

Chapter 15

Depression and Other Affective Illnesses as a Cause of School Failure and Maladaptation in Learning Disabled Children, Adolescents, and Young Adults

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Introduction

Depression is a leading cause of failure in school and in life for young people (children, adolescents, and young adults) with learning disabilities. The depressed state can be primary (familial-genetic disorder) or secondary (symptomatic depression) to medical conditions or learning disability.

Depression is feeling sad, lonely, unloved, dumb, and worthless, along with guilt feelings and beliefs of being mistreated. Depression is having trouble falling asleep (initial insomnia), restless sleep with recurrent awakenings (interval insomnia), or waking early in the morning unable to fall back to sleep (terminal insomnia). Depression often is also awakening in the morning anxious, tired, sad, or mad. Depression is a loss of interest in friends, hobbies, and school or work. Depression causes a loss of concentration, poor memory, forgetfulness, and inattention. Depression is having headaches, stomachaches, and other physical symptoms. Depression is feeling angry, picked on, grouchy, and irritable. Sometimes depression is feeling hopeless and helpless, and wishing to die. Depression is the leading cause of suicide. A clinical depression is a combination of these symptoms which persist for longer than three weeks and cause failure in the person's environments of home, work (school), or play. Depression has been demonstrated in many studies to be associated with and the possible cause of school failure, conduct disorder and delinquency, anorexia and bulimia, school phobia, and panic attacks.

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The scientific description of depression and manic-depressive disease was first prepared by Emil Kraepelin in the early 1900s. For the first half of the twentieth century, most clinicians did not accept the concept that primary depression (or manic-depressive disease: affective illness) occurred in children and adolescents. However, by the early 1970s, criteria for the recognition of depression in children and adolescents had been established (Table 1, "The Weinberg Criteria"). These criteria were based upon those used to diagnose depression and mania in adults as formulated in the Research Diagnostic Criteria (RDC) for adult psychiatric disorders by the Department of Psychiatry of the Washington University School of Medicine in St. Louis, Missouri. The Weinberg Criteria for depression in children and adolescents was published in 1973 and the criteria for mania in 1976 by Weinberg and Brumback (Table 2). In 1980, the American Psychiatric Association published the Diagnostic and Statistical Manual of Mental Disorders - Third Edition (DSM-III) [revised in 1988 as DSM-III-R and in 1994 as DSM-IV] which contained guidelines for the diagnosis of depression and mania in individuals of all ages. In the DSM-III, diagnostic criteria were added for the various temporal patterns of affective illness. These patterns are characterized by chronic, long-standing ebbs and flows, "good" and "bad" periods per day, mixed with "good" and "bad" days, but without prolonged well (stable) states. Patterns of affective illness with prominent manic symptomatology (chronic hypomania, cyclothymia, juvenile rapid cycling bipolar disorder and dysthymia with the manic feature of hostile anger) are most prevalent in pre-school years and continue through young adult age.

Depression and other forms of affective illness are probably genetic disorders based upon studies of familial clusterings, evaluation of offspring of both depressed and manic-depressive adults, and studies of

siblings and twins reared apart. Research is now underway to determine whether the various patterns of affective illness represent a unitary genetic disorder modified by other traits or are the result of multiple and varied genetic and psychosocial causes. It is possible that even genetically-based affective illness could remain dormant throughout an individual's life if inappropriate environmental stressors are not present.

Criteria For Depression In Young People

The "Weinberg Criteria" for depression (Table 1), include 10 major symptom categories: dysphoric mood, self-deprecatory ideation, agitation, sleep disturbance, change in school performance, diminished socialization, change in attitude towards school, somatic complaints, loss of usual energy, and unusual change in appetite or weight. The individual must manifest: 1) dysphoric moods and affect and 2) self-deprecatory ideation. We use the term mood to describe the young person's emotional feelings, while the term affect indicates the individual's appearance in relation to feelings. Symptoms of dysphoric mood and affect are sadness, loneliness, unhappiness, hopelessness, pessimism, mood swings, moodiness, irritability, easily annoyed, hypersensitivity, tearfulness and crying, negativity, and difficulty being pleased. Generally, a child manifesting dysphoric moods will have a multitude of these symptoms. Sadness and hopelessness can be so severe that the young person will withdraw to a room to sit and cry (melancholy). Self-deprecation is the feeling of being worthless, useless, dumb, stupid, and guilty, or beliefs of persecution. Death wishes occur in 35% of pre-pubertal children manifesting depression, suicidal ideation (thoughts and plans) in 15%, and suicidal attempts in 5%.

In addition to dysphoric moods and self-deprecation, the child must have four or more of the following eight symptoms:

- agitation
- sleep disturbance
- change in school performance
- diminished socialization
- change in attitude toward school (work)
- somatic complaints
- loss of usual energy
- unusual change in appetite or weight

Most young people fulfilling this criteria will have seven or eight of the ten major symptoms.

The agitation of depression is irritability, grouchy feelings and actions, inappropriate sudden anger or fighting, quarrelsomeness, lack of respect for authority, and difficulty getting along with others. The most

common sleep disturbance is trouble falling asleep (initial insomnia). The young child will often manifest restless sleep with frequent awakenings (interval insomnia) and enter the parents bed for comfort. Awakening too early in the morning (terminal insomnia) strongly correlates with thoughts of suicide. Common to the depressed adolescent is difficulty awakening in the morning with excessive morning sleep. Change in school performance is characterized by frequent complaints from the teachers of: daydreaming, poor concentration and memory, and inattentiveness; loss of usual work effort in school subjects with a drop in grades; loss of usual interest in extra-scholastic activities; incomplete classroom assignments; much incomplete homework; avoidance of homework activity; and disruptive behavior.

The depressed young person will often lose interest in friends and social activities. Occasionally the young person loses the ability to anticipate or experience pleasure (anhedonia). Commonly, the individual must be forced or coerced to participate in what previously were usual, fun activities, but the enjoyment is lessened and the participation does not restore interest (dyshedonia). School avoidance, school resistance, discomfort in school, and school phobia are symptomatic of depression. Somatic complaints (physical complaints) of headaches, stomachaches, joint and muscle aches and pains, and visual disturbances without any physical cause is depression until proven otherwise. The depressed young person complains of poor energy and tiredness. A change in appetite (either a loss in appetite or excessive desire to eat) associated with change in weight is less common in children than adults, but is still symptomatic of depression.

It is important to understand that depression, as a disease or illness, is a group of symptoms associated with maladaptation or worsening in usual performance in either the home, school, or play environments. Most depressed young people will be failing in school, will be difficult to live with at home, and will drop out of usual extra-scholastic pursuits. The depressed individual's judgment deteriorates, interests wane, and failure occurs.

Minor depressions are identical to major depressive episodes in duration but involve fewer symptoms and less impairment. The Weinberg Criteria for depression defines severity of a depressive episode in relation to death wishes and suicidal ideation: mild (no death wishes), moderate (positive for death wishes or suicidal thoughts), and severe (positive for suicidal plans or suicidal attempts).

Diagnosis of Depression

Using the Weinberg Criteria, the diagnosis of depression is established through interview of the individual and the primary caretaker. The criteria symptoms for depression must represent a change from the individual's usual self or a worsening of the usual self, be present for more than three weeks, and be associated with poor performance and a worsening of adaptation (or failure) in one, two, or three environments - home, school (work), or play. A symptom is accepted as positive when at least one of the characteristic behaviors listed for that symptom category is present (Table 1). Dysphoric moods and self deprecation (deteriorated self-esteem) must be reported by the young person for these symptoms to be considered positive. The other eight symptom categories can be reported by either the individual or the primary caretaker.

Petti (1978; 1985) developed the Bellevue Index of Depression (BID) from the Weinberg Criteria. The BID offers a systematic interview approach to both the young patient and the caretaker and can be used for diagnosis of depression and assessment of the severity of the depressed state. The Diagnostic Inventory for Children and Adolescents (DICA) developed by Herjanic and Reich (1982) is a structured interview based upon DSM-III criteria for depression and other psychiatric disorders. It is administered to both the patient and the caretaker. The Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS-P) developed by Puig Antich and Chambers (1978) is a structured interview modified for children based upon the Research Diagnostic Criteria (RDC) for depression and other psychiatric disorders in adults. The Children's Depression Rating Scale - Revised (CDRS-R) was developed by Poznanski and colleagues, in 1985, and modeled after the Hamilton Rating Scale for Depression for adults. It correlates with global ratings for depression.

Depression By Self-Report

It is possible to recognize young people at risk for depression through self-report instruments. The Weinberg Screening Affective Scale (WSAS) is a 56 item self-administered form requiring fourth grade reading ability (Appendix A) that takes less than five minutes to complete. The WSAS correlates with depressive illness in both outpatient and hospitalized depressed children and adolescents.

