PRACTICAL GUIDE FOR STUDENTS IN CHILD PSYCHIATRY

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3. DISORDERS USUALLY FIRST EVIDENT IN INFANCY, CHILDHOOD, OR ADOLESCENCE: DISRUPTIVE BEHAVIOR DISORDERS

DSM-IV groups together as "disorders usually first evident in infancy, childhood, or adolescence" a number of disorders that typically begin before adulthood. Usual onset may be in infancy (e.g., reactive attachment disorder), middle childhood (e.g., conduct disorder), or adolescence (e.g., anorexia nervosa). Any of these diagnoses may be used for adults if they have persisting symptoms of the disorder (e.g., attention-deficit hyperactivity disorder (ADHD) that began before age 18. Some of these disorders (e.g., anorexia nervosa) may begin in adulthood. This chapter will cover the disruptive behavior disorders.

Behavior problems are highly prevalent in both "normal" school children and child psychiatric patients. For disruptive behavior disorders, parents generally appear more distressed than the child, and typically bring a reluctant child for treatment. The child often denies symptoms and views the parents as the problem. These conditions are called externalizing disorders, emphasizing that these children and adolescents "act out" their conflicts and feelings.

ATTENTION - DEFICIT HYPERACTIVITY DISORDER

CLINICAL DESCRIPTION

Children with ADHD are impulsive, inattentive, distractible, and often hyperactive.

DSM-IV criteria are listed in the following table:

Diagnostic criteria for ADHD

A. Either (1) or (2):

(1) six (or more) of the following symptoms of Inattention have persisted for at least 6month to a degree that is maladaptive"and inconsistent with developmental level:

Inattention

(a) often fails to give close attention to details or makes careless mistake in schoolwork, work or other activities

(b) often has difficulty sustaining attention in tasks or play activities

(c) often does not seem to listen when spoken to directly

(d) often does not follow through on instruction and fails to finish schoolwork, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)

(e) often has difficulty organizing tasks and activities

often avoids, dislikes or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)

(g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books or tools)

(h) is often easily distracted by extraneous stimuli

(i) is often forgetful in daily activities

(2) six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 month to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity:

(a) often fidgets with hands or feet or squirms in seat

(b) often leaves seat in classroom or in other situations in which remaining seated is expected

(c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings or restlessness)

(d) often has difficulty playing or engaging in leisure activities quietly

(e) is often "on the go" or often acts as if "driven by a motor"

(f) often talks excessively

Impulsivity.

(g) often blurts out answers before questions have been completed

(h) often has difficulty awaiting turn

(i) often interrupts or intrudes on others (e.g., butts into conversations or games)

A. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.

B. Some impairment from the symptoms is present in two or more settings (e.g., at school/or work and at home).

C. There must be clear evidence of clinically significant impairment in social, academic or occupational functioning.

D. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorder or a Personality Disorder).

Code based on type:

314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 month

314.00 Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 month

314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 month

Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.

In the past, the joint appearance of hyperactivity, impulsivity, and inattention was labeled "minimal brain damage" (even though there is no direct evidence of brain damage), "minimal brain dysfunction" (though overt neurological damage can produce similar dysfunctions), "hyperkinetic syndrome," or "hyperactivity syndrome" (though more than motor systems alone are involved). Clinical skill and specific criteria are needed to make the diagnosis, because one-half of all children are rated as "hyperactive" by their parents and teachers.

The child may appear quite different to observers in different environments. Most children experience more environmental and affective pressure at school than at home, and impulsivity is particularly clear in the classroom. Impulsivity and inattention are also increased in noisy places or group settings.

Although DSM-IV emphasizes cognitive and motoric characteristics, pathological impulsivity is also seen in emotional lability, lack of behavior control, and aggressivity. Children with ADHD tend to be impulsive in all settings, but the degree of impulsivity increases with emotional states, excessive sensory stimulation, lack of environmental structure, and physiologic stress, such as hunger or fatigue.

Children with ADHD do not modulate their level of arousal to match the expectations of the situation and so are often over-or underaroused. They have a relative inability to motivate them-selves when tasks are difficult, long, or boring and are unusually prone to seek immediate reinforcement.

Not all children with "attention deficits" have behavior problems, hyperactivity, or excessive aggressivity. The DSM-IV diagnosis undifferentiated attention-deficit disorder is used when attention deficits occur without hyperactivity and behavioral impulsivity. These children tend to show mild anxiety and withdrawal, have fewer conduct disorder and behavior problems, and less frequently present to psychiatric settings. There is preliminary evidence that their inattention may improve with psycho-stimulant treatment.

Peers perceive ADHD children as immature and irritating and often avoid or neglect them due to their low frustration tolerance and intrusive, bossy, socially inappropriate behavior. Peers learn quickly that it is easy to tease children with ADHD, or to set them up to get into trouble with adults.

EPIDEMIOLOGY

Prevalence of ADHD is estimated at 9—10% of boys and 3% of girls. ADHD is present in 30—50% of child psychiatric outpatients and 40—70% of child psychiatric inpatients, often in combination with other psychiatric disorders.

Neurologists tend to see ADHD with seizures and mental retardation, psychiatrists see ADHD with learning disorders and other psychiatric illnesses, and pediatricians typically treat "healthy" and good-prognosis ADHD children.

Girls constitute 10—25% of ADHD children. Girls with ADHD typically show less impulsivity and aggression than boys, but more fear, mood swings, social withdrawal and rejection, and cognitive and language problems. Age at diagnosis tends to be older in girls.

ETIOLOGY

There are likely multiple types of attention deficit, with a variety of brain mechanisms. Different etiologies may be relevant for different individuals.

• Genetic factors.

ADHD runs:

- in families, particularly in male relatives of children with ADHD
- in families with mood disorders
- in families with higher incidence of antisocial personality disorder, aggression, arrest and imprisonment.

Girls with ADHD have a stronger family history for ADHD (a higher genetic loading for ADHD) than do boys with ADHD.

• Medical factors.

Various medical and neurological etiologies can produce ADHD, although children with identifiable causes represent a small proportion of the ADHD population, and many children with this trauma do not have ADHD.

Medical contributions to ADHD: prenatal: - poor maternal health

- maternal use of cigarettes, alcohol an drugs birth complications: - bleeding
    - hypoxia
    - toxemia
    - prolonged labor perinatal: - low birth weight
- postmaturity infancy: malnutrition toxic: lead poisoning

inborn errors of metabolism: phenylketonuria brain injury: - trauma - infection Neurological disorders:

- seizures and cerebral palsy - 5% of children with ADHD.
- computed tomography scans are typically normal.

EEG findings are abnormal in 20% (versus 15% of "normal" children).

- mild decrement in IQ (- cortical lesions in the right hemisphere was found in 93%
- frontal cortex function is impaired in certain children Neurocfiemicarstudies of ADHD suggest involvement of neurotransmitter systems

using dopamine,norepinephrine, serotonin, and phenylethylamine.

The regional cerebral blood flow of children with ADHD is decreased in the striatal region and increased in primary sensory regions of the occipital and temporal cortex Psychostimulant medication tends to normalize the pathologic flow distribution.

COURSE AND PROGNOSIS

Mothers of youngsters with ADHD commonly recall excessive intrauterine "kicking" or report that "when he began to walk, he ran." ADHD is diagnosable by age 36 months, but can be hard to identify before age 5_years due to overlap with normal developmental hyperactivity, impulsivity, and short attention span (the

"terrible twos"). Identification is often delayed until elementary school, where demands for physical stillness, prolonged attention, and conformity to social norms are greater, and comparison to peers is easier.

When inattention, impulsivity, and emotional changeability with or without motoric hyperactivity persist into adulthood (in 50% of patients), the diagnosis of ADHD remains valid. The residual symptoms continue to be stimulant responsive.

A major finding, confirmed in several follow-up studies, is that 25% of adults diagnosed with ADHD as children have Janti-social personality disorder. In young adulthood, there is often low self-esteem and significant compromise in social skills. These individuals

have more school failure, car accidents, moves, court appearances, felony convictions, suicide attempts' phobic anxiety, and somatization, but no general excess of alcohol or drug dependence or schizophrenia.

Complications in family functioning can potentially influence marital harmony and sibling development.

It is likely that the low self-esteem, compromised social skills, major behavior problems, aggressivity, criminality, and antisocial personality are complications (or features of associated disorders) rather than essential components ADHD.

ADHD provides an example of how a congenital or early-onset disorder, often with a genetic or neurological etiology, can be modified by life experiences and developmental processes. Socioeconomic class, family variables, education, and medical treatment become increasingly prominent in their influence on behavior and personality.

ADHD is commonly seen in association with other psychiatric disorders, particularly oppositional defiant disorder, conduct disorder, and mood disorder. There is also an increased prevalence ot ADHD in mental retardation, Tourette's disorder, and specific learning disorders.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Clinical evaluation of ADHD entails assessing etiologies, delineating concomitant and possibly treatable neurological and psychiatric disorders, and ruling out similarly presenting diagnoses.

Clinical evaluation includes:

- Physical examination
- Neurological examination before starting medication documents possible neurological deficits, neuromaturational signs (choreiform movements, overflow and minor movements, gross and fine motor function, laterality), tics, or dystonias.
- A sample of handwriting might be included.
- An EEG is generally not cost-effective, but can be considered if neurological findings are suggestive.
- Laboratory screening includes thyroid battery, blood lead level
- Psychological testing and possibly neuropsychological evaluation

Differential diagnosis:

Clinical expertise is necessary to differentiate ADHD from normal high activity level, which may be causing complaints from parents or teachers. Problems of recent onset and brief duration may represent an adjustment disorder. In addition, situational anxiety, child abuse and neglect, or simple boredom can clinically present as inattention, hyperactivity, or impulsivity.

- Conduct disorder and oppositional defiant disorder are characterized by deliberate or provocative noncompliance, as distinguished from the impulsive or inattentive failure to comply of ADHD, although these disorders often occur together.
- Major depression, bipolar disorder anxiety disorder, and possibly schizophrenia may present with deficits in attention. Mania may take uncharacteristic forms in prepubertal children and should be considered strongly in patients with a family history of bipolar disorder.
- Children with pervasive developmental disorder often show hyperactivity, inattention, and impulsivity, but this diagnosis preempts a diagnosis of ADHD.
- Children with undiagnosed mental retardation or specific developmental disorder are often mistakenly referred for stimulant treatment because of inattention, distractibility, and

and imprisonment.

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inability to follow directions in school.

Hyperthyroidism can mimic ADHD, as can the effect of certain drug treatments (theophylline, carbamazepine, benzodiazepines, or phenobarbital)

TREATMENT MODALITIES

Multimodal treatment should be targeted to the specific problems of the child and family. Environmental and drug treatments of ADHD can be sufficient in patients in whom behavior problems are not prominent, good adaptive functioning is seen, and study habits and interest in school have not been disrupted. If medications and environmental treatments do not lead to improved behavior, academic performance, and social adjustment, then additional psychiatric treatments should be considered. Individual family psychotherapy can be crucial to facilitate developmental gains.

Environmental management of sensory stimulation involves arranging the child’s home and school setting to reduce stimuli and distractions. At home, parents are advised to establish quiet spaces, remove easily damaged furnishings, keep toys put away,permit only one friend at a time to visit, avoid the supermarket and shopping mall, and encourage fine i motor exercises (e.g., jigsaw puzzles).

Education. Special education is not always required, but is useful in many patients. At school, a small and perhaps self-contained classroom, small-group activities, thoughtful selection of seating location, high teacher-to-student ratio, routine and predictable structure, one-to-one tutoring, or use of a resource room can be beneficial.

Informing school officials about the child’s strengths and problems, self-esteem, and social skills and receiving regular reports regarding behavior and academic performance are essential.

Pharmacotherapy. Medication is useful for treating impulsivity, inattention, and hyperactivity. Approximately 75% of children with ADHD respond positively to psychostimulants (methylphenidate, d-amphetamine, or pemoline) or tricyclic antidepressants.

- Psychostimulant treatment alone does not routinely lead to improved academic performance or school grades, even in children whose attention and classroom behavior improve. Stimulants alone have not been shown to produce lasting benefit in reducing aggressivity, conduct disorder, or criminality or infostering educational achievement, job functioning, marital relationships, or long-term adjustment.

Antidepressants can be considered if:

- the rebound effect of the psychostimulants is disruptive,
- tics are present or emerge, if medication cannot be given at school,
- the child is depressed or becomes dysphoric when treated with a stimulant,
- the family psychiatric history of mood disorder is strong,
- the child or family is potentially abusing drugs.
- Lithium is probably not effective in typical ADHD.

- Drug treatment may continue for years, with periodic dosage adjustments needei for changes in body weight, varying environmental or developmental stress, or developmental () changes in drug biotransformation rate

Psychotherapy. Behavior modification can improve academic achievement, reduce specifically targeted conduct problems, and decrease symptoms that are not helped by stimulants.

Both punishment (time-out and response cost) and contingent rewards are required, and consistent, intensive, and prolonged (months to years) treatment may be needed. The cooperation OT parents ana teachers is required for behavior modification to be effective.

Cognitive-behavior therapy is useful for teaching problem-solving strategies, selfmonitoring, verbal mediation (using internal speech) for self-praise and self-instruction, and seeing rather than glossing over errors.

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Diet. Dietary treatments are unestablished. Thd Feinqold diet (omitting salicylates and food dyes) has yielded contradictory findings, but food dye restriction night be helpful for a subgroup (5—10%) of children with ADHD. Parents' reports of dye effects are demonstrably not reliable. Even for the diet responders, stimulants are a more potent treatment than diet.

Caffeine has been recommended by nonprofessionals as a more "natural" treatment of hyperactivity, but studies have shown significant side effects and no evidence of efficacy.

CONDUCT DISORDER

CLINICAL DESCRIPTION

Conduct disorder, the most common diagnosis in child and adolescent patients in both clinic and hospital settings, describes youngsters who repeatedly violate important societal rules or the personal rights of others. See the following table for DSM-Icriteria:

Diagnostic criteria for 312.8 Conduct Disorder

A. A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated, as manifested by the presence of three (or more) of the following criteria in the past 12jrionth, with at least one criterion present in the past 6 month: Aggression to people and animals

(1) often bullies, threatens or intimidates others

(2) often initiates physical fights

(3) has used a weapon that can cause serious physical harm to others (e.g., a bat, brick, broken bottle, knife, gun)

u4) has been physically cruel to people

(5) has been physically cruel to animals

(6) has stolen while confronting a victim 9e.g., mugging, purse snatching, extortion, armed robbery)

(7) has forced someone into sexual activity

Destruction of property

(8) has deliberately engaged in fire setting with the intention of causing serious damage

(9) as deliberately destroyed others' property (other than by fire setting)

Deceitfulness or theft

(10) has broken into someone else' house, building or car

(11) often lies to obtain goods or favors or to avoid obligations (i. g., "cons" others)

(12) has stolen items of nontrivial value without confronting a victim (e.g., shoplifting, but without breaking and entering; forgery)

Serious violations of rules

(13) often stays at night despite parental prohibition beginning before age 13 years

(14) has run away from home over night at least twice while living in parental or parental surrogate home (or once without returning for a lengthy period)

(15) is often truant from school beginning before age13 years

B. The disturbance in behavior causes clinically significant impairment in social academic or occupational functioning

C. If the individual is age 18 years or older criteria are not met for Antisocial Personality Disorder.

Specify severity.

Mild: few if any conduct problems in excess of those required to make the diagnosis and conduct problems cause only minor harm to others.

Moderate: number of conduct problems and effect on others intermediate between "mild" and "severe".

Severe: many conduct problems in excess of those required to make the diagnosis or conduct problems cause considerable harm to others.

Specify type based on age at onset:

Childhood-Onset Type: onset of at least one criterion characteristic of Conduct Disorder prior to age 10 years.

Adolescent-Onset Type: absence of any criteria characteristic of Conduct Disorder prior to age 10 years.

Despite the diversity of behavioral symptoms and severity in conduct disorders, certain psychological characteristics are common: attention deficits - learning disorders impulsivity

impaired mood - sullenness, impulsive anger impaired cognition:

- distortions of size and time awereness
- lack of or distorted conection between prior events and consequences
- limited problem - solving ability

-pathological defense mechanisms:

-minimizing

-avoiding

- externalizing

- unconscious manipulation

- denial

- identification with the aggressor

-interpersonal impairment:

- suspiciousness or paranoia, with cognitive distortions
- misperception of others' actions as hostile
- difficulty relating to peers and adults
- lack of guilt, of empathy

EPIDEMIOLOGY

There is a male predominance for violent crimes (8:1) and property crimes (4:1), but the prevalence of conduct disorder has been increasing in females in recent years. When self-report data are used instead oficial statistics, the prevalence of misconduct and delinquent behaviors becomes much greater, and the male predominance lowers to about 2:1.

ETIOLOGY

A wide variety of etiological factors have been proposed for conduct disorder:

- - intrapsychic: defective superego
- sociological: - social deprivation-poverty and cultural disadvantage
- cultural behavior norms - street-corner gangs
- genetic transmission of: - antisocial personality disorder

- substance abuse
- mood disorders -ADHD
- schizophrenia
- learning disorders

- borderline IQ or mental retardation: - easily led by peers
  - lack of more adaptive coping strategies
- difficult temperament: resistant to parental discipline
- brain damage: - seizures
  - victim of physical abuse
  - head and face injuries
- concomitant psychiatric disorders: - ADHD
  - mood disorders
  - learning disorders
  - psychosis
- parenting characteristics: - large family
  - absent or alcoholic father
  - depresed or irritable parent
  - parental rejection or abandonment
  - harsh inconsistent unpredictable discipline
  - inadequate supervision and limit setting '
  - modeling of impulsivity, aggression or,antisocial behavior
  - institutional placement

No single factor is able to account for more than 50% of the variance in the occurrence of conduct disorder. No combination of factors can account for more than 70% of the variance. Many children with these risk factors do not develop a conduct disorder.

COURSE AND PROGNOSIS

Many youths with conduct disorder are rehabilitated, but some lead lives of delinquency or undergo long-term incarceration. A relatively small number of chronic offenders account for the majority of delinquency. Recidivists are more likely to have early onset, poor school grades, and low socioeconomic status. Only a fraction of children and adolescents with conduct disorders receive psychiatric treatment. When child guidance clinic patients with antisocial behavior are followed into adulthood, over one-third are maladjusted and show severe psychopathology-including antisocial behavior, alcohol abuse, psychiatric

hospitalization, child neglect, nonsupport, financial dependence, and poor envelopment and military records. Their own children are at high risk for truancy, running away, theft, and high school dropout.

Complications of conduct disorder are numerous: school failure, school suspension, legal problems, injuries due to fighting or retaliation, accidents, sexually transmitted diseases, teenage pregnancy, prostitution, being raped or murdered, fugitive status, abandonment of family, drug addiction, suicide, and homicide.

Typically, concomitant psychiatric or neurological pathology is observed in association with serious conduct disorder and contributes to severity and chronicity.

Despite the high incidence of major psychopathology? maladjustment, and incarceration, many children with conduct disorder are able to achieve a favorable adult adjustment. More adaptive social skills and more positive peer experiences predict a better long-term outcome. Patients with the group type of conduct disorder generally have a less serious prognosis.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

The comprehensive biopsychosocial evaluation of conduct disorder involves a multidisciplinary team including a psychiatrist, psychologist, pediatrician, neurologist, educational consultant, speech and language specialist, occupational and recreational therapist, social worker, legal advisor, school liaison, and case manager.

It is essential to evaluate the fuil range of Axes j. and JL psychiatric diagnoses, neurological status, intelligence, neuropsychological features, language and speech abilities, educational skills and deficits, social adaptiveness and assertiveness, and family functioning.

Approximately one-third of prepubertal children with depressive mood disorders will develop a DSM-IV conduct disorder by age 19. Childhood bipolar disorders are associated with an even higher prevalence of conduct disorder. Psychotic children and adolescents may fulfill criteria for conduct disorder. Substance use is often present and is commonly underdiagnosed. ADHD appears with conduct disorder in 70% of clinical cases. Differential diagnosis includes a spectrum of less severe disorders a (DSM-IV code):

- oppositional defiant disorder,
- adjustment disorder with disturbance of conduct,
- childhood or adolescent antisocial behavior,
- normal mischief.

