966; 35:861-867.
7. Silver LB: Controversial approaches to treating learning disabilities and attention deficit disorder. Am J Dis Child 1986; 140:1045-1052.
8. Shaywitz SE, Shaywitz BA: Attention deficit disorder: current perspectives. Pediatr Neurol 1987; 3:129-135.
9. Bird HR, Canino G, Rubio-Stipec M, et al: Estimates of the prevalence of childhood maladjustment in a community survey in Puerto Rico. Arch Gen Psychiatry 1988; 45:1120-1126.
10. Costello EJ, Costello AJ, Edelbrock C, et al: Psychiatric disorders in pediatric primary care. Arch Gen Psychiatry 1988; 45:1107-1116.
11. Weinberg WA, McLean A: A diagnostic approach to developmental specific learning disorders. J Child Neurol 1986; 1:158-172.

12. Weinberg WA, Emslie GJ: Adolescents and school problems: Depression, suicide and learning disorders, in Feldman RA, Stiffman AR (eds): Advances in Adolescent Mental Health, Vol 2: Depression and Suicide. Greenwich, Conn, JAI Press, 1988, pp 181-205.

13. Kovacs M, Feinberg TL, Crouse-Novak MA, et al: Depressive disorders in childhood. I. A longitudinal prospective study of characteristics and recovery. Arch Gen Psychiatry 1984; 41:229-234.

14. Kovacs M, Paulauskos S, Gatsonis C, Richards C: Depressive disorders in childhood III. A longitudinal study of comorbidity with and risk for conduct disorder. J Affective Disorders 1988; 15:205-217.

15. Weinberg WA, Rutman A, Sullivan J, et al: Depression in children referred to an educational diagnostic center: Diagnosis and treatment. J Pediatr 1973; 83:1065-1073.

16. Brumback RA, Weinberg WA: Relationship of hyperactivity and depression in children. Percept Mot Skills 1977; 45:247-251.

17. Weinberg WA, Rehmet A: Childhood affective disorder and school problems, in Cantwell DP, Carlson GA (eds): Affective Disorders In Childhood and Adolescence: An Update. Jamaica, NY, Spectrum Publications, 1983, pp 109-128.

18. Herjanic B, Reich W: Development of a structured psychiatric interview for children: agreement between child and parent on individual symptoms. J Abnorm Child Psychol 1982; 10(3):307-324.

19. Reich W, Herjanic B, Welner A, Gandby P: Development of a structured psychiatric interview for children. Agreement on diagnosis comparing child and parent interviews. J Abnorm Child Psychol 1982; 10:325-336.

20. Kelly PC, Cohen ML, Walker WO, et al: Self-esteem in children medically managed for attention deficit disorder. Pediatrics 1989; 83:211-217.

21. Trommer BL, Hoeppner JB, Lorber R, Armstrong KJ: The go-no-go paradigm in attention deficit disorder. Ann Neurol 1988; 24:610-614.

22. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders. Third Edition. Washington, DC: American Psychiatric Association, 1980.

23. Weinberg WA, McLean A, Snider RL, et al: Depression, learning disability, and school behavior problems. Psychol Rep 1989; 64:275-283.

24. Brumback RA, Staton RD, Wilson H: Right cerebral hemisphere dysfunction. Arch Neurol 1984; 41:248-250.

25. Brumback RA, Staton RD: Learning disability and childhood depression. Am J Orthopsychiatry 1983; 53:269-281.

26. Zrull J, McDermott J, Poznanski E: Hyperkinetic syndrome: The role of depression. Child Psychiatry and Hum Dev 1970; 1:33-40.

27. Carlson GA, Cantwell DP: Diagnosis of childhood depression: A comparison of the Weinberg and DSM-III criteria. J Am Acad Child Psychiatry 1982; 21:247-250.

28. Poznanski EO, Mokros HB, Grossman J, et al: Diagnostic criteria in childhood depression. Am J Psychiatry 1985; 142:1168-1173.

29. Emslie GJ, Weinberg WA, Rush AJ, et al: Depression and dexamethasone suppression testing in children and adolescents. J Child Neurol 1987; 2:31-37.

30. Feighner JP, Robbins E, Guze SP, et al: Diagnostic criteria for use in psychiatric research. Arch Gen Psychiatry 1972; 26:57-63.

31. Weinberg W, Emslie G: Weinberg screening affective scales (WSAS and WSAS-SF). J Child Neurol 1988; 3:294-296.

32. Winokur G: Depressive spectrum disease: Description and family study. Comprehensive Psychiatry 1972; 13:3-8.

33. Emslie GJ, Roffwarg HP, Rush AJ, et al: Sleep EEG findings in depressed children and adolescents. Am J Psychiatry 1987; 144:668-670.