Two large populations of high school students have been studied for depression by self-report using the WSAS. The first study group consisted of 3,292 students in a large metropolitan urban school district,

and the second population had 1,298 students from a rural middle-size college community school district. These two population studies (in which 96% of the students participated) demonstrated that about 14% of high school students manifest depression by self-report. Gender and ethnic group differences were evident. Hispanic females had the highest percentage of depression by self-report (22%), while white males (8%) had the lowest. Black students, white females, and Hispanic males were intermediate in the prevalence of depression. Three percent (3%) of the students related suicidal thoughts ("I think a lot about killing myself") These studies concluded that depressive symptomatology is prominent in adolescents, large numbers of young people are actively depressed, and a significant number are at risk for suicide. The WSAS can be used for the screening of depression in young people and is a strong confirmation instrument in the clinical setting. Two other widely used self-report forms for depression are the Beck Depression Inventory (BDI) which has been used in adolescents and young adults and the Children's Depression Inventory (CDI) developed by Kovacs in 1987 for ages 7-17 years.

Dysthymia: Chronic or Long-standing Depressive Symptomatology

Dysthymia is characterized by chronic long-standing depressive symptomatology with ebbs and flows in severity, periods each day of depressive moods (mixed days), occasional fully-depressed days, and an occasional depression-free day; but no prolonged well states. Periods of major depression are common in individuals with dysthymic disorder. Dysthymia limits functioning, performance, and enjoyment, but is not associated with the same degree of impairment that characterizes minor or major depressive episodes.

The onset of the dysthymic pattern is usually during the pre-school years (ages 2-5 years), with major or minor depressive episodes occurring later in elementary and secondary school years. School phobia with the start of school can be the presenting symptoms of a major depressive episode in the anxious dysthymic young child. The learning disabled dysthymic child will often develop a major depressive episode during the third or fourth grade school years associated with the increased stress of school tasks.

Criteria for Mania in Young People

The hallmarks of mania (Table 2) are euphoria and hostile anger. Euphoria has two presentations: a) an elated mood with inappropriate feelings of well being, cheerfulness, giddiness, and silliness. However, it can also be b) the inappropriate denial of problems or

symptoms despite behavior that is obvious to observers as inappropriate, disruptive, and destructive. The manic young person can be hyperactive, intrusive, destructive, or agitated, or display hostile anger, while at the same time reporting, "I have no problems or concerns."

The irritability of mania is hostile anger, with ranting rages, driven impulsive destructiveness, or uncontrollable antisocial behavior. The hostile anger of mania can lead to homicidal wishes, thoughts, threats, or attempts.

Utilizing the criteria established by Weinberg and Brumback (1976) for the recognition of mania in children and adolescents, the individual must manifest either euphoria or hostile anger with three or more of the following six symptoms:

- hyperactivity (mental, motor, or sexual), motor drivenness, intrusiveness;
- push of speech;
- flight of ideas, racing thoughts, the inability to maintain thinking on one idea;
- grandiosity with heightened self-worth, self-power, and self-influence which can be delusional;
- sleep disturbance with decreased need for sleep and nighttime hyperwakefulness (night-owl insomnia);
- shortened attention span, inability to concentrate, and distractibility.

Each symptom must be a discrete change from the individual's usual self, either a new behavior or a worsening of an old behavior. Although the original criteria stated that this symptom complex must be present for more than one month, further studies indicate that a duration of only one week is necessary.

Diagnosis of mania is established by interview and observation of the young person and interview with the primary caretaker utilizing a semi-structured, closed-end interview technique. A symptom is accepted as positive when at least one of the characteristic behaviors listed for the category is present. Euphoria (most commonly denial of any problems) must be reported by the individual, but the other symptoms are observational and can be reported by the individual, the caretaker, or knowledgeable others.

The course of childhood onset classic manic-depressive disease is exemplified by the child with no previous disturbance of mood and affect who begins to manifest the symptom complex of depression. On a rare occasion, a manic episode will precede the first period of depression. Subsequently there are discrete periods of depression usually lasting six to eighteen months and

mania persisting for one to six weeks, with interspersed prolonged well states. Although classic manic-depressive disease is uncommon in pre-pubertal children, the patterns of dysthymia, hypomania, cyclothymia, and juvenile rapid cycling bipolar disorder often begin during pre-school years, and are mislabeled as attention deficit hyperactivity disorder (ADHD), conduct or oppositional defiant disorder, and over-anxious disorder. As the child ages, classic manic-depressive disease becomes evident.

Hypomania, Cyclothymia, And Juvenile Rapid Cycling Bipolar Disorder (See Glossary)

One of the confusing problems in the recognition and appropriate management of mania in children and adolescents is the observation that hypomania is chronic with a very early onset. Hypomania can present in utero as a hyperactive fetus; as overly alert, irritable, colicky infant with poor sleep habits; or as a "supermarket toddler" requiring restriction in the grocery store cart. The hypomanic child progresses through preschool years with significant hyperactivity, excessive cheerfulness, frequent sexual naughtiness, inappropriate silliness, giddiness, and elation, and nightowl insomnia. As the child ages, periodic rants, rages, hostile anger (mania), and even homicidal threats limit the hypomanic child's ability to function. Hypomania is a common cause of attention deficit disorder with persistent hyperactivity (ADHD): "hypomanic/hyperactivity." Increasing manic moods and classic manic-depressive disease become apparent as the child ages.

Cyclothymia (See Glossary)

Cyclothymia is long-standing (chronic) hypomania with moments per day of both depressive moods and "mini" moments of significant anger. Most of the days are "mixed days" with an occasional all "bad" day, but rarely an "all good day." The cyclothymic disorder begins in the toddler or early pre-school years and progresses during late childhood and young adolescent years to discrete periods of major depression or manic episodes.

Juvenile Rapid Cycling Bipolar Disorder (JRCBD) (See Glossary)

The term juvenile rapid cycling bipolar disorder (JRCBD) is applied to a group of manic-depressive young people who are free of continuous hypomanic/hyperactivity but manifest moment-to-moment, day-to-day ("all mixed days") of depressive moods, actions, and feelings interspersed with hostile anger, ranting, or rages. Juvenile rapid cycling bipolar disorder most often is evident during preschool years

and worsens with aging to major depressive or manic episodes during the adolescent years.

Family History

The pattern of affective illness in the young person predicts family history. If the pattern in the young person is discrete cycles of depression with or without a manic episode and with prolonged well states, the family history will only be positive for typical depression or classic manic-depressive disease. Dysthymia or cyclothymia in the young person predicts a family history of depression or manic-depressive disease along with alcoholism.

Depressed children, who are oppositional and defiant or have a conduct disorder, have a positive family history of sociopathy, or alcoholism, along with depression or manic-depressive disease. Hallucinations during depression indicate a family member with schizophrenia. Depressed children with prominent somatic complaints frequently have a female family member with Briquet's syndrome (a disorder characterized by severe hypochondriasis and often multiple operations without recognizable underlying medical disease). Inherited traits of these other disorders become apparent as additional symptoms during the depressive episodes in young people. When that young person is free of depression or manic-depressive episodes, those traits become inapparent. Thus, it is important to try to identify an underlying depression or manic-depressive state in any children presenting with an apparent conduct disorder, somatic complaints, or other behavior complexes.

It is also noteworthy that 30-40% of affectively ill children will have a biologic parent who is also affectively ill (most commonly depression) at the time the young person presents for initial evaluation.

The Neurology and Biology of Affective Illness in Young People

The neurologic examination is very important in both establishing and understanding the localization of depression in the brain and the influence of this metabolic (chemical) disorder of the brain on other brain functions. During a depressive episode the young person will often manifest neurologic abnormalities of tone, posture, or reflexes in the left arm and leg indicating dysfunction of the right side of the brain (right cerebral hemisphere). The manic young person will have similar neurologic abnormalities that involve the right arm and leg indicating dysfunction of the left side of the brain (left cerebral hemisphere). Depressed young people have a variety of problems that indicate

disturbances in right parietal lobe functions: they have impaired coordination and are clumsy (dyspraxias); handwriting deteriorates (dysgraphia); they have language skills ("math"), wit, and humor suffer. In mania, the individual demonstrates errors indicative of left cerebral hemisphere dysfunction: specific word finding (naming) difficulties, problems with clarity of speech, and abnormal verbalizations. During well states, these motor findings and disturbances of higher brain communicative-cognitive functions improve to the individual's more usual abilities. An important concept is that affective illness can cause a learning disability or worsen an underlying learning disability.