TREATMENT

Treatment of conduct disorder may take a variety of forms: legal sanctions, family interventions, social support, behaviour modification, psychopharmacology, psychiatric treatment of the individual or a family

member, or neuromedical treatment. The treatment site can be home, school, hospital, residential school, or specialized delinquency program.

A single treatment modality can be decisive for some individuals with conduct disorder, but the vast majority require multimodal treatment.

Limit setting at home may be compromised by parental conflict, parental absence, inconsistent discipline, vague or minimal expectations for appropriate behavior or parental depression or other psychiatric illness. Creating or reinforcing limits can involve parent counseling, psychiatric treatment of parents, increased supervision at home, surveillance at school, or use of legal mechanism.

Pharmacotherapy. Psychiatric treatment of conduct disorder depends more on individual variables than on diagnostic category.

Some children with conduct disorders (especially those in psychiatric inpatient units) will respond to treatment with antidepressants or lithium.They may be viewed as having “affective conduct disorder," or as having drug-responsive behavior disorders independent of mood disorder. If the conduct disorder is secondary to a major depression, successful treatmenfof the depression with antidepressants may lead to remission of the conduct symptoms.

Lithium may be considered in the treatment of severe impulsive aggression, especially when it is accompanied by explosiv affect. In a well-designed controlled study, lithium was shown to be equal or superior to haloperidol in reducing aggression, hostility, and tantrums, with fewer side effects. Patients with severe impulsive aggression with emotional lability and irritability who have an abnormal EEG, a strong clinical suggestion of epileptic phenomena, or nonresponse to lithium may indicate a trial of carbamazepine. Propranolol, a beta- adrenergic blocker, may be useful in patients with otherwise uncontrollable rage reactions and impulsive aggression, especially those with evidence of organicity.

In patients with coexisting ADHD, stimulant treatment may decrease impulsive conduct symptoms as well as overactivity and inattention. Neuroleptics may reduce aggression, hostility, negativism, and explosiveness in severely aggressive children.

Psychotherapy. Although conventional insight-oriented individual psychotherapy is not useful, recent developments in/cognitive - behavioral problem-solving therapy are promising in the treatment of conduct disorder. Social skills training can remedy.specific deficits.

Functional or family behavior therapy can be useful in correcting dysfunctional interaction patterns. Parent guidance or training in behavior modification can help management of difficult behaviors and promote effective limit setting and positive reinforcement.

Group therapy, particularly in residential treatment or group-oriented facilities, often permits the “gang orientation" to promote positive change and to improve socialization skills.

School interventions can include special attention to behavior control, individualized educational programming, vocational training, speech, and other specific learning disorders.

OPPOSITIONAL DEFIANT DISORDER

CLINICAL DESCRIPTION

Children with oppositional defiant disorder (ODD) show argumentative and disobedient behavior, without serious violation of the rights of other people. They are stubborn, negativistic, and provocative. See following table for DSM-IV criteria:

Diagnostic criteria for 313.81 Oppositional Defiant Disorder

A. The disturbance in behavior of least 6 month during which at least five of the following are present:

(1) often lose temper

(2) often argues with adults

(3) often actively defies or refuses to comply with adults' requests or rules

(4) often deliberately annoys people

(5) often blames others for his or her mistakes or misbehavior

(6) is often touchy or easily annoyed by others

(7) is often angry and resentful

(8) is often spiteful or vindictive

Note: consider a criterion met only if the behavior occurs more frequently than is tipically observed in individuals of comparable age and developmental level.

B. The disturbance in behavior causes clinically significant impairment in social, academic or occupational functioning.

C. The behaviors do not occur exclusively during the course of a Psychotic or Mood Disorder.

D. Criteria are not met for Conduct Disorder and if the individual is age 18 years or older, criteria are not met for Antisocial Personality Disorder.

Anger-related symptoms are typically directed at parents and teachers. A lesser degree of anger dyscontrol may be seen in peer relationships.

A crucial feature of ODD is the self-defeating stand that these children take in arguments. They may be willing to lose something they want (a privilege or toy) rather than lose a battle or lose face. The oppositional struggle takes on a life of its own in the child's mind and becomes more important than the reality of the situation. "Rational" objections voiced to the child become counterproductive, and the child may experience these interventions as continuing arguments.

At times, oppositional children can also be quite compliant, or even perfectionist.

EPIDEMIOLOGY

Perhaps 2—20% of children have ODD, with a male predominance of 2—3:1.ODD is common in psychiatric clinics and in classrooms for emotionally disturbed and learning- disabled children. It is frequently seen concurrently with ADHD.

ETIOLOGY

Psychosocial mechanisms have been hypothesized for ODD:

- Parental problems in disciplining, structuring, and limit setting
- Identification by the child with an impulse-disordered parent who sets a role model for oppositional and defiant interactions with other people
- Parental unavailability

Genetic, neurobiological, and temperament factors may also contribute.

COURSE AND PROGNOSIS

The long-term outcome of ODD is undetermined. It is thought that at least some children with ODD go on to develop a conduct disorder.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

The oppositional and defiant symptoms are generally reported by parents and caretakers. The children do not view themselves as oppositional or argumentative and externalize blame onto parents, authority figures, and peers. Symptoms are more prominent when the child is with familiar people.

Psychiatric evaluation of the child and family is needed to rule out possible conduct disorder, mood disorders, and learning disorders and to investigate family and psychosocial factors.

These children might engage in bullying or psychological cruelty, but would stop short of the pattern of serious behavior problems seen in conduct disorder.

Discret periods of oppositional of defiant behavior may be secondary to major affective episodes (depression or hypomania), psychosis, or an adjustment disorder. Although oppositional and defiant behavior can be normal for children age 1T8-36] months and for adolescents, the 6-month duration criterion for ODD ordinarily excludesihese developmental phenomena. Some children are simply temperamentally stubborn.The intentional and provocative noncompliance characteristic of ODD should be differentiated from the noncompliance resulting from impulsivity and inattention in ADHD although both are often present.

Oppositional behavior that is restricted to the school setting may be a result of mental retardation, borderline intelligence, or a specific developmental disorder or may be a function of lack of training in cultural norms and expectations.

TREATMENT

Parent management training in behavior management techniques such as positive reinforcement, giving more effective commands, "time-out," and token economies can modify oppositionality. Traditional individual and family psychotherapy are not generally helpful with the primary symptoms of ODD.

In children with coexisting ADHD and ODD, stimulant or antidepressant medication may reduce oppositional behavior and improve compliance.

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4.OTHER DISORDERS USUALLY FIRST EVIDENT IN INFANCY, CHILDHOOD, OR ADOLESCENCE

ANXIETY DISORDERS OF CHILDHOOD OR ADOLESCENCE

Some types of anxiety have a developmental basis, and can be considered normative:

Common normal fears:

birth to 6 month: - loss of physical support
- loud noises
- large rapidly approaching objects 7-12 months: strangers

1-5 years: - loud noises
- storms
- animals
- the dark
- separation from parents

3-5 years: - monsters
- ghosts 6-12 years: - bodily injury
- burglars
- being sent to the principal

- being punished
- failure

12-18 years: - tests in school
- social embarrassment

Panic disorder, obsessive-compulsive disorder, phobias, and posttraumatic stress disorder can appear in children, although DSM-IV treats these conditions as adult-onset anxiety disorders.Two anxiety disorders usually first evident in childhood are designated in DSM-IV: separation anxiety disorder and overanxious disorder.

SEPARATION ANXIETY DISORDER

Clinical description. In separation anxiety disorder, cognitive, affective, somatic, or behavioral symptoms appear in response to genuine or fantasized separation from attachment figures. The following table shows DSM-IV criteria:

Diagnostic criteria for 309.21 Separation Anxiety Disorder

A. Developmental inappropriate excessive anxiety concerning separation from home or from those to whom the individual is attached, as evidenced by three (or more) of the following:

(1) recurrent excessive distress when separation from home or majoi attachment figures occurs or is anticipated

(2) persistent and excessive worry about losing or about possible ham befalling' major attachment figures

(3) persistent and excessive worry that an untoward event will lead t separation from a major attachment figure (e.g., getting lost or bein kidnapped)

(4) persistent reluctance or refusal to go to school or elsewhere because c separation

(5) persistently and excessively fearful or reluctant to be alone or without major attachment figures at home or without significant adults in other settings

(6) persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home

(7) repeated nightmares involving the theme of separation (8) repeated compliants of physical symptoms (such as headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated

B. The duration of the disturbance is at least 4 weeks

C. The onset is before age 18 years.

D. The disturbance causes clinically significant distress or impairment in social, academic (occupational) or other important areas of functioning.

E. The disturbance does not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia or other Psychotic Disorder and, in adolescents and adults, is not better accounted for by Panic Disorder With Agoraphobia.

Specify if:
Early Onset: if onset occurs before age 6 years.

Separation anxiety disorder can present clinically in a variety of ways, including difficulty in falling asleep and school absenteeism.

Although school absenteeism has various etiologies in addition to separation anxiety disorder, these two conditions will be discussed together because DSM-IV deals with “school phobia" as a specific presentation of separation anxiety disorder. Not all children with school absenteeism have separation anxiety, and not all children with separation anxiety disorder have school absenteeism.

The symptoms of separation anxiety disorder may not be observed until the child experiences a sense of separation from the attachment object, usually a parent or caretaker but sometimes a favorite toy or familiar place. Typically, even a young child with separation anxiety disorder can specify the attachment object that gives a sense of protection or safety from anxiety. Although the anxiety is usually centered on separation from a parent, it can instead manifest as an anticipatory fear of being injured, kidnapped, or killed. Interference with autonomous functioning can extend to inability to [sleep alone, attend school, visit friends, go on errands, or stay at camp.

Children with separation anxiety disorder are typically overly compliant and perfectionist. They will often use excuses such as stomachaches or headaches, or claim that peers or teachers pick on them, to avoid leaving home.

Epidemiology. Separation anxiety disorder has a prevalence of 3%.The gender ratio is equal or slightly female predominant (2:1). School absenteeism is much more common and is a pervasive problem at certain inner-city schools.

Etiology. Developmental theorists have speculated about mechanisms contributing to separation anxiety disorder: Unconscious internal conflicts regarding aggressive and sexual impulses, uncertainty regarding the location of the caretaker after the toddler is able to walk away, and parent-induced anxious attachment.?

Learning theorists have emphasized the maintenance of symptoms by conditioned fear, based on stimulus generalization and reinforcement.

Biological theorists focus on temperamental factors and the possible relationship to childhood mood and adult anxiety disorders!Over half of children with separation anxiety disorder have parents with mood or anxiety disorders]

A physiological component is suggested in follow-up of children identified at age 2 J years as extremely inhibited, quiet, and restrained in unfamiliar settings. At age 7 years, ithey remain shy and socially avoidant, with excessive sympathetic reactivity (measured b\ heart rate acceleration and early morning salivary cortisol levels).

In many cases of school absenteeism, parent and child axe bound to each other b} psychodynamically based fears of separation.

COURSE AND PROGNOSIS. Separation anxiety disorder is usually recognized in early 01 middle childhood. It is typically a recurrent disorder, with acute exacerbations at times o actual separations, deaths, family moves, or crises. Symptoms may worsen during or afte medical illness. Multiple medical evaluations are commonly sought by the child or parents.

As many as one-third of children presenting with separation anxiety disorder also have major depression, and a similar number have concurrent overanxious disorder.

Follow-up studies of school absenteeism have demonstrated a high prevalence o adult maladaptation and psychopathology. The psychopathology takes a wide variety o forms and is not exclusively mood and anxiety disorders. In adulthood, these individuals art highly symptomatic (with somatization and problems with aggression), dependent, and home oriented. Agoraphobic mothers report a high incidence of stomachaches during childhooc and report that their children have a high incidence of school absenteeism an stomachaches. School absenteeism often show academic underachievement and socia avoidance, and can be at risk for excessive absenteeism or chronic unemployment ii adulthood.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. Children may initially need to be interviews with a parent, if separation is too difficult. The workup includes a search for other anxiet disorders or mood disorder in the child and parents. Physical symptoms such as nausea vomiting, abdominal pain, or headaches may require a basic, but not exhaustive, medics evaluation. In separation anxiety disorder, somatic complaints are worse on evenings am mornings before school, and absent on weekends and holidays, except as schoc approaches.

Separation anxiety is a normal developmental phenomenon at ages/18—30 months For children less than 6 years old, separation anxiety typically reappears in stressfi situations. Children with schizophrenia, autistic disorder, or pervasive development disorder not otherwise specified may present with separation anxiety, but these disorder cause severe dysfunction in multiple areas, and their diagnosis excludes separation anxiet disorder.

The most common cause of school absenteeism is truancy, a feature of condui disorder.

Treatment. Separation anxiety disorder is treated with individual psychotherap combined with parent guidance or family therapy and often with an antidepressar medication.

For children with separation anxiety disorder, the child's experience of psychotherap is organized around the actual separations within the therapy. Preparation of the parents a well as the child and active management of the practicalities surrounding expecte separations help in the management of these patients.

Another pitfall in the treatment of these children and their parents involves pathologic; compliance. Some parents and children have an overly good or nice presentation of self, ar their perfectionist manner of managing themselves can prevent them from exposing deep thoughts and feelings. “Pathological pleasantness” in the child or parents needs to t

monitored during the course of treatment and not mistaken for substantial understanding, support, or change.

Limit setting in the treatment of separation anxiety disorder can be useful or disruptive. If the child also has a panic disorder or major depressive episode, then limit setting can be counterpro- ductive and aggravate the symptomatology. Forcing a child with panic disorder to attend school can result in a temper tantrum or physical assault. However, therapeutic coercion can be sensitively applied (“It’s important for you to put yourself into a situation where you may experience some anxiety so you can come to master it”). In treating truancy, limit setting is a useful and necessary measure for establishing a routine daily structure in the child’s life.

Sever separation anxiety disorder that result in total avoidance of school and does not respond to environmental psychotherapeutic and pharmacologic intervention may require psychiatric hospitalisation.

OVERANXIOUS DISORDER

Clinical description. Children with overanxious disorder show generalized anxiety or a need for reassurance that is not restrictive to any particular feared object or anxiety provoking situation.These children suffer from a variety of worries. The most characteristic symptom is unrealistic worry about future events.

In addition to showing anxiety, worry, tension, and somatization, children with overanxious disorder often appear shy, self-doubting, and self-deprecating. Some of these children appear pseudomature and overly serious. Perfectionism and excessive compliance with authority can be carried with apparent pride and superficial self-confidence. They may show habit disturbances, such as nail biting, hair pulling, or thumb sucking. Somatic complaints may result in requests for excessive medical evaluations. Family characteristics include parents who demand high performance, even if they superficially deny placing pressure on the child.

Epidemiology.

- equal gender prevalence or slight male predominance (before puberty)
- 7% of mid-adolescents suffering from overanxious disorder.

Etiology.

- psychodinamic factors: parental pressure, identification with parental anxiety
- neurodevelopmental or genetic contribution.
- temperamental or physiological factors

COURSE AND PROGNOSIS. Some children with overanxious disorder appear to have a chronic course and are hypothesized to be at risk for anxiety disorders, mood disorders, or somatization disorder in adulthood. Potential complications are impairments of self-esteem, learning, and peer relations. Phobic disorders are common codiagnoses.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. Other anxiety disorders and the possibility of mood disorder need to be considered in the differential diagnosis of overanxious disorder. Unlike children with attention-deficit hyperactivity disorder (ADHD), who are also restless and fidgety, overanxious children show obsessive worrying or constant preoccupations. Due to their numerous somatic complaints, various physical illnesses can be a part of the differential diagnosis.

Treatment. Various treatment modalities are used for overanxious disorder:

Behavioral methods include relaxation, desensitization by progressive exposure or by imagining different anxiety-provoking situations, and contingent reinforcement of approach to feared objects or situations.

Psychotherapy is often oriented toward promoting psychological individuation and autonomy in the child and family.

Cognitive therapy is directed at changing self-defeating and pessimistic attitudes. Assertiveness training can be helpful, especially in a group setting.

Benzodiazepines can be considered for the anticipatory anxiety.

Treatment of the parents is often important, particularly if a parent has symptomatic anxiety or an anxiety disorder. In working with parents, it may be helpful to anticipate future developmental events that can lead to parental overprotectiveness or overdemandingness.

EATING DISORDERS

ANOREXIA NERVOSA

Clinical description. Anorexia nervosa is a disorder characterized by severe weight loss or failure to reach expected wei gain due to purposeful strict dieting and/or other extreme measures, frequently including excessive exercise.

The disturbance of body image may be of delusional proportions. The name of the disorder is misleading because there is no loss of appetite (except sometimes in the advanced stages).

Anorexic patients can be divided into those who severely restrict their diet and those who have episodes of binge eating. Restricters may also induce vomiting, or take large amounts of laxatives or diuretics; bulimic anorexics almost always do.

Certain psychological symptoms of anorexia nervosa may be effects of starvation, including social withdrawal, impaired concentration, apathy, mood swings, overactivity, preoccupation with food, and food-related rituals and stereotyped behaviors.

Patients with anorexia nervosa have a generalized fear of loss of control. Low selfesteem and a sense of helplessness and inadequacy are common. Academic overachievement, preoccupation with getting perfect grades in school, excessive studying, and anxiety regarding school performance are common. See attached DSM-IV criteria.

Diagnostic criteria for Anorexia Nervosa

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on selfevaluation, or denial of the seriousness of the current low body weight.

D. In postmenarcheal females, amenorrhea, i.e., the absence of at least three consecutive mentrual cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen, administration.)

Specify type:

Restricting Type: during the current episode of Anorexia Nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Binge-Eating/Purging Type: during the current episode of Anorexia Nervosa, the person has regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).

Epidemiology. Over 90% of patients with anorexia nervosa are female, and they comprise about 1% of adolescent and young adult women. They are predominantly Caucasian and from comfortable economic circumstances or upwardly striving families. The
disorder occurs much more rarely in males.

Etiology. The factors that initiate weight loss are difficult to specify and may differ - from factors that maintain the behaviors, such as the effects of starvation and negative reinforcement. Cultural overvaluation of thinness results in overconcern with dieting and fear of obesity in many girls, as early as elementary school, and may account for the high rate of anorexia nervosa among ballet dancers, models, and gymnasts.

Psychodynamic theories describe these children and adolescents as overly compliant and "perfect." The extreme focus on thinness is seen as an attempt to gain control of a part of their life, and to stave off adolescence and sexuality and remain in or return to a more childlike state. Family dynamics include parental

overinvolvement, lack of appropriate boundaries within the family, and insufficient autonomy. It is important, however, not to confuse the effect on a family of having a child starving herself to death with family characteristics that may have caused the problem.

A genetic component is suggested by the increased incidence of eating disorders in relatives.

COURSE AND PROGNOSIS. The most common onset of anorexia nervosa is during adolescence, soon after the rapid growth spurt at the beginning of puberty, with its weight gain, increase in body fat, and change in body shape. Onset before puberty is less common. The disorder may follow dieting in slightly plump girls. Many of these girls vividly recall being teased about their weight.

Physiological changes are common. Medical complications may require hospitalization:

- dehydration
- electrolyte imbalance constipation and/or diarrhea
- cardiovascular: hypotention, bradycardia, arrhythias,cardiac arrest, edema and congestive heart failure during refeeding, cardiac failure secondary to cardiomyopathy from ipecac (emetine) poisoning

-neuroendocrine: amenorrhea or irregular menses, low basal metabolism rate, abnormal glucose tolerance test with insuline resistance, hypothermia, elevated levels of gowth hormone and cortisole, sleep disturbance abnormal urinalysis

-hematologic: leukopenia, anemia, low sedimantation rate dermatologic: dry skin, lanugo

-oral, esophageal and gastric damage from vomiting and/or binge eating.

The course of anorexia nervosa is typically prolonged, with 75% of patients eventually attaining normal weight, but frequently retaining abnormal eating behavior, especially bulimia. The long-term mortality rate is 5—18%, from suicide and medical causes. There is an increased lifetime prevalence of affective and anxiety disorders in these patients. A subgroup develop obsessions and compulsions extending beyond preoccupation with food and fear of weight gain and may warrant an additional diagnosis of obsessive-compulsive disorder.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. A complete history, physical examination, and routine laboratory studies are necessary to rule out a medical cause for weight or appetite loss. An elaborate medical workup is not usually indicated in the absence of clues from these. Potential complications must be sought and evaluated. Signs of infection may be masked by leukopenia and hypothermia.