34. Freeman RL, Galaburda AM, Cabal RD, Geschwind N: The neurology of depression: cognitive and behavioral deficits with focal findings in depression and resolution after electroconvulsive therapy. Arch Neurol 1985; 42:289-291.

35. Brumback RA, Staton RD: An hypothesis regarding the commonality of right hemisphere involvement in learning disability, attentional disorder and childhood major depressive disorder. Percep Mot Skills 1982; 55:1091-1097.

36. Ross ED, Rush AJ: Diagnosis and neuroanatomical correlates of depression in brain-damaged patients. Arch Gen Psychiatry 1981; 38:1344-1354.

37. Lenhart RE, Katkin ES. Cerebral lateralization in depression (Reply to letter to the editor). Am J Psychiatry 1986; 143:1631-1632.

38. Bolla-Wilson K, Robinson RG, Starkstein SE, Boston MA, Price TR: Lateralization of dementia of depression in stroke patients. Am J Psychiatry 1989; 146:627-634.

39. Robinson RG, Bolla-Wilson K, Kaplan E, Lipsey JR, Price TR: Depression influences intellectual impairment in stroke patients. Brit J Psychiat. 1986; 148:541-547.

40. Lipsey JR, Robinson RG, Pearlson GD, Rao K, Price TR: Mood change following bilateral hemisphere brain injury. Brit J Psychiat 1983; 143:266-273.

41. Hollister LE: Drug therapy: Tricyclic antidepressants. N Engl J Med 1978; 229:1106-1109, 1168-1171.

42. Camfield CS, Chaplin S, Doyle A-B, et al: Side effects of phenobarbital in toddlers: behavioral and cognitive aspects. J Pediatr 1979; 95:361-365.

43. Brent D, Crumrine P, Varma R, Allan M, Allman C: Phenobarbital treatment and major depressive disorder in children with epilepsy. Pediatrics 1987; 80:909-917.

44. Weinberg WA: Epilepsy and interictal behavior disorders in children and adolescents. Int Pediatr 1987; 2:196-204.

45. Ling W, Oftedal G, Weinberg WA: Depressive illness in childhood presenting as severe headache. Am J Dis Child 1970; 120:122-124.

46. Puig-Antich J: Major depression and conduct disorder in prepuberty. J Am Acad Child Psychiatry 1982; 21:118-128.

47. Kashani JH, Carbon GA, Beck NC, et al: Depression, depressive symptoms and depressed mood among a community sample of adolescents. Am J Psychiatry 1987; 144:931-934.

48. Weller EB, Weller RA, Preskorn SH: Depression in children: Effects of antidepressant therapy. J KS Med Soc 1983; 83:117-119.

49. Geller B, Cooper TB, Chestnut E, et al: Nortriptyline pharmacokinetic parameters in depressed children and adolescents: preliminary data. J Clin Psychopharmacol 1984; 4:265-269.

50. Weinberg WA, Brumback R: Mania in childhood: Case studies and literature review. Am J Dis Child 1976; 130:380-385.

51. Akiskal HA, Down J, Jordan P, et al: Affective disorders in referred children and younger siblings of manic-depressives. Arch Gen Psychiatry 1985; 42:996-1003.

52. Strober M, Morrell W, Burroughs J, Lampert C, Danforth H, Freeman R: A family study of bipolar disorder in adolescence. J Affective Disord. 1988; 15:255-268.

53. Brumback RA, Weinberg WA: Mania in childhood. II. Therapeutic trial of lithium carbonate and further description of manic-depressive illness in children. Am J Dis Child 1977; 131:1122-1126.

54. Delong R: Lithium carbonate treatment of select behavior disorders in children suggesting manic-depressive illness. J Pediatr 1978; 26:389-394.

55. Delong R, Aldershof A: Long-term experience with lithium treatment in childhood: correlation with clinical diagnosis. J Amer Acad Child Adol Psychiat. 1987; 26:389-394.

56. Davies DR, Parasuraman R: The Psychology of Vigilance. New York, Academic Press Inc. (London) LTD, 1981.

57. Mesulam MM: A cortical network for directed attention and unilateral neglect. Ann Neurol 1981; 10:309-325.

58. Heilman KM, Watson RT, Valenstein E: Neglect and related disorders, in Heilman KM, Valenstein E (ed): Clinical Neuropsychology. New York, Oxford University Press, 1985, pp 234-294.