The right posterior parietotemporal cortex appears to be involved in the production of the dysphoric and vegetative symptoms of depression. The left mesial temporal and entorhinal cortex appears to be responsible for the key symptoms of mania: euphoria (denial) and hostile anger. It is likely that obsessions, compulsions, and oppositional defiant behavior are minor manifestations of the hostile anger of mania. It is prudent to mention that some research concerning adult stroke patients has implicated left frontal lobe lesions in depression and right frontotemporal lesions in mania. However, studies by Freeman and colleagues (1985) and the preponderance of studies reviewed by Brumback (1985: 1993), indicate that the right parietotemporal area plays a major role in the genesis of depression. Our ongoing observations of children and adolescents confirm this brain localization for depression.

Biological correlates for depression in young people are similar to those reported in adults (Table 3). The most reproducible have been abnormalities of sleep and disturbances in hypothalamic-pituitary hormonal activity (abnormal dexamethasone suppression test and blunted growth hormone and thyrotropin releasing hormone). The strongest biological correlate is the proven successful treatment of depression with certain specific medications. Originally, the monoamine (MAO) inhibitors, and for the past twenty five years, the tricyclic antidepressant medications (TCAs) have provided beneficial therapy. Both these agents augment the activities of the neurotransmitters norepinephrine and serotonin. Serotonin is produced by neurons in the raphe nuclei of the brain stem and travels up the axons of these neurons to the cerebral hemispheres, more right than left, where it is released into the synaptic clefts. Norepinephrine is produced by another group of nerve cells, the locus ceruleus, of the brain stem and also travels up axons to the cerebral hemispheres. In depressed states, there is a relative deficiency of these two neurotransmitters. The tricyclic antidepressants, amitriptyline (Elavil) and doxepin (Sinequan), block

the inactivation of serotonin thus allowing this neurotransmitter to be available for needed use by cerebral cortical nerve cells (Table 4). Desipramine (Norpramin) and protriptyline (Vivactil) block the inactivation of norepinephrine, while nortriptyline (Pamelor) and imipramine (Tofranil) prevent inactivation of both serotonin and norepinephrine. The second generation antidepressant trazodone (Desyrel) prevents inactivation of serotonin and maprotiline (Ludiomil) prevents inactivation of norepinephrine. The new third generation antidepressants sertraline (Zoloft), fluoxetine (Prozac), and paroxetine (Paxil) are very specific in preventing inactivation of serotonin. The underlying mechanisms leading to this relative deficiency of serotonin and norepinephrine in the cerebral cortex is unknown, but we believe that a dysfunction of modulating control from the right posterior temporoparietal cortex initiates a cascade of events causing altered activity of the key brain stem neurons and consequent deficiency of norepinephrine and serotonin release by their axons in the cerebral cortex.

The biology of mania (Table 3) is less well understood and cannot be explained by any available bioamine (neurotransmitter) theory. The major tranquilizers widely used to treat mania in adults block the effect of the neurotransmitter dopamine. However, the disorder of absent dopamine, e.g., Parkinson's disease, is not characterized by mania. (due to the severe progressive motor problems associated with the major tranquilizers, we actively avoid their use in young people). The two most successful drugs in the treatment and prevention of manic episodes, lithium carbonate and carbamazepine (Tegretol), have many different biological actions, none of which are unique to mania. Although, right limb motor abnormalities, suggesting left brain dysfunction, is a reproducible observation, various brain imaging studies have been inconclusive, possibly due to heterogeneous study populations.

Concurrent Diagnoses

Concurrent diagnoses (a combination of two or more diagnoses occurring in the same individual at the same point in time) are common in young people with affective illness. There is a high prevalence of specific learning disabilities, overanxious disorder, school phobia, and anorexia in depressed young people. Conduct disorder and oppositional defiant disorder are common in children with hypomania, cyclothymia, or juvenile rapid cycling bipolar disorder.

It has been reported (and we concur) that 35-45% of delinquent adolescents have affective illness prior to their incarceration for delinquent activity. In this same

context, drug-abusing, learning-disabled adolescents frequently have had major depressive episodes, chronic minor depression (dysthymia), or long-standing cyclothymia prior to initiating drug abusing behavior in adolescence. It has also been reported (and we concur) that at least 50% of the older adolescent and young adults committing suicide, with a prior history of drug abuse, had evidence of depression predating the abuse of drugs and suicide.

Nearly twenty years ago (1977), we reported that hyperactivity beginning after age two years was most often symptomatic of an underlying depression. Fifty-five percent (55%) of hyperactive pre-pubertal children were only hyperactive when in a state of depression (Table 5). The so-called "rebound" side effects and vegetative symptoms (loss of appetite, insomnia) attributed to stimulant medications used to treat hyperactive children are actually the result of an underlying depression.

The terms "hyperkinetic impulse disorder" (1950s), "minimal brain dysfunction" (MBD;1960s), "hyperactive child syndrome" (1970s), "attention deficit disorder with or without hyperactivity" (ADD with or without H; 1980s) and now "attention deficit hyperactivity disorder" (ADHD;1990s) have successively been applied to a heterogeneous (mixed) group of children, adolescents, and young adults whose clinical problem can be more correctly explained by one or more of the following disorders: affective illness, learning disabilities, and primary disorder of vigilance (Table 6). ADHD is just a group of symptoms that demands more accurate evaluation and correct diagnosis (Figure 1).

The recently described primary disorder of vigilance (PDV) (Table 7) is the proper diagnosis of a large group of young people fulfilling criteria for ADHD without hyperactivity or with only minimal hyperactivity. These young people are busy, inattentive, and daydream while trying to stay awake due to difficulties remaining alert (hypovigilance). The individual with primary disorder of vigilance, when depressed, becomes excessively sleepy and can "sleep the depression off." Such individuals can go to bed for weeks, months, or until the depression lifts. Interestingly, during a manic episode, the individual with primary disorder of vigilance continues to have vigilance problems ("sleepy") when trying to concentrate.

Learning Disabilities, Depression, and School Failure

Children, adolescents, and young adults with specific learning disorders have a high prevalence of

depression. Sixty to eighty percent (60-80%) of learning disabled children failing in school will fulfill criteria for depression at the time of initial clinical evaluation. Evaluation of individuals failing in school necessitates careful assessment of emotions and cognition and communication skills (higher brain functions; Table 8), along with standard neurologic and physical examinations. This total evaluation, including history of the present problem, developmental, health and family histories should take less than 90-120 minutes to complete. Too often an evaluator takes the short-cut of only evaluating part of the young person rather than the whole person: the evaluation for learning disability will be omitted; affective illness will be excluded; none of the young person's brain functions will be examined (with only a motor examination performed); or without any examination, the determination will be made that the environment (home or school) is the sole cause of the difficulties; or the label ADHD suggested and a prescription of stimulant medication offered. It is important to undertake a complete systematic evaluation to establish correct diagnosis and develop a successful clinical management program.

For children, adolescents, and young adults with learning disabilities, school can be an unpleasant, noxious, and highly frustrating environment. Young people with various specific learning disabilities often manifest poor attention, restlessness, daydreaming, and limited diligence when asked to do tasks that they are unable to do. It is postulated that continuous stimulation of a genetically maldeveloped area of the cerebral hemisphere makes that area, surrounding cerebral cortex, and homologous cerebral cortex in the opposite hemisphere all work less well. For example, language-impaired and dyslexic children (left hemisphere learning disability) often become moody, irritable, and angry when asked to speak, read, or recall words. Poor spellers (left hemisphere learning disability), practicing spelling for the spelling test, become increasingly dysphoric, anxious, irritable, and dysattentive as the school year progresses. Some will cry, show poor self-esteem and express death wishes. The repeated attempts to perform language functions by these children stimulates poorly functioning left cerebral hemisphere which leads to dysfunction of the opposite right hemisphere areas with resultant depressive symptomatology. These examples represent the interaction between environmental stress and cerebral dysfunction.

Right hemisphere learning-communication disorders (RHD) are characterized by difficulties with social communication including social discomfort, social dysemotionality, dysprosody (difficulties understanding

the gestures or speech tones of others, difficulties expressing appropriate gesture or speech tones, or both), ordering problems (difficulties with sequencing, timing, and context), motor and social dyspraxias (clumsiness), disturbed attention (both hypovigilance and neglect), difficulties with calculation (dyscalculia for the sequencing of numbers), and poor graphic skills (Table 8). Young people with right hemisphere learning communication disorders, during adolescence and adulthood, are at high risk for major depressive disorder often exacerbated by the inappropriate stress promoted by education directed at their communication difficulties. This population can be divided into an under-expressive group (hypoprosodic; developmental expressive hypodysprosodia) and an over-expressive group (Asperger's Syndrome; developmental expressive hyperdysprosodia).