In the psychiatric interview of a patient with markedly low weight for height, patients with anorexia nervosa can be distinguished by their denial that they are too thin, although sophisticated patients may have learned to disguise their continuing wish to lose weight. Anorexic patients are often unreliable historians and will claim to have lost their appetite, attempt to hide excessive exercise, claim to eat more than they do, or hide heavy objects in their clothes or drink large amounts of water before lacing weighed. Information from other sources is essential.

Differential diagnoses of anorexia nervosa:

-normal thinness

-phisical disorders causing weight lose: hyperthyroidism, other endocrine disorder, gastrointestinal disorder, malignancy, chronic infection

-psychiatric disorder causing lose of appetite and weight lose: depression, schizophrenia or psychotic depression,conversion disorder, phobia of choking

-hypothyroidism producting hypothremia and amenorrhea.

Treatment. The denial and fears of loss of control and of becoming fat generate severe-resistance to treatment characteristic of anorexia nervosa. A firm focus on weight is helpful, with a balanced emphasis on individual and family issues. A gradual weight gain (0,1—0,2 kg/day) is recommended to reach target weight. Forced weight gain alone and too-rapid weight gain exacerbates fears of loss of control and may be medically hazardous.Tube or intravenous feeding should be reserved for medical emergencies, because these methods are viewed by patients as punitive. They can be sabotaged by the patient and can even be dangerous (bleeding from site of pulled IV, aspiration of tube feedings).

Goals of family therapy are to stop fighting over the patient's weight, to address dysfunctional patterns that have been identified in the evaluation, and to help the family deal with changes as the patient becomes more independent and assertive, less "perfect" and compliant, and more interested in sexual activities.

Behavior modification is useful in producing initial gain to a minimal healthy weight. This lessens the medical risk as well as the negative emotional and behavioral effects of starvation.

Cognitive behavior modification is useful in changing incorrect beliefs and dysfunctional cognitions, developing more adaptive ways of maintaining control, and dealing with stress. A nutritionist can assess .current dietary patterns, design a refeeding program, educate the patient, and remove the therapist from constant haggling over diet and weight.

Long-term outpatient treatment is often necessary, in the form of individual or group therapy. Medication is not especially useful for anorexia nervosa per se, and there is an increased risk of side effects, but it may be useful for concomitant psychiatric disorders (mood disorder or obsessive-compulsive symptoms). Hospitalization may be required for medical instability, lack of improvement, or relapse during outpatient treatment.

BULIMIA NERVOSA

Clinical description. Bulimia nervosa is characterized by repeated episodes of uncontrollable binge eating of huge amounts of food, which continue for up to several hours, as long as the person is able to obtain food or until stopped by abdominal discomfort, sleep, interruption by another person, or vomiting.

Patients with bulimia nervosa are usually of normal weight, but may be obese or thin, and have frequent weight fluctuations. Binge eating is typically done in secret and is initially pleasurable. Guilt, depression, shame, fear of loss of control and weight gain, and reduced self-esteem appear immediately after the binge.

Self-induced vomiting or use of large amounts of laxatives and/or diuretics (purging) decreases discomfort from bloating, may relieve depression and guilt, and controls weight without dieting. The binge-purge cycle may be repeated many times a day, and normal cues for eating and satiety are lost. See attached DSM IV criteria.

Diagnostic criteria Bulimia Nervosa

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:

(1) eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances

(2) a sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)

B. Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as selfinduced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of Anorexia Nervosa.

Specify type:

Purging Type: during the current episode of Bulimia Nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

Nonpurging Type: during the current episode of Bulimia Nervosa, the person has used other inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.

Epidemiology.It is associated with female gender, Caucasian race, higher socioeconomic status, and more developed countries. In a school-based questionnaire survey, 13% of I 5-year-otds reported overconcern about weight and at least occasional use of self-induced vomiting, laxatives, and/or diuretics.

Etiology. Chronic strict food restriction usually precedes bulimia. When hunger breaks through, a binge results. Vomiting and/or purging are discovered as ways to “undo” the binge. Binge eating generalizes as a nonspecific way of dealing with stress, boredom, and negative affects, with the compensatory behaviors to avoid weight gain also becoming chronic.

A family history of obesity, depression or alcoholism is common.

COURSE AND PROGNOSIS. The peak time of onset of bulimia nervosa is late adolescence or early adulthood, somewhat later than for anorexia nervosa, although there is considerable overlap. Anorexia nervosa may precede the development of bulimia nervosa.The course is typically chronic, with repeated relapses. Medical complications of bulimia were mention before, at anorexia nervosa.

Social complications can be severe, with time and finances depleted by obtaining food, binge eating, and purging. School and peer functioning typically suffer.

Associated disorders include depression, substance abuse and borderline personality disorder. The risk of suicide or death from medical complications is significant.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. Bulimic patients often come to medical or psychiatric attention late in the course of the illness. In the absence of severe weight loss, they can hide the disorder, even from family members

living in the same household The evaluation is similar to that for anorexia nervosa. The symptom of binge eating may be secondary to a neurological, endocrine, or genetic disorder (e.g., Prader-Willi syndrome).

Treatment.

The goals of treatment include:

- eliminating the binge-purge cycle,
- establishing healthy eating habits,
- promoting new strategies and skills to deal with emotions and problem situations.

Nutritional consultation can facilitate eating regular, well-balanced meals, avoiding the hunger that triggers the urge to binge.

Hospitalization is indicated in the presence of:

- suicidality,
- out-of-control eating and vomiting,
- metabolic instability,
- failure of outpatient treatment.
- In the hospital, a behavior contract can be implemented, with activities and privileges contingent on eating regular meals and not vomiting. Patients must be watched closely for hiding or stealing food and for secretive vomiting.

Cognitive-behavior modification, conducted in an individual or group setting, can help patients overcome feelings of helplessness and the habit of using food to deal with all of their uncomfortable feelings. Skills and strategies for problem solving, coping with stress, identifying feelings, and avoiding relapse are learned. Recording events, emotions, and desires to binge in a diary is a useful technique. Imipramine, desipramine, phenelzine, lithium, and possibly carbarnazepine may be helpful in the treatment of bulimia nervosa, although medical complications increase the risk of side effects.

PICA

Clinical description. A pattern of eating nonfood materials can be seen in young children, mentally retarded individuals, and pregnant women and appears culturally normative in certain region of the world. Pica is minimally documented in the psychiatric literature, despite its presumed biopsychosocial etiology and major behavioral, cognitive, neurological, and developmental complications. See the following table for DSM-I criteria for pica.

Diagnostic criteria for 307.52 Pica

A. Persistent eating of nonnutritive substance for a period of at least 1 month.

B. The eating of nonnutritive substance is inappropriate to the developmental level.

C. The eating behavior is not part of a culturally sanctioned practice.

D. If the eating behavior occurs exclusively during the course of another mental disorder (e.g., Mental Retardation, Pervasive Developmental Disorder, Schizophrenia), it is sufficiently severe to warrant independent clinical attention.

Pica in children is often observed in association with behavior and other medical problems, and the risk of accidental poisoning is significant.

Epidemiology. About 10—20% of children are believed to show pica as a symptom at some point in their lives. Low socioeconomic class and male gender are over represented. **Etiology.**

The findings of increased childhood pica in households with pets, and of children's eating of pets' food, suggest imitation as a contributing factor.

Disorders of parent-child nurturance and psychosocial deprivation

Parental neglect and poor supervision are related to the risk for toxic ingestions in children.

- Mothers of children with pica are often described as immature, emotionally unavailable, and overwhelmed by parenting tasks.

COURSE AND PROGNOSIS. In children, pica usually starts at age 12—24 months, and resolves by age 6 years. Pica can last into adulthood, particularly in the mentally retarded. Medical complications of pica are numerous and potentially severe:

- Constipation and gastrointestinal malabsorption are common.
- Anemias,
- Repetitive fecal impaction,- Intestinal obstruction,
- Salt imbalances,
- Vomiting,

Parasitic infections.

Lead toxicity, from ingestion of paint, plaster, or earth, can lead to learning impairment and hyperactivity, fatigue, weight loss, and constipation, and finally to toxic encephalopathy At least 30% of children with pica show lead-related symptoms, and approximately 80% of lead-poisoned children have pica.

Delayed motor and mental development, neurological deficits, and behavior abnormalities may predate (and perhaps contribute) to or result from pica.

Evaluation. Children are typically brought for evaluation of other problems, and the pica is often missed. Pica should be considered in all cases of developmental deviation, learning difficulties, mental retardation, unusual behavioral symptoms, and chronic constipation.

Evaluation of children with pica involve:

- behavioral and psychiatric evaluation of the child and parents,
- psychosocial evaluation of the home environment, nutritional status, ~
- feeding history, lead exposure. -

Unexplained fatigue or weight loss, learning impairments, mental retardation, or children who show gingival "leadlines" should alert the clinician to the possibility of lead poisoning.

Treatment.

Behavior therapy has been successfully used for mentally retarded individuals and may be applicable to children with pica. Components include:

- rewarding appropriate eating,
- teaching the differentiation of edible foods,
- overcorrection (enforced immediate oral hygiene) and negative reinforcement (timeouts, physical restraint) for instances of pica.

Psychosocial interventions include:

- promotion of maternal supervision
- stimulation, improvement of play opportunities,
- placement in day care.

Concomitant treatment of medical complications may be required.

RUMINATION DISORDER OF INFANCY

Clinical description Rumination disorder of infancy, a potentially fatal eating disorder, is particularly apparent when infants are alone and reflects abnormal development of early self-stimuiation and physiological regulation. The DSM-I criteria are:

Diagnostic criteria for 307.53 Rumination Disorder

A. Repeated regurgitation and rechewing of food a period of at least 1 month following a period of normal functioning.

B. The behavior is not due to an associated gastrointestinal or other general medical condition (e.g., esophageal reflux).

C. The behavior does not occur exclusively during the course of Anorexia Nervosa or Bulimia Nervosa. If the symptoms occur exclusively during the course of Mental Retardation or a Pervasive Developmental Disorder, they are sufficiently severe to warrant independent clinical attention.

It may be a result or cause of disrupted parent-child attachments and is associated with major developmental delays and mental retardation.

These infants show a pleasurable relaxation as they regurgiate, rechew, and reswallow their food, usually in the absence of caretakers or other sources of stimulation. Their continuing self-stimulation, apparent satisfaction, and languorous obliviousness highlight 52Xrtill engrossment in rumination.

Rumination may start with the infant placing fingers or clothes in the mouth to induce vomiting, with rhythmic body or neck motions, or without any apparent initiating action. During rumination, the infant

generally lies quietly, looks happy or "spacey," or may hold body and head in a characteristic arching position, while sucking. There is no apparent nausea, discomfort or disgust. If observed, the infant usually stops and fixes visual attention on the intruder; when no longer observed, sucking and tongue movements restart in seconds. When not ruminating, the infant may appear apathetic and withdrawn, become irritable and fussy, or seem quite normal.

Etiology. Organic and environmental factors contribute to the etiology of rumination disorder.

- Certain cases of rumination disorder from gastroesophageal reflux due to an esophageal sphincter disorder (hiatal hernia) and can be treated by media or surgical intervention.

Obstetric complications are seen in one-third of these infants, and one-quarter have developmental delays (associate with mental retardation or pervasive developmental disorder).

Perinatal brain damage contributes to the appearance of rumination disorder Prenatal pathology (for example, impaired psychomotor or visceromotor capacities) can contribute to subsequent perinatal and postnatal difficulties

- Although an understimulating or overstimulating environment can contribute to the appearance of rumination disorder, some infants with the disorder appear happy and have parents who are emotionally supportive and interactive. For these infants the usual explanations do not appear to apply, and these infants may be particularly likely to respond to simple behavioral interventions .

COURSE AND PROGNOSIS. Rumination does not typically appear until the infant is age 3—12 months. It usually resolves by the end of the 2nd year, but may persist until the 3rd or 4th year. Spontaneous remissions are common. More persistent cases generally involve mental retardation.

Dehydration, electrolyte imbalance, slow weight gain, failure to thrive, and malnutrition can result from rumination disorder. Mortality may be as high as 25%, usually due to malnutrition.

CLINICAL DESCRIPTION

Boys with gender identity disorder of childhood display effeminate mannerisms, dress up in female clothes (improvising if necessary), and avoid rough-and-tumble play. Girls refuse to wear skirts or dresses or to engage in culturally expected female play, such as dolls, dress-up, or house (except as the father). Parents often tolerate or even encourage cross-gender behavior early on. As the child grows older, however, differences between the parents' perceptions of the seriousness of the problem can generate significant marital conflict. See attached DSM IV criteria.

Diagnostic criteria for Gender Identity Disorder

A. A strong and persistent cross-gender identification (not merely a desire for any perceived cultural advantages of being the other sex).

In children, the disturbance is manifested by four (or more) of the following:

(1) repeatedly stated desire to be, or insistence that he or she is, the other sex

(2) in boys, preference for cross-dressing or simulating female attire; in girls, insistence on wearing only stereotypical masculine clothing

(3) strong and persistent preferences for cross-sex roles in makebelieve play or persistent fantasies of being the other sex

(4) intense desire to participate in the stereotypical games and pastimes of the other sex

(5) strong preference for playmates of the other sex

In adolescents and adults, the disturbance is manifested by symptoms such as a stated desire to be treated as the other sex, or the conviction that he or she has the typical feelings and reactions of the other sex.

B. Persistent discomfort with his or her sex or sense of inappropriateness in the gender role of that sex.

In children, the disturbance is manifested by any of the following: in boys, assertion that his penis or testes are disgusting or will disappearor assertion that it would be better not to have a penis, or aversion toward rough-and-tumble play and rejection of male stereotypical toys, games, and activities; in girls, rejection of urinating in a sitting, position, assertion that she has or will grow a penis, or assertion that she does not want to grow breasts or menstruate, or maked aversion toward normative feminine clothing.

In adolescents and adults, the disturbance is manifested by symptoms such as preoccupation with getting rid of primary and secondary sexcharacteristics (e.g., request for hormones, surgery, or other procedures to physically alter sexual characteristics to simulate the other sex) or belief that he or she was born the wrong sex.

C. The disturbance is not concurrent with a physical intersex condition.

D. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if (for sexually mature individuals):

Sexually Attracted to Males; Sexually Attracted to Females; Sexually Attracted to Both; Sexually Attracted to Neither

• EPIDEMIOLOGY

Cross-gender behavior of clinical significance, but not meeting criteria for gender identity disorder, is common in 5- to 12-year-old boys referred to a psychiatric clinic for various other emotional and behavioral symptoms.

The estimated prevalence of true gender identity disorder in children is in 100,000.

•ETIOLOGY

Various causative factors for gender identity disorder of children have been proposed, including biological, psychodynamic, and psychosocial models.

No genetic or hormonal factor has yet been identified.

- Family characteristics, such as:
  - lack of appropriate gender role modeling;

- parental wish for opposite-sex child;
- parent treating child as opposite sex;
- absent, distant or depressed same-sex parent;
- disturbed mother-child relationship;
- mother who is dissatisfied with her own gender or gender role;
- family violence, have been identified in individual patients.

Interestingly, children raised in transsexual or lesbian households show no evidence of problems in gender identity, role behavior, or sexual object choice.

Girls whose mothers were treated with masculinizing progestogens during pregnancy are more likely to be tomboys, preferring intense outdoor play and male peers and avoiding doll play, but they are not more likely to become transsexual or homosexual.

COURSE AND PROGNOSIS

Peer relationships are usually significantly impaired in children with gender identity disorders. Children ages 6-12 years have rigid gender role expectations and do not tolerate a child who does not conform to these, although "tomboys" have somewhat more latitude than effeminate boys, who are teased and ostracized. The child's pervasive discomfort also interferes with school performance.

Gender identity disorder of childhood may coexist with oppositional defiant disorder, conduct disorder, or a mood or anxiety disorder.

Follow-up studies of clinical populations show that some effeminate boys become transsexual adults and generally have serious difficulty in social adaptation. A larger number of boys with gender-atypical characteristics develop a homosexual orientation in adulthood and do not carry a psychiatric diagnosis. Clinically referred girls also show a variety of gender and sexual orientations as adults, although nonreferred "tomboys" almost always develop more typically feminine interests at puberty. Early treatment of gender identity disorder is important because the treatment of transsexualism in adults is medically and psychiatrically difficult.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Pediatric evaluation is indicated to rule out a physical or hormonal abnormality such as hermaphroditism, congenital adrenal hyperplasia, or androgen-insensitivity syndrome. Children with gender identity disorder must be differentiated from those who simply do not meet the gender role expectations of their families or their environment.

TREATMENT

The best documented treatment for gender identity disorder of childhood is behavior modification. Changes in role behavior can lead to changes in gender identity, as well as improved self-esteem and decreased depression. Techniques include:

- social reinforcement of gender-concordant behaviors,

- ignoring cross-gender behaviors,
- reinforcement of play with appropriate toys,
- response cost for gender-inappropriate speech or play,
- modeling and shaping of absent skills.

Generalization is not automatic, and results are best if the program is conducted both at home and in school, as well as at the clinic, and several behaviors are addressed.

Years of intensive treatment may be required.

Cognitive-behavior self-monitoring of mannerisms may also be useful.

Dynamic individual psychotherapy has been successful in some patients.

Family therapy is often indicated, as is treatment of coexisting mood or conduct disorders.

Parent counseling is always needed.

TOURETTE'S DISORDER AND OTHER TIC DISORDERS

CLINICAL DESCRIPTION

Tic disorders are stereotyped abnormalities of semi-involuntary motor movement or vocalization, presumably relating to dysfunction in the basal ganglia. Symptoms of these disorders are subject to moment-to-moment influences from environmental and internal stimuli. Although experienced as involuntary, they may be temporarily suppressed by conscious effort. All forms of tics are often exacerbated by stress. They typically - diminish markedly during sleep and may become attenuated during absorbing activities.

Tic disorders are subdivided into transient tic disorder, chronic tic disorder, and Tourette's disorder.

Neurological symptoms are typically observed, including "soft" signs (50%) or choreiform movements (30%). Approximately 50% of patients show abnormal EEG findings, particularly immature patterns (excess slow waves and posterior sharp waves). Computed tomography scans are usually normX. See attached DSM IV criteria.

Diagnostic criteria for Tourett's Disorder

A. Both multiple motor and one or more vocal tics have been present at some time during the illness, although not necessarily concurrently. (A tic is a sudden, rapid, recurrent, nonrhythmic, stereotyped motor movement or vocalization.)

B. The tics occur many times a day (usually in bouts) nearly every day or intermittently throughout a period of more than 1 year, and during this period there was never a tic-free period of more than 3 consecutive months.

C. The disturbance causes marked distress or significant impairment in social, occupational, or other important areas of functioning.

D. The onset is before age 18 years.

E. The disturbance is not due to the direct physiological effects of a substance (e.g., stimulants) or a general medical condition (e.g., Huntington's disease or postviral encephalitis).

EPIDEMIOLOGY

Simple tics are seen in up to 25% of boys.

Tourette's disorder has a prevalence of about 30 in 100,000.

Tourette's and the other tic disorders show a male predominance of 3-10:1.

ETIOLOGY

Two-thirds of all relatives of Tourette's disorder patients have transient and/or chronic tics. A familial predisposition to both tic disorders and obsessive-compulsive disorder appears to be guverned by a single gene with autosomal dominant transmission. There may also be a link to ADHD.

A "gender threshold effect" is observed for Tourette's disorder. Both the male predominance and the higher risk of Tourette's disorder in boys than in girls at a given level of genetic loading can be explained by a lower penetrance in girls for the genetic form of the disorder. When all forms of the disorder are considered, the penetrance for males is 100% and for females is 71%. Within affected families, males are more likely to have tic disorders, and females to have obsessive-compulsive disorder. About 10% of individuals with Tourette's disorder have a nonfamilial version, which is a phenocopy similar to the genetic form.

Stimulants and some antidepressants may aggravate tics but it is unclear whether they can cause tic disorders or hasten the appearance of tics in a vulnerable child, or whether their use simply coincidental with the onset of tics.