59. Zentall S: Optimal stimulation as theoretical basis for hyperactivity. Am J Orthopsychiatry 1975; 45:549-563.

60. Douglass AB, Harris L, Pazderka F: Monozygotic twins concordant for the narcoleptic syndrome. Neurology 1989; 38:140-141.

61. Lenn NJ: HLA-DR2 in childhood narcolepsy. Pediatr Neurol 1986; 2:314-315.

62. Yoss RE, Daly D: Treatment of narcolepsy with Ritalin. Neurology 1959; 9:171-173.

63. Honda Y, Hishkawa YA: Long-term treatment of narcolepsy and excessive daytime sleepiness with pemoline. Curr Ther Res 1980; 27:429-441.

64. Sherrington CS: The Integrative Action of the Nervous System. New Haven, University Press, 1906.

65. Williamson PD, Spencer DD, Spencer SS, et al: Episodic aphemia and epileptic focus in nondominant hemisphere: Relieved by section of corpus callosum. Neurology 1985; 35:1069-1071.

66. Musiek FE, Reeves AG, Baran JA: Release from central auditory competition in the split-brain patient. Neurology 1985; 35:983-987.

67. Gold PW, Goodwin FK, Chrousos GP: Clinical and biochemical manifestations of depression: relation to the neurobiology of stress. N Engl J Med 1988; 319:348-413, 413-420.

68. Weinbtraub S, Mesulam MM: Developmental learning disabilities of the right hemisphere: Emotional, interpersonal, and cognitive components. Arch Neurol 1983; 40:463-468.

69. Denckla MB. The neuropsychology of social-emotional learning disabilities. Arch Neurol 1983; 40:461-462.

70. Voeller KKS: Right-hemisphere deficit syndrome in children. Am J Psychiatry 1986; 143:1004-1009.

71. Voeller KKS, Henson JA, Wendt RN: Facial affect recognition in children: a comparison of the performance of children with right and left hemisphere lesions. Neurology 1988; 38:1744-1748.

72. Voeller KKS, Heilman KM: Attention deficit disorder in children: a neglect syndrome? Neurology 1988; 38:806-808.

73. Silberberg N, Silberberg M: The bookless curriculum: an educational alternative. J Learn Dis 1969; 2:302-307.

74. Silberberg N, Silberberg M: Myths in remedial education. J Learn Dis 1969; 2:34-42.

75. Galaburda AM, Sherman GF, Rosen GD, et al: Developmental dyslexia: four consecutive patients with cortical anomalies. Ann Neurol 1985; 18:222-233.

76. Cohen M, Campbell R, Yaghami F: Neuropathological abnormalities in developmental dysphasia. Ann Neurol 1989; 25:567-570.

77. Spitzer RL, Endicott J, Robins E: Research diagnostic criteria: rationale and reliability. Arch Gen Psychiatry 1978; 35:773-782.

78. Lhermitte F, Pillon B, Serdaru M: Human autonomy and the frontal lobes. Part 1: Initiation and utilization behavior; a neuropsychological study of 75 patients. Ann Neurol 1988; 19:326-334.

79. Lhermitte F: Human anatomy and the frontal lobes. Part II: Patient behavior in complex and social situations: the "environmental dependency syndrome". Ann Neurol 1986; 19:335-343.

80. Mesulam MM: Frontal cortex and behavior. Ann Neurol 1986; 19:320-324.

81. Ferro JM, Matins IP, Tavora L: Neglect in children. Ann Neurol 1984; 15:281-284.

82. Joseph J: Learning disability due to a primary deficiency in planning, organization, and motivation. Neurology 1987; 37(Suppl 1):221.

83. Eslinger P, Damasio A: Severe disturbance of higher cognition after bilateral frontal lobe ablation: patient EVR. Neurology 1985; 35:1731-1741.

84. Nieman G, Delong R: Use of the personality inventory for children as an aid in differentiating children with mania from children with attention deficit disorder with hyperactivity. J Amer Acad Child Adol Psychiat. 1987; 25, 3:381-388.