In contrast, it is uncommon to see on a referred basis a pure left hemisphere learning disability (Table 8). Most, if not all, children, adolescents, and young adults referred because of school failure have evidence of bihemispherical (right and left cerebral hemisphere) learning disorder. With sixty percent (60%) or greater of these bihemispherical learning disabled children manifesting affective illness, it is often the exacerbation of the affective illness that tips the balance from marginal passing to failure. Within this context, there are several major questions: "Why is this young person now depressed? Can depression be induced or promoted by asking the right cerebral hemisphere to do repetitively what it is unable to do? Is it likely that pure left hemisphere disorders (pure 'classic' dyslexia; pure 'classic' dysphasia) (Table 8), with no genetic basis for depression, will struggle in the school setting but be free of depression? (Population studies of non-referred young people suggest this interpretation). Does asking the learning disabled individual to work repetitively at tasks he or she is unable to do cause depression? Is there a biological basis for a 'reactive' depression?"

The multiple threshold theorem best explains induction of affective illness. For those individuals with a high genetic load for depression little or no environmental stressors are needed. These individuals will have spontaneous episodes of affective illness throughout their lives. In contrast, for those individuals with a low genetic load, external factors are important in the induction of a depressive or manic episode. Such episodes can be induced by head trauma, viral encephalitis, medical illnesses (including hyperthyroidism, systemic lupus erythematosus, and infectious mononucleosis), and various drugs and medications (including minor and major tranquilizers, anti-hypertensives, and anti-convulsants). Inappropriate educational stress (as indicated above) can also induce

depression. Young people with specific learning disabilities who are failing in school and functioning poorly outside of school must be considered to have depression until proven otherwise. The treatment of specific learning disabilities should avoid induction of depression or worsening of other patterns of affective illness by providing an appropriate educational environment. If manic or depressive episodes occur, specific medical management should be undertaken (Table 9).

Treatment of Depression, other Patterns of Affective Illness, and Specific Learning Disabilities

Depression and other patterns of manic-depressive disease are chemical disorders of the brain that can occur spontaneously or be promoted or induced by other medical illnesses, drugs and medications, and environmental events. Often the school environment is a major stressor for the learning disabled individual. Treatment strategies for learning disabled young people should address appropriate educational input/output systems assuring success in school, including college preparatory and college classes. The goal of formal education must be the acquisition and utilization of meaningful information and the pursuit of the individual's assets, talents, and creativity. The educator provides a structured and disciplined environment to accomplish these goals. Reading, spelling, numeric language, graphic skills, vocabulary, and word usage improve with aging and maturation of the nervous system, something that cannot be accelerated by remediation or other programs. Young people with specific learning disabilities are willing, able, capable, and competent learners if appropriate input/output systems are used. Table 10 summarizes the bypass/compensatory strategies and assistive technologies that must be implemented for the learning disabled person to prevent the induction of affective illness episodes.

The management of children, adolescents, and young adults with affective illness is multimodal (Table 9). They must be informed about the nature, course, and treatment of this disease. The environments must be supportive and reassuring. There must be an emphasis on positive interaction, and the individual must be assisted with appropriate decision making. The depressed young person should not be allowed to "drop out" of usual pursuits.

Cognitive coaching on a "mini" daily basis should be offered by parents and significant others (teachers, coaches, employers, or friends). Cognitive coaching involves recognizing that actions should dictate feelings and intelligence should overrule emotions: "It is not

how one feels but it is how one acts"; "act positive-feel better"; "feel sad and act sad, then you will be sad"; "feel sad and act happy, then you will feel better"; "feel mad and act with anger, trouble!"; "feel mad and act civil, then good things can happen". Clinical cognitive psychological counseling may be indicated for both the individual and the primary caretakers (Wilkes and colleagues, 1994). The emphasis is for all concerned parties to act using intelligence and not emotions, with such interaction being positive, rewarding, reassuring, and supportive, while intelligently ignoring the disturbing mood or emotion.

The hallmark of successful treatment is for the brain to be free of the "chemical imbalance" of affective illness. For this to occur, the treatment should include appropriate medication management. Prior to instituting antidepressant medication, it has become standard to obtain an electrocardiogram (EKG). Complete blood count with differential, blood chemistry profile, and thyroid profile are also often indicated (particularly before use of antimanic drugs). An electroencephalogram (EEG) is obtained if the person has had a seizure in the past, if there is a positive family history for epilepsy, or prior to the use of antimanic drugs. Sometimes an EEG, CT scan, or MRI is indicated as a result of abnormal neurologic findings.

The drug management of depression, dysthymia, and cyclothymia with prominent dysphoria continues to be with the first generation antidepressants (tricyclic antidepressants) in children and young adolescents. The choice of drug is dependent upon the body type of the individual and prior treatment response in family members. Amitriptyline (Elavil) is the first choice in young people who are not overweight. If not effective, this is followed by doxepin (Sinequan). These two medications increase appetite and can increase weight. Imipramine (Tofranil) followed by desipramine (Norpramin), the metabolite of imipramine, are the first drugs in overweight young people. Nortriptyline (Pamelor), the metabolite of amitriptyline (Elavil), is sometimes the third choice but can also cause excessive weight gain. If the depressed child or young adolescent does not respond to the above medications, the third generation antidepressants are then offered. For most, we use fluoxetine (Prozac) first, followed by sertraline (Zoloft).

In mature adolescents and young adults with depression, dysthymia, and cyclothymia with prominent dysphoria, the third generation antidepressants are becoming the first choice rather than the tricyclic antidepressants. For those free of prominent hypomania or manic moods, we prefer fluoxetine (Prozac),

followed by sertraline (Zoloft) and then either imipramine (Tofranil) (a first generation antidepressant), or paroxetine (Paxil). Adolescents manifesting dysthymia with bipolar features, juvenile rapid cycling bipolar disorder, or cyclothymia are initially offered imipramine (Tofranil) followed by sertraline (Zoloft), prior to antimanic medication.

For successful management of depression and dysthymia in adolescents and young adults, a combination of a third generation antidepressant and a tricyclic is often needed. The third generation antidepressant is taken in the morning and the tricyclic at bedtime.

Table 11 lists the antidepressant medications, the dosage range, serum levels, indications, contraindications, and potential adverse effects. These medications are not approved by the FDA for use in children less than 12 years of age, except for the use of imipramine for bedwetting. However, they are successful in the treatment of depression in all ages and their use has become a standard of practice.

There is a group of depressed learning disabled young people who also have inherited the primary disorder of vigilance (Table 7). If this group fails with the above approach to drug management, protriptyline (Vivactil) is sometimes successful. Occasionally a combination of protriptyline during daytime hours with another tricyclic antidepressant at bedtime proves beneficial.

The treatment of mania in classic manic-depressive disease, juvenile rapid cycling bipolar disorder, and cyclothymia will often require antimanic drugs (Table 12). However, we continue to offer individuals with these patterns of affective illness an initial trial of antidepressant medication. If manic symptomatology is promoted then antimanic medication must be started. The antimanic drugs primarily used are carbamazepine (Tegretol) and lithium carbonate. If the manic state is characterized primarily by hypomania and elated moods, then lithium is the first choice. If the mania is manifested as hostile anger without elation, the first antimanic drug is carbamazepine. Most individuals whose mania is successfully controlled with lithium (or carbamazepine) will require the addition of an antidepressant medication to be free of depression.

All the antidepressants can promote or induce mania. Likewise, the antimanic drugs can promote depression. For most depressed individuals, an antidepressant medication will be successful. For some the addition of lithium or carbamazepine with the antidepressant will be needed to achieve stable moods and good functioning.

Serum medication levels are obtained during the course of treatment. Repeat EKG, blood chemistries profile, and thyroid profile are performed at appropriate intervals depending upon the medication being used and age of the patient.

Frequently the young patient (and parents) will ask: "Do I have to take medicine forever?" Our answer is "No." Once a well state is established for a period of 6-9 months, medication can be tapered on a trial basis. Although affective illness is a cyclic disorder, recurrent cycles can be expected but not anticipated or predicted. Sometimes recurrence will be seasonal and on other occasions associated with stressful events. If, with aging through adolescence, the dysthymia, hypomania, cyclothymia, or rapid cycling bipolar disorder patterns do not stabilize, then appropriate medication will be needed for a long period of time: "But nothing is forever." However, individuals requiring and benefiting from lithium can anticipate long term use of the medication.

A complex issue is the treatment of attentional symptoms with the two stimulant medications: methylphenidate (Ritalin) and pemoline (Cylert). These two agents heighten vigilance and can be successfully used in the management of the primary disorder of vigilance. They also lower the hyperactivity of hypomania (Table 12). However, these drugs can promote depression. The mood disturbance ("rebound"), insomnia, and loss of appetite associated with these two medications are symptomatic of an underlying depression. Rarely are these two agents helpful in the management of affectively-ill, learning-disabled young people. With aging and freedom from depression, those with the primary disorder of vigilance will stay busy to be alert, to learn, and to perform. If depression occurs, appropriate antidepressant medication should lift the depression thus restoring normal vigilance which was worsened by the depression.