COURSE AND PROGNOSIS

Transient tic disorder, whether single or recurrent episodes, usually begins in middle childhood (ages 5—10 years) or early adolescence. If recurrent, the frequency and severity of symptoms typically abate over the course of years. Chronic motor or vocal tic disorder also appears at ages 5-10 years. In about two-thirds of patients, symptoms cease in adolescence, but may persist in mild form for years or even decades.

Tourette's disorder is a lifelong disease that shows a typic pattern of waxing and waning over time. Frequency and severity of symptoms vary widely, and clinical presentation can change during the course of development. Onset is typically during childhood (ages 2—I 3 years) and is rarely postpubertal. In retrospect, symptoms of ADHD often appear before any tics. Average age onset of motor symptoms is £.years, typically first with a single motor tic, with a rostrocaudal progression over time (head before a trunk and limbs). At an average age of 11 years phonic and vocal tics may appear, along with obsessions and compulsive behavior. Vocal tics may start as a single syllable, progressing to longer exclamations and occasionally to complex gestures. Classic coprolalia is observed in 60% of patients with Tourette's disorder, with an initial appearance typically in early adolescents. Copropraxia (complex obscene gestures) may appear later, as coprolalia resolves. Complex motor tics may appear purposeless, or may be camouflaged by being blended into purposeful

movements. Sometimes these complex motor tics may be selfdestructive (e.g., scratching or cutting) or violent (e.g., temper tantrums, assaults).

Complications of Tourette's disorder generally include impaired self-esteem and social performance. Teasing, shame, self-consciousness, and social ostracism are standard features of these patients' lives, leading to reluctance to involve themselves in social demanding situations. Particularly if the symptoms are se severe, these patients may avoid entering intimate relationships, marriage, and other interpersonally gratifying activities. The rate of unemployment in adults with Tourette's disorder is reportedly as high as 50%.

The tic disorders vary in intensity over time, usually increasing during psychosocial stress, intrapsychic conflict, and positive or negative emotional excitement. Symptoms may be exacerbated at the start of the school year, as a result of physical fatigue, or if parents separate or divorce. Tics typically decrease in frequency and severity during focused mental activity, concentration, or sudden alerting, and disappear during sleep.

ADHD and conduct disorder are frequently associated with Tourette's disorder, particularly in psychiatric clinic samples. Obsessive-compulsive disorder or obsessive or compulsive symptoms are common.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Individual tics may be seen occasionally in normal children, but they persist for at least 2 weeks.

Neurological evaluation is needed to rule out other movement disorders (e.g., Wilson's disease). An assessment of baseline dyskinesias is needed before the start of neuroleptic drug treatment. An EEG is helpful in ruling out myoclonic seizures and other neurological disorders. Use of prescribed and over-the-counter medications should be determined.

Psychiatric evaluation focuses on concomitant anxiety, mood, behavior, and developmental disorders and identifying stressors that may be aggravating symptoms. Psychometric evaluation documents level of intellectual functioning, school achievement, and possible learning disabilities (specific developmental disorders).

School reports including academic performance, general behavior, severity of tics, and social skills are useful. The child's self-consciousness, management of teasing and social ostracism, and assertiveness are assessed. Evaluation of tic disorders in relatives may be considered.

The clinical boundaries between Tourette's disorder, ADHD, and obsessive- compulsive disorder are blurred in many patients who combine features of these disorders.

TREATMENT

For transient tics, treatment is not usually required.

It is helpful to advise the family to reduce attention to the symptom and avoid criticizing the child. The patient and family may be provided with education and reassurance and encouraged to return if symptoms persist.

For chronic tic disorder, behavior, drug, or psychosocial interventions are used. Behavioral relaxation techniques, medication (minor tranquilizers, low doses of major tranquilizers), brief psychotherapy may be helpful for anxiety management and tic control.

For Tourette's disorder, psychosocial interventions often should precede pharmacologic treatment and may be far more important. In many cases, the control of tics is less important than the treatment of the accompanying attentional, behavioral, obsessive or compulsive symptoms

Education of parents, teachers, peers, and the patient ameliorates the consequences of less severe tics.

Psychotherapy may help an individual deal with the stigma of illness, promote selfesteem, improve interpersonal comfort and social skills, and reduce anxiety and symptom severity.

Behavior modification at home and school is often require to deal with symptoms of ADHD and oppositional defiant disorder.

Special education placement may be indicated.

Low dosage of nonsedating neuroleptics or clonidine can be considered.

Lithium or calcium antagonists may be used when the usual medications are ineffective.

- The use of stimulants to treat symptoms of ADHD in the presence of tics is highly controversial, but most experts recommend avoiding the risk of agravating tics.

ELIMINATION DISORDERS

FUNCTIONAL ENCOPRESIS

Clinical description. This disorder includes fecal soiling of clothes or excretion in inappropriate places, occurring after age 4_years when full bowel control is developmentally expected. Medical evaluation for structural and other nonfunctional abnormalities must be obtained before labeling as functional. See the following table DSM-IV criteria.

Diagnostic criteria for Encopresis

A. Repeated passage of feces into inappropriate places (e.g., clothing or floor) whether involuntary or intentional.

B. At least one such event a month for at least_3jnonth.

C. Chronological age is at least 4 years (or equivalent developmental level)

D. The behavior is not due exclusively to the direct physiological effects of a substance (e.g., laxatives) or a general medical condition except through a mechanism involving constipation.

Code as follows:

787.6 With Constipation and Overflow Incontinence

307.7 Without Constipation and Overflow Incontinence

Daytime encopresis is much more common than nocturnal. In half of these patients, bowel control is not yet learned, so the encopresis is termed primary. In secondary encopresis, the child initially learned bowel control, had been continent for at least 1 year, and then regressed.

Certain children with encopresis show neurodevelopmental symptoms, including inattention, hyperactivity, impulsivity, low frustration tolerance, and dyscoordination.

Typically, children with functional encopresis often experience shame and embarrassment, feel "dirty," and have low self-esteem. They may suffer accusations from parents and siblings, fear discovery by peers, and hide physically and emotionally.

Epidemiology.

- Prevalence of encopresis is about 1,5% after age 5 years, dimi with age and becoming rare in adolescents.
- There is a 4:1 male predominance.
- Higher rates are observed among individuals with moderate or severe mental retardation and in lower socioeconomic classes.

Etiology.

Functional encopresis may result from:

- painful defecation,
- inadequate or punitive toilet training,
- physical discomfort associated with inadequate physical support during toilet training (in feet do not touch the ground),
- improper management of toilet-related fears,

Stress-related factors appear causative in one half of patients related with secondary encopresis.

If involuntary encopresis can be related to constipation, impaction, or retention (with overflow incontinence). If soiling is deliberate, the child is typically impulsive or hostile; antisocial or major psychiatric disorders may be associated. Smearing of feces may be accidental or voluntary.

Individual and family psychopathology are over represented in functional encopresis. Although there is a familial occurrence of functional encopresis, with 15% of fathers having had childhood encopresis, there is no evidence of a genetic etiology.

Course and prognoses.

Secondary encopresis usually starts by age 8 years.

Substantial follow-up studies are unavailable, bur psychiatric or medical comorbidity may be the primary determinant of prognosis.

Behavior problems such as conduct disorder are more common in the psychiatrically referred population than in those seen by pediatricians.

In the psychiatric population, 25% of children with functional encopresis also have functional enuresis;
In the pediatric population, this diagnostic overlap is less common. Some children withhold both urine and feces, and may have megabladder and megacolon.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

- A detailed history of bowel function, nature and pattern of spiling, previous attempts to trait or treat, and bathroom habits or environment is needed. Initial medical evaluation is Tequired to evaluate possible structural abnormalities and/or fecal impaction, and may require barium enema.

Urinalysis will detect an associated urinary tract infection, common in girls with encopresis

Medical causes of fecal incontinence include thyroid disease, hypercalcemia, lactase deficiency, overeating of fried and fatty foods, rectal stenosis, anal fissure, congenital aganglionic 'megacolon (although Hirschsprung's disease is usually associated with large feces rather than incontinence), and neurologic disorders such as myelodysplasia. These medical causes of fecal soiling exclude the diagnosis of functional encopresis.

Retention of stools may be initiated by painful defecation, caused by diaper rash in infants, constipation, or anal fissure. Once retention is initiated by emotional or medical factors, if may then bemiintalned independently.

Pathophysiologic mechanisms include altered colon motility and contraction patterns, stretching and thinning of colon walls (megacolon), and decreased sensation or perception. Liquid stool leaks around the impaction, and the child is unaware and unable to exert control.

Psychiatric evaluation includes assessment of associated psychiatric disorders, including mental retardation, ADHD, oppositional defiant disorder, conduct disorder, mood disorders,
and psychosis.

Oppositional children may soil willfully.

Children with ADHD do not plan ahead, so may be caught by an urge to defecate when a bathroom is not available.

Timid or phobic children maybe intimidated by perceived or real dangers or humiliations in school bathrooms and avoid defecation, only to have an "accident" on the way home.

Treatment.

Most cases of functional encopresis can be treated in a pediatric model, but more complex cases may need psychiatric intervention.

Education of the child and parents regarding bowel function is essential.

For encopresis without constipation, a behavioral program is used that rewards sitting on the toilet and later moving the bowels appropriately (with or without a negative consequence for soiling).

For children with severe stool retention, impaction, and loss of bowel tone, an initial bowel clean out (e.g., enemas or suppositories) followed by bowel "retraining" (e.g. mineral oil, high roughage diet, development of a toileting routine, use of a mild suppository if necessary) may be used in conjunction with the behavioral program. Repeated administration of enemas by parents is harmful to the parent-child relationship.

In resistant cases, or if aggressive pediatric treatment becomes counterproductive, individual and family psychiatric intervention are indicated.

The focus of treatment then shifts to the associated psychiatric disorders.

FUNCTIONAL ENURESIS

Clinical description. Urinary incontinence may be seen in youngehildren and occasionally in older children after the completion of toilet training as normal developmental phenomen.

FunctionaPenuresis is diagnosed when the frequency of medically unexplained urinary incontinence exceeds developmental (age-specific) expected norms. See the following table for DSM-lV criteria.

Diagnostic criteria for 307.6 Enuresis

A. Repeated voiding of urine into bed or clothes (whether involuntary or intentional).

B. The behavior is clinically significant as manifested by either a frequency of twice a week for at least 3 consecutive months of the presence of clinically significant distress or impairement in social, academic (occupational) or other important areasof functioning.

C. Cronological age is at least 5 years (or equivalent developmental level).

D. The behavior is not due exclusively to the direct physiological effect of a substance (e.g.. diuretic) or general medical condition (e.g., diabetes, spina bifida, seizure disorder).

Specify type:

Nocturnal only.

Diurnal only.

Nocturnal and diurnal.

In functional enuresis bed wetting is more common then daytime urinary incontinence. Nocturnal enuresis typically occurs 30 minutes to 3 hours after sleep onset, but may occur at any time during the night. Most children with daytime enuresis also have nocturnal enuresis.

In 80% children with enuresis, bladder control has not yet been attained and the enuresis is primary.

In 20% urinary incontinence is secondary reappearing after competent functioning is maintaining for ar least 1 year.

Epidemiology

at age 5 years: 7% of boys, 3% of girls;

the male predominance degreases with age;

at age 10 years 3% of boys and 2% of girls;

in adulthood general prevalence is 1%;

a higher prevalence is on moderate or severe mental retardetion.

Etiology.

Approximately 70% of children with enuresis (particularly boys) have a first-degree relative with functional enuresis.

Studies of monozygotic and dizygotic twins show a strong genetic factor.

- A "maturational" etiology is suggested in some cases by the findings of small volume of voidings, short stature, low mean bone age, and delayed sexual maturation in adolescence.

The presentation is usually primary, and there is a spontaneous remission rate of 15% per year.

Common physiologic causes of diurnal enuresis in girls are vaginal reflux of urine, "giggle incontinence," and urgency incontinence.

Enuresis is occasionally caused by psychiatric disorders:

Anxious children may experience urinary frequency, resulting in daytime incontinence if toilet facilities are not readily available, or if the child is fearful of certain bathrooms.

- In oppositional defiant disorder, refusal to use the toilet may be part of the child's battle for control.
- Many children with ADHD wait until the last minute to urinate, then lose control on the way to the bathroom.
- Voluntary enuresis may imply psychopathology (oppositional defiant disorder, conduct disorder, or psychosis), but may be difficult to identify in individual patients. Secondary enuresis may be related to stress, trauma, or psychosocial crisis, such as the birth of a sibling, the start of school, a move, hospitalization, or parental absence. Among these patients, enuresis is typically equally prevalent in boys and girls.

Children with primary enuresis seen by pediatricians rarely have psychopathology, other than affective symptoms secondary to the wetting.

COURSE AND PROGNOSIS.

Primary enuresis has a high rate of spontaneous remission.

Only about 1% of boys (and fewer girls) still have this condition at age 18 years.

Onset of the disorder in adolescence, however, may signify more psychopathology and less favorable outcome.

Complications include: embarrassment,anger from and punishment by caretakers, teasing by peers, avoidance of overnight visiting and camp, social withdrawal, angry outbursts.

If not properly managed, these may have more impact on long-term outcome than the enuresis itself.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

Initial medical assessment is required to rule out the various medical causes of enuresis which are more common in secondary am diurnal enuresis than in primary or nocturnal enuresis:

urinary tract infection

urethritis: bubble bath and sexual abuse

diabetes mellitus

diabetes insipidus

sickle cell trait

seizure disorders

neurogenic bladder

physiologic genitourinary abnormality

- congenital malformation of genitourinar tract
- urinary obstruction.

In the absence of suggestive findings on the history and physical examination, a urinalysis is a sufficient screen.

Psychiatric evaluation of the child with enuresis and his or her family includes assessment of associated psychopathology, recent psychosocial stressors, and evaluation of family concern and management of the symptoms. Psychological and developmental evaluation can identify the child who is not sufficiently mature to attain continence.

Treatment.

- For younger children who wet only at night the most useful strategy is to minimize symptoms while awaiting maturation by discouraging the parents from punishing or ridiculing the child.
- For older children who are motivated to stop bed wetting a monitoring and reward procedure may be effective: behavioral program (bladder training, delaying bladder emptying, interruption of stream while urinating).
- If simple intervention is unsuccessful an urine alarm is recommended.
- Children with secondary enuresis may need treatment of comorbid psychopatology and management of psychosocial stressor.
- Low dosage of tricycle antidepressant can be helpful in the short term.
- Psychotherapy is indicated only for the uncommon patient in whom the symptom is interpersonally cathected (for example, into an oppositional struggle, or into expressing rage against parents) or in whom there is clear associated psychopathology.

STUTTERING

CLINICAL DESCRIPTION

Stuttering, a disruption of normal speech flow, is characterized by involuntary and irregular hesitations, prolongations, repetitions, or blocks of sounds, syllables, or words. The dysfluency Tnay show transient worsening during periods of performance anxiety or communicative stress. The symptoms are often absent during singing, reading aloud, talking in unison, or talking to pet or inanimate objects. Stutterers are extremely aware of their symptoms and cannot readily improve their speech by slowing or by focusing attention on their speech rate or rhythm.

Diagnostic criteria for 307.0 Stuttering

A. Disturbance in the normal fluency and time pattering of speech (inappropriate for the individual age) characterized by frequent occurrences of one or more of the following:

(1) Sound and syllable repetitions

(2) Sound prolongation

(3) Interjection

(4) Broken words (e.g., pauses within a word)

(5) Audible or silent blocking (filled or unfilled pauses in speech)

(6) Circumvolutions (word substitutions to avoid problematic words)

(7) Words produced with an excess and physical tension

(8) Monosyllabic whole-word repetitions (e.g., "I-I-I see him")

B. The disturbance in fluency interferes with academic or occupational achievement or with social communication.

C. If a speech motor or sensory deficit is present the speech difficulties are in excess of those usually associated with these problems.

Coding note: If a speech motor or sensory deficit or neurological condition is present, code the condition on Axes III.

EPIDEMIOLOGY

Approximately 2—4% of children and 1% of adolescents stutter.

There is a male predominance of 3—4:1.

ETIOLOGY

Etiological theories of stuttering include:

genetic,

neurological

psychodynamic,

behavioral concepts, probably reflecting several etiological subtypes.

Sixty percent of stutterers have a familial component, with the disorder appearing in about 20—40% of first-degree relatives (especially males).

A strikingly higher concordance in monozygotic than in dizygotic twins suggests a genetic factor. Current data fit a polygenic model with lower penetrance in females.

Left handedness or QTixeddominQce is over represented in stutterers (and their ives), suggesting that stuttering may be associated with anomalous cortical organization. Stuttering is also associated with mental retardation. In rare cases, stutterlike dysfluency can be caused by psychotropic medications (e.g., tricyclic antidepressants, neuroleptics, lithjum, allEEazolam).

Behavior theories have focused on the role of reinforcement in maintaining dysfluencies, and secondary aggravation by frustration. There is no evidence for anxiety, neurosis,.or family dynamics as causal factors.

COURSE AND PROGNOSIS

For toddlers, stuttering is usually a transient developmental symptom lasting less than 6 months, but 25% of patients with early onset of stuttering have persistent stuttering beyond age 12 years.

- Stuttering usually begins at ages 2—4 years, sometimes atages 5—7 years, and rarely during adolescence. Spontaneous improvement occurs in 50-80% of patients.

At the onset of illness, the child is usually unaware of the symptom. The disorder typically waxes and wanes during childhood, either gradually improving, or worsening and leading a chronic course.

Complications include:

- fearful anticipation,
- eye blinking,
- involuntary tension of the jaw and face muscles,
- tics,
- avoidance of problematic words and situations

Negative reaction by family and peers (embarrassment, guilt, anger, teasing), may affect self-image, social skills, and language development and lead to academic impairment, occupational problems, and social withdrawal.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Evaluation of stuttering includes:

-a full developmental history -neurological and audiological examinations.

-referral to a speech and language pathologist

-it is helpful to assess the dysfluency in monologue, conversation, play

Behavior assessment documents secondary restrictions in social interactions and activities.

TREATMENT

Speech therapy involves:

- intensive training of fluent speech skills,
- fostering self-esteem and social assertiveness,
- elements of behavior therapy, such as:
  - modifying environmental and conversational factors that trigger stuttering,
  - relaxation,
  - role-playing feedback,
  - dealing with accessory body movements,
  - imitation, /v

- rhythm control,
- practice in speaking (reading aloud, choral reading, conversing),
- talking in different settings (alone, in group, in front of a classroom, on a telephone)

and with different, people (parents, relatives, friends, strangers).

Education and counseling of family members are advised.

Psychotherapy is not generally indicated, but might be considered for secondary symptoms or associated problems. Antianxiety drugs have minimal value.

SELECTIVE MUTISM

CLINICAL DESCRIPTION

Children with selective mutism do not speak in one or several of the major environments in which they live, despite having the ability to speak and to comprehend spoken language. Typically, speech is normal at home when the child is alone with parents and siblings, but partial or total muteness appears in the presence of teachers, peers, and strangers, or selectively in unfamiliar places or particular social situations. When separated from a familiar or comfortable setting, these children might use gestures, nods, monosyllabic responses, written notes or whispers, but avoid full vocalization.

Social behavior is usually impaired. Many of these children are shy, timid, anxious, submissive, and excessively dependent. They cling to their parents, sulk with strangers, throw temper tantrums, and are prone to immature behaviors when under stress. At times, these children may be surprisingly oppositional, demanding, and disobedient. School absenteeism, problems with separation, and obsessive or compulsive features are common.

Four subtypes of elective mutism have been distinguished on the basis of psychodvnamic and behavioral features. The symbiotic form, the largest subgroup, involves a dominant mother who is openly jealous of the child's relationships with other people, a passive or nonverbal father, and a child who appears submissive but can be intensely manipulative.

Passive-aggressive children with selective mutism use silence in a_defiant and hostile manner, display antisocial and often aggressive behaviors, and generally have parents with overfantisocial features.

Reactive (or perhaps depressed) children show depressive features and social withdrawal. A parent suffers from a mood disorder, and there is often a family history of shyness.

Speech phobic children appear literally afraid to hear their own voices, show autonomic excitatory reactions in response to hearing themselves talk (even on tape), and exhibit obvious ritualistic and compulsive behaviors. See attached DSM IV criteria.

Diagnostic criteria for Selective Mutism

A. Consistent failure to speak in specific social situations (in which there is an expectation for speaking, e.g., at school) despite speaking in other situations.