TABLE 1

ATTENTION DEFICIT HYPERACTIVITY DISORDER

DIFFERENTIAL DIAGNOSIS

1. DEPRESSION

2. MANIA

3. NARCOLEPSY

4. PRIMARY DISORDER OF VIGILANCE

5. TASK DEPENDENT

6. CONDUCT DISORDER

7. ACQUIRED FOCAL NEUROLOGICAL DEFICITS

   A. NEGLECT

   B. INATTENTION

TABLE 2

PREVALENCE OF ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD), DEVELOPMENTAL SPECIFIC LEARNING DISORDERS (DSLD), AND DEPRESSION (DP). (N = 100)

DIAGNOSIS	N
ADHD - Alone	4
DSLD - Alone	9
DP - Alone	11
ADHD + DSLD	13
ADHD + DP	6
DSLD + DP	17
ADHD + DSLD + DP	40
TOTAL	100

SUMMARY

	N/100
TOTAL ADHD	63/100
TOTAL DSLD	79/100
TOTAL DP	74/100

TABLE 3

CONSECUTIVELY REFERRED NORMALLY INTELLIGENT

LEARNING DISABLED PREPUBERTAL CHILDREN (N = 223)[16]

"HYPERACTIVE" CHILDREN		
TOTAL HYPERACTIVES:	117/223	(52.5%)
NOT DEPRESSED	31/117	(26.0%)
POSITIVE FOR DEPRESSION	86/117	(74.0%)
HYPERACTIVE ONLY WITH DEPRESSION	64/117	(55.0%)
"DEPRESSED" CHILDREN		
TOTAL WITH DEPRESSION:	136/223	(61.0%)
NOT HYPERACTIVE	50/136	(37.0%)
HYPERACTIVE	86/136	(63.0%)
SUMMARY		
HYPERACTIVITY PLUS DEPRESSION	86/223	(38.5%)

TABLE 4

Comparison of Criteria for Primary Depression in Children and Adults

(adapted from Weinberg et al[15], Emslie et al[29], and Feighner et al[30])

Childhood Depression	Adult Depression
A. The ten symptoms and the characteristic behaviors for each symptom. Presence of both symptoms I and II and four or more of the remaining eight symptoms (III-X). I. Dysphoric Mood Statements of sadness, loneliness, unhappiness, hopelessness and/or pessimism. Mood swings, moodiness Irritable, easily annoyed Hypersensitive, cries easily Negative, difficult to please II. Self-deprecatory Ideation Feelings of being worthless, useless dumb, stupid, ugly, guilty Beliefs of persecution Death wishes Suicidal thoughts Suicidal attempts III. Agitation Difficult to get along with Quarrelsome Disrespectful of authority Belligerent, hostile, agitated Excessive fighting or sudden anger IV. Sleep Disturbance Initial insomnia Interval insomnia Terminal insomnia Difficulty awakening in morning V. A Change in School Performance Frequent complaints from teachers ("daydreaming", "poor concentration", "poor memory") Loss of usual work effort in school subjects Loss of usual interest in non-academic school activities Many incomplete classroom assignments Much incomplete homework A drop in usual grades Finds homework difficult	A. Presence of symptom 1 and five or more of the remaining eight symptoms (2-9). 1. Dysphoric mood (depressed, sad, blue, despondent, hopeless, "down in the dumps", irritable, fearful, worried, discouraged). (I,II)* 2. Poor appetite or weight loss (positive if 2 or more pounds a week or 10 pounds a year when not dieting. (X) 3. Sleep difficulty (including insomnia or hypersomnia). (IV) 4. Loss of energy, e.g., fatigability, tiredness. (IX) 5. Agitation (III) or retardation. (IX) 6. Loss of interest in usual activities (VI,VII) or decrease in sexual drive. 7. Feeling of self-reproach or guilt (may be delusional). (II) 8. Complaints of or actually diminished ability to think or concentrate, such as slowed thinking or mixed-up thoughts (V) 9. Recurrent thoughts of death or suicide, includes thoughts of "wishing to be dead". (II) * Roman Numerals correspond to criteria symptoms for Childhood Depression.

TABLE 4 (Continued)

Childhood Depression - Continued

VI. Diminished Socialization
Less group participation
Less friendly; less outgoing
Socially withdrawing
Loss of usual social interests

VII. Change in Attitude Toward School
Does not enjoy school activities
Does not want or refuses to attend school.

VIII. Somatic Complaints
Non-migraine headaches
Abdominal pain
Muscle aches or pains
Other somatic concerns or complaints

IX. Loss of Usual Energy
Loss of usual personal interests or pursuits (other than school, e.g., hobbies, sports)
Decreased energy; mental and/or physical fatigue

X. Unusual Change in Appetite and/or Weight
Anorexia or polyphagia
Unusual weight change in past 4 months.

B. Interview of patient and historian(s) is conducted utilizing a semi-structured, closed-end technique.

C. A symptom is accepted as positive when at least one of the characteristic behaviors listed for the category is present.