Summary

A common cause of school failure in learning disabled young people is affective illness. Depression is generated by the right cerebral hemisphere and manic symptoms by the left hemisphere. Affective illness is a genetic disorder that can present in several patterns: depression, dysthymia, mania, hypomania, cyclothymia, juvenile rapid cycling bipolar disorder, and classic manic-depressive disease. The treatment is multimodal involving the use of appropriate medication in addition to appropriate environmental (home and school) communication systems, cognitive coaching, and counseling. Affective illness is recognizable, treatable, and should have a good prognosis.

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From *Secondary Education and Beyond, LDA, 1995*

Table 1. Childhood Depression

- A. The 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, pessimism; mood swings, moodiness, irritable, easily annoyed; cries easily, hypersensitive; negative; difficult to please)
 - II. Self-deprecatory ideation*
(feelings of being worthless, useless, dumb, stupid, ugly, guilty; beliefs of persecution; death wishes; suicidal thoughts and/or attempts)
 - III. Agitation*
(difficult to get along with; quarrelsome; disrespectful of authority; belligerent, hostile, agitated; excessive fighting or sudden anger)
 - IV. Sleep disturbance
(initial, interval, and/or terminal insomnia; difficulty awakening in the morning)
 - V. Change in school performance*
(frequent complaints from teachers: "daydreaming, poor concentration, poor memory;" loss of usual effort in school subjects; loss of usual interest in nonacademic school activities; incomplete classroom assignments; much incomplete homework; drop in usual grades; finds homework difficult)
 - 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
(nonmigraine headaches; abdominal pain; muscle aches or pains; other somatic concerns)
 - IX. Loss of usual energy
(loss of usual personal interest or pursuits other than school; 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 primary caretaker (historians) is conducted utilizing a semistructured closed-end technique.
- C. A symptom is accepted as positive when at least one characteristic behavior listed in brackets for the category is present.
- D. Symptoms I and II must be reported by the patient to be considered positive, but symptoms III to IX to be considered positive can be reported by either patient or historian.
- E. Each symptom must be discrete change in usual self (new or worsening of old behavior); symptom complex must be present for more than 1 month and associated with maladaptation.
-

*Denotes psychological symptoms

Adapted from Levy HB, Harper CR, Weinberg WA: **A practical approach to children failing in school.** *Pediatr Clin North Am* 39:895-928, 1992 and Weinberg WA, Rutman J, et al: **Depression in children referred to an educational diagnostic center: Diagnosis and treatment.** *J Pediatr* 83: 1065-1072, 1973

Table 2. Childhood Mania

- A. The presence of either or both symptoms 1 and 2 and three or more of the remaining six symptoms (3-8):
 - 1. Euphoria
(denial of problems or illness: “nothing wrong with me”, inappropriate feelings of well-being, inappropriate cheerfulness, giddiness, silliness)
 - 2. Irritability and/or agitation
(belligerence: hostile anger; destructiveness: inappropriate crudeness, rudeness, vulgarity)
 - 3. Hyperactive, “motor driven,” intrusive/disruptive
 - 4. Push of speech (may become unintelligible), garrulousness, “motor-mouth”
 - 5. Flight of ideas (racing thoughts)
 - 6. Grandiosity (may be delusional)
 - 7. Sleep disturbance (decreased sleep)
 - 8. Distractibility, shortened attention span, inability to concentrate
 - B. Interview of patient and primary caretakers (historians) is conducted utilizing a semistructured, 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 to be considered positive, but symptoms 2 to 8 to be considered positive can be reported by either patient or historian.
 - E. Each symptom must be discrete change in usual self (new or worsening of old behavior); symptom complex must be present for more than 2 weeks and associated with change to maladaptation.
-

Adapted from Levy HB, Harper CR, Weinberg WA: **A practical approach to children failing in school.** *Pediatr Clin North Am* 39:895-928,1992 and Weinberg WA, Brumback RA: **Mania in childhood: Case studies and literature review.** *Am J Dis Child* 130:380-385. 1976.

Table 3. Biological Correlates

Depression

Abnormal neuroamine metabolism
Sleep rhythm disturbance
 Early-onset REM sleep
 Fragmented stage IV sleep
Aberrant neuroendocrine secretion
 Nonsuppressible cortisol secretion
 Blunted thyrotropin release following TRH
 Blunted growth hormone release
Abnormal left limb electrodermal response
Left limb motor hemisyndromes
Right cerebral (parietal) cognitive dysfunction
Hypovigilance
Right cerebral hypometabolism
Response to neuroamine re-uptake blocking drugs

Mania

Abnormal neuroamine metabolism
Right limb motor hemisyndromes
Left cerebral cognitive dysfunction
Response to dopamine receptor blocking drugs
Response to lithium

Primary Disorder of Vigilance

Left limb motor hemisyndromes
Hypovigilance
Hypersomnolence

Narcolepsy

Inappropriate REM-onset sleep activity
Hypovigilance
HLA-DR2 phenotype

Learning Disability (Right Hemisphere)

Subtle left limb hemiatrophy
Left limb motor hemisyndromes
Right cerebral hypometabolism

Sociopathy

Absence of cognitive dysfunction
Absence of parietal-temporal dysfunction
Absence of motor abnormalities

Learning Disability (Left Hemisphere)

Subtle right limb hemiatrophy
Right limb motor hemiatrophy
Left cerebral hypometabolism

Adapted from Weinberg WA, Brumback RA: **The myth of attention deficit hyperactivity disorder: Symptoms resulting from multiple etiologies.** *J Child Neurol* 7:431-445, 1992.

Table 4. Summary of Pharmacologic Differences among Six Tricyclic Antidepressants

<u>Drug</u>	<u>Sedation</u>	<u>Anticholinergic Effects</u>	<u>Block of Amine Pump</u>	
			<u>Serotonin</u>	<u>Norepinephrine</u>
Imipramine	++	++	++	++
Amitriptyline	+++	+++	+++	+
Desipramine	+	+	0	+++
Nortriptyline	++	++	+	++
Doxepin	+++	+++	Weak	
Protriptyline	0	++	Not known	

0 = none; + = slight; ++ = moderate; +++ = high

Adapted from Holister LE: **Drug therapy: Tricyclic antidepressants**, *N Engl J Med* 229:1106-1109, 1168-1171, 1978 and with permission in Weinberg WA, Emslie GJ: **Attention deficit hyperactivity disorder: The differential diagnosis**. *J Child Neurol* 6(Suppl):S23-S36, 1991.

Table 5. Consecutively Referred Normally Intelligent Learning Disabled Prepubertal Children

<u>Diagnosis</u>	<u>Number/Total (%)</u>
Total with hyperactivity	117/223 (52.5)
Not depressed	31/117 (26.0)
Positive for depression	86/117 (74.0)
Hyperactive only while depressed	64/117 (55.0)
Total with depression	136/223 (61.0)
Not hyperactive	50/136 (37.0)
Hyperactive	86/136 (63.0)
Hyperactivity plus depression	86/223 (38.5)

Adapted from Weinberg WA, Emslie GJ: **Attention deficit hyperactivity disorder: The differential diagnosis**. *J Child Neurol* 6(Suppl):S23-S36, 1991 and Brumback RA, Weinberg WA: **Relationship of hyperactivity and depression in children**. *Percept Mot Skills* 45:247-251, 1977.

Table 6. Prominent Causes Of Attention Deficit Hyperactivity Disorder

DEVELOPMENTAL HYPERACTIVITY

AFFECTIVE ILLNESS (BIPOLAR DISORDER)

Dysthymia

Hypomania

Cyclothymia

Depression

Mania

Classic manic-depressive disease

Juvenile rapid cycling bipolar disease

PRIMARY DISORDER OF VIGILANCE

LEARNING DISABILITIES (task dependent)

NARCOLEPSY

PRIMARY CONDUCT DISORDER

CLASSIC FOCAL NEUROLOGICAL DEFICIT

Neglect

Inattention

Adapted from Weinberg WA, Emslie GJ: **Attention deficit hyperactivity disorder: The differential diagnosis.** *J Child Neurol* 6(Suppl):S23-S36, 1991.