B. The disturbance interferes with educational or occupational achievement or with social communication.

C. The duration of the disturbance is at least Ijnonth (not limited to the first month of school).

D. The failure to speak is not due to a lack of knowledge of, or comfort with, the spoken language required in the social situation.'

E. The disturbance is not better accounted for by a Communication Disorder (e.g., Stuttering) and does not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder.

EPIDEMIOLOGY

The prevalence of selective mutism is in the range of 30—80 cases per 100,000, but broader definitional criteria could increase this estimate tenfold.

There is a slight female predominance.

Increased prevalence in immigrant families is reported.

ETIOLOGY

The selective appearance of the symptoms of selective mutism implies the operation of psychodynamic factors:

A posttraumatic etiology is supported by the high incidence in the histories of physical and sexual abuse, parental violence, early trauma to the mouth or face, and punishments such as slapping the face or washing out the mouth, especially during the period of speech development.

Psychodynamic theorists have emphasized mechanisms involving oral inhibition, anal control, separation anxiety, and abandonment fears.

The role of separation anxiety is consistent with the observed resistance to separation, as well as the strong maternal ties, family history of mood disorder, and a history of geographic or cultural change.

Overly strong emotional ties to the mother are emphasized in many case reports. There are frequent descriptions of parental fear, anxiety, shyness, and over protectiveness but also of parental violence, aggressivity, and use of silence as a weapon ofangerricf a means of coercion.

Approximately one-third of children with selective mutism also have language disorders, and one-half have a speech disorder or.delayed speech development.

There is an increased prevalence of mental retardation and neurological disorders. These associations suggest a neurqdevelgpmental etiology or that developmental disorders may aggravate communication impairments.

COURSE AND PROGNOSIS

Selective mutism typically starts at ages 3-5 years when developmental normal children may still show brief periods of mutism on meeting strangers or in new settings (e.g., staffing kindergarten). Although early "shyness" may be identified retrospective selective mutism is typically diagnosed at ages 5—10 years, usually after symptoms appear at school. Symptoms may last for weeks, months, or years.

Some patients with onset in adolescence completely cease speaking and show prominent passive-aggressive or antisocial features.

Complications of selective mutism include: academic underachievement, impaired peer relations, reliance on the secondary my gains of illness (excessive protection and personal attention) The child's persistent silence may lead to inappropriate special class and school placements.

Prognosis varies, with a minority of cases becoming chronic, despite treatment. Abnormal social behavior, interpersonal manipulations, shyness, and oppositionality may persist beyond the period of muteness.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

A full psychiatric evaluation of the child and parents, neurological assessment for possible brain damage, psychometric assessment for mental retardation, and speech and language evaluation, including familial patterns of communication, silence, anger, are warranted.

The possible presence of physical and sexual abuse, depression, antisocial behavior, and shyness should be evaluated in the child and the family.

The differential diagnosis of failure to speak includes:

hearing impairment,

mental retardation,

pervasive developmental disorders,

developmental language disorder,

aphasia,

schizophrenia and conversion disorder.

Only patients in the latter two categories may show speech in some situations and not others.

TREATMENT

Behavior therapy (using contingency management, positive reinforcement, desensitization, and assertiveness training).

Parent counseling.

- Short-term therapy may be effective.

Resistant patients may require hospitalization.

Speech and language therapy are often indicated.

Although accommodations to the child's muteness are often made by teachers and parents, it is better to maintain a clear expectation that the child talk and communicate, at least for a structured period of each session.

The parents, especially the symbiotic parent, should be explicit that the child is expected to talk at school and in therapy. For certain children, minimizing the "directness" of verbal interaction can reduce the subjective feeling of threat or aggressivity that may be experienced in communicating.

Therapists have employed methods such as covering their own mouths during speech, reducing eye contact with the child, using averted body positions, speaking in gestures (pantomime), simple questioning that requires

one-word responses or tapping of the therapist's hand, and silent mouthing of words. In general, parents should be discouraged from reinforcing the child's passivity or contributing to the secondary gain.

The treatment of anxiety and fear associated with communication, management of separation anxiety, promotion of autonomy, and enhancement of personal assertiveness may involve treatment of both the child and the parents. Once the child's speech is improving, associated psychiatric disorders may continue to require clinical attention.

IDENTITY DISORDER

CLINICAL DESCRIPTION

Identity disorder was first delineated in DSM-IV has been little studied. It requires both a failure to develop an integrated, coherent, acceptable sense of self compared with what would be expected age and IQ and severe subjective distress regarding this failure.

EPIDEMIOLOGY

Identity disorder is believed to be more common now than in the past, partly because of the dramatic increase in ideological, social, and occupational choices available to young people.

ETIOLOGY: Causative factors have not been studied.

COURSE AND PROGNOSIS

Age at onset of identity disorder is most commonly in late adolescence, when identity independent of the family is normally established, at least in a preliminary way, and important choices for adult life are made. Academic, vocational, and social functioning may be impaired, acutely or chronically.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Individual, family, and social context and expectations must be considered. Identity disorder may be considered to fall on a spectrum between normal adolescent developmental conflicts and the pervasive disturbance of borderline personality disorder.

TREATMENT

Individual psychotherapy appears to be the first-choice treatment for identity disorder.

Family therapy as an adjunctive treatment could be considered, especially if the young person is still living at home.

REACTIVE ATTACHMENT DISORDER OF INFANCY OR EARLY CHILDHOOD

CLINICAL DESCRIPTION

Reactive attachment disorder encompasses both decreased and inappropriately increased social interactiveness after inadequate care or trauma in infancy or early childhood.

Diagnostic criteria for 313.89 Reactive Attachment Disorder of Infancy or Early Childhood

A. Markedly disturbed ofolevelopmentally inappropriate social relatedness in most contexts beginning before age 5 years as evidenced by either (1) or (2):

(1) Persistent failure to initiate or respond in a developmentally appropriate fashion to most social interaction, as manifest by excessively inhibited hypervigilant, or highly ambivalent and contradictory responses (e.g., the child may respond to caregivers with a mixture of approach, avoidance and resistance to comforting, or may exhibit frozen watchfulness).

(2) Diffuse attachment as manifest by indiscriminate sociability with marked inadaptability to exhibit appropriate selective attachments (e.g., excessive familiarity with relative strangers or lack of selectivity in choice of attachment figures.

B. The disturbance in criterion A is not accounted for sole by developmental delay (as in Mental Retardation) and does not meet criteria for a Pervasive Developmental Disorder.

C. Pathogenic care as evidenced by at least one of the following:

(1) Persistent disregard of the children basic emotional needs for comfort, stimulation and affection.

(2) Persistent disregard of the children basic physical needs.

(3) Repeated changes of primary caregiver that prevent formation of stabile attachments (e.g., frequent changes in foster care).

D. There is a presumption that the care in criterion C is responsible for the disturbed behavior in criterion A (e.g., disturbances in criterion A began following the pathogenic care in criterion C).

Specify type:

Inhibited type: if criterion A1 predominates in the clinical presentation.

Disinhibited type: if criterion A2 predominates in the clinical presentation.

The related pediatric diagnosis of failure to thrive (FTT) describes children whose growth in height and weight and development are delayed. “Organic” FTT can result from chronic physical illness (e.g., congenital acquired immunodeficiency syndrome [AIDS]), neurological disease, sensory deficit, or virtually any serious pediatric disease.

Reactive attachment disorder is a subset of “nonorganic” FTT, which includes:

pathological food refusal,

disorders of regulation of sleep and feeding,

protein-calorie malnutrition,

social and emotional factors interfering with adequate nutrition.

Any deviation in development may be labeled as reactive attachment disorder if preceded by clear failures in child care. Various behavioral, cognitive, and affective presentations may be seen at different ages.

In early infancy, diagnosis is based on the failure to achieve developmental expectations:

-tack of eye tracking or responsive smiling by age 2 months,

-failure to play simple games or to reach out to be picked up by age 5 months,

-failure to show overt behavioral signs of attachment and bonding to a parent by age 8 monthf.

Infants appear lethargic, show little body movement or activity, have excessive and disrupted sleep, gain weight slowly, and resist being held.

In childhood, odd social responsiveness, weak interpersonal attachment, inappropriate excitability, and mood abnormalities are seen. The children may appear withdrawn, passive, and disinterested in people or, alternatively, may display excessive interest, overly rapid familiarity, inappropriate touching, clinging, or immediate emotional involvement that seems odd or unusual.

EPIDEMIOLOGY

Approximately 1—5% of pediatric hospitalizations are due to non-organic FTT. Epidemiological data concerning the subgroup of reactive attachment disorder are unavailable.

ETIOLOGY

The etiology of reactive attachment disorder is written into the DSM-IV definition.

prior abuse, neglect, or impaired care taking is required;

a causal connection to symptoms is inferred;

disruption of interpersonal attachment is implied by the disease label.

Parents or caretakers may suffer from major depression, psychosis, substance abuse, or mental retardation. They may be poor, uneducated, or isolated from social and emotional supports. They may be hostile or indifferent to the child, or simply have insufficient skills, supports, and frustration tolerance to deal with a “difficult" child. Family life may be grossly disturbed.

COURSE AND PROGNOSIS

The course of reactive attachment disorder may vary from spontaneous remission to malnutrition, infection, or death. Either nutritional or psychosocial deprivation may result in long-term behavior changes, hyperactivity, short stature, and lowered IQ.

If emotional deprivation continues but enforced feeding is provided, children may show improved body growth but with “depression” and developmental delays.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Reactive attachment disorder is diagnosed by observation of patent-child interactions, home visit, and symptomatic response to adequate care.

Assessment of caretaker-child interaction includes:

observation of physical and emotional nurturance, including the capacity for empathy, appropriateness of level and timing of stimulation,

attentiveness to the child’s behavior, whether expectations of the child are appropriate to developmental level,

caretaker emotional reactions to the child such as anxiety, anger, or indifference.

A home visit is generally indicated to evaluate the adequacy of housing, safety, and nutrition.

Psychiatric evaluation of the parents is essential.

Physical or sexual abuse or neglect may not be quickly or easily identifiable.

For infants or very young children, hospitalization is useful for diagnosis. Removal from the home environment may permit establishment of normal feeding and sleeping patterns, and evaluating and remediating parental care giving capacity.

Medical assessment is required to rule out chronic physical illness that may result in organic FTT, sleep and feeding disorders, food refusal, malnutrition,neurological disease, and sensory deficit.

A major clinical improvement in response to hospitalization or treatment is considered confirmation of the diagnoses. Clinical non responsiveness implies the presence of a different disorder, or that extreme medical complications with physical damage occurred before treatment.

Psychiatric disorders that should be considered as alternatives are pervasive developmental disorder and depression.

TREATMENT

Basic medical care, provision of adequate care taking, parent education, and parent psychiatric treatment WP nftpn needed.

Legal intervention may be indicated.

Given the complexity of medical and psychiatric interventions, hospitalization is often justified.

If treatment is initiated long after the period of abuse and. neglect, when early attachment problems are part of the past history and abnormal social behavior is prominent, then standard psychiatric evaluation and treatment of the child and the parents.

Reference:

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5. "ADULT" DISORDERS THAT MAY BEGIN IN CHILDHOOD OR ADOLESCENCE

PSYCHOACTIVE SUBSTANCE USE DISORDERS

CLINICAL DESCRIPTION

The DSM-IV criteria for the psychoactive substance use disorders do not change with age. The continuum of adolescent substance use ranges from nonusers, through experimental and casual users, to compulsive users. The line between use and abuse is I crossed more easily by young persons than by adults.

EPIDEMIOLOGY

Although a majority of young people experiment with alcohol or drugs, use of cocaine I decreased; the use of "crack," LSD, heroin, and opiates showed little change; inhalant abuse continued to increase; and there was no decrease in alcohol consumption or cigarette j smoking.

ETIOLOGY

Psychosocial risk factors for psychoactive substance abuse are:

rebelliousness

aggression

impulsivity

low self-esteem

elementary school underachievement failure to value education absence of strong religious conviction peers with behavior problems who use drugs alienation from parents

family lacking in clear discipline, praise and positive relationships experimentation with drugs before age 15 years

Children of substance abusers appear to be particularly vulnerable, probably due to a combination of genetic and family dynamic factors and attitudes toward substance use. Genetic contributions to alcoholism in males are particularly strong. Peer influence mediates both initiation and maintenance of substance use, as well as avoidance of drugs. Disturbances of.behavior and emotions commonly predate the onset of drug use.

Substances may be used to produce positive feelings and avoid unpleasant ones, relieve tension and stress, reduce disturbing emotions, alleviate depression or anxiety, and gain peer acceptance. Drug use is more correlated with school failure, low perceived risk of I smoking, and peer use of marijuana than with depression. Whatever the contributing factors, | any illegal use of substances constitutes risk-taking behavior.

COURSE AND PROGNOSIS

Adolescence is the critical period for initiation of drug abuse. Onset is rare in adulthood, at least for nonprescription drugs. Each stage serves as a gateway to the next from abstinence to beer or wine and cigarettes, to hard liquor, to marijuana, and then to

other illicit drugs. Progression is not inevitable, but stages are rarely skipped. In general, drugs from each stage are continued into the next, leading to a pattern of multiple drug abuse. One exception is that children may begin with abuse of inhalants easily available volatile substances which ceases as they gain access to other drugs.

Potential impairment is more severe in children and adolescents than in adults, due to interference with developing cognitive, social, and physical abilities. Chronic use of marijuana, and possibly of sedatives or

narcotics, may result in an amotivational syndrome, which is particularly damaging to young persons. Critical developmental experiences that are missed may be difficult or impossible to replace. The risk is high for impairment of future functioning in every sphere.

The risk of death from intentional or accidental overdose, dangerous behavior while intoxicated (especially automobile accidents), homicide related to drug dealing, or acquired immunodeficiency syndrome (AIDS) is significant. Intravenous-drug use is currently the major vehicle for the spread of AIDS among in adolescents.

Virtually any psychi c disorder may be seen in association with substance use as cause, effect, or comorbidity. Most common are attention-deficit hyperactivity disorder (ADHD), conduct disorder, and specific developmental disorders. Depression may predate substance abuse or be a pharmacologic or situational effect. Prepubertal alcoholism suggests the presence of a bipolar mood disorder.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Virtually any change in emotional state, behavior, social activities, or academic performance can signal a problem with substance use.

All adolescent patients and older children should be questioned regarding substance use, in a manner that is nonjudgmental and encourages reporting. Questions should be asked regarding every category of substance, with details of amount, frequency, impairment, and social and emotional context.

Verification by parents, teachers, other professionals, or peers may be crucial, because underreporting is common.

Because many young substance users have "dual diagnoses" (primary psychiatric disorders in addition to substance use disorders), it is helpful to attempt to establish chronological

correlates of substance use with emotional and behavioral symptoms. A detailed family history of psychiatric disorders and substance use is essential.

Physical or neurological examination may disclose both direct and secondary effects. Laboratory screening for drug use can provide valuable information, although false positives and false negatives occur, and verification and integration with the rest of the assessment is essential.

TREATMENT

- Specific medical detoxification is rarely necessary in adolescents.
- Common features of treatment programs for substance abuse include:

-abstinence,

-group therapy with other substance abusers,

-participation in self-help "12-step" groups such as Alcoholics Anonymous (AA) and

Narcotics Anonymous (NA),

-the concept of "recovery" rather than cure.

- conventional individual psychotherapy is not useful in treating substance abuse per se. Many inpatient treatment units specialize in the care of substance-abusing

adolescents. Both the patient and family are actively involved in group treatment and education regarding drugs. In addition, various psychotherapeutic interventions may be required to deal with specific deficits or psychopathology.

Psychotropic medications may alleviate concomitant disorders, reduce withdrawal symptoms, or facilitate abstinence. Residential treatment from 6 months to 2 years in duration may be necessary for more severe, complex, or recalcitrant cases.

Long-term continuation of treatment is important, whether outpatient or conducted in a day treatment program or halfway house. Intensive participation in AA or NA is often required. Adolescents probably do best in groups with other adolescents rather than mixed with adults.

Family therapy is an integral part of treatment. Relapses are common and should be viewed as predictable complications rather than as catastrophes or reasons for terminating treatment. Specific attention to situations where drug use is likely, with training in coping strategies, may reduce the number and severity of relapses. Periodic urine testing can facilitate abstinence.

SCHIZOPHRENIA

CLINICAL DESCRIPTION

DSM-Icriteria for schizophrenia do not change with age, except that in children, failure to reach expected levels of adaptive functioning may be seen instead of deterioration. The presentation of schizophrenia in adolescence is similar to that in adulthood, whereas features of schizophrenia in children are rather different.

Schizophrenia in children is characterized by markedly uneven development and gradual onset. Language and social behavior are usually delayed and are qualitatively different from that seen in normal children at any developmental stage. Peers rapidly identify schizophrenic children as different. Visual hallucinations are more common in children than in adults, but when they are present, they are virtually always accompanied by auditory hallucinations.

EPIDEMIOLOGY

-The prevalence of childhood schizophrenia has been estimated at 5 per 10,000, with a male predominance.

- After puberty, the prevalence increases and approaches adult levels in late adolescence.

ETIOLOGY

- Etiologic factors for schizophrenia similar to those in adults have been implicated in children and adolescents.
- There is no evidence that parental behavior can produce schizophrenia.
- Environmental stressors may precipitate psychotic episodes.

COURSE AND PROGNOSIS

- Onset of schizophrenia is rare in childhood, and uncommon until late adolescence.

- Before puberty, onset is usually insidious, and course is chronic, whereas adolescents have episodes more similar to those seen in adults.
- In children with prepubertal onset of schizophrenia, common premorbid symptoms are short attention span, hyperactivity, conduct disturbances, and symptoms usually associated with pervasive developmental disorder, such as echolalia, rituals, stereotypies, and language and motor delays.
- The most frequent concomitant psychiatric disorders are conduct disorder, atypical depression, dysthymia, enuresis, and encopresis.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

- Acute hallucinations are not uncommon in normal children, resulting from acute phobic reactions, physical illness with fever or metabolic aberration, or medications. Children may have difficulty distinguishing among true hallucinations and dreams, illusions, and hallucinations occurring while fallingnasleep (hypnagogic) or awakening (hypnopompic).
- In young children, it may be difficult to distinguish delusions and hallucination from fantasy play at the extreme end of the normal range, or from play with normal imaginary companions. Apparent delusions or hallucinations may be reflections of shared religious or cultural phenomena.
- In young children who are nonverbal, it is not possible to make the diagnosis of schizophrenia. Some of these children receive a diagnosis of autistic disorder. As they develop language, evidence of delusions, hallucinations, and thought disorder emerges, enabling the correct diagnosis to be made.
- True autistic disorder does not “change into" classic schizophrenia, although very rarely, both may be present.
- Children who are mentally retarded have multiple delays in development, but they are consistent, and they do not have the peculiarities of thought and behavior characteristic of schizophrenia.
- Apparent thought disorder may actually be due to deafness or aphasia
- Schizophrenic children may be overactive and distractible, but the diagnosis of ADHD is preempted.
- Other differential diagnostic possibilities include organic syndromes, substance abuse, obsessive-compulsive disorder, dissociative states, psychotic depression, and mania.

TREATMENT

- The cornerstone of treatment of childhood schizophrenia is an intensive school-based program that incorporates multiple methods of intervention.
- Hospitalization or long-term residential treatment may be needed.
- Individual psychotherapy may be useful as a part of a comprehensive treatment plan. The therapist must be prepared to provide structure, to limit regression and fantasy, and to focus on reality testing and development of stronger defense mechanisms and healthier coping skills.
- Family psycho-educational treatment may prove beneficial.

- Major tranquilizers should be used only in conjunction with a comprehensive treatment program. Target symptoms that may respond include over activity, aggression, agitation, stereotyped movements, delusions, and hallucinations.
- Neuroleptics are less likely to be effective in prepubertal schizophrenic children than in schizophrenic adolescents or adults, and they are more likely to have troublesome sedation, especially from low-potency neuroteptics. To avoid sedation, a higher-potency drug (e.g., haloperidol, fluphenazine, trifluoperazine, or perphenazine) may be best.

MOOD DISORDERS

CLINICAL DESCRIPTION

Although DSM-Icriteria for mood disorders are essentially the same for children as for adults, the behaviors may be manifested in different ways at different developmental levels. Key indicators of depression in young people may be declining school performance, withdrawal from social activities, somatic complaints (especially headaches and abdominal pain), sleep difficulties, or conduct problems.