D. Symptoms I & II must be reported by the patient for it to be considered positive. Symptoms III - X can be reported by either patient or historian to be considered positive.

E. Each symptom must be discrete change in usual self (a new behavior or worsening of an old behavior). The symptom complex must be present for more than one month and associated with a change to maladaptation.

Adult Depression - Continued

B. Must be discrete psychiatric illness lasting at least one month with no pre-existing psychiatric conditions.

C. Patients with life-threatening or incapacitating medical illness preceding or paralleling the depression are excluded from the diagnosis of primary depression

TABLE 5

Summary of Pharmacologic Differences Among Six Tricyclic Antidepressants[41].

Drug	Sedation	Anticholinergic Effects	Block of Amine Pump For Serotonin	Block of Amine Pump For Norepinephrine
Imipramine	++	++	++	++
Amitriptyline	+++	+++	+++	+
Desipramine	+	+	0	+++
Nortriptyline	++	++	+	++
Doxepin	+++	+++	Weak	
Protriptyline	0	++	Not Known	

0 indicates none, + slight, ++ moderate, & +++ high.

TABLE 6

MANAGEMENT OF CHILDREN AND ADOLESCENTS WITH AFFECTIVE ILLNESS[12]

I. Individual, Family and Environmental Counselling

1) Remove Inappropriate Stressors: use bypass strategies; demands and tasks in keeping with the child/adolescent's facilities.

2) Informative: emphasis on genetics, biology and maturation with potential cycles.

3) Educative; emphasis on what is known and not known; avoid rationalization and misinformation.

4) Supportive: be a positive advocate.

5) Reassuring: a treatable and self limiting condition with anticipation of long periods of well states.

6) Assist With Order and Planning: towards school, work, play and pursuit of assets and talents.

7) Assist with decision making: "Continue Usual Pursuits" / "Do Not Drop-Out".

8) Cognitive Coaching on a "Mini" Daily Basis: "Learn to Think Positive ---- Act Positive:" "Come to know that actions should dictate feelings;" "Intelligence should overrule emotions".

II. Psychopharmacological Treatment

1) Tricyclic antidepressants (TCADs): amitriptyline, imipramine, desipramine, nortriptyline, protriptyline, doxepin.

2) thioridazine (and rarely haloperidol - other major tranquilizers are not presently being used).

3) carbamazepine

4) lithium

Table 7

Comparison of Criteria for Mania in Children and Adults

(adapted from Weinberg and Brumback[50] and Feighner, et al[30])

A. The presence of either or both symptoms 1 and 2 and three or more or the remaining six symptoms (3-8)

1. Euphoria
   a. Denial of problems or illness; &/or
   b. Inappropriate feelings of well-being; inappropriate cheerfulness; giddiness and silliness
2. Irritability and/or agitation (particularly belligerence, hostile anger, destructiveness, and anti-social behavior)
3. Hyperactivity, "motor driven", intrusiveness, interruptiveness
4. Push of speech (may become unintelligible), garrulousness
5. Flight of ideas (racing thoughts
6. Grandiosity (may be delusional)
7. Sleep disturbance (decreased sleep and unusual sleep pattern)
8. Distractibility, shortened attention span, and inability to concentrate.

B. Interview of patient and historian(s) is conducted utilizing a semi-structured, closed-end technique.

C. A symptom is accepted as positive when at least one of the characteristic behaviors listed for the category is present.

D. Symptom (1) must be reported by the patient. Symptoms (2-8) are observational and reported by the historian(s).

E. Each symptom must be discrete change in usual self (a new behavior or worsening of an old behavior). The symptom complex must be present for more than two weeks and associated with a change to maladaption.

A. The presence of either or both symptoms 1 and 2 and three or more of the other six symptoms (3-8)

1. Euphoria
2. Irritability
3. Hyperactivity - may be social, motor, sexual
4. Push of speech (pressure to keep talking)
5. Flight of ideas (racing thoughts)
6. Grandiosity (may be delusional)
7. Decreased sleep
8. Distractibility

B. A discrete psychiatric illness lasting two weeks with no other preexisting psychiatric condition

Table 8

DIAGNOSTIC CRITERIA FOR

TASK DEPENDENT DISORDER OF ATTENTION

Asking brain to do that which it is unable to do: Developmental specific learning disorders (DSLD).

Good select attention

Good continuous mental performance and good continuous task performance in areas of brain talents, interest, and assets.

Attention is a problem or concern primarily with school tasks and associated with a clinically determined DSLD

Summation: WHO TEACHES BIRDS TO FLY?