Table 7. Primary Disorder of Vigilance

- A. A disorder of at least 1 year duration with symptoms from all five major symptom categories:
1. Decreasing ability to sustain alertness, wakefulness, arousal, and watchfulness during continuous mental (or other task) performance
 - a. Complaints of tiredness, drowsiness, sleepiness, lethargy
 - b. Yawning, stretching, sleepy-eyed (glassy-eyed) appearance
 - c. Falling asleep; excessive napping
 2. Decreasing attention to present activities
 - a. Daydreaming
 - b. Difficulty focusing attention; loses place in activities and conversation
 - c. Poor performance
 - d. Slow, delayed, or incomplete tasks
 - e. Disorganized
 3. Avoidance of structured or repetitive activities
 - a. Loss of interest in or complaint that structured activities are dull, boring, monotonous, tedious, uninteresting (or no longer interesting)
 - b. Preference for shifting activities that have random or irregular changes in schedule or activity (orderly randomization)
 4. Motor restlessness and behaviors to improve alertness
 - a. Fidgeting
 - b. Talkativeness
 - c. Moving about
 - d. Busyness
 5. Caring, compassionate, affectionate, kind temperament
- B. A major symptom category is accepted as positive when the symptom or one or more of its behaviors is identified in a semi-structured, closed-end interview of patient and primary caretakers (historians).
- C. This symptom complex must precede the onset of other medical disorders (including depression, narcolepsy, various medications, alcohol and drug abuse, hypothyroidism) that can cause secondary hypovigilance.
- D. Although symptomatology may be identified at any age, the disorder generally becomes more symptomatic with schooling and aging and can result in increasing maladaptation.

Adapted from Weinberg WA, Brumback RA **Primary disorder of vigilance: A novel explanation of inattentiveness, daydreaming, boredom, restlessness, and sleepiness.** *J Pediatr* 116:720-725, 1990 and Weinberg WA and Harper CR: **Vigilance and its disorders.** *Neurol Clin* 11:59-78, 1993.

Table 8. Left And Right Cerebral Hemisphere Functions

Left Cerebral Hemisphere Functions

Left Parietal Temporal Cortex

Primary Sensory Functions
Sensation of right body
Perception of right visual field
Appreciation of sound from right ear

Cognitive Functions

Language Skills: Comprehension and expression of oral and written language including storage and recall of symbols and nominals; storage of common nouns and action verbs (inner vocabulary); rules of grammar and structure of language; and verbal word recognition (inner speech)

Praxias - command type

Emotional Functions

Denial, oppositional, non-compliance, and hostile anger (mania)

Obsessions and compulsions

“Learned” pessimism and negativity

Pedantic, rigid responses

Rationalization

Attentional Functions

Attentiveness to right space

Minor role in vigilance (tonic state)

Left Frontal Lobe Functions

Fundamental movement of right body

Right voluntary gaze

Clarity of verbal thought (freedom from auditory-verbal hallucinations and delusions)

Adapted from Weinberg WA, Emslie GJ: **Adolescents and school problems: Depression, suicide, and learning disorders.** In Stiffman AR, Feldman RA (eds): *Advances in Adolescent Mental Health, vol 3*, Greenwich, CT, JAI Press, 1988 and Weinberg WA, Harper CR, Brumback RA: **Use of the Symbol language and communication battery in the physicians office for assessment of higher brain functions (particularly the temporal and parietal lobes).** *Child Neurol* 1995; 10(Suppl 1): 233-S31.

Right Cerebral Hemisphere Functions

Right Parietal Temporal Cortex

Primary Sensory Functions

Sensation of left body
Perception of left visual field
Appreciation of sound from left ear

Cognitive Functions

Spatial orientation
Spatial relations (right-left discrimination)
Sequencing of symbols, objects, and events
Timing and time perception
Music appreciation
Recognition of objects and faces
Geometric communication
Non-verbal communication
Praxias - coordinated motor behavior

Emotional Functions

Prosody
Primary emotionality
Empathy and comprehension of emotionality
Affective behavior (depression)
Wit and humor

Attentional Functions

Arousal
Vigilance: alertness - wakefulness (phasic states)
Attentiveness: Right and left space

Primary Visual Imagery

Picture-to-picture storage and representation

Symbolization (symbolic representation)

Picture-to-word storage and representation
(understanding the surrounding world)

Right Frontal lobe functions

Fundamental movement of left body
Left voluntary gaze
Motor persistence
Order (formal type: seeing the world as a series of interrelated entities)
Planning
Volition - intention (“the will”)
Diligence - work ethic - drive
Executive control
Abiding by rules and regulations: (social conduct); reputation

Table 9. Management of Children and Adolescents with Affective Illness

Individual, family, and environmental counseling

Remove inappropriate stressors: Use bypass strategies; demands and tasks in keeping with the young person's facilities.

Informative: Emphasis on genetics, biology, and maturation with potential cycles.

Educative: Emphasis on what is known and not known; avoid rationalization and misinformation.

Supportive: Be a positive advocate.

Reassuring: A treatable and self-limiting condition with anticipation of long periods of well states.

Assist with order and planning: Toward school, work, play, and pursuit of assets and talents.

Assist with decision making "Continue usual pursuits"; "Do not drop out." 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."

Psychopharmacologic treatment

Tricyclic antidepressants: Amitriptyline, imipramine, desipramine, nortriptyline, protriptyline, doxepin, clomipramine

Newer antidepressants: trazodone, maprotiline, fluoxetine, sertraline, paroxetine.

Thioridazine (and rarely haloperidol — other major tranquilizers are not presently being used).

Carbamazepine.

Lithium.

Adapted from Levy HB, Harper CR, Weinberg WA: **A practical approach to children failing in school** *Pediatr Clin North Am* 39:895-928, 1992; Weinberg WA, Emslie GJ: **Attention deficit hyperactivity disorder: The differential diagnosis.** *J Child Neurol* 6(Suppl):S23-S36, 1991; and Weinberg WA, Emslie GJ: **Adolescents and school problems: Depression, suicide, and learning disorders.** In Stiffman AR, Feldman RA (eds): *Advances in Adolescent Mental Health, vol 3*, Greenwich, CT, JAI Press, 1988, pp 181-205.

**Table 10. Bypass Compensatory Strategies to Reduce Stress
in Children with Cerebral Dysfunction**

Problem Area	Strategy
Reading	<ol style="list-style-type: none"> 1. Listen to “good readers” read age-appropriate (not “skill” level) material while eyeing the printed page; avoid listening to poor readers reading 2. Use talking books, listen to tapes, and view films and filmstrips for information; if necessary, read salient material using well-prepared course handouts and worksheets. 3. Read silently for meaning, but never read aloud. 4. Test orally and provide a reader and recorder for necessary written tests (such as the college entrance examinations and group achievement tests).
Word finding Vocabulary Reading comprehension Picture-word association	<ol style="list-style-type: none"> 1. Use picture-to-word matching tasks (including associating names and faces) for vocabulary development. 2. Test both orally and in writing using multiple-choice, true-false, yes-no, or circle-the-correct-answer formats; avoid both oral and written “fill-in the blank” testing situations.
Spelling	<ol style="list-style-type: none"> 1. Look at word while simultaneously hearing the word spoken. 2. Test spelling by multiple-choice format using age-appropriate (not “skill” level) words. 3. Use <i>The Bad Speller’s Dictionary</i> (New York, Random House, 1974). 4. Use a good speller as a secretary or write using computer word processor with appropriate spell-checking software.
Writing	<ol style="list-style-type: none"> 1. Encourage oral expression or demonstration instead of written tasks. 2. Allow the child to dictate information or answers into a tape recorder or directly to a secretary. 3. Use a circle-the-correct-answer written text format. 4. Provide computer with word-processing software that contains a built-in dictionary and thesaurus.
Computation	<ol style="list-style-type: none"> 1. Pursue instruction of age-appropriate (not “skill level”) mathematical concepts and Arithmetic Mathematics principles, avoiding specific calculations. 2. For instruction of computation, teach by example showing both correct and incorrect responses. 3. Provide calculators and/or computers with appropriate software to perform the necessary computations at age-appropriate (not skill”) level. 4. Utilize pick-the-correct-answer testing formats.
Organization	<ol style="list-style-type: none"> 1. Offer one small task at a time, or divide larger tasks into several mini-tasks. 2. As necessary, provide reminders to child of tasks; avoid nagging and avoid checklists. 3. Avoid punishment for “forgetting,” not finishing, or being late with assignments or tasks. 4. Provide the child with a pictorial system for self-reminding of tasks, chores, and events. 5. Offer all testing using an untimed format.

Adapted from Levy HB, Harper CR, Weinberg WA: **A practical approach to children failing in school.** *Pediatr Clin North Am* 39:895-928. 1992 and Brumback RA, Weinberg WA: **Pediatric behavioral neurology: An update of the neurologic aspects of depression, hyperactivity, and learning disabilities.** *Neurol Clin* 8:677-703, 1990.