EPIDEMIOLOGY

- The prevalence of major depression has been estimated at 2% in prepubertal children and 4.7% in adolescents.
- Dysthymic disorder without coexisting major depression is found in 3.3% of adolescents.
- Before puberty, depression is more common in boys than in girls, with a change to the adult gender ratio in adolescence.
- Mania is rare before middle adolescence, but becomes more common in late adolescence.

ETIOLOGY

- Etiologic factors for mood disorders are presumed similar to those in adults.
- A depressed parent may be a powerful contributing factor to depression in genetically vulnerable young people, via modeling, emotional unavailability, and decreased capacity for care taking activities.
- Abuse and neglect may be significant precipitants, especially in very young children.

AND PROGNOSIS

- Mood disorders are serious and potentially fatal problems.
- Dysthymia is more chronic, with an average episode length of 3 years, often progressing to a major depressive episode.
- Adjustment disorder with depressed mood has an average duration of 6 months.
- Among those in mid-adolescence with major depression, subsequent bipolarity is predicted by precipitous onset of symptoms, psychomotor retardation, psychotic features psychopharmacologically precipitated hypomania, and family history of bipolar disorder.

- Approximately one-third of prepubertal children with depressive mood disorder will develop a DSM-IV conduct disorder by age 19.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

Children can be asked direct questions related to depression. Young children have more difficulty recognizing and verbalizing their feelings and may use idiosyncratic words, such as “bored.” to describe dysphoria or anhedonia. “Cranky" may mean more to a child than iritable. Both parent and child repels are essential.

Mania and hypomania are difficult to diagnose, especially because children” and adolescents are prone to rapid, brief cycles of irritability, agitation, and impulsivity. Severe temper outbursts lasting more then 30 minutes, heedless risk taking, highly energized affect, and inappropriate sexual preoccupation and behavior are useful markers. Family history and
longitudinal course are important.

It is crucial to assess the degree of suicidality and other dangerous behavior, children can be questioned regarding ideation, plans, and attempts, just as adults.

Anxiety disorders, ADHD, or conduct 'disorders frequently coexist with a mood disorder. Substance abuse may make diagnosis of a mood disorder impossible without drug- free observation.

A search should be made for child abuse, parental substance abuse, or physical or emotional neglect.

Separation anxiety disorder may resemble major depression or dysthymic disorder, or may coexist with it. Children younger than age 4 years may develop a clinical picture similar to major depression when separated from their parents. Children suffering from reactive attachment disorder secondary to parental abuse or neglect who present with lethargy, apathy, and withdrawal may appear depressed.

Both mania and agitated depression may be confused with ADHD, but mood disorders are episodic, whereas ADHD is a chronic condition with onset in early childhood. Adolescent mania is frequently misdiagnosed as schizophrenia.

Secondary mania may result from prescribed medication (e.g., steroids, carbamazepine, tricyclic antidepressants) or illegal drugs (e.g., cocaine, amphetamines), metabolic abnormalities (especially thyroid), neoplasm, or epilepsy.

TREATMENT

If risk-taking behavior or suicidal ideation is present, close parental and psychiatric supervision are needed. Psychiatric hospitalization may be required for youngsters who are psychotic or seriously suicidal or who do not respond to outpatient treatment.

Even after successful treatment with medication, impaired interpersonal relations with peers and family members may require individual, group, or family therapy to address developmental deficits or sequelae of the depression. Cognitive therapy techniques developed for the treatment of depression in adults are being adapted for use with children and adolescents.

Remedial education or tutoring may be needed when illness has interfered with learning in school.

The evidence for efficacy of pharmacotherapy is less than that for adults.

For nonpsychotic depression, psychotherapy is the first step, with medication added if there is no improvement in 4—6 weeks. Although there are multiple reports of successful treatment of depression in prepubertal children with amitriptyline or imipramine, a high rate of placebo response has made it difficult to demonstrate efficacy in controlled trials.

In adolescents with carefully diagnosed major depression, 6 weeks of treatment with imipramine at a dosage of 4—5 mg/kg per day results in complete or substantial resolution of depressed mood or anhedonia in 44%, a rate significantly lower than that for adults. The coexistence of separation anxiety is associated with poorer prognosis.

Monoamineoxidase inhibitors may be useful in the treatment of unipolar and bipolar depressed children and adolescents who do not respond to tricyclics. Suicidal and impulsive outpatients should be excluded because of the risk of dietary and drug interactions. Even for responsible youngsters, close supervision and repeated careful dietary instruction are necessary.

Lithium may be considered in the treatment of children and adolescents with bipolar affective disorder (mixed or manic) or as a supplement to antidepressants for treatment- resistant patients.

As in adults, anticonvulsants (carbamazepine, valproic acid) may be useful in treating adolescents with rapidly cycling bipolar disorder or mania resistant to lithium and neuroleptics.

ANXIETY DISORDERS PHOBIAS

Clinical description. The DSM-IV criteria for simple phobia and social phobia are the same in young people and adults, although cognitive immaturity may limit recognition that fear is excessive or unreasonable.

Epidemiology. About 10% of children and 2—3% of adolescents have significant fears, but many children with phobias are never seen in a clinical setting.

Etiology. The wide variety of proposed etiologies of phobias may operate to varying degrees in different patients: psychodynamic - Freud classical conditioning theory modeling or “contagion” from fearful adults genetic

psychophysiologic COURSE AND PROGNOSIS.

isolated phobias in childhood commonly remit spontaneously.

social phobia is more common in adolescents than in younger children. In these adolescents, answering questions in a group and speaking in front of the class are commonly feared, leading to impaired grades in school, despite adequate learning. If severe, the phobia may lead to an avoidance of school altogether.

phobic disorders are commonly found in association with other anxiety disorders.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

Parents are often unaware of phobic symptoms in their children, so the clinical interview is especially important. The history includes:

- a description of the feared stimulus,
- circumstances surrounding the development of the phobia,
- behavior in response to the phobic objector situation,
- anticipatory or avoidant behaviors,
- secondary gain.

Behavior observations may be useful.

Differential diagnosis includes:

- panic disorder,
- agoraphobia,
- separation anxiety disorder,
- schizophrenia with delusions,
- posttraumatic stress disorder,
- obsessive-compulsive disorder,
- an eating disorders (fear of eating or of gaining weight).

Treatment.

Individual psychotherapy, which may include the use of play for young children, may be indicated for the child with multiple anxiety-related symptoms.

Behavior treatments are the best studied and the most efficient, and are generally the treatment of choice for children with one or two phobias.

Systematic desensitization techniques can be used as in adults, with techniques adapted for developmental level.

In vivo desensitization appears to be more effective than imaginal techniques, especially if a young child has difficulty learning the relaxation techniques and imagining the stimuli.

Emotive imagery using stories the therapist creates, pairing the child with a powerful hero who helps the child confront the hierarchy of feared stimuli is appropriate for 4- to 8-year-olds.

A second group of behavior techniques are based on modeling or observation learning.

This type of treatment has the advantage of simultaneously reducing anxiety and teaching skills.

In operant conditioning approaches, phobic avoidance is eliminated by changing the positive and negative contingencies that maintain the phobia.

Cognitive therapy techniques aim to change feelings and behavior by specifically addressing maladaptive, self-defeating thoughts or self-statements.

POSTTRAUMATIC STRESS DISORDER

Clinical description. Posttraumatic stress disorder (PTSD) involves specific, long- lasting emotional and behavioral symptoms following a shocking, unexpected event that is outside the range of usual human experience, that makes the individual feel intensely fearful and helpless and that is so extreme that it could be expected to overwhelm anyone's coping strategies.

Symptoms include reexperiencing the traumatic event, avoidance of reminders of the event, generalized emotional numbing and increased arousal.

The precipitant may be experienced directly (e.g., rape, physical injury, kidnapping), by observation (witness to trauma to another person), or vicariously (after learning about a traumatic event affecting a close friend or relative). Although the DSM-Icriteria for PTSD are essentially the same at all ages, symptoms in children differ in some ways.

Immediate effects include fear of separation from parent(s), of death and of further fear. Children withdraw from new experiences.

Although many details of the experience are accurately remembered, sequencing or duration of events is often distorted.

In children, reexperiencing the event is likely to occur in the form of nightmares, daydreams, or repetitive and potentially dangerous reenactment in symbolic play or in actual behavior (rather than in flash backs). Even children younger than age 3 years demonstrate through play or dreams, memories of traumatic events that they cannot describe verbally. The denial, repression, and psychic numbing experienced by many adults are not typically seen after children suffer a single traumatic event.

Children manifest:

- sleep disturbance
- somatic symptoms, particularly headaches and stomachaches.
- regression (behavior characteristic of a previous developmental stage)
- guilt is often experienced. (See attached DSM IV criteria.)

Diagnostic criteria for Posttraumatic Stress Disorder

A. The person has been exposed to a traumatic event in which both of the following wereF present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved! actual or threatened death or serious injury, or a threat to the physical integrity of self or other I

(2) the person's response involved intense fear, helplessness, or horror.

Note: In children, this may be expressed instead by disorganized or agitated behavior

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.

Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) Recurrent distressing dreams of the event. Note: In children, there may be frightening dreams! without recognizable content.

(3) Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific reenactment may occur.

(4) Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

(5) Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feeling, or conversations associated with the trauma

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (e.g., unable to have loving feelings)

(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or, a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep

(2) irritability or outbursts of anger

(3) difficulty concentrating

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, a other important areas of functioning.

Specify if:

Acute: if duration of symptoms is less than 3 months

Chronic: if duration of symptoms is 3 months or more

Specify if:

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor

Epidemiology. Among young people exposed to a sudden, overwhelming, dangerous situation, the incidence and severity PTSD vary with their previous history, the severity of the event, and the degree of exposure.

Children who have experienced multiple stressors prior loss, anxiety, oi depression are vulnerable to more severe and. prolonged symptoms, but a sufficient!] severe stressor can produce the disorder in a person without any predisposition.

Proposed mechanisms for the development of PTSD symptoms include destruction of "basic trust,'' cognitive information overload, and a classical conditioning model to explain extension of anxiety to otherwise harmless stimuli present at the time of the traumatic event.

COURSE AND PROGNOSIS. Changes in living circumstance caused by disasters, such as loss of home, isolation from usual social supports, and even death of parents or other family members, can exacerbate PTSD. Symptoms may be partially ameliorated by a stable, cohesive, supportive family.

Anxiety (especially separation anxiety disorder) or depression may be prominent. Impulsivity, difficulty concentrating, and decreased motivation may interfere with school performance.

Many traumatized children develop a chronic sense of pessimism and hopelessness about the future. A screen or cover memory may be created, which loses the emotional intensity of the original memories. As late as 4—5 years after the event, children remain fearful, many with continuing disturbances of sleep and play, and deeply ashamed of their helplessness in the face oh danger.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. The clinician may or may not be aware of the traumatic event. PTSD should be suspected in any child or adolescent who has had a significant change in behavior. Both the parents and child should be questioned, because the child may have difficulty describing the symptoms, and the parents may not be fully aware of them.

A clinical interview technique developed for child and adolescent victims begins with the use of play and fantasy, using a projective drawing and storytelling task. The interviewer then structures a detailed recounting of the traumatic event, including affective responses and fantasies of revenge. The concluding stage includes a review of the child's current life concerns, a reassessment of the traumatic experience, anticipatory guidance regarding reactions that the child may experience, and efforts to support the child's self-esteem.

Developmental history may indicate sources of increased vulnerability. Teacher reports document change in behavior and achievement at school.

An anxiety or mood disorder is also diagnosed, if criteria are met. PTSD is distinguished from adjustment disorder by the severity of the stressor and the distinctive symptoms, such as repetitive reexperiencing of the traumatic event. Some of the symptoms of PTSD may be mistaken for psychosis or organic brain damage.

Treatment. Individual insight-oriented play and verbal psychotherapy is the modality that has been most commonly used in treatment of PTSD. Discussions with the child about tactics to cope with the danger experienced (with specific instructions and demonstrations) may help in reducing the child's guilt regarding the event, as the child recognizes that he or she did not have the necessary skills to prevent the traumatic event. This technique can also alleviate the child's current feelings of helplessness.

Time-limited focal psychotherapy is effective in some patients. Systematic desensitization of specific trauma-related fears may be useful in conjunction with other interventions.

The use of psychotropic medication to treat PTSD in children has not been extensively evaluated. Beta-blockers may offer some help. Other agents would be appropriate for concurrent mood or anxiety disorder.

Group therapy with victims who have been exposed to the same even may be helpful in decreasing distortions and reducing the spread of posttraumatic fears and symptoms.

Supportive therapy for parents and siblings can provide information about the child's symptoms and their cause, deal with vicarious trauma experienced by family members, and reduce contagion.

OBSESSIVE - COMPULSIVE DISORDER

Clinical description. The DSM- criteria for obsessive-compulsive disorder (OCD) are the same in children and adults. The most frequent symptoms in young people are cleaning, counting, and checking rituals and ego-dystonic thoughts of violence or sex.

Epidemiology. OCD is more common than clinical populations would suggest, with a prevalence in high school students of at least 0.33%. Boys suffer from an earlier age at onset, and a rate twice that found in girls.

Etiology.

A current biological hypothesis involves failure of the frontal lobe to inhibit latent behavior patterns stored in the basal ganglia. OCD may follow neurological disorders affecting the basal ganglia (e.g., Sydenham's chorea).

- A family history of OCD is found in one-fourth of patients.

A genetic mechanism is likely, because child-rearing practices (such as overly strict toilet training or parental perfectionism) are not causal, and there is little evidence for modeling of symptoms.

COURSE AND PROGNOSIS.

Over one-third of adult cases of OCD begin by age 15.

In child and adolescent cases, a premorbid pattern of obsessive traits is rarely found. Onset of OCD symptoms is typically acute or subacute (over several months) and may begin at ages 3-5 years.

Rituals are typically hidden from adults at first, until progressive severity makes this impossible. Partial control is common, with suppression of rituals outside of the home, but inability to do so with the family.

The rituals may increase in number, severity, and interference with daily activities, become chronic, and extend into adulthood.

Associated disorders include depression, anorexia nervosa, Tourette’s disorder, conduct disorder, anxiety disorders, and specific developmental disorders.

EVALUATION AND DIFFERENTIAL DIAGNOSIS. Children and adolescents are often secretive about obsessions and compulsions, so questions must be specific, and collateral sources used. Alternative diagnoses include phobic disorder. Tourette's disorder, and schizophrenia. There is a superficial resemblance to the rituals seen in pervasive developmental disorder.

Treatment.

Clomipramine hydrochloride is effective regardless of the presence or absence of symptoms of depression, whereas desipramine is not.

Fluoxetine, another potent blocker of serotonin reuptake, may also be useful.

Behavior treatments such as response prevention for overt rituals, thought stopping for rumination and covert compulsions, and systematic desensitization with in vivo exposure have some promise, but are largely untested and require a highly motivated patient. Symptom substitution does not occur.

- Family involvement is crucial. As in adults, psychodynamic psychotherapy is rarely useful in removing obsessions or compulsions.

PANIC DISORDER

Clinical description. Panic disorder and agoraphobia occur in children and adolescents, with the same DSM-I criteria and physical symptom profile as in adults. Cognitive immaturity may preclude some characteristic cognitions during an attack (fear or dying, going crazy, doing something uncontrolled).

Epidemiology. Although 11.6% of ninth graders have had at least one four-symptom panic attack , the full syndrome of panic disorder is less common than other anxiety disorders in childhood and adolescence.

Etiology. In a controlled study, panic disorder was found only iii children and adolescents with a parent with panic disorder and/or depression, suggesting contributions from genetics and/or modeling. Shy children with constitutionally exaggerated sympathetic nervous system reactivity may be at risk for panic disorder.

COURSE AND PROGNOSIS. Adults with panic disorder have a peak age at onset of 15- 19 years, but some cases begin before puberty. The course may be chronic or recurrent. Panic attacks often begin at the onset of or during an episode of major depression or separation anxiety disorder

EVALUATION AND DIFFERENTIAL DIAGNOSIS. Some children can report current panic attacks, but parent report is required to verify duration and past history. In practice, panic disorder in children may be under diagnosed, with the symptoms attributed to separation anxiety disorder, hyperventilation syndrome, or situational or "normal" anxiety.

Treatment. Case reports suggest clonazepam, alprazolam, and imipramine may be effective drugs for treatment of panic disorder. Supportive and educational individual and family psychotherapy are useful adjuncts.

SLEEP DISORDERS

EVALUATION OF SLEEP-RELATED COMPLAINTS

Assessment is similar in all children with sleep disorders.

Pediatric history and physical examination are required, with particular attention to: obesity, enlarged tonsils, middle ear problems, a seizure disorder, allergies, asthma, and medication use.

Developmental and psychiatric histories are necessary to assess possible psychiatric etiology or comorbidity.

Sleep history includes details of the physical environment while sleeping, sleep habits, stressors, parental reactions to sleep-related problems, caffeine consumption, substance abuse in older children or adolescents, and the effects of prior behavioral and pharmacologic interventions.

If sleep apnea or nocturnal epilepsy is being considered, sleep laboratory evaluation (polysomnography) may be indicated, including sleep EEG, eye movements, electromyogram, air flow, respiratory effort, ECG, and video monitoring. Sleep- deprived EEG is useful in the evaluation of possible seizures.

- A drug screen may be needed in adolescents.

Dyssomnias are characterized by disturbance in the amount, quality, or timing of sleep.

PRIMARY INSOMNIA

Clinical description. Patients with insomnia disorder have difficulty initiating or maintaining sleep, compared with norms for age.

Epidemiology.

Most infants sleep through the night (or at least do not fuss when they waken) by age 6—9 months. Up to one-half of all infants, however, have irregular sleep patterns and occasional or persistent night wakening throughout the 1st year.

A recent study of normal children found that 21% of 18- to 23-month-olds awakened during the night. Thirty-one percent of the 24 to 29 month old, took more than 30 minutes to fall asleep on more than three nights in a week.

Children ages 30—36 months are most likely to have difficulty settling for the night (16%) ail to express fears of the dark (24%).

Etiology. Chronic childhood insomnia is much more frequent children with psychiatric disorders. It is often related to behavior habit problems in settling for the night (especially in the child with ADHD, oppositional defiant disorder, or separation anxiety disorder) or may be a symptom of mood disorder, pervasive developmental disorder, or schizophrenia.

Insomnia may be secondary to prescribed or over-the counter medication such as phenobarbital, theophylline, decongestants, or stimulants, to caffeine, or to substance abuse.

Treatment. After any psychiatric or medical cause is address in young children the first steps are the removal of environmental factors interfering with sleep and enhancing structure that encourages sleep (e.g., a bedtime routine, the use of a transition, object, or a night-light for the child afraid of the dark). Behavior treatment removes the secondary gain of parental attention at night and provides positive reinforcement for the child staying quietly in his or her own room. Older children and adolescents may benefit from hypnosis or relaxation techniques.

In adolescents, behavior therapy can disrupt the conditioned association between bedtime and anxiety regarding inability to sleep. Sleep hygiene is improved by use of the bed only for sleeping, establishing a regular wake-up time, and avoiding naps.

Chloralhydrate or an antihistamine (such as diphenhydramine hydrochloride) may be indicated for short-term use in a crisis. Hypnotic medications are not recommended for chronic use. Many children respond with paradoxical agitation to sedatives. If there is extreme fear or anxiety, a short-acting benzodiazepine (such as triazolam) may be used briefly.

PRIMARY HIPERSOMNIA - See attached DSM IV criteria.

Diagnostic criteria for Primary Hypersomnia

A. The predominant complaint is excessive sleepiness for at least 1 month (or less if recurrent) as evidenced by either prolonged sleep episodes or daytime sleep episodes that occur almost daily.

B. The excessive sleepiness causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The excessive sleepiness is not better accounted for by insomnia and does not occur exclusively during the course of another Sleep Disorder (e. g., Narcolepsy, Breathing-Related Sleep Disorder, Circadian Rhytm Sleep Disorder, or a Parasomnia) and cannot be accounted for by an inadequate amount of sleep.

D. The disturbance does not occur exclusively during the course of another mental disorder.

E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Specify if:

Recurrent: if there are periods of excesive sleepiness that last at least 3 days occurring several times a year for at least 2 years

NARCOLEPSY

Clinical description. Narcolepsy, one of the hypersomnias, is characterized by sudden, uncontrollable attacks of rapid eye movement (REM) sleep during wakefulness. Sleep is usually resisted, but the patient eventually succumbs. Features in children are similar to those seen in adults, including cataplexy, hypnagogic or hypnopompic auditory or visual hallucinations, and sleep paralysis. These frightening symptoms may lead to reluctance or refusal to go to bed.

Etiology. Narcolepsy is a genetic disorder of REM sleep-regulating mechanisms.

COURSE AND PROGNOSIS. Onset of narcolepsy is typically around the time of puberty, although in one-fifth of narcoleptic adults, the onset is before puberty. Peak incidence is during the late teens and early 20s. Sleep attacks and cataplexy may significantly interfere with schoolwork and peer relations.

Differential diagnosis.

The most likely alternative diagnosis to narcolepsy is the normal increase in daytime sleep and cornplants of sleepiness in adolescence.

Excessive daytime sleep may be an avoidance mechanism, even in the classroom, or secondary to insomnia, environmental interference with sleep, or sleep apnea Sleep onset or waking hallucinations may be misidentified as symptoms of psychosis, and cataplexy or sleep attacks may be confused with a seizure disorder.

Treatment. Stimulant drugs (methylphenidate, dextroamphetamine, pemoline) are used for sleep attacks. Tricyclic antidepressants may reduce cataplexy and sleep paralysis. Environmental support and scheduled naps may be helpful.

SLEEP APNEA

Clinical description. Sleep apnea is characterized by repeated cycles of loud snoring followed by an apneic period, terminated by a brief arousal from sleep, leading to excessive daytime sleepiness.

Epidemiology. Sleep apnea is seen primarily in males

Etiology. Medical causes of sleep apnea include:

gross obesity (Pickwickian syndrome),

enlarged tonsils and/or adenoids,

nocturnal asthma,

lax upper airway structures,

maxillofacial abnormalities,

hypothyroidism,

dysfunction of central control of breathing.

COURSE AND PROGNOSIS.

Somatic complications of sleep apnea include:

- morning headache and systemic
- pulmonary hypertension.

Daytime sleepiness substantially interferes with development and performance in daily activities. The child may be misdiagnosed with borderline mental retardation.

Treatment. The treatment of sleep apnea is medical, with correction of any obstruction when possible. Some patients require tracheostomy. If the cause is central, a home apnea monitor may be required. Certain tricyclic antidepressants can be useful.

PARASOMNIAS

This event is the primary complaint, not its effect on sleep or daytime alertness.

NIGHTMARE DISORDER (DREAM ANXIETY DISORDER)

Clinical description. Occasional nightmares are considered normal in children and adolescents. They occur during REM sleep, more commonly in the second half of the night. If the youngster awakens, he or she rapidly becomes oriented and alert and can recount the dream.

DSM-I criteria for dream anxiety disorder require “repeated" awakening due to nightmares with “significant distress” and “detailed recall”.The boundary between normally occurring frightening dreams and dream anxiety disorder is not clear.

Epidemiology. Among normal 1-to 3-year-olds, the 1-week prevalence of nightmares is 10%. Nightmares generally decrease in frequency with age. There are no data on the prevalence and dream anxiety disorder.

Etiology. There is no consistently associated psychopathology in children.
Nightmares tend to increase with stress, fatigue, and change in sleep environment. Frequency of nightmares waxes and wanes as the child develops. Tricyclic antidepressants can cause severe nightmares.

Differential diagnosis. The main alternative diagnoses to nightmare disorder are sleep terror disorder and drug-induced nightmares (e.g., as a result of antidepressants or caffeine).

Treatment. No treatment is ordinarily indicated, beyond education and support. The disorder generally disappears with time.

Clinical description. Episodes typically occur during the first third of the night, in non-REM sleep stages 3 and 4, lasting 1-10 minutes. The child appears terrified, screams, and appears to be staring, with dilated pupils, sweating, rapid pulse, and hyperventilation. The child is agitated and confused and cannot be comforted. Subsequently, when alert, the child typically has no memory of the episode, but rarely has brief recall of a feeling of terror or of dream fragments. The child returns rapidly to sleep when the episode is over and has complete amnesia in the morning. The parents are far more distressed than the child. See attached DSM IV criteria.

Diagnostic criteria for Sleep Terror Disorder

A. Recurrent episodes of abrupt awakening from sleep, usually occurring during the first third of the major sleep episode and beginning with a panicky scream.

B. Intense fear and signs of autonomic arousal, such as tachycardia, rapid breathing, and sweating, during each episode.

C. Relative unresponsiveness to efforts of others to comfort the person durring the episode.

D. No detailed dream is recalled and there is amnesia for the episode.

E. The episodes cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

F. The disturbance is not due to the direct physiological effects of a substance (e. g., a drug of abuse, a medication) or a general medical condition.

Epidemiology. Sleep terror disorder is often seen in children ages 3—6 years, but isolated episodes are common throughout childhood. The estimated prevalence in children of the full disorder is 1—4%. It is more common in boys than in girls. A recent study of sleep habits in middle-class 1- to 3-year-olds found the 1-week prevalence of at least one episode to be 7%.

Etiology. Stress, exhaustion, or a febrile illness may increase the frequency of sleep terror disorder. A family history of similar parasomnias is common.

COURSE AND PROGNOSIS. The age at onset of sleep terror disorder is typically between 4 and 12 years, with spontaneous resolution by, adolescence. The number and frequency of episodes are highly variable. In children, there is no typically associated psychopathology.

Differential diagnosis. Simple nightmares, or dream anxiety disorder, usually occur in the latter half of the night, and affected children show less physiologic arousal and confusion, and clear recollection of the dream. Other alternatives are hypnagogic or hypnoponipic hallucinations, or epileptic seizures during sleep with postictal confusion.

Treatment. Sleep terror disorder usually responds to support and education while waiting for the child to outgrow the problem. Medication is used only if the episodes are frequent, severely disrupting the family, or interfering with daytime functioning. A low dose of imipramine or diazepam may be given at bedtime.

SLEEPWALKING DISORDER (SOMNAMBULISM)

Clinical description.

Somnambulism is characterized by repeated episodes of arising from bed and engaging in motor activities while still asleep.

Episodes, which last a few minutes to half an hour, typically occur 1—3 hours after the child falls asleep, during stage 3 and 4 delta (non-REM) sleep.

The child or adolescent arises quietly and engages in pereseverative, stereotyped movements (such as picking at blankets), which may progress to walking and other complex behaviors.

He or she is difficult to awaken, and coordination is poor. Although the child may be able to see, the risk of injury is high.

Speech, when present, is usually incomprehensible.

The youngster may awaken and be confused, may return to bed, or may lie down somewhere else and continue sleeping.

Morning amnesia is typical.

Epidemiology. Sleepwalking is common in children ages 6—12 years. It is often seen in children who had night terrors when younger.

Etiology. Likelihood of sleepwalking is increased when the child is overtired or under stress.

COURSE AND PROGNOSIS. The onset of sleepwalking is usually before age 10, with spontaneous remission by age 15.

Differential diagnosis. Diagnostic alternatives to sleepwalking disorder include nocturnal seizures and children who wake and wander in the night.

Treatment. Parents should be guided to remove hazards in the environment and may need to lock the child's door. If sleepwalking is frequent, or dangerous, a low dose of imipramine at bedtime may be used.

ADJUSTMENT DISORDER

CLINICAL DESCRIPTION

Common stressors in childhood and adolescence include parental divorce, changing schools and acute physical illness, abuse or neglect, parental unemployment.

See attached DSM IV criteria.

Diagnostic criteria for Adjustment Disorders

A. The development of emotional or behavioral symptoms in response to an identifiable stressor(s) occurring within 3 months of the onset of the stressor(s).

B. These symptoms or behaviors are clinically significant as evidenced by either of the following:

(1) marked distress that is in excess of what would be expected from exposure to the stressor

(2) significant impairment in social or occupational (academic) functioning

C. The stress-related disturbance does not meet the criteria for another specific Axis I disorder and is not merely an exacerbation of a preexisting Axis I or Axis II disorder.

D. The symptoms do not represent Bereavement.

E. Once the stressor (or its consequences) has terminated, the symptoms do not persist for more than an additional 6 months.

Specify if:

Acute: if the disturbance lasts less than 6 months Cronic:

Adjustment Disorders are coded based on the subtype, which is selected according to the predominant symptoms. The Specific stressor(s) can be specified on Axis IV.

ETIOLOGY

The precipitant of adjustment disorder is presumed to be the identified psychosocial stressor, although this may not be the case.

It is assumed that the symptoms of adjustment disorder will remit when the stressor is removed or when a new level of adaptation is reached. By definition, if the disorder lasts for more than 6 months, it must be given a different classification.

The prognosis depends on the severity and duration of the stressor and its meaning to the child or adolescent, the vulnerability of the individual, and the response of the environment to both the stressor and the young person's reaction. In general, the prognosis is assumed to be benign, although it may be quite severe, or even fatal, as in a successful suicide attempt in reaction to a perceived failure or rejection.

An adjustment disorder may be diagnosed in a person suffering from another mental disorder, if an identifiable stressor is followed by the development of symptoms that are not characteristic of the original disorder.

EVALUATION AND DIFFERENTIAL DIAGNOSIS

A thorough search should be made for stressors, including reports from child, parent, and teacher, rather than assuming that the first-reported or obvious stressor is the crucial one.

Efforts should be made to identify any other diagnosis that is masquerading as an adjustment disorder. Unfortunately, past overuse of the diagnosis of adjustment disorder (in an effort to avoid "labeling" children and adolescents) has often obscured another psychiatric diagnosis.

Adjustment disorder is a residual category and should be used only if the patient's symptoms do not meet criteria for another DSM-I disorder.

If functional impairment is not evident, or if the degree of emotional or behavioral reaction is considered "normal and expectable," a V code, such as other interpersonal problem, phase of life problem, or other life circumstance problem should be used.

If the primary reaction to a stressor is exacerbation of a documented physical disorder (e.g., asthma or diabetes), the appropriate diagnosis is psychological factors affecting physical condition. If the stressor is sudden and catastrophic or potentially so, a diagnosis of PTSD should be made, if other criteria are met.

TREATMENT

Crisis intervention and time-limited psychotherapy techniques may be useful for treatment of adjustment disorder. Cognitive therapy techniques to improve coping skills and problem-solving abilities and to reduce dysfunctional thoughts and beliefs in reaction to the stressor may be beneficial.

Environmental intervention may be indicated, to remove or ameliorate the stressor.

Reference:

1. Russell AT, Bott L, Sammons C: The phenomenology of schizophrenia occuring in childhood. J Am Acad Child Adolesc Psycciatry 28:399-407,1989

2. Stroberg M, Carson G: Bipolar illness in adolescents with major depression. Arch Gen Psychiatry 39:549-555,1982
3. Hajward C, Killen JD, Taylor CB: Panic attacks in young adolescent. Am J Psychiatry 146:1061-1062,1989

6. DEVELOPMENTAL DISORDERS

The developmental disorders, which include mental retardation, pervasive developmental disorders (including autistic disorder), and a series of specific developmental disorders (teaming disorders), are coded on DSM-IV Axis II, to distinguish them from and to prevent their being obscured by concurrent Axis I diagnoses.

MENTAL RETARDATION CLINICAL DESCRIPTION

The diagnosis of mental retardation requires both low intelligence and deficits in adaptive functioning. Onset must be during childhood.

Developmental slowness is primarily evident in cognition and intellectual functioning, but may appear across all areas of motor, sensory, neurologic, social, and emotional functioning. There may be a wide "scatter" of strengths and weaknesses among various intellectual and adaptive skills and areas of psychological development. Characteristics vary according to the degree of retardetion:

Mild: - IQ = 50-55 to 70

- of 85% of mentally retarded population
- educable
- community residence
- manages a job Moderat: - IQ = 35-40 to 50-55
  - of 10% of mentally retarded population
  - trainable
  - sheltered residence
  - usually able to manage pocket money Severe: - IQ = 20-25 to 30-35
  - of 4% of mentally retarded population
  - untrainable
  - mostly living in highly structured and closely supervised settings
  - can take notes to stores when shopping Profound: - IQ below 20-25
- of 1% of mentally retarded population
- untrainable
- mostly living in highly structured and closely supervised settings
- dependent on others See attached DSM IV criteria.

Diagnostic criteria for Mental Retardation

A. Significantly subaverage intellectual functioning: an IQ of approximately 70 or below on an individually administred IQ test (for infants, a clinical judgment of significantly subaverage intellectual functioning).

B. Concurrent deficits or impairments in present adaptive functioning (i. e., the person's effectiveness in meeting the standards expected for his or her age by his or her cultural group) in at least two of the following areas: communication, self-care, home living, social/interpersonal skills, use of community resources, self-direction, functional academic skills, work, leisure, health, and safety.

C. The onset is before age 18 years.

There is a male predominance at all levels of mental retardation (overall, about 1.5:1). In the lowest socioeconomic class, there is a 10—30% prevalence of mental retardation in the school-age population.

ETIOLOGY

The most common forms of mild retardation are idiopathic, associated with sociocultural or psychosocial disadvantage, and familial, typically seen in the offspring of retarded parents.

Intellectual and adaptive deficits are presumed to be determined by the interaction of a polygenic mechanism and intervening social factors.

There is high comorbid transmission of conduct, attention-deficit and language disorders.

Moderate, severe, and profound retardation are less likely to be idiopathic. There are more than 200 recognized biological syndromes involving mental retardation, entailing disruptions in virtually any sector of brain biochemistry or physiology.

Known biomedical etiologies are identified in 25% of all cases of mental retardation, and in 60—90% of severe or profound mental retardation:

Biological causes of mental retardation:

Genetic:

-single gene defects-dominant

- inborn error of metabolism-recessive
- chromosomal abnormalities:

- Trisomy 21 (Down's syndrome) is most common
- Fragile X chromosome syndrome
- polygenic inheritage Prenatal:
  - maternal illness
  - maternal infection passed to fetus
  - toxins
- brain malformation Perinatal:
  - extreme prematurity
  - blood group incompatibility

  - brain trauma
- cerebrovascular accident Aquired in infancy or childhood:
  - brain infection
- head trauma
- neurological disease
- brain tumor
- hypothyroidism
- irradiation -asphyxia
- several malnutrition

Fragile X chromosome syndrome is the second most common inherited cause of moderate and severe mental retardation, with a prevalence of over I per 1000 in the general
population. The classic triad of physical stigmata, seen most clearly at puberty, is long face, prominent ears, and macro-orchidism. Other physical features may include hyperextensible finger joints, flat feet, strabismus, pectus excavatum, mitral valve prolapse, single palmar crease, long narrow palpebral fissures, and a high-arched palate.

Cognitive impairment in fragile X chromosome syndrom ranges from normal IQ with learning disabilities to profound retardation. Behavior abnormalities are present in 80% of males, including hyperactivity, short attention span, and autistic-like features, sometimes meeting criteria for autistic disorder or pervasive developmental disorder not otherwise specified.

In female heterozygotes, affective disturbances and schizotypal featurs are more typical. Language and speech deficits include poor abstraction, immature syntax, unusual speech rhythm, expressive and receptive language deficits, and articulation problems. In females (who are partially protected by having two X chromosomes), a “carrier” state is associated with mild mental retardation or specific developmental disorders

Oral folate reduces the frequency of fragile sites in vivo and improves behavior and attention in prepubertal children. Stimulant medication improves attention and over activity.

COURSE AND PROGNOSIS

The course of mental retardation is influenced by environment variables, including parental intelligence, psychological resilience, and material resources, as well as community supports and barriers.

More severe levels of mental retardation are diagnosed before the child is school-age. In moderate to mild retardation, diagnosis is uncommon before age 5 years, rises sharply in the early school years, and peaks in the later school years. For mental retardation at all levels of severity the developmental course is slow but not “deviant." Although the normal sequence of cognitive developmental stages is observed, the speed of developmental change is slow, and there is a ceiling on ultimate achievement.

Delays in speech and language development may limit ability to express negative affect, leading to impulsive anger and low frustration tolerance. Insufficient financial resources, inappropriate or inadequate

educational programming, and prejudices of communities and health care personnel can result in a wide variety of developmental, social, and medical complications.

It cannot be overemphasized that all psychiatric diagnoses may co-occur with mental retardation. About one-half of children with mental retardation have "dual diagnoses," i.e., an additional psychiatric diagnosis. Approximately one-third to one-half have attention-deficit hyperactivity disorder (ADHD). Several other disorders occur at higher rates in association with mental retardation: pica, stereotypy/habit disorder, cluttering, stuttering, and the other Axis II disorders (autistic disorder, pervasive developmental disorder not otherwise specified, and language and speech disorders).

It is clear that depression can be a complication of mental retardation, for example, in response to extra burdens, poor self-image, and stigma. However, individuals with mental retardation may also have unipolar or bipolar mood disorders and anxiety disorders.

Family reactions may include parental disappointment, anger, guilt, over protectiveness, infantilization, over involvement, or detachment.

Current data on the course of mental retardation reflect varying degrees of agressivity in rehabilitative efforts.

Adaptive capacities may be judged by many means, including standardized instruments for assessing social maturity and adaptive skills. Information provided by a semistructured interview of a parent or caregiver yields a multidimensional measure of adaptive behaviors in five "domains": communication, daily living skills, socialization, motor skills, and maladaptive behaviors. Age-dependent expected competency scores of adaptive skills are established for children from infancy to age 18 years with different levels of mental retardation.

Medical evaluation includes physical examination (seeking physical stigmata) and laboratory tests, including chromosome studies. Neurological evaluation, including EEG and computed tomography (CT) scan, seeks possible treatable causes of mental retardation, seizure disorders, deafness, and blindness.

Hyperactivity, aggression, sadness, lack of enthusiasm, excessive anxiety, or formal thought disorder are not primary features of mental retardation and should lead to a full psychiatric evaluation, as should a dramatic change in mood, behavior, or level of adaptive functioning. The family can be assessed by interviews and a home visit with respect to the level of stimulation, emotional support, help-seeking abilities, and skills in making decisions and planning for the future.

TREATMENT

For treatment of the multiple handicaps and complications commonly associated with mental retardation, multimodal treatment with a developmental orientation is optimal. The specifically psychiatric component includes the coordination of medical and; psychiatric evaluations, parent guidance (support, education, behavior management, educational and environmental planning, long-term monitoring, and advocacy), and the usual variety of psychiatric therapist.

Specialized professionals may provide educational and developmental training to enhance speech and language, motor cognitive, social and occupational functioning and adaptive skills such as toilet training, dressing and grooming, and eating.

Developmentally oriented psychotherapeutic interventions may be effective for crisis management or long-term psychosocial goals. For certain adolescents with mild mental retardation, psychotherapy may promote self-esteem, identity formation, social development, emotional and behavioral control, appropriate expression of emotions, and ability to set realistic goals. Technical modifications include specific goals; positive reinforcement; brief, clear verbalizations; supportive limit setting; focus on current events and feelings; reinforcement of reality-fantasy differentiation; and teaching about the nature of emotional life.

PERVASIVE DEVELOPMENTAL DISORDERS

Pervasive developmental disorders (PDD) are divided into the more severe autistic disorder (formerly called infantile autistic and pervasive developmental disorder not otherwise specified (PDD NOS) to accommodate atypical or less severe cases. In both, development is not merely delayed, but also "atypical" or deviant". PDD is not simply a collection of delays in specific skills or domains, but implies broad disruption of functions. This majority of these individuals also have mental retardation.

AUTISTIC DISORDER

Clinical description. The detailed DSM-Icriteria for autistic disorder focus on severe impairment, relative to chronological and mental age, in three key areas:

- Reciprocal social interaction
- Verbal and nonverbal communication and imaginative —activities
- Repertoire of activities and interests

Diagnostic criteria for 299.00 Autistic Disorder

A. A total of 6 (or more) items from (1), (2) and (30 with at least two from (1) and one each from (2) and (3):

(1) Qualitative impairment in social interaction, as manifested by at least two of the following:

(a) marked impairment in the use of multiple nonverbal behaviors such as eye to eye gaze, facial expression, body posture and gestures to regulate social interaction.