Table 11. Antidepressant Pharmacotherapy

Medication	Dosage Range	Serum level (mg/ml)	Indications	Contraindications	Potential Adverse Effects
Amitriptyline	Children 1-3 mg/kg/d (up to 5 mg/kg/d if monitored) Adolescents 110-200	100-250 (sum of amitriptyline plus nortriptyline)	Primary drug of choice for depression in non-obese preadolescents, or underweight adolescents, with a history of "pure familial" type of affective disorder Tertiary drug for depression in adolescents or if family history of "depressive spectrum biogeny"	Obesity Excessive daytime sleepiness History of paroxysmal atrial tachycardia other cardiac conduction disturbances Liver or renal disease unless dose and serum levels can be closely monitored	Induction or promotion of mania Atropinic side effects Mild tremor Worsening of depressive symptoms Increased appetite and excess Excessive daytime sleepiness Rash EKG evidence of cardiac conduction changes + Hypertension
Nortriptyline (A metabolite of amitriptyline)	Children 1-3 mg/kg/d Adolescents 50-150 mg/d	50-150	Secondary drug for depression in children and adolescents	History of paroxysmal atrial tachycardia or other cardiac conduction disturbances Liver or renal disease unless dose and serum levels can be closely monitored	Same as amitriptyline except with less effect on appetite and weight "Therapeutic window" in which sub-therapeutic dosages worsen depression
Imipramine	Children 1-3 mg/kg/d (up to 5 mg/kg/d if monitored) Adolescents 100-200 mg/d	150-250 (sum of imipramine plus desipramine)	Primary drug of choice for depression in adolescents, obese children, or with a family history of "depressive spectrum biogeny"	Significantly under-weight or with anorexia History of paroxysmal atrial tachycardia or other cardiac conduction disturbance Liver or renal disease unless dose and serum levels can be closely monitored	Induction or promotion of mania Atropinic side effects Mild tremor Worsening of depressive symptoms Decreased appetite and excess weight loss Excessive daytime sleepiness Rash EKG evidence of cardiac conduction changes Hypertension

Table 11. Antidepressant Pharmacotherapy (Continued)

Medication	Dosage Range	Serum level (mg/ml)	Indications	Contraindications	Potential Adverse Effects
Desipramine (a metabolite of imipramine)	Children 1-3 mg/kg/d (up to 5 mg/kg/d if monitored) Adolescents 100-200 mg/d	50-300	Primary or secondary drug of choice for depression in children and adolescents with excessive daytime sleepiness Secondary drug for depression in adolescents, obese children, or with a family history of “depressive spectrum biogeny”	Same as nortriptyline	Same as imipramine, plus insomnia
Doxepin	Children 1-3 mg/kg/d Adolescents 100-200 mg/d	75-200	Primary drug of choice for depression in non-obese children and adolescents with prominent anxiety, phobias, and somatic symptoms Tertiary drug for depression in non-obese children and adolescents without excessive daytime sleepiness	Same as amitriptyline	Same as amitriptyline, except infrequently induces or promotes mania
Protriptyline	children 5-10 mg bid or tid* Adolescents 5-15 mg bid or tid*	70-260	Secondary drug for depression in association with excessive daytime sleepiness (the primary disorder of vigilance)	Same as nortriptyline	Induction or promotion of mania Atropinic side effects Mild tremor Worsening of depressive symptoms Decreased appetite and excess weight loss Insomnia and hyperalertness Rash EKG evidence of cardiac conduction changes+ Hypertension

Table 11. Antidepressant Pharmacotherapy (Continued)

Medication	Dosage Range	Serum level (mg/ml)	Indications	Contraindications	Potential Adverse Effects
Trazodone	Adolescents 50-600 mg/d	800-1600	Alternate drug for depression in non-obese individuals or without excessive daytime sleepiness	Same as amitriptyline	Same as amitriptyline, plus priapism
Maprotiline	Adolescents 75-300 mg/d	200-600	Alternate drug for depression in obese individuals or with excessive daytime sleepiness	Same as imipramine	Same as imipramine, plus insomnia
Fluoxetine	Children 20 mg qam Adolescents 20-40 mg bid*	100-900 (combined)	Alternate drug for depression, particularly in obese individuals or with excessive daytime sleepiness	Same as imipramine	Decreased appetite and excess weight loss Confusion Incoordination Insomnia Induction or promotion of mania
Paroxetine HCl	10-40 mgms qam	86 +/- 61	Same as fluoxetine	Same as fluoxetine	Same as fluoxetine
Sertraline HCl	50-100 mgms qam	30-200	Same as fluoxetine	Same as fluoxetine	Same as fluoxetine

* Last dose should not be given after 4 pm

+ EKG = electrocardiogram

Adapted from Brumback RA, Weinberg WA: Pediatric behavioral neurology: An update on the neurologic aspects of depression, hyperactivity, and learning disabilities. *Neurol Clin.* 8:677-703, 1990 and adapted from Levy HB, Harner CR, Weinberg WA: A practical approach to children failing in school. *Pediatr Clin North Am* 39:895-928, 1992

Table 12. Antimanic Pharmacotherapy

Medication	Dosage Range	Serum level	Indications	Contraindications	Potential Adverse Effects
Lithium carbonate	Children 20-40 mg/kg/d (600-1200 mg/d) Adolescents 600-1800 mg/d (use tid dosage schedule for regular tablets and bid schedule for sustained-release tablets)	0.8-1.4 mEq/L	Drug of choice in manic-depressive disease when other conventional regimens for depressive and manic symptomatology fail, often used in combination with an antidepressant	Renal disease Unrecognized thyroid disease	Induction or promotion of depression Tremor Increased appetite and excess weight gain Excessive daytime sleepiness Polydipsia and polyuria with resultant hyponatremia Hypothyroidism; nontoxic goiter Parathyroid dysfunction EEG spike discharges+ EKG conduction changes+
Thioridazine	Children 10-15 mg bid, tid, or qid Adolescents 10-25 mg bid, tid or qid	Not available	Primary drug of choice for minor manic states and as an additional drug for manic states induced or promoted by antidepressant drugs	Obesity Hepatic disease Excessive daytime sleepiness	Induction or promotion of depression Increased appetite and excess weight gain Excessive daytime sleepiness Rash Dyskinesias Retinitis
Carbamazepine	Children 10-20 mg/kg/d Adolescents 200-1200 mg/d (use bid or tid dosage schedule)	4-12 g/ml	Primary drug of choice for manic rages if relatively free of depression; excessive daytime sleepiness, and petit mal epilepsy May be added to other drug regimens to control manic rages	Excessive daytime sleepiness Depressive irritability and anger Petit mal epilepsy Hepatic disease Blood dyscrasia	Excessive daytime sleepiness Induction or promotion of depression Hepatotoxicity Hematologic abnormalities Rash
Methylphenidate	10-60 mg/d (bid or tid dosages*)	Not available	Primary drug of choice for chronic hypomania and for excessive daytime sleepiness (hypovigilance) May be added to antidepressant drug regimen in rapidly cycling manic-depressive disease (or cyclothymia)	Depressive symptomatology Hypertension Chronic tic syndrome	Hypertension Induction or promotion of depressive symptomatology particularly vegetative symptoms (anorexia, weight loss insomnia, somatic complaints) Transient growth retardation Tics
Pemoline	37.5-112.5 mg/d (in bid or tid dosages)	Not available	Secondary drug for chronic hypomanic and for excessive daytime sleepiness	Same as methylphenidate	Same as methylphenidate, plus elevation of liver enzymes

* Last dose should not be given after 4 pm + EKG = electrocardiogram + EEG = electroencephalogram

From Brumback RA, Weinberg WA: Pediatric behavioral neurology: An update on the neurologic aspects of depression, hyperactivity, and learning disabilities. *Neurol Clin* 8:677-703, 1990 and adapted from Levy HB, Harper CR, Weinberg WA: A practical approach to children failing in school. *Pediatr Clin North Am* 39:895-928, 1992

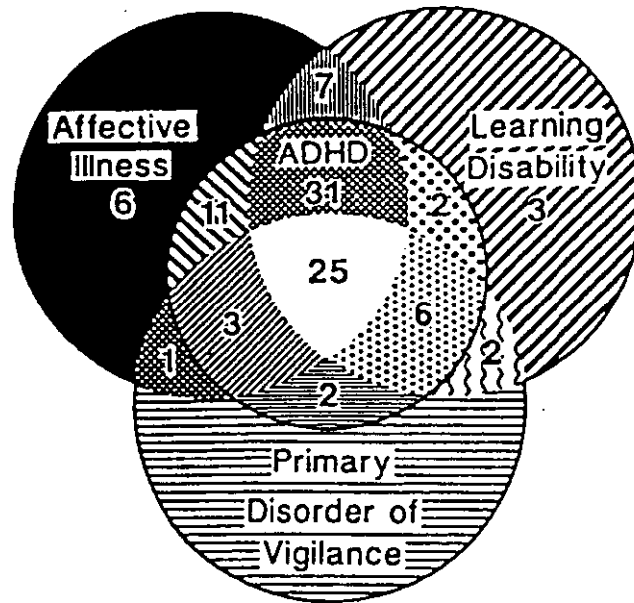


FIGURE 1

Distribution of diagnoses for 100 children consecutively referred to the outpatient Pediatric Behavioral Neurology Program at the Children's Medical Center of Dallas (only 99 children are represented because one child with affective illness, primary disorder of vigilance, and learning disability did not fulfill criteria for ADHD). The mean age of the group was 10.3 years (range, 5 years 5 months to 18 years 9 months), with 73 boys and 27 girls. Racial distribution of the population was 93 white, six black, and one Oriental, and all were of at least middle-class socioeconomic status. A large number of the children had more than one specific diagnosis (comorbidity of disease and all ADHD (inner circle) could be explained by one or more of the specific entities of affective disorder, primary disorder of vigilance, and learning disability).

Adapted from Weinberg WA, Brumback RA: The myth of attention deficit hyperactivity disorder: Symptoms resulting from multiple etiologies. *J Child Neurol* 7:431-445,1992

APPENDIX A

Weinberg Screening Affective Scale (WSAS)

INSTRUCTIONS:

We would like to ask you some very serious and very important questions. We want to know how you feel about yourself. If you agree with the statement, circle yes. If you do not agree with the statement, circle no. We consider these questions and your answers very important.

- | | | |
|--|-----|----|
| 1. I will try to give my honest feelings on these questions. | yes | no |
| 2. I feel dumb and stupid too much of the time. | yes | no |
| 3. I can't do my homework anymore. | yes | no |
| 4. I wish that I could stay in bed all day. | yes | no |
| 5. I can't do anything right. | yes | no |
| 6. Sometimes I wish I were dead. | yes | no |
| 7. I don't like other people. | yes | no |
| 8. I don't like school anymore. | yes | no |
| 9. I feel sad too much of the time. | yes | no |
| 10. I can't do my school work anymore, it's too hard. | yes | no |
| 11. It's hard to have any fun anymore. | yes | no |
| 12. School makes me feel sick. | yes | no |
| 13. I have too many bad moods. | yes | no |
| 14. This is not a good world. | yes | no |
| 15. I don't like to eat anymore. | yes | no |
| 16. I feel lonely too much of the time. | yes | no |
| 17. I have too much trouble remembering things. | yes | no |
| 18. Nothing is ever done the way I like it. | yes | no |
| 19. I eat too much. | yes | no |
| 20. I am not as good as other people. | yes | no |
| 21. It seems like I'm always in trouble for fighting and that is not fair. | yes | no |
| 22. I have gained too much weight. | yes | no |
| 23. I have too many headaches. | yes | no |
| 24. I don't want to go to school anymore. | yes | no |
| 25. I don't have fun playing with my friends anymore. | yes | no |
| 26. I feel too tired to play. | yes | no |
| 27. It seems like some part of my body always hurts me. | yes | no |
| 28. It makes me feel good to tease other people. | yes | no |
| 29. People are always talking about me when I'm not there. | yes | no |
| 30. I can't sit still and that is a problem for me. | yes | no |

32. My friends don't want to be with me anymore.	yes	no
33. I can't concentrate on my work.	yes	no
34. I daydream too much in school.	yes	no
35. I never seem to be able to finish my work in school.	yes	no
36. I have too many stomach aches.	yes	no
37. I have too many aches and pains in my muscles.	yes	no
38. I don't want to get out of bed in the morning.	yes	no
39. I talk too much and that causes a problem for me.	yes	no
40. I'm always grouchy and that's bad.	yes	no
41. It's hard to fall asleep and that bothers me.	yes	no
42. My friends don't like me anymore.	yes	no
43. When I wake up at night, it is hard to go back to sleep	yes	no
44. I am losing too much weight.	yes	no
45. I cause trouble for everybody.	yes	no
46. I don't want to be with my friends anymore.	yes	no
47. Everybody picks on me.	yes	no
48. I get angry easily.	yes	no
49. School makes me feel nervous.	yes	no
50. I cry a lot.	yes	no
51. I talk back to grown-ups.	yes	no
52. I wake up too early in the morning and it is hard to go back to sleep.	yes	no
53. I can't have any fun anymore.	yes	no
54. I think a lot about killing myself.	yes	no
55. My answers are how I have been feeling most of the time.	yes	no
56. These answers represent my honest feelings.	yes	no

Score Sheet

Weinberg Screening Affective Scale (WSAS)

Criteria for depression by self-report

- A. I and II plus four (4) or more of III - X.
- B. Two or more positive items per major symptom category: I- X.
- C. "Yes" response on Question 55.

	<u>Number of Positive Items</u>	<u>Criteria</u>	
I: 9,13,14,16,18,40,48,50	_____	yes	no
II: 2,5,6,20,21,29,31,42,47,54	_____	yes	no
III: 28,32,45,51	_____	yes	no
IV: 38,41,43,52	_____	yes	no
V: 3,10,17,33,34,35	_____	yes	no
VI: 7,25,46	_____	yes	no
VII: 8,12,24,49	_____	yes	no
VIII: 23,27,36,37	_____	yes	no
IX: 4,11,26,53	_____	yes	no
X: 15,19,22,44	_____	yes	no
TOTAL:	_____		

Total number of positive categories

I	II	III	IV	V	VI	VII	VIII	IX	X	_____		
Response to Question 55												
										:	yes	no
DEPRESSION BY SELF-REPORT										:	yes	no
Death wish - positive on item 6										:	yes	no
Suicidal ideation - positive on item 54										:	yes	no

Adapted from Weinberg WA, Emslie GJ: **Weinberg screening affective scales (WSAS and WSAS-SF)**, *J Child Neurol* 3:294-296, 1988.

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**Emphasized selected readings*

GLOSSARY

Affective Illness: Disorder(s) of mood and affect with associated vegetative symptoms producing discomfort and maladaptation-malfunction (see below for various patterns).

Bipolar disorder: Classic manic-depressive disease characterized by discrete periods of depression and mania interspersed with well states.

Cyclothymia: Chronic, long-standing (greater than one year) state of hypomanic/ hyperactivity with moment to moment, day to day (mixed days) of both manic and depressive moods and feelings with no prolonged, stable (well) states.

Depression: A discrete period of symptoms persisting for more than 34 weeks and associated with poor functioning either at work (school), home, or in play. The two major symptoms are: 1) dysphoric mood characterized by both statements and appearance of sadness, unhappiness, pessimism, loneliness with associated moodiness and often being difficult to please; and 2) low self-worth (self-deprecation/poor self-esteem): feelings of worthlessness, uselessness, and being dumb, stupid, ugly, or guilty, along with beliefs of persecution, death wishes, and suicidal thoughts. Four or more of the following symptoms are usually present during the depressive episode: agitation, insomnia, lowered school performance, diminished socialization, (or change to an unacceptable social group), loss of interest in school, physical complaints, loss of usual energy and dropping out of usual fun, sport, or aesthetic activities, and change in appetite or weight. Depending upon the number of symptoms and severity of malfunctioning, depression is characterized as either “minor” or “major”.

Dysthymia: (chronic minor depression) Chronic, long standing (greater than one year) depressive moods and feelings (dysphoria) along with variable vegetative symptoms of insomnia, low energy, and decreased interest that fluctuates in the pattern of mixed days with occasional “all bad days” but with no prolonged, stable (well) states.

Dysthymia With Bipolar Features: Dysthymia with brief, recurrent “moments” (periods) of hostile anger.

Hypomania: (Chronic hypomanic/hyperactivity) Persistent hyperactivity with inappropriate cheerfulness, silliness, giddiness, intrusiveness, and interruptive-disruptive behavior. Racing thoughts with push of speech, inappropriate, provocative sexual activity, irritable moods, and decreased need for sleep can become apparent in older children (ages 3-8 years).

Juvenile Rapid Cycling Bipolar Disorder (JRCBD): Chronic, long standing (greater than one year) moment to moment or day to day (mixed days) fluctuations of both manic and depressive moods and feelings with no prolonged, stable (well) states but without any evidence of chronic hypomanic/ hyperactivity.

Mania: The hallmark of mania is two symptoms: 1) euphoria (most commonly a total denial of any problems as one is disrupting the environment and life of others and/or inappropriate feeling of well-being); and 2) rages, hostile anger (ire) that can be associated with homicidal thoughts and statements. An episode of mania is characterized by these two symptoms persisting for a period greater than 1-2 weeks along with the symptoms and behaviors of hypomania. Associated with these symptoms, there must be malfunctioning in three environments of school, home, and play. (Occasionally the individual will have malfunctioning in only one or two of the environments.)

Manic-depressive Disease (Classic Bipolar Disorder): Episodes of depression persisting for more than 2-4 weeks and mania persisting for more than 1-2 weeks interspersed with long periods (months or years) of stable (well) states.

Primary Disorder of Vigilance: A dominantly inherited condition characterized by loss of alertness/wakefulness (vigilance) during activities requiring continuous mental performance (attention-concentration) or continuous task performance. Individuals with the Primary Disorder of Vigilance have a unique temperament: kind, caring, affectionate, compassionate, and the inability to hold a grudge.