(b) Failure to develop peer relationship appropriate to developmental level.

(c) A lack of spontaneous seeking to share enjoyment, interests, or achievement to other people (e.g., by a lack of showing, bringing or pointing out object of interest).

(d) Lack of social and emotional reciprocity.

(2) Qualitative impairments in communication as manifested by at least one of the following:

(a) delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime.

(b) in individuals with adequate speech, marked impairment in the ability to initiate or sustained a conversation with others.

(c) Stereotyped and repetitive use of language or idiosyncratic language.

(d) Lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level.

(3) Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:

(a) encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus

b) apparently inflexible adherence to specific, nonfunctional routines or rituals.

(c) Stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole-body movements)

(d) Persistent preoccupation with parts of objects.

B. Delays or abnormal functioning in at least one of the following areas, with onset prior to age ,3. years: (1) social interaction, (2) language as used in social communication, or (3) symbolic or imaginative play.

C. The disturbance is not better accounted for by Rett's Disorder or Childhood Disintegrative Disorder.

Autistic disorder presents in a wide spectrum of severity:

- Socially, these individuals appear passive and aloof, and initially avoid social contacts, but can come to enjoy and seek interpersonal experiences.
- They show persistent deficits in appreciating the feelings and thoughts of other people and in understanding the process and nuances of social communications. Communicative speech and gesturing are limited or difficult to understand due to echolalia, pronoun reversals, and idiosyncratic meanings. Phonological (sound production) and syntactic (grammar) functions may be relatively spared, with more significant impairments of semantics (sociocultural meanings) and pragmatics (rules of interpersonal exchange), and nonverbal aspects of communication. Imaginative and symbolic function (e.g., use of toys in play) may be deeply affected.
- Cognitive deficits include impairments in abstraction, sequencing, and integration.
- There may be distorted perception of smell, taste, or touch, and underdevelopment of visual and auditory processing.
- Rituals, stereotypies (rocking, whirling), self-stimulation, self-mutilation, and unusual mannerisms are common.
- Affective responses may be "shallow," overly responsive to small changes, oblivious to large changes in the environment, and unpredictably labile and odd.
- The majority of individuals with autistic disorder show subnormal intelligence, but a few show significant improvement in measured IQ during development or treatment.

- Unusual or special capacities (savant skills) in music, drawing, arithmetic, or calendar calculation may be present.
- Autistic disorder is not associated with delusions, hallucinations, or loose associations.

Epidemiology. The most recent prevalence estimate, based on strict DSM-IV criteria for autism, is 4 per 10,000 in the general population. PDD NOS may occur at a rate of 10-20 per 10,000. There is a male predominance of 3—4:1, and no correlation with socioeconomic class.

Etiology

Genetic and biological factors appear to predominate.

- There is no evidence that psychosocial factors or parenting abnormalities cause autistic disorder.

The prevalence of autism in siblings is 4.5%, and autism is 215 times more frequent among siblings born after an autistic child than in the general population.

There islirfeievated incidence of early developmental problems such as:

- postnatal neurological infections,'
- congenital rubella,
- phenylketomiria.

Seizure disorders appear in 35-50% of patients by age 20 years.

CT scan showchanges (ventricular enlargement, left temporal abnormalities, abnormal symmetry)

MRI shows developmental hypoplasia of two discrete areas in the cerebellum and forebrain morphological abnormalities.

Neuropsychological testing shows numerous abnormalities. Forty percent of autistic patients appear to have specific [autoantibodies to serotonin-IA receptors, which are not present in brain-damaged or normal patients.

Various ofOtDtrfffnune disorders are over represented.

COURSE AND PROGNOSIS.

Autistic disorder is often apparent in early infancy, and parents may seek a medical opinion during the 1st year (often suspecting deafness)A

In some patients, the disorder is not apparent until after age 3 years. An initial period of apparently normal development may be followed by developmental arrest or by regression, with loss of previously developed abilities.

In most cases, there is gradual but erratic improvement. There may be episodes of regression during medical illness, environmental stress, and especially at puberty, and periods of rapid developmental progress unexplained by environmental factors.

More severely autistic persons can learn some adaptive skills.

For the less severely impaired, treatment may yield social skills and adaptations that permit employment and independent or group-home living. Predictors of good adaptive outcome are: higher IQ, better language skills, greater social and communicative skills, and later onset.

As adults, autistic individuals continue to show a gradual clearing of symptoms, but retain clinical evidence of residual organic brain damage. Depending on the severity of the autistic disorder, perhaps 2—15% achieve a nonretarded level of cognitive and adaptive function.

Adults:

- remain socially aloof, and often oppositional.
- expressive and receptive language may become normal.

"Obsessional" symptoms persist, including stereotyped pacing, rocking, perseveration, and stuttering.

-There are no delusions or hallucinations.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

-a workup of autistic disorder includes assessment of language, cognition, social skills, and adaptive functioning.

-neurological examination evaluates possible inborn metabolic and degenerative diseases. Screening for phenylketonuria,) is probably cost-effective. An EEG (for seizure disorder) and a chromosome analysis (for fragile_X) may be advised,

-audiological examination for possible deafness and examinations for other sensory deficits may be obtained.

- evaluation of intellectual potential must be done by a psychologist experienced with these difficult youngsters.

Differential diagnosis includes:

- congenital deafness (though deaf children typically learn an alternative lip or sign language, lose their isolative behaviors, and develop sensitive expressive communication),
- congenital blindness (though blind children relate socially),
- developmental expressive and receptive language disorders (though these children are typically more sociable communicate well in gestures),
- juvenile-onset schizophrenia (distinguished by hallucinations, delusions, and thought disorder).
- Children with pure mental retardation have a more even pattern of delays and do not have bizarre behaviors or deficits in social relatedness.
- In very young children, reactive attachment disorder may be a consideration.
- Degenerative diseases may resemble PDD.

Children or adults with autistic disorder may also have mood disorders, anxiety disorders, or symptoms of over activity, inattention, and impulsivity (the diagnosis of ADHD is excluded in the presence of PDD).

Treatment.

-multimodal treatment may have slow but dramatic improvement.

-the milieu should be highly structured and include: special education, vocational training, and teaching of adaptive skills.

-behavior therapy:

- may help control unwanted symptoms;

- promote speech, social interaction, and assertiveness;
- increase self-reliance;
- and facilitate exploration, sometimes, residential treatment is needed.

Low dosages of nonsedating neuroleptics, in conjunction with a highly structured treatment program may be helpful in:

- controlling behavior symptoms,
- reducing excessive activity levels,
- enhancing the effect of behavior therapy.

Psychostimulant medication may decrease symptoms of impulsivity, over activity, and distractibility. The serotonergic agent fenfluramine is usually unhelpful, but a small subgroup of patients may show a short-term reduction in hyperactivity and stereotypic behaviors.

Preliminary reports suggest that an opiate-receptor blocking agent (naltrexone) may improve affective availability, promote social reciprocity, and reduce stereotyped motor and self-injurious behaviors.

Buspirone and beta-blocking agents have been suggested to address aggression in autistic persons.

Rett's Disorder See attached DSM IV criteria.

Diagnostic criteria for Rett's Disorder

All of the following: apparently normal prenatal and perinatal development

apparently normal psychomotor development through the first 5 months after birth normal head circumference at birth

Onset of all of the following after the period of normal development: deceleration of head grwth between ages 5 and 48 months

loss of previously acquired purposeful hand skills between ages 5 and 30 months with the subsequent development of stereotyped hand movements (e. g., hand-wringing or hand washing)

loss of social engagement early in the course (although often social interaction develops later) appearance of poorly coordinated gait or trunk movements

severely impaired expressive and receptive language development with severe psychomotor retardation

See attached DSM IV criteria.

Diagnostic criteria for Asperger's Disorder

A. Qualitative impairment in social interaction, as manifested by at least two of the following: (1)marked impairment in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body postures, and gestures to regulate social interaction

(2)failure to develop peer relationships appropriate to developmental level

(3)a lack of spontaneous seeking to share enjoiment, interests, or archievements with other people (e. g., by a lack of showing, bringing, or pointing out objects of interest to other people) lack of social or emotional reciprocity

B. Restricted repetitive and stereotyped patterns of behavior, interests, and activities, as manifested by at least one of the following:

(1)encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus

(2)apparently inflexible adherence to specific, nonfunctional routines or rituals

(3)stereotyped and repetitive motor mannerisms (e. g., hand or finger flapping or twisting, or complex whole-body-movements) persistent preoccupation with parts of objects

C. The disturbance causes clinically significant impairment in social, occupational, or other important areas of functioning.

D. There is no clinically significant general delay in language (e. g., single words used by age 2 years, communicative phrases used by age 3 years).

E. There is no clinically significant delay in cognitive development or in the development of age- appropriate self-help skills, adaptive behavior (other than in social interaction), and curiosity about the environment in childhood.

F. Criteria are not met for another specific Pervasive Developmental Disorder or Schizophrenia.

SPECIFIC DEVELOPMENTAL DISORDERS

The specific developmental disorders are a series of learning disorders, each affecting a specific domain. The notion of a "specific" developmental disorder implies that a particular area of functioning is impaired relative to general intelligence. For certain conditions, the diagnosis requires a measurement of IQ and a demonstration of specific impairment relative to IQ. ~

EPIDEMIOLOGY

Estimated prevalence of each of the specific developmental disorders in elementary school-age children ranges from 2 to 10%.

ETIOLOGY

Lower socioeconomic classes are over represented in the specific developmental disorders

- There is a male predominance of 3—4:1, with the notable exception of developmental receptive language disorder, where there is no gender difference.

Each specific developmental disorder is probably an etiologically heterogeneous group.

Etiology is presumably related to cortical deficits, attributed to organic damage, delayed maturation, or genetics.

Frequent comorbidity with other specific developmental disorders suggests that these impairments reflect multiple cerebral dysfunctions Numerous theories regarding hemisphere development and specialization have been proposed to explain the developmental disorders.

- A study comparing strongly left-handed and right-handed people demonstrated that left-handers (and their relatives) have more dyslexia, stuttering, and immune disorders, supporting a speculation that a perinatal testosterone spurt may alter development of the brain and other body functions.

Family psychiatric histories show an overrepresentation of reading, speech, and language disorders in the siblings and parents of affected children. The concordance rate in identical twins approaches 100%.

Maternal smoking, low birth weight, prenatal and perinatal mishaps, overt neurological disorders, and EEG abnormalities appear to be associated with the appearance of developmental reading disorder.

LEARNING DISORDER'S (ACADEMIC SKILLS DISORDERS)

Clinical description. DSM-IV criteria for academic skills disorders require skills in the specified academic area to be markedly below the level expected for IQ (as measured by individually administered standardized tests) and for exposure to education. Academic or adaptive impairment is required and cannot be secondary to a sensory defect or known neurologic disorder. In addition, left-right orientation, sound discrimination, and perceptual- motor skills are often impaired in academic skills disorders. Attention difficulties are common, as are unevenness in social skills and emotional development.

COURSE AND PROGNOSIS.

Diagnosis of academic skills disorders is often made initially during grade school. In later school years, problems involving organizational skills (note taking, time management, and maintenance of books and papers) may be signs of cortical deficits, even if basic skills

were well remedied. In college and graduate school, individuals may have difficulty in learning foreign languages, writing efficiently, or reading for fun.

Academic skills disorders are commonly associated with psychological complications, but there is also an overrepresentation of Axis I disorders in the families of patients with these disorders.

Complications include:

- - low self-esteem (“feeling stupid”),

poor frustration tolerance,

- „ lack of enjoyment in learning,

passivity, rigidity in new learning situations, truancy, and school dropout.

Disruptive behavior disorder may also be a complication

After school years, weakness in certain academic skills is not socially stigmatic and may not be a direct source of personal distress. Many adults make accommodations in their lives and work to manage a residue of dysfunctions that were more evident during school years.

EVALUATION AND DIFFERENTIAL DIAGNOSIS Evaluation of academic skills disorders includes:

assessment of a full range of abilities (specific academic skills, speech and language,

and motor function),

cognitive tests (including IQ),

observation of the child's behavior in the classroom.

It is also essential to evaluate the child for possible: mental retardation,

ADHD, mood disorder (causing low motivation), anxiety

disorder (causing reduced attention), and other psychiatric and neurological disorders. Sensory perception tests are needed to rule out impairments of vision or hearing. Concomitant conduct disorder is common.

Treatment.

Treatment for specific developmental disorders in public schools is mandated by law.

In practice, remediation of only basic skills is funded, and deficits must be severe to qualify (e.g., 2 years behind expected level). An "individual educational plan" (IEP) is designed for each child, but the quality of evaluation and treatment services is variable. Part- time resource rooms, full-time self-contained classrooms, and mainstream classrooms (with special-education consultants) provide the major part of special education. Sometimes, specialized schools and residential treatment programs are employed. Depending on the type of disorder, a child might be encourged to:

- make use of a calculator, typewriter, or word processor; be permitted to take time-extended tests;

be tutored individually or in a small group;

use self-paced programmed texts or computerized self-instruction.

Behavior techniques are often used to emphasize success, foster enjoyment of new skills, develop pride, reduce rigidity in general learning, and promote application in new situations.

Parents of children with specific developmental disorders should be included in educational planning. Supportive counseling may help them to avoid contributing to a climate of negativity and criticism and to adjust their child's and their own expectations to anticipate slower-than-standard learning, but (unlike mental retardation) with no clear "ceiling" on educational outcome.

Individual psychotherapy may be useful in reducing secondary psychological complications such as low self-esteem, temper tantrums, lack of assertiveness, and inflexibility.

Psychopharmacological therapy is not helpful for specific developmental disorders, but may be for concurrent disorders.

LANGUAGE AND SPEECH DISORDERS

Clinical description (according with ICD 10). Language and speech disorders are defined by delays in specific speech or language abilities that interfere with academic or adaptive functioning, and that are not due to PDD, mental retardation, sensory deficit, or structural or neurological disorder.

There is a continuum of severity of impairment from developmental articulation disorder, through developmental expressive language disorder, to developmental receptive language disorder.

In developmental receptive language disorder, both decoding (comprehension) and encoding (expression) are impaired, leading to more academic and social disruption. Multiple cortical deficits are usually observed, including sensory, integrative, recall, and sequencing functions. In adolescence, social awkwardness, stereotypies, resistance to change, and low frustration tolerance may approach the severity of autistic disorder, but with better social skills, environmental awareness, and abstraction. Nonverbal comprehension may be preserved or disrupted.

Epidemiology.

Communication problems are seen in 7—12% of the general population and are even more common in people with psychiatric disorders, mental retardation, or hearing impairment.

Among child psychiatric patients, about 25% have language and speech disorders. Males outnumber females 3—4:1 among patients with developmental expressive language disorder

Etiology.

By age 5 years, children are expected to speak fluently and to comprehend speech. Adult articulation skills should be present by age 8 years.

Speech and language are highly dependent on the linguistic characteristics of the environment and may interact with other environmental factors in influencing development and adult skills.

In social interactions, verbal and nonverbal communication include:

-word finding (access and retrieval of verbal information),

-word relationships (semantics),

- sentence formation (syntax),

- giving and receiving feedback,

- following conversational structure and flow,

- responding to the context,

- adapting to meanings and external events (pragmatics),

- responding to one own internal sense of events,

- monitoring one's own communicative productions (metalinguistic skills).

The etiology of deficits is often unknown, but contributing factors may include:

faulty speech models within the family, lack of stimulation of language, neurocortical deficits.

Hearing is crucial in the development of speech and language, and hearing loss plays a significant role in the etiology of the language and speech disorders. Even a mild hearing loss (25—40 decibels) (resulting from chronic otitis media or perforation of the tympanic membrane) may delay development of articulation, expressive and receptive language, reading, and spelling.

COURSE AND PROGNOSIS.

Deficits in articulation (speech sound production), expression (oral language production and use), and reception (comprehension) may be observable by ages 2—3 years. Delays in speech and language frequently improve during development, so that early delays are not strongly predictive of subsequent learning disorders Complications of language and speech disorders include:

- progressive academic impairment,
- psychological distress,
- low self-esteem,
- rigidity regarding learning,
- school dropout.

These children may have difficulty maintaining a conversation or expanding on a topic. About 50% of children with language and speech disorders have concomitant Axis I diagnoses, and another 20% have other developmental disorders, especially academic skills disorders.

EVALUATION AND DIFFERENTIAL DIAGNOSIS.

Specialized language and speech evaluation includes articulation, receptive skills (understanding single words, word combinations, and sentences), and expressive language skills (syntactic structures, vocabulary, and social appropriateness) that are compared with developmental norms.

Family characteristics and free speech between parents and child may be observed, assessing social skills and nonverbal communication (vocalizations, gestures, and gazes).

Hearing acuity should be tested by audiometry or by auditory evoked response (which does not require the child's cooperation). Auditory attention (losing flow of conversation, inability to hear in a crowd, distractibility), discrimination, and memory are evaluated. Differential diagnosis includes:

- pervasive developmental disorder,
- elective mutism,
- deafness,
- mental retardation,
- aphasia

Treatment.

Deficits in hearing should be addressed, whenever possible, and aggressive treatment of otitis media is prudent.

Social involvement, imitation, and imaginative play are encouraged to increase verbal, communicative, and symbolic skills.

- Referral to a speech and language pathologist is essential.

Special education should be maintained until symptoms improve. Once a child is mainstreamed speech and language therapy and supplemental academic supports may still be required.

- Psychiatric treatment for concurrent attention and behavior problems and educational management of academic skills disorders may be involved.

MOTOR SKILLS DISORDER

Developmental coordination disorder refers to impaired learning of motor skills, sufficient to cause functional impairment, and not due to a known physical disorder. About 5% of children have significant impairments of gross or fine motor functions, which are manifest in running, throwing a ball, buttoning, holding a pencil, or general awkwardness and clumsiness. ADHD is commonly also present.

Frequent complications include scapegoating, sports avoidance, and vocational impairment. Because absence of athletic ability or competent performance of fine motor tasks may lead to impaired peer relations, academic impairment (via slow and illegible handwriting), low self-esteem, and even economic handicap as an adult, remediation is warranted.

Reference:

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7. SPECIAL CLINICAL CIRCUMSTANCES EMERGENCIES

EMERGENCIES

ASSESSMENT AND TRIAGE

Emergencies present most often in psychiatric, pediatric, or general hospital emergency rooms, but they may be seen in a school, psychiatric, or pediatric outpatient setting. Rapid clinical assessment emphasizes the potential for physical danger or acute psychiatric deterioration. Support systems are assessed, treatment may be initiated, and a disposition is made for further evaluation and treatment.

An emergency evaluation must be brief and focused. It is useful to talk with the child or adolescent and the relevant adults alone and together. Important outside information can be gathered by telephone, from as many informants as possible. To ensure patient and staff safety in the emergency room, assess immediately for:

- Acute medical illness
- Head trauma
- Suicidal overdose
- Drug or alcohol intoxication
- Drug or alcohol withdrawal
- Need for physical restraint to prevent aggression or leaving the emergency room
- Need to prevent a parent from removing the child from the emergency room
- Drugs or weapons with which the patient could hurt himself or herself or others

A careful mental status examination is crucial in an emergency situation, with special focus on signs of psychosis, organicity, intoxication, suicidality, poor judgment, or impulsivity. The cause of a sudden, drastic change in behavior should be considered organic until proven otherwise. "Medical clearance" does not guarantee the absence of physical disorder.

It is often difficult to make a definitive DSM-IV diagnosis in a single evaluation in an emergency setting, especially if collateral information is limited. Often only a triage decision regarding further evaluation and treatment is possible. The decision to hospitalize a child or adolescent is often determined by the inability of supervising adults to tolerate the young person's behavior or to ensure safety.

SUICIDE

If a child or adolescent is seen after a suicide attempt, a detailed evaluation should be made of the circumstances preceding and following the attempt, history of substance abuse or impulsive behavior, wishes to die or to influence others at the time of the attempt, whether a friend or family member has committed suicide, and coping skills and supports in the patient and family.

Frequent precipitants of adolescent suicide attempts are arguments with parents, breaking up with a boyfriend or girlfriend, and/or failing at school.

All suicide attempts should be taken seriously as a communication of desperation and an indication of limited problem-solving skills.

A brief hospitalization may be useful even for the youth who denies continuing suicidality, to complete a more detailed assessment and begin the treatment process.